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Letter to the Editor

In Order to Apply Specific Anti-Infective Treatment in Neonatal GBS, Identification of the Causative Pathogen is Essential

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Letter to the Editor

We read with interest the article by Pasawal *et al*. about a 24-day-old boy who developed floppy infant syndrome and respiratory failure on day 18 of life, which was attributed to demyelinating Guillain-Barré syndrome (GBS) [1]. The boy made a full recovery after administration of intravenous immunoglobulins (IVIG) [1]. The study is interesting but raises some questions.

Firstly, the trigger for GBS could not be identified [1]. Triggers for GBS include infections, vaccinations, and surgery [1]. Infections can be caused by viruses, bacteria, or protozoa. The most common pathogens that cause GBS include Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae, HCV, HEV, HSV, VZV, SARS-CoV-2, dengue virus, Zika virus, CMV, HIV, HBV, EBV, Plasmodium falciparum, leptospirosis, Orientia tsutsugamushi, Treponema pallidum, and Mycobacterium tuberculosis [2]. Identifying the causative pathogen is crucial, as treating the infection is just as important as treating the neurological disorder.

Second, the increase in IgG antibodies against the SARS-CoV-2 virus does not necessarily mean that the patient was recently infected with SARS-CoV-2. Since IgG antibodies can cross the placenta and be transferred from mother to fetus [3], it is conceivable that the elevated antibody titer in the index patient originated from the mother and not from the patient himself. Had the mother tested positive for SARS-CoV-2 before or during pregnancy?

Third, it is unclear why a hereditary disease was initially suspected [1]. The patient developed normally during the first few days of life and only began to show a deterioration in motor function from day 18 onwards. Reports indicate that the patient was healthy until day 10, when he developed fever, cough, runny nose, and diarrhea [1]. This is a strong evidence that motor dysfunction developed after an infectious disease in an initially healthy individual, suggesting that the muscle weakness was acquired rather than congenital.

The fourth point concerns the fetal stress that necessitated a cesarean section [1]. What specific complications occurred that made this procedure necessary? Was the mother a primiparous woman or had she given birth before? In this context, the Agpar score at birth is missing; was it normal or pathological?

The fifth point concerns the enhancement of the cauda equina. However, since the patient developed tetraparesis and cranial nerve involvement, contrast enhancement of the cervical or cranial nerve roots is to be expected.

The sixth point concerns the missing information on the motor, sensory, or mixed nerves examined. Knowledge of the detailed nerve conduction velocity test results is crucial to assess whether only motor or also sensory fibers were affected by the condition.

Finally, the reference ranges for the blood tests were not provided. Ganglioside antibody titers are also missing. Among the missing cerebrospinal fluid parameters are interleukin-6 and IL-8. IL-18, MCP-1, TNF-alpha, chemokines, glial factors, neurofilament light chain, antibodies against SARS-CoV-2 and a panel for viral particle including SARS-CoV-2.

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