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Prognostic value of systemic immune-inflammation index (SII), systemic inflammation response index (SIRI) and white blood cell–mean platelet volume ratio (WMR) in predicting mortality in acute ischemic stroke

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Abstract

Background & Objective: Systemic inflammatory markers have recently gained attention as potential predictors of clinical outcomes in acute ischemic stroke. This study aimed to evaluate the prognostic value of the Systemic Immune-Inflammation Index (SII), Systemic Inflammation Response Index (SIRI), and White Blood Cell–Mean Platelet Volume Ratio (WMR) in predicting early mortality and neurological status at hospital admission. *Methods:* This retrospective study included 140 patients diagnosed with acute ischemic stroke. Demographic data, laboratory parameters, and clinical scores (NIHSS, mRS, GCS) at admission were recorded. Patients were grouped as survivors and non-survivors. Between-group comparisons, correlation analyses, ROC curves, and binary logistic regression were performed to evaluate the predictive role of SII, SIRI, and WMR. *Results:* Non-survivors had significantly higher SIRI, SII, and WMR values compared with survivors ($p<0.001$ for all). SIRI and SII showed moderate positive correlations with baseline NIHSS ($r=0.346$ and $r=0.401$) and mRS scores ($r=0.368$ and $r=0.402$), while correlating negatively with GCS ($r=–0.336$ and $r=–0.393$). ROC analyses demonstrated good diagnostic performance for SIRI (AUC=0.760), SII (AUC=0.724), and WMR (AUC=0.678) in distinguishing mortality. In multivariable logistic regression, SII (OR=36.42; $p=0.001$), SIRI (OR=1.41; $p=0.023$), and WMR (OR=0.05; $p=0.003$) were identified as independent predictors of mortality.
Conclusion: Higher SIRI, SII, and WMR values are strongly associated with early mortality and worse neurological status in acute ischemic stroke. These readily available and low-cost inflammatory indices may assist in rapid risk stratification and early clinical decision-making in the acute phase.

Keywords: Acute ischemic stroke, systemic inflammation, SIRI, SII, WMR, mortality, prognosis

INTRODUCTION

Acute ischemic stroke remains a leading cause of mortality and long-term disability, and early identification of high-risk patients is essential for improving outcomes. A growing body of evidence indicates that the systemic inflammatory cascade triggered immediately after cerebral ischemia contributes significantly to secondary neuronal injury, infarct progression, and functional decline.[1] Conventional inflammatory markers provide limited insight into this complex process, prompting interest in composite indices that better reflect the balance between innate and adaptive immune responses.

In recent years, hemogram-derived inflammatory indices such as the Systemic Immune-Inflammation Index (SII) and the Systemic Inflammation Response Index (SIRI) have emerged as promising biomarkers in cardiovascular and systemic inflammatory conditions.[2] These markers integrate neutrophils, lymphocytes, monocytes, and platelets into a single calculation and have been increasingly studied in the context of stroke.

Several studies have demonstrated that elevated SII and SIRI values are associated with greater stroke severity, early neurological deterioration, and unfavorable functional outcomes.[3-5] Alongside these indices, the White Blood Cell–Mean

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Platelet Volume Ratio (WMR) has been proposed as another marker of heightened inflammatory activation and has been linked to early neurological worsening in ischemic stroke populations.[6] Together, these parameters appear to offer a broader understanding of the inflammatory milieu observed in acute stroke.

Recent analyses further suggest that systemic inflammation may influence not only the acute neurological picture but also the development of stroke-associated complications, thereby affecting long-term prognosis.[7] Additionally, composite inflammatory indices have shown potential value in predicting early mortality after stroke, highlighting their usefulness as rapid and inexpensive prognostic tools.[8,9] Although these findings are encouraging, the combined predictive value of SII, SIRI, and WMR in real-world acute ischemic stroke cohorts remains insufficiently explored. Most existing studies have included smaller patient groups, focused on selective clinical scenarios, or lacked detailed multivariable analyses.

Given that these markers are inexpensive, routinely available, and can be calculated immediately upon admission, demonstrating their prognostic reliability could aid clinicians in early risk stratification and clinical decision-making. Therefore, the present study aims to investigate the association of SII, SIRI, and WMR with early mortality and neurological severity scores (NIHSS, mRS, GCS) in patients with acute ischemic stroke. By incorporating comprehensive laboratory and clinical data, this study intends to provide practical and clinically applicable evidence supporting the integration of systemic inflammatory indices into routine stroke evaluation.

METHODS

This retrospective observational study was carried out in the Neurology Clinic and the Neurology Intensive Care Unit of Gaziantep City Hospital. Medical records of patients diagnosed with acute ischemic stroke were reviewed. A total of 140 patients who met the eligibility criteria were included, and they were classified into survivors (n=79) and non-survivors (n=61) based on in-hospital outcomes. The study was approved by the Institutional Review Board of Gaziantep City Hospital, and all procedures adhered to the principles of the Declaration of Helsinki.

Patients aged 18 years and older with radiologically confirmed acute ischemic stroke and complete laboratory and clinical data obtained at admission were eligible for inclusion. Individuals with hemorrhagic stroke, transient ischemic attack, active infection, documented chronic inflammatory or malignant disease, chronic renal or hepatic failure, or incomplete medical records were excluded to prevent confounding effects on inflammatory biomarkers.

Clinical and demographic data were obtained from the electronic hospital database and included age, sex, neurological status, and survival at discharge. Neurological severity at admission was assessed using the National Institutes of Health Stroke Scale (NIHSS), the modified Rankin Scale (mRS), and the Glasgow Coma Scale (GCS). Routine laboratory parameters collected at admission encompassed complete blood count results—neutrophils, lymphocytes, monocytes, platelets, and mean platelet volume—as well as biochemical markers including urea, creatinine, electrolytes, albumin, total protein, CRP, fibrinogen, procalcitonin, uric acid, and D-dimer. From these measurements, systemic inflammatory indices were calculated using established formulas: the Systemic Immune-Inflammation Index (SII) as neutrophil × platelet count / lymphocyte count; the Systemic Inflammation Response Index (SIRI) as neutrophil × monocyte count / lymphocyte count; and the White Blood Cell–Mean Platelet Volume Ratio (WMR) as total leukocyte count / mean platelet volume. The primary endpoint of the study was in-hospital mortality, while secondary analyses explored the associations between inflammatory indices and admission NIHSS, mRS, and GCS scores.

All statistical analyses were performed using IBM SPSS Statistics version 23.0 and R software. The distribution of continuous variables was assessed with the Kolmogorov–Smirnov test. Variables with normal distribution were compared using the independent samples t-test, whereas non-normally distributed variables were analyzed with the Mann–Whitney U test. Categorical variables were compared using the chi-square test. The relationships between SII, SIRI, WMR, and neurological scores were examined using Spearman's rank correlation analysis. Receiver Operating Characteristic (ROC) curve analysis was used to evaluate the diagnostic performance of inflammatory indices and other laboratory parameters in predicting mortality, and area under the curve (AUC), optimal cut-off values, sensitivity, specificity, positive predictive value, and negative predictive value were reported accordingly. Finally, binary logistic regression analysis was performed to determine independent predictors of mortality; variables with a p-value

Table 1: Baseline demographic and clinical characteristics of the patients

Variable	Value
Sex (Female/Male)	78 (55.7%) / 62 (44.3%)
Survival status (survivor/non-survivor)	79 (56.4%) / 61 (43.6%)
Age (years)	67.66 ± 13.79 (median: 69; range: 25–113)
Admission NIHSS	13.5 ± 6.69 (median: 13; range: 1–26)
Admission mRS	3.77 ± 1.26 (median: 4; range: 1–5)
Admission GCS	10.6 ± 3.64 (median: 12; range: 4–15)

NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale; GCS, Glasgow Coma Scale.

below 0.10 in univariate analyses were entered into a multivariable model. Statistical significance was set at $p<0.05$.

RESULTS

A total of 140 patients were included in the final analysis, consisting of 78 women (55.7%) and 62 men (44.3%). The mean age was 67.66 ± 13.79 years, and 61 patients (43.6%) died during hospitalization. Baseline clinical characteristics, including a median NIHSS score of 13, mRS of 4, and GCS of 12, are summarized in Table 1. These values indicate that most patients presented with moderate-to-severe neurological impairment.

When survivors and non-survivors were compared, marked differences were observed across several laboratory parameters (Table 2). Non-survivors exhibited significantly higher levels of urea (107.05 vs. 29 mg/dL; $p < 0.001$), creatinine (1.98 vs. 0.77 mg/dL; $p < 0.001$), CRP (108.5 vs. 15.2 mg/L; $p < 0.001$), procalcitonin

Table 2: Comparison of laboratory parameters between survivors and non-survivors

Parameter	Survivors	Non-survivors	p-value
AST	24	49	<0.001
Urea (mg/dL)	29	107.05	<0.001
Creatinine (mg/dL)	0.77	1.98	<0.001
Magnesium (mg/dL)	1.91 ± 0.25	2.32 ± 0.50	<0.001
Phosphorus (mg/dL)	3.33	4.59	<0.001
Calcium (mg/dL)	8.73 ± 0.76	8.16 ± 0.88	<0.001
Total Protein (g/L)	61	52	<0.001
Albumin (g/L)	34.08 ± 5.53	27.90 ± 5.20	<0.001
CRP (mg/L)	15.2	108.5	<0.001
Procalcitonin (ng/mL)	0.09	2.62	<0.001
D-dimer (mg/L)	1.09	6.21	<0.001
Fibrinogen (mg/dL)	351.4	486	<0.001
Uric Acid (mg/dL)	4.68 ± 1.93	6.91 ± 3.05	<0.001
WBC ($\times10^3/\mu L$)	8.6	12.8	<0.001
RBC ($\times10^6/\mu L$)	4.28 ± 0.84	3.74 ± 0.95	0.001
Hematocrit (%)	36.28 ± 5.46	32.94 ± 8.21	0.007
Platelets ($\times10^3/\mu L$)	278	205	0.001
MPV (fL)	8.7	9.8	<0.001
Neutrophils ($\times10^3/\mu L$)	5.3	10.6	<0.001
Lymphocytes ($\times10^3/\mu L$)	1.89 ± 0.69	1.41 ± 0.88	0.001
Monocytes ($\times10^3/\mu L$)	0.7	0.8	0.369
SIRI	2.07	6.21	<0.001
SII	806.73	1691.25	<0.001
WMR	0.97	1.18	<0.001
Admission NIHSS	9	20	<0.001
Admission mRS	3	5	<0.001
Admission GCS	13	7	<0.001

SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; WMR, White Blood Cell–Mean Platelet Volume Ratio; WBC, white blood cell count; RBC, red blood cell count; MPV, mean platelet volume; CRP, C-reactive protein; AST, aspartate aminotransferase.

Table 3: Correlation between inflammatory indices and clinical severity scores

Clinical Variable	SIRI r (p)	SII r (p)	WMR r (p)
Admission NIHSS	0.346 (<0.001)	0.401 (<0.001)	0.237 (0.005)
Admission mRS	0.368 (<0.001)	0.402 (<0.001)	0.245 (0.004)
Admission GCS	–0.336 (<0.001)	–0.393 (<0.001)	–0.233 (0.006)

SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; WMR, White Blood Cell–Mean Platelet Volume Ratio; NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale; GCS, Glasgow Coma Scale.

(2.62 vs. 0.09 ng/mL; $p < 0.001$), D-dimer (6.21 vs. 1.09 mg/L; $p < 0.001$), and fibrinogen (486 vs. 351.4 mg/dL; $p < 0.001$). Inflammatory cell parameters also showed significant differences: WBC count (12.8 vs. 8.6 $\times 10^3/\mu L$; $p < 0.001$), neutrophils (10.6 vs. 5.3 $\times 10^3/\mu L$; $p < 0.001$), and MPV (9.8 vs. 8.7 fL; $p < 0.001$) were all elevated in the non-survivor group. Conversely, albumin (27.9 vs. 34.08 g/L; $p < 0.001$), total protein (52 vs. 61 g/L; $p < 0.001$), hematocrit, and lymphocyte counts were significantly lower among patients who died.

Inflammatory indices demonstrated clear and consistent differences between groups. Median SIRI (6.21 vs. 2.07), SII (1691.25 vs. 806.73), and WMR (1.18 vs. 0.97) were all significantly higher in non-survivors ($p < 0.001$ for all), supporting a strong association between systemic inflammation and mortality. Likewise, admission NIHSS and mRS scores were markedly higher, while GCS was lower in the non-survivor group (NIHSS: 20 vs. 9; mRS: 5 vs. 3; GCS: 7 vs. 13), reflecting more severe neurological compromise at presentation.

Correlation analyses revealed significant associations between the inflammatory indices and clinical severity scores (Table 3). SIRI demonstrated weak-to-moderate correlations with baseline NIHSS ($r = 0.346$, $p < 0.001$) and mRS ($r = 0.368$, $p < 0.001$), and a negative correlation with GCS ($r = –0.336$, $p < 0.001$). SII showed the strongest correlations among the three markers, displaying moderate relationships with NIHSS ($r = 0.401$, $p < 0.001$) and mRS ($r = 0.402$, $p < 0.001$), and a significant negative correlation with GCS ($r = –0.393$, $p < 0.001$). WMR was also significantly correlated with all neurological scores, though to a lesser extent.

Receiver operating characteristic (ROC) analysis demonstrated that the inflammatory indices had acceptable discriminative performance for predicting in-hospital mortality (Table 4). SIRI showed the highest diagnostic accuracy among these markers (AUC = 0.760) with an optimal cut-off of ≥3.313, yielding 72.13% sensitivity and 77.22% specificity. SII yielded an AUC of 0.724 (cut-off ≥1809.176), characterized by high specificity (88.61%) but lower sensitivity (49.18%). WMR demonstrated an AUC of 0.678, with high specificity (89.87%) but modest sensitivity (45.9%). Procalcitonin showed the highest AUC overall (0.870), while platelet-to-lymphocyte ratio did not differ significantly between groups and had no discriminatory value ($p = 0.380$).

Binary logistic regression analysis revealed that SII, SIRI, and WMR were independently associated with in-hospital mortality after adjustment for potential confounders (Table 5). In univariate analysis, increases in SII (OR = 2.58; $p < 0.001$), SIRI (OR = 1.32; $p < 0.001$), and WMR (OR = 4.34; $p = 0.001$) were strongly associated with mortality. In the multivariable model, SII remained a powerful independent predictor (OR = 36.42; $p = 0.001$), along with SIRI (OR = 1.41; $p = 0.023$). Interestingly, WMR showed an inverse

Table 4: ROC curve analysis for predicting in-hospital mortality

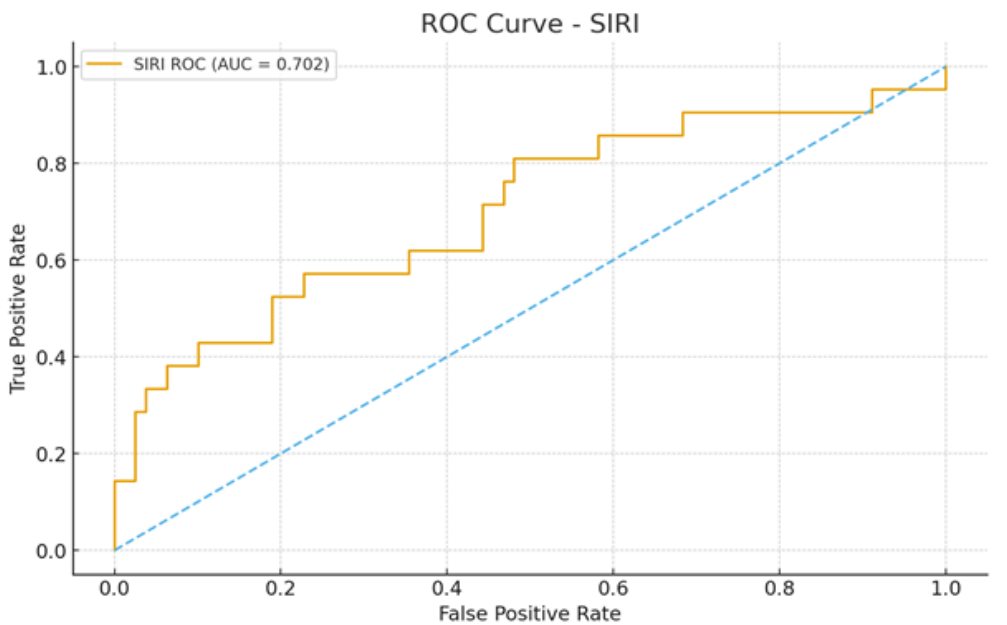
Marker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)
SIRI	0.760	≥3.313	72.13	77.22
SII	0.724	≥1809.176	49.18	88.61
WMR	0.678	≥1.391	45.90	89.87
Procalcitonin	0.870	≥0.285	78.69	83.54
LYM/MONO	0.700	≤5.571	11.67	97.47
PLT/LYM	0.543	–	–	–

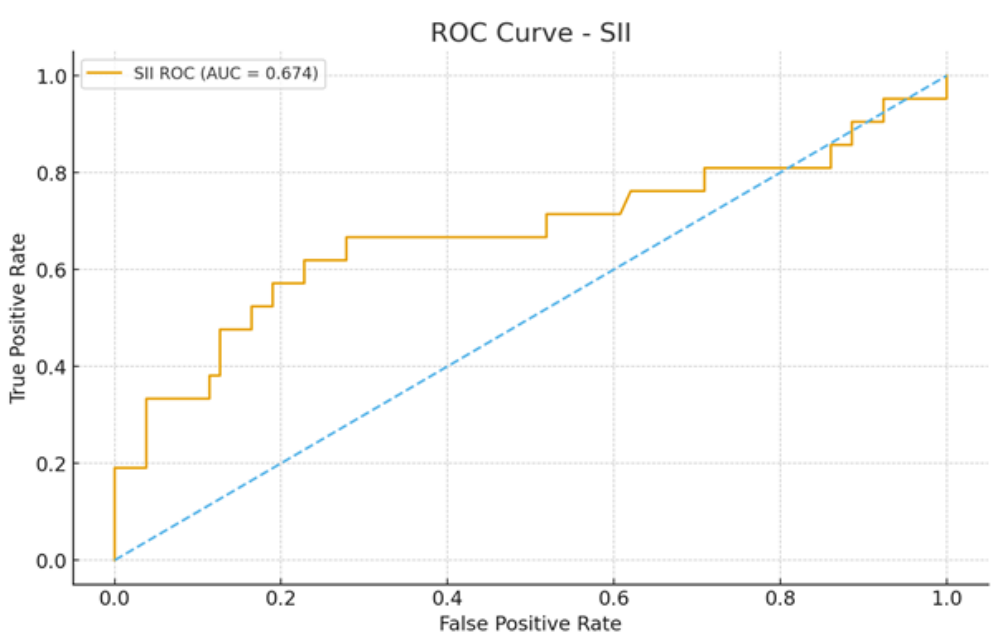
ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval; SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; WMR, White Blood Cell–Mean Platelet Volume Ratio.

Table 5: Binary logistic regression analysis of predictors of mortality

Variable	Univariate OR (95% CI)	p	Multivariate OR (95% CI)	p
SIRI	1.32 (1.16–1.50)	<0.001	1.41 (1.05–1.89)	0.023
SII	2.58 (1.64–4.06)	<0.001	36.42 (3.99–332.48)	0.001
WMR	4.34 (1.89–9.98)	0.001	0.05 (0.01–0.36)	0.003
LYM/MONO	0.92 (0.77–1.10)	0.353	1.54 (1.02–2.33)	0.041
PLT/LYM	1.23 (0.75–2.00)	0.411	0.02 (0.00–0.19)	0.001
Procalcitonin	1.01 (0.97–1.03)	0.134	1.00 (0.99–1.02)	0.824

OR, odds ratio; CI, confidence interval; SII, Systemic Immune-Inflammation Index; SIRI, Systemic Inflammation Response Index; WMR, White Blood Cell–Mean Platelet Volume Ratio.





association with mortality (OR = 0.05; p = 0.003) after adjustment, likely reflecting interactions between leukocyte and platelet parameters in the multivariable context. Lymphocyte-to-monocyte ratio also emerged as an independent predictor (OR = 1.54; p = 0.041), whereas procalcitonin lost significance in the adjusted model.

Overall, the non-survivor group displayed a distinct inflammatory and biochemical profile, characterized by elevated systemic inflammation indices, severe neurological impairment at presentation, and significantly worse clinical outcomes. These findings underscore the importance of SIRI, SII, and WMR as accessible biomarkers for early mortality prediction in acute ischemic stroke.

DİSCUSSİON

In this study, we evaluated the prognostic value of hemogram-derived systemic inflammatory indices—SIRI, SII, and WMR—in predicting early mortality among patients with acute ischemic stroke. Our findings demonstrate that all three indices were significantly higher in non-survivors and were strongly associated with greater neurological severity at admission. In multivariable analysis, SIRI and SII remained independent predictors of in-hospital mortality, while WMR showed a complex but significant relationship after adjustment. These results support the growing body of evidence suggesting that systemic inflammation is closely linked to early deterioration and mortality in ischemic stroke.[1]

The inflammatory cascade following cerebral ischemia contributes to secondary neuronal injury, blood–brain barrier disruption, and systemic immune activation. Therefore, markers capturing both innate and adaptive immune responses may offer valuable prognostic information. Our results are in line with previous studies reporting that SII and SIRI are associated with stroke severity, early neurological worsening, and poor functional outcomes.[3-5] In particular, we found moderate correlations between these markers and NIHSS and mRS scores at admission, reinforcing earlier observations that higher inflammatory burden reflects more profound neurological impairment.[3,4]

SIRI emerged as one of the strongest predictors in our analysis. Elevated SIRI reflects a combination of neutrophilia and monocytosis—both hallmarks of acute inflammatory activation—together with lymphopenia, which indicates immune dysregulation. This combination has previously been shown to predict early complications and adverse outcomes in mild and moderate ischemic stroke.[10] The AUC value for SIRI in our study (0.76) is comparable with earlier reports and indicates good discriminatory ability for short-term mortality.

SII also demonstrated a strong independent association with in-hospital mortality, with the highest odds ratio among all variables included in the multivariable model. SII combines neutrophil and platelet counts, both of which are known contributors to thrombus formation, microvascular obstruction, and ischemic tissue damage. Prior studies have shown that higher SII values correlate with increased stroke severity and unfavorable 90-day outcomes, particularly in patients treated with intravenous thrombolysis.[2] Our findings extend these observations by showing that SII is also a powerful marker for early mortality, even in a heterogeneous acute stroke population.

The prognostic value of WMR has been investigated less frequently compared to SIRI and SII. In our cohort, non-survivors had significantly higher WMR values, and WMR correlated with all clinical severity scores. Although its AUC was lower than that of SIRI and SII, WMR showed high specificity for mortality. Interestingly, the direction of association changed in the multivariable model, likely due to interactions with platelet count and other inflammatory markers. Similar inconsistencies have been reported in the literature, highlighting the need for further research into the biological mechanisms underlying WMR changes in acute stroke.[6]

Another important observation is that the non-survivor group exhibited pronounced systemic inflammation overall, with markedly elevated CRP, procalcitonin, D-dimer, fibrinogen, urea, creatinine, and neutrophil counts, along with reduced albumin and lymphocyte levels. This constellation reflects a state of acute physiological stress and systemic decompensation that likely contributes to poor outcomes. Previous studies have emphasized that inflammation not only aggravates ischemic injury but also increases the risk of secondary complications such as infection and organ dysfunction.[7,8] The strong associations we observed between inflammatory indices and neurological severity further reinforce this pathophysiological link.

The clinical implications of our findings are noteworthy. SIRI, SII, and WMR can be calculated rapidly and inexpensively upon admission using routine laboratory tests. Their integration into early risk stratification models may help identify high-risk patients who require closer monitoring

or more aggressive management, particularly in settings where advanced imaging or biomarker assays are not readily available. Moreover, these indices may complement established scoring systems, such as NIHSS and mRS, by providing additional insight into the systemic inflammatory status of the patient.

This study has several limitations. First, its retrospective design may introduce bias related to data completeness and patient selection. Second, the analysis was based on a single-center cohort, and external validation is needed before generalizing the findings. Third, we evaluated inflammatory markers only at baseline; dynamic changes over time may provide additional prognostic information that was not captured in this study. Despite these limitations, the study benefits from a well-characterized dataset and comprehensive statistical analysis.

In conclusion, our results demonstrate that SIRI, SII, and WMR are valuable and accessible biomarkers for predicting early mortality in acute ischemic stroke. Their strong associations with neurological severity and adverse outcomes underscore the importance of systemic inflammation in stroke pathophysiology. Incorporating these indices into clinical decision-making may improve early identification of high-risk patients and enhance individualized care strategies.

DISCLOSURE

Ethics: This study was approved by the Gaziantep City Hospital Non-Interventional Clinical Research Ethics Committee.

Data availability: The anonymized dataset supporting the conclusions of this study is available from the corresponding author upon reasonable request.

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Conflict of interest: None

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