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Before Attributing Polyradiculitis to SARS-CoV-2 Vaccination, All Other Potential Triggers Must Be Ruled Out

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LETTER TO THE EDITOR

We read with interest Zhu. et al's article on a 58year-old male who developed Guillain-Barre syndrome (GBS), subtype acute, inflammatory demyelinating polyneuropathy (AIDP), one week after diarrhoea for two days and 55 days after the second dose of a SARS-CoV-2 vaccination [Zhu, C. et al., 2024]. His medical history was positive for polyradiculitis at the of age 18, which completely disappeared [Zhu, C. et al., 2024]. The patient had progressive quadriparesis and dysarthria on admission. and respiratory insufficiency and cardiac arrest 12 hours after admission, requiring resuscitation, intubation, and mechanical ventilation [Zhu, C. et al., 2024]. GBS was diagnosed three days after admission and treated with six cycles of immunoglobulins (IVIG), three cycles of plasmapheresis, and glucocorticoids, resulting in incomplete recovery at 13 months [Zhu, C. et al., 2024]. The study is compelling but some points require discussion.

The first point is that the causal relationship between SARS-CoV-2 vaccination and GBS in the index patient remains unproven [Zhu, C. *et al.*, 2024]. Since the latency period between the second vaccination and the onset of GBS was 55 days, a causal relationship is unlikely.

The second point is that alternative causes of GBS have not been sufficiently ruled out. Since the patient suffered from diarrhea a week before the onset of GBS, it would have been imperative to exclude a bacterial or viral gastrointestinal infection, particularly Campylobacter jejuni, which remains the most common cause of GBS [Finsterer, J. *et al.*, 2022]. Other common tiggers that should have been excluded include Epstein–Barr virus (EBV), cytomegalovirus (CMV), influenza virus, Zika virus, dengue virus, and mycoplasma pneurmoniae [Finsterer, J. *et al.*, 2022].

The third point is that the latency between the onset of symptoms and the diagnosis of GBS was

three days. Why wasn't GBS suspected upon admission or at least during first 12 hours after hospitalisation? The longer the latency between the onset of symptoms and the start of treatment, the longer the time to recovery.

The fourth point is that the conclusions are not justified [Zhu, C. *et al.*, 2024]. It cannot be concluded from an individual case that the interval between SARS-CoV-2 doses should be increased in patients with a history of GBS [Zhu, C. *et al.*, 2024]. Appropriately designed studies are needed to draw general conclusions on the interval between SARS-CoV-2 vaccine doses in patients with a history of immunological disease.

Other points that should be addressed are that the vaccine brand should be specified, an explanation should be given why methylprednisolone was administered, which is known that it is ineffective in GBS, whether cardiorespiratory insufficiency was due to Takotsubo syndrome, and whether the respiratory failure caused ventricular tachycardia, ventricular fibrillation, or asystole.

In summary, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before the relapse of GBS can be attributed to the SARS-CoV-2 vaccination, alternative triggers of GBS must be thoroughly ruled out. Treatment for GBS should be started as soon as suspicion is raised.

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