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Letter to the Editor

Involvement of Cranial and Autonomic Nerves in Guillain-Barré Syndrome Caused by Mycoplasma Pneumoniae should not be Classified as Atypical

Josef Finsterer
Neurology & Neurophysiology Center, Vienna, Austria

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Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Chakrabarti *et al*. regarding a previously healthy 17-year-old female who developed progressive quadriparesis four days after a Mycoplasma pneumoniae respiratory tract infection [1]. Subsequently - 11, 13, and 15 days after the onset of limb muscle weakness - she developed respiratory muscle weakness, facial palsy, and dysautonomia [1]. Guillain-Barré syndrome (GBS) of the acute inflammatory demyelinating polyneuropathy (AIDP) subtype was diagnosed [1] Following repeated administration of intravenous immunoglobulins (IVIG) and plasmapheresis, the patient began to recover gradually 22 days after the onset of quadriparesis [1]. The study is promising, yet certain points warrant discussion.

First, we disagree with the statement that the clinical presentation was atypical [1]. GBS is known for its potentially rapid course and deterioration leading to respiratory insufficiency due to muscle weakness [2]. Moreover, progressive muscle weakness in the lower and upper extremities had already been present for ten days prior to admission, and quadriplegia did not occur until 16 days after the onset of ascending weakness [1]. This pattern corresponds to the typical clinical course of the AIDP subtype of GBS and cannot be classified as "atypical" [3].

Secondly, we disagree with the view that there was an overlap of multiple GBS subtypes [1]. Involvement of cranial nerves and nerves innervating the respiratory muscles is not unusual in AIDP [4] and does not justify classifying the clinical presentation as an overlap of different GBS subtypes. Even dysautonomia can be part of the clinical presentation of AIDP [5]. Only when cranial or autonomic nerves are affected without concurrent involvement of nerves innervating the limb or respiratory muscles should the condition be classified as an isolated cranial nerve subtype or an isolated dysautonomia subtype of GBS.

Thirdly, there was no explanation as to why contrast-enhanced spinal MRI showed contrast enhancement at the anterior and posterior nerve roots of the thoracic and lumbar spine, but not at the cervical nerve roots. Given that the patient also exhibited upper-extremity weakness, contrast enhancement at the cervical nerve roots would also have been expected. Were there signs of recovery in the upper extremities prior to the recovery of the respiratory and leg muscles?

A fourth point concerns the lack of long-term follow-up [1]. Data from follow-up examinations at 6 and 12 months would have been helpful in assessing whether the patient benefited from rehabilitation. Did she fully regain her ability to walk and her independence? What score did she achieve on the modified Rankin Scale after rehabilitation?

A fifth point is that no explanation was provided as to why azithromycin was ineffective against Mycoplasma pneumoniae [1]. Was Mycoplasma pneumoniae also detected in sputum culture, and was azithromycin administered empirically or based on susceptibility testing? Was doxycycline suitable for the pathogen?

In summary, AIDP should not be classified as atypical if cranial nerves and autonomic nerves are also affected. Pneumonia caused by Mycoplasma pneumoniae should be treated based on susceptibility testing rather than empirically.

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Keywords: Guillain-Barre Syndrome, Acute Inflammatory Demyelinating Polyneuropathy, Intravenous Immunoglobulins, Mycoplasma Pneumoniae, Nerve Conduction Studies

References
1. Chakrabarti MP, Sultan R, Ruderfer D, Hickey K. Atypical Presentation of Rapidly Progressive Guillain-Barré Syndrome with Dysautonomia in an Adolescent with Persistent *Mycoplasma pneumoniae* Infection: A Case Report. Case Rep Pediatr, Jun 30, 2026, 5766838. Doi: 10.1155/crpe/5766838
2. Bellanti R, Rinaldi S. Guillain-Barré syndrome: A comprehensive review. Eur J Neurol, Aug 2024; 31(8):e16365. Doi: 10.1111/ene.16365
3. Patnaik AP, Mininni J, Porter NC, Morris NA. A Severe Course of Relapsing-Remitting Acute-Onset Chronic Inflammatory Demyelinating Polyneuropathy in a Young Man. Case Rep Neurol, Feb 4, 2021; 13(1):73-77. Doi: 10.1159/000511956
4. Shibuya K, Tsuneyama A, Misawa S, Suichi T, Suzuki Y, Kojima Y, *et al*. Cranial nerve involvement in typical and atypical chronic inflammatory demyelinating polyneuropathies. Eur J Neurol, Dec 2020; 27(12):2658-2661. Doi: 10.1111/ene.14497
5. Cuciureanu D, Prodan R. Manifestări disautonomice în polineuropatiile demielinizante inflamatorii acute [Autonomic dysfunction in acute inflammatory demyelinating polyneuropathy]. Rev Med Chir Soc Med Nat Iasi, Jan-Mar 2004; 108(1):155-158.