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LETTER TO THE EDITOR

Nerve conduction studies support the classification of SARS-CoV-2 associated Guillain-Barre subtypes

Letter to the Editor

We read with interest the article by da Silva et al. about a 62-year-old female who developed paresthesias of the distal limbs 18 days after the first dose of AstraZeneca's vaccine for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] The patient developed weakness of the lower and upper limbs 2 and 3 weeks later, respectively,[1] and was admitted to hospital 2 months after the onset of these symptoms because of facial diplegia and quadriparesis, dysphagia, and absent tendon reflexes.[1] The patient was diagnosed with Guillain–Barre syndrome (GBS), with a score of 2 according to the Brighton criteria based on the clinical presentation and the dissociation cyto-albuminique in the cerebrospinal fluid (CSF).[1] The patient only partially recovered with administration of immunoglobulins.[1] The study is appealing but raises concerns that need to be discussed.

We disagree with the conclusion that there is only a "temporal association" between SARS-CoV-2 vaccination and GBS. Arguments that speak for not only a temporal but also a causal connection are that GBS occurs not only after vaccinations against but also after infection with SARS-CoV-2; that, in addition to GBS, other neuro-immunological disorders can also occur after SARS-CoV-2 infections or vaccinations; that no other triggers of GBS, such as Campylobacter jejuni, cytomegalovirus, influenza, Mycoplasma pneumoniae, flaviviruses, or alpha-viruses, have been identified in patients with SARS-CoV-2-associated GBS; that GBS can be associated with other neuro-immunological diseases, such as myasthenia[2]; that vaccinations for H1N1 or influenza can also occasionally trigger GBS[1]; and that cytokines and chemokines are increased in the CSF of patients with SARS-CoV-2-associated GBS.[3]

We disagree that only a few cases of SARS-CoV-2-vaccination-associated GBS (SC2VaG) have been reported.[1] In a recent review of the neurological side effects of SARS-CoV-2 vaccines, 300 cases of SC2VaG were described.[4]

We should be told why it took 2 months from the onset of GBS for the patient to be diagnosed with SC2VaG. The sooner immunoglobulins or plasmapheresis are initiated in patients with GBS, the better the outcome. The 2-month delay between onset of symptoms and administration of immunoglobulins may have contributed to the index patient's outcome.

We should know why cytokines (interleukin [IL]-6, IL-8, IL-1a, tumour necrosis factor-α), chemokines, or glial markers were not determined in the CSF to document the post-vaccination cytokine storm.

A shortcoming of the study is that no nerve conduction studies (NCSs) were performed. NCSs are crucial for the classification of GBS subtypes. Determining the subtype of GBS is critical because treatment and outcomes can vary between axonal and demyelinating GBS.

The GBS subtype in the index patient was apparently GBS with polyneuritis cranialis. We should know whether only cranial nerves 7, 9, and 10 were affected or other cranial nerves as well.

Overall, this interesting study has some limitations that call into question the results and their interpretation. There are more arguments for than against a causal relationship between SARS-CoV-2 vaccinations and GBS. For this reason, GBS can be considered a definite complication of SARS-CoV-2 vaccinations.

KEYWORDS

COVID-19, neuropathy, polyradiculitis, SARS-CoV-2

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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AUTHOR CONTRIBUTION

JF: design, literature search, discussion, first draft, critical comments. Informed consent was obtained. The study was approved by the institutional review board.

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REFERENCES

1. da Silva GF, da Silva CF, Oliveira R, Romancini F, Mendes RM, Locks A, et al. Guillain-Barré syndrome after coronavirus disease 2019 vaccine: a temporal association. Clin Exp Neuroimmunol. 2021. https://doi.org/10.1111/cen3.12678
2. Yuan J, Zhang J, Zhang B, Hu W. The clinical features of patients concurrent with Guillain-Barre syndrome and myasthenia gravis. Neurosciences (Riyadh). 2018;23(1):66–70. https://doi.org/10.17712/nsj.2018.1.20170209
3. Gigli GL, Vogrig A, Nilo A, Fabris M, Biasotto A, Curcio F, et al. HLA and immunological features of SARS-CoV-2-induced Guillain-Barré syndrome. Neurol Sci. 2020;41(12):3391–4. https://doi.org/10.1007/s10072-020-04787-7
4. Finsterer J. Neurological side effects of SARS-CoV-2 vaccinations. Acta Neurol Scand. 2022;145(1):5–9. https://doi.org/10.1111/ane.13550

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