

## In Reply: SARS-CoV-2 Vaccination Related, Pediatric Guillain-Barre Syndrome Requires the Same Management as in Adults

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Thank you for your comments. We have reviewed your response letter and have the following comments.

We are fully aware that no cerebrospinal fluid (CSF) investigations were carried out, and it has been stated in the case illustration as well. However, according to the National Institute of Neurological Disorders and Stroke which is also restated in Consensus Statement by Leonhard *et al.* [1] and seminar article by Shahrizaila *et al.* [2], features required for the diagnosis of Guillain-Barre Syndrome (GBS) are progressive bilateral weakness along with absent or decreased tendon reflexes in affected limbs – both which are clearly found in this case. Electrodiagnostic features along with albumino-cytological dissociation in CSF are both features that strongly support the diagnosis of GBS. It is also clearly stated in the consensus that CSF examination is mainly used to rule out causes of weakness other than GBS and should be performed during the initial evaluation of the patient.

In our case, patient presented to the neurophysiologic clinic 3 weeks after the onset of limb weakness, which is no longer in the initial phase of GBS. If CSF examination were carried out in the late phase, the features of albumino-cytological dissociation will only be seen in smaller percentage of the case, and normal CSF protein levels would not actually rule out the diagnosis of GBS [1,3]. On the other hand, electrodiagnostic studies reveal a sensorimotor polyradiculoneuropathy, indicated by reduced sensorymotor conduction velocities, abnormal temporal dispersion, and prolongation of F-wave as shown in Figure 1. Electrodiagnostic study – unlike CSF examination – are

more likely to show abnormalities 2–3 weeks after onset and are most likely normal in the acute settings [1]. Therefore, in this case, electrodiagnostic study is a more suitable ancillary investigation to support the diagnosis of GBS.

For the diagnosis of acute inflammatory demyelinating polyneuropathy, we used the Hadden *et al.* [4], Meulstee *et al.* [5], and Kashnathan *et al.* [6] electrophysiological criteria for the diagnosis of childhood GBS. It is true that in Acute Motor Axonal Neuropathy, distal latency can be prolonged as well. However it is unlikely in this case, according to the Hadden and Dutch GBS electrodiagnostic criteria. And, no – we do not use our own reference limits for nerve conduction studies parameters, but according to the electrodiagnostic criteria and consensus guideline in making analysis.

The pathophysiological explanation of how vaccination trigger GBS is a theoretically possible mechanism and there were no statement that there is a clear evidence that SARS-CoV-2 vaccines trigger a viral infection.

The sentence ‘vaccination outweighs the risk of contracting the virus’ was made based on previous reports that SARS-Cov-2 infection have a higher probability of inducing GBS than vaccine itself (which was also stated in the article).

We never say that there were only five pediatric patients with SARS-CoV-2 vaccination associated GBS have been reported. What we stated was ‘At least two from five previous report have highlighted the development of GBS after CoronaVac vaccine’. This sentence clearly shows that there weren’t only five cases, and that we reported only the cases associated with CoronaVac (and not all SARS-CoV-2 vaccination) at the time the article was written.

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■ **Conflicts of Interest**

No potential conflict of interest relevant to this article was reported.

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**REFERENCES**

1. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. *Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol* 2019;15:671-683.
2. Shahrizaila N, Lehmann HC, Kuwabara S. *Guillain-Barré syndrome. Lancet* 2021;397:1214-1228.
3. Wong AH, Umapathi T, Nishimoto Y, Wang YZ, Chan YC, Yuki N. *Cytoalbuminologic dissociation in Asian patients with Guillain-Barré and Miller Fisher syndromes. J Peripher Nerv Syst* 2015;20:47-51.
4. Hadden RD, Cornblath DR, Hughes RA, Zielasek J, Hartung HP, Toyka KV, et al. *Electrophysiological classification of Guillain-Barré syndrome: clinical associations and outcome. Plasma Exchange/Sandoglobulin Guillain-Barré Syndrome Trial Group. Ann Neurol* 1998;44:780-788.
5. Meulstee J, van der Meché FG. *Electrodiagnostic criteria for polyneuropathy and demyelination: application in 135 patients with Guillain-Barré syndrome. Dutch Guillain-Barré Study Group. J Neurol Neurosurg Psychiatry* 1995;59:482-486.
6. Kasinathan A, Saini AG, Suthar R, Saini L, Sahu JK, Singhi P, et al. *Comparison of five different electrophysiological criteria for childhood Guillain Barre syndrome. Ann Indian Acad Neurol* 2021;24:542-546.