

Association Between Systemic Immune-Inflammation Index and in-Hospital and Long-Term All-Cause Mortality in Patients with Type 2 Diabetes Mellitus Complicated by Spontaneous Intracerebral Hemorrhage

Yongze Shen^{1,*}, Aili Gao^{2,*}, Yingjie Shen¹, Shang Gao¹, Yiwei Rong¹, Zhaoxin Fan¹, Qi Li¹, Tie Lin¹, Shanshan Yang³, Hongsheng Liang¹

¹Department of Neurosurgery, The First Affiliated Hospital of Harbin Medical University, Harbin, 150001, People's Republic of China; ²School of Life Science, Northeast Agricultural University, Harbin, 150006, People's Republic of China; ³Department of Neurology, The First Affiliated Hospital of Harbin Medical University, Harbin, 150001, People's Republic of China

*These authors contributed equally to this work

Correspondence: Shanshan Yang; Hongsheng Liang, Email yangshanshan81@hrbmu.edu.cn; lianghongsheng@hrbmu.edu.cn

Objective: Patients with type 2 diabetes mellitus (T2DM) and spontaneous intracerebral hemorrhage (sICH) face high mortality. Systemic inflammation may play a key role, but the prognostic value of the systemic immune-inflammation index (SII) in this population remains unclear. This study aimed to investigate the association between SII and all-cause mortality in T2DM with sICH.

Methods: A retrospective cohort study was conducted on 891 patients with T2DM and sICH hospitalized at the First Affiliated Hospital of Harbin Medical University from January 2021 to December 2024. SII was calculated using the formula: platelet count × neutrophil count/lymphocyte count. Patients were categorized into high and low SII groups based on the median value (817.5). Kaplan-Meier survival analysis, multivariate logistic and Cox regression models were used to assess the association of SII with in-hospital and long-term mortality. Subgroup and sensitivity analyses were performed to test the robustness of findings. Receiver operating characteristic (ROC) curves further evaluated predictive performance.

Results: In fully adjusted models, high SII was independently associated with a significantly increased risk of in-hospital mortality (OR = 3.122, 95% CI: 1.759–5.544, $P < 0.001$) and long-term all-cause mortality (median follow-up 20.6 months, HR = 2.755, 95% CI: 1.938–3.919, $P < 0.001$). Each 1-standard deviation increase in SII was linked to a 75.9% higher in-hospital death risk (OR = 1.759), and a 17.0% increase in long-term mortality risk (HR = 1.170). ROC analysis demonstrated moderate discriminative ability for SII (AUC = 0.722 for in-hospital mortality, AUC = 0.748 for long-term mortality). Kaplan-Meier analysis revealed significantly poorer survival in the high SII group over a 50-month follow-up (log-rank $P = 0.015$). These associations remained stable across subgroups and after adjusting for confounding comorbidities and clinical parameters.

Conclusion: Elevated SII is an independent predictor of both in-hospital and long-term all-cause mortality in patients with T2DM and sICH. This may be because high SII reflects an imbalance of inflammation and immunity, leading to increased mortality risk.

Keywords: systemic immune-inflammation index, type 2 diabetes mellitus, spontaneous intracerebral hemorrhage, mortality, prognostic biomarker

Introduction

Spontaneous intracerebral hemorrhage (sICH) refers to a non-traumatic rupture of blood vessels within the brain parenchyma, leading to the accumulation of blood in the brain tissue. It is an acute cerebrovascular disease typically presenting with sudden severe headache, frequent vomiting, and varying degrees of consciousness disturbance, often

accompanied by neurological deficits such as hemiplegia and aphasia.¹ sICH is characterized by rapid onset, severe clinical presentation, fast progression, high disability rate, and high mortality rate. It is one of the leading causes of stroke-related death and disability worldwide.² Treatment primarily focuses on preventing secondary brain injury [eg, hematoma expansion, cerebral edema, intraventricular hemorrhage (IVH)], promoting neurological recovery, and minimizing complications. Although sICH accounts for only 10%–15% of all strokes, its 30-day mortality rate reaches 30%–50%, significantly higher than that of ischemic stroke (IS).³ Among survivors, about 80% experience varying degrees of functional impairment, severely affecting their quality of life.⁴ In China, the annual incidence of sICH is 50–80 per 100,000 people, much higher than in Western countries (10–15 per 100,000),⁵ with higher incidence rates observed in northern regions and during winter, possibly due to cold-induced vasoconstriction and poor hypertension control.⁶ The main risk factors for sICH include hypertension, cerebral atherosclerosis, cerebrovascular structural abnormalities (eg, arteriovenous malformations, intracranial aneurysms), coagulation disorders (eg, thrombocytopenia, coagulation factor deficiency, or long-term use of anticoagulants/antiplatelet agents), unhealthy lifestyle habits (eg, smoking, alcohol consumption), metabolic diseases (eg, diabetes, hyperlipidemia), age, sex, and genetic factors.⁷

Among these, type 2 diabetes mellitus (T2DM), a common chronic metabolic disease, is closely associated with the occurrence, progression, and prognosis of sICH. First, T2DM increases the risk of sICH. Chronic hyperglycemia leads to metabolic disorders, vascular endothelial damage, activation of oxidative stress and inflammatory pathways, resulting in hyaline degeneration and atherosclerosis of small vessels. These changes weaken vessel walls, making them more prone to rupture during blood pressure fluctuations.^{8,9} Furthermore, in a hyperglycemic state, platelet activity increases, red blood cell deformability decreases, and fibrinolysis is inhibited, resulting in a hypercoagulable state and further increasing the risk of cerebral vessel rupture.¹⁰ Second, T2DM exacerbates secondary injury after sICH. Hyperglycemia intensifies oxidative stress and promotes the release of inflammatory mediators, leading to additional neuronal damage. High osmolarity causes cerebral edema, and elevated blood viscosity reduces cerebral perfusion, worsening ischemia and hypoxia, thereby accelerating disease progression.¹¹ Third, T2DM patients have lower immunity and are more susceptible to complications such as pulmonary and urinary tract infections, which impair recovery and increase mortality. Surgical patients may also experience delayed wound healing, while peripheral neuropathy due to chronic hyperglycemia affects the rehabilitation process, leading to poor neurological recovery. Given that recent global data estimate over 529 million people are currently living with diabetes—96% of whom have T2DM—and project that this number will exceed 1.3 billion by 2050, mainly driven by rising obesity, the urgency to explore early prognostic markers in high-risk populations such as patients with T2DM and sICH is increasingly evident.¹² Thus, identifying risk factors for poor outcomes in patients with T2DM and sICH is crucial for reducing premature death and improving survival and quality of life.

In recent years, the central role of inflammation in the pathophysiology of intracerebral hemorrhage has attracted increasing attention, particularly in the context of metabolic diseases such as T2DM. After brain hemorrhage, immune-inflammatory responses are rapidly activated in the perihematomal region, releasing a large number of inflammatory mediators that trigger neuronal apoptosis and blood-brain barrier (BBB) disruption. T2DM, being a chronic low-grade inflammatory condition, can further amplify these effects. Therefore, the assessment of inflammatory markers is essential for evaluating disease progression and prognosis. Traditional inflammatory blood markers—such as white blood cell count (WBC), neutrophil count, lymphocyte count, monocyte count, C-reactive protein (CRP), and cytokines like interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and interleukin-1 beta (IL-1 β)—have been widely used in clinical inflammation monitoring and stroke prognosis analysis.^{13,14} However, these indicators often suffer from time sensitivity, high variability, and limited assessment scope, making them inadequate for reflecting the overall systemic immune-inflammatory status.

To overcome these limitations, several composite inflammatory scores have been proposed, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and lymphocyte-to-monocyte ratio (LMR). Among them, the systemic immune-inflammation index (SII)—calculated as platelet count \times neutrophil count/lymphocyte count—has gained attention for its ability to integrate three types of immune cells simultaneously, reflecting both pro-inflammatory and immune-suppressive processes in a single metric.¹⁵ Compared to NLR and PLR, SII incorporates platelet levels, which play an important role in thromboinflammation and secondary brain injury following hemorrhage, thus offering

better biological plausibility and broader clinical relevance. In cardiovascular diseases (CVD), SII has not only been shown to be significantly associated with the risk of major adverse cardiovascular and cerebrovascular events (MACCE) in patients with coronary artery disease (CAD), but also demonstrated superior predictive performance compared to CRP and other traditional inflammatory markers, independently predicting the occurrence of MACCE in CAD patients.^{16,17} In neurological conditions, SII has been validated as a predictor for delayed cerebral vasospasm after aneurysmal subarachnoid hemorrhage (aSAH).¹⁸ Besides, Yang et al, in a study including 379 patients with acute ischemic stroke due to large vessel occlusion (AIS-LVO) who underwent endovascular treatment (EVT), demonstrated that a higher admission SII was independently associated with an increased risk of sICH, with the risk rising in a dose-dependent manner—specifically, every 10-unit increase in SII was linked to greater odds of sICH, and patients in the highest SII tertile had a significantly elevated risk compared to those in the lowest tertile.¹⁹ Furthermore, Cao et al retrospectively analyzed 482 patients with AIS-LVO who underwent successful EVT, and found that SII on the first postoperative day was an independent predictor of both poor 90-day functional outcome and sICH.²⁰ These findings suggest that SII may outperform other composite indices by capturing broader aspects of the systemic inflammatory response. However, current studies have not systematically evaluated the role of SII in predicting in-hospital and long-term all-cause mortality among patients with sICH complicated by T2DM. Moreover, its independent predictive significance in this comorbid, high-risk population remains unclear.

Therefore, this study aims to investigate the prognostic value of SII in patients with T2DM complicated by sICH, specifically focusing on its association with in-hospital and long-term all-cause mortality. Clarifying this relationship may help establish SII as a reliable biomarker for early risk stratification and personalized intervention strategies in clinical practice.

Methods

Study Population

This study adopted a retrospective cohort design. The study population consisted of patients with T2DM complicated by sICH who were hospitalized at the First Affiliated Hospital of Harbin Medical University between January 1, 2021, and December 31, 2024. After an initial screening of 915 cases, a total of 891 patients were ultimately included in the final analysis based on the inclusion and exclusion criteria. This study protocol complies with the Declaration of Helsinki and was reviewed and approved by the First Affiliated Hospital of Harbin Medical University. Written informed consent was obtained from all patients.

Inclusion and Exclusion Criteria

Inclusion criteria were as follows: (1) Definite diagnosis of sICH, meeting the diagnostic criteria of the Chinese Stroke Association guidelines;²¹ (2) Diagnosis of T2DM in accordance with the 2020 criteria of the American Diabetes Association (ADA), including fasting blood glucose (FBG) ≥ 7.0 mmol/L, 2-hour postprandial blood glucose ≥ 11.1 mmol/L, or hemoglobin A1c (HbA1c) $\geq 6.5\%$;²² (3) All laboratory parameters were collected within 24 hours of hospital admission; (4) Age ≥ 18 years; (5) Complete availability of clinical data, imaging studies, and laboratory results.

Exclusion criteria were as follows: (1) Hemorrhage secondary to trauma, arteriovenous malformation, tumor, aneurysm, moyamoya disease, or other non-spontaneous causes; (2) History of anticoagulant (eg, warfarin), antiplatelet (eg, aspirin), or immunosuppressant use prior to admission, or known coagulation disorders; (3) Presence of severe systemic diseases [eg, malignancy, heart failure (HF), chronic infection, or immune system disorders] that may affect prognostic evaluation; (4) Incomplete or missing follow-up data.

Data Collection

All baseline information was collected and organized from the hospital's electronic medical record system. This included demographic data (such as age and sex), lifestyle factors, medical history, medication use, and comorbidities. A family history of cerebrovascular disease was defined as having a first-degree relative with either intracerebral hemorrhage or IS. Smoking history was defined as current or former smoking with a cumulative duration of more than six months, while alcohol consumption was defined as drinking alcohol at least once per week on average.

Hypertension was diagnosed according to the 2024 Revised Chinese Guidelines for the Prevention and Treatment of Hypertension, which define hypertension as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg on at least three non-consecutive measurements, or a previously confirmed diagnosis.²³ Hyperlipidemia was defined based on the 2016 Revised Chinese Guidelines for the Prevention and Treatment of Dyslipidemia in Adults, with diagnostic thresholds including total cholesterol (TC) ≥ 6.2 mmol/L, triglycerides ≥ 2.3 mmol/L, or low-density lipoprotein cholesterol (LDL-C) ≥ 4.1 mmol/L.²⁴ T2DM was diagnosed according to the 2020 criteria of the ADA, defined as FBG ≥ 7.0 mmol/L, 2-hour plasma glucose during an oral glucose tolerance test ≥ 11.1 mmol/L, or HbA1c $\geq 6.5\%$.²² Coronary heart disease (CHD) was defined as myocardial ischemia or infarction confirmed by imaging or electrocardiography.²⁵ IS referred to prior cerebral infarction or transient ischemic attack confirmed by imaging. Atrial fibrillation (AF) was diagnosed by electrocardiogram or Holter monitoring and classified as either persistent or paroxysmal. HF was diagnosed based on clinical presentation and echocardiographic findings, including reduced ejection fraction or diastolic dysfunction.²⁶ Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m² lasting for more than three months.²⁷ A history of previous sICH was also documented to provide a comprehensive assessment of cerebrovascular risk.

Medication use included antihypertensive, lipid-lowering, and hypoglycemic drugs. Antihypertensive medications included calcium channel blockers (CCB), angiotensin-converting enzyme inhibitors (ACEI), or angiotensin receptor blockers (ARB), diuretics, and beta-blockers. Lipid-lowering drugs primarily included statins and fibrates. Hypoglycemic therapy included oral antidiabetic drugs (such as metformin and sulfonylureas) and insulin injections.

Clinical data included body mass index (BMI), calculated as weight (kg) divided by the square of height (m²), and blood pressure measurements (SBP and DBP) recorded in mmHg at the time of admission. Neurological function was evaluated using the Glasgow Coma Scale (GCS) for consciousness and the Modified Rankin Scale (mRS) for functional disability.²⁸ The presence of hemiplegia and vomiting at admission was determined from physical examination and nursing records. Levels of consciousness were categorized into five grades: alert, drowsy, confused, stuporous, and comatose. Time from onset to admission (TFA) was recorded in hours.

Hematoma volume was calculated using the ABC/2 formula, where A is the longest diameter of the hematoma on the largest axial slice, B is the diameter perpendicular to A, and C is the number of slices with hemorrhage multiplied by the slice thickness in centimeters, then multiplied by 0.5.²⁹ Additional imaging data included the presence of midline shift, hemorrhage location (basal ganglia, thalamus, lobar, cerebellum, brainstem), intraventricular extension, and the presence of obstructive hydrocephalus. Treatment modality was categorized as either conservative or surgical, and intraoperative or postoperative complications—including rebleeding, reoperation, pulmonary infection, and seizures—were also recorded.

Laboratory parameters were collected within 24 hours of admission and included WBC, neutrophil count, lymphocyte count, monocyte count, hemoglobin, platelet count, FBG, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin (TB), albumin, uric acid (UA), blood urea nitrogen (BUN), creatinine, electrolytes (potassium, sodium, chloride, calcium, bicarbonate), fibrinogen, and D-dimer.

Assessment and Measurement of SII

In this study, all patients had venous blood samples collected within 24 hours of hospital admission. The samples were analyzed by the Clinical Laboratory Center of the First Affiliated Hospital of Harbin Medical University to obtain the parameters required for calculating the SII. The SII was calculated using the following formula: $SII = \text{neutrophil count} (\times 10^9/L) \times \text{platelet count} (\times 10^9/L) / \text{lymphocyte count} (\times 10^9/L)$.¹⁵

To comprehensively assess the predictive value of SII for all-cause mortality, the index was incorporated into regression analyses in three forms: as a continuous variable, a log-transformed variable (log₁₀ transformation), and a standardized variable (Z-score). Additionally, SII was analyzed as a categorical variable. Based on the median value of 817.5, patients were divided into two groups: a low SII group (SII < 817.5 , $n = 352$) and a high SII group (SII ≥ 817.5 , $n = 352$).

Follow-Up and Outcomes

Based on in-hospital mortality status, the overall study population was divided into two groups: the non-in-hospital death group ($n = 704$) and the in-hospital death group ($n = 187$). Patients in the non-in-hospital death group were followed up,

and follow-up data were obtained through telephone interviews, outpatient visits, and collaboration with community healthcare institutions. The starting point of follow-up was defined as the date of hospital discharge. Follow-up was conducted at 6 months, 1 year, and then annually thereafter to ensure standardized long-term outcome assessment. The final follow-up date was March 31, 2025. All-cause mortality was defined as death from any cause during the follow-up period. Finally, the non-in-hospital mortality group was further divided based on the occurrence of all-cause mortality into the non-all-cause mortality group ($n = 503$) and the all-cause mortality group ($n = 201$).

Statistical Analysis

All statistical analyses were performed using SPSS version 27.0, with a two-sided P -value < 0.05 considered statistically significant. Continuous variables with a normal distribution were expressed as mean \pm standard deviation, and comparisons between two groups were conducted using the independent samples t -test. Continuous variables not conforming to a normal distribution were presented as median and interquartile range, and comparisons were made using non-parametric tests. Categorical variables were expressed as frequencies and percentages, and comparisons between groups were performed using the chi-square test or Fisher's exact test, as appropriate.

Kaplan-Meier survival curves were plotted to compare long-term all-cause mortality between the high and low SII groups, and the Log rank test was used to assess the statistical significance of differences between survival curves. Univariate logistic regression analysis and univariate Cox regression analysis were used to identify variables with $P < 0.05$, which were subsequently included in multivariate logistic regression and multivariate Cox regression models. Three regression models were constructed to evaluate the association between SII—as a categorical variable and as a continuous variable—and both in-hospital mortality and all-cause mortality. To ensure the stability of the regression models, multicollinearity diagnostics were performed for all variables included in the multivariate logistic and Cox regression models. Variance inflation factors (VIFs) were calculated, and no significant multicollinearity was detected among the covariates in either model. Further, stratified analyses were performed using fully adjusted multivariate logistic regression and Cox regression models across various subgroups, including: sex, age, smoking status, alcohol consumption, presence of IS, presence of CHD, BMI ($< 28 \text{ kg/m}^2$ or $\geq 28 \text{ kg/m}^2$, based on the obesity threshold defined for Chinese adults), GCS (< 13 or ≥ 13 , based on the median GCS score), mRS (≤ 4 or > 4 , based on the median mRS score), presence of consciousness disturbance, hemorrhage in the basal ganglia, IVH, $\text{WBC} \leq 10 \times 10^9/\text{L}$, $\text{WBC} > 10 \times 10^9/\text{L}$, conservative treatment, surgical treatment, and presence or absence of pulmonary infection. These subgroup analyses aimed to further validate the stratified association between SII and both in-hospital and all-cause mortality. Additionally, robustness analyses were conducted using fully adjusted multivariate logistic and Cox regression models after excluding patients with hyperlipidemia, a history of sICH, CKD, HF, AF, hydrocephalus, postoperative rebleeding, reoperation, and seizures. Receiver operating characteristic (ROC) curve analysis was used to assess and compare the predictive value of SII, GCS, hematoma volume, and degree of midline shift for in-hospital mortality and long-term all-cause mortality.

Results

Baseline Characteristics Stratified by SII

As shown in Table 1, the overall study population was divided into two groups based on the SII level: the low SII group ($n = 445$) and the high SII group ($n = 446$). Compared with the low SII group, the high SII group exhibited higher levels of SBP, DBP, mRS score, hematoma volume, degree of midline shift, WBC, neutrophil count, monocyte count, platelet count, FBG, AST, albumin, and serum calcium ($P < 0.05$). Additionally, the high SII group had higher proportions of patients presenting with hemiplegia, stupor, coma, vomiting, IVH, and obstructive hydrocephalus ($P < 0.05$). Regarding treatment, the high SII group had a higher rate of surgical intervention ($P < 0.05$). In terms of postoperative complications, the high SII group had higher incidences of rebleeding, reoperation, pulmonary infection, and seizures ($P < 0.05$). However, compared with the low SII group, the high SII group had a lower mean age, a lower frequency of alcohol consumption, and lower levels of GCS score, lymphocyte count, serum chloride, and bicarbonate ($P < 0.05$).

Table 1 Baseline Characteristics Stratified by SII

Variables	Total Population	Low SII	High SII	P Value
N	891	445	446	
Age, years	61.65 ± 10.91	62.58 ± 10.79	60.73 ± 10.96	0.012
Male, n (%)	581 (65.21%)	288 (64.72%)	293 (65.70%)	0.760
Family history of cerebrovascular disease, n (%)	34 (3.82%)	22 (4.94%)	12 (2.69%)	0.079
Smoking, n (%)	183 (20.54%)	98 (22.02%)	85 (19.06%)	0.273
Alcohol consumption, n (%)	159 (17.85%)	92 (20.67%)	67 (15.02%)	0.028
Hypertension, n (%)	723 (81.14%)	356 (80.00%)	367 (82.29%)	0.383
Antihypertensive drugs, n (%)	705 (79.12%)	410 (92.13%)	406 (91.03%)	0.553
Hyperlipidemia, n (%)	16 (1.80%)	7 (1.57%)	9 (2.02%)	0.617
Lipid-lowering drugs, n (%)	18 (2.02%)	9 (2.02%)	9 (2.02%)	0.996
Antidiabetic drugs, n (%)	816 (91.58%)	410 (92.13%)	406 (91.03%)	0.553
CHD, n (%)	181 (20.31%)	92 (20.67%)	89 (19.96%)	0.790
Ischemic stroke, n (%)	223 (25.03%)	113 (25.39%)	110 (24.66%)	0.802
Atrial fibrillation, n (%)	26 (2.92%)	14 (3.15%)	12 (2.69%)	0.686
Heart failure, n (%)	53 (5.95%)	31 (6.97%)	22 (4.93%)	0.199
CKD, n (%)	74 (8.31%)	34 (7.64%)	40 (8.97%)	0.473
History of intracerebral hemorrhage, n (%)	72 (8.08%)	33 (7.42%)	39 (8.74%)	0.467
BMI, kg/m ²	26.08 ± 4.02	26.03 ± 4.23	26.13 ± 3.80	0.716
SBP, mmHg	171.68 ± 27.88	169.05 ± 27.42	174.31 ± 28.11	0.005
DBP, mmHg	95.47 ± 17.24	94.13 ± 16.86	96.81 ± 17.54	0.020
GCS	13.00 (9.00, 15.00)	14.00 (11.00, 15.00)	11.50 (7.00, 14.00)	<0.001
mRS	4.00 (3.00, 5.00)	3.46 ± 1.19	3.98 ± 1.14	<0.001
Hemiplegia, n (%)	551 (61.84%)	234 (52.58%)	317 (71.08%)	<0.001
Level of consciousness disturbance, n (%)				<0.001
Alert	307 (34.46%)	190 (42.70%)	117 (26.23%)	
Drowsy	112 (12.57%)	60 (13.48%)	52 (11.66%)	
Confused	200 (22.45%)	109 (24.49%)	91 (20.40%)	
Stuporous	34 (3.82%)	14 (3.15%)	20 (4.48%)	
Comatose	238 (26.71%)	72 (16.18%)	166 (37.22%)	
Vomiting, n (%)	401 (45.01%)	151 (33.93%)	250 (56.05%)	<0.001
TFA, h	4.00 (2.00, 10.00)	4.00 (2.00, 12.00)	4.00 (2.62, 7.75)	0.430
Hematoma volume, mL	12.17 (5.67, 32.25)	8.89 (4.76, 20.98)	16.41 (7.24, 43.70)	<0.001
Degree of midline shift, cm	0.25 ± 0.43	0.15 ± 0.33	0.35 ± 0.49	<0.001
Hemorrhage location, n (%)				0.612
Basal ganglia	427 (47.92%)	220 (49.44%)	207 (46.41%)	
Thalamus	198 (22.22%)	98 (22.02%)	100 (22.42%)	
Lobar	132 (14.81%)	66 (14.83%)	66 (14.80%)	
Cerebellum	75 (8.42%)	31 (6.97%)	44 (9.87%)	
Brainstem	59 (6.62%)	30 (6.74%)	29 (6.50%)	
Intraventricular hemorrhage, n (%)	429 (48.20%)	155 (34.83%)	274 (61.57%)	<0.001
Obstructive hydrocephalus, n (%)	73 (8.20%)	23 (5.17%)	50 (11.24%)	<0.001
WBC, ×10 ⁹ /L	10.02 (7.47, 13.04)	8.05 (6.25, 9.83)	12.36 (10.06, 15.72)	<0.001
Neutrophil count, ×10 ⁹ /L	7.94 (5.51, 11.01)	5.76 (4.15, 7.33)	10.67 (8.43, 13.78)	<0.001
Lymphocyte count, ×10 ⁹ /L	1.29 (0.91, 1.81)	1.62 (1.20, 2.12)	1.00 (0.72, 1.35)	<0.001
Monocyte count, ×10 ⁹ /L	0.45 (0.32, 0.62)	0.43 (0.31, 0.55)	0.46 (0.32, 0.71)	0.009
Hemoglobin, g/L	143.11 ± 21.31	143.67 ± 20.29	142.55 ± 22.30	0.433
Platelet count, ×10 ⁹ /L	219.00 (176.00, 271.00)	201.00 (159.00, 244.00)	240.00 (199.00, 294.00)	<0.001
FBG, mmol/L	11.07 (8.68, 13.84)	10.76 (7.81, 13.19)	11.44 (9.35, 14.43)	<0.001
ALT, U/L	20.30 (15.00, 29.90)	20.15 (14.60, 30.70)	20.35 (15.62, 28.50)	0.589
AST, U/L	21.90 (17.25, 29.70)	21.00 (16.40, 29.70)	23.00 (18.00, 29.67)	0.028
TB, umol/L	12.99 (9.20, 18.12)	12.75 (9.05, 17.21)	13.20 (9.55, 18.87)	0.154

(Continued)

Table 1 (Continued).

Variables	Total Population	Low SII	High SII	P Value
Albumin, g/L	41.72 ± 6.39	40.66 ± 6.41	42.79 ± 6.19	<0.001
Uric acid, umol/L	333.49 ± 111.14	329.74 ± 104.79	337.24 ± 117.11	0.314
BUN, mmol/L	6.12 (4.79, 7.96)	6.01 (4.78, 7.51)	6.22 (4.81, 8.49)	0.106
Creatinine, umol/L	68.60 (54.70, 88.92)	68.15 (54.38, 85.73)	69.75 (55.42, 94.77)	0.125
Potassium, mmol/L	3.96 ± 0.63	3.96 ± 0.57	3.97 ± 0.69	0.803
Sodium, mmol/L	139.14 ± 4.48	139.17 ± 3.93	139.10 ± 4.97	0.818
Chloride, mmol/L	102.41 ± 4.92	103.10 ± 4.42	101.72 ± 5.29	<0.001
Calcium, mmol/L	2.30 (2.21, 2.40)	2.29 (2.20, 2.38)	2.32 (2.21, 2.40)	0.007
Bicarbonate, mmol/L	24.21 ± 3.82	24.64 ± 3.54	23.78 ± 4.04	<0.001
Fibrinogen, g/L	2.93 (2.40, 3.84)	2.79 (2.35, 3.57)	3.13 (2.50, 4.14)	<0.001
D-dimer, mg/L	0.94 (0.37, 1.28)	0.93 (0.37, 1.28)	0.97 (0.38, 1.28)	0.160
Treatment modality, n (%)				<0.001
Conservative	653 (73.29%)	360 (80.90%)	293 (65.70%)	
Surgical	238 (26.71%)	85 (19.10%)	153 (34.30%)	
Rebleeding, n (%)	36 (4.04%)	11 (2.47%)	25 (5.61%)	0.018
Reoperation, n (%)	11 (1.23%)	2 (0.45%)	9 (2.02%)	0.034
Pulmonary infection, n (%)	365 (40.97%)	148 (33.26%)	217 (48.65%)	<0.001
Seizure, n (%)	24 (2.69%)	4 (0.90%)	20 (4.48%)	<0.001

Notes: Low SII: SII < 817.5; High SII: SII ≥ 817.5.

Abbreviations: SII, systemic immune-inflammation index; CHD, coronary heart disease; CKD, chronic kidney disease; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; GCS, Glasgow coma scale; mRS, modified Rankin scale; TFA, time from onset to admission; WBC, white blood cell count; FBG, fasting blood glucose; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TB, total bilirubin; BUN, blood urea nitrogen.

Among these variables, the most striking differences were observed in hematoma volume, degree of midline shift, and GCS score ($P < 0.001$), which were well-established predictors of poor prognosis in sICH, suggesting that patients with higher SII tended to present with more severe neurological impairment and thereby supporting the biological plausibility of SII as a prognostic marker.

Multivariate Logistic Regression Analysis of SII and In-Hospital Mortality

As shown in Table 2, variables with $P < 0.05$ in the univariate logistic regression analysis were included in the multivariate logistic regression, and three regression models were constructed. Model 1 was adjusted for smoking, alcohol consumption, use of antihypertensive drugs, SBP, and DBP. Model 2 was further adjusted for GCS score, mRS score, presence of hemiplegia,

Table 2 Multivariate Logistic Regression Analysis of SII and In-Hospital Mortality

	Model 1			Model 2			Model 3		
	OR	95% CI	P Value	OR	95% CI	P Value	OR	95% CI	P Value
Categorical variable									
Low SII	Ref			Ref			Ref		
High SII	4.931	3.333–7.294	<0.001	3.146	1.908–5.188	<0.001	3.122	1.759–5.544	<0.001
Continuous variable									
SII	1.000	1.000–1.000	<0.001	1.000	1.000–1.000	<0.001	1.000	1.000–1.000	0.001
Log ₁₀ SII	11.158	6.560–18.978	<0.001	4.141	2.165–7.920	<0.001	4.914	2.291–10.540	<0.001
Standardized SII	2.339	1.876–2.916	<0.001	1.634	1.272–2.099	<0.001	1.759	1.276–2.426	0.001

Notes: Model 1: Adjusted for smoking, alcohol consumption, antihypertensive drugs, SBP, and DBP. Model 2: Adjusted for variables in Model 1 plus GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, TFA, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, and obstructive hydrocephalus. Model 3: Adjusted for variables in Model 2 plus WBC, FBG, AST, total bilirubin, blood urea nitrogen, chloride, bicarbonate, D-dimer, treatment modality, rebleeding, reoperation, and seizure.

Abbreviations: SII, systemic immune-inflammation index; OR, odds ratio; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; GCS, Glasgow coma scale; mRS, modified Rankin scale; TFA, time from onset to admission; WBC, white blood cell count; FBG, fasting blood glucose; AST, aspartate aminotransferase; TB, total bilirubin; BUN, blood urea nitrogen.

level of consciousness disturbance, vomiting, TFA, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, and obstructive hydrocephalus based on Model 1. Model 3 was additionally adjusted for WBC, FBG, AST, TB, BUN, serum chloride, bicarbonate, D-dimer, treatment modality, rebleeding, reoperation, and seizures based on Model 2.

In all three models—whether SII was treated as a categorical or continuous variable—SII remained significantly associated with in-hospital mortality ($P < 0.05$). Specifically, in the fully adjusted Model 3, the high SII group had a 3.122-fold higher risk of in-hospital mortality compared to the low SII group (OR = 3.122, 95% CI: 1.759–5.544). Additionally, for each 1-unit increase in SII, 1-standard deviation increase in SII, and 1-unit increase in log-transformed SII ($\text{Log}_{10}\text{SII}$), the risk of in-hospital mortality significantly increased (OR = 1.000, 95% CI: 1.000–1.000; OR = 1.759, 95% CI: 1.276–2.426; OR = 4.914, 95% CI: 2.291–10.540, respectively).

Multivariate Stratified Association Between SII and In-Hospital Mortality

In the subgroup analysis of Table 3, when SII was analyzed as a categorical variable (high vs low), high SII was significantly associated with increased in-hospital mortality in multiple subgroups: males, older patients aged ≥ 60 years, patients without IS, those without CHD, BMI $< 28 \text{ kg/m}^2$, GCS < 13 , mRS > 4 , with consciousness disturbance, basal ganglia hemorrhage, non-basal ganglia hemorrhage, with and without IVH, WBC $\leq 10 \times 10^9/\text{L}$ and $> 10 \times 10^9/\text{L}$, conservative and surgical treatment, with and without pulmonary infection (all $P < 0.05$).

When SII was analyzed as a continuous variable, including raw values, log-transformed values ($\text{Log}_{10}\text{SII}$), and standardized SII, consistent positive associations with in-hospital mortality were observed in many subgroups. $\text{Log}_{10}\text{SII}$ and standardized SII showed significant associations in females, age ≥ 60 , those without IS, both CHD subgroups, BMI $< 28 \text{ kg/m}^2$, both GCS and mRS subgroups, patients with consciousness disturbance, both hemorrhage locations, both IVH groups, WBC $\leq 10 \times 10^9/\text{L}$, surgical treatment, and patients with pulmonary infection (all $P < 0.05$). Additionally, all P for interaction values were > 0.05 , indicating no statistically significant interactions across subgroups.

SII and In-Hospital Mortality: Multivariate Logistic Regression Analysis - Sensitivity Analysis

As shown in Table 4, sensitivity analyses were conducted by sequentially excluding patients with potential confounding conditions, including hyperlipidemia, history of IVH, CKD, HF, AF, hydrocephalus, as well as those with postoperative rebleeding, reoperation, or seizures. In all scenarios, the high SII group consistently showed a significantly increased risk of in-hospital mortality compared to the low SII group, with ORs ranging from 2.800 to 3.719 (all $P < 0.05$). Furthermore, in each subgroup, increases in SII (per unit and per standard deviation), as well as in log-transformed SII ($\text{Log}_{10}\text{SII}$), were all significantly associated with elevated in-hospital mortality risk (all $P < 0.05$), confirming the robustness of the association between SII and in-hospital mortality.

Kaplan-Meier Survival Analysis of Long-Term All-Cause Mortality by SII Levels

The Kaplan-Meier survival curve of Figure 1 illustrated the difference in long-term survival probability between patients with high and low SII levels. Over a follow-up period of approximately 50 months, the high SII group (red line) consistently showed a lower survival probability compared to the low SII group (blue line). The difference in survival between the two groups was statistically significant, as indicated by the Log rank test ($P = 0.015$).

Multivariate Cox Regression Analysis of SII and Long-Term All-Cause Mortality

As shown in Table 5, multivariate Cox regression analysis included variables with $P < 0.05$ from the univariate Cox regression analysis, and three regression models were constructed. Model 1 was adjusted for age, smoking, and SBP. Model 2 further adjusted for GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, and obstructive hydrocephalus based on Model 1. Model 3 further adjusted for WBC, D-dimer, ALT, treatment modality, and pulmonary infection based on Model 2.

In Models 1, 2, and 3, whether SII was treated as a categorical or continuous variable, it was significantly associated with all-cause mortality ($P < 0.05$). Specifically, in the fully adjusted Model 3, the high SII group had a 2.755-fold higher risk of all-

Table 3 Multivariate Stratified Association Between SII and In-Hospital Mortality

	High SII vs Low SII		SII		Log ₁₀ SII		Standardized SII		P for Interaction
	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	
Sex									0.623
Male	4.253 (2.076–8.714)	<0.001	1.000 (1.000–1.000)	0.212	5.874 (2.313–14.916)	<0.001	1.359 (0.839–2.200)	0.212	
Female	2.698 (0.675–10.789)	0.161	1.000 (1.000–1.001)	0.006	8.590 (2.285–32.293)	0.001	2.268 (1.262–4.074)	0.006	
Age									0.288
< 60 years	2.659 (0.538–13.137)	0.230	1.000 (1.000–1.001)	0.128	4.867 (1.230–19.256)	0.024	1.996 (0.824–4.692)	0.128	
≥ 60 years	5.011 (2.353–10.673)	<0.001	1.000 (1.000–1.001)	0.005	6.158 (2.297–16.509)	<0.001	2.002 (1.231–3.255)	0.005	
Ischemic stroke									0.461
No	3.858 (2.000–7.445)	<0.001	1.000 (1.000–1.000)	0.001	5.392 (2.327–12.492)	<0.001	1.805 (1.555–2.595)	0.001	
Yes	4.170 (0.435–40.007)	0.216	1.001 (1.000–1.001)	0.075	36.021 (0.652–1991.418)	0.080	3.449 (0.883–13.496)	0.075	
Coronary heart disease									0.223
No	2.907 (1.487–5.686)	0.002	1.000 (1.000–1.000)	0.005	3.839 (1.592–9.257)	0.003	1.692 (1.169–2.450)	0.005	
Yes	–	0.960	1.000 (1.000–1.001)	0.005	12.570 (2.434–64.921)	0.003	2.756 (1.351–5.619)	0.005	
Body mass index									0.903
< 28 kg/m ²	3.623 (1.735–7.568)	<0.001	1.000 (1.000–1.000)	0.003	5.050 (1.990–12.816)	<0.001	1.713 (1.195–2.457)	0.003	
≥ 28 kg/m ²	6.113 (0.975–38.594)	0.053	1.000 (1.000–1.001)	0.101	8.784 (0.674–114.418)	0.097	2.373 (0.844–6.670)	0.101	
GCS									0.357
< 13	4.326 (2.191–8.543)	<0.001	1.000 (1.000–1.001)	<0.001	9.908 (3.643–26.947)	<0.001	2.363 (1.496–3.732)	<0.001	
≥ 13	5.346 (0.502–59.926)	0.165	1.001 (1.000–1.002)	0.033	24.509 (0.576–1043.382)	0.095	8.522 (1.184–61.335)	0.033	
mRs									0.983
≤ 4	2.853 (0.633–12.849)	0.173	1.001 (1.000–1.001)	0.003	3.626 (1.043–12.609)	0.043	3.971 (1.620–9.731)	0.003	
> 4	4.233 (1.942–9.225)	<0.001	1.000 (1.000–1.000)	0.026	5.238 (1.865–14.701)	0.002	1.667 (1.062–2.619)	0.026	
Consciousness disturbance									0.432
No	5.585 (0.066–472.108)	0.447	1.001 (0.999–1.003)	0.356	75.506 (0.083–68,690.417)	0.214	7.806 (0.100–611.239)	0.356	
Yes	4.039 (2.080–7.840)	<0.001	1.000 (1.000–1.000)	<0.001	7.109 (2.976–16.982)	<0.001	2.057 (1.408–3.006)	<0.001	
Hemorrhage location									0.161
Basal ganglia	2.671 (1.258–5.443)	0.010	1.000 (1.000–1.001)	0.006	5.293 (1.970–14.221)	<0.001	2.010 (1.222–3.306)	0.006	
Non-basal ganglia	6.478 (2.223–18.880)	<0.001	1.000 (1.000–1.000)	0.235	4.997 (1.422–17.561)	0.012	1.447 (0.775–2.815)	0.235	
Intraventricular hemorrhage									0.460
No	7.346 (2.293–23.532)	<0.001	1.000 (1.000–1.001)	0.439	23.404 (4.171–131.328)	<0.001	1.827 (0.397–8.398)	0.439	
Yes	2.568 (1.350–5.053)	0.006	1.000 (1.000–1.000)	0.006	3.784 (1.553–9.221)	0.003	1.678 (1.163–2.422)	0.006	
White blood cell count									0.129
≤ 10 × 10 ⁹ /L	4.614 (1.897–11.224)	<0.001	1.001 (1.000–1.001)	0.002	14.969 (3.293–68.048)	<0.001	3.507 (1.607–7.655)	0.002	
> 10 × 10 ⁹ /L	2.925 (1.225–6.982)	0.016	1.000 (1.000–1.000)	0.081	3.916 (1.335–11.485)	0.013	1.607 (0.944–2.736)	0.081	
Treatment modality									0.224
Conservative	2.845 (1.209–6.693)	0.017	1.000 (1.000–1.001)	0.112	4.263 (1.248–16.564)	0.021	1.993 (0.852–4.659)	0.112	
Surgical	3.841 (1.717–8.596)	0.001	1.000 (1.000–1.000)	0.008	5.824 (2.017–16.814)	0.001	1.800 (1.167–2.778)	0.008	
Pulmonary infection									0.909
No	3.571 (1.391–9.168)	0.008	1.000 (1.000–1.001)	0.345	3.974 (1.142–13.832)	0.030	1.500 (0.646–3.482)	0.345	
Yes	4.020 (1.930–8.372)	<0.001	1.000 (1.000–1.000)	<0.001	6.945 (2.675–18.003)	<0.001	1.844 (1.302–2.611)	<0.001	

Notes: The subgroup analysis was adjusted for smoking, alcohol consumption, antihypertensive drugs, SBP, DBP, GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, TFA, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, obstructive hydrocephalus, white blood cell count, FBG, AST, total bilirubin, blood urea nitrogen, chloride, bicarbonate, D-dimer, treatment modality, rebleeding, reoperation, and seizure.

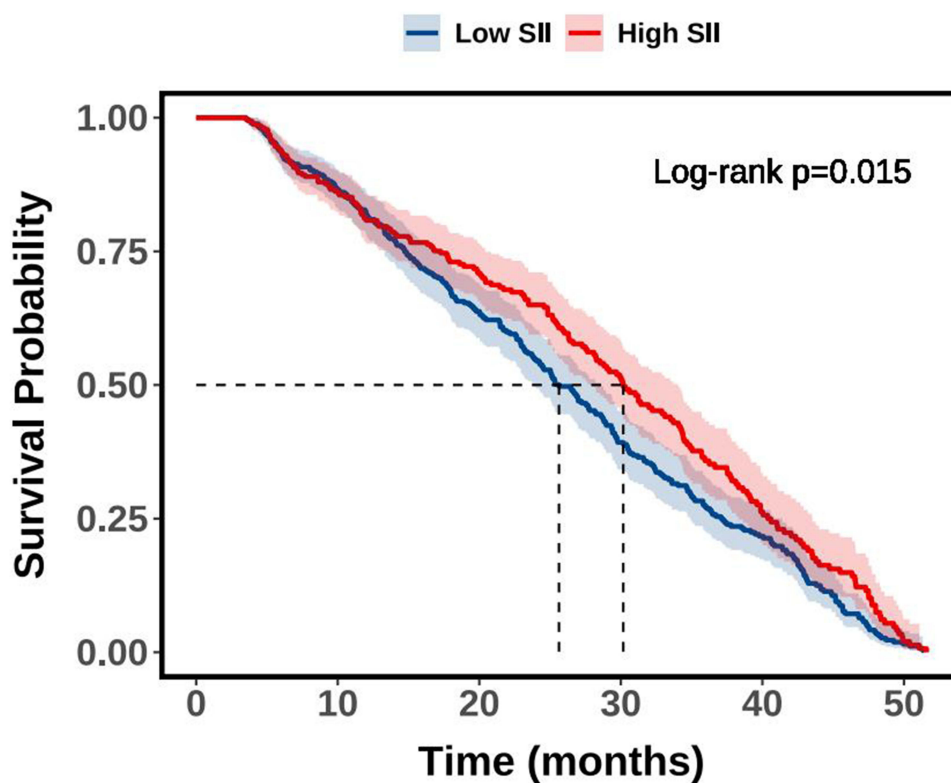
Abbreviations: SII, systemic immune-inflammation index; OR, odds ratio; CI, confidence interval; GCS, Glasgow coma scale; mRS, modified Rankin scale; TFA, time from onset to admission; FBG, fasting blood glucose; AST, aspartate aminotransferase.

Table 4 Multivariate Logistic Regression Analysis of SII and In-Hospital Mortality: Sensitivity Analysis

	Excluding Patients with Hyperlipidemia		Excluding Patients with a History of Intracerebral Hemorrhage		Excluding Patients with Chronic Kidney Disease		Excluding Patients with Heart Failure		Excluding Patients with Atrial Fibrillation		Excluding Patients with Hydrocephalus		Excluding Patients with Postoperative Rebleeding, Reoperation, and Seizures	
	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value
Low SII	Ref		Ref		Ref		Ref		Ref		Ref		Ref	
High SII	3.488 (1.973–6.166)	<0.001	3.201 (1.779–5.758)	<0.001	3.607 (1.989–6.539)	<0.001	3.719 (2.055–6.732)	<0.001	3.275 (1.832–1.854)	<0.001	3.443 (1.803–6.573)	<0.001	2.800 (1.511–5.191)	0.001
SII	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001	1.000 (1.000–1.000)	<0.001
Log ₁₀ SII	5.977 (2.830–16.622)	<0.001	5.423 (2.478–11.907)	<0.001	5.960 (2.624–13.537)	<0.001	4.792 (2.220–10.342)	<0.001	4.819 (2.251–10.313)	<0.001	6.214 (2.587–14.929)	<0.001	5.355 (2.181–13.148)	<0.001
Standardized SII	1.826 (1.371–2.433)	<0.001	1.715 (1.251–2.351)	<0.001	1.903 (1.388–2.609)	<0.001	1.764 (1.308–2.379)	<0.001	1.689 (1.243–2.295)	<0.001	2.077 (1.443–2.991)	<0.001	1.831 (1.288–2.604)	<0.001

Notes: The analysis adjusted for smoking, alcohol consumption, antihypertensive drugs, systolic blood pressure, diastolic blood pressure, GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, TFA, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, obstructive hydrocephalus, white blood cell count, fasting blood glucose, aspartate aminotransferase, total bilirubin, blood urea nitrogen, chloride, bicarbonate, D-dimer, treatment modality, rebleeding, reoperation, and seizure.

Abbreviations: SII, systemic immune-inflammation index; OR, odds ratio; CI, confidence interval; GCS, Glasgow coma scale; mRS, modified Rankin scale; TFA, time from onset to admission.



Low SII		High SII				
At Risk	352	294	201	112	59	4
Events	0	46	121	193	242	296
High SII		Low SII				
At Risk	352	247	166	93	41	3
Events	0	42	82	122	164	200

Figure 1 Kaplan-Meier survival analysis of long-term all-cause mortality by SII levels.

Abbreviation: SII, systemic immune-inflammation index.

cause mortality compared to the low SII group (HR = 2.755, 95% CI: 1.938–3.919). Additionally, for each 1-unit increase in SII, 1-standard deviation increase in SII, and 1-unit increase in $\text{Log}_{10}\text{SII}$, the risk of all-cause mortality significantly increased (HR = 1.000, 95% CI: 1.000–1.000; HR = 1.170, 95% CI: 1.040–1.316; HR = 1.221, 95% CI: 1.175–1.268).

Multivariate Stratified Association Between SII and Long-Term All-Cause Mortality

In the subgroup analysis of Table 6, when SII was analyzed as a categorical variable (high vs low), high SII was significantly associated with increased long-term all-cause mortality in males, females, patients aged 60 years or older, non-smokers, alcohol users, patients with or without IS, with or without CHD, those with BMI less than 28 kg/m^2 , patients with GCS scores 13 or above, mRS scores 4 or below, with or without consciousness disturbance, those with basal ganglia or non-basal ganglia hemorrhage, with or without IVH, with WBC either ≤ 10 or $> 10 \times 10^9/\text{L}$, those receiving conservative or surgical treatment, and those with or without pulmonary infection (all $P < 0.05$).

When SII was treated as a continuous variable (including raw, log-transformed, and standardized SII), it remained significantly associated with long-term mortality in females, patients aged 60 years or older, alcohol users, patients with or without IS, with or without CHD, those with BMI less than 28 kg/m^2 , both GCS subgroups, mRS ≤ 4 subgroup, patients with or without consciousness disturbance, both hemorrhage location groups, both IVH subgroups, WBC $\leq 10 \times 10^9/\text{L}$,

Table 5 Multivariate Cox Regression Analysis of SII and Long-Term All-Cause Mortality

	Model 1			Model 2			Model 3		
	HR	95% CI	P Value	HR	95% CI	P Value	HR	95% CI	P Value
Categorical variable									
Low SII	Ref			Ref			Ref		
High SII	3.763	2.738–5.173	<0.001	3.384	2.441–4.691	<0.001	2.755	1.938–3.919	<0.001
Continuous variable									
SII	1.000	1.000–1.000	<0.001	1.000	1.000–1.000	<0.001	1.000	1.000–1.000	0.009
Log ₁₀ SII	1.229	1.184–1.277	<0.001	1.222	1.176–1.270	<0.001	1.221	1.175–1.268	<0.001
Standardized SII	1.341	1.253–1.436	<0.001	1.350	1.253–1.455	<0.001	1.170	1.040–1.316	0.009

Notes: Model 1: Adjusted for age, smoking, and systolic blood pressure. Model 2: Adjusted for age, smoking, systolic blood pressure, GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, and obstructive hydrocephalus. Model 3: Adjusted for variables in Model 2 plus white blood cell count, D-dimer, alanine aminotransferase, treatment modality, and pulmonary infection.

Abbreviations: SII, systemic immune-inflammation index; HR, hazard ratio; CI, confidence interval; GCS, Glasgow coma scale; mRS, modified Rankin scale.

surgical treatment subgroup, and patients without pulmonary infection (all $P < 0.05$). Additionally, all P for interaction values were > 0.05 , indicating no statistically significant interactions across subgroups.

Multivariate Cox Regression Analysis of SII and Long-Term All-Cause Mortality: Sensitivity Analysis

In the sensitivity analyses of [Table 7](#), high SII was significantly associated with increased long-term all-cause mortality across all exclusion subgroups. Specifically, after excluding patients with hyperlipidemia, the HR was 2.698 (95% CI: 1.897–3.835); after excluding those with a history of IVH, HR = 2.630 (95% CI: 1.829–3.783); after excluding patients with CKD, HR = 2.607 (95% CI: 1.812–3.752); after excluding HF patients, HR = 2.520 (95% CI: 1.750–3.629); after excluding AF, HR = 2.918 (95% CI: 2.025–4.207); after excluding hydrocephalus, HR = 2.697 (95% CI: 1.858–3.915); and after excluding patients with postoperative rebleeding, reoperation, or seizures, HR = 2.406 (95% CI: 1.683–3.439).

For continuous forms of SII, all three indicators—raw SII, log₁₀-transformed SII, and standardized SII—were significantly associated with long-term all-cause mortality across all sensitivity analyses. The log₁₀SII yielded HRs ranging from 1.207 to 1.227, all with $P < 0.001$ and 95% CIs that did not cross 1, indicating stable and robust associations. Similarly, standardized SII showed HRs between 1.136 and 1.174, with all P values ≤ 0.042 and 95% CIs indicating statistical significance. Although raw SII values had HRs approximately equal to 1.000, the narrow CIs and P values (ranging from 0.008 to 0.042) suggest a consistent, albeit small, linear effect on risk.

Predictive Performance of SII for Mortality Outcomes

[Figure 2](#) presented the ROC curves comparing the predictive performance of SII with established clinical indicators, including GCS, hematoma volume, and degree of midline shift, for both in-hospital and long-term all-cause mortality. For in-hospital mortality (Panel A), SII yielded an AUC of 0.722 (95% CI: 0.681–0.763, $P < 0.001$), indicating a moderate discriminative ability, with SII having about a 72% probability of correctly assigning higher risk to a non-survivor when randomly comparing one in-hospital death with one survivor. However, its predictive performance was lower than that of GCS (AUC: 0.878, 95% CI: 0.847–0.901), hematoma volume (AUC: 0.778, 95% CI: 0.738–0.817), and midline shift (AUC: 0.760, 95% CI: 0.716–0.805).

In contrast, for long-term all-cause mortality (Panel B), SII achieved the highest AUC among all variables at 0.748 (95% CI: 0.709–0.787, $P < 0.001$), suggesting good discriminative capacity with about a 75% probability of correctly distinguishing a patient who will die during follow-up from one who will survive, and outperforming hematoma volume (AUC: 0.662, 95% CI: 0.617–0.708), GCS (AUC: 0.634, 95% CI: 0.587–0.681), and midline shift (AUC: 0.580, 95% CI: 0.543–0.616).

Table 6 Multivariate Stratified Association Between SII and Long-Term All-Cause Mortality

	High SII vs Low SII		SII		Log ₁₀ SII		Standardized SII		P for Interaction
	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	
Sex									0.547
Male	3.076 (1.956–4.837)	<0.001	1.000 (1.000–1.000)	0.056	1.212 (1.155–1.271)	<0.001	1.151 (0.997–1.329)	0.056	
Female	2.182 (1.235–3.857)	0.007	1.000 (1.000–1.000)	0.003	1.239 (1.155–1.329)	<0.001	1.387 (1.116–1.725)	0.003	
Age									0.166
< 60 years	1.505 (0.784–2.887)	0.219	1.000 (1.000–1.000)	0.002	1.174 (1.091–1.262)	<0.001	1.226 (1.075–1.398)	0.002	
≥ 60 years	3.546 (2.312–5.440)	<0.001	1.000 (1.000–1.000)	<0.001	1.254 (1.193–1.318)	<0.001	1.677 (1.476–1.904)	<0.001	
Smoking									0.154
No	2.883 (1.941–4.281)	<0.001	1.000 (1.000–1.000)	0.010	1.219 (1.170–1.271)	<0.001	1.182 (1.041–1.343)	0.010	
Yes	2.384 (0.839–6.770)	0.103	1.001 (1.000–1.001)	0.770	1.230 (1.090–1.388)	<0.001	1.107 (0.560–2.191)	0.770	
Alcohol consumption									0.378
No	2.439 (1.653–3.598)	<0.001	1.000 (1.000–1.000)	0.025	1.212 (1.163–1.263)	<0.001	1.151 (1.018–1.302)	0.025	
Yes	4.761 (2.179–10.404)	<0.001	1.001 (1.000–1.001)	<0.001	1.388 (1.212–1.591)	<0.001	2.877 (1.814–4.564)	<0.001	
Ischemic stroke									0.307
No	2.068 (1.382–3.093)	<0.001	1.000 (1.000–1.000)	<0.001	1.159 (1.095–1.227)	<0.001	1.292 (1.118–1.493)	<0.001	
Yes	5.207 (2.653–10.220)	<0.001	1.000 (1.000–1.000)	0.001	1.217 (1.135–1.304)	<0.001	1.255 (1.094–1.439)	0.001	
Coronary heart disease									0.609
No	2.388 (1.613–3.535)	<0.001	1.000 (1.000–1.000)	0.014	1.217 (1.165–1.272)	<0.001	1.186 (1.036–1.359)	0.014	
Yes	3.283 (1.567–6.877)	0.002	1.000 (1.000–1.000)	0.037	1.310 (1.182–1.452)	<0.001	1.281 (1.015–1.617)	0.037	
Body mass index									0.434
< 28 kg/m ²	3.690 (2.546–5.347)	<0.001	1.000 (1.000–1.000)	<0.001	1.213 (1.161–1.267)	<0.001	1.338 (1.229–1.456)	<0.001	
≥ 28 kg/m ²	1.749 (0.734–4.169)	0.207	1.000 (1.000–1.000)	0.470	1.050 (0.931–1.184)	0.424	0.880 (0.621–1.246)	0.470	
GCS									0.298
< 13	1.946 (1.175–3.344)	0.012	1.000 (1.000–1.000)	0.084	1.144 (1.067–1.227)	<0.001	1.176 (0.978–1.413)	0.084	
≥ 13	3.840 (2.470–5.969)	<0.001	1.000 (1.000–1.000)	<0.001	1.227 (1.162–1.294)	<0.001	1.291 (1.169–1.425)	<0.001	
mRs									0.242
≤ 4	3.306 (2.142–5.102)	<0.001	1.000 (1.000–1.000)	0.020	1.246 (1.189–1.305)	<0.001	1.179 (1.026–1.355)	0.020	
> 4	1.574 (0.825–3.006)	0.169	1.000 (1.000–1.000)	0.372	1.097 (1.000–1.203)	0.050	1.126 (0.868–1.461)	0.372	
Consciousness disturbance									0.113
No	5.491 (3.095–9.742)	<0.001	1.000 (1.000–1.000)	<0.001	1.247 (1.171–1.328)	<0.001	1.275 (1.133–1.435)	<0.001	
Yes	1.999 (1.301–3.071)	0.002	1.000 (1.000–1.000)	0.001	1.156 (1.087–1.228)	<0.001	1.287 (1.102–1.505)	0.001	
Hemorrhage location									0.469
Basal ganglia	1.907 (1.053–3.452)	0.033	1.000 (1.000–1.001)	<0.001	1.229 (1.148–1.316)	<0.001	1.778 (1.462–2.187)	<0.001	
Non-basal ganglia	3.636 (2.262–5.843)	<0.001	1.000 (1.000–1.000)	0.138	1.226 (1.169–1.286)	<0.001	1.125 (0.963–1.314)	0.138	
Intraventricular hemorrhage									0.146
No	3.339 (2.057–5.418)	<0.001	1.000 (1.000–1.000)	<0.001	1.244 (1.179–1.312)	<0.001	1.321 (1.197–1.457)	<0.001	
Yes	2.017 (1.210–3.363)	0.007	1.000 (1.000–1.000)	0.031	1.134 (1.058–1.215)	<0.001	1.215 (1.018–1.448)	0.031	

(Continued)

Table 6 (Continued).

	High SII vs Low SII		SII		Log ₁₀ SII		Standardized SII		P for Interaction
	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	
White blood cell count									
≤ 10 ×10 ⁹ /L	2.808 (1.808–4.360)	<0.001	1.000 (1.000–1.000)	<0.001	1.243 (1.168–1.324)	<0.001	1.366 (1.136–1.642)	<0.001	0.710
> 10 ×10 ⁹ /L	3.525 (1.951–6.371)	<0.001	1.000 (1.000–1.000)	0.179	1.161 (1.093–1.233)	<0.001	1.117 (0.950–1.313)	0.179	
Treatment modality									0.296
Conservative	3.005 (1.947–4.639)	<0.001	1.000 (1.000–1.000)	0.482	1.230 (1.172–1.291)	<0.001	1.067 (0.890–1.279)	0.482	
Surgical	2.025 (1.149–3.567)	0.015	1.000 (1.000–1.000)	<0.001	1.172 (1.098–1.250)	<0.001	1.353 (1.164–1.572)	<0.001	
Pulmonary infection									0.147
No	2.987 (1.912–4.666)	<0.001	1.000 (1.000–1.000)	<0.001	1.237 (1.164–1.315)	<0.001	1.631 (1.390–1.915)	<0.001	
Yes	2.717 (1.607–4.593)	<0.001	1.000 (1.000–1.000)	0.804	1.194 (1.132–1.259)	<0.001	1.022 (0.860–1.214)	0.804	

Notes: The subgroup analysis was adjusted for age, smoking, systolic blood pressure, GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, obstructive hydrocephalus, white blood cell count, D-dimer, alanine aminotransferase, treatment modality, and pulmonary infection.

Abbreviations: SII, systemic immune-inflammation index; HR, hazard ratio; CI, confidence interval; GCS, Glasgow coma scale; mRS, modified Rankin scale.

Table 7 Multivariate Cox Regression Analysis of SII and Long-Term All-Cause Mortality: Sensitivity Analysis

	Excluding Patients with Hyperlipidemia		Excluding Patients with a History of Intracerebral Hemorrhage		Excluding Patients with Chronic Kidney Disease		Excluding Patients with Heart Failure		Excluding Patients with Atrial Fibrillation		Excluding Patients with Hydrocephalus		Excluding Patients with Postoperative Rebleeding, Reoperation, and Seizures	
	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value
Low SII	Ref		Ref		Ref		Ref		Ref		Ref		Ref	
High SII	2.698 (1.897–3.835)	<0.001	2.630 (1.829–3.783)	<0.001	2.607 (1.812–3.752)	<0.001	2.520 (1.750–3.629)	<0.001	2.918 (2.025–4.207)	<0.001	2.697 (1.858–3.915)	<0.001	2.406 (1.683–3.439)	<0.001
SII	1.000 (1.000–1.000)	0.011	1.000 (1.000–1.000)	0.016	1.000 (1.000–1.000)	0.038	1.000 (1.000–1.000)	0.042	1.000 (1.000–1.000)	0.008	1.000 (1.000–1.000)	0.012	1.000 (1.000–1.000)	0.019
Log ₁₀ SII	1.220 (1.175–1.268)	<0.001	1.208 (1.161–1.258)	<0.001	1.227 (1.178–1.278)	<0.001	1.222 (1.174–1.271)	<0.001	1.221 (1.175–1.270)	<0.001	1.218 (1.170–1.268)	<0.001	1.207 (1.159–1.257)	<0.001
Standardized SII	1.165 (1.035–1.310)	0.011	1.161 (1.028–1.312)	0.016	1.143 (1.007–1.296)	0.038	1.136 (1.005–1.286)	0.042	1.174 (1.043–1.323)	0.008	1.169 (1.034–1.321)	0.012	1.150 (1.023–1.293)	0.019

Notes: The analysis adjusted for age, smoking, systolic blood pressure, GCS, mRS, hemiplegia, level of consciousness disturbance, vomiting, hematoma volume, degree of midline shift, hemorrhage location, intraventricular extension, obstructive hydrocephalus, white blood cell count, D-dimer, alanine aminotransferase, treatment modality, and pulmonary infection.

Abbreviations: SII, systemic immune-inflammation index; HR, hazard ratio; CI, confidence interval; GCS, Glasgow coma scale; mRS, modified Rankin scale.

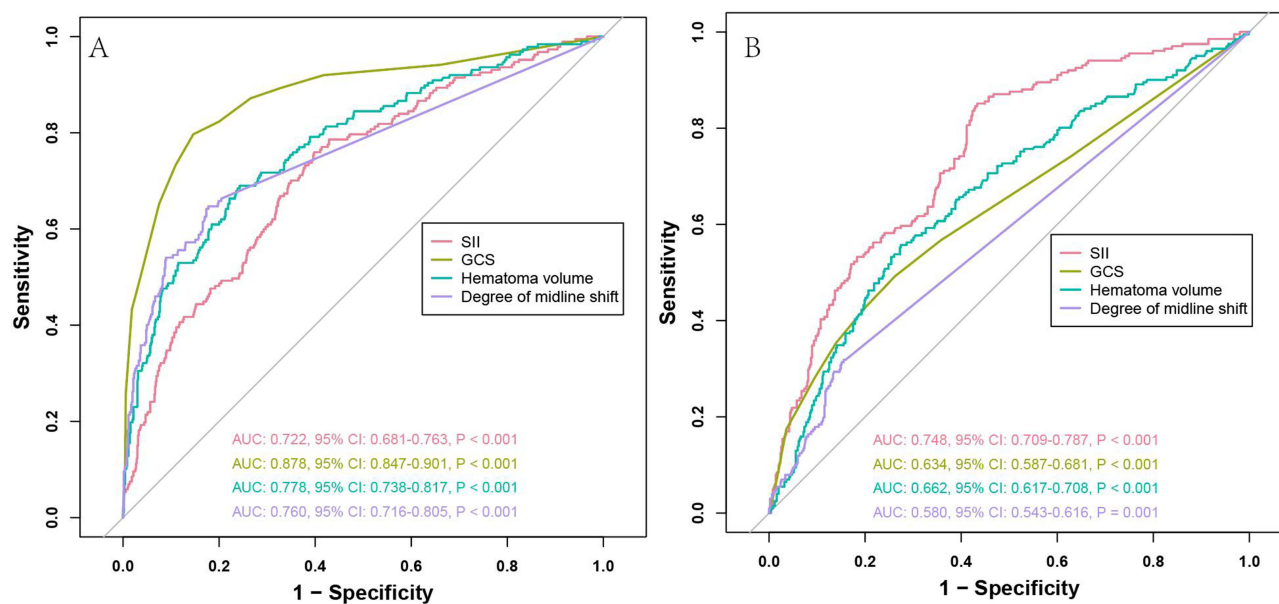


Figure 2 ROC curves comparing the predictive performance of SII, GCS, hematoma volume, and degree of midline shift for in-hospital mortality (**A**) and long-term all-cause mortality (**B**).

Abbreviations: ROC, receiver operating characteristic; SII, systemic immune-inflammation index; GCS, Glasgow Coma Scale; AUC, area under the curve; CI, confidence interval.

Discussion

In this large retrospective cohort study of patients with T2DM complicated by sICH, we found that SII was independently associated with both in-hospital and long-term all-cause mortality. These associations were established through multivariate logistic regression for in-hospital mortality and multivariate Cox regression for long-term mortality, and remained robust in subgroup and sensitivity analyses excluding major comorbidities. ROC curve analyses further showed that SII had a higher AUC than hematoma volume, midline shift, and GCS in predicting long-term out-of-hospital mortality, suggesting better performance for long-term risk prediction. However, for in-hospital mortality, SII showed a lower AUC than these conventional indicators, indicating limited utility in short-term prognostication where acute neurological factors are dominant. These findings support the clinical utility of SII as a simple, inexpensive, and accessible biomarker for early risk stratification in patients with T2DM and sICH. While it may be less effective than traditional markers for short-term outcomes, SII demonstrates stronger value in predicting long-term prognosis, particularly after discharge when systemic inflammation may continue to influence recovery. Incorporating SII into clinical assessment may aid in identifying high-risk patients and guiding timely, individualized interventions.

Currently, studies investigating the prognostic value of the SII in cerebrovascular diseases have primarily focused on general stroke populations or treated SII as a single inflammatory biomarker. There is still a lack of systematic research exploring its prognostic significance specifically in patients with sICH complicated by T2DM. For example, Geraghty et al conducted a retrospective study of 246 patients with SAH to examine the association between SII and delayed cerebral vasospasm.³⁰ The results showed that approximately 67.5% (166 cases) developed imaging-confirmed vasospasm, and SII at admission was significantly elevated in those patients. In multivariate logistic regression analysis, SII remained an independent predictor even after adjusting for age, aneurysm location, T2DM, and hyperlipidemia. ROC curve analysis demonstrated a good predictive value of SII for vasospasm, suggesting that early elevation of SII may serve as an independent predictor of delayed cerebral vasospasm in aneurysmal SAH, providing reference for clinical risk stratification and intervention.

In another study by Wang et al on patients with ICH, SII was associated with stroke-associated pneumonia (SAP) in univariate analysis, but ROC curve analysis revealed limited predictive ability, and SII was not identified as an independent predictor in multivariate analysis.³¹ Additionally, a retrospective study by Chen et al that included 333

SAH patients found that SII was an independent predictor of delayed cerebral ischemia (DCI).³² Multivariate regression showed a significant association between elevated SII and DCI risk. The ROC curve revealed high predictive accuracy for DCI (AUC = 0.860), with an optimal cutoff of 1,424, yielding a sensitivity of 93.1% and specificity of 68.1%. Patients with higher SII were also more likely to develop acute hydrocephalus and DCI, had higher modified Fisher scores and Hunt-Hess grades, and worse clinical outcomes, indicating that SII could serve as a practical biomarker for early DCI risk identification in aneurysmal SAH.

Furthermore, in a retrospective study by Liu et al, 105 patients with sICH were included to evaluate the prognostic value of SII for disease severity and outcomes.³³ Patients were divided into good and poor prognosis groups based on the 3-month mRS score. Results showed that SII was significantly higher in the poor prognosis group and correlated with GCS, NIHSS, and mRS scores at admission. Multivariate regression confirmed SII as an independent risk factor for poor outcomes. ROC analysis showed that SII had an AUC of 0.838, outperforming other markers with higher predictive accuracy. Thus, SII can be used as a valid predictor of disease severity and 3-month outcomes in sICH patients. Wang et al also conducted a single-center retrospective study evaluating the ability of SII to predict acute kidney injury (AKI) and short-term outcomes in post-operative sICH patients.³⁴ Among the 305 patients who underwent craniotomy, 129 (42.3%) developed AKI. Multivariate analysis identified SII as an independent predictor of AKI. Furthermore, in AKI patients, elevated SII was significantly associated with poor postoperative outcomes (AUC = 0.886; sensitivity 78.9%, specificity 88.2%). This study demonstrated that SII may predict both AKI risk and short-term prognosis in sICH patients undergoing surgery, offering clinical guidance. Liao et al conducted a systematic review to assess the prognostic value of SII in patients with aSAH, ultimately including four eligible studies from PubMed and Embase.³⁵ All studies found that elevated SII at admission was significantly associated with worse outcomes, including DCI, cerebral vasospasm, and poor functional recovery. Although SII has been widely used in other medical fields for outcome prediction, this review was the first to systematically confirm its potential for predicting DCI and poor prognosis in aSAH patients, suggesting its value as an early risk stratification and prognostic tool. In a retrospective study by Trifan et al involving 239 patients with supratentorial sICH, SII was found to be an independent predictor of poor discharge outcomes.³⁶ In multivariate analysis, SII was the only inflammatory index significantly associated with poor outcomes, and ROC curve analysis showed good discriminatory ability (AUC = 0.89), with an optimal cutoff value of 0.73 (sensitivity 95%, specificity 71%), indicating that early SII can be used to predict short-term poor outcomes in supratentorial sICH patients. However, in a retrospective analysis of 869 critically ill ICH patients from the MIMIC-IV database, Zhao et al assessed the predictive power of five inflammatory markers for ICU mortality risk.³⁷ Although SII was included in the analysis, it did not outperform other markers and was not the most significant independent predictor in multivariate regression. Thus, while SII was considered in this study, its predictive value for ICU mortality in critically ill ICH patients was relatively limited.

Despite growing evidence suggesting that SII holds prognostic value in stroke, current research remains limited in several important ways. Most existing studies focus on general stroke populations—especially non-diabetic or mixed cohorts—without stratifying or specifically analyzing patients with coexisting T2DM. As a result, they fail to capture the unique inflammatory, immunologic, and coagulopathic disturbances characteristic of this high-risk subgroup. Moreover, most prior studies emphasized short-term functional outcomes, complications, or hospital mortality, while systematic evaluations of SII in predicting mid-to-long-term mortality (eg, post-discharge all-cause death) are still lacking. In addition, many of the available studies suffer from methodological limitations, such as small sample sizes and insufficient confounder adjustment, which restrict the generalizability and statistical power of their findings. These gaps limit our understanding of how SII might inform long-term risk stratification and individualized management strategies in patients with sICH, particularly those with T2DM. The present study addresses these limitations and contributes several novel insights. First, it is the first to focus specifically on patients with T2DM complicated by sICH—a population at significantly higher risk of mortality and disability—thereby enhancing the clinical relevance and applicability of the findings. Second, it comprehensively evaluates the association between SII and both in-hospital and long-term all-cause mortality, using robust statistical techniques including multivariate regression, subgroup analysis, interaction testing, and sensitivity analysis. Third, the study demonstrates that SII not only retains independent predictive power for short-term outcomes but also outperforms traditional indicators such as GCS, hematoma volume, and midline shift in predicting long-term mortality, highlighting its added value in post-discharge prognostication. With a relatively large cohort and rigorous model validation,

this study provides the first systematic evidence that SII can serve as a reliable and cost-effective biomarker for early risk stratification in patients with T2DM and sICH. These findings fill a critical gap in the literature and lay the groundwork for future individualized treatment and monitoring strategies in this vulnerable population.

This study further explored the potential biological mechanisms underlying the prognostic value of the SII in patients with T2DM and sICH, highlighting its central role in the interplay between inflammation and immunity (Figure S1). From a mechanistic perspective, SII is a composite inflammatory marker derived from neutrophil, lymphocyte, and platelet counts. It provides a more comprehensive reflection of peripheral inflammatory burden and immune status than any single parameter alone. After cerebral hemorrhage, neutrophils are rapidly mobilized to the site of injury, releasing elastase, myeloperoxidase, and other reactive species that induce neuronal apoptosis and BBB disruption. A reduction in lymphocytes reflects an immunosuppressive state, increasing susceptibility to post-hemorrhagic infections such as pneumonia. Platelet activation contributes to the inflammation-coagulation cascade, promoting microthrombus formation and exacerbating tissue ischemia and necrosis.³⁸ By integrating these three components, SII captures the complexity of this pathological process. Beyond inflammation, other mechanisms may also explain the elevation of SII in patients with T2DM and sICH. Chronic hyperglycemia induces endothelial dysfunction through oxidative stress and advanced glycation end products, leading to impaired vascular reactivity and increased permeability, which aggravates BBB disruption.^{39,40} In addition, T2DM is frequently associated with coagulopathy, including platelet hyperreactivity, impaired fibrinolysis, and abnormal activation of the coagulation cascade.⁴¹ These abnormalities not only elevate SII through increased platelet counts and neutrophil activation but also contribute to secondary ischemic damage and hematoma expansion. Thus, endothelial dysfunction and coagulopathy may represent important pathophysiological links complementing the role of systemic inflammation. In the context of T2DM, due to chronic low-grade inflammation and platelet hyperreactivity, SII levels are often persistently elevated, representing a triad of inflammation, immune dysregulation, and coagulation abnormalities. Thus, in patients with T2DM and sICH, SII may serve as a “sensitive indicator” of inflammation amplification, reflecting both the degree of tissue damage and the likelihood of poor outcomes. Dynamic monitoring of SII could potentially inform early risk identification and personalized intervention strategies. Importantly, SII encapsulates components of both the coagulation and immune-inflammatory pathways. While these systems function independently, in the complex pathological environment of T2DM combined with sICH, a synergistic interaction may exist. On the one hand, T2DM induces platelet hyperactivity and enhanced leukocyte adhesion, triggering mutual activation of both pathways and exacerbating thrombosis and tissue injury.⁴² On the other hand, an imbalance in the proportions of platelets and neutrophils within the SII may reciprocally affect coagulation, forming a vicious cycle of “inflammation–immunity–coagulation”.⁴³ Therefore, SII not only overcomes the limitations of single-pathway markers but also enables multidimensional risk assessment in this high-risk population. In summary, as a representative biomarker of both inflammation and coagulation, SII plays a dual but complementary role in the disease progression of patients with T2DM and sICH. By also reflecting endothelial dysfunction and coagulopathy, SII provides a broader mechanistic framework that captures the interplay of vascular injury, immune imbalance, and hemostatic disturbances. Its application can capture the pathophysiological process from multiple perspectives, offering vital biological and theoretical support for personalized treatment strategies, precise risk stratification, and multi-target interventions in clinical practice.

Although this study was carefully designed and rigorously conducted, with stable and reliable results, several important limitations should be acknowledged. First, this was a single-center retrospective study, with all data derived from the First Affiliated Hospital of Harbin Medical University. This may introduce regional bias and limits the generalizability of the findings to national or international populations of patients with T2DM and sICH; thus, caution is warranted in extrapolating the results. Second, data collection relied on existing medical records. Despite thorough cross-checking, some degree of missing information or inconsistent data entry was inevitable, which may have introduced information bias. Third, although multiple confounding variables were adjusted for, important factors such as infection severity, specific inflammatory biomarkers (eg, CRP, IL-6, high mobility group box 1), and the use of anti-inflammatory or anti-infective therapies were not included due to limitations in routine clinical testing and data completeness. However, we note that most patients included in this study were admitted in the acute phase of sICH without concurrent infection or systemic inflammation, and only a small proportion received anti-inflammatory or anti-

infective treatments. Thus, the potential confounding impact of these variables is likely limited, but their absence still restricts the comprehensiveness of the analysis. Fourth, this study only measured SII within the first 24 hours of admission and lacked dynamic monitoring throughout the disease course. Therefore, the temporal evolution of SII and its predictive value at different stages of illness could not be assessed. Fifth, although SII demonstrated good predictive performance in this sample, external validation in independent cohorts has not yet been conducted. Thus, its generalizability and practical clinical utility require further evidence. Sixth, although ROC curve analysis showed that SII had a relatively higher AUC for predicting long-term all-cause mortality compared to traditional indicators such as hematoma volume, GCS, and midline shift, its performance in predicting in-hospital mortality was only moderate and clearly lower than that of those established predictors. This limits the utility of SII in early in-hospital risk stratification and should be interpreted with caution. Additionally, the study did not examine the potential modulatory effects of treatments such as anticoagulation, glucose-lowering, or anti-inflammatory therapies on SII, nor did it explore interactions between interventions and biomarker changes, limiting the model's capacity to evaluate treatment response. Some subgroups (eg, those receiving anti-infective therapy or reoperation) had relatively small sample sizes, resulting in insufficient statistical power for interaction analyses and affecting interpretability. Therefore, future research should not only focus on multicenter prospective validation but also investigate specific mechanistic pathways linking SII with vascular injury, immune dysfunction, and coagulation abnormalities, and further evaluate whether SII-guided therapeutic strategies—such as intensified anti-inflammatory therapy, endothelial-protective interventions, or personalized anticoagulation management—can effectively reduce mortality and improve long-term recovery in this vulnerable population.

Conclusions

In conclusion, this study confirms that the SII is an independent predictor of both in-hospital and long-term all-cause mortality in patients with T2DM complicated by sICH. Given its ability to reflect the balance between systemic inflammation and immune response, SII may help clinicians identify patients at higher risk for poor outcomes at an early stage, thereby guiding the intensity of monitoring and therapeutic decisions such as the use of anti-inflammatory agents, immunomodulatory support, or more aggressive glucose and blood pressure control. This suggests that SII has immediate clinical utility as a simple and accessible biomarker for early risk stratification and individualized management during hospitalization and after discharge. In the future, SII may also serve as a useful tool for tailoring treatment strategies, monitoring response to therapy, and integrating into clinical decision-making algorithms, particularly in precision medicine approaches. Future multicenter, prospective studies are warranted to further validate its prognostic value, investigate the underlying mechanisms, and explore its broader clinical applications.

Data Sharing Statement

The datasets used during the current study are available from the corresponding author (Hongsheng Liang) on reasonable request.

Ethics Approval and Consent to Participate

This study protocol complies with the Declaration of Helsinki and was reviewed and approved by the First Affiliated Hospital of Harbin Medical University. Written informed consent was obtained from all patients.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests.

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