

Fulminant Guillain–Barré Syndrome in A Young Adult: Case Report

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INTRODUCTION

Guillain–Barré syndrome is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness and areflexia. Although the demyelinating form, acute inflammatory demyelinating polyradiculoneuropathy (AIDP), predominates in Western countries, axonal variants such as acute motor axonal neuropathy (AMAN) are also recognized and may result in more severe neurological impairment and delayed recovery [1]. A preceding infectious episode, particularly gastrointestinal infection, is frequently reported. We present a case of fulminant axonal GBS in a previously healthy young adult who rapidly developed respiratory failure requiring mechanical ventilation.

CASE PRESENTATION

A 25-year-old North African man with no significant medical history presented to the emergency department with rapidly progressive flaccid tetraparesis that had developed over approximately 12 hours. The weakness was ascending, initially affecting the lower limbs before progressing to the upper limbs. There were no sensory symptoms or sphincter disturbances. However, the patient reported increasing shortness of breath and a history of acute diarrhea seven days earlier.

On admission, he was dyspneic, with an oxygen saturation of 92% on room air, while hemodynamic parameters remained stable. Neurological examination revealed a Glasgow Coma Scale score of 15/15, complete motor paralysis of all four limbs (0/5), and absent deep tendon reflexes. Temperature and capillary blood glucose levels were normal.

The patient rapidly developed worsening respiratory distress, characterized by sweating and agitation, prompting urgent endotracheal intubation and mechanical ventilation. Based on the clinical presentation, GBS was suspected and IVIg therapy was initiated at 0.4 g/kg/day for five consecutive days, together with supportive management.

Cerebrospinal fluid analysis showed a normal cell count (<3 cells/mm³) and protein concentration (0.45 g/L). Early nerve conduction studies demonstrated findings compatible with an axonal variant of GBS. The patient was transferred to the

intensive care unit and subsequently underwent tracheostomy because of the anticipated need for prolonged ventilatory support. Neurological recovery was gradual over a three-week period.

DISCUSSION

GBS typically presents with symmetrical, rapidly progressive weakness and generalized areflexia. Fulminant presentations are uncommon but carry a substantial risk of acute respiratory failure [2]. In the present case, the combination of rapidly ascending paralysis, areflexia, preserved consciousness, and early respiratory deterioration was strongly suggestive of GBS. According to the Brighton diagnostic criteria, the patient did not fulfill Level 1 because the cerebrospinal fluid protein concentration was not elevated. However, the clinical presentation and electrophysiological findings were strongly supportive of Guillain–Barré syndrome. The normal cerebrospinal fluid protein level may be explained by the early timing of lumbar puncture in the course of the disease [3,7].

The AMAN subtype is an axonal variant characterized predominantly by motor involvement without significant sensory manifestations. It has been associated particularly with preceding *Campylobacter jejuni* infection and anti-GM1 antibodies [4,5]. Although antibody testing was unavailable in our setting, the recent diarrheal illness, absence of sensory symptoms, and early electrophysiological evidence of axonal involvement supported the diagnosis.

Respiratory muscle weakness is a major complication of GBS and may progress rapidly, requiring intensive care and mechanical ventilation [6]. Early recognition of respiratory deterioration is therefore essential. Although cerebrospinal fluid albuminocytologic dissociation is a classical finding, it may be absent during the early phase of the disease, as observed in our patient [7].

IVIg is an established treatment for GBS, and early administration is recommended to limit disease progression and improve recovery [8]. Axonal variants may be associated with slower motor recovery and prolonged rehabilitation, with an increased risk of residual neurological deficits [9].

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Prolonged mechanical ventilation may necessitate tracheostomy, as occurred in this case [10].

CONCLUSION

Fulminant GBS, particularly the axonal variant, can progress rapidly to respiratory failure in otherwise healthy young adults. Early clinical recognition, prompt respiratory support, and timely initiation of IVIg are essential. Close monitoring in an intensive care setting and appropriate airway management are crucial to minimize complications and improve neurological outcomes.

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