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Case Report

A case of Guillain-Barré syndrome in a 24-year-old man

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ABSTRACT

Guillain-Barré syndrome (GBS) is an acute, immune-mediated neurological disorder that affects approximately 1.2 to 2.3 individuals per 100,000 population annually, with notable regional and seasonal variations. In recent years, an increase in reported cases in certain regions of India has highlighted its emerging public health significance. GBS is characterized by rapid onset of muscle weakness and can progress to severe paralysis if not promptly managed. Early diagnosis and timely therapeutic interventions, such as immunotherapy and supportive care, are essential to improve patient outcomes and reduce the risk of long-term disability. This case report describes a 24-year-old male diagnosed with severe motor axonal polyneuropathy, a variant of GBS associated with more aggressive progression. Following a thorough clinical evaluation and diagnostic investigations, the patient was initiated on plasmapheresis. Currently, his condition is stable, with no further neurological deterioration, indicating a positive response to treatment and ongoing medical management.

Keywords: Autoimmune disease of the nervous system, Autoimmune neuropathy, Guillain-Barré syndrome, Young adult

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute, immune-mediated disorder of the peripheral nervous system characterized by an aberrant immune response that targets peripheral nerves. This response results in muscle weakness, sensory disturbances, and, in severe cases, paralysis.[1] Both motor and sensory nerves may be involved, leading to impaired movement and altered sensations such as pain, temperature, and touch. Symptoms typically develop over hours to weeks and frequently follow a respiratory or gastrointestinal infection. GBS affects individuals of all ages and occurs equally in males and females. Campylobacter jejuni is the most commonly identified antecedent pathogen, usually transmitted through contaminated food or water.[2] Viral infections, including Zika virus, have also been associated with GBS, and in rare cases, vaccinations may act as potential triggers. However, the precise immunopathogenic mechanisms underlying the disease remain incompletely understood. Immune-mediated injury to the myelin sheath or axons disrupts nerve conduction, leading to muscle weakness and sensory loss. Because peripheral nerves supplying the limbs are long, symptoms often begin in the hands and feet and progress proximally in an ascending pattern. GBS was first described by Landry in 1859 and later characterized by Guillain, Barré, and Strohl in 1916.[3] GBS is classified into subtypes, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and Miller-Fisher syndrome (MFS), which presents with ophthalmoplegia,

ataxia, and areflexia. Globally, GBS affects approximately 1.2–2.3 individuals per 100,000 population annually, with recent increases reported in several regions of India, highlighting its growing public health significance.[4]

CASE REPORT

On December 14, 2025, a 24-year-old male patient was admitted to the hospital with the chief complaints of persistent cough accompanied by expectoration that had a whitish appearance for the past four days. Additionally, the patient experienced fever for the past three days, bilateral lower limb weakness for the past two days, and breathlessness for the past one day. Following a comprehensive physical examination and laboratory investigations, the patient was diagnosed with severe motor axonal polyneuropathy (GBS).

Clinical signs

The patient was brought into the hospital with the chief complaints of cough with whitish expectoration consistently 10-15 ml/day, which was non-foul smelling, non-blood-tinted. The patient was also having fever on and off, relieved on taking medicine. The patient has developed weakness, which was insidious in onset, gradually progressed in the bi-lateral lower limbs. On physical examination, the patient's vital signs were: Temp was 102.7 degrees Fahrenheit, Blood pressure was 90/50, SpO2 was 95%, Pulse rate was 108 beats/minute, Respiration was 23 breaths/minute. On general examination, the patient was looking drowsy. However, on systemic examination, CVS - S1, S2 positive, on respiratory system bi-lateral numbness along with wheezing was present, P/A was soft and non-tender. CNS examination shows reflexes are absent (E1VTM1).

Investigation

Relevant investigation was done on the arrival of the patient at the hospital. Hematology results show HB 10.2 g/dl, RBC count 3.64 million/cumm, total leucocytes count 19390 cells, platelet count was 1.00 lakhs/cumm, neutrophil 79%, lymphocytes 12%. Liver function test shows that C-reactive protein 102.14%. Renal function test shows that serum urea 31 mg/dl, serum creatinine 1.0 mg/dl, serum sodium 132 mmol/l, serum potassium 3.6 mmol/l, serum chloride 100 mmol/l. ABG analysis shows pH7.41, PCO2 34.1, PO2 111.3, HCO3 21.3, LAC 0.9. On examination of heart activity, ECG shows sinus tachycardia, abnormal Q wave, ST abnormality at v2, right axis deviation. On CT chest, it shows collapse consolidation with nodular opacities in the basal segment of bi-lateral lobes and dependent regions of right upper lobe. MRI of brain shows no intracranial bleeding, no significant abnormality, MRI cervical spine shows no abnormality, Lumbar region L1-L2 shows mild bulging with L4-L5, L5-S1 mild stenosis, Bi-lateral neural foraminal stenosis, and the patient was on mechanical ventilation with SIMV modes.

Therapeutic intervention

After confirmation of the diagnosis, the patient was prescribed for injection Piptaz 4.5 g IV X OD, Injection Meropenem 1 g/IV × TID, Injection PCT 1 gm × TID, Injection Optineuron 1 ampule in 100 ml NS × OD, Tablet Shelcal HD 500 mg × OD, Infusion Midazolam 42 ml with injection Dexmedetomidine 1 ampule × 5 ml/hour and injection Tigecycline 50 mg × BD. The patient was also receiving over-the-counter IV infusion of NS and RL. Recently, the patient was also started with plasmapheresis procedure and physiotherapy.

DISCUSSION

GBS is a heterogeneous immune-mediated polyradiculoneuropathy with variable clinical presentations, subtypes, and outcomes. The present case describes a young adult male with acute motor axonal neuropathy (AMAN) who developed rapidly progressive bilateral lower limb weakness with respiratory compromise following a presumed respiratory infection. This clinical pattern aligns with earlier reports highlighting the aggressive nature of axonal variants of GBS, particularly in younger patients from developing regions. In the current case, antecedent respiratory symptoms preceded neurological manifestations by a few days, consistent with established literature identifying infections as a major trigger for GBS. Asbury et al described immune-mediated inflammatory processes targeting peripheral nerves following infections, emphasizing molecular mimicry as a key pathogenic mechanism.[5]

Similarly, Griffin et al demonstrated that AMAN is characterized by early nodal and axonal damage without prominent demyelination, explaining the rapid progression and severity observed in such patients.[6] The early respiratory failure and need for mechanical ventilation in our patient are in agreement with these pathological findings. Several epidemiological studies have highlighted the predominance of axonal variants such as AMAN in Asian populations. Islam et al reported a strong association between Campylobacter jejuni infection and axonal GBS in Bangladesh, with patients exhibiting severe motor weakness and higher rates of ventilatory support.[7] Although C. jejuni testing was not documented in the present case, the clinical phenotype closely mirrors these findings, suggesting a possible infectious trigger. In India, GBS shows notable seasonal and regional variation. Epidemiological analyses indicate an increased incidence during monsoon and post-monsoon periods, coinciding with a rise in respiratory and gastrointestinal infections.[2] The recent surge in GBS cases reported across multiple Indian states further underscores the public health relevance of this disorder.[8,9] The age and gender profile of the present patient is consistent with

Indian epidemiological data, which report a male predominance and higher incidence among young adults.[10] The absence of sensory deficits and reflexes in this case is characteristic of AMAN and contrasts with the demyelinating subtype (AIDP), which typically presents with sensory involvement. Griffin et al. emphasized that selective motor axonal injury explains the pure motor presentation and areflexia seen in AMAN patients.[2]

Similar findings were observed in previous Indian and Asian cohort studies, supporting the diagnostic classification in this case. Early initiation of plasmapheresis in the present patient aligns with established treatment guidelines and prior evidence supporting its efficacy in severe GBS. Bragazzi et al, in a global burden analysis, emphasized that timely immunomodulatory therapy significantly reduces disability and improves functional outcomes, particularly in severe cases requiring intensive care.[8] Although intravenous immunoglobulin (IVIG) is commonly used, plasmapheresis remains equally effective, especially in resource-limited settings.

Compared to Miller Fisher syndrome, which represents a rare variant of GBS characterized by ophthalmoplegia and ataxia, the present case lacked cranial nerve involvement. Recent reports on Miller Fisher syndrome emphasize its relatively benign course compared to axonal variants, further highlighting the severity of AMAN observed in this patient.[11] Overall, this case reinforces previous findings that AMAN is a severe GBS subtype with rapid progression, high ventilatory requirement, and significant morbidity, particularly in young male patients following infections. Early recognition, aggressive supportive care, and prompt immunotherapy are crucial to improving outcomes.

CONCLUSION

GBS is a serious immune-mediated peripheral neuropathy with diverse clinical presentations and significant global burden. Early recognition, prompt management, and improved understanding of its immunopathogenesis are essential to reduce morbidity, disability, and emerging public health concerns worldwide.

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