Rapid clinical reversal of fulminant Guillain-Barre syndrome (AMAN variant) following therapeutic plasma exchange: a case report

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ABSTRACT

Background: Acute motor axonal neuropathy (AMAN) is a severe Guillain-Barré syndrome variant that may deteriorate rapidly, particularly when early bulbar weakness and respiratory failure occur. Cerebrospinal fluid (CSF) protein may remain normal early in the disease course, and coexisting upper-airway findings can create diagnostic uncertainty.

Case Presentation: A 43-year-old previously healthy man developed severe dysphagia, sialorrhea, and nasal speech after febrile pharyngitis, followed by rapidly progressive limb weakness. Examination showed flaccid tetraparesis, preserved sensation, and evolving bulbar dysfunction. Nasopharyngoscopy demonstrated mild non-obstructive epiglottic inflammation, initially suggesting an upper-airway or infectious cause. Within 24 hours, he developed ineffective cough, neck flexor weakness, and impending respiratory failure requiring intensive care admission and endotracheal intubation. Post-intubation examination showed generalized areflexia and autonomic instability. Day 1 CSF showed no pleocytosis and normal protein. Day 2 nerve conduction studies demonstrated severe predominantly motor axonal polyneuropathy with relatively preserved sensory responses, consistent with AMAN. Intravenous immunoglobulin was initially started but discontinued before completion of a full therapeutic course, and five plasma exchange (PLEX) sessions were performed alongside intensive supportive care. Clinical improvement was observed during hospitalization, allowing extubation by Day 5 and discharge on Day 10 with mild residual upper-limb weakness.

Conclusion: Fulminant bulbar-predominant AMAN may mimic upper-airway pathology and present before CSF protein elevation. In this case, neurological improvement occurred after initiation of PLEX and intensive supportive care; however, causality cannot be inferred from a single case. Normal early CSF findings and mild non-obstructive ENT abnormalities should not delay electrodiagnostic evaluation, respiratory monitoring, and timely immunomodulatory treatment.

Keywords: Guillain-Barré syndrome, acute motor axonal neuropathy, dysphagia, plasma exchange, respiratory failure, peripheral nervous system diseases.

Type of Article: Case Report **Specialty:** Neurology (with critical care/ internal medicine relevance)

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Background

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and a leading cause of acute flaccid paralysis worldwide, with an estimated incidence of 1-2 per 100,000 person-years [1-3]. It often follows a respiratory or gastrointestinal infection and may progress rapidly, making early recognition and escalation of care essential [1,2]. Bulbar dysfunction and dysautonomia are associated with severe disease, and some patients require mechanical ventilation due to neuromuscular respiratory failure [1,2].

GBS includes electrophysiological subtypes with distinct mechanisms and prognoses, including the axonal variant acute motor axonal neuropathy (AMAN) [3-5]. Diagnosis is primarily clinical and supported by cerebrospinal fluid (CSF) analysis and electrodiagnostic studies; however, both may be normal or non-diagnostic early in the disease course [1,2]. We report a fulminant bulbar-predominant AMAN case following acute pharyngitis in which mild non-obstructive epiglottic inflammation acted as a diagnostic distractor, highlighting the importance of avoiding premature diagnostic closure and maintaining early respiratory monitoring in suspected GBS [2,5].

Case Presentation

A 43-year-old previously healthy Bangladeshi man presented with severe dysphagia and rapidly progressive generalized weakness, after a 7-day prodrome of subjective

fever and pharyngitis, for which he had received empirical oral amoxicillin-clavulanate and paracetamol. Two days before admission, he developed sore throat, odynophagia, productive cough, sialorrhea, and worsening dysphagia. One day before presentation, he developed rapidly progressive limb weakness that was initially more prominent in the upper limbs, particularly proximally, and subsequently involved the lower limbs, resulting in two falls.

He had no known chronic medical or neuromuscular disease, no relevant family history of neurological or autoimmune disease, and no contributory occupational or exposure history.

On arrival, he was afebrile, hemodynamically stable, and saturating 97% on room air. He was alert and fully oriented but had nasal speech, continuous drooling, and an inability to swallow saliva. Neurological examination showed flaccid weakness in all four limbs with proximal predominance and mild asymmetry, with upper-limb power of 2/5 on the right and 3/5 on the left, and lower-limb power of 3-4/5 bilaterally. Sensation was preserved, sphincter function was intact, and bulbar involvement was evident, with dysarthria and dysphagia.

Nasopharyngoscopy demonstrated mild non-obstructive epiglottic inflammation without critical airway narrowing. This initially raised concern for an upper-airway or infectious cause of dysphagia; however, the concurrent generalized limb weakness and evolving neuromuscular signs suggested that isolated epiglottitis did not fully explain the presentation.

Within 24 hours of admission, his bulbar dysfunction worsened, with ineffective cough, repeated choking on secretions, and new neck flexor weakness. Despite a Glasgow Coma Scale score of 15 and intact extraocular movements, impending respiratory failure prompted urgent transfer to the intensive care unit (ICU) for endotracheal intubation and airway protection [6]. Post-intubation examination showed profound generalized weakness, global areflexia, and sinus bradycardia consistent with early dysautonomia. Sensory assessment was limited by sedation, but there was no sensory level or sphincter disturbance. The first plasma exchange (PLEX) session was performed on Day 1.

Investigations

Initial laboratory testing showed mild neutrophil-predominant leukocytosis and mildly elevated C-reactive protein, with low procalcitonin and otherwise unremarkable blood count indices (Table 1). Metabolic testing showed mild hyponatremia and hyperchloremia, with preserved renal function and euglycemia. Ionized calcium was initially normal but decreased during PLEX and required replacement. Baseline coagulation parameters were normal; however, hypofibrinogenemia developed after PLEX, reaching a nadir of 1.23 g/l on Day 7, and improved by discharge without bleeding.

CSF analysis on Day 1, obtained within the first week of weakness, showed clear fluid with no pleocytosis, normal glucose, normal protein, and negative Gram stain and culture. This did not exclude GBS because albuminocytologic dissociation may be absent early in the disease course, and CSF protein can remain normal during the first week after symptom onset. Therefore, diagnostic emphasis was placed on the clinical progression, areflexia, respiratory involvement, and early nerve conduction studies (NCS).

Microbiological investigations, including blood, urine, CSF, and endotracheal aspirate cultures, multidrug-resistant organism screening, and respiratory and meningitis multiplex polymerase chain reaction (PCR) panels, were negative. Acetylcholine receptor antibody testing, *Mycoplasma* IgM, and urine *Legionella* antigen were also negative. Ganglioside antibodies were sent during admission and became available on follow-up, showing a positive anti-ganglioside panel with significantly elevated titers (>300), including GD1a, GD1b, and GQ1b IgG/IgM antibodies.

Chest radiograph demonstrated a new left basal infiltrate, consistent with aspiration or early hospital-acquired pneumonia in the setting of severe bulbar dysfunction and impaired airway protection (Figure 1). Contrast-enhanced computed tomography (CT) neck showed bilateral cervical

Table 1. *Clinically relevant laboratory findings during admission.*

PARAMETER	RESULT	INTERPRETATION
WBC	11.4 × 10^9/l	Mild leukocytosis
Neutrophils	76.3%	Neutrophil predominance
CRP	5.0-5.3 mg/l	Mild inflammatory response
Procalcitonin	0.06 ng/ml	Bacterial sepsis less likely
Sodium	134 mmol/l	Mild hyponatremia
Chloride	112 mmol/l	Mild hyperchloremia
Ionized calcium	Initially normal; decreased during PLEX	Monitored/replaced during PLEX
Fibrinogen	Nadir 1.23 g/l after PLEX	Monitored; no bleeding

Abbreviations: WBC, white blood cell count; CRP, C-reactive protein; PLEX, plasma exchange.

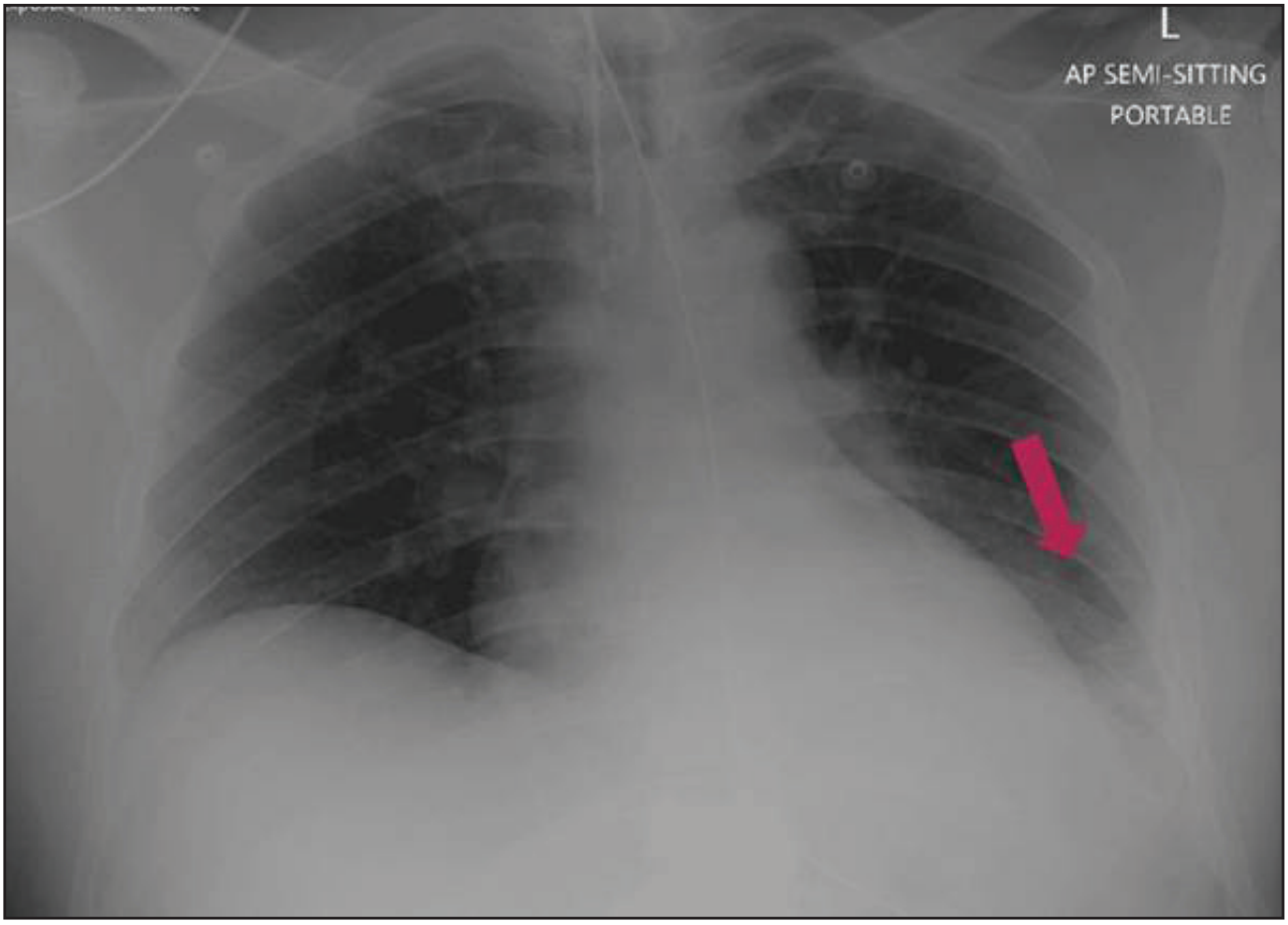

Figure 1. *Portable chest radiograph demonstrating a new left basal infiltrate, supporting aspiration or early hospital-acquired pneumonia in the context of severe bulbar dysfunction and impaired airway protection.*

lymphadenopathy, asymmetric tonsillar prominence, and a small left tonsillar fluid collection, supporting acute tonsillitis or upper-airway inflammation as a diagnostic distractor and possible infectious trigger, without critical airway narrowing (Figure 2). CT head was normal. Magnetic resonance imaging (MRI) brain showed no acute infarction or demyelination. MRI of the cervical and thoracic spine showed no myelitis, spinal cord infarction, or compressive pathology.

Electrodiagnostic studies on Day 2 demonstrated a severe, predominantly motor axonal polyneuropathy with lower-limb predominance. Sensory NCS showed preserved lower-limb, ulnar, and radial sensory responses, with absent left median sensory response. Motor studies showed absent distal compound muscle action potential (CMAPs) in bilateral peroneal and tibial nerves, markedly reduced CMAP amplitudes with relatively preserved conduction velocities, and unrecordable F-waves in all tested nerves. The overall electrodiagnostic pattern supported AMAN.

Differential diagnosis

The main diagnostic alternatives were myasthenic crisis and botulism, as both can present with prominent bulbar dysfunction and respiratory failure. Myasthenic crisis was considered because of severe dysphagia, dysphonia, and evolving ventilatory failure. However, the absence of fatigable or ocular weakness, the development of generalized areflexia, negative acetylcholine receptor antibodies, and NCS evidence of diffuse motor axonal polyneuropathy favored AMAN rather than a neuromuscular junction disorder.

Botulism was also considered because it may cause bulbar weakness and respiratory failure. However, the absence of pupillary involvement, lack of compatible foodborne or wound exposure, preserved sensorium, and electrodiagnostic findings were not supportive. Infectious polyradiculitis, meningitis, and encephalitis were less likely given normal CSF cellularity, negative cultures and multiplex PCR testing, and lack of supportive neuroimaging findings. Acute myelitis or spinal cord ischemia was also considered less likely because there was no sensory level or sphincter dysfunction, and spinal MRI showed no myelitis, infarction, or compressive pathology.

Overall, the combination of recent infectious prodrome, rapidly progressive flaccid weakness, generalized areflexia, preserved sensory responses, and severe motor axonal involvement on NCS supported AMAN.

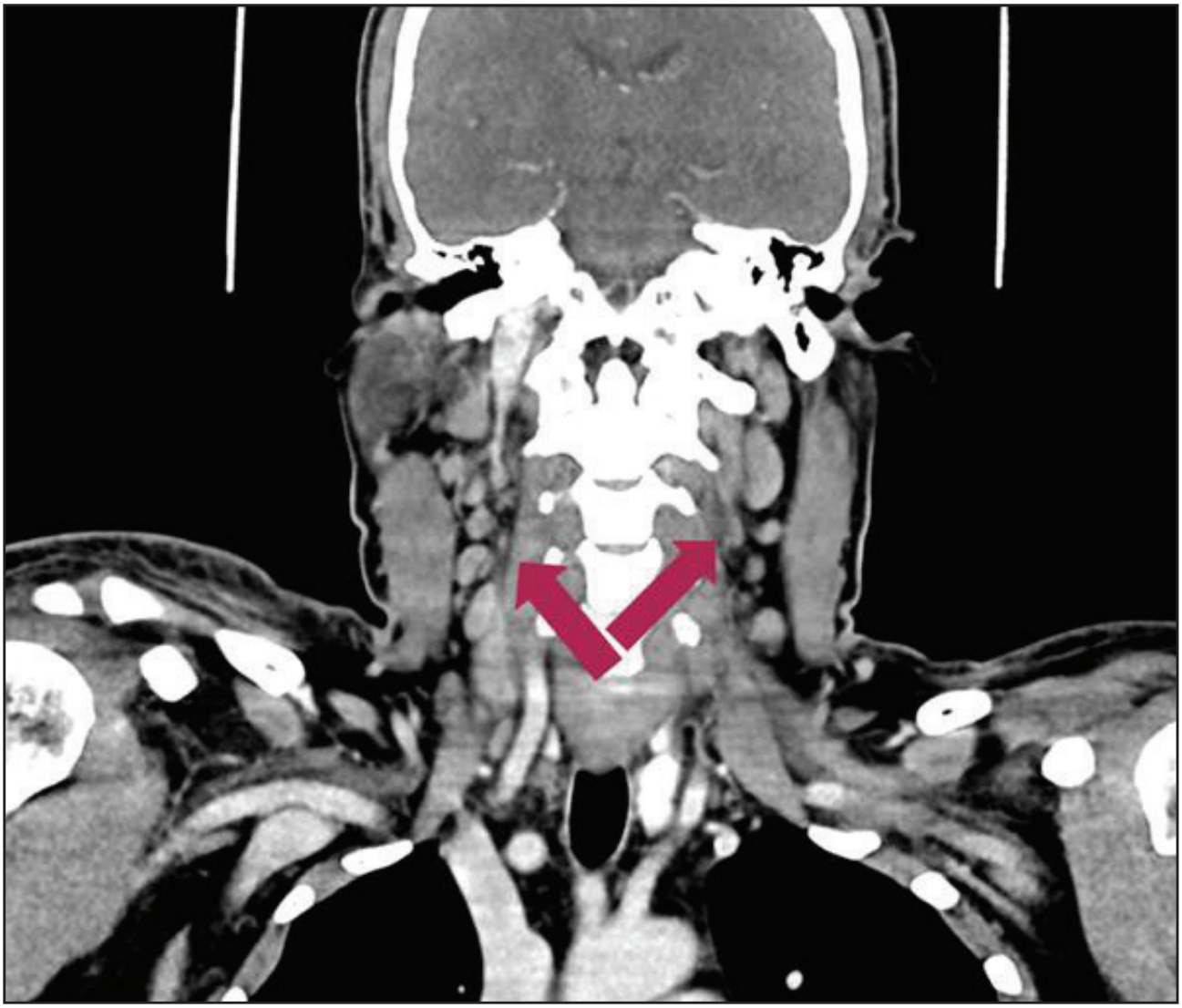

Figure 2. *Contrast-enhanced CT neck demonstrating bilateral cervical lymphadenopathy, asymmetric tonsillar prominence, and a small left tonsillar fluid collection, supporting acute tonsillitis or upper-airway inflammation as a diagnostic distractor and possible infectious trigger, without evidence of critical airway narrowing.*

Therapeutic intervention

Before admission, the patient had received empirical oral amoxicillin-clavulanate and paracetamol for presumed upper-respiratory infection; however, oral therapy could not be continued after presentation because of severe dysphagia. In the emergency department, he received a single dose of intravenous dexamethasone for suspected epiglottitis. After chest radiography on Day 1 showed a new left basal infiltrate, antibiotics were escalated to piperacillin-tazobactam for 5 days and then de-escalated to intravenous amoxicillin-clavulanate to complete a 10-day course for aspiration pneumonia and acute tonsillitis.

Immunomodulatory treatment was initially started with intravenous immunoglobulin while the working diagnosis included severe GBS and other immune-mediated neuromuscular disorders. IVIG was discontinued before completion of a full therapeutic course, and PLEX was selected as the definitive immunomodulatory therapy because of the fulminant bulbar and respiratory involvement, ICU setting, and treating team's judgment. This was not intended as sequential combination therapy.

PLEX was performed via a right internal jugular apheresis catheter in five sessions over 9 days on Days 1, 3, 5, 7, and 9. Approximately 1 to 1.5 plasma volumes were exchanged per session using 5% albumin and saline replacement. Calcium and fibrinogen were monitored throughout treatment, and treatment-related hypocalcemia and hypofibrinogenemia were corrected without bleeding or hemodynamic complications.

The patient underwent endotracheal intubation and mechanical ventilation on Day 1 for airway protection because of severe bulbar dysfunction, ineffective cough, and inability to manage secretions. Sedation was initiated with fentanyl and propofol and later transitioned to dexmedetomidine to facilitate neurological assessment and ventilator weaning. Low-dose norepinephrine was used

briefly for blood pressure support. Supportive ICU care included electrolyte correction, venous thromboembolism prophylaxis, enteral nutrition, and physiotherapy.

Follow-up and outcomes

Clinical improvement was observed after initiation of PLEX and intensive supportive care. After the first exchange session, he became more alert and demonstrated improved limb movement while still mechanically ventilated. By Day 5, after three exchange sessions, limb strength and bulbar function had improved sufficiently to allow extubation, removal of the nasogastric tube, and transfer from the ICU to the medical floor. Over the subsequent days, swallowing normalized, dysautonomia resolved, and he progressed from assisted transfers to independent ambulation within his room.

All five planned PLEX sessions were completed. Tolerability was assessed clinically and with serial laboratory monitoring, including calcium and fibrinogen. Treatment-related hypocalcemia and hypofibrinogenemia were corrected, with no major bleeding, hemodynamic instability, thromboembolic event, catheter-related complication, ventilator-associated pneumonia, or significant adverse drug reaction.

At discharge on Day 10, he was alert, breathing comfortably on room air, and hemodynamically stable. Neurological examination showed full strength in both lower limbs and mild residual proximal weakness in the upper limbs. Distal sensory symptoms in the legs were improving, and aspiration pneumonia had resolved with antibiotic therapy.

At early outpatient follow-up 10 days after discharge, he reported significant overall improvement, with only mild residual right upper-limb weakness and no sensory loss. Examination showed normal neck flexion and extension, normal facial power, normal tongue movements, full lower-limb power bilaterally, and mild distal-predominant upper-limb weakness involving the finger extensors and intrinsic hand muscles. Upper-limb and ankle reflexes remained absent, knee reflexes were present with reinforcement, plantar responses were downgoing bilaterally, and pinprick sensation was normal in all limbs.

Results reviewed at follow-up included a positive anti-ganglioside antibody panel with significantly elevated titers >300, negative acetylcholine receptor antibody, Day 1 CSF with normal protein and no pleocytosis, NCS consistent with AMAN, elevated creatine kinase trending downward, and normal MRI brain and cervical spine. Because follow-up was short, longer-term neurological recovery could not be fully assessed. A repeat NCS after 5 months and neurology clinic follow-up after 6 months were planned, with earlier reassessment advised if weakness, dysphagia, sensory symptoms, or respiratory symptoms recurred.

Discussion

This case describes bulbar-predominant onset with rapid progression to areflexic flaccid quadriparesis and early respiratory compromise, ultimately diagnosed as AMAN, an axonal subtype of GBS [1,3-5]. Early dysphagia and drooling after acute pharyngitis, together with mild non-obstructive epiglottic inflammation on nasopharyngoscopy, created a plausible upper-airway explanation and risk of diagnostic anchoring while neuromuscular respiratory failure was evolving [1,3].

This case highlights two practical diagnostic pitfalls in suspected GBS: over-reliance on early CSF protein elevation and premature closure on an anatomic or infectious explanation for dysphagia [1-3]. Normal CSF protein on Day 1 did not exclude GBS, as CSF protein may remain normal during the first week of illness [1,3]. Although electrodiagnostic testing may be non-classifying early, this patient's NCS showed a severe motor axonal pattern with preserved sensory responses, supporting AMAN and arguing against a primary neuromuscular junction disorder [1,4]. Myasthenic crisis and botulism were reasonable differentials at the time of impending ventilatory failure [7,8]. However, generalized areflexia, absence of fatigable ocular weakness, lack of pupillary involvement or compatible exposure history, and the electrophysiologic pattern favored AMAN rather than a neuromuscular junction disorder or botulism [7-9].

A major management strength was early recognition of the need for airway protection and ICU monitoring in the setting of severe bulbar dysfunction and ineffective cough [1,2]. Respiratory failure in GBS may develop without prominent dyspnea, and tools such as the Erasmus GBS Respiratory Insufficiency Score may support earlier ICU triage in similar presentations [2,10]. In non-ambulant GBS, intravenous immunoglobulin and PLEX have broadly similar efficacy when delivered within an appropriate treatment window [1,2,11-13]. The 2023 European Academy of Neurology guideline supports either intravenous immunoglobulin or PLEX, typically four to five exchanges over 1 to 2 weeks, and advises against routine sequential combination therapy [2,13].

Clinical improvement was observed after initiation of PLEX and intensive supportive care; however, causality cannot be established from a single case [1,4,14]. Early improvement in AMAN may reflect reversible nodal or paranodal dysfunction, which can permit faster recovery than would be expected from irreversible axonal degeneration alone [5,15]. The positive anti-ganglioside antibody panel further supported an immune-mediated axonal GBS phenotype. Anti-GD1a antibodies are particularly associated with AMAN and may target motor axolemmal or nodal/paranodal structures, consistent with this patient's predominantly motor axonal NCS pattern and preserved sensory responses [4,5]. The reported high titers (>300) support strong seropositivity, but interpretation should

remain cautious because assay units and cutoffs vary between laboratories, and no validated titer threshold reliably predicts prognosis in an individual patient. PLEX was well tolerated overall; hypocalcemia and hypofibrinogenemia were monitored and corrected without bleeding or hemodynamic instability, highlighting the importance of structured monitoring during therapy.

Strengths of this report include the detailed clinical chronology, early electrodiagnostic characterization, and documentation of short-term recovery in a fulminant bulbar-predominant presentation. Limitations include the single-case design, inability to infer causality regarding PLEX response, delayed availability of ganglioside antibody results, and short follow-up beyond the early post-discharge visit. Longer-term neurological recovery could not be fully assessed, although repeat NCS at 5 months and neurology follow-up at 6 months were planned.

Conclusion

Fulminant AMAN may initially mimic upper-airway pathology such as epiglottitis. GBS should remain in the differential diagnosis when dysphagia occurs with progressive limb weakness, even when ear, nose, and throat findings appear explanatory. Normal early CSF protein does not exclude GBS, particularly during the first week of illness. In this case, neurological improvement was observed after initiation of PLEX and intensive supportive care; however, causality cannot be inferred from a single case. Early recognition, respiratory monitoring, electrodiagnostic assessment, and timely immunomodulatory treatment remain central to management.

Patient perspective

The patient reported that the most distressing symptoms were the sudden inability to swallow and rapid loss of strength. He was grateful that his swallowing and mobility had improved before discharge. At follow-up, he reported continued improvement, with only mild residual right arm weakness.

What is new?

Fulminant bulbar-predominant AMAN can initially mimic upper-airway pathology, including mild epiglottic inflammation, creating a risk of diagnostic anchoring while neuromuscular respiratory failure is evolving. This case also highlights that normal early CSF protein does not exclude GBS and that early electrodiagnostic testing plus timely PLEX may be associated with rapid clinical recovery. Follow-up anti-ganglioside serology further supported an immune-mediated AMAN phenotype.

List of Abbreviations

ABG	Arterial blood gas
AChR	Acetylcholine receptor
AMAN	Acute motor axonal neuropathy
CMAP	Compound muscle action potential
CRP	C-reactive protein
CSF	Cerebrospinal fluid
CT	Computed tomography
ENT	Ear, nose, and throat
GBS	Guillain-Barre syndrome
ICU	Intensive care unit
MRI	Magnetic resonance imaging
NCS	Nerve conduction studies
PCR	Polymerase chain reaction
PLEX	Plasma exchange

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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None.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the journal upon request.

Ethical approval

Ethical approval is not required at our institution for anonymous case reports.

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Summary of the case

NO.	ITEM	DETAILS
1	Patient (gender, age)	43 years, male
2	Final diagnosis	GBS, AMAN variant
3	Symptoms	Severe dysphagia, sialorrhea, nasal speech, rapidly progressive generalized weakness, bulbar dysfunction, evolving respiratory failure
4	Medications	Empirical amoxicillin-clavulanate and paracetamol before admission; dexamethasone in the emergency department; piperacillin-tazobactam followed by amoxicillin-clavulanate for aspiration pneumonia and acute tonsillitis; ICU supportive medications including sedation and brief norepinephrine
5	Clinical procedure	Endotracheal intubation and mechanical ventilation; five PLEX sessions via right internal jugular apheresis catheter; ICU supportive care and rehabilitation
6	Specialty	Neurology (with critical care/internal medicine relevance)