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RAPID CLINICAL RECOVERY IN GUILLAIN–BARRÉ SYNDROME WITH MIXED AXONAL AND DEMYELINATING SENSORIMOTOR POLYRADICULONEUROPATHY FOLLOWING INTRAVENOUS IMMUNOGLOBULIN THERAPY: A CASE REPORT

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ABSTRACT

Background: Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness and areflexia. Mixed axonal and demyelinating variants are relatively uncommon and may present with severe quadriparesis. **Case Presentation:** A 60-year-old male with a history of hypertension and ischemic heart disease presented with rapidly progressive bilateral lower limb weakness followed by upper limb weakness. Neurological examination revealed flaccid quadriparesis (MRC grade 0/5 in all four limbs), generalized areflexia, bilateral flexor plantar responses, and distal sensory abnormalities. Routine laboratory investigations were within normal limits except for an elevated CK-MB of 1834 U/L. MRI of the cervical and lumbar spine demonstrated only mild degenerative changes without spinal cord pathology. Nerve conduction studies showed symmetrical mixed axonal and demyelinating sensorimotor polyradiculoneuropathy involving all four limbs, consistent with Guillain–Barré syndrome. The patient received intravenous immunoglobulin (IVIG) at 2 g/kg over 5 days, resulting in marked neurological improvement. **Conclusion:** Early diagnosis using clinical examination and electrophysiological studies, followed by prompt IVIG therapy, resulted in rapid neurological recovery.

KEYWORDS

Guillain–barré Syndrome, IVIG, Axonal Neuropathy, Demyelinating Neuropathy, Quadriparesis

INTRODUCTION:
GBS is the most common cause of acute flaccid paralysis and requires prompt recognition and immunotherapy.

CASE REPORT:
A 65-year-old male with hypertension and ischemic heart disease presented with a two-day history of ascending weakness involving both lower limbs followed by upper limbs. Neurological examination revealed flaccid quadriparesis (MRC 0/5), generalized areflexia, bilateral flexor plantar responses and distal sensory impairment.

Routine laboratory investigations were unremarkable. Nerve conduction study revealed symmetrical mixed axonal and demyelinating sensorimotor polyradiculoneuropathy involving all four limbs, consistent with GBS. CSF examination was not performed.

The patient received IVIG 2 g/kg over five days. By day 5, deep tendon reflexes returned and power improved to 4/5 in both upper limbs and 5/5 in both lower limbs.

DISCUSSION:
Early clinical recognition together with electrophysiological confirmation enabled timely IVIG therapy and excellent neurological recovery.

CONCLUSION: Prompt diagnosis and treatment of GBS remain crucial for favorable outcomes.

Figure 1: Nerve conduction study demonstrating mixed axonal and demyelinating sensorimotor polyradiculoneuropathy.