RESEARCH PAPER

Intravenous Immunoglobulin Therapy in ICU-Managed Guillain–Barré Syndrome: Clinical Profile and Treatment Outcomes

Dr. Dharmesh Chauhan [1], Dr. Ami Patel[2], Dr. Hemant Toshikhane[3], Dr. Misbah Rangwala[4], Dr. Harish Daga[5]

[1]Associate Professor, Department of RSBK, Parul Institute of Ayurveda, Parul University
[2]IDCCM Fellowship Student, Parul University
[3]Dean Faculty of Ayurveda, Parul University, Parul Institute of Ayurveda, Parul University
[4]Chief Intensivist, Parul Sevashram Hospital
[5]Associate Professor, Department of Shalya Tantra, Parul Institute of Ayurveda, Parul University

Received: 15th Feb, 2026; Revised: 27th Feb 2026; Accepted: 20th Mar, 2026; Available Online: 5th Apr, 2026

ABSTRACT

Background: Guillain–Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness and variable clinical severity, frequently necessitating intensive care support. Axonal variants, particularly in Indian populations, are associated with more severe disease, higher ventilatory requirements, and poorer outcomes. Despite the established efficacy of intravenous immunoglobulin (IVIG), morbidity and mortality remain significant among critically ill patients.

Objectives: To evaluate the demographic profile, clinical characteristics, electrophysiological subtypes, intensive care course, and in-hospital outcomes of adult patients with GBS treated with IVIG in a tertiary-care intensive care unit.

Methods: A retrospective observational case series was conducted from January to December 2025 in the ICU of a tertiary-care center. Adult patients (≥18 years) meeting Brighton diagnostic criteria (Levels 1–3) and treated with standard IVIG therapy (0.4 g/kg/day for 5 days) were included. Data on demographics, antecedent events, clinical presentation, electrophysiological subtype, need for mechanical ventilation, ICU duration, complications, and outcomes were analyzed using descriptive statistics.

Results: A total of 15 patients were included (mean age: 35.5 years; 66.7% male). Antecedent infections were identified in 73.3% of cases. The predominant electrophysiological subtype was Acute Motor Axonal Neuropathy (53.3%), followed by Acute Motor-Sensory Axonal Neuropathy (26.7%) and Acute Inflammatory Demyelinating Polyneuropathy (20%). Mechanical ventilation was required in 40% of patients. The mean ICU stay was 21 days. Major ICU complications included respiratory failure, sepsis, ventilator-associated pneumonia, and pulmonary atelectasis. The overall in-hospital mortality rate was 13.3%.

Conclusion: This ICU-based case series highlights a predominance of axonal variants of Guillain–Barré Syndrome, associated with significant ventilatory support and prolonged intensive care utilization. Despite timely administration of IVIG, mortality remains considerable in severe presentations. Early diagnosis, prompt risk stratification, and optimized multidisciplinary critical care management are crucial for improving patient outcomes.

Keywords: Guillain–Barré syndrome, AMAN, Intravenous immunoglobulin, Mechanical ventilation, Intensive care unit, Axonal neuropathy

How to cite this article: Toshikhane H, Patel A, Chauhan D, Rangwala M, Daga H. Intravenous Immunoglobulin Therapy in ICU-Managed Guillain–Barré Syndrome: Clinical Profile and Treatment Outcomes. Int J Drug Deliv Technol. 2026;16(4): 43-48. DOI: 10.25258/ijddt.16.4.6

Source of support: Nil.

Conflict of interest: None

INTRODUCTION

Guillain–Barré Syndrome (GBS) is an acute, immune-mediated poly radiculoneuropathy and remains one of the most common causes of acute flaccid paralysis worldwide. It is clinically characterized by rapidly progressive, symmetrical limb weakness, areflexia, and

varying degrees of sensory, bulbar, and autonomic dysfunction. The disease exhibits a heterogeneous clinical spectrum, ranging from mild, self-limiting weakness to severe, life-threatening presentations requiring admission to the intensive care unit (ICU) and mechanical ventilatory support.

Epidemiological and clinical studies from India have demonstrated considerable regional variation in disease patterns, particularly in terms of electrophysiological subtypes and clinical severity. Notably, axonal variants—such as Acute Motor Axonal Neuropathy (AMAN) and Acute Motor-Sensory Axonal Neuropathy (AMSAN)—are reported to be more prevalent in Indian populations and are frequently associated with rapid progression, respiratory involvement, and poorer outcomes compared to the demyelinating subtype.

Current evidence supports the use of intravenous immunoglobulin (IVIG) and plasma exchange as effective disease-modifying therapies for GBS. Among these, IVIG is widely preferred in clinical practice due to its ease of administration, accessibility, and favorable safety profile. Previous studies and case series have demonstrated the efficacy of IVIG in improving functional recovery; however, critically ill patients continue to experience substantial morbidity, including prolonged ICU stay, ventilatory dependence, and infectious complications. Clinical outcomes are influenced by several factors, including disease severity at presentation, presence of autonomic dysfunction, electrophysiological subtype, and timing of therapeutic intervention.

Despite the growing body of literature, there remains a relative paucity of data focusing specifically on ICU-admitted GBS patients managed exclusively with IVIG, particularly from tertiary care centers in Gujarat. Therefore, the present case series aims to evaluate the demographic profile, clinical characteristics, electrophysiological patterns, ICU course, and in-hospital outcomes of patients with GBS admitted to the ICU of Parul Sevashram Hospital and treated with IVIG as the primary therapeutic modality.

METHODOLOGY

This retrospective observational case series was conducted in the Intensive Care Unit (ICU) of Parul Sevashram Hospital, a tertiary care referral center, over a period of 12 months from January to December 2025. Adult patients (≥18 years) admitted with a diagnosis of Guillain–Barré Syndrome were considered for inclusion. The diagnosis was established based on the Brighton diagnostic criteria (Levels 1–3), ensuring standardized diagnostic certainty for clinical research.

Only patients who received standard intravenous immunoglobulin (IVIG) therapy at a dose of 0.4 g/kg/day for five consecutive days were included, as IVIG is a well-established first-line treatment with efficacy comparable to plasma exchange. Patients with incomplete medical records, those managed with plasma exchange, or those with alternative diagnoses mimicking GBS were excluded from the study.

Data were extracted from electronic medical records and ICU charts using a structured data collection proforma adapted from previously published Indian case series. The variables recorded included demographic details (age, sex), antecedent events (recent respiratory or gastrointestinal infections), and clinical features at presentation, such as pattern of weakness, cranial nerve involvement, and autonomic dysfunction. Electrophysiological classification was based on nerve conduction studies and categorized into Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor-Sensory Axonal Neuropathy (AMSAN), and Miller Fisher variant.

Treatment-related parameters included timing of IVIG initiation following admission. ICU-related variables comprised requirement of mechanical ventilation, duration of ventilatory support, length of ICU stay, and development of complications such as respiratory failure, sepsis, and ventilator-associated pneumonia.

The primary outcome measure was functional status at discharge, while secondary outcomes included duration of ICU stay, need for mechanical ventilation, and in-hospital mortality. These outcome parameters were selected in accordance with previously published studies to ensure comparability.

Statistical analysis was performed using descriptive methods. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages. Due to the limited sample size, inferential statistical analysis was not performed.

RESULTS

During the 12-month study period (January–December 2025), a total of 15 adult patients diagnosed with Guillain–Barré Syndrome met the inclusion criteria and were included in the analysis. All patients fulfilled the Brighton diagnostic criteria (Levels 1–3) and received standard intravenous immunoglobulin (IVIG) therapy.

Demographic and Baseline Characteristics

The mean age of the study population was 35.5 years, reflecting a predominance of young to middle-aged adults. This distribution is consistent with previously reported Indian cohorts, where GBS tends to affect a relatively younger population compared to Western data. A male predominance was observed, with a male-to-female ratio of 2:1, in line with earlier studies reporting higher incidence among males.

Antecedent events were identified in a majority of patients, most commonly upper respiratory tract infections and gastrointestinal illnesses preceding the onset of neurological symptoms. These antecedent infections are well-recognized immunological triggers in GBS and have been consistently reported across multiple studies.

Clinical Profile at Presentation

The most common presenting feature was progressive limb weakness, with ascending paralysis observed as the predominant pattern. Quadriparesis was noted in a substantial proportion of patients, while sensory involvement was variably present. Deep tendon reflexes were diminished or absent in most patients at the time of admission, consistent with the classical clinical presentation of GBS.

Cranial nerve involvement was observed in a subset of patients, most frequently presenting as facial nerve palsy. Features of autonomic dysfunction, including fluctuations in blood pressure, were also documented in selected cases, indicating more severe disease involvement.

Motor weakness was assessed using the Medical Research Council (MRC) grading system, and MRC sum scores were calculated at admission. Several patients demonstrated low MRC sum scores, reflecting severe motor impairment at presentation. Functional disability was evaluated using the Hughes GBS Disability Scale at both admission and discharge to assess disease severity and recovery trajectory.

Electrophysiological Findings

Nerve conduction studies were performed in all patients. The predominant electrophysiological subtype identified was Acute Motor Axonal Neuropathy (AMAN), followed by Acute Motor-Sensory Axonal Neuropathy (AMSAN) and Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP). The observed predominance of axonal variants in patients with Guillain–Barré Syndrome is consistent with findings from several Indian studies, which report a higher frequency of AMAN and AMSAN compared to Western populations.

Axonal subtypes are typically associated with more severe clinical presentation, rapid progression, and prolonged recovery, which may contribute to increased ICU utilization and poorer functional outcomes.

ICU Course and Ventilatory Requirement

Mechanical ventilation was required in 40% (6/15) of patients due to respiratory muscle involvement. This proportion is comparable to previously reported Indian ICU-based studies. Patients requiring ventilatory support demonstrated longer ICU stays and a higher incidence of complications.

The mean duration of ICU stay was 21 days, indicating significant utilization of critical care resources. The duration of mechanical ventilation varied among patients. Common ICU-related complications included respiratory failure, ventilator-associated pneumonia, sepsis, autonomic instability, and metabolic disturbances. Prolonged ICU stay and increased complication rates have been similarly reported in other tertiary care studies involving severe GBS.

Treatment Details

All patients received standard intravenous immunoglobulin (IVIG) therapy at a dose of 0.4 g/kg/day for five consecutive days. The interval between symptom onset and initiation of IVIG therapy was documented. Early initiation of treatment has been associated with improved clinical outcomes in previous studies; however, no inferential statistical analysis was performed in the present study due to the limited sample size.

Mortality and Functional Outcomes

The overall in-hospital mortality rate was 13.3% (2/15 patients). This is comparable to mortality rates reported in Indian studies, which typically range between 5% and 15%, depending on disease severity and availability of intensive care support. The observed mortality in this series may be attributed to severe axonal involvement and respiratory compromise.

Among survivors, functional recovery at discharge was variable. Although improvement in disability scores was observed in several patients, a proportion required continued rehabilitation at the time of discharge. Axonal variants are known to be associated with slower and often incomplete recovery compared to demyelinating subtypes.

Demographic Characteristics

Variable	**n (%)**
Mean age (years)	35.5
Male	10 (66.7%)
Female	5 (33.3%)
Male: Female ratio	2: 1

Age >50 years	4 (26.7%)
Rural residence	13 (86.7%)
Urban residence	2 (13.3%)
Comorbidities present	4 (26.7%)
Hypertension	1
Diabetes mellitus	2
Sickle cell disease	1

Antecedent Events

Antecedent Event	n (%)
Any antecedent illness	11 (73.3%)
Upper respiratory infection	6 (40%)
Diarrheal illness	2 (13.3%)
Fever (isolated)	2 (13.3%)
No antecedent event	4 (26.7%)

ICU Course and Treatment

Variable	n (%) or Mean
Mechanical ventilation required	6 (40%)
Mean ICU stay (days)	21
Mean hospital stay (days)	26
Delayed treatment (>7 days)	6 (40%)
ICU complications (any)	6 (40%)

Clinical Profile at Admission

Variable	n (%)
Limb weakness (initial symptom)	15 (100%)
Ascending paralysis	10 (66.7%)
Quadriparesis	3 (20%)
Descending weakness	2 (13.3%)
Sensory symptoms	5 (33.3%)
Cranial nerve involvement	2 (13.3%)
Autonomic dysfunction	2 (13.3%)
Respiratory involvement at presentation	4 (26.7%)
Reduced/Absent reflexes	13 (86.7%)
Mean MRC Sum Score	31
Low MRC score (<40)	8 (53.3%)

Electrophysiological Subtype

Subtype	n (%)
AMAN	8 (53.3%)
AMSAN	4 (26.7%)
AIDP	3 (20%)

Documented ICU Complications:

Respiratory failure	3
Sepsis	2
Ventilator-associated pneumonia	1
Pneumonia	1
Lung collapse (atelectasis)	1
Septicaemic shock	1

Outcomes

Outcome	n (%)
In-hospital mortality	2 (13.3%)
Survived	13 (86.7%)
Required rehabilitation at discharge	4 (26.7%)
Residual weakness at follow-up	3 (20%)

DISCUSSION

In this retrospective ICU-based case series of 15 patients with Guillain–Barré Syndrome, a predominance of young adult males, higher frequency of axonal variants, significant ventilatory requirement, prolonged ICU stay, and a mortality rate of 13.3% were observed. These findings underscore the substantial burden of severe GBS in a tertiary care setting.

The mean age of 35.5 years in the present study is consistent with several Indian studies, which have reported a relatively younger affected population compared to Western cohorts. The observed male predominance (2:1) is also in agreement with previous literature indicating a higher incidence among males. A majority of patients had antecedent respiratory or gastrointestinal infections, supporting the well-

established immune-mediated pathogenesis of GBS triggered by preceding infections.

Electrophysiological evaluation revealed Acute Motor Axonal Neuropathy (AMAN) as the predominant subtype (53.3%), followed by AMSAN and AIDP. This pattern aligns with existing Indian data demonstrating a higher prevalence of axonal variants, in contrast to Western populations where AIDP is more common. Axonal variants are known to be associated with more severe motor involvement, rapid progression, and delayed recovery, which likely contributed to the increased need for intensive care and ventilatory support observed in this cohort.

Mechanical ventilation was required in 40% of patients, which is comparable to rates reported in Indian ICU-based studies. Patients requiring ventilatory support experienced prolonged ICU stays and a higher incidence of complications, including sepsis, ventilator-associated pneumonia, and pulmonary complications. These findings are consistent with previous reports highlighting respiratory involvement as a key determinant of disease severity and adverse outcomes.

The in-hospital mortality rate of 13.3% observed in this study falls within the range reported in Indian literature (5–15%). Mortality in severe GBS is commonly attributed to respiratory failure, autonomic dysfunction, and secondary infections, even with appropriate IVIG therapy. Despite all patients receiving standard IVIG treatment, variability in outcomes emphasizes the critical role of early diagnosis, timely initiation of therapy, and comprehensive supportive ICU management.

Overall, the findings of this study are in concordance with existing Indian data, demonstrating a predominance of axonal subtypes, significant ventilatory requirement, and considerable ICU burden in patients with severe Guillain–Barré Syndrome. However, the study is limited by its small sample size and retrospective design, which restricts generalizability and precludes inferential analysis. Further large-scale, prospective studies are warranted to identify prognostic factors and optimize management strategies in critically ill GBS patients.

CONCLUSION

In conclusion, this ICU-based case series of patients with Guillain–Barré Syndrome highlights the significant clinical severity and healthcare burden associated with the disease in a tertiary care setting. The predominance of axonal variants, particularly Acute Motor Axonal Neuropathy (AMAN), along with a high requirement for mechanical ventilation and prolonged ICU stay, reflects a more aggressive disease profile.

Despite timely administration of standard intravenous immunoglobulin (IVIG) therapy, in-hospital mortality remained notable (13.3%), indicating that severe GBS continues to be associated with considerable risk, especially in patients with respiratory involvement and systemic complications.

The variability in functional recovery observed among survivors emphasizes the importance of early diagnosis, prompt risk stratification, close respiratory monitoring, and comprehensive multidisciplinary care, including timely rehabilitation.

Further large-scale, prospective, and multicentric studies are warranted to identify reliable prognostic factors and optimize management strategies, with the goal of improving both survival and long-term functional outcomes in critically ill patients.

Declaration of Interest

The authors declare that there are no conflicts of interest related to the publication of this manuscript.

Data Availability

De-identified data underlying this study are available from the corresponding author upon reasonable request.

Funding

No external funding was received for this study.

Reference -

1. Willison HJ, Jacobs BC, van Doorn PA. Guillain–Barré syndrome. *Lancet*. 2016;388(10045):717–727.
2. Hughes RA, Cornblath DR. Guillain–Barré syndrome. *Lancet*. 2005;366(9497):1653–1666.
3. van den Berg B, Walgaard C, Drenthen J, et al. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. *Nat Rev Neurol*. 2014;10(8):469–482.
4. Yuki N, Hartung HP. Guillain–Barré syndrome. *N Engl J Med*. 2012;366(24):2294–2304.
5. Meena AK, Khadilkar SV, Murthy JM. Treatment guidelines for Guillain–Barré syndrome. *Ann Indian Acad Neurol*. 2011;14(Suppl 1):S73–S81.
6. Kalita J, Misra UK, Das M. Neurophysiological subtypes and outcome of Guillain–Barré syndrome in India. *J Neurol Sci*. 2014;346(1–2):251–255.
7. Sharma G, Sood S, Sharma S. Clinical profile and predictors of outcome in GBS. *J Assoc Physicians India*. 2018;66:45–48.
8. Sudulagunta SR, Sodalagunta MB, Sepehrar M, et al. Guillain–Barré syndrome: clinical profile and management. *J Neurosci Rural Pract*. 2015;6(3):345–350.

9. Lawn ND, Fletcher DD, Henderson RD, et al. Anticipating mechanical ventilation in GBS. *Arch Neurol*. 2001;58(6):893–898.
10. Sharshar T, Chevret S, Bourdain F, Raphaël JC. Early predictors of mechanical ventilation in GBS. *Crit Care Med*. 2003;31(1):278–283.
11. Walgaard C, Lingsma HF, Ruts L, et al. Early recognition of poor prognosis in GBS. *Neurology*. 2011;76(11):968–975.
12. van Koningsveld R, Steyerberg EW, Hughes RA, et al. A clinical prognostic scoring system for GBS. *Lancet Neurol*. 2007;6(7):589–594.
13. Sejvar JJ, Kohl KS, Gidudu J, et al. Guillain–Barré syndrome and Brighton criteria. *Vaccine*. 2011;29(3):599–612.
14. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for GBS. *Cochrane Database Syst Rev*. 2014;(9):CD002063.
15. Raphael JC, Chevret S, Hughes RA, Annane D. Plasma exchange vs IVIG for GBS. *Lancet*. 2001;357(9270):1925–1929.
16. Kleyweg RP, van der Meché FG, Schmitz PI. Interobserver agreement in GBS disability scale. *Neurology*. 1991;41(7):1027–1030.
17. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for GBS. *Ann Neurol*. 1990;27(S1):S21–S24.
18. Ropper AH. The Guillain–Barré syndrome. *N Engl J Med*. 1992;326(17):1130–1136.
19. Wijdicks EF, Klein CJ. Guillain–Barré syndrome. *Mayo Clin Proc*. 2017;92(3):467–479.
20. Dhar R, Stitt L, Hahn AF. The morbidity and outcome of GBS in ICU. *J Neurol Sci*. 2008;264(1–2):121–128.
21. Chio A, Cocito D, Leone M, et al. Guillain–Barré syndrome: a prospective population-based study. *Neurology*. 2003;60(7):1146–1150.
22. McGrogan A, Madle GC, Seaman HE, de Vries CS. Epidemiology of GBS worldwide. *Neuroepidemiology*. 2009;32(2):150–163.
23. Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiological classification of GBS. *Ann Neurol*. 1998;44(5):780–788.
24. Rajabally YA, Uncini A. Outcome and electrophysiological classification in GBS. *J Neurol Neurosurg Psychiatry*. 2012;83(7):711–718.
25. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of GBS in ten steps. *Nat Rev Neurol*. 2019;15(11):671–683.