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Letter to the Editor

Not only Polyneuritis Cranialis is in Need of Treatment, but also the Infection that Causes it

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Letter to the Editor

We read with interest the article by Memarian *et al*. on a 9-year-old boy with Guillain-Bare syndrome (GBS), polyneuritis cranialis (PNC) subtype, one week after a bout of influenza and bloody diarrhea two days before admission [1]. He presented with ptosis, ophthalmoplegia, facial paralysis, masseter and temporalis muscle weakness, a reduced gag reflex and nasal speech [1]. The patient benefited significantly from intravenous immunoglobulins (IVIGs) and made a full recovery at one-month follow-up [1]. The study is excellent, but some issues need to be clarified.

The first point is that the causative infectious agent was not specified [1]. The patient had a flu-like infection and bloody diarrhoea, but the infectious agents causing these infections were not specified [1]. GBS is usually due to a gastrointestinal or pulmonary infection or a vaccination. Infectious agents that can cause GBS include viruses, bacteria and protozoa. The most common pathogen is Campylobacter jejuni [2]. Other infectious agents that cause GBS are Haemophilus influenza, Mycoplasma pneumoniae, SARS-CoV-2 virus, cytomegalovirus, Zika virus, HIV, influenza A and B virus, parainfluenza virus, hepatitis B virus, hepatitis C virus, hepatitis A virus, hepatitis E virus, adenovirus, dengue virus, Epstein-Barr virus, herpes simplex virus, varicella zoster virus, malaria, Leptospira, Tsutsugamushi fever, and syphilis [3]. These pathogens should have been excluded as causes of PNC.

Second, electrophysiological examination revealed acute axonal cranial polyneuropathy involving cranial nerves 3, 4, 5, 6, 7, 9, and 10 [1]. However, only the facial nerve was reported to have been electrophysiologically examined. We should know which cranial nerves, other than the 7th cranial nerve, were accessible for nerve conduction velocity studies and what the detailed results were. What methods were used to examine cranial nerves 3, 4, 5, 6, 9 and 10?

Third, MRI of the cranial nerve roots with contrast medium was not performed [1]. Various reports have shown that enlargement of the cranial nerve roots is a typical finding in the GBS subtype with multicranial nerve involvement [4]. Cranial nerve root thickening may also be present [5].

The fourth point is that the 11th and 12th cranial nerves were not involved [1]. We should know the reason for this. Since both are purely motor nerves, one would expect these two nerves to be particularly involved in GBS.

In Table 1, the headings of the “Results” and “Normal Range” columns have been swapped [1].

In summary, this interesting study has limitations that qualify the results and their interpretation. Addressing these limitations could strengthen the conclusions and reinforce the study’s message. All outstanding questions should be clarified before readers uncritically accept the study’s conclusions. In patients with GBS, subtype PNC, the pathogen should be identified to treat not only the GBS but also the underlying infection.

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