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Frequency of Variant of Gullian-Barré Syndrome (GBS) in Bolan Medical Complex Hospital, Quetta

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ABSTRACT

Background: Guillain-Barré Syndrome (GBS) is a heterogeneous immune-mediated neuropathy with varying clinical and electrophysiological variants. This study investigates the frequency and characteristics of GBS variants in Quetta, Pakistan. **Methods**: A prospective cross-sectional study was conducted at Bolan Medical Complex Hospital from November 2024 to April 2025, enrolling 67 patients aged ≥12 years meeting Brighton Criteria for GBS. Data on demographics, clinical features, electrophysiological findings, and outcomes were analyzed using R software, with statistical significance set at $p<0.05$. **Results**: AIDP was the most common variant (46.3%, 95% CI: 34.2–58.8%), followed by AMAN (26.9%), AMSAN (11.9%), MFS (9.0%), and PCB (4.5%). Axonal variants (38.8%) were prevalent, linked to Campylobacter jejuni in 23.1% of cases (OR 5.2, p=0.003). Poor outcomes (HDS ≥4) occurred in 34.3%, with axonal variants (OR 3.8, p=0.023), mechanical ventilation (OR 8.5, p<0.001), age >60 years (OR 4.1, p=0.016), and rapid progression (OR 2.9, p=0.049) as predictors. AMAN showed prolonged recovery (42 days) versus AIDP (28 days, p=0.002). **Conclusion**: The high prevalence of axonal variants in Quetta underscores the need for tailored diagnostic and therapeutic strategies in resource-limited settings.

INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute, post-infectious, immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness, diminished reflexes, and sensory disturbances[1-3]. It is the most common cause of acute flaccid paralysis worldwide, affecting individuals across all age groups[4-6]. While the classic presentation involves ascending symmetrical weakness, GBS is a heterogeneous disorder with several recognized clinical variants, each differing in terms of clinical features, electrophysiological findings, and sometimes, prognosis and response to treatment[7-10].

Understanding the prevalence and characteristics of these variants is crucial for accurate diagnosis, appropriate management, and predicting patient outcomes. The spectrum of GBS variants includes, but is not limited to, Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor-Sensory Axonal Neuropathy (AMSAN), and Miller Fisher Syndrome (MFS)[11-13]. The classic *Acute Inflammatory Demyelinating Polyneuropathy* (AIDP) predominates in Western populations, while *axonal variants* (AMAN, AMSAN) are more frequent in Asia and Latin America, often linked to antecedent *Campylobacter jejuni* infections and anti-ganglioside antibodies[9, 14-17]. The frequency of these variants can differ geographically, influenced by factors such as genetic predisposition, environmental triggers, and the prevalence of antecedent infections[18].

The variability in GBS presentation and the differing frequencies of its variants across populations underscore the importance of conducting localized epidemiological studies. Such regional data are vital for understanding the specific burden of GBS and its subtypes within a particular geographical area, which can inform public health strategies, resource allocation, and the development of tailored clinical guidelines.

This study aims to determine the frequency of various clinical and electrophysiological variants of Guillain-Barré

Syndrome among patients admitted to Bolan Medical College Hospital (BMCH) in Quetta, Pakistan. By analyzing the demographic, clinical, and electrophysiological profiles of GBS patients presenting to a tertiary care center in this region, we seek to contribute valuable epidemiological data specific to our local population. Such data is essential for enhancing diagnostic accuracy, optimizing therapeutic strategies, and improving the overall management of GBS patients within the context of healthcare resources available in Quetta.

METHODOLOGY

Study Design and Setting

This study employed a prospective cross-sectional design to investigate the frequency of variants of Guillain-Barré Syndrome (GBS). The research was conducted at the Neurology Department of Bolan Medical Complex Hospital (BMCH) in Quetta, Pakistan, a tertiary care center serving a diverse patient population. Data collection for this study spanned a continuous six-month period, commencing on November 1, 2024, and concluding on April 30, 2025. During this period, all patients aged 12 years and above admitted with a diagnosis of GBS were included. Before the initiation of any data collection activities, comprehensive ethical approval for the study protocol was secured from the institutional review boards of both the College of Physicians and Surgeons Pakistan (CPSP) (Reference: CPSP/REU/NEU-2023-001-805) and BMCH.

Inclusion and Exclusion Criteria

The following criteria were applied for patient selection:

Inclusion	- Fulfilled Brighton Criteria for GBS diagnosis.
	- Complete clinical, electrophysiological, and laboratory records.
Exclusion	- Alternative diagnoses (CIDP, myasthenia gravis, metabolic neuropathies).
	- Incomplete diagnostic workup or loss to follow-up.

Sampling and Sample Size

Consecutive sampling was employed to enroll all eligible GBS cases during the specified study period. The final sample size comprised all 67 GBS cases that met the inclusion criteria. This sample size was determined to be sufficient to detect a 20% difference in variant frequency with 80% power at a significance level (α) of 0.05.

Data Collection Protocol

Data were extracted from various sources within the hospital records, including clinical records (admission notes, progress charts, discharge summaries), diagnostic archives (Nerve Conduction Studies (NCS) reports, Cerebrospinal Fluid (CSF) analysis results for protein and cell count.

Operational Definitions

GBS Variants: Classified according to the National Institute of Neurological Disorders and Stroke (NINDS) diagnostic criteria[19].

- *AIDP*: Defined by demyelinating NCS findings coupled with cytoalbuminologic dissociation in CSF.
- *AMAN*: Defined by axonal NCS findings in conjunction with anti-GM1 antibody positivity.

Poor Outcome: Defined as a Hughes Disability Score (HDS) of ≥4 at the time of discharge[20].

Statistical Analysis

All statistical analyses were performed using R statistical software version 4.2.1, utilizing packages such as tidyverse, survival, and lmerTest.

Analytical Approach

Descriptive Statistics: Categorical variables were summarized using frequencies and percentages, accompanied by 95% confidence intervals (CI). Continuous variables were presented as mean ± standard deviation (SD) for normally distributed data, and as median [interquartile range (IQR)] for skewed data.

Inferential Statistics:

- Comparisons between GBS variants for categorical variables were performed using Chi-square tests or Fisher's Exact tests, as appropriate.
- For continuous variables, comparisons between variants were conducted using Analysis of Variance (ANOVA) for normally distributed data or Kruskal-Wallis tests for skewed data.
- Predictors of outcomes were assessed using binary logistic regression, reporting adjusted odds ratios (aOR) with 95% CI.
- The correlation between NCS patterns and HDS was evaluated using Spearman's rank correlation coefficient (ρ).

Survival Analysis: Time to independent walking (defined as HDS ≤2) was analyzed using Kaplan-Meier curves, with comparisons between groups performed using the log-rank test.

Significance Threshold: A two-sided *p*-value of less than 0.05 was considered statistically significant.

Quality Control

Rigorous quality control measures were implemented to ensure data accuracy and reliability. Data validation involved random verification of 20% of the patient records by an independent neurologist, achieving a Kappa (κ) coefficient greater than 0.85 for variant classification, indicating substantial agreement. Nerve Conduction Study (NCS) interpretations were re-read in a blinded fashion by two neurophysiologists, demonstrating an inter-rater agreement of 92%. Cases with more than 10% incomplete variables were excluded from the analysis; however, no such cases were found in the final cohort.

Ethical Considerations

The study protocol received ethical approval from the Institutional Review Board of BMCH (Reference: BMCH-IRB-2023-GBS-067). Given the prospective nature of the study and the use of anonymized data, the requirement for individual patient consent was waived. Patient confidentiality was maintained by replacing all personal identifiers with unique study codes, and the collected data were stored in an encrypted format to ensure privacy and data security.

RESULTS

Cohort and Demographic Characteristics

A comprehensive analysis of our patient cohort confirmed the diagnosis of Guillain-Barré Syndrome (GBS) in 67

individuals. The patient population exhibited a notable gender disparity, with males constituting 61.2% and females 38.8% of the cases. The mean age of the cohort was 43.2±18.1 years, indicating a broad age distribution of the disease presentation. A majority of the patients, 56.7%, hailed from the urban center of Quetta, while the remainder were from surrounding rural areas. This demographic distribution suggests a higher incidence or better access to diagnostic services within the urban population.

Preceding illnesses were a common feature in the clinical history of our patients, documented in a significant 77.6% of the cohort. The most frequent antecedents were gastroenteritis (55.8%) and upper respiratory infections (30.8%), consistent with prior research on GBS triggers. Further microbiological investigations identified *Campylobacter jejuni* infection in 23.1% of these patients, providing a key etiological link within the study population.

Distribution of GBS Variants and Diagnostic Findings

The study identified a diverse distribution of GBS variants. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) was the most prevalent subtype, diagnosed in 46.3% of cases (95% CI: 34.2–58.8%). This finding is consistent with the global prevalence of AIDP. However, our data also revealed a high proportion of axonal variants, with Acute Motor Axonal Neuropathy (AMAN) accounting for 26.9% and Acute Motor and Sensory Axonal Neuropathy (AMSAN) for 11.9% of the cases. Collectively, the axonal subtypes represented a substantial 38.8% of the cohort, which is higher than typically reported in Western populations and may reflect a regional predisposition or environmental factors.

Electrophysiological studies were instrumental in the subtyping of GBS cases. Demyelinating patterns were observed in 53.7% of patients, while axonal patterns were present in 46.3%, providing a direct electrodiagnostic correlation with the clinical variant distribution. The presence of albuminocytologic dissociation in the cerebrospinal fluid (CSF), characterized by elevated protein levels without a corresponding increase in white blood cells, was noted in 76.1% of patients, reinforcing the diagnostic criteria for GBS. Additionally, anti-ganglioside antibodies, which are known to target specific nerve components in axonal variants, were detected in 35.8% of the patients tested.

Clinical Outcomes and Key Associations

Our analysis of patient outcomes at discharge revealed significant morbidity and mortality. A poor outcome, defined by a Hughes Disability Score (HDS) of ≥ 4 (inability to walk independently), was observed in 34.3% of patients. The severity of the disease was further underscored by the need for mechanical ventilation in 28.4% of patients, with a resulting mortality rate of 7.5%.

A compelling association was established between the AMAN variant and a preceding *C. jejuni* infection. The odds of an AMAN diagnosis were 5.2 times higher in patients with a confirmed *C. jejuni* infection (95% CI: 1.7–15.9; p = 0.003). This strong association supports the hypothesis of molecular mimicry, where the immune response to bacterial lipooligosaccharides cross-reacts with gangliosides on the surface of motor nerves, leading to an axonal-predominant neuropathy.

Several factors were identified as robust independent predictors of poor outcomes in our cohort. Patients with axonal variants had 3.8 times higher odds of a poor outcome (95% CI: 1.2–12.1; p = 0.023). The most profound predictor of mortality was the need for mechanical ventilation, which increased the risk of death 8.5-fold (95% CI: 2.5–28.9; p < 0.001). Furthermore, older age (>60 years) and a rapid disease progression (peak disability in less than 7 days) were significant risk factors, with odds ratios of 4.1 (p = 0.016) and 2.9 (p = 0.049) for a poor outcome, respectively. These findings highlight the importance of early identification and aggressive management in these high-risk patient subgroups. A negative correlation was also observed between slower nerve conduction velocities and HDS improvement (ρ=0.41; p = 0.001), indicating that the extent of nerve damage is a critical determinant of functional recovery.

Survival Analysis and Recovery Trajectories

Time-to-event analysis provided a detailed understanding of the recovery trajectories for different GBS variants. We observed a significant difference in the median time to achieve independent walking. Patients with the axonal AMAN variant required a median of 42 days for recovery, whereas those with the demyelinating AIDP variant recovered more quickly, with a median time of 28 days. This difference was statistically significant (p = 0.002, Log-rank test), confirming that axonal variants are associated with a more prolonged and challenging recovery period. This is mechanistically explained by the fundamental difference between demyelinating and axonal pathology; while demyelination allows for relatively rapid remyelination, axonal damage requires slow and often incomplete axonal regeneration. The findings from our survival analysis thus underscore the prognostic importance of accurate electrophysiological subtyping in GBS.

Table 1

Demographic Profile of Patients with GBS

Variable	Value
Age (years)	Mean: 43.2 ± 18.1; Median: 45 (IQR: 28–58)
Sex	Male: 41 (61.2%) Female: 26 (38.8%)
Geographical Origin	Urban Quetta: 38 (56.7%) Rural: 29 (43.3%)

Table 2

Clinical Characteristics

Variable	Categories / Statistics
GBS Variants	AIDP: 31 (46.3%) AMAN: 18 (26.9%) AMSAN: 8 (11.9%) MFS: 6 (9.0%) PCB: 3 (4.5%) Other: 1 (1.5%)
Preceding Illness	Present: 52 (77.6%) Gastroenteritis: 29 (55.8%) Upper Respiratory Infection: 16 (30.8%) *C. jejuni* positive: 12 (23.1%)
Time to Peak Disability	Median: 7 days (IQR: 4–11)
Hughes Disability Score (HDS) at Admission	Median: 4 (IQR: 3–5)

Table 3
Diagnostic Findings

Variable	Findings
NCS Pattern	Demyelinating: 36 (53.7%) Axonal: 31 (46.3%)
CSF Albuminocytologic Dissociation	Present: 51 (76.1%)
Anti-Ganglioside Antibodies	Positive: 24 (35.8%)

Table 4
Outcomes

Outcome Variable	Value
Mechanical Ventilation	Required: 19 (28.4%)
Hospital Stay	Median: 14 days (IQR: 9–21)
Mortality	5 (7.5%)
HDS Improvement (≥1 point)	48 (71.6%)

Table 5
Predictors of Poor Outcomes (Binary Logistic Regression, Outcome = HDS ≥4 at Discharge)

Factor	Odds Ratio (95% CI)	p-value
Age >60 years	4.1 (1.3–13.0)	0.016
Axonal Variant (AMAN/AMSAN)	3.8 (1.2–12.1)	0.023
Mechanical Ventilation	8.5 (2.5–28.9)	<0.001
Time to Peak <7 days	2.9 (1.0–8.6)	0.049

Table 6
Survival Analysis: Time to Independent Walking (HDS ≤2)

Variant	Median Recovery Time (days)
AIDP	28
AMAN	42
MFS	21
Log-Rank Test	$\chi^2 = 12.3$, p = 0.002

DISCUSSION

The present study offers valuable insights into the epidemiological profile of Guillain-Barré Syndrome (GBS) variants in Quetta, Pakistan, a region that is underrepresented in the global literature on this disorder. Among the 67 patients analyzed, Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) emerged as the predominant subtype at 46.3%, followed by axonal forms including Acute Motor Axonal Neuropathy (AMAN) at 26.9% and Acute Motor-Sensory Axonal Neuropathy (AMSAN) at 11.9%, collectively accounting for 38.8% of cases. Miller Fisher Syndrome (MFS) was observed in 9.0%, Pharyngeal-Cervical-Brachial (PCB) variant in 4.5%, and other unspecified variants in 1.5%. This distribution reflects a hybrid pattern, with AIDP still leading but axonal subtypes notably elevated compared to Western cohorts, aligning with broader trends in Asian and low- to middle-income countries (LMICs) where environmental and infectious factors may influence subtype prevalence.

Comparative analyses with recent regional studies underscore geographical variability in GBS subtypes. In Pakistan, our findings contrast with higher axonal predominance reported in other provinces; for instance, a 2023 retrospective study from Peshawar involving 39 patients identified AMAN as the most common subtype at 59%, AMSAN at 25.6%, and AIDP at only 15.3%, with axonal variants comprising 84.6% of cases[21]. Similarly, another 2023 Pakistani cohort of 117 patients from Lahore reported AMAN at 46.2%, AMSAN at 34.2%, and AIDP at 16.2%, highlighting a consistent trend toward axonal dominance in northern and central Pakistan[22]. The relatively higher AIDP frequency in our Quetta-based sample (46.3%) may reflect local differences in antecedent infections or genetic factors, though axonal forms remain substantially more frequent than in Western populations (typically 6-17%)[23]. This intermediate profile in Quetta could indicate a transitional epidemiological zone influenced by Balochistan's unique socio-environmental context, including rural-urban divides and varying access to healthcare.

Broadening to Asia, our results resonate with patterns in East and South Asia, where axonal variants are more prevalent due to higher rates of Campylobacter jejuni infections[24]. A 2025 Taiwanese study of 185 patients from 2011-2022 reported AIDP at 42.7% and axonal GBS at 30.9%, with MFS and GBS/MFS overlap at 25%—figures comparable to our axonal (38.8%) and MFS (9.0%) rates, and notably higher than Western estimates (MFS 3-11%, axonal 3-17%)[23]. In contrast, a 2025 Indian study from Delhi with 137 patients showed AIDP dominance at 70%, axonal forms at 18.2%, and MFS-inclusive variants at 11%, suggesting subcontinental heterogeneity possibly linked to differing infectious burdens[25].

A 2024 study from the same tertiary center in Quetta, Pakistan, involving 141 patients (91.5% GBS), further contextualizes our variant distribution by demonstrating the efficacy of therapeutic plasma exchange (TPE) in managing acute immune-mediated neuropathies, with significant improvements in muscle strength and functional outcomes. In that cohort, TPE led to 51.8% of patients walking without assistance after four weeks, highlighting its role in recovery, particularly for GBS subtypes[26]. Globally, a 2024 meta-analysis estimated GBS incidence at 1.12 per 100,000 person-years, with higher rates in South Asia (1.78 per 100,000), reinforcing the elevated axonal subtype frequency in this region as observed in our cohort[27]. These disparities emphasize the role of molecular mimicry in axonal GBS pathogenesis, particularly in LMICs where sanitation and infection control challenges amplify C. jejuni exposure[24].

The strong association between AMAN and preceding C. jejuni infection in our study (odds ratio 5.2, p=0.003) corroborates established mechanisms of immune cross-reactivity with gangliosides, a finding echoed in recent LMIC research[24]. Preceding illnesses were noted in 77.6% of our patients, with gastroenteritis (55.8%) predominant, aligning with a 2021 global review indicating that up to 40% of GBS cases in Asia link to C. jejuni, far exceeding Western rates (10-20%)[24]. Electrophysiologically, our demyelinating (53.7%) versus axonal (46.3%) patterns, alongside anti-ganglioside positivity in 35.8%, support subtype-specific diagnostics, consistent with a 2024 update on global GBS burden emphasizing the prognostic value of such classifications[28].

Regarding outcomes, 34.3% of patients experienced poor recovery (Hughes Disability Score ≥4 at discharge), with axonal variants conferring 3.8-fold higher odds (p=0.023), mechanical ventilation 8.5-fold (p<0.001), age >60 years 4.1-fold (p=0.016), and rapid progression (<7 days) 2.9-fold (p=0.049). Mortality stood at 7.5%, within the 2-10% global range reported in 2024[28]. Survival analysis

revealed prolonged recovery in AMAN (median 42 days to independent walking) versus AIDP (28 days, p=0.002), attributable to axonal regeneration challenges, as noted in a 2025 East Asian review[23]. These predictors align with a 2023 Pakistani study identifying axonal subtypes and ventilation as key mortality risks, underscoring the need for early intervention in resource-limited settings[29].

This study's strengths include its prospective design, rigorous electrophysiological classification (92% inter-rater agreement), and focus on a tertiary center in an underserved region. However, limitations warrant consideration: the sample size (n=67) may limit generalizability, potentially underrepresenting rare variants or rural cases due to referral bias. The six-month data collection period (November 2024-April 2025) might overlook seasonal influences, though no such trends were evident. Absence of long-term follow-up beyond discharge restricts insights into chronic outcomes, and reliance on hospital records could introduce documentation biases, despite high data completeness.

Clinically, these findings advocate for heightened awareness of axonal GBS in Pakistan, informing tailored immunotherapy and supportive care, such as plasma exchange in resource-constrained environments. Public health implications include targeted surveillance for C. jejuni in high-risk areas to mitigate triggers. Future research should encompass multicenter, longitudinal studies across Pakistan to elucidate genetic-environmental interactions, incorporating advanced biomarkers like neurofilament light chain for prognostication, as suggested in recent global guidelines[30]. In conclusion, this Quetta cohort bridges gaps in regional GBS epidemiology, highlighting a distinct variant distribution that demands context-specific management strategies to optimize patient outcomes in LMICs.

CONCLUSION

This study provides critical insights into the epidemiology of Guillain-Barré Syndrome (GBS) variants in Quetta, Pakistan, revealing a hybrid pattern with Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) as the predominant subtype (46.3%) and a significant proportion of axonal variants (38.8%), notably Acute Motor Axonal Neuropathy (AMAN) and Acute Motor-Sensory Axonal Neuropathy (AMSAN). The high prevalence of axonal subtypes, strongly associated with *Campylobacter jejuni* infections, underscores regional differences compared to Western populations and highlights the role of environmental and infectious triggers in low- to middle-income countries (LMICs). Poor outcomes, observed in 34.3% of patients, were linked to axonal variants, older age, rapid disease progression, and mechanical ventilation, with AMAN patients exhibiting prolonged recovery times. These findings emphasize the need for enhanced diagnostic precision, early intervention, and tailored therapeutic strategies, such as plasma exchange, to improve outcomes in resource-constrained settings. Future multicenter studies in Pakistan incorporating biomarkers like neurofilament light chain are recommended to further elucidate GBS pathophysiology and optimize management in LMICs.

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