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Acute motor axonal neuropathy in Ecuador: a retrospective case series with electrophysiological and functional correlation

Neuropatía axonal motora aguda en Ecuador: Una serie de casos retrospectiva con correlación electrofisiológica y functional

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ABSTRACT

Acute Motor Axonal Neuropathy (AMAN) is a severe Guillain–Barré Syndrome (GBS) variant characterized by acute flaccid paralysis and pure motor axonal involvement. In Latin America, its diagnosis remains challenging due to limited access to neurophysiological tools. This case series describes three Ecuadorian male patients diagnosed with AMAN between 2017 and 2025, each with recent gastrointestinal infection. Electromyography confirmed axonal motor neuropathy in all cases. One patient, a high-performance cyclist, required mechanical ventilation and fully recovered after IVIG. Another, a retired officer, showed partial improvement and remains orthotic-dependent. The third, a former conscript, developed irreversible quadriplegia despite treatment and prolonged ICU stay. Antiganglioside antibodies were tested in one case, revealing GD1a, GD1b, and GT1b positivity. These cases demonstrate the clinical heterogeneity

of AMAN and the importance of early diagnosis, timely immunotherapy, and structured rehabilitation. Increased awareness and improved diagnostic capacity are essential to optimize outcomes in AMAN patients in resource-limited settings.

Keywords: Guillain–Barré Syndrome, AMAN, acute flaccid paralysis, axonal neuropathy, neurophysiology

RESUMEN

La neuropatía axonal motora aguda (AMAN) es una variante grave del síndrome de Guillain-Barré (SGB), caracterizada por parálisis flácida aguda de origen motor sin compromiso sensitivo. Su diagnóstico en América Latina es complejo debido al limitado acceso a estudios electrofisiológicos. Esta serie de casos describe tres pacientes varones ecuatorianos diagnosticados con AMAN entre 2017 y 2025, todos con antecedentes de infección gastrointestinal. Los estudios de electromiografía confirmaron neuropatía axonal motora pura en los tres casos. Un paciente, ciclista de alto rendimiento, requirió ventilación mecánica transitoria y recuperó su funcionalidad tras recibir inmunoglobulina intravenosa. Otro, militar retirado, presentó recuperación parcial y actualmente depende de órtesis. El tercero, exconscripto, desarrolló cuadriplejía irreversible a pesar del tratamiento y una estancia prolongada en UCI. Solo uno de ellos fue evaluado con anticuerpos antiganglósido, encontrándose positividad a GD1a, GD1b y GT1b. Estos casos evidencian la heterogeneidad clínica del AMAN y refuerzan la necesidad de un diagnóstico precoz, tratamiento oportuno y rehabilitación estructurada. Es indispensable fortalecer la capacidad diagnóstica y la conciencia clínica sobre esta variante del SGB en contextos con recursos limitados.

Palabras clave: Síndrome de Guillain-Barré, AMAN, parálisis flácida aguda, neuropatía axonal, neurofisiología

INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive, symmetrical muscle weakness and generalized areflexia. In severe cases, it may compromise respiratory function and lead to autonomic dysfunction. Among its clinical variants, Acute Motor Axonal Neuropathy (AMAN) is distinguished by predominant involvement of motor axons without significant sensory abnormalities. It has been more frequently reported in Asia and Latin America (Uncini & Yuki, 2012; Willison et al., 2016).

From an immunological standpoint, the pathogenesis of AMAN involves molecular mimicry, wherein certain pathogens share epitopes with peripheral nerve gangliosides, particularly GM1 and GD1a. This similarity induces an autoimmune response mediated by antibodies, complement activation, and subsequent axonal damage at the nodes of Ranvier (Yuki & Hartung, 2012; Susuki et al., 2007). This mechanism is well documented in post-infectious cases, especially those following *Campylobacter jejuni* infection.

However, increasing evidence suggests that various immune stressors—including infections, physical overexertion, or dermal trauma—may act as triggering events in genetically predisposed individuals. These stimuli can activate antigen-presenting cells and provoke cross-reactive autoimmune responses through loss of tolerance or bystander activation (Kuwabara et al., 2015; Luijten et al., 2022). As such, the etiological spectrum of AMAN is broader than previously assumed and should be interpreted within an integrative immunopathological framework.

Despite its well-established clinical and neurophysiological features, AMAN remains underdiagnosed in many Latin American settings due to limited access to electrophysiological studies and antiganglioside antibody testing. In Ecuador, published reports are scarce, and formal documentation is limited. This case series contributes to addressing this data gap by presenting three confirmed cases from a public hospital, supported by detailed clinical records.

All three patients developed rapidly progressive symmetrical motor weakness with absent or minimal sensory involvement, preserved consciousness, and neurophysiological findings consistent with AMAN. Their antecedents included gastrointestinal infections and, in one case, recent intense physical exertion. These observations reflect the heterogeneity of AMAN presentation and support a post-infectious autoimmune hypothesis in resource-limited settings where diagnostic tools are often unavailable.

This report presents a retrospective case series of three Ecuadorian patients diagnosed with Acute Motor Axonal Neuropathy (AMAN), each confirmed by clinical and electrophysiological criteria. By documenting their demographic, functional, and respiratory profiles, this study contributes local evidence of AMAN in Latin America and highlights the clinical heterogeneity and recovery patterns associated with post-infectious immune-mediated neuropathies.

METHODOLOGY

A retrospective, observational, and descriptive case series study was conducted involving three patients diagnosed with the acute motor axonal neuropathy (AMAN) variant of Guillain-Barré Syndrome (GBS). These cases were treated between 2017 and 2025 at a public hospital in Ecuador. This time frame was chosen to encompass all clinically compatible AMAN, three clinical cases were included, all diagnosed with the AMAN variant of GBS by nerve conduction studies. They were managed at the same ecuadorian hospital between 2017 and 2025.

Clinical information was obtained through institutional medical record review and direct interviews with patients or their relatives, using a structured data collection form specifically designed for this study. The form was reviewed by clinical professionals to ensure its relevance and comprehensiveness.

Inclusion criteria were: confirmed diagnosis of AMAN based on clinical and neurophysiological findings; recent exposure to an immunological stressor such as gastrointestinal infection or physical overexertion; and availability of complete clinical data. Cases with incomplete documentation or uncertain diagnostic confirmation were excluded.

The following variables were collected: demographic data (age, sex, occupation), relevant antecedents, latency between exposure and symptom onset, neurological presentation, cerebrospinal fluid (CSF) findings, electromyography (EMG) results, type of immunotherapy received (IVIG or plasma exchange and functional status at hospital discharge and up to three months post-onset, regardless of recovery.

Data were organized into a comparative clinical characteristics table and analyzed using qualitative descriptive methods. No inferential statistics were applied due to the limited sample size. All patient data were fully anonymized, and the hospital name was omitted to protect confidentiality. Verbal informed consent was obtained from all participants for academic and research purposes.

CASE PRESENTATION

Case 1

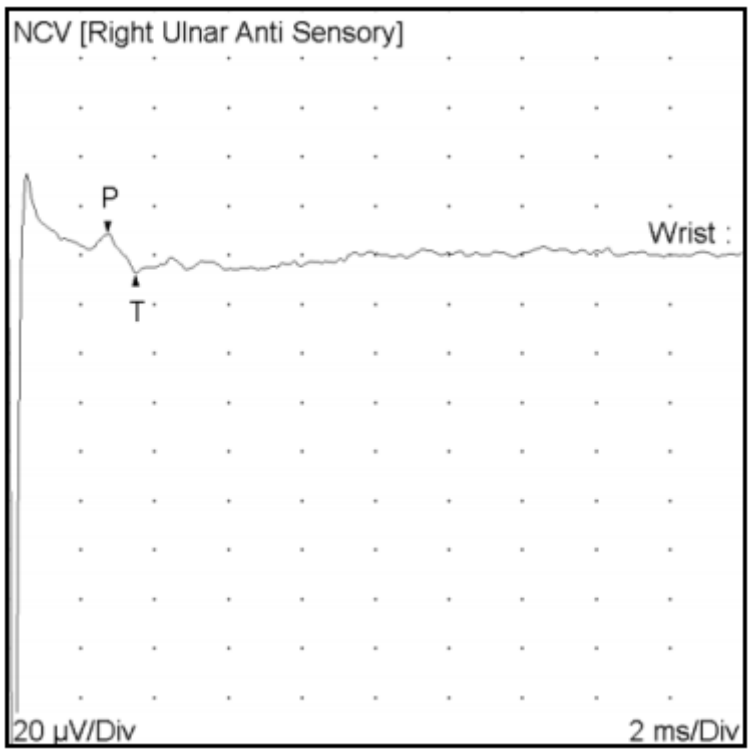
A 37-year-old Ecuadorian male, active-duty military officer and competitive cyclist, presented on April 30, 2025, with acute ascending weakness and generalized fatigue following a febrile diarrheal illness. He had no history of chronic disease, recent vaccination, or substance use, but reported ingestion of food from a street market days before symptom onset. Neurological examination on admission revealed flaccid tetraparesis (MRC 1/5 upper and lower limbs), generalized areflexia, and intact cranial nerves. Cerebrospinal fluid (CSF) analysis on May 13 showed normal cytology and biochemistry. Nerve conduction studies performed on May 16 revealed findings consistent with acute motor axonal neuropathy (AMAN). Antiganglioside antibodies (GD1a, GD1b, GT1b) were positive. The patient's initial forced vital capacity (FVC)

was 0.88 L, and he required invasive mechanical ventilation from May 16. Intravenous immunoglobulin (IVIG) was administered over five days, starting on May 14. He remained in the intensive care unit (ICU) for seven days. Upon ICU discharge, strength improved to MRC 3/5 in upper limbs and 5/5 in lower limbs. He was discharged home on May 26, 2025, walking independently and performing daily activities without assistance. He did not require tracheostomy and currently shows full neurological recovery.

A nerve conduction study and electromyography were performed, confirming the diagnosis of acute motor axonal neuropathy (AMAN). The key findings are illustrated in **Figure 1**, which displays the electrophysiological profile of the patient.

Figure 1

Nerve conduction study of the right ulnar sensory nerve in a patient with AMAN variant of Guillain-Barré Syndrome (Case 1)



The waveform shows markedly reduced amplitude and preserved latency, consistent with a predominantly axonal pattern. These findings are compatible with severe motor axonal involvement and early conduction failure.

Case 2

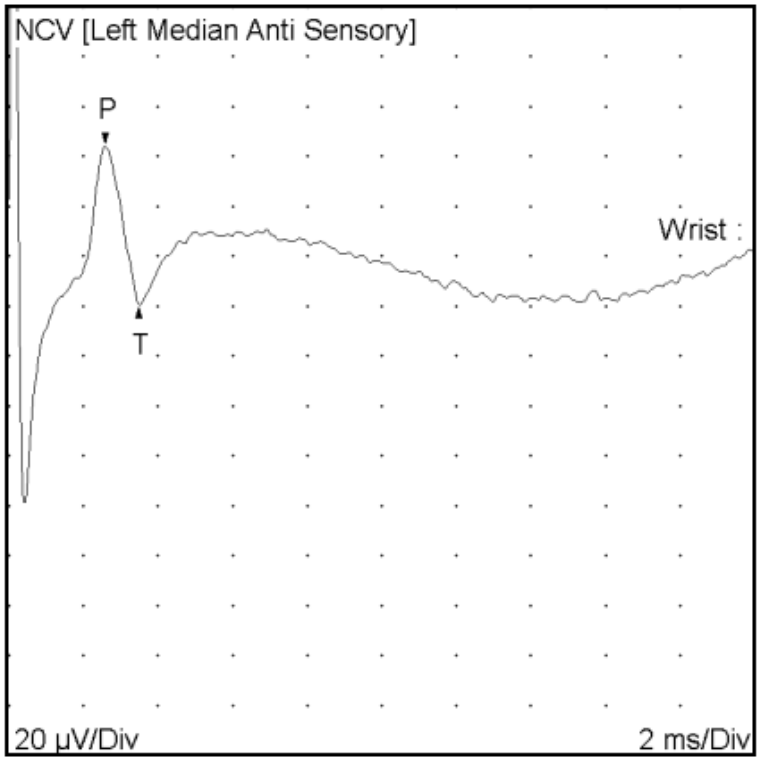
A 38-year-old Ecuadorian male, retired military officer, was admitted on April 11, 2023, with progressive lower limb weakness and areflexia that began four days after an episode of gastroenteritis (fever, diarrhea) following seafood consumption. On admission, he presented with flaccid tetraparesis (MRC 2/5), preserved consciousness, and no bulbar involvement. CSF analysis was normal. Nerve conduction studies performed the same day revealed an acute motor axonal pattern consistent with AMAN. FVC was 2.21 L. The patient received a five-day course

of IVIG starting April 11. He remained in the ICU for eight days and was discharged to the general ward with MRC scores of 3/5 in upper limbs and 1/5 in lower limbs. He was discharged home on April 26, 2023, using a wheelchair, with only minimal voluntary movement in the lower limbs. At one-year follow-up, he is partially dependent, uses orthotic devices for ambulation, and exhibits preserved reflexes and sensitivity but limited motor coordination.

The patient underwent an EMG which supported a diagnosis compatible with the AMAN variant. The relevant findings are shown in **Figure 2**, summarizing the neurophysiological characteristics.

Figure 2

Left median sensory nerve conduction in a patient with AMAN (Case 2)



The graph shows diminished amplitude and preserved latency, suggestive of axonal damage without significant demyelination. This electrophysiological profile supported the diagnosis of acute motor axonal neuropathy.

Case 3

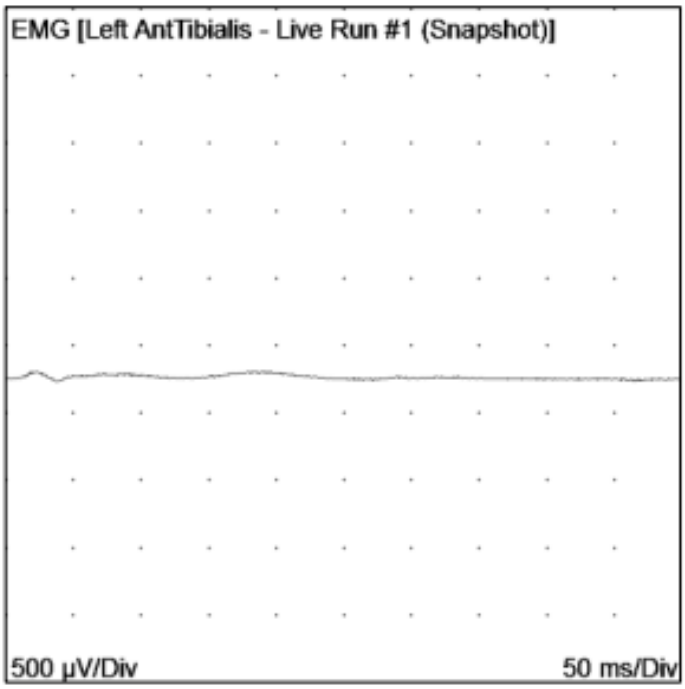
A 27-year-old Ecuadorian male, former military conscript, was admitted to a public hospital on December 6, 2017, with acute symmetrical flaccid paralysis following fever and diarrhea reported the previous week. Neurological examination revealed complete paralysis (MRC 0/5) of all limbs and absent deep tendon reflexes. CSF was normal. Electromyography on December 9 showed pure motor axonal neuropathy consistent with AMAN. His initial FVC was recorded at 20 L, though this value likely reflects a data entry error. He developed respiratory failure and was intubated on December 7. IVIG was administered over five days starting the same

day. He remained in ICU for 157 days, required tracheostomy, and experienced nosocomial infection. He was discharged from ICU on May 28, 2018, and from the hospital on June 18. At discharge, he remained wheelchair-bound, with absent reflexes, no motor or sensory recovery, and full dependency for activities of daily living. Although decannulated prior to discharge, he remains fully dependent and has not regained motor function to date.

An extensive neurophysiological assessment revealed severe motor axonal involvement with poor prognosis. The corresponding EMG patterns are depicted in **Figure 3**.

Figure 3

Electromyography (EMG) recording of the left anterior tibialis muscle in a patient with severe AMAN variant of Guillain-Barré Syndrome (Case 3)



The absence of spontaneous activity and motor unit potentials reflects complete axonal degeneration and denervation. The findings are consistent with the poor functional outcome documented in this patient.

Table 1

Clinical, Electrophysiological, and Functional Characteristics of Three Ecuadorian Patients with Acute Motor Axonal Neuropathy (AMAN), 2017–2025

Variable	Case 1	Case 2	Case 3
Sex / Age	Male, 37 years	Male, 38 years	Male, 27 years
Year of Onset	2025	2023	2017
Institutional Status	Active military / high-performance cyclist	Retired military	Former military conscript

Variable	Case 1	Case 2	Case 3
Triggering Event	Febrile gastroenteritis	Gastroenteritis (seafood-related)	Fever and diarrhea (possible arboviral illness)
Days from Trigger to Symptoms	2 days	4 days	5 days
Initial Symptoms	Flaccid tetraparesis, fatigue	Progressive paraparesis	Complete quadriplegia
Cranial Nerve Involvement	None	None	None
CSF Findings	Normal	Normal	Normal
EMG Findings	Pure motor axonal neuropathy (AMAN)	Pure motor axonal neuropathy (AMAN)	Pure motor axonal neuropathy (AMAN)
Anti-ganglioside Antibodies	GD1a, GD1b, GT1b positive	Not available	Not available
CVF at Admission	0.88 L	2.21 L	20 L* (likely error)
Ventilatory Support	Invasive (7 days)	No	Invasive (with tracheostomy, 157 days ICU)
Immunotherapy	IVIG (5 days)	IVIG (5 days)	IVIG (5 days)
Length of ICU Stay	7 days	8 days	157 days
Functional Status at Discharge	Independent ambulation	Wheelchair, partial leg movement	Total dependency, no motor or sensory recovery
Hughes Scale at Discharge	Not documented	Not documented	Not documented
Current Functional Status	Full recovery	Orthotic-dependent, mild coordination deficit	Fully dependent, no recovery

Note: The CVF value of 20 L in Case 3 was extracted directly from the original clinical record. This value is physiologically inconsistent and likely a documentation error. It is presented here as recorded, but should be interpreted with caution.*

The three patients were attended at different time points between 2017 and 2025 in a public hospital in Ecuador. Clinical data were collected retrospectively using a standardized data

collection form validated for the diagnosis of Guillain-Barré Syndrome variants, including Acute Motor Axonal Neuropathy (AMAN).

DISCUSSION

Guillain-Barré syndrome (GBS) is recognized as the leading cause of acute flaccid paralysis in the post-polio era. Among its variants, acute motor axonal neuropathy (AMAN) is characterized by rapid progression of motor weakness, absence of demyelinating features, and a variable prognosis depending on early intervention and axonal preservation (Yuki & Hartung, 2012; Willison et al., 2016). The three clinical cases presented here demonstrate the heterogeneity of AMAN in terms of onset, severity, treatment response, and functional outcomes.

Case 1 involved a high-performance military cyclist who exhibited an abrupt onset of bilateral motor weakness following a gastrointestinal episode. The patient received early IVIG therapy and was admitted to intensive care with an initial vital capacity of 0.88 L. Notably, he responded favorably to treatment and achieved full recovery without residual deficits, returning to baseline physical activity. This case aligns with previous literature emphasizing the importance of early immunomodulatory treatment in reducing disease burden (Yuki & Hartung, 2012; Shi et al., 2019). As shown in **Figure 1**, the patient's EMG demonstrates severe axonal loss without evidence of demyelination, supporting a diagnosis of pure AMAN. These findings underscore the potential for favorable outcomes in young, physically fit patients when treatment is administered promptly.

Case 2, in contrast, illustrates an incomplete recovery despite early IVIG administration. The patient, a 38-year-old with no significant prior comorbidities, presented with symmetrical weakness and documented AMAN via electromyography on the day of admission. Although respiratory compromise was avoided, the patient remained partially dependent on orthotic support for ambulation at three months. The nerve conduction study (**Figure 2**) revealed axonal involvement of both sensory and motor fibers, consistent with broader axonal injury. According to Uncini et al. (2018), such cases may reflect a continuum between AMAN and AMSAN, leading to a more protracted recovery. As outlined in **Table 1**, this case differed from Case 1 primarily in EMG severity and delayed axonal regeneration.

Case 3 presented the most severe trajectory. A 27-year-old male with no previous systemic disease developed ascending flaccid paralysis and required invasive ventilation, prolonged ICU stay (157 days), and tracheostomy. He was discharged in a functionally dependent state and remains wheelchair-bound. EMG findings (**Figure 3**) indicated complete loss of motor unit potentials and absence of reflex arcs, consistent with extensive axonal degeneration. Infections and prolonged critical illness neuropathy likely contributed to poor recovery (Willison et al., 2016; Patone et al., 2021). This case illustrates the extreme end of the AMAN spectrum, where

early intervention was insufficient to prevent long-term disability, possibly due to the magnitude of the immune-mediated injury.

Comparative analysis of the three cases emphasizes the relevance of early diagnosis, timely initiation of immunotherapy, and access to intensive rehabilitation services. While the pathophysiology of AMAN centers on antibody-mediated ganglioside disruption at the nodes of Ranvier, recent studies suggest that host factors such as genetic predisposition, physical conditioning, and early respiratory involvement significantly modulate clinical outcomes (Uncini et al., 2018; Willison et al., 2016). The disparities in recovery highlight the importance of establishing prognostic biomarkers and individualized rehabilitation protocols in Latin American contexts, where resource limitations are common.

This case series highlights the broad clinical and functional spectrum of AMAN, underscoring that early administration of intravenous immunoglobulin (IVIG), while beneficial in many cases, does not uniformly prevent poor outcomes—particularly in patients with extensive axonal degeneration. The results emphasize the critical role of timely electrophysiological assessments, such as EMG and vital capacity monitoring, to guide prognosis and intervention strategies. Additionally, they draw attention to the pressing need for accessible post-acute neurorehabilitation services and greater clinical awareness of AMAN subtypes in Latin America, where diagnostic and therapeutic resources may be constrained.

CONCLUSION

In conclusion, this case series highlights the clinical variability and functional outcomes associated with Acute Motor Axonal Neuropathy (AMAN) in Ecuadorian patients, primarily following gastrointestinal infections. The cases presented demonstrate that while early recognition and immunotherapy can lead to full recovery in some individuals, others may experience partial improvement or persistent neurological disability despite timely intervention. These findings reinforce the importance of neurophysiological confirmation in suspected cases of AMAN and the need to consider this diagnosis in patients with acute flaccid paralysis, particularly in post-infectious contexts. Strengthening access to diagnostic tools and multidisciplinary rehabilitation services is essential to improving outcomes for patients with immune-mediated neuropathies in Latin America.

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