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Letter to the Editor

Rapid Progression of Guillain-Barre Syndrome is not Uncommon and should not Prevent its Diagnosis

Josef Finsterer
Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

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Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Matsubayashi *et al*. about a 36-year-old man with Guillain-Barré syndrome (GBS) of the acute motor axonal neuropathy (AMAN) subtype following a previous respiratory tract infection [1]. The case was considered unique because the nadir was reached within 16 hours of onset [1]. The study is worthy of discussion.

First of all, we disagree with the subclassification of GBS as AMAN. The patient complained of dysesthesia, which clearly indicated sensory impairment, although sensory tests and sensory nerve conduction studies (NCS) were described as normal on the 17th day of hospitalization [1]. GBS can manifest not only in large fibers but also in small A-delta and C-fibers. When small fibers are involved, sensory and motor NCS are normal [2].

The second point is that the trigger for GBS has not been specified in detail. GBS is most commonly triggered by Campylobacter jejuni, followed by infections with Haemophilus influenza, Mycoplasma pneumoniae, and the Zika virus [3]. Other, less common triggers of GBS are SARS-CoV-2, HIV, hepatitis-B virus, hepatitis-C virus, hepatitis-A virus, hepatitis-E virus, Epstein-Barr virus, cytomegalovirus, herpes simplex virus, measles virus, enterovirus, influenza-A virus (H1N1), chikungunya virus, parecho virus, Toscana virus, Japanese encephalitis virus, varicella zoster virus, Plasmodium falciparum, Leptospira, Orientia tsutsugamushi, syphilis, and dengue. Non-infectious triggers of GBS include vaccinations (e.g., SARS-CoV-2, influenza, polio, rabies, hepatitis A and B), lymphomas, kidney transplants, intravenous administration of gangliosides, and trauma [3]. Since the distribution of motor, sensory, and autonomic nerve involvement in GBS can depend on the pathogen, the causes of GBS should be specified precisely. It is also helpful to know the trigger, as it may need to be treated itself.

The third point is that the MRI of the spine was described as normal, but it is not specified whether contrast medium was used. An MRI of the spine with contrast medium often shows thickening and enhancement of the spinal roots, which supports the diagnosis of GBS [4].

The fourth point is that it is incomprehensible why it took four days to start intravenous administration of immunoglobulins. The patient showed rapidly progressing flaccid tetraparesis and absent tendon reflexes. Normal findings in the cerebrospinal fluid and NCS on admission do not rule out GBS.

The fifth point is that no long-term results were reported [1]. Given the initial rapid progression and delayed start of treatment, we should know whether this led to a poor outcome or did not prevent a full recovery without sequelae.

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