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Ratio of neutrophil to lymphocytes, albumin and sodium levels as predictive markers of Post-Plasmapheresis clinical response in Guillain Barre Syndrome (SGB) patients

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Abstract

Guillain–Barré Syndrome (GBS) is an acute autoimmune disorder of the peripheral nervous system that can cause progressive paralysis and may be life-threatening. Plasmapheresis has long been established as a primary immunomodulatory therapy for patients with GBS; however, post-treatment clinical responses vary considerably among patients. Therefore, simple, readily available, and clinically applicable predictive markers are needed to help estimate patient outcomes. This study aimed to analyze the roles of the neutrophil-to-lymphocyte ratio (NLR), serum albumin levels, and serum sodium levels as predictive markers of clinical response following plasmapheresis in patients with Guillain–Barré syndrome. This study employed a cross-sectional design using secondary data obtained from the medical records of GBS patients who underwent plasmapheresis at Dr. Saiful Anwar General Hospital, Malang. A total of 84 patients who met the inclusion criteria were analyzed and classified into good and poor clinical response groups based on post-treatment outcomes. Statistical analyses were performed using the Mann–Whitney test and prevalence ratio calculations. The results demonstrated that patients with normal or low NLR values were 2.5 times more likely to exhibit a good clinical response compared with those with high NLR values ($p = 0.001$). In addition, normal or high serum albumin levels were significantly associated with better clinical responses (PR = 2.13; $p = 0.003$). In contrast, serum sodium levels showed no statistically significant association with clinical response after plasmapheresis ($p = 0.970$). In conclusion, NLR and serum albumin levels have the potential to serve as predictive markers of clinical response in patients with GBS following plasmapheresis, whereas serum sodium levels were not shown to be significant predictors. These findings are expected to support clinical decision-making and improve risk stratification in patients with Guillain–Barré syndrome.

Keywords: Guillain–Barré syndrome, Plasmapheresis, Neutrophil-To-Lymphocyte ratio, Albumin, Sodium.

Introduction

Guillain–Barré Syndrome (GBS) is an inflammatory disorder of the peripheral nervous system and represents the most common cause of acute flaccid paralysis worldwide, with an estimated annual global incidence of approximately 1–2 cases per 100,000 populations. GBS occurs more frequently in males than in females, and its incidence increases with advancing age, although individuals of all age groups may be affected. Patients with GBS typically present with progressive muscle weakness and sensory disturbances that begin in the lower limbs and may ascend to involve the upper extremities and cranial muscles. Despite these characteristic features, the clinical presentation of GBS is heterogeneous, with several recognized clinical variants. The diagnosis of GBS is primarily based on clinical history and supported by neurological examination, electrophysiological studies, and Cerebrospinal Fluid (CSF) analysis (Leonhard et al., 2019; Jam et al., 2018).

Guillain–Barré syndrome is commonly preceded by an infectious episode or other immune stimulation that triggers an aberrant autoimmune response targeting peripheral nerves and spinal nerve roots. Molecular mimicry between microbial antigens and neural components is widely recognized as a key pathogenic mechanism, particularly in cases associated with Campylobacter jejuni infection. However, the complex interaction between microbial factors and host immune responses that determines the diversion of immunity toward pathological autoreactivity remains incompletely understood (Willison et al., 2016).

Guillain–Barré syndrome is a potentially life-threatening condition, requiring prompt general medical care and immunomodulatory treatment. Plasmapheresis has been accepted as a gold-standard therapy for GBS for nearly two decades. The effectiveness of both plasmapheresis and intravenous immunoglobulin (IVIg) has been demonstrated in multiple randomized controlled trials. Nevertheless, following evidence from

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randomized trials showing that IVIg has efficacy comparable to plasmapheresis, IVIg has gradually replaced plasmapheresis as the preferred treatment for severe GBS in many healthcare settings, primarily due to its greater convenience and ease of administration (Leonhard et al., 2019).

Despite advances in treatment, Guillain–Barré syndrome remains associated with substantial morbidity. Patients may die during the acute progressive phase of the disease, most commonly due to respiratory complications or autonomic dysfunction, including cardiac arrhythmias. Survivors frequently experience residual neurological deficits that significantly impair daily functioning and quality of life. Improving outcomes in GBS therefore requires not only effective therapeutic interventions but also accurate assessment of prognosis. Prognostic models play a critical role in identifying patients who may require intensified treatment, closer monitoring, and prolonged rehabilitation (Willison et al., 2016).

Plasmapheresis, which represents a primary therapeutic modality for Guillain–Barré syndrome (GBS), involves the separation of plasma from the cellular components of blood by centrifugation, followed by the removal and replacement of the patient's plasma with an equal volume of replacement fluid consisting of fresh frozen plasma (FFP) and 5% albumin solution to maintain adequate oncotic pressure, while the remaining cellular elements are reinfused into the patient. In GBS, this procedure modulates immune activity by altering the ratio of T helper 1 (Th1) to Th2 cells, modifying the number and activation of B and T lymphocytes, and reducing the circulating concentrations of pathogenic plasma components such as autoantibodies, immune complexes, complement factors, cytokines, and other immunologically active substances. According to a recent Cochrane review, the therapeutic benefit of plasmapheresis is greatest when treatment is initiated early, particularly within 7 days of disease onset, although beneficial effects may still be observed when initiated within the first 4 weeks (Huy et al., 2019).

According to the 2019 guidelines of the American Society for Apheresis (ASFA), plasmapheresis for GBS is classified as an ASFA Category I indication with a Level 1A recommendation. The guidelines recommend exchanging 1–1.5 plasma volumes per session, administered 5–6 times over a period of 10–14 days on an every-other-day schedule. Plasmapheresis typically removes approximately 60–70% of substances from the intravascular compartment. Intervals between procedures are necessary to allow re-equilibration of the intravascular space and to reduce the risk of bleeding associated with depletion of anticoagulation factors, particularly fibrinogen. The most commonly reported adverse effects of plasmapheresis include allergic reactions to plasma (such as chills, fever, rash, urticaria, dyspnea, and stridor), chest pain, dizziness, headache, abdominal pain, anxiety, hypotension, nausea, and vomiting, with an incidence of approximately 11% compared with patients receiving 5% albumin solution as replacement fluid. Other adverse effects include symptoms of hypocalcemia when citrate is used as an anticoagulant, due to its binding to ionized calcium in the blood. Albumin-based replacement fluids are primarily associated with adverse effects such as hypotension, nausea, and vomiting, largely attributable to hypo-oncotic effects (Padmanabhan et al., 2019).

The neutrophil-to-lymphocyte ratio (NLR) is an inflammatory marker that reflects the balance between acute inflammatory responses mediated by neutrophils and adaptive immune responses mediated by lymphocytes. Elevated NLR has been associated with systemic inflammation and adverse outcomes in a variety of diseases, including neurological disorders such as GBS. A study by Erdem et al. (2018) demonstrated that higher NLR values were predictive of disease severity and poorer clinical outcomes in patients with GBS.

Another biomarker relevant to GBS is serum albumin. Albumin serves as the primary source of sulfhydryl and thiol groups and plays a crucial role in scavenging reactive oxygen species, nitrogen radicals, and toxins. In addition, albumin contributes to the maintenance of colloid osmotic pressure, substance binding and transport, antioxidant activity, platelet inhibition, anticoagulation, erythrocyte aggregation, preservation of vascular permeability, and acid–base balance (Makris et al., 2016).

The pathophysiology of GBS leads to decreased serum albumin levels, primarily due to systemic acute inflammatory responses. Hypoalbuminemia is associated with acute-phase protein reactions and elevated C-Reactive Protein (CRP) levels, reflecting ongoing inflammatory processes (Ethemoglu et al., 2018). A study conducted in the Special Region of Yogyakarta in 2021 reported that 14% of patients diagnosed with GBS exhibited hypoalbuminemia, defined as serum albumin levels below 3.5 g/dL (Harahap et al., 2021).

Serum albumin is therefore an important biomarker, as it functions as a negative acute-phase protein that reflects both nutritional status and systemic inflammation. Low albumin levels are frequently associated with more severe inflammatory states and malnutrition, both of which may adversely affect the prognosis of patients with GBS. A study by Kim et al. (2017) demonstrated that lower serum albumin levels were associated with greater clinical severity in GBS and could serve as a predictor of treatment response.

Hydroelectrolyte disturbances are frequently associated with severe neurological diseases and are considered unfavorable prognostic factors, with sodium being the electrolyte most commonly involved. Hyponatremia is one of the most frequent electrolyte abnormalities observed in hospitalized patients, regardless of the underlying primary disease, and is particularly common in GBS. The reported prevalence of hyponatremia ranges from 21.5% to 48% among hospitalized patients and has been identified as a poor prognostic factor, associated with alcohol misuse, anemia, hypertension, coagulopathy, malignancy, diuretic use, and a history of diarrhea. Hyponatremia is not typically a pre-existing condition prior to disease onset but rather a complication arising from disease-related pathophysiological mechanisms, such as the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Hyponatremia may progress in severity, and fluid restriction remains the most commonly employed therapeutic approach (Cui et al., 2019).

In addition, serum sodium plays a critical role in maintaining electrolyte balance, and disturbances in sodium homeostasis can adversely affect neural and cardiovascular function. Reduced sodium levels have been reported to be associated with poorer clinical outcomes in patients with GBS (Patel et al., 2020). A more recent study by Martinez et al. (2023) found that 14% of patients with GBS developed hyponatremia, which was associated with dysautonomia and prolonged hospital length of stay. Based on these considerations, this study aimed to investigate whether NLR, serum albumin levels, and serum sodium levels could serve as predictive markers of clinical response following plasmapheresis in patients with Guillain–Barré syndrome.

Methods

Study design

This study employed a cross-sectional design using secondary data obtained from the medical records of patients with Guillain–Barré Syndrome (GBS) who underwent plasmapheresis. Patients were classified into two groups based on post-treatment clinical outcomes: a good outcome group and a poor outcome group. The Neutrophil-To-Lymphocyte Ratio (NLR), serum albumin levels, and serum sodium levels measured prior to plasmapheresis were analyzed. The quantitative strength of the predictive association was assessed using prevalence ratios, which are appropriate for a cross-sectional study design.

Study population and subjects

The study population consisted of patients with Guillain–Barré syndrome who sought medical care at Dr. Saiful Anwar General Hospital, Malang. The study subjects were patients who had been clinically diagnosed with GBS and subsequently underwent plasmapheresis at the same institution. Diagnosis was established based on clinical evaluation by consultant neurologists and internal medicine specialists, supported by relevant laboratory findings.

The control group comprised GBS patients who underwent plasmapheresis and demonstrated a good clinical outcome, whereas the case group consisted of GBS patients who underwent plasmapheresis and exhibited a poor clinical outcome.

All study subjects underwent comprehensive clinical assessment, including medical history taking (anamnesis), diagnostic physical examination, and laboratory blood tests, which included measurements of the neutrophil-To-Lymphocyte Ratio (NLR), serum albumin levels, and serum sodium levels.

Sample size

The sample size for this study was calculated using Slovin's formula. This study was designed as an unpaired numerical comparative study. The required sample size was calculated using the following formula:

$$n = \frac{N}{1 + Ne^2}$$

where:

n = sample size
N = population size
e^2 = margin of error (significance level) of 5% (0.05)

Based on the calculation using a margin of error of 5% and a population size (N) of 84, the required sample size for this study was determined to be 80 patients.

A consecutive sampling method was applied, including all eligible patients who met the inclusion and exclusion criteria at the Neurology Outpatient Clinic and the Apheresis Unit of the Division of Medical Hematology and Oncology, Faculty of Medicine, Universitas Brawijaya – Dr. Saiful Anwar General Hospital, Malang.

Study variables

This study involved two types of variables: independent and dependent variables. The independent variable was Guillain–Barré syndrome patients who received plasmapheresis. The dependent variables were the Neutrophil-To-Lymphocyte Ratio (NLR), serum albumin levels, and serum sodium levels.

Study location and period

The study was conducted at Dr. Saiful Anwar General Hospital, Malang. Data collection was carried out over a seven-year period, from January 2019 to December 2025.

Research flow

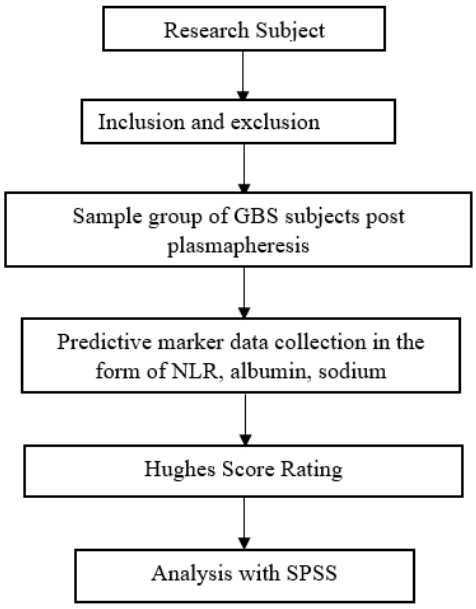

Figure 1 Research flow

Data collection and statistical analysis

Tabel 1. Results of the normality test

	Kolmogorov-Smirnov			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
NLR	.274	84	.000	.497	84	.000
Albumin	.143	84	.000	.969	84	.038
Sodium	.097	84	.051	.951	84	.003
Score Hughes	.426	84	.000	.595	84	.000

Based on the Kolmogorov–Smirnov and Shapiro–Wilk normality tests, the distribution of data across all study variables demonstrated heterogeneous results. A total of 84 samples met the inclusion criteria in this study; therefore, normality testing was primarily evaluated using the Kolmogorov–Smirnov method. The Kolmogorov–Smirnov test showed significant results for the neutrophil-to-lymphocyte ratio (NLR) ($p < 0.001$), serum albumin levels ($p < 0.001$), serum sodium levels ($p = 0.051$), and post-plasmapheresis Hughes scores ($p < 0.001$).

Although the Kolmogorov–Smirnov test for serum sodium yielded a borderline value ($p = 0.051$), the Shapiro–Wilk test remained significant ($p < 0.05$); therefore, the sodium data were also considered non-normally distributed. Consequently, numerical data are presented as medians with interquartile ranges (IQRs), and bivariate analyses were conducted using the non-parametric Mann–Whitney U test.

Baseline characteristics

Table 2. Basic characteristics of numerical data

	N	Range	Min	Max	Sum	Median	Variance
NLR	84	92.47	.53	93.00	686.83	4.47	161.959
Albumin	84	3.32	2.01	5.33	326.06	3.91	.625
Natrium	84	28	118	146	11356	135.50	22.686
Valid N	84						

Based on the numerical data characteristics, the median neutrophil-to-lymphocyte ratio (NLR) among all subjects was 4.47, with a minimum value of 0.53 and a maximum value of 93.00, indicating a very wide range of inter-patient variability. The median serum albumin level was 3.91 g/dL, with a range between 2.01 and 5.33 g/dL. Meanwhile, median serum sodium level was 135.50 mmol/L, with values ranging from 118 mmol/L to 146 mmol/L. All numerical variables were analyzed across a total of 84 subjects. The considerable variability observed in these three parameters reflects the heterogeneity of clinical conditions and biochemical status among patients with Guillain–Barré syndrome undergoing plasmapheresis.

Distribution of baseline variable characteristics

Table 3. NLR variable distribution

Category NLR Sample	N	(%)
Low	0	0
Normal	12	14.28
High	72	85.72
Total	84	100.00

The distribution of neutrophil-to-lymphocyte ratio (NLR) categories demonstrated varying levels of systemic inflammation among the study subjects. The majority of patients were classified within the high inflammatory category in 72 patients (85.72%). Only a small proportion of patients were within the normal NLR category in 12 patients (14.28%), and no patients were classified as having low NLR category. These findings indicate that most patients with Guillain–Barré syndrome in this study were experiencing elevated levels of systemic inflammation.

Table 4. Variable distribution of albumin

Categories Albumin Samples	N	(%)
Low	22	26.19
Normal	62	73.81
High	0	0.00
Total	84	100.00

Based on the distribution of albumin categories, the majority of study subjects had serum albumin levels within the normal range, comprising 62 patients (73.81%). Meanwhile, 22 patients (26.19%) were found to have hypoalbuminemia, and no patients exhibited albumin levels above the normal range.

These findings indicate that although most patients with Guillain–Barré syndrome undergoing plasmapheresis had normal albumin levels, a substantial proportion still experienced

hypoalbuminemia, which may potentially influence post-treatment clinical responses.

Table 5. Variable distribution of sodium

Category Sodium Sample	N	(%)
Low	33	39.26
Normal	50	59.52
High	1	1.19
Total	84	100.00

The distribution of serum sodium levels showed that the majority of patients had sodium concentrations within the normal range, accounting for 50 patients (59.52%). Hyponatremia was observed in 33 patients (39.26%), while only one patient (1.19%) exhibited serum sodium levels above the normal range. These findings indicate that disturbances in sodium balance, particularly hyponatremia, are relatively common among patients with Guillain–Barré syndrome undergoing plasmapheresis and may potentially influence the clinical course of the disease.

Neutrophil-to-Lymphocyte Ratio (NLR) as a predictive marker of clinical response in GBS after plasmapheresis

Table 6. Results of the Mann-Whitney Test of NLR Variables

Clinical Response / NLR	Good	Bad	Total	*p*	Prevalence Ratio
Normal/Low	17	15	32	.001	2.50
%	60.7	26.8	38.1		
High	11	41	52		
%	39.3	73.2	61.9		
Total	28	56	84		
%	100	100	100		

Based on the study results, of the 84 patients analyzed, 32 patients (38.1%) had normal or low NLR values, while 52 patients (61.9%) had high NLR values prior to therapy. A good clinical response was observed in 17 patients (53.1%) with normal or low NLR and in 11 patients (21.2%) with high NLR. The prevalence ratio for a good clinical response was significantly higher in the normal/low NLR group compared with the high NLR group (PR = 2.50; p = 0.001). These findings indicate that patients with normal or low NLR had a 2.5-fold higher prevalence of a good clinical response than those with high NLR, and this difference was statistically significant.

From a diagnostic performance perspective, normal or low NLR demonstrated a sensitivity of 60.7% and a specificity of 73.2% in predicting a good clinical response, indicating moderate sensitivity with relatively good specificity.

Serum Albumin as a Predictive Marker of Clinical Response in GBS After Plasmapheresis

Table 7. Sodium Variable Mann-Whitney test results

Clinical Response / Albumin	Good	Bad	Total	*p*	Prevalence Ratio
Normal/High	24	38	62	.003	2.13
%	85.7	67.9	73.8		
Low	4	18	22		
%	14.3	32.1	26.2		
Total	28	56	84		
%	100	100	100		

Based on the study findings, of the 84 patients analyzed, 62 patients (73.8%) had normal or high serum albumin levels, while 22 patients (26.2%) had low serum albumin levels prior to therapy. A good clinical response was observed in 24 patients (38.7%) with normal or high albumin levels and in 4 patients (18.2%) with low albumin levels.

The prevalence of a good clinical response was significantly higher in the normal/high albumin group compared with the low albumin group (PR = 2.13; p = 0.003). These results indicate that patients with normal or high albumin levels had approximately a 2.13-fold higher prevalence of a good clinical response than those with low albumin levels, and this difference was statistically significant.

In terms of diagnostic performance, normal or high serum albumin levels demonstrated a sensitivity of 85.7% and a specificity of 32.1% for predicting a good clinical response, indicating high sensitivity but low specificity.

Serum sodium as a predictive marker of clinical response in GBS after plasmapheresis

Table 8. Results of the Mann-Whitney sodium variable test

Clinical Response / Sodium	Good	Bad	Total	*p*	Prevalence Ratio
Normal/High	19	32	51	.970	1.37
%	67.9	57.1	60.7		
Low	9	24	33		
%	32.1	42.9	39.3		
Total	28	56	84		
%	100	100	100		

Of the total 84 patients, 51 patients (60.7%) had normal or high serum sodium levels, while 33 patients (39.3%) had low serum sodium levels prior to plasmapheresis. Clinical improvement was observed in 19 patients (37.3%) with normal or high sodium levels and in 9 patients (27.3%) with low sodium levels. The prevalence of clinical improvement in the normal/high sodium group was only slightly higher than that in the low sodium group (PR = 1.37; p = 0.970). These findings indicate that patients with normal or high sodium levels had a 1.37-fold higher prevalence of clinical improvement compared with those with low sodium levels; however, this association was not statistically significant. Sensitivity analysis showed that normal or high sodium levels had a sensitivity of 67.9% for detecting clinical improvement, with a specificity of 42.9%, indicating limited predictive performance.

Discussion

The Role of the Neutrophil-to-Lymphocyte Ratio (NLR) as a Predictive Marker of Clinical Response in GBS After Plasmapheresis

Based on the results of the normality tests, the neutrophil-to-lymphocyte ratio (NLR) data were not normally distributed. Therefore, Hypothesis I was tested using the non-parametric Mann–Whitney U test to evaluate differences in NLR values between post-plasmapheresis clinical outcome groups in patients with Guillain–Barré syndrome (GBS). The Mann–Whitney U analysis yielded a statistically significant result, with an Asymp. Sig. (2-tailed) value of 0.001. As this value was below the predefined significance threshold of $\alpha = 0.05$, a statistically significant difference in NLR values between the clinical outcome groups was confirmed. These findings indicate that NLR is significantly associated with clinical response in GBS patients following plasmapheresis. Accordingly, Hypothesis I was accepted, suggesting that NLR has potential utility as a predictive marker of clinical response after plasmapheresis in patients with GBS.

From a theoretical perspective, NLR serves as a marker of systemic inflammation that reflects the balance between innate immune activity mediated by neutrophils and adaptive immune responses mediated by lymphocytes. In GBS, disease pathogenesis is predominantly driven by aberrant immune responses triggered by molecular mimicry, in which antibodies and immune cells generated in response to preceding infections mistakenly target gangliosides in peripheral nerves. This process leads to immune activation, release of pro-inflammatory cytokines, immune cell infiltration, and macrophage activation, all of which contribute to demyelination and axonal damage.

Plasmapheresis exerts its therapeutic effect by removing circulating autoantibodies, immune complexes, cytokines, and inflammatory mediators, thereby modulating immune responses and improving clinical outcomes in patients with GBS. Patients with lower NLR values tend to exhibit a lower degree of systemic inflammation, which may allow for a more favorable response to plasmapheresis. In contrast, elevated NLR values reflect more severe inflammation and complex immune dysregulation, which may be associated with suboptimal clinical responses following therapy. Increased NLR in patients with GBS represents a pro-inflammatory systemic state characterized by neutrophilia due to acute inflammatory responses and lymphopenia as a manifestation of impaired adaptive immunity. This imbalance indicates a higher degree of inflammatory activity, which is clinically associated with greater disease severity and poorer outcomes.

Within the context of plasmapheresis, patients with higher NLR values are more likely to experience poorer clinical responses due to a greater inflammatory burden and heightened pathological

immune activity, thereby limiting clinical improvement despite the removal of pathogenic immune mediators. The findings of this study are consistent with those reported by Erdem et al. (2018), who demonstrated that elevated NLR correlates with increased disease severity and worse clinical outcomes in patients with GBS.

Taken together, both theoretical considerations and empirical evidence support the use of NLR as a simple, inexpensive, and readily available biomarker for predicting clinical response in patients with GBS following plasmapheresis. The results of this study further strengthen the theoretical framework and existing evidence indicating that NLR is a clinically meaningful inflammatory biomarker. Incorporating NLR into routine clinical assessment may assist clinicians in risk stratification, therapeutic planning, and monitoring treatment response in patients with Guillain–Barré syndrome more effectively.

Based on the results of the normality test, serum albumin levels were not normally distributed. Therefore, Hypothesis II was tested using the non-parametric Mann–Whitney U test to evaluate differences in albumin levels between post-plasmapheresis clinical outcome groups (good and poor outcomes) in patients with Guillain–Barré syndrome (GBS). The Mann–Whitney U analysis yielded an Asymp. Sig. (2-tailed) value of 0.03, which was below the predefined significance threshold of $\alpha = 0.05$. This finding indicates a statistically significant difference in serum albumin levels between the post-plasmapheresis clinical outcome groups. These results demonstrate that serum albumin levels are significantly associated with clinical response in GBS patients following plasmapheresis. Accordingly, Hypothesis II was accepted, suggesting that serum albumin has potential utility as a predictive marker of clinical response after plasmapheresis in patients with GBS.

From a theoretical perspective, albumin is the principal plasma protein with multiple essential biological functions, including maintenance of oncotic pressure and its role as a negative acute-phase protein that reflects both inflammatory status and nutritional condition. Albumin exerts antioxidant effects through sulfhydryl and thiol groups, enabling the binding and neutralization of reactive oxygen species, reactive nitrogen species, cytokines, and other inflammatory mediators. In addition, albumin contributes to vascular permeability regulation, acid–base balance, and microcirculatory stability, all of which are integral to systemic homeostasis—in acute systemic inflammatory conditions such as GBS, increased vascular permeability and enhanced pro-inflammatory cytokine activity lead to reduced serum albumin concentrations. Hypoalbuminemia in patients with GBS therefore reflects a higher degree of inflammation and impaired metabolic status, which may adversely affect clinical response to plasmapheresis.

From a pathophysiological standpoint, the autoimmune process in GBS induces sustained systemic inflammation. Under these conditions, albumin behaves as a negative acute-phase reactant, with reduced synthesis in parallel with increasing levels of inflammatory mediators such as C-Reactive Protein (CRP). This observation is consistent with existing theoretical frameworks suggesting that lower serum albumin levels correlate with greater disease severity and poorer prognosis in GBS. Conversely, higher albumin levels indicate more controlled inflammation and better physiological reserves, thereby facilitating a more favorable response to plasmapheresis.

Furthermore, albumin plays several critical biological roles, including maintenance of plasma oncotic pressure, antioxidant activity, binding of toxins and inflammatory mediators, and preservation of endothelial stability. These functions are particularly relevant in the context of plasmapheresis, which aims to remove circulating autoantibodies and inflammatory mediators. Patients with higher serum albumin levels before or during therapy are more likely to maintain adequate homeostasis, resulting in more optimal clinical responses following plasmapheresis. Higher albumin levels reflect a relatively stable systemic condition and controlled inflammatory burden, allowing clinical improvement to be achieved more readily.

These findings are consistent with previous studies, including that of Kim et al. (2017), which demonstrated that lower serum albumin levels were associated with greater clinical severity and poorer outcomes in patients with GBS. Similarly, Ethemoglu

et al. (2018) reported that hypoalbuminemia was associated with elevated inflammatory markers such as CRP, reinforcing the role of albumin as an indicator of inflammatory activity. In addition, a study conducted in Indonesia by Harahap et al. (2021) identified a proportion of GBS patients with hypoalbuminemia that was associated with more severe clinical conditions. Collectively, these findings support the use of serum albumin as a simple, readily available biomarker with clinical relevance in predicting response to plasmapheresis.

In summary, the results of this study strengthen both the theoretical foundation and empirical evidence supporting serum albumin as a simple, accessible biomarker with prognostic value in predicting clinical response among patients with GBS undergoing plasmapheresis. Incorporating serum albumin into clinical assessment may assist clinicians in risk stratification, evaluation of systemic condition, and comprehensive planning and monitoring of plasmapheresis therapy.

Based on the results of the normality test, serum sodium levels were also not normally distributed. Therefore, Hypothesis III was tested using the non-parametric Mann–Whitney U test to assess differences in sodium levels between post-plasmapheresis clinical outcome groups (good and poor outcomes) in patients with Guillain–Barré syndrome (GBS). The Mann–Whitney U analysis yielded an Asymp. Sig. (2-tailed) value of 0.97, which exceeded the predefined significance threshold of $\alpha = 0.05$. Thus, no statistically significant difference in serum sodium levels was observed between the post-plasmapheresis outcome groups. These findings indicate that serum sodium levels are not significantly associated with clinical response in patients with GBS following plasmapheresis. Accordingly, Hypothesis III was rejected, suggesting that serum sodium does not serve as a predictive marker of clinical response after plasmapheresis in GBS.

From a theoretical perspective, sodium is the principal extracellular electrolyte and plays a critical role in nerve impulse transmission, muscle contractility, and regulation of autonomic nervous system function. Disturbances in sodium balance, particularly hyponatremia, may result in neurological dysfunction, altered consciousness, and worsening of clinical status in patients with acute neurological disorders. Electrolyte imbalances—especially hyponatremia—are frequently observed in patients with GBS, particularly during the acute phase of the disease. One of the primary mechanisms underlying this condition is autonomic dysfunction and dysregulation of antidiuretic hormone secretion, leading to the syndrome of inappropriate antidiuretic hormone secretion (SIADH).

From a pathophysiological standpoint, sodium is essential for maintaining neuronal membrane stability, neuromuscular transmission, and action potential propagation. Reduced sodium levels may disrupt neuronal signaling, exacerbate muscle weakness, and delay neurological recovery. Patients with GBS who experience electrolyte disturbances—particularly hyponatremia—tend to have poorer overall clinical outcomes. In the context of plasmapheresis, adequate electrolyte balance is essential to support therapeutic effectiveness. Although plasmapheresis removes circulating autoantibodies and inflammatory mediators, optimal clinical response depends heavily on metabolic and physiological stability.

Serum sodium levels are clinically relevant during plasmapheresis because the procedure can influence fluid and electrolyte balance. Patients with lower sodium levels before or during plasmapheresis may have greater systemic instability, potentially limiting clinical improvement. Conversely, sodium levels closer to normal reflect better homeostatic balance and neurological stability, which may facilitate improved responses to therapy.

The findings of this study are consistent with those reported by Zhang et al. (2021), who observed that hyponatremia is relatively common in patients with GBS, particularly during the acute phase, but does not independently predict short-term clinical outcomes or response to immunomodulatory therapies, including plasmapheresis. Their study concluded that hyponatremia more accurately reflects the patient's systemic condition rather than the effectiveness of treatment.

Similar results were reported by Algahtani et al. (2023), who investigated electrolyte disturbances in

acute inflammatory neuropathies. Although hyponatremia was associated with disease severity, no significant association was found between baseline sodium levels and neurological recovery following specific treatments. These findings support the results of the present study, indicating that serum sodium is not a reliable predictive marker of clinical response after plasmapheresis.

Furthermore, Van den Berg et al. (2020) and Leonhard et al. (2020) emphasized that hyponatremia in GBS is frequently associated with autonomic dysfunction and SIADH, reflecting disease activity rather than factors directly influencing plasmapheresis efficacy. Thus, although hyponatremia may be more prevalent among patients with poorer clinical responses, the relationship is not causal.

Moreover, Rinaldi et al. (2022) highlighted that the primary determinants of clinical response in GBS after plasmapheresis include baseline weakness severity, respiratory involvement, patient age, and timing of therapy initiation. Laboratory parameters such as serum electrolytes are not considered major predictors of clinical outcomes, further supporting the conclusion that serum sodium alone cannot serve as a predictive marker of clinical response.

In conclusion, the findings of this study indicate that serum sodium levels are not significantly associated with clinical response in patients with GBS following plasmapheresis and therefore cannot be used as a standalone predictive marker. Nevertheless, monitoring serum sodium remains clinically important for detecting metabolic complications and autonomic disturbances that may exacerbate the patient's overall condition.

Implications for clinical practice

The findings of this study have important implications for the field of medicine, particularly in the clinical management of patients with Guillain–Barré syndrome (GBS) undergoing plasmapheresis therapy. The observed significant associations between the neutrophil-to-lymphocyte ratio (NLR) and serum albumin levels with post-plasmapheresis clinical response indicate that these parameters have potential utility as simple, accessible, and practical predictive markers in routine clinical practice.

As a marker of systemic inflammation, NLR holds particular relevance in neurology and internal medicine. Elevated NLR values reflect a higher degree of inflammation and immune activation in patients with GBS. Incorporating NLR into the initial clinical evaluation may assist clinicians in assessing inflammatory severity, predicting response to plasmapheresis, and performing early risk stratification. This, in turn, enables more informed clinical decisions regarding the intensity of monitoring and therapeutic management.

Serum albumin, as an indicator of inflammatory status and overall systemic condition, also has important clinical implications within a holistic approach to GBS management. Lower albumin levels were associated with poorer clinical responses following plasmapheresis, suggesting that albumin monitoring may help identify patients with reduced physiological reserves and a higher risk of unfavorable outcomes. These findings further emphasize the importance of optimizing metabolic and inflammatory conditions—including nutritional and inflammatory status—as integral components of comprehensive GBS management.

Overall, the integration of NLR and serum albumin measurements may enhance prognostic assessment and therapeutic monitoring in patients with GBS undergoing plasmapheresis. The use of these routine laboratory-based markers allows for early identification of high-risk patients, supports individualized treatment strategies, and improves the quality of clinical decision-making, which may ultimately contribute to better clinical outcomes in GBS.

Study limitations

This study has several limitations that should be considered when interpreting the findings. First, the observational study design precludes the establishment of a direct causal relationship between the Neutrophil-To-Lymphocyte Ratio (NLR), serum albumin levels, serum sodium levels, and post-plasmapheresis clinical response in patients with Guillain–Barré Syndrome (GBS). The observed associations may be influenced by other clinical factors that could not be fully controlled.

Second, laboratory data were collected at a single

time point, limiting the ability to evaluate longitudinal changes in NLR, albumin, and sodium levels before, during, and after plasmapheresis. Serial assessment of these biomarkers over time may provide more comprehensive prognostic insights into treatment response and disease progression in GBS.

In addition, potential confounding factors that may influence biomarker levels—such as concurrent infections, nutritional status, other inflammatory conditions, renal dysfunction, use of specific medications, and variability in supportive care—were not fully accounted for in this study. These factors may independently affect biomarker values and clinical outcomes.

Furthermore, the relatively small sample size and single-center study setting may limit the generalizability of the findings to broader GBS populations with different clinical and demographic characteristics. Therefore, caution is warranted when applying these results to other clinical settings.

Nevertheless, these limitations do not diminish the significance of the study's primary findings. Instead, they highlight the need for future research, particularly multicenter and longitudinal studies, to strengthen the evidence base regarding the role of NLR, albumin, and sodium as predictive markers of clinical response in patients with Guillain–Barré syndrome undergoing plasmapheresis.

Conclusion

Based on the results of the analyses and discussions, it can be concluded that the Neutrophil-To-Lymphocyte Ratio (NLR) and serum albumin levels play important roles as predictive markers of clinical response in patients with Guillain–Barré Syndrome (GBS) following plasmapheresis. Both parameters reflect inflammatory status, systemic condition, and homeostatic balance, which collectively contribute to the clinical effectiveness of plasmapheresis therapy.

The Neutrophil-To-Lymphocyte Ratio (NLR) serves as a predictive marker of clinical response in GBS after plasmapheresis. Elevated NLR values were associated with poorer clinical responses, indicating that higher degrees of systemic inflammation and immune activation may hinder clinical improvement in patients with GBS. These findings reinforce the role of NLR as an inflammatory indicator that not only reflects disease severity but is also relevant for predicting response to immunomodulatory therapy.

Serum albumin levels also function as a predictive marker of clinical response in GBS after plasmapheresis. Higher albumin levels were observed in patients who demonstrated better clinical responses, suggesting that a more stable systemic condition and lower inflammatory burden support therapeutic effectiveness. Albumin reflects not only nutritional status but also inflammatory activity and physiological reserve, thereby providing prognostic value in the clinical course of GBS. In contrast, serum sodium levels were not found to be predictive of clinical response in GBS following plasmapheresis. Nevertheless, lower sodium levels in patients with GBS have been identified as a poor prognostic factor for clinical outcomes in patients not undergoing plasmapheresis, as reported in previous studies by Van den Berg et al., Leonhard et al., Zhang et al., Rinaldi et al., and Algahtani et al. Overall, the findings of this study add to the growing body of evidence supporting the integrated use of inflammatory markers (NLR) and systemic condition markers (serum albumin). The simultaneous application of these parameters may facilitate a more targeted clinical approach, enable personalized therapy, and support more accurate clinical decision-making in the management of patients with Guillain–Barré syndrome.

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