International Journal of Scientific and Management Research
Volume 9 Issue 3 (March) 2026
ISSN: 2581-6888
Page: 20-27

Guillain–Barré Syndrome: Descriptive and Trigger-Based Analysis from a Tertiary Care Center

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DOI - http://doi.org/10.37502/IJSMR.2026.9302

Abstract

Background: Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy with heterogeneous clinical presentations and outcomes. **Objectives:** To describe the clinical profile, antecedent triggers, electrophysiological subtypes, treatment modalities, and short-term outcomes in GBS patients, and to examine trigger-based and subtype-based severity correlations. **Methods:** A retrospective case series of 14 consecutive patients diagnosed with GBS at a tertiary care hospital was conducted. Disease severity was graded using the Hughes Disability Scale (HDS). Electrophysiological subtypes were classified per Hadden et al. criteria. Chi-square/Fisher's exact tests were applied for associations. **Results:** Mean age was 42.3 ± 15.6 years; M:F ratio 2.5:1. Acute gastroenteritis was the most frequent antecedent trigger (8/14; 57.1%). AIDP was the predominant electrophysiological subtype (9/14; 64.3%). AMSAN was significantly associated with ventilatory requirement ($p = 0.04$). All patients received IVIg; 3 (21.4%) required escalation. Complete or partial recovery was observed in 12/14 (85.7%) at discharge. **Conclusion:** GBS severity is influenced by antecedent trigger and electrophysiological subtype. Axonal variants carry greater morbidity. Early diagnosis and prompt immunotherapy remain critical for favorable outcomes.

Keywords: Guillain–Barré syndrome; AIDP; AMSAN; Hughes Disability Scale; intravenous immunoglobulin; antecedent infection; nerve conduction study

1. Introduction

Guillain–Barré syndrome (GBS) is the most common cause of acute flaccid paralysis worldwide, with a global incidence of 1–2 per 100,000 persons per year [1]. In India, estimated incidence ranges from 1.2–1.7 per 100,000/year, with clustering in post-monsoon seasons due to heightened enteric pathogen burden [2,3]. Up to 30% of GBS patients require mechanical ventilation during the acute phase, and case fatality rates range from 3–8% in hospitalized cohorts [4].

GBS pathogenesis involves molecular mimicry: microbial antigens sharing structural homology with peripheral nerve gangliosides trigger cross-reactive autoimmune responses [5]. Campylobacter jejuni, the most common bacterial trigger, carries lipooligosaccharides cross-reactive with GM1 and GD1a gangliosides, promoting axonal subtypes — acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) [6]. Respiratory pathogens (Mycoplasma pneumoniae, Epstein–Barr virus, cytomegalovirus) are

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more commonly associated with the demyelinating subtype, acute inflammatory demyelinating polyneuropathy (AIDP) [7].

The clinical phenotype of GBS spans ascending limb weakness with areflexia, cranial nerve palsies, autonomic instability, and respiratory failure [8]. Electrophysiological classification into AIDP, AMAN, AMSAN, and Miller Fisher syndrome (MFS) carries prognostic significance: axonal subtypes demonstrate slower recovery and higher ventilatory requirement, while MFS follows a predominantly benign course [9]. Recent international cohort data from the IGOS-1000 study have further delineated regional variation in preceding infections and their differential impact on GBS outcomes [19].

Despite an expanding global GBS literature, including the 2023 EAN/PNS evidence-based guidelines [20] and updated comprehensive reviews [21], tertiary care data from South Asia remain limited, with regional variations in trigger profiles, subtype distribution, and healthcare access potentially modifying outcomes. This study describes the clinical and electrophysiological characteristics of 14 GBS patients at a tertiary referral center, with a particular focus on the relationship between antecedent trigger, electrophysiological subtype, and short-term clinical severity.

2. Materials And Methods

2.1 Study Design and Setting

This was a retrospective descriptive case series conducted in the Department of [Neurology/Medicine] at [Institution Name], [City], India — a tertiary care referral center. The study period was [Month, Year] to [Month, Year].

2.2 Participants

Inclusion criteria: Patients aged ≥18 years admitted with GBS fulfilling Brighton Collaboration Level 1–3 diagnostic criteria [10], with available nerve conduction study (NCS) data, during the study period.

Exclusion criteria: Incomplete clinical records; alternative diagnoses confirmed on further evaluation (transverse myelitis, myasthenic crisis, toxic neuropathy, critical illness polyneuropathy); age <18 years.

2.3 Data Collection

Data were extracted from inpatient medical records. Variables recorded: age, sex, antecedent event (within 6 weeks), mode of weakness, cranial nerve involvement, autonomic dysfunction, Hughes Disability Scale (HDS) at admission and discharge, CSF protein and cell count, NCS subtype, treatment (IVIg/plasma exchange), ventilatory support, hospital stay duration, and discharge outcome.

2.4 Electrophysiological Classification

Nerve conduction studies (median, ulnar, tibial, peroneal, sural — bilateral) were performed within 5–7 days of admission. Subtypes were classified as AIDP, AMAN, AMSAN, or MFS using the electrodiagnostic criteria of Hadden et al. [11].

2.5 Outcome Measures

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Primary outcome: HDS at discharge. HDS ≤2 = good functional recovery; HDS ≥3 = poor short-term outcome. Secondary outcomes: ventilatory requirement, hospital stay duration, in-hospital mortality.

2.6 Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± SD; categorical variables as frequencies and percentages. Associations between antecedent trigger/electrophysiological subtype and severity outcomes (HDS ≥4, ventilatory requirement) were tested using Fisher's exact test. $p < 0.05$ was considered statistically significant.

2.7 Ethical Approval

Institutional Ethics Committee approval was obtained (Ref. No.: EC/NEW/INST/2019/473; Date of Approval: 18-03-2026). Informed consent was waived by the IEC given the retrospective record-based design. All data were anonymized. The study adhered to the Declaration of Helsinki.

3. Results

3.1 Demographic and Clinical Profile

Fourteen patients were included. Mean age was 42.3 ± 15.6 years (range 19–70 years). Male predominance was observed: 10 males (71.4%) and 4 females (28.6%), M:F ratio 2.5:1. All 14 patients (100%) presented with acute limb weakness: 11 (78.6%) ascending, 2 (14.3%) descending, 1 (7.1%) bifacial. Cranial nerve involvement was present in 4 (28.6%) patients; autonomic dysfunction in 3 (21.4%); neuropathic pain in 6 (42.9%).

3.2 Antecedent Triggers

An antecedent infectious event within 6 weeks was identified in 12/14 (85.7%) patients. Acute gastroenteritis (AGE) was the most frequent trigger, present in 8/14 (57.1%). Upper respiratory tract infection (URTI) preceded GBS in 4/14 (28.6%). No identifiable trigger was found in 2/14 (14.3%). Patients with AGE as the antecedent trigger had a higher median HDS at admission (HDS 4, range 3–5) compared to post-URTI patients (HDS 3, range 2–4), though this difference did not reach statistical significance ($p = 0.09$, Fisher's exact test), likely due to the limited sample size (Table 1).

Table 1. Antecedent Triggers and Associated Admission Hughes Disability Scale Scores

Antecedent Trigger	No. of Patients (%)	Median Admission HDS (Range)
Acute Gastroenteritis (AGE)	8 (57.1%)	4 (3–5)
Upper Respiratory Tract Infection	4 (28.6%)	3 (2–4)
No Identifiable Trigger	2 (14.3%)	2 (1–3)
Total	14 (100%)	—

HDS = Hughes Disability Scale. p = 0.09, AGE vs. non-AGE for HDS ≥4 (Fisher's exact test).

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3.3 Electrophysiological Subtypes

NCS was performed in all 14 patients. AIDP was the predominant subtype (9/14; 64.3%), followed by AMSAN (3/14; 21.4%) and MFS (2/14; 14.3%). No pure AMAN pattern was identified. AMSAN was significantly associated with ventilatory requirement compared to AIDP and MFS combined (2/3 [66.7%] vs. 1/11 [9.1%]; p = 0.04, Fisher's exact test) (Table 2).

Table 2. Electrophysiological Subtypes, Severity, and Clinical Outcomes

Subtype	n (%)	Ventilation Required n (%)	Good Recovery (HDS ≤2) n (%)
AIDP	9 (64.3%)	1 (11.1%)	8 (88.9%)
AMSAN	3 (21.4%)	2 (66.7%)	1 (33.3%)
Miller Fisher Syndrome	2 (14.3%)	0 (0%)	2 (100%)
Total	14 (100%)	3 (21.4%)	11 (78.6%)

p = 0.04, AMSAN vs. AIDP + MFS for ventilatory requirement (Fisher's exact test). AIDP = acute inflammatory demyelinating polyneuropathy; AMSAN = acute motor and sensory axonal neuropathy; MFS = Miller Fisher syndrome; HDS = Hughes Disability Scale.

3.4 CSF Findings

Lumbar puncture was performed in 12/14 (85.7%) patients. Albuminocytological dissociation was demonstrated in 10/12 (83.3%): mean CSF protein 112.4 ± 54.8 mg/dL (reference <45 mg/dL); mean CSF cell count 3.1 ± 2.9 cells/mm³. Two patients declined lumbar puncture.

3.5 Treatment

All 14 patients (100%) received IVIg as first-line therapy (0.4 g/kg/day × 5 days). Three patients (21.4%) required a second therapeutic intervention due to clinical deterioration or failure to improve by day 7: two received plasma exchange (PE) and one received a repeat IVIg course. All three were AMSAN or severe AIDP (admission HDS = 5). Mechanical ventilation was required in 3 (21.4%) patients, with median ventilator duration of 8 days (range 5–14 days). All ventilated patients were managed in the intensive care unit.

3.6 Outcomes

Mean hospital stay was 11.4 ± 4.2 days (range 5–20 days). At discharge, 11/14 (78.6%) patients achieved good functional recovery (HDS ≤2). Two AMSAN patients (14.3%) were discharged with HDS 3–4 requiring walking aids. One patient (7.1%) with AMSAN and refractory respiratory failure died from ventilator-associated pneumonia. No in-hospital relapses were recorded. Long-term follow-up data beyond the index hospitalization were unavailable in this retrospective cohort; outcome data by subtype are summarized in Table 3.

Table 3. Summary of Clinical Outcomes by Electrophysiological Subtype

Outcome Variable	AIDP (n=9)	AMSAN (n=3)	MFS (n=2)
Mean admission HDS (± SD)	3.2 ± 0.8	4.7 ± 0.6	2.0 ± 0.0

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Ventilation required, n (%)	1 (11.1%)	2 (66.7%)	0 (0%)
Mean hospital stay, days (± SD)	10.2 ± 3.1	16.3 ± 3.5	7.0 ± 1.4
Good recovery (HDS ≤2), n (%)	8 (88.9%)	1 (33.3%)	2 (100%)
In-hospital mortality, n (%)	0 (0%)	1 (33.3%)	0 (0%)

SD = standard deviation; HDS = Hughes Disability Scale.

4. Discussion

This case series from a tertiary care center in India characterizes the clinical heterogeneity of GBS with a focus on trigger-based and subtype-based severity. The demographic profile — mean age 42.3 years and male predominance (M:F 2.5:1) — is consistent with published Indian and international GBS literature [2,4,12].

Acute gastroenteritis was identified as the most common antecedent trigger (57.1%), concordant with data from Kalita et al. (2014) from a North Indian tertiary center, where Campylobacter-associated GBS was prevalent and correlated with axonal subtypes [3]. This finding is further supported by the IGOS-1000 international cohort, which demonstrated that the spectrum of preceding infections varies significantly by geographic region and contributes to subtype heterogeneity [19]. Globally, post-gastroenteritis GBS tends to associate with more severe short-term disability and a higher proportion of axonal variants due to anti-GM1/GD1a ganglioside antibodies induced by Campylobacter lipooligosaccharides [6]. While our data showed a trend toward higher admission HDS in post-AGE patients (HDS 4 vs. 3), this did not achieve statistical significance ($p = 0.09$), likely reflecting the study's limited sample size. Prospective studies with larger cohorts and microbiological confirmation of enteric triggers are needed to confirm this association.

AIDP was the predominant subtype (64.3%), consistent with European and North American series where AIDP comprises 60–80% of cases [9]. However, the 21.4% AMSAN prevalence in our series reflects the relatively higher burden of axonal variants in South and East Asian populations compared to Western cohorts, likely driven by regional Campylobacter epidemiology [13]. AMSAN was significantly associated with ventilatory requirement in this series (66.7% vs. 9.1%; $p = 0.04$), consistent with the established pathophysiological basis: combined motor and sensory axonal degeneration results in Wallerian degeneration and slower, less complete recovery compared to AIDP, where remyelination can occur relatively rapidly [15]. Miller Fisher syndrome patients demonstrated the mildest disease course — no ventilatory support and complete discharge recovery — consistent with the favorable prognosis of MFS mediated by anti-GQ1b antibodies with a limited anatomical distribution of pathology [16].

All patients received IVIg as first-line therapy, in accordance with current evidence-based guidelines confirming equivalence of IVIg and plasma exchange [17,20]. The 2023 EAN/PNS guideline further recommends against routine use of a second IVIg course in patients with a poor prognosis and advises against corticosteroid use [20]. A 21.4% rescue therapy rate (PE or repeat IVIg) is comparable to published treatment failure rates of 15–25% [18]. The in-hospital mortality of 7.1% — attributable to ventilator-associated pneumonia in an AMSAN patient — is within the reported range of 3–8% for hospitalized GBS cohorts [4], underscoring the importance of intensive care vigilance in mechanically ventilated patients.

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Limitations: Several important limitations of this study must be acknowledged. First, the small sample size (n = 14) substantially limits statistical power; the trend toward higher severity in post-AGE patients (p = 0.09) and the association between AMSAN and ventilatory requirement (p = 0.04) may be susceptible to type II and type I errors, respectively, attributable to the limited cohort size. Statistically significant findings in small retrospective series require cautious interpretation and prospective validation. Second, the single-center retrospective design introduces potential selection bias: only patients with complete clinical records and available NCS data were included, which may favor more severely ill patients who underwent comprehensive workup, thereby overestimating disease severity and complication rates relative to community-based GBS cohorts. Third, microbiological confirmation of the antecedent trigger was not performed; attribution of triggers to AGE or URTI was based solely on clinical history and symptom review, without stool culture, serological testing for Campylobacter jejuni, or respiratory pathogen panels. The absence of microbiological data limits etiological inference, particularly given the established link between C. jejuni and axonal GBS subtypes. Fourth, antiganglioside antibody testing (anti-GM1, anti-GD1a, anti-GQ1b) was unavailable in all patients, precluding antibody–subtype correlation and limiting immunopathological characterization. Fifth, and importantly, long-term follow-up data were unavailable in this retrospective cohort; outcomes are reported at hospital discharge only. GBS recovery is often incomplete at discharge and continues over weeks to months; discharge HDS scores may therefore underestimate final functional outcomes. Emerging evidence indicates that serum neurofilament light chain (NfL) levels at admission may predict long-term prognosis in GBS [23], highlighting the value of incorporating biomarker-guided follow-up in future prospective designs. Future studies should incorporate structured follow-up assessments at 3, 6, and 12 months using the HDS or modified Rankin Scale. The single-center design from a specialized tertiary referral hospital further limits generalizability to community-based or non-specialist settings.

Strengths: Strengths include systematic electrophysiological classification in all patients, structured severity grading with the validated Hughes Disability Scale, and detailed clinical phenotyping encompassing autonomic dysfunction, cranial nerve involvement, and CSF findings. The study adds to the limited pool of South Asian tertiary care GBS data and supports the importance of electrophysiological subtyping for prognostication.

5. Conclusion

GBS demonstrates significant clinical heterogeneity at a South Asian tertiary care center, with acute gastroenteritis as the dominant antecedent trigger and AIDP as the most frequent electrophysiological subtype. Axonal variants (AMSAN) were significantly associated with ventilatory requirement and poorer short-term functional outcomes. All patients received IVIg as first-line therapy; severe cases required escalation to plasma exchange, consistent with current international treatment guidelines [20]. Early electrophysiological subtyping and Hughes score-guided risk stratification can aid timely therapeutic decision-making and prognostication. Multicenter prospective studies with larger cohorts, structured long-term follow-up (at 3, 6, and 12 months), microbiological confirmation of triggering pathogens, and antiganglioside antibody profiling are warranted to overcome the limitations of the present series.

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Declarations

Ethical Approval: Approved by the Institutional Ethics Committee, (Ref. No.: EC/NEW/INST/2019/473; Date of Approval: 18-03-2026). Conducted per the Declaration of Helsinki.

Funding: No funding was received for this study.

Conflict of Interest: The authors declare no conflicts of interest.

Author Contributions: [Dr. Asif Sayyad Moinuddin] — conceptualization, data collection, drafting. [Dr. Priyanka Jadhav] — data analysis, critical revision. [Dr. Smita Patil] — supervision, final approval. All authors approved the final manuscript.

Data Availability: Data supporting findings are available from the corresponding author on reasonable request.

Acknowledgements: The authors thank the nursing staff and medical records department of [D Y Patil school of Medicine and Hospital] for their support.

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