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ORIGINAL ARTICLE

Clinical Progression and Determinants of Unfavorable Functional Outcomes in Guillain-Barré Syndrome – A Retrospective Analysis

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ABSTRACT

Background: *Guillain–Barré Syndrome (GBS) is an acute immune-mediated neuropathy causing motor, sensory, and autonomic dysfunction, often leading to long-term disability. Despite standard treatments, some patients experience rapid progression and unfavorable functional outcomes. Identifying predictors of poor recovery is crucial for early intervention and prognosis.* ***Aim of the study:*** *To evaluate the clinical progression of GBS and determine the factors associated with unfavorable short-term functional outcomes.* ***Methods & Materials:*** *A retrospective observational study was conducted at Dhaka Medical College Hospital from January 2017 to December 2018, including 50 adult patients with first-episode GBS admitted within seven days of symptom onset. Demographic, clinical, and laboratory data were collected. Functional outcomes at six weeks were assessed using the GBS Disability Scale. Univariate and multivariate analyses were performed to identify predictors of poor outcome.* ***Result:*** *Among 50 patients, 42 (84%) had favorable outcomes, and 8 (16%) experienced poor functional recovery. Advanced age (≥50 years), severe motor weakness (MRC score ≤10), pain at onset, hyponatremia (<135 mmol/L), and ICU admission were independent predictors of unfavorable outcomes. Significant correlations were observed between MRC sum score, CSF protein, serum sodium, and GBS disability scores.* ***Conclusion:*** *Advanced age, severe initial motor weakness, pain at onset, hyponatremia, and ICU admission are key determinants of poor short-term functional outcomes in GBS. Early identification of these high-risk patients may guide intensive monitoring, targeted therapy, and rehabilitation strategies to improve recovery.*

Keywords: *Guillain–Barré Syndrome, Functional Outcome, Predictors, MRC Score, Hyponatremia, ICU Admission*

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INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that affects the peripheral nervous system, resulting in varying levels of motor weakness, sensory deficits, and autonomic disturbances [1]. It is the leading cause of acute flaccid paralysis and may develop at any age. Its overall incidence is approximately 1 to 2 cases per 100,000 people each year [2]. Globally, the incidence of GBS is estimated to range between 0.6 and 2.4 cases per 100,000 population annually [3,4]. The acute inflammatory demyelinating polyradiculoneuropathy (AIDP) variant represents the predominant subtype in Europe, accounting for approximately 90% of all GBS cases [5]. Although current treatments for GBS are generally effective and the overall prognosis is favorable, the disease can still be severely debilitating. Approximately 5% of patients die, and up to 20% remain unable to walk independently six months to one year after onset, significantly affecting their daily activities and quality of life (QoL) [6,7]. GBS shows significant regional differences in its incidence, demographic patterns, preceding triggers, clinical features, electrophysiological variants, diagnostic methods, treatment strategies, and prognostic outcomes across various parts of the world [8,9]. Regional differences in GBS arise from a combination of infectious, environmental, genetic, and socioeconomic factors. Variations in the prevalence and strains of pathogens such as CMV, EBV, and Campylobacter jejuni, along with differences in hygiene standards, dietary habits, and nutritional status, influence disease incidence and progression [10,11]. Environmental exposures and population-specific genetic variations further modulate susceptibility and clinical severity. Additionally, disparities in healthcare infrastructure and limited access to advanced diagnostic tools in some regions contribute to the underdiagnosis or misclassification of GBS subtypes, leading to inaccurate estimates of disease burden across different populations [12]. Although current clinical treatments offer substantial benefits for many GBS patients, a subset still experiences severe disease progression requiring mechanical ventilation and may suffer lasting complications such as muscle weakness and sensory deficits, greatly diminish quality of life and increasing

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socioeconomic burden [13,14]. In some cases, the disease progresses rapidly, and conventional therapies fail to yield significant improvement. Furthermore, a double-blind, randomized, placebo-controlled trial found no evidence that a second IVIG course benefits patients with poor prognosis, while it may increase the risk of serious adverse events [15]. GBS is an acute immune-mediated neuropathy causing motor, sensory, and autonomic dysfunction, often leading to long-term disability. Although standard treatments are generally effective, some patients experience rapid progression, require mechanical ventilation, or suffer persistent sequelae, impairing quality of life and increasing socioeconomic burden. Incidence, clinical features, and outcomes vary regionally due to infectious, genetic, environmental, and healthcare factors. This retrospective study aims to evaluate the clinical progression of GBS and identify determinants of unfavorable functional outcomes to improve prognosis and guide management.

METHODOS & MATERIALS

This retrospective observational study was conducted in the Department of Neurology and the Department of Medicine at Dhaka Medical College Hospital, Dhaka. The study covered two years from January 2017 to December 2018 and included hospitalized patients diagnosed with GBS. The objective was to evaluate the clinical course and identify factors associated with unfavorable short-term functional outcomes in GBS patients.

Inclusion Criteria:

Patients aged 18 years and above, of either sex, with a first episode of GBS and admitted within seven days of symptom onset were included. The diagnosis of GBS was established according to the criteria proposed by **Asbury and Cornblath** [16].

Exclusion Criteria:

Ethical approval was obtained from the Ethical Review Committee of Dhaka Medical College, Dhaka. The purpose, procedures, potential risks, and benefits of the study were explained to all participants and their close relatives in simple language. Informed written consent was secured before enrollment. Confidentiality and anonymity were maintained throughout the study, and participation was entirely voluntary without any financial incentives.

Ethical Considerations

Ethical considerations were integral to the study design and execution. The study adhered to ethical principles of confidentiality and privacy, ensuring that all patient data were anonymized and securely stored. Informed consent was obtained where applicable, and the research protocol was reviewed and approved by the relevant institutional ethics committee, ensuring compliance with ethical standards in human subject research.

Data Collection

Relevant clinical and laboratory data were extracted from hospital records using a structured data collection sheet. Variables included demographic information (age, sex, residence), antecedent infections, clinical manifestations (such as pattern of weakness, cranial and bulbar nerve involvement, autonomic dysfunction, and pain at onset), and disease severity assessed using the Medical Research Council (MRC) sum score at admission. Biochemical investigations included random blood sugar, serum sodium, and cerebrospinal fluid (CSF) analysis (protein concentration and cell count). Nerve conduction studies (NCS) were performed to determine GBS subtypes—acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor–sensory axonal neuropathy (AMSAN). The functional outcome was assessed at six weeks post-admission using the GBS Disability Scale (Hughes scale). A *good outcome* was defined as the ability to walk independently (GBS disability score ≤2), while a *poor outcome* was defined as the inability to walk independently (score ≥3).

Statistical Analysis

All data were analyzed using SPSS version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation (SD), while categorical data were expressed as frequencies and percentages. The Chi-square test and independent t-test were used for univariate comparisons between good and poor outcome groups. Variables showing statistical significance ($p < 0.05$) in univariate analysis were further assessed using multiple logistic regression to identify independent predictors of unfavorable functional outcomes. The strength of associations was expressed as odds ratios (OR) with 95% confidence intervals (CI). Correlation analyses were performed to examine the relationships between biochemical parameters and functional outcomes using Pearson's correlation coefficient (r). A p-value of <0.05 was considered statistically significant.

RESULT

Table I showed that the mean age was 37.24±10.12 years, with most aged 28–37 years (36.00%). Males comprised 64.00%, and 64.00% resided in rural areas. Half reported a preceding gastrointestinal infection, 30.00% an upper respiratory infection, 8.00% nonspecific infections, and 12% had no antecedent infection. Electrophysiologically, AIDP was most common (70.00%), followed by AMAN (20.00%) and AMSAN (10.00%). Pain was present in 36.00% of patients. Biochemical and laboratory findings are summarized in Table II. The mean random blood sugar was 6.3±1.5 mmol/L (range 4.1–9.2), serum sodium 135.6±4.8 mmol/L (128–142), CSF protein 118±38 mg/dL (72–210), and CSF cell count 2.1±1.3 cells/mm^3 (0–5). Ascending paralysis was the most common feature (80.00%), followed by cranial nerve involvement and bulbar involvement (both 54.00%) (Table III). Sensory symptoms were present in 44.00%, pain at onset in 36.00%, and autonomic dysfunction in 8.00%. Severe muscle weakness (MRC score 0–10) was seen in 14.00%, and 10.00% of patients required ICU admission. Patients with poor outcomes were older (48.3±8.1 vs. 35.6±9.2 years, p=0.03) and more likely to have severe MRC scores (≤10, 62.50% vs. 4.80%, p<0.001), autonomic dysfunction (37.50% vs. 2.40%, p=0.01), bulbar involvement (87.50% vs. 47.60%, p=0.04), pain at onset (75.00% vs. 28.60%, p=0.03), hyponatremia (sodium <135 mmol/L, 50.00% vs. 14.30%, p=0.04), and ICU admission (37.50% vs. 4.80%, p=0.02). Elevated CSF protein (≥120 mg/dL) was more frequent in poor outcomes (75.00% vs. 42.90%) but did not reach statistical significance (p=0.08) (Table IV). Logistic regression analysis identified several independent predictors of unfavorable functional outcomes (Table V). Age ≥50 years (OR=5.72, 95% CI:1.01–32.4, p=0.04), severe MRC score ≤10 (OR=8.15, 95% CI:1.10–60.1, p=0.04), pain at onset (OR=3.88, 95% CI:1.05–14.3, p=0.04), hyponatremia (sodium <135 mmol/L, OR=5.66, 95% CI:1.14–28.1, p=0.03), and ICU admission (OR=4.88, 95% CI:1.09–21.9, p=0.04) were significant independent predictors. Autonomic

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dysfunction showed a trend toward significance (OR=4.12, 95% CI:0.92–18.4, p=0.06). The MRC sum score was strongly inversely correlated with GBS disability scale (r=–0.82, p<0.001), indicating that lower muscle strength was associated with greater disability (Table VI). CSF protein showed a moderate positive correlation with disability (r=0.46, p=0.002), while serum sodium was moderately inversely correlated with disability (r=–0.41, p=0.004).

Table – I: Demographic and clinical characteristics of GBS patients (*n*=50)

Variables	Frequency (n)	Percentage (%)
Age (years)		
18-27	10	20
28-37	18	36
38-47	15	30
48-57	7	14
Mean ± SD	37.24 ± 10.12	
Sex		
Male	32	64
Female	18	36
Residence		
Rural	32	64
Urban	18	36
Antecedent Infection		
Gastrointestinal infection	25	50
Upper respiratory tract infection	15	30
Nonspecific infection	4	8
None	6	12
GBS variant (NCS type)		
AIDP	35	70.00
AMAN	10	20.00
AMSAN	5	10.00
Presence of pain		
Yes	18	36.00
No	32	64.00

Table – II: Biochemical and laboratory findings of participants (*n*=50)

Parameter	Mean ± SD	Range	Reference
Random blood sugar (mmol/L)	6.3 ± 1.5	4.1–9.2	3.9–7.8
Serum sodium (mmol/L)	135.6 ± 4.8	128–142	135–145
CSF protein (mg/dL)	118 ± 38	72–210	<45
CSF cell count (cells/mm^3)	2.1 ± 1.3	0–5	<10

Table – III: Clinical features and disease severity at admission (*n*=50)

Clinical Feature	Frequency (n)	Percentage (%)
Ascending paralysis	40	80
Cranial nerve involvement	27	54
Bulbar involvement	27	54
Sensory symptoms	22	44
Autonomic dysfunction	4	8
Pain at onset	18	36
Severe MRC score (0–10)	7	14
ICU admission required	5	10

Table – IV: Determinants of unfavorable functional outcome (*n*=50)

Predictor Variable	Good Outcome (n=42)		Poor Outcome (n=8)		P-value
	n	%	n	%	
Mean age (years)	35.6 ± 9.2		48.3 ± 8.1		0.03
Severe MRC score (≤10)	2	4.80	5	62.50	<0.001
Autonomic dysfunction	1	2.40	3	37.50	0.01
Bulbar involvement	20	47.60	7	87.50	0.04
Pain at onset	12	28.60	6	75.00	0.03
CSF protein ≥120 mg/dL	18	42.90	6	75.00	0.08
Sodium <135 mmol/L	6	14.30	4	50.00	0.04
ICU admission	2	4.80	3	37.50	0.02

Table – V: Logistic regression analysis of predictors of unfavorable outcome

Predictor	Odds Ratio (OR)	95% CI	P-value
Age ≥50 years	5.72	1.01–32.4	0.04
Severe MRC score (≤10)	8.15	1.10–60.1	0.04
Autonomic dysfunction	4.12	0.92–18.4	0.06
Pain at onset	3.88	1.05–14.3	0.04
Hyponatremia (<135 mmol/L)	5.66	1.14–28.1	0.03
ICU admission	4.88	1.09–21.9	0.04

Table – VI: Correlation between biochemical parameters and functional outcome

Parameter	r-value	P-value
MRC sum score vs. GBS disability scale	-0.82	<0.001
CSF protein vs. GBS disability	0.46	0.002
Sodium vs. GBS disability	-0.41	0.004

DISCUSSION

The clinical course of Guillain-Barré Syndrome (GBS) exhibits considerable heterogeneity, with certain patient- and disease-related factors predisposing to unfavorable functional outcomes [17]. In our study of 50 GBS patients, the majority were males (64%) with a mean age of 37.24±10.12 years, reflecting a male predominance and peak incidence in the third to fifth decades, consistent with prior studies [18]. Rural residents comprised 64% of cases, which may reflect healthcare access patterns. Antecedent infections were common, with gastrointestinal infections in 50% and upper respiratory tract infections in 30%, supporting earlier reports that infections often trigger GBS [11]. CSF analysis showed elevated protein levels (118±38 mg/dL) with normal cell counts, consistent with albuminocytologic dissociation a hallmark feature of GBS [19]. Mild hyponatremia was noted in some patients, potentially reflecting dysautonomia or SIADH, as described in previous studies [20]. Ascending paralysis was predominant (80%), with cranial nerve and bulbar involvement in 54% of patients. Sensory symptoms were reported in 44%, while autonomic dysfunction was less frequent (8%). Severe weakness at admission (MRC ≤10) occurred in 14%, and ICU admission was required in 10%. These findings align with previous studies reporting variable involvement of cranial nerves and bulbar muscles as indicators of more severe disease [21]. Patients with poor outcomes were older (48.3±8.1 years), and had higher rates of severe MRC score (62.5%), bulbar involvement (87.5%), autonomic dysfunction (37.5%), pain at onset (75%), hyponatremia (50%), and ICU admission (37.5%). Statistically significant associations were observed for age, severe MRC score, autonomic dysfunction, bulbar involvement, pain, hyponatremia, and ICU admission, highlighting their role as potential prognostic indicators. These results are consistent with prior literature emphasizing advanced age, baseline weakness, bulbar/cranial involvement, and ICU-level severity as predictors of poor outcome [22-25]. Multivariate analysis confirmed age ≥50 years (OR 5.72), severe MRC score ≤10 (OR 8.15), pain at onset (OR 3.88), hyponatremia (OR 5.66), and ICU admission (OR 4.88) as independent predictors of unfavorable outcomes. Autonomic dysfunction showed a trend toward significance (OR 4.12, p=0.06). These findings reinforce prior observations that early identification of high-risk patients can guide management and improve prognosis [24]. Strong negative correlation was observed between MRC sum score and GBS disability scale ($r = -0.82$, $p < 0.001$), indicating that lower muscle strength at admission predicts higher disability [26]. CSF protein levels and sodium were moderately correlated with disability ($r=0.46$ and -0.41, respectively), supporting their prognostic relevance, consistent with previous studies linking elevated CSF protein and hyponatremia with poor functional outcomes [27].

Limitations of the study: This study was limited by its retrospective design, relying on hospital records, which may have resulted in incomplete or missing data. The single-center setting and relatively small sample size (N=50) may limit the generalizability of the findings to broader populations. Long-term outcomes beyond six weeks were not assessed, restricting understanding of prolonged functional recovery. Additionally, variations in treatment timing and supportive care could have influenced outcomes, and potential confounding factors, such as comorbidities and prior subclinical neuropathies, were not fully controlled.

CONCLUSION

This retrospective analysis demonstrates that Guillain–Barré Syndrome exhibits considerable variability in clinical progression, with a subset of patients at higher risk for unfavorable functional outcomes. Advanced age (≥50 years), severe motor weakness at admission (MRC score ≤10), pain at onset, hyponatremia, and ICU admission emerged as significant independent predictors of poor short-term functional recovery. The findings underscore the critical importance of early recognition of these high-risk features to guide intensive monitoring, timely intervention, and tailored rehabilitation strategies. Additionally, strong correlations between MRC sum scores, CSF protein levels, serum sodium, and functional outcomes highlight the value of integrating clinical and laboratory parameters for prognosis. These insights can inform clinical decision-making and resource allocation to optimize patient outcomes in GBS.

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