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# A Case Report on Acute Motor Axonal Neuropathy Variant of Guillain-Barré Syndrome

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**ABSTRACT**

Guillain-Barré Syndrome (GBS) is a rare autoimmune disorder that affects the peripheral nerves and is known for its rapid onset of muscle weakness and unusual sensory occurrences. This case study is about a 75-year-old female who complained of numbness in her right middle finger which eventually led to bilateral lower extremity weakness and unsteady gait. Past medical history included carcinoma of the ascending colon and bronchial asthma. Initial treatments at another hospital included ceftriaxone and pregabalin, but her condition worsened. Lab tests showed anemia, high ESR, and lymphocytic pleocytosis in the CSF. From the clinical presentation and diagnostic tests, the patient was found to have an acute motor axonal neuropathy (AMAN) variant of GBS. Treatment consisted of IVIg, gabapentin, and other supportive meds. After treatment the patient responded very well to the treatment and was released with a prescription for some medicines. This example illustrates how crucial it is to recognize and treat GBS, especially the AMAN variant early, because it would have made a big difference in the patient's prognosis. It also stresses the continued research for new treatments and neuroprotective approaches in the treatment of nerve damage in GBS. This study hopes to add to the knowledge of AMAN in relation to GBS and it's treatment.

**Keywords:** Guillain-Barré Syndrome, acute motor axonal neuropathy, autoimmune disorder, peripheral nervous system, Acute Inflammatory Demyelinating Polyneuropathy.

## INTRODUCTION.

Guillain-Barré syndrome is a rare autoimmune condition that develops over a period of days to weeks, primarily affecting the peripheral nervous system, located outside the brain and spinal cord. It is characterized by an immune-mediated attack on peripheral nerves, leading to progressive muscle weakness and sensory abnormalities [1]. In around 5% of GBS cases results in mortality, and extreme muscular weakness. In addition to this motor deficits, patients may also have respiratory insufficiency, autonomic dysfunction, and discomfort. Although GBS can affect individuals of all age, it is more prevalent among adults, especially among males.[2]

The incidences of GBS varies from 0.30 to 6.08 cases per 100,000 inhabitants and 0.42 to 6.58 cases per 100,000 person-years throughout the cohort studies. The risk incidence in the self-controlled studies were estimated to be around 1.73 and 4.30 cases per 100,000 person-years and around 0.072 and 1 case per 100,000 inhabitants [3].

The GBS progresses through 3 distinct stages which includes the progressive phase, which lasts for days to four weeks; the plateau period, which lasts for days to months and shows minimal clinical change; and the healing phase. Approximately about 98% of patients will reach their lowest neurologic function by 4 weeks, and around 75% will reach by 7 days [4].

The two most prevalent subtypes of GBS includes Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). In North America and Europe, nearly 90% of GBS patients develop AIDP [6]. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is a variant of GBS characterized by immune mediated demyelination, affecting the impulse transmission through modified internodal and paranodal membranes [5]. Acute motor axonal neuropathy (AMAN) is a non-inflammatory condition in which the body's immune system attacks and destroys motor nerve cell axons specifically. The axons surrounding the myelin sheath remain intact [6]. Unlike AIDP, AMAN is distinguished by minimal lymphocytic infiltration and preservation of sensory nerves, dorsal nerve roots, and dorsal root ganglia. The initial pathological changes in AMAN include elongation of the nodes of Ranvier and macrophage recruitment to the nodal region [7].

In the general population, acute inflammatory demyelinating polyneuropathy (AIDP) affects 0.75 to 2 out of every 100,000 people annually [8]. Only 3-5% of infections in Western nations are of the axonal kinds (AMAN and acute motor-sensory axonal neuropathy, AMSAN; see this term), but 30% to 50% of GBS cases occur in Asia and Latin America [9].

This case study focuses on a patient who came with a complaint of numbness of limbs and was later diagnosed with the AMAN variant of GBS, a rare neurological disorder, and the treatment approach.

## CASE REPORT

A 75-year-old female patient came with the complaint of right middle finger numbness followed by weakness for 5 days which progressed to the left leg foot, numbness of both feet and weakness of both lower limbs. She also had difficulty in walking. She was diagnosed with Carcinoma (CA) Ascending colon, 4 months back and Bronchial Asthma (BA) 13 years back. She was admitted to another hospital and was given ceftriaxone and pregabalin. Her vitals were stable. She had an elevated Erythrocyte Sedimentation Rate (ESR) and elevated CSF lymphocytes and was anaemic, as shown in Table 1.

Table 1: Lab investigations

| LAB PARAMETERS | OBSERVED VALUE | NORMAL VALUE |
|---|---|---|
| CLINICAL HEMATOLOGY | | |
| Hemoglobin(Hb) | 10.2 g/dl | 12-16 g/dl |
| Packed Cell Volume (PCV) | 32.8% | 36-47 % |
| Total Count (TC) | 9710 /$mm^3$ | 4000-10000 /$mm^3$ |
| ESR | 70 mm/hr | 0-20 mm/hr |
| BLOOD SUGAR | | |
| Random (RBS) | 200 mg/dl | 70-140 mg/dl |
| HbA1c | 5.5% | <6.5 % |
| ELECTROLYTES | | |
| Sodium | 135 mmol/L | 135-145 mmol/L |
| Potassium | 4.29 mmol/L | 3.5-5 mmol/L |
| LIVER FUNCTION TEST | | |
| Aspartate Aminotransferase (AST) | 15 U/L | 0-40 U/L |
| Alanine Aminotransferase (ALT) | 30 U/L | 0-30 U/L |
| Total Bilirubin | 0.24 mg/dl | 0.1-1 mg/dl |
| Direct Bilirubin | 0.14 mg/dl | 0.2-0.6 mg/dl |
| RENAL FUNCTION TEST | | |
| Serum Creatinine | 0.92 mg/dl | 0.8-1.3 mg/dl |
| CSF EXAMINATION | | |
| Volume | 3.0 ml | |
| Colour | Colourless | |
| Appearance | Clear | |
| Xanthochromasa | Nil | |
| Total count | 01/ $mm^3$ | <4/$mm^3$ |
| RBC | Nil | Nil |
| Lymphocytes | 100% | 40-80 % |
| Sugar | 75 mg/dl | 40-70 mg/dl |
| Protein | 19 mg/dl | 15-45 mg/dl |

The patient also had an elevated Rheumatoid Factor (RF). Her Dorsal column spinal cord stimulation (DCS) revealed motor pre-axonal polyneuropathy involving both upper and lower extremities. Her urine culture revealed occasional gram-negative bacilli. From the subjective and objective evidence, she was diagnosed with acute polyradiculoneuropathy- an Acute Motor Axonal Neuropathy variant of Guillain-Barré Syndrome. She was treated with

Intravenous Immunoglobulin (IV-IG), gabapentin, nortryptilline, buprenorphine, ademethionine, pantoprazole and vitamins (Table 2). Once the patient's condition improved, she was discharged with the medication listed in Table 3.

Table 2: Drug Chart

| S.No | Drugs Prescribed | | Dose | Route | Frequency | Duration | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Brand Name | Generic Name | | | | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 1 | T. Tayo | Cholecalciferol | 60k | PO | Weekly once | ✓ | | | | | | |
| 2 | Inj. Pan | Pantoprazole | 40mg | PO | OD | | ✓ | ✓ | ✓ | ✓ | | |
| 3 | Inj. Renerve Plus | Multivitamin | 1cc | IV | OD | | ✓ | ✓ | ✓ | ✓ | | |
| 4 | T. Palmiges | Palmitoylethanolamide, Genistein and Daidzein | 1tab | PO | BD | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| 5 | T. Gabapentin | Gabapentin | 400mg | PO | HS | | ✓ | | | | | |
| 6 | Buvalor patch | Buprenorphine | 10mg | PO | OD | | | | ✓ | | | |
| 7 | T. Pregabalin | Vitamin B12 | 75mg | PO | OD | | | | ✓ | ✓ | ✓ | ✓ |
| 8 | T. Gabastone NT | Gabapentin and Nortriptyline | 400mg | PO | HS | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| 9 | T. Gladsam | S-Adenosyl- L-Methionine | 200mg | PO | BD | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| 10 | T. Pan | Pantoprazole | 40mg | PO | OD | | | | | | ✓ | ✓ |
| 11 | IV IG | Intravenous Immunoglobulin | 20g | IV | OD | | | ✓ | ✓ | | | |
| | | | 30g | IV | OD | | | | | ✓ | ✓ | |

Table 3: Drugs On Discharge

| S. No | DRUGS PRESCRIBED | | DOSE | ROUTE | FREQUENCY | DURATION |
|---|---|---|---|---|---|---|
| | BRAND NAME | GENERIC NAME | | | | |
| 1 | T. Gabastone NT | Gabapentin and Nortriptyline | 400mg | PO | HS | × To Continue |
| 2 | T. Palmiges | Palmitoylethanolamide, Genistein and Daidzein | 1tab | PO | BD | × To Continue |
| 3 | T. Tayo | Cholecalciferol | 60k | PO | Weekly once | × 5 weeks |
| 4 | T. Gladsam | S-Adenosyl- L-Methionine | 200mg | PO | OD | × To Continue |
| 5 | T. Pan | Pantoprazole | 40mg | PO | OD | × To Continue |
| 6 | T. Pregabalin | Vitamin B12 | 75mg | PO | OD | × To Continue |
| 7 | T. Semiultracet | Tramadol + Acetaminophen | 1 tab | PO | BD | × SOS |
| 8 | T. Clavam | Amoxicillin + Potassium Clavunalate | 625mg | PO | BD | × 5days |
| 9 | T. Chymoforte | Trypsin + Chymotrypsin | 1tab | PO | BD | × 5days |
| 10 | C. Bifilac | Probiotic | 1cap | PO | BD | × 2 days and SOS |
| 11 | T. Citravite | Vitamin C | 1tab | PO | OD | × 2 WEEKS |

## DISCUSSION

In addition to the basic appearance of GBS, clinical variations are determined by the major method of fibre injury (axonal vs demyelinating), the presence of altered awareness, and the types of nerve fibres affected (motor, sensory, sensory and motor, cranial or autonomic) [10]. Patients with AMAN collectively experience extended paralysis and respiratory failure over a few days, with a faster trend of weakening to an earlier nadir than those with AIDP. Acute

motor conduction block neuropathy was coined because AMAN might manifest as a temporary conduction block without axonal damage [11]. The primary clinical difference between the Acute Motor Axonal Neuropathy (AMAN) and Acute Inflammatory Demyelinating Polyneuropathy (AIDP) variants of Guillain-Barre Syndrome (GBS) is that AMAN usually manifests a faster progression of severe muscle weakness with minimal sensory involvement, whereas AIDP typically exhibits a slower progression of weakness accompanied by sensory symptoms due to damage to the myelin sheath on the nerves, frequently with more prominent involvement of cranial nerves. In other words, AMAN is primarily characterised by damage to the motor nerves, whereas AIDP involves both motor and sensory nerves as a result of demyelination [12]. Figure 1 represents the pattern of symptoms in variants of Guillain–Barré syndrome.

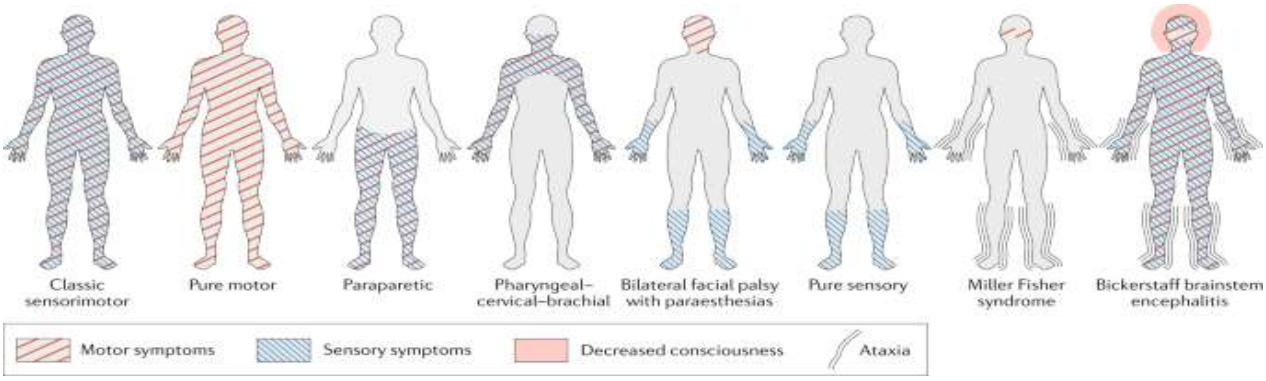


Figure 1: Pattern of symptoms in variants of Guillain–Barré syndrome

The diagnosis of GBS is made using clinical history and examination, with the help of auxiliary tests such CSF analysis and electrodiagnostic studies, in the absence of adequately sensitive and specific disease biomarkers [13]. In this case report, the patient was diagnosed based on the same criteria and was treated with intravenous immunoglobulin which is proved to be effective in improving the patient's outcome [14]

## CONCLUSION

Early diagnosis of Guillain-Barre syndrome might be challenging. Its symptoms can differ from person to person and are comparable to those of other illnesses. However, a patient's condition can be significantly improved by early GBS discovery. Patients with GBS who do not react to PE or IVIg may have additional effective choices with newly developed immunomodulatory treatments. Given our growing understanding of the processes causing nerve damage and axonal degeneration, future initiatives may involve implementing neuroprotective therapies.

**REFERENCE**

1. Marcus R. What Is Guillain-Barré Syndrome? JAMA. 2023;329(7):602.
2. van Doorn PA, Van den Bergh PYK, Hadden RDM, Avau B, Vankrunkelsven P, Attarian S, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain–Barré syndrome. J Peripher Nerv Syst. 2023;28(4):535–63.
3. van Doorn PA, Van den Bergh PYK, Hadden RDM, Avau B, Vankrunkelsven P, Attarian S, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain–Barré syndrome. J Peripher Nerv Syst. 2023;28(4):535–63.
4. Wachira VK, Farinasso CM, Silva RB, Peixoto HM, de Oliveira MRF. Incidence of Guillain-Barré syndrome in the world between 1985 and 2020: A systematic review. Glob Epidemiol. 2023;5(100098):100098.
5. Guillain–Barré syndrome (GBS) Clinical Guideline. Alderney. nhs. uk
6. Tan JK, Dhayalen K, Hazfadzila MU, Remli R, Tan HJ. P-NE006. Atypical presentation of Guillain-Barré syndrome (GBS) in acute inflammatory demyelinating polyneuropathy (AIDP) with conduction block and its association with anti-sulfatide antibody: A case report. Clin Neurophysiol. 2021;132(8):e90.
7. Marzoughi S, Marulanda L, Ngo D, Chen T. Pearls & oysters: Delayed diagnosis of acute motor axonal neuropathy with cardiac arrest. Neurology [Internet]. 2022;98(8):336–9.
8. Patel P, Shah D, Jani C, Shah J, Jani R, Kelaiya A, et al. Outcomes of patients presenting with Guillain-Barre Syndrome at a tertiary care center in India. BMC Neurol. 2022;22(1).
9. Mansour M, Ouerdiene A, Bedoui I, Kacem A, Zaouali J, Mrissa R. Acute-onset chronic inflammatory demyelinating polyneuropathy with cranial nerves and respiratory tract involvement: A case report. Clin Case Rep [Internet]. 2020;8(11):2199–203.
10. Xu L, Zhao C, Bao Y, Liu Y, Liang Y, Wei J, et al. Variation in worldwide incidence of Guillain-Barré syndrome: a population-based study in urban China and existing global evidence. Front Immunol [Internet]. 2024;15:1415986.

11. Berciano J. Axonal pathology in early stages of Guillain-Barré syndrome. Neurol (Engl Ed). 2022;37(6):466–79.

12. Sriwastava S, Kataria S, Tandon M, Patel J, Patel R, Jowkar A, et al. Guillain Barré Syndrome and its variants as a manifestation of COVID-19: A systematic review of case reports and case series. J Neurol Sci. 2021;420(117263):117263.

13. Khedr EM, Shehab MM, Mohamed MZ, Mohamed KO. Early electrophysiological study variants and their relationship with clinical presentation and outcomes of patients with Guillain-Barré syndrome. Sci Rep. 2023;13(1).

14. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Diagnosis and management of Guillain–Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671–83.

15. Sidow NO, Hassan MS. Intravenous immunoglobulin treatment with prognosis for the first six months of Guillain–Barré Syndrome in Somalia: Case series. Ann Med Surg (Lond). 2022;84.