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A CASE OF ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY (AMSAN) VARIANT OF GUILLAIN-BARRÉ SYNDROME PRESENTING WITH PARADOXICAL HYPERREFLEXIA AND INVERTED SUPINATOR REFLEX

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INTRODUCTION

Guillain-Barré Syndrome (GBS) encompasses a heterogeneous spectrum of acute, immune-mediated polyradiculoneuropathies, conventionally characterized by progressive, ascending flaccid paralysis, sensory disturbances, and areflexia. While the demyelinating form (AIDP) predominates in Western populations, axonal variants such as AMAN and AMSAN are increasingly recognized, particularly in Asian demographics. AMSAN is a fulminant and severe variant defined by primary axonal degeneration of both motor and sensory fibers, often associated with delayed recovery and significant residual disability. However, the presence of hyperreflexia—a sign classically attributed to Upper Motor Neuron (UMN) pathology—creates a profound diagnostic dilemma, often mimicking CNS etiologies like acute myelopathy or vasculitic neuropathy.

This paper presents a rare and diagnostically challenging case of a 48-year-old female, Ms. Ramalakshmi, who presented with acute-onset bilateral lower limb weakness and distal sensory loss following a two-month prodrome of polyarthralgia. The clinical picture was confounded by significant hyperreflexia (3+) and an inverted supinator reflex, a sign traditionally pathognomonic for cervical cord compression at the C5-C6 level. Extensive investigations, including MRI of the neuraxis, CSF analysis, and a comprehensive immunological panel (ANA, ANCA, C3, C4), were employed to systematically exclude compressive myelopathy, systemic vasculitis, and connective tissue disorders. NCS ultimately confirmed Sensory Motor Axonal Neuropathy, leading to the diagnosis of AMSAN variant of GBS, with a favorable response to IVIG.

The Spectrum of Guillain-Barré Syndrome

GBS represents the most frequent cause of acute flaccid paralysis worldwide, with an estimated annual incidence of 0.8 to 1.9 cases per 100,000 individuals. GBS is now understood as a clinical syndrome encompassing several distinct subtypes: AIDP, AMAN, AMSAN, and Miller Fisher Syndrome (MFS). The pathophysiological hallmark is an aberrant immune response against peripheral nerve antigens, often triggered by antecedent infection. In axonal variants (AMAN/AMSAN), primary antibody binding to gangliosides (GM1, GD1a) at Nodes of Ranvier leads to complement-mediated axonal degeneration.

Pathology of the AMSAN Variant

AMSAN is characterized by rapid and extensive degeneration of both motor and sensory axons, with wallerian-like degeneration of myelinated fibers and minimal inflammation or demyelination. The clinical presentation is typically more severe than other forms, often leading to rapid quadriparesis, profound sensory loss, and a higher incidence of respiratory failure. Because axonal regeneration proceeds at approximately 1 mm per day, recovery in AMSAN is frequently prolonged and incomplete.

Hyperreflexia in Axonal GBS

Hyperreflexia has been documented in 33–48% of axonal GBS (AMAN) cases in Chinese and Japanese cohorts. Proposed mechanisms include:

1. **Spinal Disinhibition:** Anti-ganglioside antibodies binding to inhibitory interneurons in the spinal cord.
2. **Axonal Hyperexcitability:** Altered sodium channel function at Nodes of Ranvier leading to repetitive motor neuron firing.
3. **Conduction Block Differential:** Preferential involvement of large-diameter sensory afferents disrupting the reflex arc balance.

3. CASE REPORT

Ms. Ramalakshmi, a 48-year-old female, was admitted to the Department of Internal Medicine at Tirunelveli Medical College Hospital (IP No: 131337) on November 12, 2025, and discharged on November 29, 2025.

Chief Complaints

1. Bilateral lower limb weakness of acute onset, 3 days prior to admission.
2. Difficulty walking and decreased sensation below the ankles, 5 days prior to admission.
3. Pain over multiple large joints (elbows, knees, shoulders) for 1–2 months before onset of weakness.

History of Present Illness

The patient developed insidious onset diffuse polyarthralgia approximately two months before admission. Five days prior to admission, she first noticed distal paresthesias and reduced sensation below the ankles, rapidly followed by motor weakness in the lower limbs progressing over 48 hours. The weakness was bilateral and relatively symmetric. There was no history of fever, diarrhea, respiratory infection, back pain, bowel/bladder disturbance, or recent vaccination.

General Examination

Conscious and oriented. Pallor (+). Angular stomatitis (+). Bilateral hyperpigmentation over great toes. No icterus, cyanosis, clubbing, lymphadenopathy, or pedal edema. BP: 120/80 mmHg; PR: 98 bpm; RR: 17/min; SpO2: 97% on room air; Afebrile.

Neurological Examination

Higher mental functions normal (MMSE 25/25). All cranial nerves intact. Normal bulk; no wasting or fasciculations. Tone normal in all four limbs.

Power (MRC Grade): Shoulder/Elbow/Wrist/Fingers: 4+/5 bilaterally; Hip/Knee: 4-/5 bilaterally; Ankle/Toes: 3/5 bilaterally. Weakness more pronounced distally and in lower limbs.

Reflexes: Biceps 3+ (Brisk); Triceps 3+ (Brisk); Supinator — **Inverted** (finger flexion elicited instead of elbow flexion/supination); Knee 3+ (Brisk); Ankle — **Absent** bilaterally. Plantar reflexes flexor; Hoffman's and Wartenberg's signs absent.

Sensory: Fine touch, pain, and temperature lost bilaterally below mid-leg. Vibration and position sense lost bilaterally below the ankle.

Gait: High stepping gait, characteristic of distal weakness and sensory ataxia.

3.4 Investigation Results

Hematology and Biochemistry

Test	Admission	Discharge	Comment
Hemoglobin	9.8 g/dL	12.4 g/dL	Microcytic Hypochromic Anemia
Total WBC	13,000 /cumm	—	Neutrophilic Leucocytosis
Platelets	4.62 Lakhs/cumm	3.13 Lakhs/cumm	Reactive Thrombocytosis
ESR	105 mm/hr	95 mm/hr	Significantly Elevated
CRP	24.6 mg/L	5.3 mg/L	Elevated (Inflammation)
Serum LDH	506 U/L	—	Elevated
Serum Urea	22.6 mg/dL	19.1 mg/dL	Normal
Serum Creatinine	0.9 mg/dL	0.87 mg/dL	Normal
Serum Sodium	146 mEq/L	140 mEq/L	Normal
Serum Potassium	4.2 mEq/L	3.5 mEq/L	Normal

Immunology and Serology

ANA: Positive (1+), AC-20 pattern, titer 1:100 — low/borderline, likely non-specific. c-ANCA (Anti-PR3): Negative. p-ANCA (Anti-MPO): Negative. C3: 1.450 g/L (Normal). C4: 0.450 g/L (mildly elevated — acute phase reactant). HBsAg, HCV, HIV: Negative.

CSF Analysis

Sugar: 84.6 mg/dL. Protein: 30.7 mg/dL. Cell count: Nil (acellular). Globulin: Negative. No albuminocytological dissociation — a common finding in the first week of GBS.

Electrophysiology (NCS)

Impression: **Sensory Motor Axonal Neuropathy.** This confirmed the axonal variant (AMSAN) over the demyelinating variant (AIDP), with low amplitudes rather than slow conduction velocities.

Radiological Imaging

MRI Brain: Grade 1 small vessel ischemic changes; no acute infarcts, space-occupying lesions, or demyelination. MRI Cervical Spine: Disc desiccation at all levels; mild disc bulge at C4-C5 and C5-C6 with anterior thecal sac indentation. **No compressive myeloradiculopathy or cord signal changes identified** — effectively ruling out a structural cause for the hyperreflexia and inverted supinator reflex. MRI Lumbar Spine: Diffuse disc bulge at L4-L5 with lateral recess narrowing.

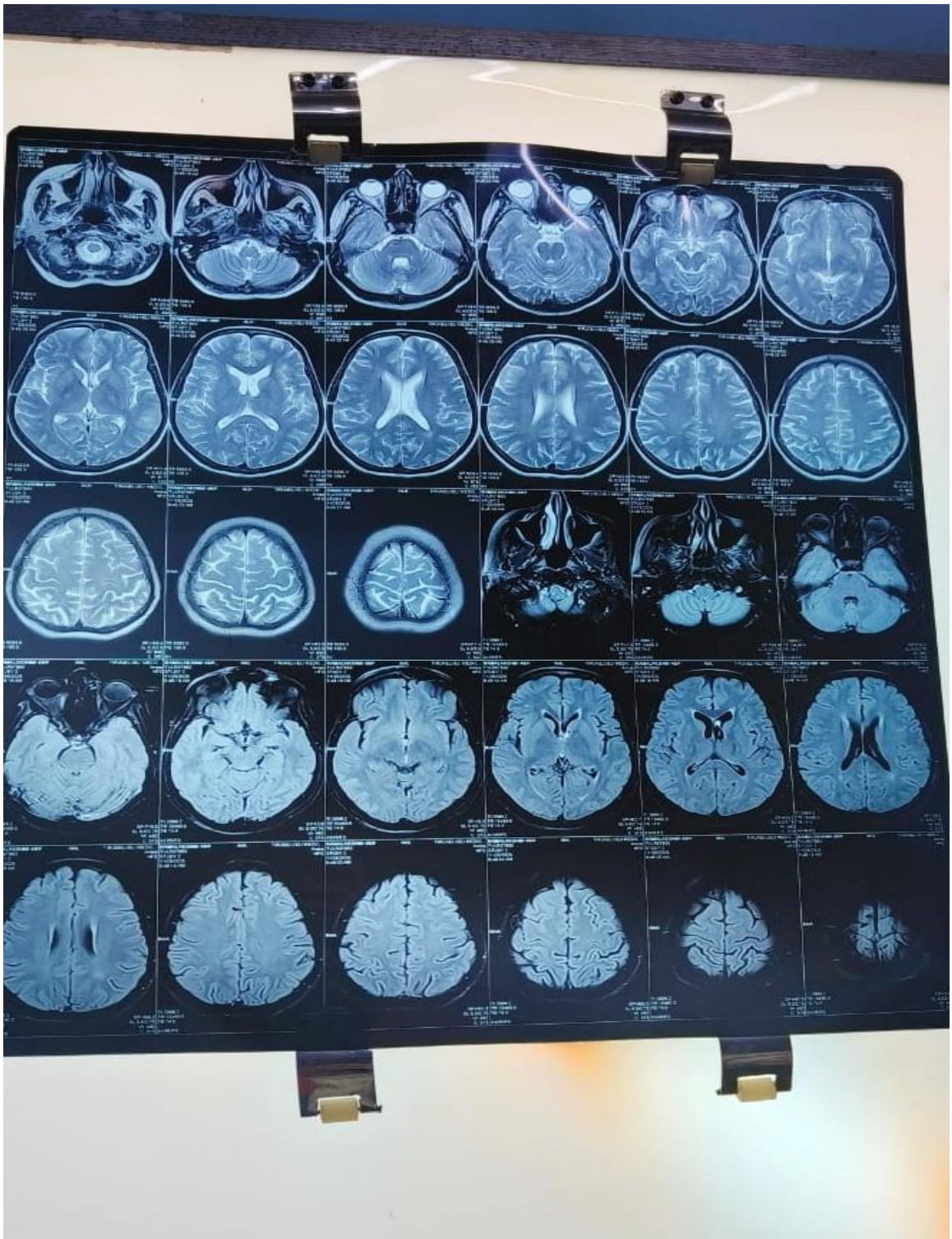

Figure 1: MRI Brain (axial sequences) of Ms. Ramalakshmi. *Multiple sequences including T2-weighted and FLAIR images are displayed. Findings showed Grade 1 small vessel ischemic changes with no acute infarcts, space-occupying lesions, or demyelinating plaques, effectively excluding central nervous system pathology as the cause of the patient's neurological signs.*

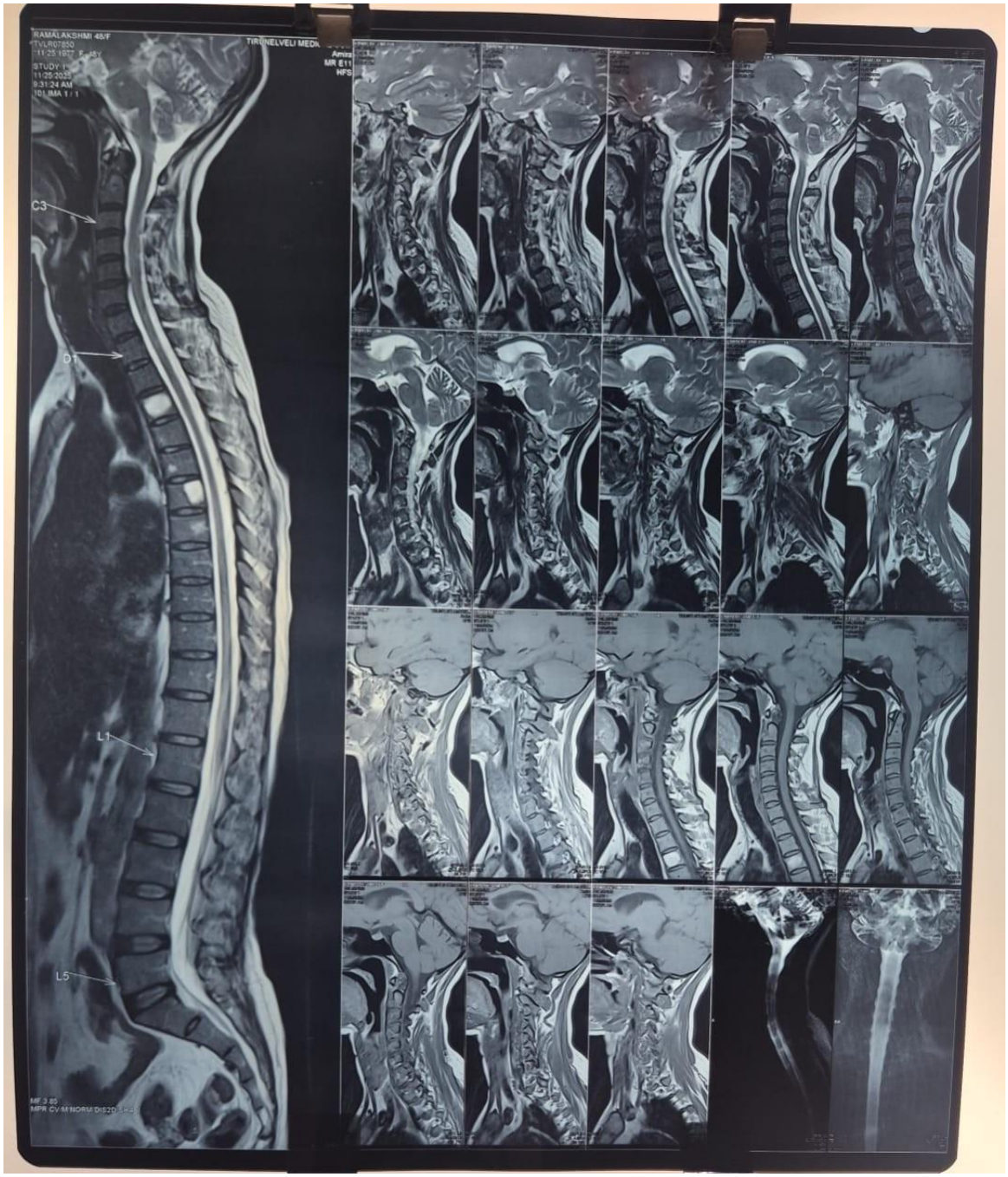

Figure 2: MRI Cervical and Lumbar Spine of Ms. Ramalakshmi (Tirunelveli Medical College). *The sagittal whole-spine scout (left) shows vertebral levels C3, D1, L1, and L5. Parasagittal sequences of the cervical spine reveal disc desiccation at all levels with mild disc bulges at C4-C5 and C5-C6 causing anterior thecal sac indentation.* ***No compressive myeloradiculopathy or cord signal changes are identified****, confirming that the paradoxical hyperreflexia and inverted supinator reflex were of peripheral/immunological origin rather than structural.*

Management

Therapeutic Regimen

1. **IVIG:** 20 grams/day IV for 5 doses.
2. **Corticosteroids:** Methylprednisolone 1 gram IV for 7 days, followed by tapering oral Prednisolone (30 mg/day reducing).
3. **Anemia Correction:** Iron Sucrose 200 mg IV OD and Vitamin B12 100 mcg IV OD for 12 days.
4. **Supportive:** Pregabalin 75 mg, Pantoprazole, IV fluids.

Complications: Superficial thrombophlebitis of the right wrist progressing to abscess formation, managed with incision and drainage (29/11/2025) and antibiotics (Amoxycillin + Cloxacillin).

Condition at Discharge: Sensory level receded from mid-leg to below the ankle. Conscious, oriented, vitals stable. Discharged with physiotherapy, oral steroid tapering, and neurology follow-up.

4. DISCUSSION

The case of Ms. Ramalakshmi deviated from the classic GBS phenotype in two critical ways: a 2-month prodromal polyarthralgia suggesting systemic inflammatory etiology, and hyperreflexia (3+) with an inverted supinator reflex pointing toward CNS pathology.

Excluding Autoimmune Vasculitis and CTD

The negative ANCA profile, normal-to-high complement levels, and symmetric onset allowed vasculitic neuropathy to be excluded. The low-titer ANA (1:100, AC-20) was deemed non-specific, found in up to 20% of healthy individuals, with no supporting features of Antisynthetase Syndrome.

Excluding Cervical Myelopathy

The inverted supinator reflex is a classic localizing sign for cervical myelopathy at C5-C6. MRI cervical spine (Figure 2) was the definitive investigation, showing only mild disc bulges with no cord compression or myelomalacia, confirming that the 'central' signs were not due to structural cord pathology. MRI brain (Figure 1) likewise excluded demyelination or acute infarcts.

Establishing the AMSAN Diagnosis

NCS demonstrated Sensory Motor Axonal Neuropathy (low amplitudes), confirming AMSAN. Normal CSF protein is consistent with the first week of GBS, where albuminocytological dissociation is absent in up to 50% of cases. The hyperreflexia is explained by spinal interneuron disinhibition via anti-ganglioside antibodies and distal axonal pathology sparing the proximal reflex arc.

Differential Diagnosis Summary

Differential Diagnosis	Points Favoring	Points Against
Cervical Myelopathy	Hyperreflexia, Inverted Supinator Reflex	MRI Spine: No compression/ signal change (Figure 2). NCS: Axonal pattern.
Vasculitic Neuropathy	Prodromal joint pain, High ESR/CRP	ANCA: Negative. C3/C4: Normal. GBS symmetric; vasculitis asymmetric.
Autoimmune (SLE/Sjogren's)	Joint pain, ANA 1+ (AC-20)	No rash/renal/mucosal involvement. ANA low titer (1:100) non-specific.
Transverse Myelitis	Paraparesis, sensory level	MRI: No cord inflammation (Figures 1 & 2). CSF: Acellular. NCS: Peripheral neuropathy.
AMSAN (GBS)	Acute course, NCS findings, Sensory loss, IVIG response	Hyperreflexia atypical (but documented in axonal variants).

CONCLUSION

This report documents a rare case of Acute Motor and Sensory Axonal Neuropathy (AMSAN) in a 48-year-old female, distinguished by the presence of hyperreflexia and an inverted supinator reflex. Diagnosis was achieved through rigorous exclusion: MRI spine (Figure 2) ruled out compressive myelopathy; MRI brain (Figure 1) excluded CNS demyelination and infarction; immunological workup (ANA, ANCA, C3, C4) ruled out vasculitic neuropathy; and NCS definitively identified Sensory Motor Axonal Neuropathy.

The patient was treated with IVIG (20g × 5 days) and corticosteroids, resulting in stabilization of weakness and improvement in sensory deficits. This case serves as a vital reminder that hyperreflexia does not exclude GBS, particularly the axonal variants (AMAN/AMSAN), and that the inverted supinator reflex can occur in peripheral neuropathies due to complex spinal reflex disinhibition. Early recognition prevents diagnostic delays and facilitates life-saving immunotherapy.