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Clinical Presentation and Outcome of Guillain-Barre Syndrome in Hospitalized Patients

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Abstract

Background: *Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness and areflexia. Clinical features, antecedent events, electrophysiological variants and outcomes vary across populations. This study aimed to evaluate the demographic characteristics, clinical presentation, treatment modalities and short-term outcomes of hospitalized GBS patients in Bangladesh.*

Methods: *This observational study was conducted in the Department of Neurology, Dhaka Medical College Hospital, Dhaka, Bangladesh, from July 2024 to June 2025. A total of 100 patients meeting the diagnostic criteria were included.*

Results: *The mean age of patients was 38.7 ± 15.2 years, with a male predominance (61%). The most common antecedent events were upper respiratory infection (33%) and gastroenteritis (24%). Facial palsy and bulbar involvement were noted in 27% and 18% of patients, respectively, while respiratory muscle weakness occurred in 21% and autonomic dysfunction in 16%. Electrophysiological studies revealed AIDP in 58%, AMAN in 27%, AMSAN in 10% and Miller Fisher Syndrome in 5% of cases. Treatment included IVIG in 71% and plasmapheresis in 19% of patients. Mechanical ventilation was required in 13% and the mean hospital stay was 14.6 ± 6.3 days. At discharge, 46% of patients were independent (Grade 0–2), 38% required aid or were bedridden (Grade 3–4), 9% remained ventilator-dependent and in-hospital mortality was 7%.*

Conclusion: *Ascending weakness and areflexia are the hallmark features of GBS, with significant cranial, respiratory and autonomic involvement in a subset of patients. Early immunotherapy and supportive care are critical, with the majority achieving functional recovery by discharge.*

Keywords: Guillain-Barré Syndrome, Clinical Features, Electrophysiology, Treatment, Outcomes.

1. INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and is recognized as the most frequent cause of acute flaccid paralysis worldwide since the decline of poliomyelitis [1]. It is characterized by rapidly progressive, symmetrical weakness of the limbs, diminished or absent deep tendon reflexes and variable degrees of sensory, cranial nerve and autonomic involvement [2]. The disease often follows an antecedent infection, most commonly upper respiratory tract infection or gastroenteritis caused by Campylobacter jejuni, although other viral and bacterial pathogens including cytomegalovirus, Epstein-Barr virus and influenza have also been implicated [3].

Vaccination, surgery and trauma are additional triggers in some cases. The clinical spectrum of GBS is heterogeneous and includes several subtypes such as acute inflammatory

demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN) and the Miller Fisher syndrome (MFS) variant [4]. While AIDP is the predominant form in Europe and North America, axonal variants such as AMAN and AMSAN are more frequently reported in Asia and Latin America [5]. Regardless of the subtype, the disease typically progresses over days to weeks and may cause life-threatening complications such as respiratory muscle paralysis, severe dysautonomia and bulbar weakness requiring intensive care support [6]. Approximately 20–30% of patients require mechanical ventilation and despite advances in immunotherapy, the mortality rate ranges from 3 to 7% in hospital-based series [7].

The pathogenesis of GBS is believed to involve molecular mimicry between microbial antigens and peripheral nerve components, leading to immune-mediated damage to myelin or axons [8]. Early recognition and timely initiation of treatment with intravenous immunoglobulin (IVIG) or plasmapheresis are crucial in halting disease progression and improving recovery [9]. However, even with appropriate therapy, a significant proportion of patients experience residual disability, prolonged hospitalization, or long-term functional impairment. Rehabilitation and supportive care therefore remain integral parts of management [10]. Nowadays, GBS is not uncommon, yet published data on its clinical profile and outcomes are relatively limited [11]. Understanding the demographic distribution, clinical presentation, subtypes, treatment practices and short-term outcomes of hospitalized patients can provide valuable insights for clinicians and help optimize management strategies in resource-limited settings [12]. The present study was undertaken to describe the clinical presentation and outcomes of patients with Guillain-Barré syndrome admitted to the Department of Neurology, Tairunnessa Memorial Medical College, Gazipur, Bangladesh, over a one-year period. By identifying the common antecedent events, spectrum of clinical features, electrophysiological variants, treatment modalities applied and discharge outcomes.

2. Methodology And Materials

This observational study was conducted in the Department of Neurology, Dhaka Medical College Hospital, Dhaka, Bangladesh, over a period of one year from July 2024 to June 2025. A total of 100 patients who fulfilled the diagnostic criteria for Guillain-Barré syndrome were included. Both male and female patients aged 18 years and above presenting with acute flaccid paralysis and supported by clinical and electrophysiological findings consistent with GBS were enrolled. Patients with other causes of acute paralysis such as hypokalemic paralysis, transverse myelitis, myasthenia gravis, or stroke were excluded. Detailed clinical information including demographic data, antecedent illnesses and mode of onset was recorded using a structured proforma. All patients underwent thorough neurological examination with emphasis on motor, sensory, cranial nerve and autonomic involvement. Electrophysiological studies were performed to categorize the subtypes of GBS into acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor and sensory axonal neuropathy, or Miller Fisher syndrome.

Laboratory investigations including routine blood tests and cerebrospinal fluid analysis were carried out where necessary. Treatment modalities were decided by the attending neurologists and included intravenous immunoglobulin, plasmapheresis, or supportive care depending on availability and clinical condition. The requirement for mechanical ventilation, duration of hospital stay and complications were documented. Functional status at discharge was assessed using the Hughes Functional Grading Scale. Data were compiled, coded and analyzed using descriptive statistics and categorical variables were expressed as frequencies and percentages while continuous variables were presented as mean with standard deviation. All data were analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0.

3. Results

Table 1. *Demographic Characteristics of the Study Population (n=100)*

Variable	Frequency (n)	Percentage (%)
Age group (years)		
<20	9	9
20–39	37	37
40–59	34	34
≥60	20	20
Mean ± SD (years)	38.7 ± 15.2	

Sex		
Male	61	61
Female	39	39

Table 1 shows the age and sex distribution of patients admitted with Guillain-Barré syndrome. The mean age of the study population was 38.7 ± 15.2 years, with the majority of patients belonging to the 20–39 year age group (37%) followed by 40–59 years (34%). Younger patients (<20 years) accounted for 9%, while 20% were aged 60 years or above. Male patients predominated (61%), resulting in a male-to-female ratio of approximately 1.6:1.

Table 2. *Antecedent Events among Patients with GBS (n=100)*

Antecedent Event	Frequency (n)	Percentage (%)
Upper respiratory infection	33	33
Gastroenteritis (diarrhea)	24	24
Vaccination	7	7
Surgery/trauma	5	5
No identifiable antecedent event	31	31

Table 2 summarizes the preceding events identified in patients before the onset of Guillain-Barré syndrome.

The most frequently reported antecedent was upper respiratory infection (33%), followed by gastroenteritis (24%). A smaller proportion of patients reported recent vaccination (7%) or surgery/trauma (5%).

No identifiable antecedent event was found in 31% of cases, indicating that nearly one-third of patients developed GBS without a clear preceding illness.

Table 3. *Clinical Features of GBS Patients (n=100)*

Clinical Feature	Frequency (n)	Percentage (%)
Ascending motor weakness	92	92
Facial palsy (unilateral/bilateral)	27	27
Bulbar involvement	18	18
Respiratory muscle weakness	21	21
Areflexia/hyporeflexia	95	95
Sensory symptoms (paresthesia, numbness)	41	41
Autonomic dysfunction (BP/HR/urinary)	16	16

Table 3 presents the spectrum of neurological manifestations observed in hospitalized GBS patients. The vast majority of patients exhibited ascending motor weakness (92%) and areflexia or hyporeflexia (95%), which are hallmark features of the syndrome. Sensory symptoms such as paresthesia or numbness were reported in 41% of patients, while cranial nerve involvement was noted as facial palsy in 27% and bulbar involvement in 18% of cases. Respiratory muscle weakness occurred in 21% of patients and autonomic dysfunction affecting blood pressure, heart rate, or urinary function was observed in 16% of cases.

Table 4. *Variants of GBS (Based on NCS Findings) (n=100)*

Variant	Frequency (n)	Percentage (%)
Acute Inflammatory Demyelinating Polyneuropathy (AIDP)	58	58
Acute Motor Axonal Neuropathy (AMAN)	27	27
Acute Motor and Sensory Axonal Neuropathy (AMSAN)	10	10
Miller Fisher Syndrome (MFS)	5	5

Table 4 shows the distribution of GBS subtypes among the hospitalized patients. The most common variant was acute inflammatory demyelinating polyneuropathy (AIDP), observed in 58% of cases. Axonal forms included acute motor axonal neuropathy (AMAN) in 27% and acute motor and sensory axonal neuropathy (AMSAN) in 10% of patients. Miller Fisher Syndrome (MFS), a rare variant characterized by ophthalmoplegia, ataxia and areflexia, was noted in 5% of patients.

Table 5. *Treatment Modalities Applied (n=100)*

Treatment Type	Frequency (n)	Percentage (%)
Intravenous Immunoglobulin (IVIG)	71	71
Plasmapheresis	19	19
Supportive care only	10	10

Table 5 summarizes the therapeutic interventions used in the study population. The majority of patients (71%) received intravenous immunoglobulin (IVIG) as the primary treatment, while 19% underwent plasmapheresis. A smaller proportion of patients (10%) received supportive care only, due to mild disease or limited access to immunotherapy.

Table 6. *Outcomes of Hospitalized GBS Patients (n=100)*

Outcome Parameter	Frequency (n)	Percentage (%)
Required mechanical ventilation	13	13
Duration of hospital stay (Mean ± SD, days)	14.6 ± 6.3	
Functional recovery at discharge (Hughes Scale)		
- Grade 0–2 (independent)	46	46
- Grade 3–4 (ambulatory with aid / bedridden)	38	38
- Grade 5 (ventilator dependent)	9	9
Mortality	7	7

Table 6 presents the short-term outcomes of patients during hospitalization. Mechanical ventilation was required in 13% of patients due to respiratory muscle involvement. The mean duration of hospital stay was 14.6 ± 6.3 days. Functional recovery at discharge, assessed using the Hughes Functional Grading Scale, showed that 46% of patients were independent (Grade 0–2), 38% were ambulatory with aid or bedridden (Grade 3–4) and 9% remained ventilator dependent (Grade 5). In-hospital mortality was 7%, reflecting the serious nature of complications such as respiratory failure and severe autonomic dysfunction.

4. DISCUSSION

In this study, we evaluated the clinical presentation, antecedent events, electrophysiological variants, treatment modalities and outcomes of 100 hospitalized patients with Guillain-Barré syndrome (GBS) in Bangladesh. The mean age of 38.7 years and male predominance (61%) observed in our cohort are consistent with findings reported by Mohebrad et al., who described a similar demographic profile in eastern Iran [13]. Al Maawali et al. also reported comparable age and sex distribution in their Omani cohort, suggesting that GBS predominantly affects middle-aged adults with a male preponderance [14].

Antecedent infections were identified in most patients, with upper respiratory tract infections (33%) and gastroenteritis (24%) being the most common triggers. This aligns with previous reports emphasizing the role of preceding infections in GBS pathogenesis [15, 16]. Notably, 31% of our patients had no identifiable antecedent event, which is consistent with the findings of Shah et al., highlighting that GBS can occur even in the absence of clear triggers [17].

The clinical profile of our patients showed that ascending motor weakness (92%) and areflexia/hyporeflexia (95%) were the hallmark features, while sensory symptoms were present in 41% of cases. Cranial nerve involvement occurred in 27%, bulbar involvement in 18%, respiratory muscle weakness in 21% and autonomic dysfunction in 16%. These observations are comparable with those reported by Barnes et al. in an Australian cohort, where motor weakness and areflexia were predominant, with cranial and autonomic involvement in a smaller subset [18]. Chakraborty et al. reported similar rates of dysautonomia in GBS patients, emphasizing its clinical significance [19].

Electrophysiological assessment revealed that acute inflammatory demyelinating polyneuropathy (AIDP) was the most frequent variant (58%), followed by acute motor axonal neuropathy (AMAN, 27%) and acute motor and sensory axonal neuropathy (AMSAN, 10%). Miller Fisher Syndrome was rare (5%). Khedr et al. and Kılıç et al. have reported similar distributions, demonstrating that demyelinating forms predominate in most regions, although axonal variants are common in certain populations [20, 21].

Treatment modalities primarily included intravenous immunoglobulin (IVIG) in 71% and plasmapheresis in 19%, reflecting global recommendations for immunotherapy in GBS [16, 22]. Supportive care alone was provided to 10% of patients, mainly due to milder disease or resource limitations. Our findings corroborate previous studies highlighting the efficacy of immunotherapy in improving functional outcomes [23, 24].

Regarding outcomes, 13% of patients required mechanical ventilation, with a mean hospital stay of 14.6 ± 6.3 days. At discharge, 46% were independent, 38% required aid or were bedridden, 9% remained ventilator-dependent and in-hospital mortality was 7%. These results are similar to those reported by Netto et al., Dhar et al. and Alshekhlee et al., highlighting the morbidity associated with respiratory failure and the importance of ICU support [25, 26, 27]. Patients requiring ventilatory support generally had poorer functional outcomes, consistent with prior observations [28].

Overall, our findings underscore that GBS in Bangladesh presents with classical ascending weakness and areflexia, with a notable proportion experiencing cranial, respiratory and autonomic involvement. Early recognition, prompt immunotherapy and adequate supportive care are essential to optimize recovery and minimize complications, as highlighted in international literature [16].

5. Limitations of the Study

This study's design and single-centre setting may limit the generalizability of the findings. Additionally, the lack of long-term follow-up data restricts our ability to assess the full recovery trajectory of GBS patients.

6. Conclusion

Our study contributes to the growing body of literature on GBS, highlighting the clinical features, treatment approaches and outcomes in a Bangladeshi cohort. The findings underscore the importance of early diagnosis and appropriate management in improving patient outcomes. Further multicentric studies with long-term follow-up are needed to better understand the long-term prognosis of GBS in our region.

7. Financial Support and Sponsorship

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8. Conflicts of Interest

There are no conflicts of interest.

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