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Correlation between Systemic Inflammatory Response Index (SIRI) and disease severity in patients with Guillain-Barré syndrome

[1]Serdar Özdemir, [1]İbrahim Altunok, [1]Hilal Sümeyye Körelçiner, [1]Yunus Diler, [2]Mehmet Arslan

[1]Ümraniye Training and Research Hospital, Istanbul, Türkiye; [2]Balıklı Göl State Hospital, Şanlıurfa, Türkiye

Abstract

Background: Guillain-Barré syndrome (GBS) is a peripheral neuropathy with variable clinical severity, and in severe cases, it can lead to significant neurological dysfunction and respiratory failure. In this study, we evaluated the relationship between the Systemic Inflammatory Response Index (SIRI), derived from a complete blood count, and neurological severity in patients with GBS. *Methods:* The data of 40 consecutive patients diagnosed with definitive GBS between January 1, 2021, and March 1, 2023, at a tertiary care center were retrospectively analyzed. Demographic data, vital signs at presentation, and laboratory results were recorded. SIRI (neutrophils × monocytes / lymphocytes) was calculated from initial blood counts. Disease severity was defined using the Hughes Functional Grading Scale (HFGS) at the worst disease stage, with a score of ≥4 considered a "severe" clinical presentation. The performance of SIRI in predicting severe GBS was evaluated using receiver operating characteristic (ROC) curve analysis, the Youden index, and logistic regression. *Results:* The median age of the patients was 58 years (interquartile range: 42–73), and 52% were male. The median SIRI was significantly higher in the severe group (n = 27, 68%) compared to the mild group (n = 13, 32%) [1.69 (1.33–2.85) vs. 1.39 (0.90–2.02); p = 0.013]. A moderate positive correlation was found between SIRI and the worst HFGS score (r = 0.554, p < 0.001). In ROC analysis, at a cut-off value of ≥1.43, SIRI predicted severe GBS with 77.4% sensitivity and 77.8% specificity (area under the curve = 0.771, Youden index = 0.552). In multivariate logistic regression, SIRI ≥ 1.43 (odds ratio = 5.8, 95% confidence interval: 2.0–17.0), initial HFGS score ≥ 3, and age ≥ 60 years remained independent predictors of a severe clinical course.

Conclusion: The SIRI value at presentation is an independent and easily applicable biomarker for predicting neurological severity in GBS. The SIRI threshold of ≥1.43 may assist in identifying patients requiring early intensive care monitoring or aggressive treatment; however, these findings should be validated in multicenter, prospective studies.

Keywords: Guillain-Barré syndrome, systemic inflammation, prognosis, neurological disability, biomarker, complete blood count, ROC analysis, peripheral neuropathy

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy of the peripheral nervous system, frequently emerging after viral infections and characterized by progressive muscle weakness. The etiopathogenesis of the disease is based on an autoimmune process in which the immune system erroneously targets peripheral nerves. The clinical course is variable, remaining limited to mild neurological symptoms in some cases while leading to severe manifestations—most notably respiratory failure, which reflects the overall disease severity—and the need for intensive care in others.[1-4] This variability in disease progression complicates prognostication and increases the need for objective markers in clinical decision-making. Given the role of inflammation in the pathophysiology of GBS, blood-based indices

Address correspondence to: Serdar Özdemir, Department of Emergency Medicine, University of Health Sciences Ümraniye Training and Research Hospital, Istanbul, Turkey. Tel: +90(505) 2673292, email: dr.serdar55@hotmail.com

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reflecting systemic inflammatory status are gaining attention as potential prognostic markers.[5-8]

Although clinical scoring systems such as the Hughes Functional Grading Scale (HFGS) are used to predict disease progression, the relationship between the severity of the systemic inflammatory response and the extent of neurological involvement remains incompletely understood (9,10). In recent years, parameters derived from complete blood counts, such as the Systemic Inflammatory Response Index (SIRI), have been proposed as prognostic markers in inflammatory processes. This index quantifies the systemic effects of the immune response by reflecting the dynamic interaction of inflammation modulators such as neutrophils, lymphocytes, and monocytes.[11,12] Our primary hypothesis is that an elevated SIRI value correlates with severe neurological dysfunction and can serve as a prognostic indicator. This study aimed to assess the correlation of various combined inflammatory indices measured at the time of presentation to the emergency department with the worst HFGS score in patients followed up with a diagnosis of GBS, thus exploring the potential of these indices to reflect disease severity.

METHODS

This study was conducted with a retrospective, descriptive, and analytical design to investigate the relationship between hematological indices reflecting systemic inflammation and disease severity in patients diagnosed with GBS. The study included 40 patients who were hospitalized in the neurology clinic of a tertiary care training and research hospital between January 1, 2021, and March 1, 2023, and had a confirmed diagnosis of GBS. The diagnosis was established by neurology specialists in accordance with the internationally recognized Brighton criteria.

Demographic data (age and sex), presentation findings (systolic and diastolic blood pressure, pulse rate, oxygen saturation, and body temperature), duration of symptoms, GBS subtype, and clinical features of the disease (cranial nerve involvement, respiratory failure, areflexia/hyporeflexia, sensory loss, and autonomic dysfunction) were recorded. Treatments administered (intravenous immunoglobulin and steroids) were also evaluated.

The functional status of the patients was assessed using HFGS at the time of admission and throughout the clinical course. Disease severity was categorized as *severe* when the worst HFGS score during hospitalization was ≥ 4, and as *non-severe* when the score was < 4. Laboratory parameters were collected from complete blood count and biochemical tests performed at presentation. From the blood samples, the following combined inflammatory indices were calculated using neutrophil, lymphocyte, monocyte, and platelet counts: All data were retrospectively obtained from the electronic patient record system. The study was conducted with the approval of the relevant local ethics committee and in accordance with the tenets of the Declaration of Helsinki. As patient data were analyzed retrospectively, the requirement for informed consent was waived by the ethics committee. To ensure patient confidentiality, all data were anonymized and made accessible only to the researchers.

Statistical analysis

All statistical analyses were performed using Jamovi version 2.3.28 software (The jamovi project). Continuous variables were reported as median (interquartile range [IQR]) due to the non-normal distribution. Categorical variables were presented as frequencies (percentages). The Mann-Whitney U test was used for the comparison of continuous variables between groups. The chi-square test was applied for the comparison of categorical variables. The relationship between the HFGS score and SIRI was evaluated using the Pearson correlation coefficient. The strength of the correlation coefficients was interpreted according to Cohen's classification, where values between 0.3 and 0.5 were considered moderate, and values above 0.5 were considered moderately strong (13). The assumption of normality was assessed with the Shapiro-Wilk test. The strength and direction of the correlation were reported with the r value. Statistical significance was assessed at the $p < 0.05$ level. Receiver operating characteristic (ROC) curve analysis was performed to determine the prognostic value of SIRI. The optimal cut-off point was determined using the Youden index. Test performance was reported with sensitivity, specificity, positive predictive value, negative predictive value, and area under the curve (AUC). Correlation analysis results were visualized using a scatter plot, and ROC analysis results were presented using a ROC curve.

RESULTS

Among the 40 patients diagnosed with GBS included in the study, the median age was 58 years (IQR: 42–73), with 52% being male (n = 21) and 48% female (n = 19). Regarding the

distribution of electrophysiological subtypes, acute motor and sensory axonal neuropathy was identified in 45% (n = 18), acute inflammatory demyelinating polyradiculoneuropathy in 28% (n = 11), acute motor axonal neuropathy in 15% (n = 6), and Miller Fisher syndrome in 12% (n = 5). The distribution of the HFGS score at presentation was as follows: 0 in 2.5% (n = 1) of the patients, 1 in 7.5% (n = 3), 2 in 22% (n = 9), 3 in 32% (n = 13), and 4 in 35% (n = 14). When evaluated at the disease's most severe stage (nadir phase), the HFGS score was 1 in 5% (n = 2) of the patients, 2 in 18% (n = 7), 3 in 18% (n = 7), 4 in 50% (n = 20), 5 in 7.5% (n = 3), and 6

Table 1: Descriptive characteristics of the clinical cohort

Characteristic	n = 40
Age (years)	58 (42–73)
Sex	
Male	21 (52%)
Female	19 (48%)
Systolic blood pressure (mmHg)	130 (120–148)
Diastolic blood pressure (mmHg)	78 (70–85)
Heart rate (beats per minute)	82 (74–90)
Fever (°C)	36.3 (36.1–36.6)
Subgroup	
AMSAN	18 (45%)
AIDP	11 (28%)
Miller Fisher syndrome	5 (12%)
AMAN	6 (15%)
Hughes Functional Grading Scale score (initial)	
0	1 (2.5%)
1	3 (7.5%)
2	9 (22%)
3	13 (32%)
4	14 (35%)
Hughes Functional Grading Scale score (worst)	
1	2 (5%)
2	7 (18%)
3	7 (18%)
4	20 (50%)
5	3 (7.5%)
6	1 (2.5%)
Cranial nerve involvement	12 (30%)
Respiratory failure	4 (10%)
Areflexia/hyporeflexia	39 (98%)
Sensory loss	31 (78%)
Autonomic dysfunction	1 (2.5%)
Treatment	
Intravenous immunoglobulin (administered)	39 (98%)
Steroid therapy (administered)	6 (15%)
Laboratory findings	
Cerebrospinal fluid protein (mg/dL)	260 (89–525)
White blood cell count ($\times 10^3/\mu L$)	8.62 (6.97–10.93)
Neutrophil count ($\times 10^3/\mu L$)	5.80 (4.56–6.96)
Lymphocyte count ($\times 10^3/\mu L$)	1.69 (1.26–2.06)
Monocyte count ($\times 10^3/\mu L$)	0.54 (0.36–0.73)

Table 1: (continued)

Hemoglobin (g/dL)	13.50 (12.23–14.65)
Hematocrit (%)	41.2 (37.1–44)
Mean corpuscular volume (fL)	86.6 (83.9–90.4)
Red cell distribution width (%)	14.40 (13.60–15.53)
Platelet count ($\times 10^3/\mu L$)	236 (195–284)
Glucose (mg/dL)	100 (95–118)
C-reactive protein (mg/L)	8 (4–18)
Creatinine (mg/dL)	0.76 (0.68–0.88)
Alanine aminotransferase (U/L)	24 (16–42)
Aspartate aminotransferase (U/L)	22 (19–33)
Alkaline phosphatase (U/L)	81 (62–98)
Gamma-glutamyl transferase (U/L)	25 (17–42)
Lactate dehydrogenase (U/L)	228 (172–305)
Amylase (U/L)	47 (39–67)
Lipase (U/L)	28 (20–37)
Total bilirubin (mg/dL)	0.60 (0.46–0.79)
Direct bilirubin (mg/dL)	0.20 (0.17–0.31)
Neutrophil-to-lymphocyte ratio	3.12 (2.64–5.49)
Systemic Immune-Inflammation Index	893 (509–1,264)
Systemic Inflammation Response Index (SIRI)	1.69 (1.11–2.74)
Systemic Inflammation Aggregate Index	398 (268–629)
Monocyte-to-lymphocyte ratio	0.31 (0.23–0.55)

AMSAN: acute motor and sensory axonal neuropathy, AIDP: acute inflammatory demyelinating polyradiculoneuropathy, AMAN: acute motor axonal neuropathy

in 2.5% (n = 1). Other descriptive characteristics are presented in Table 1.

The patients with GBS included in the study were categorized into two groups based on clinical severity: mild (n = 13, 32%) and severe (n = 27, 68%). The median age in the severe group was 65 years (IQR: 50–77), whereas it was 52 years (IQR: 38–61) in the mild group; however, this difference was not statistically significant (p = 0.089). No statistically significant difference was found between the groups regarding sex distribution (p = 0.43). In the mild group, 62% (n = 8) of the patients were male and 38% (n = 5) were female, while in the severe group, 48% (n = 13) were male and 52% (n = 14) were female. Upon comparing SIRI levels, significantly higher values were observed in the severe GBS group (median: 1.69, IQR: 1.33–2.85), and this difference was statistically significant (p = 0.013). The median SIRI in the mild group was 1.39 (IQR: 0.90–2.02). Group comparisons for other parameters are presented in Table 2.

Spearman's correlation analysis showed a moderately strong positive relationship between the SIRI and the worst HFGS score (ρ = 0.554, 95% CI 0.293–0.738, p < 0.001), consistent with a moderately strong effect size according to Cohen's classification (Figure 1).

In the ROC analysis conducted to predict prognosis in patients with GBS using SIRI, the optimal cut-off value was determined as 1.43. At this cut-off point, SIRI demonstrated a sensitivity of 77.42%, a specificity of 77.78%, a positive predictive value of 92.31%, and a negative predictive value of 50%. The Youden index was calculated as 0.552, and the AUC was 0.771 (Figure 2).

DISCUSSION

In this study, we investigated the relationship between SIRI and neurological dysfunction in GBS and obtained three principal observations: First, SIRI levels were significantly higher in the severe GBS group. Second, we identified a moderately strong positive correlation between SIRI and the worst HFGS score (r = 0.554). Lastly, ROC analysis revealed that at a cut-off value of ≥1.43, SIRI was able to distinguish severe cases with 77% sensitivity and 78% specificity (AUC = 0.77). These findings suggest that SIRI may be a practical biomarker capable of predicting clinical severity in GBS.

GBS is characterized by immune-mediated axonal or demyelinating injury involving

Table 2: Comparison of groups according to disease severity

Characteristic	Non-severe group n = 13 (32%)	Severe group n = 27 (68%)	p-value
Age (years)	52 (38–61)	65 (50–77)	0.089
Sex			0.43
Male	8 (62%)	13 (48%)	
Female	5 (38%)	14 (52%)	
Systolic blood pressure (mmHg)	130 (120–140)	130 (116–148)	0.795
Diastolic blood pressure (mmHg)	80 (71–80)	74 (70–86)	0.635
Heart rate (beats per minute)	85 (72–86)	80 (75–93)	0.734
Fever (°C)	36.2 (36–36.5)	36.3 (36.1–36.6)	0.987
Subgroup			0.14
AMSAN	5 (38%)	13 (48%)	
AIDP	3 (23%)	8 (30%)	
Miller Fisher syndrome	4 (31%)	1 (3.7%)	
AMAN	1 (7.7%)	5 (19%)	
White blood cell count ($\times 10^3/\mu L$)	9.11 (8.35–10.39)	8.47 (6.94–11.05)	0.849
Neutrophil count ($\times 10^3/\mu L$)	5.83 (4.75–7.20)	5.56 (4.09–6.84)	0.507
Lymphocyte count ($\times 10^3/\mu L$)	2.04 (1.31–3.11)	1.49 (1.25–1.98)	0.060
Monocyte count ($\times 10^3/\mu L$)	0.49 (0.36–0.8)	0.55 (0.37–0.7)	0.496
Hemoglobin (g/dL)	14.3 (12.6–15.2)	13.3 (11.55–14.1)	0.020
Hematocrit (%)	41.6 (38.5–45.4)	40.9 (36.5–43.7)	0.089
Mean corpuscular volume (fL)	86.3 (83.9–87.6)	86.8 (85.7–92)	0.935
Red cell distribution width (%)	14.1 (13.6–15.2)	14.7 (13.6–16.05)	0.120
Platelet count ($\times 10^3/\mu L$)	253 (208–310)	233 (194–274)	0.808
Glucose (mg/dL)	100 (96–105)	104 (94–128)	0.200
C-reactive protein (mg/L)	7 (1–13)	9 (4–26)	0.178
Creatinine (mg/dL)	0.74 (0.66–0.84)	0.77 (0.70–0.91)	0.582
Alanine aminotransferase (U/L)	33 (23–36)	20 (13–45)	0.127
Aspartate aminotransferase (U/L)	27 (22–33)	21 (17–34)	0.347
Alkaline phosphatase (U/L)	79 (61–90)	84 (64–99)	0.149
Gamma-glutamyl transferase (U/L)	26 (20–30)	25 (15–53)	0.967
Lactate dehydrogenase (U/L)	188 (162–227)	243 (180–331)	0.087
Amylase (U/L)	46 (39–69)	48 (39–64)	0.750
Lipase (U/L)	28 (20–36)	28 (20–41)	0.909
Total bilirubin (mg/dL)	0.60 (0.50–0.68)	0.60 (0.46–0.82)	0.453
Direct bilirubin (mg/dL)	0.21 (0.18–0.31)	0.20 (0.17–0.31)	0.515
Neutrophil-to-lymphocyte ratio	2.93 (1.60–3.57)	3.53 (2.78–5.53)	0.30
Systemic Immune-Inflammation Index	893 (307–1,243)	914 (548–1,271)	0.059
Systemic Inflammation Response Index	1.39 (0.90–2.02)	1.69 (1.33–2.85)	0.013
Systemic inflammation aggregate index	332 (176–627)	398 (300–632)	0.086
Monocyte-to-lymphocyte ratio	0.26 (0.22–0.48)	0.34 (0.24–0.55)	0.051

AMSAN: acute motor and sensory axonal neuropathy, AIDP: acute inflammatory demyelinating polyradiculoneuropathy, AMAN: acute motor axonal neuropathy

widespread inflammation and endothelial activation in peripheral nerves. Neutrophils and monocytes release pro-inflammatory cytokines (e.g., interleukin-1 beta and tumor necrosis factor alpha) and reactive oxygen species that damage the myelin sheath, while a relative decrease in lymphocytes, representative of adaptive immunity, shifts the balance toward a pro-inflammatory state.[3,14] The SIRI formula (neutrophils × monocytes / lymphocytes) precisely quantifies this imbalance. As the ratio increases, systemic inflammation is expected to intensify and,

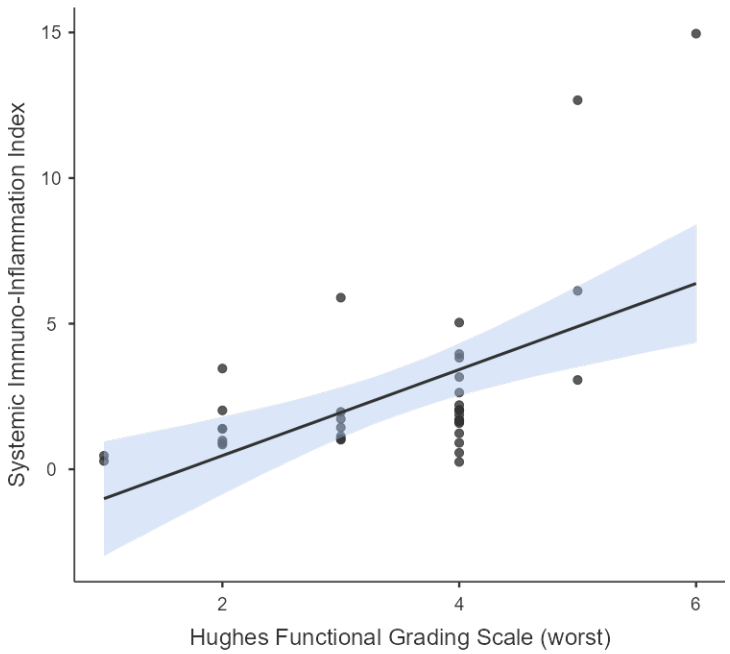

Figure 1. Scatter plot demonstrating the relationship between the worst Hughes Functional Grading Scale (HFGS) and Systemic Inflammatory Response Index (SIRI) at admission. The correlation was assessed using Spearman's rank correlation ($\rho = 0.554$, 95% CI 0.293–0.738, $p < 0.001$; $n = 40$).

indirectly, peripheral nerve damage increases. The correlation observed between SIRI and the worst HFGS score in our study supports the hypothesis that systemic inflammatory burden is reflected in peripheral neurological dysfunction.

There are studies in literature that have explored the role of combined hematologic inflammatory indices. In a study by Huang *et al*., 117 patients

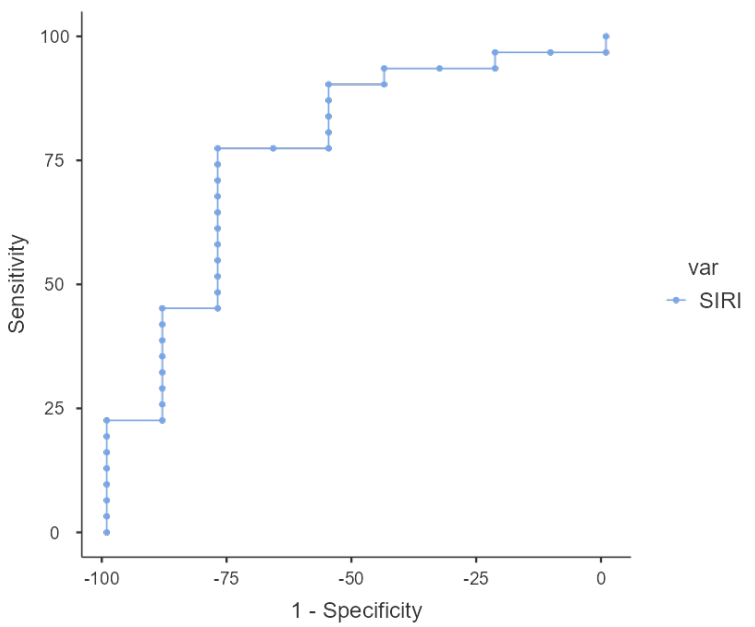

Figure 2. Receiver operating characteristic (ROC) curve showing the performance of the Systemic Inflammatory Response Index (SIRI) in predicting severe Guillain-Barré Syndrome (HFGS ≥ 4). Area under the curve (AUC) = 0.771; optimal cut-off value = 1.43 (sensitivity 77.4%, specificity 77.8%).

with GBS were compared to 217 healthy controls, and the neutrophil-to-lymphocyte ratio (NLR) and monocyte-to-lymphocyte ratio (MLR) were found to be significantly higher in the GBS group. According to ROC analysis, the cut-off value of NLR was 2.30 for predicting diagnosis and 3.05 for predicting disease severity, while the cut-off values of MLR were 0.235 for both. These indices were reported to be higher in severe cases than in mild ones.[15] A systematic review and meta-analysis combining data from six studies demonstrated that the mean NLR in patients with GBS was significantly higher than in healthy controls (mean difference = 1.76).

The sensitivity and specificity of NLR for predicting disability based on an HFGS score of ≥3 were approximately 67–82% and 67–88%, respectively, and for respiratory failure, these values were 86% and 68%. In light of these meta-analytic findings, the authors proposed NLR as a useful biomarker for assessing both disease activity and poor prognosis in GBS.[16] A retrospective study analyzing data from 136 patients suggested that elevated NLR was predictive of severe functional disability, marked muscle weakness, and poor short-term prognosis. The association between poor prognosis and NLR was particularly pronounced in patients aged ≥60 years, male sex, or those showing rapid symptom progression. The authors recommended NLR as an independent and practical biomarker in the diagnosis, severity assessment, and prognosis of GBS.[17] In another retrospective study published in 2022, NLR, MLR, and platelet-to-lymphocyte ratio were found to be significantly higher in 47 patients with GBS compared to healthy controls. These indices were even higher in the subgroup with severe disability (HFGS score ≥ 3). Especially in the acute inflammatory demyelinating polyradiculoneuropathy subtype, these three indices showed a strong positive correlation with the HFGS score, indicating that they may serve as useful biomarkers for monitoring disease activity and severity in GBS.[18]

In a study involving 369 cases, the Systemic Immune-Inflammation Index, derived from neutrophil, platelet, and lymphocyte counts, was found to independently predict respiratory failure in patients with GBS at a cut-off value of $\geq 863 \times 10^9$/L, and the AUC value of this index was reported to be 0.75.[19] These findings consistently support the capacity of hematologic inflammatory indices to reflect the clinical profile in GBS. A multiparametric prognostic model that integrates such hematological markers may facilitate more precise early risk stratification and treatment strategies.

In line with our findings, Shen *et al*. recently reported an S-shaped relationship between SIRI values and the probability of respiratory failure in GBS patients, indicating that systemic inflammation intensifies with increasing disease severity and stabilizes beyond a certain threshold.[20] Our results are consistent with theirs, showing that higher baseline SIRI levels were significantly associated with more severe neurological impairment as defined by the worst HFGS score. While Shen *et al.* focused on respiratory outcomes, our study extends this observation by linking SIRI to overall disease severity at presentation. Collectively, these findings reinforce the role of SIRI as a promising biomarker for risk stratification in GBS.

This study has certain limitations. Primarily, due to its retrospective design, it is vulnerable to potential data collection inaccuracies and recording errors, which preclude causal interpretation of the relationships between variables. Furthermore, the study was conducted at a single center and involved data from only 40 patients with GBS. This relatively small sample size limits statistical power and generalizability of the results. While the rarity of GBS explains the limited patient numbers, larger, multicenter studies are needed to substantiate the findings. In addition, temporal changes in hematological indices were not taken into account, as only presentation values were evaluated. This limitation may impede the accurate reflection of the dynamic nature of the inflammatory response. Moreover, potential confounding factors such as concurrent or preceding infections, which could elevate systemic inflammatory indices like SIRI, could not be completely excluded because of incomplete microbiological data. Lastly, no subtype-based analysis was conducted within the study cohort; therefore, the effect of different clinical variants of GBS on inflammation parameters remains unexplored. In light of these limitations, the findings should be interpreted as hypothesis-generating.

In conclusion, SIRI appears to be significantly associated with severe neurological dysfunction in GBS and demonstrates moderate-to-high accuracy in predicting clinical severity at a cut-off value of ≥1.43. Although its ease of application may render SIRI a useful adjunct tool in triage and prognostication, confirmation through larger and prospective studies remains essential.

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