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ORIGINAL ARTICLE

Monocytes and systemic inflammation in Guillain-Barré syndrome: Subtypes and relationship with prognosis

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ABSTRACT

Objectives: This study aimed to evaluate the role of hematological inflammation markers [monocyte count, neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), and C-reactive protein (CRP)] in Guillain-Barré syndrome (GBS), compare these markers across GBS subtypes, and explore their relationship with disease prognosis.

Patients and methods: This retrospective study included 65 patients (40 males, 25 females; mean age: 49.5±17.0 years; range, 18 to 89 years) with GBS (35 acute inflammatory demyelinating polyneuropathy [AIDP], 30 acute motor axonal neuropathy [AMAN] and acute motor sensory axonal neuropathy [AMSAN]) and 60 age- and sex-matched healthy controls (33 males, 27 females; mean age: 50.3±16.9 years; range, 19 to 88 years) between January 2022 and February 2025. Complete blood count parameters and CRP were recorded at diagnosis. Functional disability was assessed using the Hughes Functional Grading Scale at baseline and after three months.

Results: Compared to controls, GBS patients had significantly higher monocyte counts and NLR, SII, and CRP levels ($p<0.001$). Among subtypes, the axonal group showed a higher monocyte count ($p=0.017$) and NLR ($p=0.035$) than the demyelinating group. No significant differences were observed in SII or CRP between subtypes. Linear regression analysis revealed that a higher NLR ($\beta=-0.15$, $p=0.018$) and SII ($\beta=-0.529$, $p=0.048$) were associated with less improvement in the Hughes score.

Conclusion: Inflammation markers such as the monocyte count, NLR, and SII are elevated in GBS, particularly in axonal subtypes. Neutrophil-to-lymphocyte ratio and SII may serve as potential prognostic markers, with higher values indicating poorer clinical recovery.

Keywords: Guillain-barré syndrome, monocyte count, systemic inflammation.

Guillain-Barré syndrome (GBS) is the most common cause of acute neuromuscular paralysis worldwide. The annual incidence ranges from 0.44 to 2.12 per 100,000 people.[1] It is more common in males than in females, and the incidence of the disease increases with age.[2] In most cases, GBS occurs as a postinfectious disease; approximately two-thirds of patients report having had a gastrointestinal or respiratory tract infection before the onset of the disease. However, GBS can also develop after vaccinations and surgical interventions. Although *Campylobacter jejuni* is the most associated agent, various pathogens, including Epstein-Barr virus, cytomegalovirus, hepatitis E virus, Mycoplasma pneumoniae, *Haemophilus influenzae,* influenza A virus, and Zika virus, are also associated with GBS.[3] Vaccine-related GBS cases have been reported mostly after influenza vaccines, and there are also cases in the literature of GBS developing after herpes zoster and coronavirus disease 2019 (COVID-19) vaccines.[4]

Guillain-Barré syndrome is a neuroinflammatory disease, and both humoral and cellular immune systems play a role in its pathogenesis.[5] The fact

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that different T-cell activations were detected between acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor sensory axonal neuropathy (AMSAN) subtypes suggests that these subtypes are associated with different inflammatory mechanisms.[6]

Monocytes are a group of leukocytes originating from the bone marrow and circulating in the bloodstream. They constitute approximately 10% of leukocytes in the peripheral bloodstream. They play a crucial role in regulating the inflammatory response and releasing proinflammatory cytokines.[7] Monocytes play a pivotal role in the development of not only GBS but also other chronic inflammatory diseases, such as atherosclerosis.[8] It is also known that circulating monocytes can cross the blood-brain barrier into the central nervous system tissue and differentiate into microglial cells there. Microglial cells play a role in various neuroinflammatory and neurodegenerative processes, including Alzheimer's disease and migraine.[9]

Neutrophilia and lymphocytopenia represent the cellular response to systemic inflammation. An increase in the difference between neutrophil and lymphocyte counts reflects the severity of the inflammatory response. Therefore, the neutrophil-to-lymphocyte ratio (NLR) is used as a marker in various pathological conditions, including malignancies, chronic inflammatory diseases, and cardiovascular diseases.[10] Studies on NLR in GBS are limited. On the other hand, the systemic immune-inflammation index (SII), calculated with the formula platelet × neutrophil/lymphocyte, is a more sensitive indicator compared to other peripheral blood inflammation markers in recent years.[11] Studies in the literature on the importance of this marker in GBS are limited.

Although some inflammation markers are associated with GBS, the number of studies evaluating the association of these markers with GBS subtypes and demonstrating the relationship between these markers and the disease prognosis is insufficient. In this study, we aimed to investigate the differences in hematological and inflammatory markers, such as monocyte count, NLR, and SII, in patients with GBS compared to healthy individuals to determine whether these markers differ according to GBS subtypes and to examine their relationship with disease prognosis.

PATIENTS AND METHODS

A total of 65 patients (40 males, 25 females; mean age: 49.5±17.0; range, 18 to 89 years) hospitalized in the Neurology Clinic of the Kayseri City Hospital between January 2022 and February 2025, and diagnosed with GBS according to international diagnostic criteria, were included in this retrospective study.[12] Of these patients, 35 were classified as AIDP and 30 as AMAN or AMSAN subtypes. Complete blood count parameters obtained at the time of diagnosis included monocyte, lymphocyte, neutrophil, platelet counts, and C-reactive protein (CRP) values. The control group consisted of 60 healthy individuals (33 males, 27 females; mean age: 50.3±16.9 years; range, 19 to 88 years) matched with the patient group in terms of age and sex. In both groups, individuals with an acute or chronic infectious disease, chronic inflammatory disease, hematological disorder, or malignancy, human immunodeficiency virus carriers, and those with a history of chronic autoimmune disease were excluded from the study. Written informed consent was obtained from the participants. The study protocol was approved by the Kayseri City Hospital Clinical Research Ethics Committee (Date: 27.05.2025, No: E2-457). The studies were conducted in accordance with the principles of the Declaration of Helsinki.

The degree of functional disability in patients at the time of diagnosis was assessed using the Hughes Functional Grading Scale.[13] In this scale, 0 indicates no motor deficit; 1, mild weakness with independence in all activities; 2, ambulatory but has difficulty in activities such as climbing stairs; 3, can walk with assistance; 4, can get out of bed but cannot walk; 5, need for mechanical ventilation; 6, death. The Hughes score was reevaluated at the second visit, three months after the initial diagnosis.

Regarding treatment, eight patients in the demyelinating group and seven patients in the axonal group received plasmapheresis. All remaining patients were treated with intravenous immunoglobulin. Treatment selection was based on clinical severity and physician preference, following institutional protocols.

All laboratory studies for the patient and control groups were performed in a single center. The normal ranges for the hematological and biochemistry markers, as measured using the

Sysmex-K1000 Hematology Analyzer (Sysmex Corporation, Kobe, Japon) and Roche Cobas 8000 Modular Analyzer Series (Roche Diagnostics, Basel, Switzerland) devices were as follows: monocyte count, 0.2-0.8×10³/mm³; NLR, <3.0; SII, <500; and CRP, <5 mg/L. In the patient group, laboratory analyses were performed before treatment.

Statistical analysis

The analysis of the data obtained from the study was performed using IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to determine whether the data exhibited a normal distribution. Continuous variables were expressed as mean ± standard deviation (SD) when they showed normal distribution, and as median (min-max) values when they did not show normal distribution. Categorical variables were shown as frequency and percentages (%). In the comparison of continuous variables between two groups, the independent sample t-test was used for data showing normal distribution, and the Mann-Whitney U test was used for data not showing normal distribution.

To evaluate the differences in hematological and inflammatory markers such as monocytes, NLR, and SII between GBS demyelinating and axonal subtypes (AIDP and AMAN/AMSAN), an independent sample t-test or Mann-Whitney U test was used as appropriate. To determine the factors affecting the disease prognosis, the Hughes score was calculated at the time of admission and three months after admission. A Hughes score <2 was considered a good prognosis. The relationship between inflammation markers and prognosis among GBS subtypes was analyzed using the Spearman correlation test. Linear regression analysis was applied to examine the relationship between inflammation markers and prognosis, and statistical significance was assessed. The statistical significance level was accepted as $p<0.05$ in all analyses.

RESULTS

While the mean monocyte count was 0.69±0.28 in the GBS group, it was 0.43±0.11 in the control group. Statistically, the monocyte count was higher in the GBS group ($p<0.001$). The mean NLR was 3.79±2.93 in the GBS group and 2.21±0.76 in the control group. The NLR was statistically higher in the GBS group ($p<0.001$). The mean SII was 1027.48±728.7 in the GBS group and 566.17±252.79 in the control group. The SII was found to be statistically higher in the GBS group ($p<0.001$).

TABLE 1
Comparison of inflammatory markers between GBS and control groups

	GBS (n=65)	Control group (n=60)			
	Mean±SD	Mean±SD	t	z	*p*
Monocyte count (×10³/mm³)	0.69±0.28	0.43±0.11	6.712		<0.001
Neutrophil-to-lymphocyte ratio	3.79±2.93	2.21±0.76		4.262	<0.001
Systemic immune-inflammation index	1027.48±728.7	566.17±252.79		4.779	<0.001
C-reactive protein (mg/L)	6.01±5.4	1.58±1.31		5.951	<0.001

GBS: Guillain-Barré syndrome; SD: Standard deviation.

TABLE 2
Comparison of inflammatory markers between GBS subtypes

	Demyelinating polyneuropathy (n=35)	Axonal polyneuropathy (n=30)		
	Mean±SD	Mean±SD	z	*p*
Monocyte count (×10³/mm³)	0.61±0.25	0.77±0.28	–2.397	0.017
Neutrophil-to-lymphocyte ratio	3.12±1.94	4.53±3.62	–2.115	0.035
Systemic immune-inflammation index	921.25±722.52	1144.11±729.21	–1.944	0.053
C-reactive protein (mg/L)	5.08±4.45	7.03±6.19	–1.320	0.189

GBS: Guillain-Barré syndrome; SD: Standard deviation.

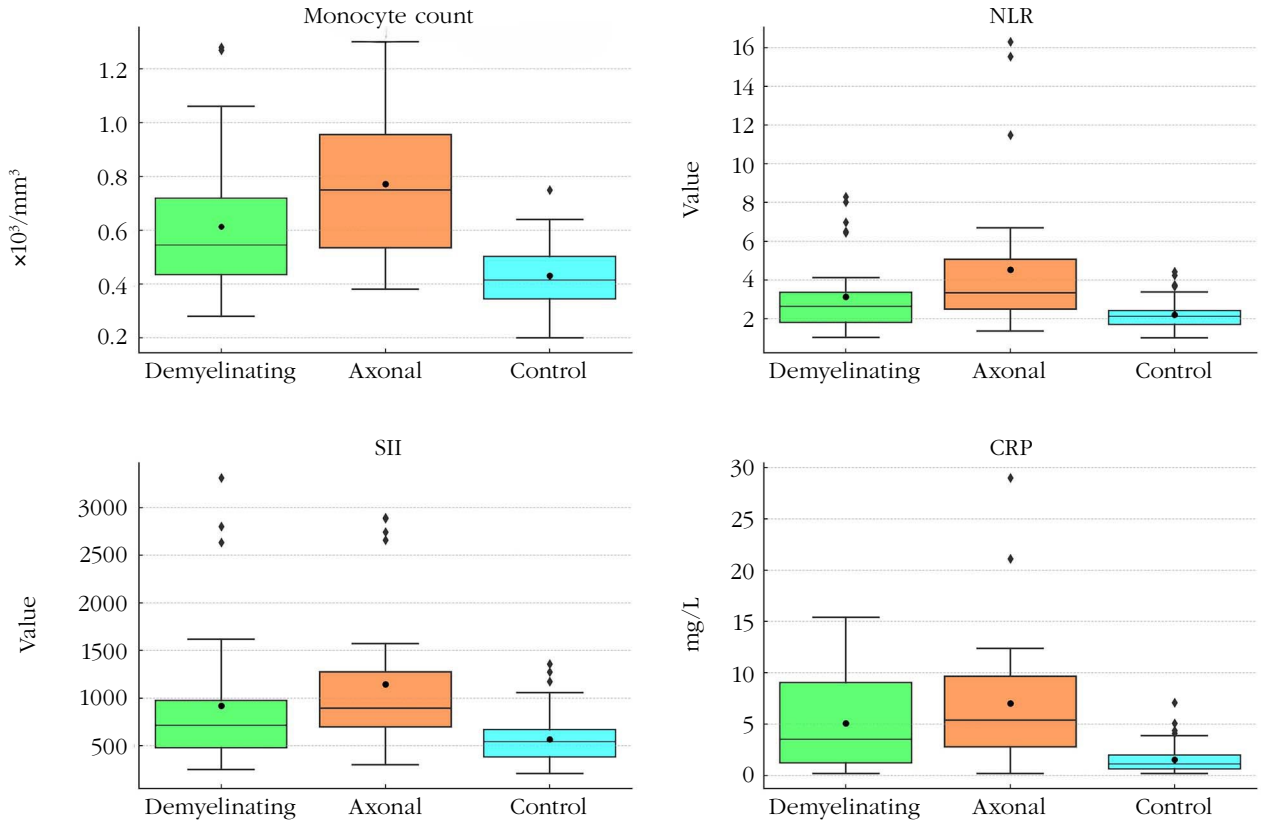

Figure 1. Comparison of mean inflammatory marker levels between study groups.
NLR: Neutrophil-to-lymphocyte ratio; SII: Systemic immune-inflammation index; CRP: C-reactive protein.

The mean CRP was found to be 6.01±5.4 in the GBS group and 1.58±1.31 in the control group. The CRP level was found to be statistically higher in the GBS group (p<0.001; Table 1).

Inflammation markers were compared between GBS subtypes. While the mean monocyte count was 0.61±0.25 in patients with demyelinating polyneuropathy, it was 0.77±0.28 in the axonal polyneuropathy group. The monocyte count was higher in patients with axonal polyneuropathy than in those with demyelinating polyneuropathy (p=0.017). While the mean NLR was 3.12±1.94 in patients with demyelinating polyneuropathy, it was 4.53±3.62 in the axonal polyneuropathy group. The NLR was higher in patients with axonal polyneuropathy than in those with demyelinating polyneuropathy (p=0.035). While the mean SII was found to be 921.25±722.52 in patients with demyelinating polyneuropathy, it was found to be 1144.11±729.21 in the axonal polyneuropathy group. No statistically significant difference was found between the SII values of the two groups (p=0.053). While the mean CRP level was found to be 5.08±4.45 in patients with demyelinating

TABLE 3
Correlation between inflammatory markers and prognosis in patients with demyelinating polyneuropathy

	Spearman rho	*p*
Monocyte count (×10³/mm³)	−0.065	0.713
Neutrophil-to-lymphocyte ratio	0.074	0.679
Systemic immune-inflammation index	0.147	0.407
C-reactive protein (mg/L)	−0.064	0.719

TABLE 4
Correlation between inflammatory markers and prognosis in patients with axonal polyneuropathy

	Spearman rho	*p*
Monocyte count (×10³/mm³)	0.127	0.496
Neutrophil-to-lymphocyte ratio	−0.048	0.798
Systemic immune-inflammation index	0.063	0.734
C-reactive protein (mg/L)	−0.254	0.167

TABLE 5
Multiple linear regression analysis

	β	SE	t	95% CI Lower	95% CI Upper	*p*
Constant	2.279	0.259	8.786	1.76	2.797	0
Monocyte count (×10³/mm³)	−0.14	0.363	−0.386	−0.866	0.586	0.701
Neutrophil-to-lymphocyte ratio	−0.15	0.062	−2.423	−0.275	−0.026	0.018
Systemic immune-inflammation index	−0.529	0.262	−2.016	−0.004	1.053	0.048
C-reactive protein (mg/L)	−0.021	0.019	−1.093	−0.058	0.017	0.278

CI: Confidence interval; Model R^2=0.124; SE: Standard error.

polyneuropathy, it was found to be 7.03±6.19 in the axonal polyneuropathy group. No statistically significant difference was found between the CRP levels of the two groups (p=0.189; Table 2, Figure 1).

Monocyte count, NLR, SII, and CRP values were not correlated with prognosis in patients with demyelinating polyneuropathy and axonal polyneuropathy (Tables 3, 4).

Inflammatory markers affecting the change in the Hughes score were determined with linear regression analysis. Accordingly, the change in the Hughes score was less in patients with high NLR values (β=−0.15, p=0.018). Similarly, the change in the Hughes score is less pronounced in patients with a high SII value (β=−0.529, p=0.048). No statistically significant relationship was found between the monocyte count, and CRP level, and the Hughes score (Table 5).

DISCUSSION

In our study, monocyte count, NLR, SII, and CRP values were statistically significantly higher in patients with both demyelinating and axonal type polyneuropathy than in the control group. While monocyte count and NLR values were statistically significantly higher in the axonal polyneuropathy group than in the demyelinating polyneuropathy group, no difference was found between the groups in terms of SII and CRP values. Hematological and inflammatory markers and CRP values were not correlated with the disease prognosis. However, in the linear regression analysis, we found that the level of recovery was lower in patients with high NLR and SII values.

Monocytes can transform into macrophages or dendritic cells in the tissue and participate in immune processes by releasing cytokines, phagocytosis, and presenting antigens.[14] The blood-brain barrier is permeable to monocytes. Monocytes can cross the blood-brain barrier and differentiate into microglia cells. This suggests that monocytes may have a neuroinflammatory role.[15] Previous studies demonstrated that microglial cells play a crucial role in neurodegenerative diseases, including Parkinson's disease and Alzheimer's disease.[16] The role of proinflammatory cytokines in the pathophysiology of GBS is essential. The roles of tumor necrosis factor alpha, interleukin (IL)-1 beta, interferon gamma, IL-6, and IL-17 were demonstrated in previous studies.[17,18] Interleukin-17 was shown to increase the release of other proinflammatory cytokines from monocytes.[18] Moreover, these cytokines also differ among GBS subtypes.[19] In our study, we found that the number of monocytes was significantly higher in GBS patients compared to the control group, and this increase was more pronounced in patients with the axonal polyneuropathy subtype than in those with the demyelinating polyneuropathy subtype. We believe that this difference may be due to the difference in the cytokine profiles released in the subtypes of GBS.

The immune response of the immune system to physiological changes is characterized by an increase in the number of neutrophils and a decrease in the number of lymphocytes.[20] Previous studies demonstrated that it was associated with poor prognosis in central nervous system diseases such as multiple sclerosis, ischemic stroke, Parkinson's disease, Alzheimer's disease, and amyotrophic lateral sclerosis.[21] In a systematic meta-analysis investigating the role of NLR in patients with GBS, conducted by Sarejloo et al.,[22] it was found that NLR count was higher than those in the control group and was a significant predictor of prognosis. However, no significant difference was found between NLR and GBS subtypes. In our study, we also found that NLR count was higher in the GBS

group than in the control group and was associated with a poor prognosis. Unlike previous studies, NLR count was found to be higher in patients with axonal neuropathy in our study.

The SII is a new inflammatory marker based on three types of inflammatory cells in the blood (platelets, neutrophils, and lymphocytes); it shows the balance of these cells in the blood.[23] This index was initially used to predict the prognosis of oncological and cardiovascular diseases. Subsequently, it was also employed to evaluate the prognosis of spontaneous intracranial hemorrhage and acute ischemic stroke.[24] In a study conducted by Liu et al.,[25] it was demonstrated that SII was higher in GBS patients than in controls and was associated with a poor prognosis; however, no relationship was found between SII and GBS subtypes. Wu et al.[26] showed that high SII values could predict respiratory failure in patients with GBS. Similarly, in our study, we demonstrated that prognosis was worse in patients with high SII, and SII values were higher in axonal subtypes than in demyelinating subtypes; however, we did not find this increase to be statistically significant.

C-reactive protein is an acute-phase reactant synthesized by the liver in response to inflammation and infection.[27] It activates the complement system via complement C1q by binding to phosphocholine on the surface of dead or near-dead cells, thus enabling the clearance of necrotic and apoptic cells from the environment.[28] In the study conducted by Altaweel et al.,[29] high CRP values at the time of diagnosis were associated with the need for mechanical ventilation and poor prognosis. In a study conducted by Paneyala et al.,[30] it was reported that the Hughes score was higher in patients with high CRP levels at the time of diagnosis, and that CRP values did not differ significantly between subtypes. Similarly, in our study, we did not observe any difference in CRP levels between subtypes, and we also showed that CRP values did not affect long-term prognosis.

This study had several limitations. First, due to the limited sample size, the axonal group could not be subdivided into AMAN and AMSAN, preventing a more detailed analysis of their potentially distinct inflammatory profiles. Second, because of the retrospective design, follow-up was limited to three months, restricting evaluation of long-term outcomes and the prognostic utility of the studied biomarkers. Third, inflammatory markers were assessed only at the time of diagnosis, before treatment initiation. Although this approach reflects baseline disease activity, it does not provide information about the dynamic changes in these markers over the disease course. Future prospective studies with larger sample sizes, extended follow-up periods, and serial biomarker measurements are warranted to better understand the role of systemic inflammation in GBS prognosis. Moreover, due to the small number of patients receiving plasmapheresis and the similar distribution across groups, treatment type was not included as a variable in statistical analyses. Since treatment modalities such as intravenous immunoglobulin and plasmapheresis may influence the levels of inflammatory biomarkers during the disease course, we preferred to use only the baseline measurements obtained at diagnosis (before treatment initiation) to ensure that the values reflected the baseline state of disease-related systemic inflammation.

In conclusion, GBS patients' monocyte counts and NLR, SII, and CRP levels were significantly higher compared to healthy individuals. The number of monocytes and NLR increased more significantly in the axonal subtype. Higher NLR and SII values were associated with worse clinical recovery. These inflammatory markers may be potential biomarkers for GBS subtypes and prognosis.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Surgical and medical practices, writing: M.A.; Concept, design, data collection or processing, analysis or interpretation: M.A, F.E, G.S.; Literature search: F.E, G.S.

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