Turkish Journal of Neurology

Turk J Neurol 2025;31(1):34-40
Doi: 10.55697/tnd.2025.232

ORIGINAL ARTICLE

Platelet-albumin-bilirubin score and systemic immune-inflammation index for assessing the prognosis of acute ischemic stroke patients

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ABSTRACT

Objectives: This study investigated the prognostic value of the systemic immune-inflammation index (SII) and platelet-albumin-bilirubin (PALBI) score in patients with acute ischemic stroke (AIS).

Patients and methods: A total of 211 patients (120 males, 91 females; mean age: 70.9±13.2 years; range, 25 to 98 years) with AIS and 145 controls (72 males, 73 females; mean age: 69.4±8.4 years; range, 53 to 88 years) were included in this prospective study between March 2023 and December 2023. Demographic data and laboratory results upon admission were recorded. The SII and PALBI scores were calculated from laboratory parameters.

Results: The SII was significantly higher in the stroke group compared to controls (p<0.001), while PALBI scores were similar between the groups (p=0.169). According to the TOAST (Trial of ORG 10172 in Acute Stroke Treatment) classification, SII was comparable across the subgroups (p=0.964), but PALBI scores were significantly higher in the cardioembolic group (p=0.002). In terms of stroke severity, SII showed no significant differences between subgroups (p=0.701); however, patients with moderate-to-severe stroke had significantly higher PALBI scores than those with minor stroke (p=0.008). Both SII (p=0.007) and PALBI scores (p=0.019) were significantly elevated in AIS patients with stroke-related complications.

Conclusion: A higher PALBI score was associated with stroke severity, and both PALBI and SII values were elevated in AIS patients with complications, indicating a poor prognosis.

Keywords: Acute ischemic stroke, platelet-albumin-bilirubin, prognosis, stroke severity, systemic immune-inflammation index.

Stroke is a leading cause of disability and the second most common cause of mortality worldwide.[1,2] Approximately 87% of all strokes are ischemic in nature.[3] During the initial phase of acute ischemic stroke (AIS), excessive oxidative stress induces both structural and functional brain injury, playing a critical role in the progression of brain damage.[4] Following ischemic injury, damage-associated molecular patterns activate local inflammatory cells, leading to the production of inflammatory cytokines and chemokines.[5] These inflammatory triggers cause the differentiation of microglia and astrocytes, breakdown of the blood-brain barrier, and migration of peripheral immune cells, including neutrophils, T cells, and platelets, into the central nervous system, thereby eliciting an immune response.[6]

Although clinical scales and neuroimaging are commonly used to predict stroke severity in daily practice, the need for reliable prognostic markers has grown, particularly for monitoring patient follow-up. One such marker is the systemic immune-inflammation index (SII), which is based on the peripheral platelet and white blood cell

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Received: June 04, 2024 **Accepted:** November 03, 2024 **Published online:** March 05, 2025
Cite this article as: Duran S, Celikbilek A, Yetis A, Sahin BE, Koysuren A, Bolacalı M, et al. Platelet-albumin-bilirubin score and systemic immune-inflammation index for assessing the prognosis of acute ischemic stroke patients. Turk J Neurol 2025;31(1):34-40. doi: 10.55697/tnd.2025.232.

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counts.[7] The SII is a novel biomarker that reflects the balance between the body's inflammatory and immune responses,[8] providing valuable insights into thrombus formation, inflammatory activity, and adaptive immune responses.[9] Recent studies demonstrated that higher SII levels were associated with worse outcomes in AIS patients.[8,9]

Another candidate biomarker is the platelet-albumin-bilirubin (PALBI) score, which is derived from the albumin-bilirubin (ALBI) score.[10] Serum bilirubin, a product of heme catabolism, is widely used as a diagnostic marker for liver, bile, and blood disorders.[11] Bilirubin functions as a potent endogenous antioxidant, but there is conflicting evidence regarding its role as either an antioxidant or an oxidant under pathological conditions. Song et al.[11] reported that elevated bilirubin levels were positively correlated with stroke severity in AIS patients, mainly due to oxidative stress. Conversely, Duan et al.[12] found that higher bilirubin levels following AIS were associated with favorable outcomes.

Serum albumin, a protein critical for maintaining plasma oncotic pressure and volume, also serves as a potent antioxidant and anti-inflammatory agent.[13] Studies demonstrated that higher albumin levels were linked to better outcomes and lower mortality rates in AIS patients.[14] However, to date, only one study has examined both the SII and PALBI scores in AIS patients and found that SII was independently associated with stroke severity, whereas PALBI scores were not.[15] The present study aimed to investigate the prognostic value of SII and PALBI scores, both of which are markers of inflammation, in AIS patients.

PATIENTS AND METHODS

This prospective case-control study included 211 patients (120 males, 91 females; mean age: 70.9±13.2 years; range, 25 to 98 years) with AIS and 145 age- and sex-matched controls (72 males, 73 females; mean age: 69.4±8.4 years; range, 53 to 88 years). The study was conducted at the Department of Neurology, Kırşehir Ahi Evran University Faculty of Medicine, between March 2023 and December 2023. Inclusion criteria were age ≥18 years, symptom presentation within 24 h, and confirmation of ischemic lesions by diffusion-weighted magnetic resonance imaging or computed tomography. Exclusion criteria included the presence of transient ischemic attack, intracerebral hemorrhage, a history of rheumatologic, hematologic, or oncologic disease, hepatobiliary system disease, infection within the last 15 days, and the use of anti-inflammatory drugs. The study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Kırşehir Ahi Evran University Faculty of Medicine Clinical Research Ethics Committee (date: 12.02.2023, no: 2023-04/26). Written informed consent was obtained from all participants.

Data, including age, sex, comorbidities, medications, National Institutes of Health Stroke Scale (NIHSS) scores on admission, duration of hospitalization, and stroke-related complications (e.g., intracerebral hemorrhage, recurrent stroke, or death), were recorded. Stroke severity was assessed using NIHSS scores, where a score >5 indicated moderate-to-severe stroke, and a score of ≤5 indicated minor stroke.[15] Stroke etiology was classified according to the TOAST (Trial of ORG 10172 in Acute Stroke Treatment) classification into four categories: large-artery atherosclerosis, cardioembolic stroke, small-vessel occlusion, and others.[16] Carotid and vertebral arteries were evaluated using Doppler ultrasonography and categorized into four groups based on the degree of stenosis: <50%, 50-69%, ≥70%, or no stenosis. Patients with atrial fibrillation on the admission electrocardiogram or paroxysmal atrial fibrillation detected on 24-h Holter monitoring were considered to have atrial fibrillation. Control subjects, who met the exclusion criteria, were selected from neurology outpatient clinics and were matched for age and sex.

Fasting venous blood samples were collected from all participants within 24 h of admission by trained nurses. Blood samples were stored at 4°C and analyzed by certified clinical laboratory technicians within 2 h of collection at a hospital-certified laboratory. Complete blood counts were performed using an LH 750 automated hematology analyzer (Beckman Coulter, Fullerton, CA, USA), and white blood cell, neutrophil, lymphocyte, and platelet counts were recorded. Serum albumin, total bilirubin, and direct bilirubin levels were measured using a Cobas c702 autoanalyzer system (Roche Diagnostics, Tokyo, Japan).

The SII was calculated using the following formula: $P \times N/L$, where P, N, and L represent platelet, neutrophil, and lymphocyte counts, respectively.[17] The PALBI score was calculated using

TABLE 1
Baseline data in control subjects and patients with acute ischemic stroke (n=356)

	Controls (n=145)					Stroke (n=211)					
	n	%	Mean±SD	Median	Min-Max	n	%	Mean±SD	Median	Min-Max	*p*
Age (year)			69.4±8.4					70.9±13.2			0.169
Sex											
Female	73	50.3				91	43.1				0.180
Comorbidities											
Hypertension	90	62.1				137	64.9				0.581
Diabetes mellitus	48	33.1				89	42.2				0.083
Coronary artery disease	22	15.2				50	23.7				**0.049**
Atrial fibrillation	2	1.4				20	9.5				**0.002**
History of stroke	0	0				49	9.5				**<0.001**
Hyperlipidemia	18	12.4				58	27.5				**<0.001**
Chronic renal failure	0	0				16	7.6				**<0.001**
Laboratory											
Albumin (g/L)			44.09±2.97					42.14±3.55			**<0.001**
White blood cells (10^9/L)				7.3	6.1-8.59				8.84	7.18-10.58	**<0.001**
Neutrophil (10^9/L)				4.05	3.21-5.2				5.48	4.28-7.12	**<0.001**
Lymphocyte (10^9/L)			2.32±0.75					2.18±1.02			0.154
Platelet (10^9/L)			252.34±63.99					265.52±78.34			0.083
Direct bilirubin (µmol/L)				3.42	2.31-4.45				2.57	1.71-3.76	**<0.001**
Total bilirubin (µmol/L)				8.55	6.58-11.97				7.01	5.3-10.95	0.091
PALBI score			–2.79±0.32					–2.73±0.37			0.169
SII (10^9/L)				460.21	331.3-605.98				675.07	437.13-1086.46	**<0.001**

SD: Standard deviation; PALBI: platelet-albumin-bilirubin; SII: Systemic immune-inflammation index.

the following formula:[18] 2.02 × ($\log_{10}$ bilirubin) – 0.37 × ($\log_{10}$ bilirubin)^2 – 0.04 × albumin – 3.48 × ($\log_{10}$ P) + 1.01 × ($\log_{10}$ P)^2.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 29.0 software (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess normality, and Levene's test was used to evaluate variance homogeneity. Continuous variables were compared using independent samples t-test, one-way analysis of variance, the Kruskal-Wallis test, or the Mann-Whitney U test, as appropriate. The chi-square test was used for categorical variables. Descriptive statistics were presented as means with standard deviations (SD) or as medians with interquartile ranges (IOR). Spearman's rank correlation was used to analyze relationships between continuous variables. Receiver operating characteristic curve and area under the curve analyses were performed to evaluate the predictive value of the PALBI score for stroke severity. A p-value of <0.05 was considered statistically significant.

RESULTS

No significant differences were observed in age or sex between patients with AIS and controls (p>0.05, Table 1). Patients with AIS had a significantly higher prevalence of comorbidities compared to controls (p<0.001, Table 1). The mean NIHSS score at admission for patients with AIS was 3.54±4.34. In the stroke group, six (2.8%) patients received thrombolytic therapy, and the median hospitalization duration was 7 days (IOR, 5-10 days). According to the TOAST classification, 74 (37.07%) patients were classified as having large-artery atherosclerosis, 64 (30.33%) as cardioembolism, 44 (20.85%) as small-vessel occlusion, and 29 (13.74%) as other types of stroke (Table 2). Stroke severity was categorized into mild (NIHSS ≤5) in 154 (72.99%) patients and moderate-to-severe (NIHSS >5) in 57 (27.01%) patients (Table 2). Stroke-related complications occurred in 11 (7.5%) patients, including four (2.7%) patients with hemorrhage, one (0.7%) with recurrent stroke, and six (4.1%) who died (Table 2).

White blood cells, neutrophils, and SII were significantly higher in patients with AIS compared to the control group (p<0.001, Table 1). Albumin and direct bilirubin levels were significantly lower in patients with AIS (p<0.001, Table 1). However, the PALBI score did not differ significantly between the AIS and control groups (p=0.169, Table 1).

When analyzing stroke subtypes based on the TOAST classification, the SII was similar across subgroups (p=0.964), but the PALBI score was significantly higher in the cardioembolic stroke group (p=0.002, Table 2). In terms of stroke severity, there was no significant difference in SII between patients with mild stroke and those with moderate-to-severe stroke (p=0.701). However, PALBI scores were significantly higher in patients

TABLE 2
The PALBI score and SII regarding TOAST classification and stroke severity in patients with acute ischemic stroke (n=211)

		PALBI score		SII		
	n	Mean±SD	*p*	Median	Min-Max	*p*
TOAST						
Large-artery atherosclerosis	74	–2.69±0.41		682.51	122.85-6832.3	
Cardioembolism	64	–2.65±0.32	0.002	611.92	104.36-11938	0.964
Small-vessel occlusion	44	–2.89±0.31		741.29	208.02-2218.14	
Other	29	–2.82±0.36		605.28	277.71-2068.28	
Stroke severity						
Mild	154	–2.77±0.34	0.008	666.92	452.93-1042.7	0.701
Moderate to severe	57	–2.63±0.41		717.77	370.23-1459.18	
Stroke-related complication						
Present	11	–2.48±0.48	0.019	791.35	476.5-1284.60	**0.007**
Absent	200	–2.75±0.35		657.36	431.93-1057.53	

PALBI: Platelet-albumin-bilirubin; SII: Systemic immune-inflammation index; TOAST: Trial of ORG 10172 in acute stroke treatment.

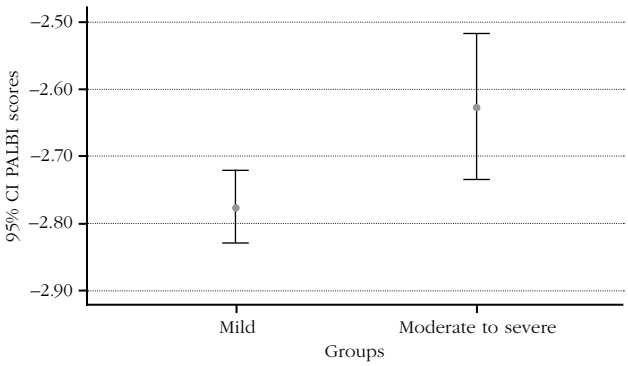

Figure 1. Platelet-albumin-bilirubin scores in patients with acute ischemic stroke.

CI: Confidence interval; PALBI: Platelet-albumin-bilirubin.

with moderate-to-severe stroke compared to those with minor stroke (p=0.008, Table 2, Figure 1). Additionally, both SII (p=0.007) and PALBI scores (p=0.019) were significantly elevated in patients with AIS who experienced stroke-related complications (Table 2).

A weak positive correlation was observed between the duration of hospitalization and the PALBI score (r=0.19, p=0.006), while no significant correlation was found between hospitalization duration and SII (p=0.451). Receiver operating characteristic analysis showed that the PALBI score had an area under the curve of 0.605 (95% confidence interval: 0.518-0.692, p=0.019), indicating that it did not identify a reliable cutoff value for predicting stroke severity.

DISCUSSION

Brain ischemia triggers a secondary neuroinflammatory response that helps clear damaged tissue and initiate the repair process.[19] This neuroinflammation is driven by resident immune cells, and involves neutrophils, macrophages, and T lymphocytes that infiltrate from the peripheral immune system.[20] Neutrophils, upon reaching the ischemic tissue, release inflammatory mediators that contribute to necrosis and apoptosis.[7,20] Leukocytes penetrate the compromised blood-brain barrier, which can lead to complications such as cerebral edema. Additionally, ischemia-reperfusion injury in the brain promotes platelet necrosis, which regulates harmful neutrophil recruitment and the formation of platelet-neutrophil aggregates, further reducing cerebral blood flow.[7] Several biomarkers that reflect systemic inflammation, including the neutrophil-to-lymphocyte ratio, have been identified as predictors of prognosis in AIS.[20,21]

In recent years, the SII has been investigated in various conditions, including cancer, acute coronary syndromes, and neurosurgery.[22] The SII may reflect underlying mechanisms of stroke, including thrombus formation, inflammatory responses, and adaptive immune pathways.[9] Some studies suggested that SII could play a predictive role in determining the prognosis of ischemic stroke.[8,9] Wang et al.,[8] in a study involving 9,107 patients from the China National Stroke Registry III, reported that higher SII levels were associated with poor short- and long-term outcomes in patients with AIS. A recent study by Huang[9] demonstrated a significant association between elevated SII and poor outcomes in patients with AIS (adjusted odds ratio [OR]=2.350, p=0.019). Similarly, Hou et al.[15] found that SII was independently associated with stroke severity (OR=1.351, p=0.007) after adjusting for confounders.

In our study, SII levels were higher in patients with AIS compared to the control group. However, we did not find an association between SII and stroke severity, which may be attributed to the fact that the majority of our patients had mild stroke. However, SII levels were elevated in patients with AIS who experienced stroke-related complications, supporting current evidence that higher SII values are associated with poorer outcomes.[7]

The PALBI score, another simple clinical parameter, was initially developed to assess liver functional reserve and has been used to predict outcomes in hepatocellular carcinoma.[18] The PALBI score is calculated based on platelet count, albumin levels, and bilirubin levels. Although bilirubin has historically been considered a harmful metabolic byproduct, recent research suggests it acts as a potent antioxidant and neuroprotective agent.[23] Conflicting results have emerged regarding the role of bilirubin in the prognosis of AIS,[11,12] leading to greater interest in derived parameters, such as the PALBI score. Bolayır et al.[24] found that the PALBI score was lower in patients with intracerebral hemorrhage compared to controls, and it decreased further with increasing hemorrhage severity both in the acute phase and at 30 days, making it an independent predictor of 30-day intracerebral hemorrhage mortality.

Data on the role of the PALBI score in AIS are limited. In contrast to our findings, a study by Hou et al.[15] did not identify significant differences in PALBI scores between patients with mild and moderate-to-severe strokes. Our study, however, revealed significantly higher PALBI scores in patients with moderate-to-severe stroke compared to those with mild stroke. Additionally, elevated PALBI scores were observed in patients with stroke-related complications, indicating a potential association with poor outcomes. This supports the notion that an elevated PALBI score may reflect ongoing neuroinflammation in AIS patients.

Pektezel et al.[10] examined the PALBI score before and 24 h after thrombolytic therapy in AIS patients and found that it was not useful in predicting the efficacy or side effects of thrombolytic treatment. Our study, however, revealed a weak positive correlation between the PALBI score and the duration of hospitalization, suggesting that higher PALBI scores may be associated with longer hospital stays in AIS patients. However, the receiver operating characteristic analysis did not identify a reliable cutoff value to predict stroke severity.

Interestingly, the PALBI score's predecessor, the ALBI score, was associated with various cardiac pathologies, including atrial septal defect and rheumatic mitral stenosis, both of which are risk factors for AIS.[25,26] Evlice et al.[25] demonstrated that a high ALBI score, indicative of liver dysfunction, could result from hypoxia secondary to heart disease. In our study, we observed significantly higher PALBI scores in patients with cardioembolic stroke, suggesting that the PALBI score may be a novel marker meriting further investigation in future studies.

This study had some limitations. First, the relatively small number of patients may limit the generalizability of the findings. Larger cohorts are necessary to confirm these results. Second, the majority of AIS patients in this study had mild stroke, which may not be representative of more severe cases. This imbalance could have influenced the results, particularly regarding correlations with stroke severity. Third, key inflammatory markers, such as tumor necrosis factor-alpha, interleukin-1, and interleukin-6, were not included in the analysis. These markers could provide a more comprehensive understanding of the inflammatory mechanisms underlying stroke. Moreover, the study relied on a single blood sample taken within the first 24 h of symptom onset. This may not fully reflect the patient's inflammatory status over time, potentially affecting the accuracy of SII and PALBI score as prognostic markers. Finally, differences in comorbid conditions between the stroke and control groups, such as a higher prevalence of coronary artery disease and atrial fibrillation in the stroke group, could have introduced bias, influencing the study outcomes.

In conclusion, the findings indicated that a high PALBI score was associated with stroke severity, and both PALBI scores and SII values were elevated in AIS patients with complications, suggesting a poor prognosis. Further studies are needed to validate these findings and explore the prognostic value of these markers in more diverse patient populations.

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

Author Contributions: Idea/concept: S.D.; Design, control/supervision, analysis and/or interpretation, writing the article, materials: S.D., A.C.; Data collection and/or processing, literature review: S.D., A.Y., B.E.S., A.K., M.B., B.P.; References and fundings: S.D., A.C., A.Y., B.E.S., A.K.; Other: S.D., M.B., B.P.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

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