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Original Article

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Role of neutrophil-to-lymphocyte ratio as a prognostic marker for Guillain-Barre syndrome: a Cross-sectional study from a tertiary care Hospital

Rabia Iqbal [a], Qamar-un-Nisa [b], Wajid Jawaid [c], Sumera Rafat Umer [c], Rabiya Khan [a], Samiksha [a]

[a] *Resident of Neurology, Dow University of Health Sciences, Dr Ruth K.M Pfau Civil Hospital, Karachi.*
[b] *Associate Professor, Department of Neurology, Dow University of Health Sciences, Dr Ruth K.M Pfau Civil Hospital, Karachi.*
[c] *Assistant Professor, Department of Neurology, Dow University of Health Sciences, Dr Ruth K.M Pfau Civil Hospital, Karachi.*
Correspondence: Wajidjawaid@outlook.com*

ABSTRACT

BACKGROUND & OBJECTIVE: Guillain-Barre Syndrome (GBS) is an acute polyradiculoneuropathy with variable course and prognosis in different patients. Neutrophil-to-lymphocyte ratio (NLR) has shown promise in predicting prognosis in Guillain-Barre syndrome. The objective of this study was to determine the role of NLR as a marker of prognosis for Guillain-Barre Syndrome.

METHODOLOGY: It was a prospective, cross-sectional study of GBS patients aged 15–70, conducted using non-probability consecutive sampling from 11th February to 10th September 2025 at the Neurology Department of Dr. Ruth K. M. Pfau Civil Hospital, Karachi. Patients were sub typed and treated with plasmapheresis. Baseline clinical data and inflammatory markers, including NLR, were collected. Treatment response was assessed using the Hughes Disability Score; poor prognosis was defined as an NLR >4.4 or non-response. SPSS version 26 was utilized for data analysis.

RESULTS: The study included 80 GBS patients (mean age 38.86 ± 15.8 years), mostly males (73.8%). The mean NLR was 7.18 ± 13.95. Higher NLR values (>4.4) were associated with poor clinical outcomes, as measured by the Hughes Disability Score. NLR showed a sensitivity of 48.48% and specificity of 80.85% and a diagnostic accuracy of 69.09% for predicting poor response. Acute motor and sensory axonal neuropathy (AMSAN) showed the highest prognostic accuracy among all studied variants of GBS.

CONCLUSION: NLR aids in early risk stratification and predicts clinical outcomes after therapeutic interventions. However, it should complement, rather than replace, comprehensive clinical evaluations.

KEYWORDS: Neutrophils, Lymphocytes, Guillain-Barré Syndrome, Plasmapheresis.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and is among the most widespread causes of acquired polyneuropathies across the global population. It is clinically defined as an acutely progressive motor weakness of the limbs, sometimes associated with sensory impairment, with a usually slow and variable recovery. It is believed that GBS affects 1 in 100,000 to 2 in 100,000 people per annum, with worldwide cases rising by 6.4 percent between 1990 and 2019 [1]. The most severe form of the disease is the axonal type, which is more common in Asia and Central and South America and has been reported at frequencies of 30% to 65%. Although even with the appropriate treatment, almost 20 percent of patients either succumb to the illness or are left with severe disabilities [2]. Jahan et al. found that the 6-month functional independence rate was poor in about 20% of patients at the time of diagnosis [3].

Although GBS is an immune-mediated disorder, the exact pathogenesis of the disease remains incompletely elucidated [4]. Furthermore, there is a shortage of biomarkers to diagnose, classify, and predict outcomes at an early stage [5]. Currently, no robust biomarkers are available that provide definitive guidance on poor prognosis. Thus, there is a growing need to identify prognostic markers that are readily accessible and reliable to enhance risk stratification in GBS and assist clinical decision-making [6].

The neutrophil-to-lymphocyte ratio (NLR) has been reported as a novel, inexpensive, and readily available marker of systemic inflammation and immune activation in

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some studies. As an indicator of the equilibrium between the innate (neutrophilic) and adaptive (lymphocytic) immune response, NLR can shed light on the inflammatory state that drives different diseases, such as autoimmune neurological conditions [7-9].

Many studies have previously explored the prognostic value of NLR in GBS. Hashim et al. studied the relationship between NLR and its prognostic value in relation to response to plasmapheresis, showing that a threshold of 4.4 was highly predictive of poor response, with a sensitivity of 81.5 percent and a specificity of 87.5 percent [10]. The focus of this study was to determine the prognostic significance of NLR in GBS and its subtypes, which has not been previously reported, according to our literature review. It also aims to generate local data to determine the predictive value of NLR for outcomes of plasmapheresis and ventilator requirements. NLR, as a universally available marker, could be beneficial for early risk stratification and clinical decision-making.

METHODOLOGY

This study was approved by the Institutional Review Board (IRB) committee of Dow University of Health Sciences on 10th February 2025 (IRB-3712/DUHS/Approval/2024/46). It was a prospective, cross-sectional investigation carried out in the Neurology Ward and the Neurology Intensive Care Unit of Dr. Ruth K.M. Pfau Civil Hospital, Karachi, a tertiary care teaching institution affiliated with Dow University of Health Sciences. The duration was seven months, from 11th February to 10th September 2025. GBS was defined as an acute, immune-mediated inflammatory polyradiculoneuropathy with progression to clinical nadir between 12 hours and 28 days.

Patients were enrolled using a non-probability, consecutive sampling methodology after obtaining informed written consent. The participants included were those with nerve conduction studies that validated one of the classical subtypes, that are acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), or acute motor and sensory axonal neuropathy (AMSAN), and were administered five alternate-day sessions of plasmapheresis

The inclusion criteria were patients of either gender, aged 15 to 70 years, with a diagnosis of GBS in accordance with the Brighton diagnostic criteria [11]. In addition to non-consenting patients, the exclusion criteria encompassed conditions that have a tendency to alter the NLR ratio, such as pregnancy, systemic infections (as indicated by an elevated total leukocyte count), serum creatinine levels exceeding 2 mg/dL, malignancies, ischemic heart diseases, congestive heart failure, and atypical variants, including Miller Fisher syndrome.

Demographic variables (age, sex, residence, and socioeconomic status) and clinical data (comorbidities, smoking, alcohol consumption, and symptom duration) were recorded. Socioeconomic status was categorized using household-income-based fixed cutoffs (with <35,000 PKR categorized as low socioeconomic status, 35,000 -80,000 PKR as middle, and > 80,000 PKR as high socioeconomic status). On admission, baseline neurological disability was assessed using the Modified Hughes Disability Scale [12]. According to this scale, the severity of disability ranges from score 0 to 6. A score of 0 indicates a healthy individual, whereas a score of 6 indicates the patient is dead. Score 1 indicates mild symptoms with no disability, including the ability to walk 5 meters without assistance, but the inability to run is labeled as score 2.

Walking five meters with assistance and wheelchair dependence/bed-bound state, each labeled as score 3 and 4, respectively, and grade 5 is labeled when the patient needed assisted ventilation. Blood samples were obtained within six hours of admission to evaluate complete blood counts. Those included absolute neutrophil and lymphocyte counts for calculation of the neutrophil-to-lymphocyte ratio (NLR), along with C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and platelet counts. Patients were then given 5 plasmapheresis sessions as part of their regular treatment protocol. Another treatment modality for GBS, intravenous immunoglobulin (IVIG), was not administered to any patient due to its unavailability in our hospital setting.

Clinical response to treatment was evaluated one day after completion of plasmapheresis. Responders were defined as those who demonstrated a reduction of at least one point in the Hughes score from their baseline pre-treatment score, while non-responders had either unchanged or worsened scores. Poor prognosis was operationally defined as either an NLR >4.4 (hereafter, positive NLR) or a clinical non-response to treatment (non-responder patient).

Statistical evaluations were conducted utilizing SPSS version 26. Continuous variables, including age and symptom duration, were reported as means ± standard deviation (SD), whereas categorical variables were reported as frequencies and percentages. The capacity of the NLR to forecast inadequate therapeutic response was examined using sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy, employing 2×2 contingency tables.

RESULTS

A total of 80 patients were enrolled in the study. The mean age of the study population was 38.86 ± 15.80 (95% CI 35.34 - 42.37). The demographic and clinical characteristics of the study population are presented in Table- I and Table -II respectively.

Table- I: Demographic Profile of Study Participants (n=80).

Demographic Characteristics	Categories	n(%)
Gender	Male	59 (73.8)
	Female	21 (26.2)
Residential Status	Urban	62 (77.5)
	Rural	18 (22.5)
Socioeconomic Status	Low	36 (45.0)
	Middle	40 (50.0)
	High	4 (5.0)

Table- II: Clinical characteristics of Study Participants (n=80).

Clinical Characteristics	Mean ± SD	95% Confidence Interval
Duration of Symptoms in Days	8.53 ± 4.21	7.59-9.47
TLC	9.26 ± 1.84	8.85- 9.68
Neutrophils	69.26 ± 12.08	66.57-71.95
Lymphocytes	20.20 ± 9.63	18.06-22.35
NLR	7.18 ± 13.95	4.07 -10.28
Platelets	284.37± 121.49	257.33 - 311.41
PLR	35.42 ± 99.16	13.35 - 57.49
CRP	37.57 ± 75.02	20.88 - 54.27
ESR	29.75 ± 16.85	25.99 - 33.50
Hughes Disability Score at Presentation	3.87 ± 0.70	3.71 -4.03
Hughes Disability Score after Treatment	3.50 ± 0.95	3.28 - 3.71
Clinical characteristics	**Type**	**n (%)**
Co-morbid Conditions	Hypertension	11 (13.8)
	Diabetes	8 (10.0)
	Others	1 (1.3)
	None	60 (75.0)
GBS Variants	AIDP	28 (35.0)
	AMAN	21 (26.2)
	AMSAN	31 (38.8)
Need for Mechanical Ventilation	Yes	13 (16.3)
	No	67 (83.7)
Precedent Illness	Respiratory Tract Infection	7 (8.8)
	Gastrointestinal Illness	6 (7.5)
	None Identified	39 (48.7)
	Others	28 (35.0)
Precedent of Dysautonomia	Yes	9 (11.3)
	No	71 (88.7)
History of Smoking	Yes	11 (13.8)
	No	69 (86.2)

*** Z-based and Binomial (Wilson/Exact) methods were used for 95% CI calculation .**
**** Abbreviations: TLC – total leukocyte count, NLR- neutrophil-lymphocyte ratio, PLR- platelet-lymphocyte ratio, CRP- C-reactive protein, ESR- erythrocyte sedimentation rate, GBS- Guillain Barre Syndrome, AIDP- acute inflammatory demyelinating polyradiculoneuropathy, AMAN- Acute motor axonal neuropathy, AMSAN- acute motor sensory polyradiculoneuropathy.**

Table-III: Comparison of NLR & poor clinical response in Guillain-Barre Syndrome (n=80).

Neutrophils to Lymphocyte Ratio	Poor Clinical Response	
	Positive	Negative
Positive	16 (48.5)	9 (19.1)
Negative	17 (51.5)	38 (80.9)

Table-III shows the association between neutrophil-to-lymphocyte ratio (NLR) and clinical response in patients with Guillain-Barré Syndrome (n=80). This demonstrates that a positive NLR is associated with worse prognosis.

Table- IV presents the diagnostic accuracy of the neutrophil-to-lymphocyte ratio (NLR) as a prognostic marker in patients with Guillain-Barré Syndrome (n=80). A positive NLR shows moderate sensitivity and high specificity along with a high positive and negative predictive value.

Table-IV: Diagnostic accuracy of NLR as a prognostic marker for GBS (n=80).

Diagnostic Variables		95% Confidence Interval
Sensitivity	48.48%	31.43 - 65.54
Specificity	80.85%	69.60 - 92.10
Positive Predictive Value	64.00%	45.18 - 82.82
Negative Predictive Value	65.36%	58.39 - 72.33
Diagnostic Accuracy	69.09%	56.88 - 81.30
Positive Likelihood Ratio	2.53	N/A
Negative Likelihood Ratio	0.64	N/A

GBS* Guillain-Barre Syndrome.

Table-V summarizes the diagnostic accuracy of the neutrophil-to-lymphocyte ratio (NLR) in differentiating variants of Guillain-Barré Syndrome.

Table-V: Diagnostic accuracy of NLR in differentiating GBS variants.

GBS Variants	Neutrophils to Lymphocyte Ratio (NLR)				
	Sensitivity	Specificity	PPV	NPV	DA
AIDP (n=28)	21.43%	71.43%	42.86%	47.62%	46.43%
AMAN (n=21)	44.44%	58.33%	44.44%	58.33%	52.38%
AMSAN (n=31)	36.36%	75.00%	44.44%	68.18%	61.29%

PPV* Positive Predictive Value; NPV* Negative Predictive Value; DA* Diagnostic Accuracy.

DISCUSSION

The NLR is a ratio that indicates inflammation and an imbalance in the immune system [7]. The normal range of NLR in adults is 1-2, with values greater than 3.0 considered abnormal [7]. A number of published studies about GBS have shown that NLR was higher among patients with GBS than in healthy controls, indicating that it may be used both to diagnose and to predict the disease. However, results have been mixed, with some studies not identifying an association [13-15]. Furthermore, many studies have demonstrated that the NLR is positively correlated with disability severity in GBS, as reflected by established clinical assessment tools such as the Hughes Disability Scale (HDS) and the Medical Research Council (MRC) sum score [16,17]. An example is a study by Kim et al., which reported that patients with GBS who achieved complete recovery without any residual disability exhibited significantly lower NLR levels than those who continued to experience residual deficits beyond 6 months [18].

Our study has evaluated the prognostic role of the NLR in patients diagnosed with GBS, employing clinical criteria based on the Brighton classification and electrophysiological confirmation of classical GBS subtypes [11]. Among the 80 patients who participated in the study, the AMSAN subtype was the most prevalent, followed by AIDP and AMAN, reflecting patterns previously observed in Asian and South American populations where axonal forms of GBS are more common [19, 20].

The results of our study demonstrated that patients with poor clinical response, as indicated by a higher Hughes disability score, had higher NLR values, suggesting its potential usefulness as a prognostic index. The specificity of NLR in our study was 80.85%, which is quite high, while its sensitivity was modest at 48.48%. This contrasts with findings from Hashim et al., who reported significantly higher sensitivity (81.5%) and specificity (87.5%) for an NLR cutoff of 4.4 in predicting poor response to plasmapheresis [8]. Such variation may result from differences in study design, sample size, treatment protocols, or underlying patient characteristics. Cabanillas Lazo et al. concluded in their systematic review and meta-analysis that elevated NLR levels were consistently associated with adverse GBS outcomes, further supporting our observations, although results across studies were not entirely homogeneous [21].

Bano S et al. showed that higher NLR and increased CSF protein levels correlated with worse prognosis, while Jawaid W et al. demonstrated that increased CSF protein levels correlated with the severity of GBS [22,23]. These studies therefore also point to the utility of inflammatory markers, such as NLR and CSF albumin, in predicting prognosis in GBS. Moreover, our mean NLR of 7.32 is much higher than those reported in earlier studies, suggesting either delayed clinical presentation or a higher inflammatory burden in our patient cohort [24].

Our study showed a moderate diagnostic accuracy of NLR (69.09%), with a positive likelihood ratio of 2.53. These findings do not establish NLR as an independent diagnostic test; however, they emphasize its value as an adjunctive tool for early prognostication, especially when used alongside other clinical scales, such as the Hughes Disability Scale. The strengths of our study include its large sample size, prospective study design, and focus on common and electro-physiologically confirmed subtypes of GBS. On the other hand, the limitations of our study included that it was conducted in a single center, that patients were not followed up for long-term prognosis, and that uncontrolled confounders, such as concurrent infections or medications, may have influenced the NLR.

CONCLUSION

This study supports the role of NLR as a potential prognostic marker in GBS patients. It could be used in GBS patients to predict clinical outcomes after therapeutic interventions. This may help physicians and neurologists triage patients based on their risk, thereby improving decision-making. However, it is very important that it be used only to complement, rather than replace, other useful clinical assessments. Additional research endeavors are recommended to validate its applicability across diverse subtypes of GBS.

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Authors Contributions:

Rabia Iqbal: Substantial contributions to the conception and design of the work.
Qamar-un-Nisa: The acquisition and analysis of data for the work.
Wajid Jawaid: Interpretation of data for the work.
Sumera Rafat Umer: Drafting the work.
Rabiya Khan: Reviewing it critically for important intellectual content.
Samiksha: Final approval of the version to be published.

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