

Frequency of Anti-Ganglioside Antibodies and Their Clinical Correlates in *Guillain-Barré Syndrome*: A Single-Centre Study in Pakistan

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ABSTRACT

Objective: To determine the frequency of anti-ganglioside autoantibodies and their clinical associations in patients clinically suspected of having *Guillain-Barré Syndrome* (GBS).

Study Design: A cross-sectional descriptive study.

Place and Duration of the Study: Department of Immunology, Armed Forces Institute of Pathology, Rawalpindi, Pakistan, from July 2024 to July 2025.

Methodology: Anti-ganglioside (IgG/IgM) antibody-positive 28 patients with suspected GBS, tested using the EUROLINE Anti-Gangliosides Profile 2 (EUROIMMUN, Germany), were included. Band intensity was quantified automatically using EUROLINeScan (EUROIMMUN, Germany). Clinical features, antecedent events, and electrophysiological findings (EMG/NCS) were documented. Normality was assessed using the Shapiro-Wilk test. The chi-square and Fisher's exact tests were applied; the independent-samples t-test was used for continuous variables. A $p < 0.05$ was considered significant.

Results: Of the 28 patients, GBS variants were the predominant clinical entities (67.8%), with axonal subtypes accounting for 39.2% and demyelinating variants comprising 28.6% of the cases. The mean age of participants was 36.4 ± 16.6 years, and 75% were male. Gastrointestinal infections were the most common antecedent event (57.9%), followed by upper respiratory tract infections (26.3%). Progressive para/tetraparesis was significantly more frequent in axonal variants ($p = 0.024$), while cranial nerve palsies were notably associated with demyelinating forms ($p = 0.005$). Anti-GM1 antibodies were the most frequently detected (46.5%) and showed a statistically significant association with axonal subtypes ($p = 0.019$).

Conclusion: Axonal subtypes predominated, with anti-GM1 antibodies correlating with motor-predominant variants. Combining serology with clinical and electrophysiological data may strengthen diagnostic accuracy. Future studies should include larger cohorts and investigate preceding infections, such as *Campylobacter jejuni*.

Key Words: *Guillain-Barré syndrome, Anti-ganglioside autoantibody, Axonal GBS, Demyelinating GBS, Peripheral neuropathy.*

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INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterised by rapidly progressive, symmetrical limb weakness, diminished or absent deep tendon reflexes, and variable sensory involvement. The disease typically follows a monophasic course and can lead to severe disability or respiratory failure if untreated.¹ It is frequently preceded by infectious illnesses, most notably *Campylobacter jejuni*, cytomegalovirus, and Epstein-Barr virus.

The underlying pathophysiology involves molecular mimicry, whereby immune responses directed against microbial antigens cross-react with neural gangliosides, resulting in peripheral nerve demyelination or axonal injury.²

GBS occurs worldwide, with an annual incidence of 1–2 cases per 100,000 population, and affects individuals of all ages, although it is slightly more common in males.³ Regional variation exists in both incidence and electrophysiological subtypes, with axonal variants such as acute motor axonal neuropathy (AMAN) being more prevalent in East and South Asia, compared to the predominance of acute inflammatory demyelinating polyneuropathy (AIDP) in Western countries. Despite advances in diagnosis and treatment, approximately 20% of patients may experience severe disability or death.⁴

A growing body of evidence has demonstrated that specific anti-ganglioside antibodies, particularly against GM1, GD1a, GD1b, GT1b, and GQ1b, are strongly associated with certain

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clinical phenotypes and electrophysiological variants of GBS. For example, anti-GM1 and anti-GD1a antibodies are typically linked to axonal forms, such as AMAN and acute motor and sensory axonal neuropathy (AMSAN), whereas anti-GQ1b antibodies are strongly correlated with Miller Fisher syndrome (MFS) and other cranial nerve-predominant presentations.⁵ These antibodies have also been detected in non-GBS disorders, including chronic inflammatory demyelinating polyneuropathy (CIDP), multifocal motor neuropathy, and certain paraneoplastic neuropathies, although their diagnostic specificity is generally higher in GBS and its variants. Their detection offers insights into disease pathogenesis, aids in early diagnosis, and may have prognostic implications.⁶ Nerve conduction studies (NCS) and electromyography (EMG) help classify electrophysiological subtypes, distinguishing between demyelinating and axonal forms.⁷

In Pakistan, data on the prevalence of anti-ganglioside antibodies in GBS are limited, with most available studies focusing primarily on clinical features and electrophysiological subtypes without evaluating serological patterns. Given the observed regional variation in GBS epidemiology and the known role of antecedent infections in shaping antibody profiles, understanding the serological patterns in the local population is crucial. This study aimed to fill this gap and determine the antibody frequency, its clinical correlations, and potential diagnostic value in the neurogenetics of GBS. To the authors' knowledge, this is among the first Pakistani studies analysing the full anti-ganglioside antibody spectrum in GBS, addressing a major gap in regional data.

METHODOLOGY

This was a cross-sectional descriptive study conducted at the Department of Immunology, Armed Forces Institute of Pathology, Rawalpindi, Pakistan, from July 2024 to July 2025, after obtaining ethical approval from the Institutional Review Board of the Armed Forces Institute of Pathology, Rawalpindi, Pakistan (Approval No. Cons-IMM-3/READ-IRB/24/3900). Sample size was calculated using the WHO formula, taking the anticipated population proportion of 1.8%,⁸ with a 95% confidence interval and 0.05 absolute precision. The study included a total of 28 patients who tested positive for anti-ganglioside antibodies (IgG and/or IgM) and were clinically suspected of having *Guillain-Barré* syndrome (GBS) or its variants. Serum samples were tested using the EUROLINE Anti-Gangliosides Profile 2 (Euroimmun, Lubeck, Germany), a semi-quantitative line immunoassay that detects IgG and IgM autoantibodies against a panel of ganglioside antigens, including *GM1*, *GM2*, *GM3*, *GD1a*, *GD1b*, *GT1b*, and *GQ1b*. Band intensity was quantified automatically using the EUROlineScan software (EUROIMMUN, Germany). Results were interpreted according to the manufacturer-defined thresholds; values <11 AU were considered negative, 11–25 AU borderline, and >25 AU as positive for the corresponding ganglioside antibody.

Electrophysiological diagnoses were obtained from available EMG and NCS for each patient and were categorised into GBS variants based on internationally accepted criteria. Patients were subtyped into axonal (AMAN, AMSAN), demyelinating (AIDP, MFS), or unclassified variants based on these findings. Clinical data were collected using a structured proforma, which included key neurological features such as pattern of motor weakness (e.g., paraparesis and tetraparesis), sensory deficits, cranial nerve involvement, reflex status, and overall disease progression. Additionally, antecedent events, occurring within four weeks prior to the onset of neurological symptoms, were recorded through patient interviews and review of clinical records.

All data were analysed using IBM SPSS Statistics, version 27. Descriptive statistics, such as frequencies and percentages, were used to summarise demographic and clinical variables, and the mean \pm standard deviation for age distribution. For continuous variables, such as age, the Shapiro-Wilk test was used to determine the normality of the distribution, and the independent-samples t-test was used to compare group means. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. The Fisher-Freeman-Halton exact test was employed for multi-category contingency tables where expected frequencies were <5. A p-value less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 28 patients with anti-ganglioside antibodies were included in the analysis. The cohort was predominantly male, comprising 75% (n = 21) males and 25% (n = 7) females, with a mean age of 36.4 ± 16.6 years. Among these, 67.8% (n = 19) were diagnosed with GBS. Electrophysiological characterisation revealed that axonal variants of GBS were the most common, accounting for 57.9% of cases. Among these, the AMAN subtype represented 25%, and AMSAN constituted 14.3%. Demyelinating variants, including AIDP and MFS, accounted for 42.1% of cases. A notable proportion of patients (28.6%) remained unclassifiable based on the available data, while one patient (3.6%) was categorised as having CIDP; both were categorised as non-GBS (n = 9, 32.1%; Table I). There were no statistically significant differences between axonal and demyelinating groups in terms of mean age (p = 0.762) or gender distribution (p = 1.000; Table II).

Antecedent infections were reported in most cases, with gastrointestinal infections being the most frequently observed, effecting 57.9% of the cohort and particularly predominant among patients with axonal variants. Upper respiratory tract infections, including influenza-like illnesses, were reported in 26.3% of cases, while non-specific febrile or rash-associated illnesses accounted for 10.5%. No statistically significant association was found between the type of antecedent event and GBS subtype (p = 0.878), although the observed trend of gastrointestinal infections preceding axonal variants aligns with known immunopathological mechanisms (Table II).

Table I: Demographic and electrophysiological characteristics of patients with GBS and related neuropathies.

Variables		Frequencies	Percentages	Mean ± SD
Gender	Male	21	75%	-
	Female	7	25%	-
Age (years)		-	-	36.4 ± 16.6
Electrophysiological diagnosis	AMAN	7	25%	-
	AMSAN	4	14.3%	-
	AIDP	6	21.4%	-
	MFS	2	7.1%	-
	CIDP	1	3.6%	-
	Unclassifiable	8	28.6%	-
	Total	28	100%	-

AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory axonal neuropathy; AIDP: Acute inflammatory demyelinating polyneuropathy; MFS: Miller Fisher syndrome; CIDP: Chronic inflammatory demyelinating polyneuropathy.

Table II: Key clinical features and association of antecedent event with GBS variants.

Variables	Axonal Variants (AMAN/AMSAN) n = 11 (57.9%)	Demyelinating variants (AIDP/MFS) n = 8 (42.1%)	Total	p-values
Age (years) Mean ± SD	34.4 ± 16.8	36.7 ± 16.5	-	0.762 [†]
Gender				1.000*
Male	8 (42.1%)	6 (31.6%)	14 (73.7%)	
Female	3 (15.8%)	2 (10.5%)	5 (26.3%)	
Antecedent event				0.878**
Gastrointestinal Infection	7 (36.8%)	4 (21.1%)	11 (57.9%)	
URTI/Flu	2 (10.5%)	3 (15.8%)	5 (26.3%)	
Fever/Rash	1 (5.26%)	1 (5.26%)	2 (10.5%)	
None	1 (5.26%)	0 (0%)	1 (5.26%)	
Clinical features				
Progressive para- or tetraparesis	9 (47.4%)	2 (10.5%)	11 (57.9%)	0.024*
Sensory deficits	5 (26.3%)	7 (36.8%)	12 (63.2%)	0.147*
Hypo- or areflexia	7 (36.8%)	8 (42.1%)	15 (78.9%)	0.103*
Cranial nerve palsies	0 (0%)	5 (26.3%)	5 (26.3%)	0.005*
Symmetric ascending weakness	9 (47.4%)	6 (31.6%)	15 (78.9%)	1.000*

[†]Independent-samples t-test; *Fisher's exact test; **Fisher-Freeman-Halton exact test; URTI: Upper respiratory tract infection.

Table III: Association of anti-ganglioside antibodies with electrophysiological diagnosis.

Anti-ganglioside antibodies (IgG/IgM)	GBS (n = 19)		Non-GBS (Unclassifiable/CIDP) n = 9	total	p-values*
	Axonal variants (AMAN/AMSAN) n = 11	Demyelinating variants (AIDP/MFS) n = 8			
Anti-GM1	8 (28.6%)	4 (14.3%)	1 (3.6%)	13 (46.5%)	0.019
Anti-GM2	4 (14.3%)	1 (3.6%)	1 (3.6%)	6 (21.4%)	0.376
Anti-GM3	1 (3.6%)	0 (0%)	2 (7.1%)	3 (10.7%)	0.473
Anti-GD1a	6 (21.4%)	1 (3.6%)	2 (7.1%)	9 (32.1%)	0.155
Anti-GD1b	3 (10.7%)	4 (14.3%)	2 (7.1%)	9 (32.1%)	0.527
Anti-GT1b	1 (3.6%)	1 (3.6%)	1 (3.6%)	3 (10.7%)	1.000
Anti-GQ1b	0 (0%)	2 (7.1%)	0 (0%)	2 (7.1%)	0.074

* Fisher-Freeman-Halton exact test. GBS: Guillain-Barré syndrome.

Progressive para- or tetraparesis was more frequently observed in patients with axonal variants and was significantly associated with this group ($p = 0.024$, $\Phi = -0.568$), with approximately threefold higher odds of developing progressive para- or tetraparesis compared with those with demyelinating variants (OR = 3.27, 95% CI: 0.95 - 11.23). In contrast, cranial nerve involvement was significantly more common in demyelinating cases ($p = 0.005$, $\Phi = 0.701$) and was absent in patients with axonal subtypes. Patients with demyelinating subtypes had a 4.7-fold higher odds of exhibiting cranial nerve palsies compared to those with axonal forms (OR = 4.67, 95% CI: 1.71-12.7). Symmetric ascending weakness, sensory disturbances, and generalised areflexia or hyporeflexia were observed across all subtypes without statistically significant differences ($p > 0.1$).

Anti-GM1 antibodies were the most prevalent overall, detected in 46.5% of patients, and demonstrated a significant association with axonal GBS, particularly AMAN and AMSAN, with 72.7% of antibody-positive patients classified within the axonal category ($p = 0.019$, $r = -0.516$). Anti-GD1a antibodies were also more frequent among axonal cases, but this did not reach statistical significance ($p = 0.155$). Notably, anti-GQ1b antibodies were identified exclusively in patients with demyelinating forms, specifically MFS, although the difference was not statistically significant ($p = 0.074$). The presence of other ganglioside antibodies (GM2, GM3, GD1b, and GT1b) was low and did not significantly differ across the groups (Table III). The distribution of anti-ganglioside antibodies across different electrophysiological diagnoses of suspected GBS is illustrated in Figure 1.

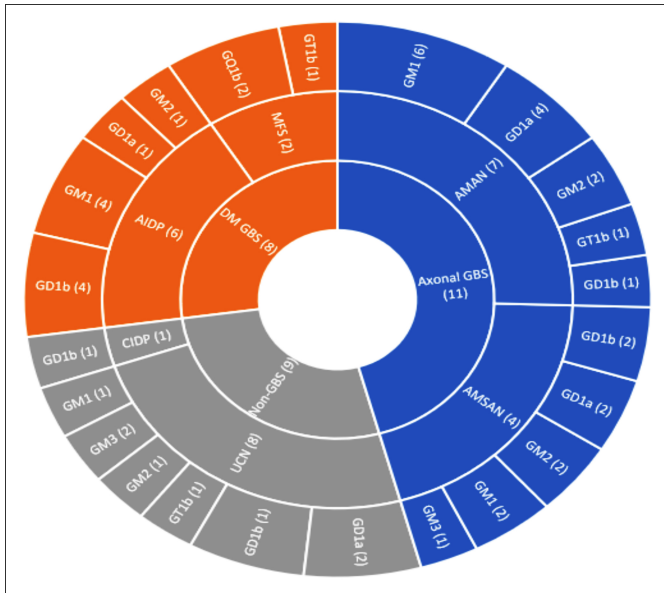


Figure 1: Distribution of detected anti-ganglioside antibodies across clinical subtypes of GBS and related neuropathies.

DISCUSSION

In this study, the majority of patients with GBS and related neuropathies were young adult males, with a mean age of 36.4 years and a male predominance of 75%. This gender distribution is consistent with global literature, in which male predominance is frequently observed in GBS, likely due to immune, hormonal, and genetic factors influencing susceptibility to peripheral nerve autoimmunity. International data reports male-to-female ratios ranging from 1.5:1 to 2:1 across various GBS subtypes.⁹ Similarly, a local study from Karachi reported a male predominance of 54% in their GBS cohort,¹⁰ corroborating the present study's findings.

Electrophysiological studies revealed that axonal variants (AMAN and AMSAN) comprised the majority (57.9%) of cases in this study. This is in contrast to Western populations, where AIDP (a demyelinating subtype) is most frequently observed.¹¹ However, several studies from East and South Asia, including China and Bangladesh, have also reported a predominance of axonal forms.^{12,13} Notably, a Pakistani study by Ayaz ul Haq *et al.* reported AMAN as the most common variant, present in 46.2% of their GBS cases, supporting a regional pattern favouring axonal subtypes.¹⁴ These geographic differences may be explained by environmental factors, variations in antecedent infections, or genetic susceptibility.

The most commonly reported antecedent illness was gastrointestinal infection (57.9%), particularly among patients with axonal subtypes. This observation aligns with the well-established association between *Campylobacter jejuni* enteritis and AMAN.¹⁵ URTIs were more commonly associated with demyelinating forms, including MFS, although the association was not statistically significant in this study. A similar trend was observed in a previous work by Guarino

et al., who demonstrated that gastrointestinal infections were highly predictive of GM1-positive axonal GBS.¹⁶ In a Pakistani cohort, Hassan *et al.* noted similar antecedent event distributions, reinforcing the role of preceding infections in shaping the disease phenotype.¹⁷

Clinically, progressive para- or tetraparesis was significantly associated with axonal GBS ($p = 0.024$), consistent with the rapid and severe motor involvement typically seen in AMAN and AMSAN.¹⁸ On the other hand, cranial nerve palsies were exclusive to the demyelinating group ($p = 0.005$), particularly MFS, where ophthalmoplegia was a defining feature. These findings are supported by international literature, which describes AIDP and MFS as more frequently associated with facial and bulbar weakness due to demyelination of cranial nerve roots.¹⁹ The co-occurrence of symmetric ascending weakness, areflexia, and sensory deficits across all groups underscores their non-specific but supportive role in the clinical diagnosis of GBS.

Serologically, anti-GM1 antibodies were the most frequently detected (46.5%) and showed a statistically significant association with axonal GBS ($p = 0.019$), particularly AMAN. This finding is in strong concordance with previous literature, in which GM1 positivity is considered a hallmark of axonal GBS, due to molecular mimicry between GM1 epitopes and *Campylobacter jejuni* lipooligosaccharides.²⁰ Anti-GD1a antibodies, also linked to motor axonal forms, were more prevalent among axonal patients, although this was not statistically significant. These antibodies have been documented as highly specific for AMAN in studies from Japan and China.^{21,22} The detection of anti-GQ1b antibodies exclusively in demyelinating cases, especially MFS, aligns with its known role in binding to oculomotor and sensory cranial nerves, confirming its high specificity for MFS.²³ Although not statistically significant ($p = 0.074$), the antibody was present in 100% of MFS patients in this study cohort, which is noteworthy, but it cannot yield definitive conclusions owing to the small sample size. However, it remains consistent with the established association reported in broader investigations.

The low frequency of other antibodies, such as GM2, GM3, GD1b, and GT1b, is consistent with their generally lower prevalence and weaker association with specific clinical phenotypes. Their presence across both axonal and demyelinating variants without significant correlation mirrors findings from other studies, suggesting a more limited diagnostic or prognostic role.²⁴ However, the presence of these antibodies may still contribute to the heterogeneity of clinical presentation and may be relevant in atypical cases or in broader panels.

This study has several inherent limitations. The relatively small cohort size and single-centre design may limit the statistical power to detect subtle associations and reduce the generalisability of the results to the broader GBS population. Inclusion was restricted to patients exhibiting anti-gan-

glioside antibody positivity; therefore, the observations pertain specifically to antibody distribution patterns rather than overall seroprevalence among all clinically suspected cases. The absence of microbiological confirmation of antecedent infections, particularly *Campylobacter jejuni*, precluded a comprehensive assessment of pathogen-antibody interactions. Furthermore, the cross-sectional nature of the study precluded the assessment of disease progression, treatment response, or long-term outcomes. Nonetheless, the study provides novel regional insights into anti-ganglioside antibody patterns and their clinical correlations in GBS, and highlights directions for future multicentre, longitudinal research.

Although taken together, these findings emphasise the importance of integrating electrophysiological findings with detailed clinical assessment and anti-ganglioside antibody profiling for a more accurate and early classification of GBS subtypes. These observations may support future diagnostic algorithms and early intervention strategies in resource-limited settings.

CONCLUSION

This study demonstrates a predominance of axonal GBS variants in a Pakistani cohort, with anti-GM1 antibodies significantly associated with motor predominant forms. Anti-GQ1b antibodies were specific to demyelinating subtypes, particularly MFS. Clinical features and antecedent infections varied by subtype, underscoring the value of combining serological, clinical, and electrophysiological data for accurate diagnosis and subtype classification in GBS. A multicentre study with larger sample sizes and longitudinal follow-up to validate the clinical utility of anti-ganglioside antibody profiling in Pakistan is recommended.

ETHICAL APPROVAL:

Ethical approval was obtained prior to the initiation of this study from the Ethical Review Committee of the Armed Forces Institute of Pathology, Rawalpindi, Pakistan (Approval No. Cons-IMM-3/READ-IRB/24/3900).

PATIENTS' CONSENT:

Informed consent was obtained from the patients.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

MA: Drafting, data analysis, and critical revision.

HNT: Conception and study design, data acquisition, analysis, and critical revision.

AH: Study design, data analysis, and critical revision.

MH: Data acquisition, analysis, and drafting of work.

WWM: Data analysis and critical revision.

All authors approved the final version of the manuscript to be published.

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