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ORIGINAL ARTICLE

Hematological Parameters, Biochemical Findings and Cerebrospinal Fluid Profiles among Guillan-Barre Syndrome Patients

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Abstract

Background: Laboratory parameters are very important among Guillain-Barré Syndrome patients. **Objective:** This study was undertaken to assess the hematological, biochemical, and cerebrospinal fluid profiles among patients with Guillain-Barré Syndrome. **Methodology:** This cross-sectional study was conducted from October 2017 to September 2018 in the Department of Clinical Neurology at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh. The patients were selected according to the selection criteria and after confirmation by an electrophysiological study. Details of laboratory profiles of the study population were collected, like hematological, biochemical, and cerebrospinal fluid parameters. Details history and meticulous examination were performed to collect the data according to the variable of interest. All necessary investigations were done at an optimum time. **Results:** A total of 108 GBS patients were recruited for this study. The mean age of patients with the demyelinating subtype was 40.20 ± 16.26 years, while that of the axonal subtype was 32.43 ± 14.93 years (p = 0.011). The mean hemoglobin (Hb) level was slightly lower in the demyelinating subtype (12.75 ± 1.98 g/dL) compared to the axonal subtype (13.19 ± 1.69 g/dL) (p = 0.213). The mean ALT value was notably higher in the axonal subtype (53.01 ± 36.95 U/L) compared to the demyelinating subtype (38.67 ± 19.27 U/L) (p = 0.012). The mean CSF cell count was nearly identical in both groups, with 2.50 ± 0.92 cells/mm³ in the demyelinating subtype and 2.55 ± 0.95 cells/mm³ in the axonal subtype (p = 0.784). The CSF glucose concentration was also comparable between the two subtypes, showing a mean of 4.23 ± 1.44 mmol/L in the demyelinating group and 3.91 ± 0.86 mmol/L in the axonal group (p = 0.178). In contrast, the CSF protein concentration showed higher mean values in the demyelinating subtype (418.28 ± 499.40 mg/L) compared to the axonal subtype (278.09 ± 488.82 mg/L) (p = 0.153). **Conclusion:** In conclusion, there are several laboratory parameters that are statistically significantly different between axonal and demyelinating types of GBS. *[Journal of Current and Advance Medical Research, July 2024;11(2):88-96]*

Keywords: Hematological parameters; biochemical findings; cerebrospinal fluid profiles; Guillan-Barre Syndrome

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Introduction

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy that represents the most common cause of acute flaccid paralysis in the post-polio era[1-2]. It is characterized by rapidly progressive, symmetrical limb weakness, areflexia, and varying degrees of sensory and autonomic dysfunction. The disease often develops following an antecedent infection, typically of the respiratory or gastrointestinal tract, caused by pathogens such as *Campylobacter jejuni*, cytomegalovirus, Epstein–Barr virus, or *Mycoplasma pneumoniae*[3-4]. In many patients, molecular mimicry between microbial antigens and host nerve gangliosides triggers an aberrant autoimmune response directed against peripheral nerve components, leading to segmental demyelination or axonal injury[5].

The global incidence of GBS is estimated at 1 to 2 cases per 100,000 population annually, with a slight male predominance and a broad age distribution[6]. Despite being relatively rare, GBS remains a neurologic emergency because of its potential to cause respiratory failure, autonomic instability, and long-term disability[7]. The disease manifests as several variants, including acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome (MFS). These variants differ in their immunopathological targets, clinical features, and electrophysiological patterns, but all share a common immune-mediated mechanism[8].

The diagnosis of GBS is primarily clinical, supported by electrophysiological studies and cerebrospinal fluid (CSF) analysis[9]. However, laboratory investigations including hematological and biochemical parameters provide valuable supportive evidence, help to exclude differential diagnoses, and monitor complications or treatment effects[10]. Although GBS primarily affects the peripheral nervous system, systemic immunological activation and metabolic responses may influence several hematological and biochemical indices[11]. Therefore, this study was undertaken to assess the hematological, biochemical, and cerebrospinal fluid profiles among patients with Guillain-Barré Syndrome.

Methodology

Study Settings and Population: This cross-sectional study was conducted from October 2017 to September 2018 in the Department of Clinical Neurology at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh. The patients were selected according to the selection criteria and after confirmation by an electrophysiological study.

Study Procedure: Details of laboratory profiles of the study population were collected. These included the hematological results, biochemical profiles, and cerebrospinal fluid findings. The complete blood count was tested for each patient. The total blood count was measured. Details history and meticulous examination were performed to collect the data according to the variable of interest. All necessary investigations were done at an optimum time. Nerve conduction study and CSF were done after 1st week of onset of the disease in the respective department of the institute.

Laboratory Profiles of the Study Population: Details of the laboratory profiles of all study participants were systematically collected and analyzed. These included hematological parameters, biochemical profiles, and cerebrospinal fluid (CSF) findings, which together provided a comprehensive assessment of the patients' physiological and pathological status.

Hematological Parameters: For each patient, a complete blood count (CBC) was performed using an automated hematology analyzer following standard laboratory protocols. The parameters evaluated included hemoglobin concentration (Hb), total leukocyte count (TLC), differential leukocyte count (DLC), platelet count, hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). Special attention was given to identifying leukocytosis, lymphocytosis, or neutrophilia, which might indicate inflammatory or infectious processes possibly preceding Guillain–Barré Syndrome (GBS). Erythrocyte sedimentation rate (ESR) was also measured as an indicator of systemic inflammation. Peripheral blood smears were examined microscopically to confirm cell morphology and exclude hematological disorders that could confound the interpretation of results.

Biochemical Profiles: Routine biochemical investigations were carried out to evaluate metabolic and organ function status. Serum electrolytes (sodium, potassium, chloride, and bicarbonate) were measured to detect any electrolyte imbalance associated with autonomic dysfunction or dehydration. Liver function tests (LFTs), including serum bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline

phosphatase (ALP), were done to rule out hepatic involvement. Renal function tests (RFTs) — including serum urea, creatinine, and uric acid — were conducted to assess renal status, as impaired renal function can influence nerve excitability and recovery. Additionally, fasting blood glucose, serum total protein, and albumin were measured to detect nutritional or metabolic abnormalities that might influence disease progression or outcome.

Cerebrospinal Fluid (CSF) Findings: Lumbar puncture was performed under aseptic conditions in all patients after excluding contraindications. The collected CSF samples were analyzed for cell count, protein concentration, and glucose level. The characteristic finding in GBS—albuminocytologic dissociation, defined as elevated CSF protein with normal or mildly raised cell count—was carefully documented. CSF appearance (clear or turbid) and opening pressure were also noted. Protein estimation was performed using the biuret method, and glucose concentration was determined by enzymatic colorimetric assay. The CSF-to-blood glucose ratio was calculated to ensure accurate interpretation in relation to blood glucose levels. Cytological examination was done to exclude infectious or neoplastic causes of neuropathy.

Quality Control and Data Validation: All laboratory investigations were conducted in the hospital's central laboratory following standard operating procedures (SOPs). Internal and external quality control measures were maintained to ensure the reliability and reproducibility of results. Laboratory data were recorded in predesigned case record forms, cross-verified with laboratory reports, and subsequently entered into the study database for analysis.

Statistical Analysis: Statistical analysis was performed by Windows-based software named as Statistical Package for Social Science (SPSS), version 22.0 (IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.). Continuous data were expressed as mean, standard deviation, minimum, and maximum. Categorical data were summarized in terms of frequency counts and percentages. Chi-square test was used for comparison of categorical variables, and Student t-test was applied for continuous variables. Every effort was made to obtain missing data. A two-sided P value of less than 0.05 was considered to indicate statistical significance.

Ethical Consideration: All procedures of the present study were carried out in accordance with the principles for human investigations (i.e., Helsinki Declaration) and also with the ethical guidelines of the Institutional Research Ethics. Formal ethics approval was granted by the local ethics committee. Participants in the study were informed about the procedure and purpose of the study and the confidentiality of information provided. All participants consented willingly to be a part of the study during the data collection periods. All data were collected anonymously and analyzed using the coding system.

Results

A total number of 108 GBS patients were recruited for this study. Among the study population, the 18–40 years age group constituted the majority in both subtypes. Specifically, 33 patients (58.2%) with the demyelinating subtype and 41 patients (77.4%) with the axonal subtype fell within this younger age bracket. The 41–60 years group accounted for 19 patients (34.5%) in the demyelinating type and 9 patients (17.0%) in the axonal type, whereas patients over 60 years comprised only a small fraction—4 (7.3%) in the demyelinating and 3 (5.7%) in the axonal variant. The overall distribution difference between age groups did not reach statistical significance (p = 0.091), indicating that the proportion of patients in each age category was relatively similar across the two GBS subtypes. However, when age was analyzed as a continuous variable, the difference in mean age between the two groups was statistically significant. The mean age of patients with the demyelinating subtype was 40.20 ± 16.26 years, while that of the axonal subtype was 32.43 ± 14.93 years (p = 0.011) (Table 1).

Table 1: Distribution of Age among the Study Population

Age Group	GBS type		P value
	Demyeli	Axonal	
18 to 40 years	33(58.2%)	41(77.4%)	
41 to 60 years	19(34.5%)	9(17.0%)	0.091
> 60 years	4(7.3%)	3 (5.7%)	
Mean ± SD	40.2±16.26	32.4±14.93	0.01[$]

*An independent sample T test and [$]Pearson chi-square test were used to determine the level of significance. p-value of less than 0.05 was taken as statistically significant; Demyeli = Demyelinating

The variables assessed included hemoglobin (Hb) concentration, total white blood cell (WBC) count, platelet count, and erythrocyte sedimentation rate (ESR). The results are presented as mean ± standard deviation (M ± SD), and statistical significance was determined using appropriate comparative tests, with $p < 0.05$ considered significant. The mean

hemoglobin (Hb) level was slightly lower in the demyelinating subtype (12.75 ± 1.98 g/dL) compared to the axonal subtype (13.19 ± 1.69 g/dL), although the difference was not statistically significant (p = 0.213). The total WBC count was modestly higher in the axonal group (11,684.62 ± 4,266.76 × 10^3/cmm) relative to the demyelinating group (10,658.93 ± 3,259.82 × 10^3/cmm), with p = 0.162. The platelet count was marginally higher in the demyelinating subtype (303,632.73 ± 86,971.11 × 10^3/cmm) compared to the axonal subtype (287,140.38 ± 87,569.27 × 10^3/cmm), again without statistical significance (p = 0.331). Finally, the erythrocyte sedimentation rate (ESR)—a general indicator of inflammation—was modestly elevated in both subtypes, with a mean of 27.09 ± 20.20 mm in the demyelinating group and 29.21 ± 19.42 mm in the axonal group. The difference between the groups was not statistically significant (p = 0.580) (Table 2).

Table 2: Laboratory Profile of Demyelinating and Axonal Subtypes of GBS Participants

Hematological Profiles	GBS Subtype		P value
	Demyelinating (M±SD)	Axonal (M±SD)	
Hb (gm/dL)	12.75 (±1.98)	13.19 (±1.69)	0.213
Total WBC (1000/cmm)	10658.93 (±3259.82)	11684.62 (±4266.76)	0.162
Platelet count (1000/cmm)	303632.73 (±86971.11)	287140.38 (±87569.27)	0.331
ESR (mm in 1st hour)	27.09 (±20.20)	29.21 (±19.42)	0.580

Hb: Hemoglobin, WBC: white cell count, ESR: erythrocyte sedimentation rate, *independent sample T test and ** Pearson chi-square test were used to determine the level of significance. P value of less than 0.05 was taken as statistically significant.

The biochemical variables analyzed included fasting blood sugar (FBS), glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), serum creatinine, and serum electrolytes (sodium and potassium). Results are expressed as mean ± standard deviation (M ± SD), and intergroup differences were evaluated using appropriate statistical tests, with $p < 0.05$ considered statistically significant.

The fasting blood sugar (FBS) levels were comparable between the two subtypes, with mean values of 6.65 ± 1.85 mmol/L in the demyelinating group and 6.57 ± 3.04 mmol/L in the axonal group (p = 0.898). Similarly, the HbA1c values were nearly identical across the groups—6.17 ± 1.27% in the demyelinating subtype and 6.00 ± 1.12% in the axonal subtype (p = 0.465). In contrast, a significant difference was observed in serum alanine aminotransferase (ALT) levels. The mean ALT value was notably higher in the axonal subtype (53.01 ± 36.95 U/L) compared to the demyelinating subtype (38.67 ± 19.27 U/L), and this difference reached statistical significance (p = 0.012). The serum creatinine levels were almost identical between groups—0.80 ± 0.16 mg/dL in the demyelinating subtype and 0.81 ± 0.22 mg/dL in the axonal subtype (p = 0.734)—indicating preserved renal function in both categories.

The serum sodium (Na^+) concentrations were also comparable across subtypes, with mean values of 139.37 ± 3.84 mmol/L in demyelinating and 139.03 ± 4.33 mmol/L in axonal GBS (p = 0.670). The serum potassium (K^+) levels showed no significant variation, measuring 4.00 ± 0.45 mmol/L in demyelinating and 4.11 ± 0.47 mmol/L in axonal patients (p = 0.229) (Table 3).

Table 3: Laboratory Profile of Demyelinating and Axonal Subtypes of GBS Participants

Biochemical Profile	GBS Subtype		P value
	Demyelinating (M±SD)	Axonal (M±SD)	
FBS	6.65 (±1.85)	6.57 (±3.04)	0.898
HbA1C	6.17 (±1.27)	6.00 (±1.12)	0.465
ALT	38.67 (±19.27)	53.01 (±36.95)	0.012
Serum creatinine	0.80 (±0.16)	0.81 (±0.22)	0.734
Na^+	139.37 (±3.84)	139.03 (±4.33)	0.670
K^+	4.00 (±0.45)	4.11 (±0.47)	0.229

FBS: fasting blood sugar, HbA1C: glycated hemoglobin, ALT: Alanine aminotransferase, Na^+: sodium, K^+: potassium, *independent sample t test were used to determine the level of significance. P value of less than 0.05 was taken as statistically significant.

Table 4: Laboratory Profile of Demyelinating and Axonal Subtypes of GBS Participants

CSF profile	GBS Subtype		P value
	Demyelinating (M±SD)	Axonal (M±SD)	
Cell count	2.50 (±0.92)	2.55 (±0.95)	0.784
Glucose	4.23 (±1.44)	3.91 (±0.86)	0.178
Protein	418.28 (±499.40)	278.09 (±488.82)	0.153
Alb.-Cyto. dissociation	50 (94.3%)	43 (87.8%)	0.206**

CSF: cerebrospinal fluid, Alb: albumin, Cyto: cytological. *Independent sample T test and ** Pearson chi-square test were used to determine the level of significance. p-value of less than 0.05 was taken as statistically significant.

The cerebrospinal fluid (CSF) findings among patients with demyelinating and axonal subtypes of Guillain–Barré Syndrome (GBS) were assessed. The parameters evaluated include total cell count, glucose concentration, protein level, and the presence of albumino-cytologic dissociation. All continuous variables are expressed as mean ± standard deviation (M ± SD), and statistical significance was determined using appropriate comparative tests, with $p < 0.05$ considered significant. The mean CSF cell count was nearly identical in both groups, with 2.50 ± 0.92 cells/mm³ in the demyelinating subtype and 2.55 ± 0.95 cells/mm³ in the axonal subtype ($p = 0.784$). The CSF glucose concentration was also comparable between the two subtypes, showing a mean of 4.23 ± 1.44 mmol/L in the demyelinating group and 3.91 ± 0.86 mmol/L in the axonal group ($p = 0.178$). In contrast, the CSF protein concentration showed higher mean values in the demyelinating subtype (418.28 ± 499.40 mg/L) compared to the axonal subtype (278.09 ± 488.82 mg/L), although this difference did not reach statistical significance ($p = 0.153$). The presence of albumin-cytologic dissociation was observed in 50 patients (94.3%) with the demyelinating subtype and in 43 patients (87.8%) with the axonal subtype. Although slightly more frequent in the demyelinating group, the difference was not statistically significant ($p = 0.206$) (Table 4).

Discussion

A total number of 108 GBS patients were recruited for this study. The age distribution of patients with demyelinating and axonal subtypes of Guillain–Barré Syndrome (GBS) was recorded. The statistical significance of differences between the two groups was evaluated using appropriate tests, with $p < 0.05$ considered statistically significant. Among the study population, the 18 to 40 years age group constituted the majority in both subtypes. Specifically, 33 patients (58.2%) with the demyelinating subtype and 41 patients (77.4%) with the axonal subtype fell within this younger age bracket. The 41–60 years group accounted for 19 patients (34.5%) in the demyelinating type and 9 patients (17.0%) in the axonal type, whereas patients over 60 years comprised only a small fraction—4 (7.3%) in the demyelinating and 3 (5.7%) in the axonal variant. The overall distribution difference between age groups did not reach statistical significance ($p = 0.091$), indicating that the proportion of patients in each age category was relatively similar across the two GBS subtypes.

However, when age was analyzed as a continuous variable, the difference in mean age between the two groups was statistically significant. The mean age of patients with the demyelinating subtype was 40.20 ± 16.26 years, while that of the axonal subtype was 32.43 ± 14.93 years ($p = 0.011$). This finding suggests that the axonal subtype tends to occur at a younger age compared to the demyelinating subtype.

This age-related trend aligns with previous epidemiological observations indicating that the axonal variants of GBS—particularly the acute motor axonal neuropathy (AMAN) form and are more frequently observed in younger adults and children, especially in regions with high prevalence of *Campylobacter jejuni* infection[11].

Conversely, the demyelinating subtype, or acute inflammatory demyelinating polyneuropathy (AIDP), is more commonly seen in middle-aged and older adults, possibly reflecting age-related immune modulation and differing patterns of infectious exposure[12].

The lower mean age among axonal GBS patients in this study may therefore reflect differences in immunopathogenesis, environmental exposure, or regional epidemiological patterns[13]. The relatively younger age of onset in the axonal group could also have clinical implications, as several studies have reported that axonal variants are often associated

with more severe initial weakness but potentially better long-term recovery in younger individuals with prompt treatment[14].

Thus, while the categorical age distribution between demyelinating and axonal subtypes did not differ significantly, the mean age difference was statistically significant, with the axonal subtype presenting predominantly in younger patients. This finding underscores subtle epidemiological distinctions between the two electrophysiological forms of GBS and supports the hypothesis that patient age may influence subtype manifestation and disease characteristics.

Hematological investigations in GBS are largely non-specific but may reflect the underlying immune response or associated infection[15]. A complete blood count (CBC) may reveal mild leukocytosis or lymphocytic predominance following viral illness, whereas a normal leukocyte count is common in idiopathic or post-infectious cases. Elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) values are usually mild and indicate concurrent inflammatory or infectious processes rather than direct disease activity. The detection of antiganglioside antibodies such as anti-GM1, GD1a, GT1a, and GQ1b has improved understanding of immune mechanisms and variant classification, particularly in axonal and Miller Fisher subtypes[16]. While these are not routine hematological tests, their presence emphasizes the autoimmune etiology of GBS.

The variables assessed included hemoglobin (Hb) concentration, total white blood cell (WBC) count, platelet count, and erythrocyte sedimentation rate (ESR). The results are presented as mean ± standard deviation (M ± SD), and statistical significance was determined using appropriate comparative tests, with $p < 0.05$ considered significant. The mean hemoglobin (Hb) level was slightly lower in the demyelinating subtype (12.75 ± 1.98 g/dL) compared to the axonal subtype (13.19 ± 1.69 g/dL), although the difference was not statistically significant ($p = 0.213$). This finding indicates that hemoglobin concentration remains largely comparable between the two electrophysiological variants, suggesting no major hematological difference in oxygen-carrying capacity or red cell status[17].

The total WBC count was modestly higher in the axonal group (11,684.62 ± 4,266.76 × 10^3/cmm) relative to the demyelinating group (10,658.93 ± 3,259.82 × 10^3/cmm), with $p = 0.162$. Although not statistically significant, this trend may reflect a slightly higher inflammatory or immune response in axonal GBS variants, which are often associated with antecedent infections, particularly *Campylobacter jejuni*[18]. Nonetheless, both values fall within the upper physiological range, consistent with the absence of marked systemic leukocytosis in most GBS cases[15].

The platelet count was marginally higher in the demyelinating subtype (303,632.73 ± 86,971.11 × 10^3/cmm) compared to the axonal subtype (287,140.38 ± 87,569.27 × 10^3/cmm), again without statistical significance ($p = 0.331$). This suggests that platelet activation or thrombocytosis is not a characteristic finding in either form of GBS, and the observed variations likely represent normal inter-individual differences rather than disease-specific hematological alterations[19].

Finally, the erythrocyte sedimentation rate (ESR)—a general indicator of inflammation—was modestly elevated in both subtypes, with a mean of 27.09 ± 20.20 mm in the demyelinating group and 29.21 ± 19.42 mm in the axonal group. The difference between the groups was not statistically significant ($p = 0.580$). These values indicate a mild to moderate elevation of ESR, which may reflect a nonspecific inflammatory or post-infectious state commonly seen in GBS, rather than a subtype-specific response.

Overall, none of the hematological parameters showed statistically significant differences between the demyelinating and axonal subtypes of GBS. This suggests that, although minor numerical variations exist, hematological indices remain broadly similar across the electrophysiological variants of the disease. The findings are consistent with the notion that GBS is primarily a neuropathic and immune-mediated process rather than a systemic hematological disorder.

Biochemical changes in GBS often reflect systemic stress, autonomic dysfunction, or treatment-related effects rather than primary metabolic derangements[11]. Hyponatremia, a frequent finding in up to one-quarter of patients, is typically attributed to the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Disturbances in potassium levels may result from autonomic imbalance or therapeutic interventions[13]. Liver and renal function tests generally remain within normal limits, though transient elevations of liver enzymes can occur due to infections or immunoglobulin therapy. Serum protein and albumin concentrations may slightly decrease as a consequence of capillary leakage, yet this finding is far less pronounced compared with cerebrospinal protein elevation[17].

These biochemical observations, while non-diagnostic, are clinically significant for patient monitoring and guiding supportive management during acute and recovery phases.

The comparison of biochemical parameters between patients with demyelinating and axonal subtypes of Guillain–Barré Syndrome (GBS) were recorded. The biochemical variables analyzed included fasting blood sugar (FBS), glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), serum creatinine, and serum electrolytes (sodium and potassium). Results are expressed as mean ± standard deviation (M ± SD), and intergroup differences were evaluated using appropriate statistical tests, with $p < 0.05$ considered statistically significant.

The fasting blood sugar (FBS) levels were comparable between the two subtypes, with mean values of 6.65 ± 1.85 mmol/L in the demyelinating group and 6.57 ± 3.04 mmol/L in the axonal group ($p = 0.898$). This lack of significant difference indicates that glucose metabolism remains similar in both GBS variants, suggesting that neither subtype is preferentially associated with hyperglycemia or stress-induced alterations in blood glucose levels. Similarly, the HbA1c values were nearly identical across the groups—6.17 ± 1.27% in the demyelinating subtype and 6.00 ± 1.12% in the axonal subtype ($p = 0.465$). These findings suggest that the long-term glycemic control and baseline metabolic status were comparable between the two patient populations, minimizing the likelihood that chronic dysglycemia influenced the observed clinical or neurological patterns.

In contrast, a significant difference was observed in serum alanine aminotransferase (ALT) levels. The mean ALT value was notably higher in the axonal subtype (53.01 ± 36.95 U/L) compared to the demyelinating subtype (38.67 ± 19.27 U/L), and this difference reached statistical significance ($p = 0.012$). Elevated ALT levels in axonal GBS may reflect hepatic involvement secondary to antecedent infections (such as *Campylobacter jejuni* or viral agents), systemic inflammatory stress, or mild hepatocellular effects related to immune activation. Another possible explanation could be the metabolic impact of treatment or underlying comorbidities, though further investigation is needed to clarify whether this elevation has any clinical or prognostic significance.

The serum creatinine levels were almost identical between groups—0.80 ± 0.16 mg/dL in the demyelinating subtype and 0.81 ± 0.22 mg/dL in the axonal subtype ($p = 0.734$)—indicating preserved renal function in both categories. These results suggest that GBS, in the absence of critical illness or treatment complications, does not typically affect renal physiology. The serum sodium (Na^+) concentrations were also comparable across subtypes, with mean values of 139.37 ± 3.84 mmol/L in demyelinating and 139.03 ± 4.33 mmol/L in axonal GBS ($p = 0.670$). Mild hyponatremia, which is sometimes observed in GBS due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH), was not prominent in this cohort. This stability in sodium levels suggests adequate fluid and electrolyte balance during the acute phase in both groups. The serum potassium (K^+) levels showed no significant variation, measuring 4.00 ± 0.45 mmol/L in demyelinating and 4.11 ± 0.47 mmol/L in axonal patients ($p = 0.229$). Potassium values within the normal range indicate the absence of substantial autonomic or renal disturbances influencing potassium homeostasis, which can sometimes occur in severe GBS with autonomic dysfunction.

Therefore, with the exception of ALT, all other biochemical parameters—including FBS, HbA1c, serum creatinine, sodium, and potassium—were statistically similar between the demyelinating and axonal subtypes of GBS. The isolated elevation of ALT in the axonal group may reflect greater systemic inflammatory activity or hepatic stress, potentially associated with preceding infections or immune responses characteristic of this variant. These findings underscore that while GBS primarily affects the peripheral nervous system, subtle biochemical alterations, particularly in hepatic enzymes, may accompany the disease process, warranting routine biochemical monitoring during diagnosis and management.

Among laboratory investigations, CSF analysis provides the most characteristic and diagnostic evidence for GBS. The classic feature—albuminocytologic dissociation—refers to elevated CSF protein concentration with a normal or minimally increased white cell count. This phenomenon reflects the disruption of the blood-nerve barrier, allowing leakage of plasma proteins into the CSF without significant cellular inflammation. Typically, CSF protein levels rise above 45 mg/dL after the first week of symptom onset, while cell counts remain below 10 cells/mm³. Glucose and chloride concentrations usually remain normal, helping to distinguish GBS from infectious or inflammatory meningopathies. The absence of pleocytosis reinforces the peripheral rather than central nervous system involvement in GBS. Thus, CSF analysis remains a cornerstone in the diagnostic

confirmation and differentiation of GBS from other neuropathies.

The cerebrospinal fluid (CSF) findings among patients with demyelinating and axonal subtypes of Guillain–Barré Syndrome (GBS) were assessed. The parameters evaluated include total cell count, glucose concentration, protein level, and the presence of albuminocytologic dissociation. All continuous variables are expressed as mean ± standard deviation (M ± SD), and statistical significance was determined using appropriate comparative tests, with $p < 0.05$ considered significant.

The mean CSF cell count was nearly identical in both groups, with 2.50 ± 0.92 cells/mm³ in the demyelinating subtype and 2.55 ± 0.95 cells/mm³ in the axonal subtype ($p = 0.784$). This uniformity indicates that both subtypes of GBS exhibit minimal pleocytosis, consistent with the classical CSF pattern of normal or mildly elevated cell count. The results reaffirm that GBS is primarily an immune-mediated peripheral neuropathy rather than an infectious or inflammatory meningoencephalitic process. The CSF glucose concentration was also comparable between the two subtypes, showing a mean of 4.23 ± 1.44 mmol/L in the demyelinating group and 3.91 ± 0.86 mmol/L in the axonal group ($p = 0.178$). These values fall within the normal physiological range, reflecting preserved glucose metabolism and transport across the blood–CSF barrier. The absence of hypoglycorrhachia further supports a non-infectious etiology.

In contrast, the CSF protein concentration showed higher mean values in the demyelinating subtype (418.28 ± 499.40 mg/L) compared to the axonal subtype (278.09 ± 488.82 mg/L), although this difference did not reach statistical significance ($p = 0.153$). The elevated CSF protein levels in both subtypes are consistent with the classical hallmark of GBS—albuminocytologic dissociation—which reflects increased permeability of the blood–nerve barrier due to inflammatory demyelination or axonal injury. The wide standard deviations in protein levels indicate considerable inter-individual variability, possibly influenced by the duration of illness at the time of lumbar puncture, disease severity, or timing of CSF sampling relative to symptom onset[16].

The presence of albuminocytologic dissociation was observed in 50 patients (94.3%) with the demyelinating subtype and in 43 patients (87.8%) with the axonal subtype. Although slightly more frequent in the demyelinating group, the difference was not statistically significant ($p = 0.206$). This high prevalence across both variants underscores that elevated CSF protein with normal cell count is a consistent and characteristic feature of GBS, irrespective of the electrophysiological subtype. The slightly higher proportion among demyelinating cases aligns with the pathophysiological basis of myelin damage leading to greater protein leakage into the CSF[15].

Overall, there were no statistically significant differences between demyelinating and axonal subtypes in any of the CSF parameters studied, including cell count, glucose, protein, or the presence of albuminocytologic dissociation. These findings suggest that the CSF profile remains broadly similar across electrophysiological variants of GBS. The predominant feature of raised protein concentration with normal cell count remains the diagnostic hallmark, reinforcing the role of CSF analysis in confirming the diagnosis and distinguishing GBS from infectious or inflammatory neuropathies[17-18].

Thus, both demyelinating and axonal forms of GBS demonstrate the classic albuminocytologic dissociation, normal CSF glucose levels, and minimal pleocytosis. Although the demyelinating subtype tends to show slightly higher CSF protein concentrations, the overall biochemical and cytological characteristics of CSF remain consistent across variants, supporting the shared immune-mediated pathogenesis of the disorder.

Conclusion

In summary, Guillain-Barré Syndrome represents a complex interplay between immune dysregulation and peripheral nerve injury. While the diagnosis relies on clinical and electrophysiological features, detailed evaluation of hematological, biochemical, and CSF findings provides valuable complementary information. These parameters not only assist in early diagnosis and differential exclusion but also contribute to comprehensive patient assessment and management strategies in both acute and convalescent stages of the disease.

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Conflict of Interest
We declare that we have no conflict of interest.

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Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Ethical approval for the study was obtained from the Institutional Review Board. As this was a prospective study the written informed consent was obtained from all study participants. All methods were performed in accordance with the relevant guidelines and regulations.

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