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CASE REPORT

Guillain–Barré Syndrome in a Young Male: Pharyngeal–Cervical–Brachial or Descending Acute Motor Axonal Neuropathy

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ABSTRACT

Introduction: Guillain–Barré Syndrome (GBS) comprises a heterogeneous group of acute immune-mediated polyradiculoneuropathies. While the classic ascending pattern is most common, rare variants, such as the pharyngeal–cervical–brachial (PCB) variant and acute motor axonal neuropathy (AMAN), can present diagnostic challenges, particularly when overlapping features are present.

Case Report: We report the case of an 18-year-old previously healthy male who presented with rapidly progressive dysphagia, neck weakness, and bilateral upper limb weakness, followed by descending motor involvement without sensory deficits. Neurophysiological studies showed reduced compound muscle action potentials with preserved sensory nerve action potentials, consistent with an axonal motor neuropathy.

Discussion: Nerve conduction studies showed AMAN, whereas bulbar and upper limb weakness suggested the PCB variant. The clinical picture, i.e. descending paralysis (hands to feet) indicated a possible descending AMAN or PCB-AMAN overlap.

Conclusion: This case underscores the importance of recognizing overlapping GBS variants to facilitate early diagnosis and timely initiation of immunotherapy. Awareness of such presentations can help avoid misdiagnosis, especially when cranial and cervicobrachial weakness predominate.

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INTRODUCTION

Guillain–Barré Syndrome (GBS) is a rare, acute, immune-mediated neurological disorder in which the immune system attacks the peripheral nerves, leading to inflammatory demyelination or axonal degeneration of motor and sensory fibres.[1] GBS is characterized by a rapid-onset muscle weakness, areflexia, and sensory disturbances that often progress symmetrically, and in severe cases, respiratory failure or paralysis may occur.[2] GBS is frequently preceded by an infection, most commonly respiratory or gastrointestinal. The leading infectious trigger is *Campylobacter jejuni*, followed by *Cytomegalovirus*, *Epstein–Barr virus*, *Mycoplasma pneumoniae*, and, more recently, *SARS-CoV-2*.[3] GBS represents the most common cause of acute flaccid paralysis worldwide, with an incidence of 1–2 per 100,000 population annually.[3] Although the classic form features ascending paralysis, several variants exist, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor–sensory axonal neuropathy (AMSAN), Miller Fisher syndrome (MFS), and the rare pharyngeal–cervical–brachial (PCB) variant.[3–5]

The PCB variant is rare and primarily affects the oropharyngeal, neck, and upper limb muscles with relative sparing of the lower extremities and sensory function.[6] In contrast, AMAN is characterized by selective motor axonal degeneration and typically demonstrates a rapidly progressive, predominantly limb weakness pattern. AMAN also shows association with *Campylobacter jejuni* infection and anti-GD1a antibodies.[1,5] Overlapping features between PCB and AMAN are uncommon but have been documented, suggesting shared immunopathological mechanisms involving anti-ganglioside antibodies, such as anti-GT1a and anti-GD1a.[5,7] Distinguishing these variants is clinically important because prompt treatment significantly improves outcomes.

Here, we present a young male with descending motor weakness, initially affecting bulbar and cervicobrachial regions, later demonstrating electrophysiological features consistent with an AMAN. This unusual presentation points towards the diagnostic overlap between PCB and descending AMAN (*Table 1*).

Table 1: Subtypes of Guillain–Barré Syndrome with Key Features and Prognosis

Subtype	Pathology	Key Features	Antibodies (often)	Prognosis
AIDP (Acute Inflammatory Demyelinating Polyradiculoneuropathy)	Demyelination of peripheral nerves	Ascending weakness, areflexia, mild sensory loss	Not strongly linked to specific gangliosides	Usually good recovery; most common in Europe/US
AMAN (Acute Motor Axonal Neuropathy)	Axonal degeneration (motor only)	Pure motor weakness, no sensory symptoms	Anti-GM1, GD1a	Often more severe, but recovery possible with good treatment; common in Asia/LatAm
AMSAN (Acute Motor-Sensory Axonal Neuropathy)	Axonal degeneration (motor + sensory)	Motor weakness and sensory loss	Anti-GD1a, GM1	Poorer prognosis; slower/ incomplete recovery
MFS (Miller Fisher Syndrome)	Demyelination/axonal injury in cranial nerves	Classic triad: ophthalmoplegia, ataxia, areflexia	Anti-GQ1b	Usually, excellent recovery within weeks
PCB (Pharyngeal–Cervical–Brachial) Variant	Neuropathy of cranial/ upper limb nerves	Weakness in face, neck, arms	Anti-GT1a, GQ1b	Variable; usually better than axonal types
Bickerstaff Brainstem Encephalitis (overlap with MFS)	Brainstem involvement	Ophthalmoplegia + ataxia + altered consciousness	Anti-GQ1b	Good recovery in most cases

Subtype	Pathology	Key Features	Antibodies (often)	Prognosis
Paraparetic GBS	Restricted to lumbosacral nerves	Weakness only in legs, mimics spinal cord lesion	None specific	Recovery usually favorable
Pure Sensory GBS (rare)	Sensory nerve involvement	Numbness, paresthesias, ataxia without weakness	Not well defined	Prognosis variable

Case Report

An 18-year-old previously healthy male presented with history of rapid progressive weakness of both the upper limbs and neck muscles, followed by involvement of lower limb muscles within 48 hours. The patient was apparently well 2 days back, when he started developing weakness in both shoulders and upper arms. Over the next 48 hours, the patient developed progressive bilateral lower limb weakness. The patient denied limb paraesthesia, diplopia, respiratory difficulty, or recent vaccination. The patient had a history of acute gastroenteritis 10 days prior and a sore throat, indicating upper respiratory tract infection 4 days before admission, consistent with a common trigger for AMAN.[7]

Examination (*Tables 2 and 3*):

- Vital signs were stable.
- On head-to-toe examination, the left upper limb strength was 2/5 and right upper limb strength was 4/5.
- Bilateral lower limbs exhibited normal strength (5/5).
- Bilateral plantar reflex were mute.
- Bilateral knee jerk reflexes were brisk.
- Bilateral biceps and triceps tendon reflexes were absent
- Sensation was intact throughout.
- No ataxia was present.
- Rest of the physical examination was normal.

Table 2: Reflex Examination Findings

Reflex Exam	Finding
Bilateral plantar response	Mute
Bilateral knee jerk reflex	Brisk
Bilateral biceps and triceps tendon reflexes	Absent

Table 3: Muscle Power Assessment

Region	Power
Left Upper Limb	2/5
Right Upper Limb	4/5
Bilateral Lower Limb	5/5

Investigations:

- Routine hematology and biochemistry were within normal limits.
- MRI brain was done, which was sugestive of a normal scan.
- MRI whole spine screening was done, which was suggestive of subtle early posterior disc bulges in mid-lower cervical and lumbar regions with no obvious significant compression on exiting nerve roots seen.
- Nerve conduction studies revealed:
  - F wave latency absent in right median, both ulnar, common peroneal and posterior tibial nerve.
  - F wave latency normal in left median nerve.
  - H reflex absent bilaterally
  - Normal sensory nerve action potentials

These findings were suggestive of electrophysiological abnormality of an acute motor neuropathy, which was consistent with an **AMAN** pattern.

Treatment and Outcome:

The patient was started on intravenous immunoglobulin (IVIG) therapy (0.4 g/kg/day for 5 days @ 0.01 ml/kg/min) post admission in ICU. 24 hours post admission, the patient developed poor swallowing and weak voice. Ryle's tube (RT) was inserted via nasal root, and the patient was started on RT feed. After 48 hours of admission, the patient developed weakened respiratory efforts and poor cough reflex, leading to retention of secretions. As a result, the patient was intubated and was put on a ventilator to protect the airway. The patient was started on Fentanyl and midazolam infusion. Bilateral upper limb strength further reduced to 1/5 and bilateral lower limb strength reduced to 2/5. Four days post admission, the patient was tracheostomised in view of prolonged need of ventilation. Over the subsequent 2 weeks, the patient was managed conservatively with frequent

tracheostomy tube suctioning, antibiotics, IV fluids, and RT. With each passing day, his condition improved. Swallowing improved substantially, neck strength recovered to 4/5, and upper limb strength improved to 4/5 and lower limb to 5/5. At 4-week follow-up, the patient had regained near-normal motor function and returned to baseline activities and the patient's tracheostomy tube was also removed.

DISCUSSION

PCB-GBS is a rare variant characterized by selective involvement of the oropharyngeal, cervical, and brachial muscles.[6] Patients typically present with rapidly progressive dysphagia, neck weakness, and upper limb weakness, often with preserved lower limb strength and sensation. Whereas, AMAN is a pure motor neuropathy, occurring in an ascending pattern, involving only motor nerves and sparing sensory nerves. Anti-GT1a and anti-GD1a antibodies have been reported in PCB and are also strongly associated with AMAN, suggesting pathophysiological overlap.[4,7] In India, AMAN accounts for 14% of GBS cases. The descending variant of AMAN is extremely rare, with no exact percentage reported. A study[7] found that antibodies linked to AMAN (GM1, GM1b, GD1a, and GalNAc-GD1a) were present in 27% of PCB patients (*Figure 1*).

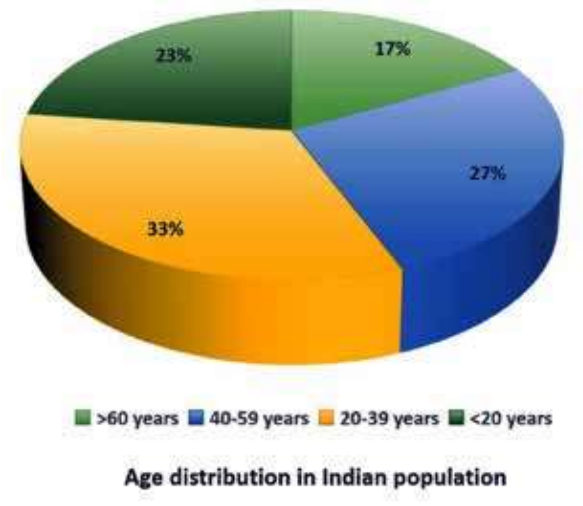

Figure 1: Pie chart showing the proportion of PCB patients with antibodies linked to AMAN[7]

The present case showed PCB variant-related features, including pharyngeal involvement, neck weakness, and upper limb dominant weakness. Nerve conduction studies showed a pure motor axonal pattern, which is typical of AMAN. The descending paralysis (hands → feet) indicated a possible descending AMAN or PCB–AMAN overlap. These overlapping signs suggest that different GBS variants may be a part of the same clinical and immune spectrum rather than being completely separate conditions. Recognizing these atypical presentations is crucial, as the early symptoms may mimic brainstem stroke, myasthenia gravis, botulism, or diphtheritic neuropathy.[4] Early neurophysiological testing, cerebrospinal fluid analysis, and response to immunotherapy can assist in confirming the diagnosis. Antiganglioside antibody analysis can provide additional diagnostic support but was beyond the scope of this case study.

This case highlights that PCB involvement does not exclude an underlying axonal subtype and that descending AMAN-like presentations should be considered when electrophysiological findings support motor axonopathy. Early initiation of IVIG or plasmapheresis is essential because both therapies significantly reduce disability and hasten recovery.[8]

CONCLUSION

This case describes a young male with an atypical presentation of GBS demonstrating overlapping features of the PCB and descending AMAN variants. Prompt recognition and early IVIG administration resulted in favourable recovery. Clinicians should remain aware of such variant overlaps to avoid diagnostic delay and initiate timely treatment.

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Conflict of Interest: There are no conflicts to declare.

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