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Research Article

The Systemic Immune Inflammation Index as a Post-Stroke Disability Predictor in Acute Ischaemic Stroke Patients

Peran Indeks Sistem Imun-Inflamasi Sebagai Faktor Prediktor Prognosis Disabilitas Pasien Stroke Iskemik Akut

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Abstract

The Systemic Immune Inflammation (SII) Index is a biomarker that may help predict stroke prognosis. However, studies on the SII Index are still limited, particularly in Indonesia. The purpose of this study was to measure the SII Index as a predictor factor of disability-prognosis for acute ischemic stroke patients. This study used a retrospective cohort design. Subjects' data (112 subjects) were collected from STROKE REGISTRY RS Bethesda Yogyakarta. In this study, the independent variable was SII Index, whereas the dependent variables were mRS and BI. SII Index was analysed via ROC/AUC with mRS output to find the best cut-off value. Bivariate analyses were performed using the Spearman rho test to observe the correlation between risk factors and clinical outcomes. Risk factors with significant correlations would be re-analyzed with regression tests to observe the correlations affected by one another. Of 112 patients, the best cut-off for SII Index was 773.242 (sensitivity 92.9%; specificity 62.2%). Significant correlations were found between the SII Index and stroke severity with both mRS and BI ($p < 0.01$ for all). Significant results were shown on the regression tests between the SII Index and both mRS and BI ($p < 0.01$ for all). SII Index has been proven as a predictor factor of disability-prognosis for acute ischaemic stroke.

Keywords*: acute ischemic stroke, Systemic Immune Inflammation Index, modified Rankin Scale, Barthel Index, post stroke disability*

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Introduction

Stroke is defined by the World Health Organization as rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin. Stroke is the second leading cause of mortality and the leading cause of morbidity in the world.[1,2] According to the World Stroke Organization (WSO), more than 94 million people were living with ischemic stroke in 2024, with 12 million new strokes and 7 million deaths from strokes annually.[3] The prevalence of stroke in Indonesia was reported to be 8.2 per 1,000 population.[4]

Systemic Immune Inflammation (SII) Index is a prognostic biomarker in stroke, calculated as the absolute platelet count multiplied by the absolute neutrophil count, divided by the absolute lymphocyte count.[5] In acute ischemic stroke, an increasing SII Index describes an increasing inflammatory response in the brain, which correlates with the injury to the brain itself.[6–8] Compared with traditional inflammatory markers such as CRP or NLR, the SII Index was used because it integrates various immune cell components, providing a more comprehensive view of systemic inflammation and its impact on stroke outcomes.[9]

In inflammation of acute ischemic stroke, increased neutrophils interact with thrombocytes and form NETs. Alongside that process, lymphocyte senescence decreases lymphocyte production, hence producing a higher SII Index.[6–8] Increased SII Index correlates with poor prognosis in acute ischemic stroke patients shown by poor mRS (>2) and dependency-result on BI.[10,11] Several studies about SII on acute ischemic stroke proposed a cut-off value of SII, but no consensus has been reached. This study aimed to propose a cut-off for the SII Index and to determine its correlation with clinical outcomes of mRS for measuring disability and BI for measuring basic activity of daily living.

Methods

This study utilized a retrospective cohort design and consecutive sampling method with a population of 112 acute ischemic stroke patients from the neurological departments of Bethesda Hospital. All the risk factors data (both lab and clinical) and medications data were recorded upon hospital admission. The SII Index and stroke severity (NIHSS) were calculated on admission, and post-stroke disability was measured using mRS and BI 30 days after hospital admission. The NIHSS score is divided into low-to-moderate severity (score ≤ 10) and moderate-to-high severity (> 10).[12] The disability outcome, mRS categorized into well mRS (≤2) and poor mRS (>2).[11] The basic activity of daily living (BI) was categorized as total dependency (0–4), severe dependency (5–12), moderate dependency (13–18), mild dependency (19), or independent (20).[13] The SII

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Index formula was mentioned in the introduction, and the cut-off was found through AUC/ROC analysis on the result. This study was approved by the ethical committee of the Bethesda Hospital in ethical clearance No. 08/KEPK-RSB/II/24.

This study included patients who met the following criteria: 1) diagnosed with acute ischemic stroke through head CT-Scan or neurologist diagnosis; 2) over 18 years of age; 3) admitted to the hospital less than 24 hours; and 4) undergoing routine treatment at Bethesda Hospital Yogyakarta for 30 to 90 days as evidenced by medical record data. Patients were excluded from the study if they met any of the following criteria: 1) incomplete stroke registry data and medical records; 2) referred patient; 3) recurrent stroke; 4) active infection; 5) history of hematology and/or autoimmune disease; or 6) death before 30 days post diagnosis.

Data were analyzed using R Commander v 4.3.2. ROC analysis was performed to find the optimal cut-off of the SII Index. Univariate analysis was used to determine the subject characteristics. Spearman's bivariate analysis was used to find the correlation between input and output variables. Multivariate analysis (logistic regression) was used to find the weight of input variables with significantly correlated outputs in bivariate analysis.

Results

According to Table 1, 112 ischemic stroke patients were dominated by age <65 years, male, and without cardiovascular nor diabetes mellitus history. Acute ischemic stroke patients' severity was dominated by mild-moderate severity (99 people). Routine hematology laboratory results from subjects at admission were recorded. Median, IQR, and KS are shown in Table 2. Those data are presented as the median and interquartile range (IQR), also significance from Kolmogorov-Smirnov normality test (Table 2).

ROC Analysis (Figure 1) was used to find the optimum cut-off of the SII Index in this research. The ROC Analysis result is AUC, sensitivity, and specificity (Table 3 and Tables 6 and 7). Based on the analysis, the optimal cut-off of SII Index obtained was 773.242, with 92.9% sensitivity and 62.2% specificity. Then, the SII Index is categorized as high SII Index value (≥773.242) and low SII Index value (<773.242) for use in the Spearman rank test. From the Spearman rank correlation test, there were fair and significant correlations between the input variables (SII Index and stroke severity) and both output variables (mRS and BI) (p<0.05).

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Table 1 Basic Non-Laboratory Characteristics of the Patients

Characteristics	Frequency (n)	Percentage (%)
Age [14]		
• <65 (Young)	70	62.5%
• ≥65 (Old)	42	37.5%
Sex		
• Male	68	60.7%
• Female	44	39.3%
Diabetes Mellitus History		
• Positive	35	31.3%
• Negative	77	68.7%
Cardiovascular Disease History		
• Positive	19	17.0%
• Negative	93	83.0%
NIHSS Onset [12]		
• > 10 (moderate-high severity)	13	11.6%
• ≤ 10 (low-moderate severity)	99	88.4%
Disability (mRS) 30 days		
• Well (<=2)	98	87.5%
• Poor (>2)	14	12.5%
Activity of Daily Living (BI) 30 days [13]		
• Total dependency (0-4)	1	0.9%
• Severe dependency (5-12)	9	8.0%
• Moderate dependency (13-18)	15	13.4%
• Mild dependency (19)	25	22.3%
• Independent (20)	62	55.4%

Table 2 Basic Laboratory Characteristics of the Patients

Characteristics	Median (Q1-Q3 [IQR])
Absolute Leukocyte Count	8.760 (7.468 - 10.923 [3.455])
Absolute Neutrophil Count	5.819 (1.459 - 2.540 [1.080])
Absolute Lymphocyte Count	1.975 (4.452 - 7.034 [2.582])
Absolute Platelet Count	275.500 (219.750 - 322.250 [102.500])
Systemic Immune Inflammation Index	689.552 (484.465 - 1142.441 [657.976])

*Significant (p <0.05) by Kolmogorov-Smirnov Normality Test

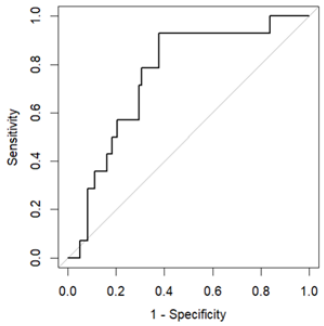

Figure 1 ROC Curve - SII Index based on mRS

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The other input variables are not significantly correlated with the output variables (p>0.05). Both SII Index and NIHSS were going to be tested on regression test (Table 4). The regression test found that the predictors (SII Index and NIHSS) significantly affect the clinical outcomes (mRS and BI). In the multivariate regression test, it could be seen how the predictors affected each other's clinical outcomes (Table 6 and Table 7).

Table 3 ROC Analysis Result

Cut-off	Sensitivity	Specificity	Youden Index
773.242	92.9%	62.2%	0.551

Table 4 AUC Analysis Result

Area	Standard error	*p* value	95% CI
0.75	0.026	<0.001*	0.63 – 0.88

*Significant (p <0.05) by AUC/ROC Test

Table 5 Spearman Bivariate Test

Variable	mRS		BI	
	Rho (*r*)	*p value*	Rho (*r*)	*p value*
SII Index	0.37	<0.001*	-0.39	<0.001*
Age	-0.01	0.88	-0.04	0.69
Sex (male)	0.08	0.38	0.10	0.28
Diabetes Mellitus	-0.02	0.82	-0.14	0.14
Cardiovascular Disease	0.12	0.22	-0.06	0.54
Stroke Severity (NIHSS)	0.37	<0.001*	-0.38	<0.001*

*Significant (p <0.05) by Spearman Rank Correlation test

Table 6 Logistic regression with outcome variable mRS

Covariate	Univariate regression		Multivariate regression	
	OR (95% CI)	*p* value	*OR* (95% CI)	*p* value
SII Index	21.43 (4.02 – 397.33)	0.004*	22.44 (3.86 – 436.86)	0.005*
NIHSS	9.75 (2.61 – 37.18)	<0.001*	10.45 (2.30 – 57.43)	0.003*

*Significant (p <0.05) by Logistic Regression Test

Table 7 Ordered Logistic Regression with outcome variable BI

Variables		Coef	SE	*OR*	*p* value
Univariate ordered logistic regression with predictor SII Index					
SII Index		-1.55	0.39	4.72‡	<0.01*
BI	≥1†	-5.68	1.04	<0.01§	<0.01*
	≥2†	-3.22	-0.42	0.04§	<0.01*
	≥3†	-2.07	-0.33	0.13§	<0.01*
	≥4†	-0.92	-0.28	0.40§	<0.01*
Univariate ordered logistic regression with predictor NIHSS					
NIHSS		-2.43	0.59	11.41‡	<0.01*
BI	≥1†	-5.46	1.05	<0.01§	<0.01*
	≥2†	-2.90	0.40	0.06§	<0.01*

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Variables		Coef	SE	*OR*	*p* value
	≥3†	-1.62	0.26	0.20 §	<0.01*
	≥4†	-0.44	0.20	0.64 §	0.03*
Multivariate ordered logistic regression					
SII Index		-1.60	0.3970	4.95‡	<0.01*
NIHSS		-2.5	0.6047	12.37‡	<0.01*
BI	≥1†	-6.64	1.1241	<0.01§	<0.01*
	≥2†	-3.87	0.4978	0.02§	<0.01*
	≥3†	-2.47	0.3640	0.08§	<0.01*
	≥4†	-1.17	0.2960	0.31§	<0.01*

*Significant (p <0.05) by Ordered Logistic Regression Test; † BI interpretations, 0 = total dependency, 1 = severe dependency, 2 = moderate dependency, 3 = mild dependency, 4 = independent ‡ base *OR* (e^{coef}); § *OR* (e^{-coef}); Coef = Coefficient

Discussion

In acute ischemic stroke, cytokines play a major role in the neuroinflammatory process. Pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 increase neural tissue damage by worsening blood-brain barrier permeability, triggering oedema, and aggravating neuronal injury, whereas anti-inflammatory cytokines such as IL-10 function to protect tissue from further damage.[6,15] In addition to cytokines, immune cells also play an important role, particularly neutrophils, lymphocytes, and platelets. Neutrophils will interact with platelets through P-selectin/PSGL1 and stabilize the thrombus with NETs (Neutrophil-Extracellular Traps) formation, thereby worsening atherosclerosis and plaque stability.[16]

Lymphocytes, especially Th (T helper) cells, played a critical role in both inflammation and anti-inflammation. Th cells are categorized mainly into Th1 and Th2 cells. Th1 cells would respond to various proinflammatory cytokines in the area surrounding the ischemic lesion to attract CD8+ cells to the lesion area, while anti-inflammatory cytotoxic cells will attract Th2 cells to the lesion core to further release anti-inflammatory cytokines.[7,15] In addition, lymphocytes Treg worked to suppress excessive cytotoxins released by CD8+ cells and also release anti-inflammatory cytokines, especially IL-10.[6,7]

The dynamics of lymphocyte composition were influenced by many factors. The mutually affecting proinflammatory and anti-inflammatory composition leads to decreased costimulatory signals in T cells.[17] The decreases in co-stimulation, along with ischemia, resulted in the programmed apoptosis or senescence of these T cells.[17,18] Senescent T cells experienced reduced sensitization and could become resistant to apoptosis signals.[6,7] Anergic T cells, such as Treg cells, could suppress inflammation in cerebral ischemia, but $CD28^{null}$ T cells could lead to a decrease in T cell progenitors and a reduction in mature B cells in the bone marrow while still increasing cytotoxicity and enhancing proinflammatory cytokines.[17]

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Those immune mechanisms could be affected by several factors such as non-modifiable factors (age and sex) and modifiable factors (diabetes and cardiovascular disease).[6,19] Age and sex have been shown to affect immune responses and susceptibility to stroke by hormonal changes (especially in women) and decline in vascular capability and regenerative ability, respectively.[14,19] However, previous studies have not found a significant correlation between these two intrinsic factors and clinical stroke outcomes.[11]

Diabetes mellitus promotes inflammation in the vasculature, reduces vascular elasticity, and may even form emboli that could block the blood flow to the brain.[20] The phenomena of changes in vascular elasticity and emboli could also be achieved by cardiovascular diseases.[21] A history of diabetes mellitus and cardiovascular disease over the long term brought changes to the vasculature and the performance of organs due to immune inflammatory responses that adapt to become higher than in people without that history of diseases.[22,23] The higher the inflammatory response, the greater the thrombo-inflammation via NETs formation, hence worsening the outcome of stroke. [6,20,21,24,25] Interestingly, in the analysis conducted, no significant relationship was found between the comorbid disease group and clinical stroke outcomes in this study. According to the previous study, this might be due to the use of prophylaxis, as well as the stable condition of patients' diabetes mellitus and/or cardiovascular disease, but these data were not further analysed in this study.[26–29]

The results on the SII index were predictive of the prognosis of acute ischemic stroke disability. This study used the SII Index cut-off found in the study, which was 773.242, with sensitivity (92.9%) and specificity (62.2%). The SII Index was fairly and significantly correlated with the clinical outcomes mRS ($r = 0.37$, $p < 0.001$) and BI ($r = -0.39$ and $p < 0.01$). These results support previous research stating that the SII Index was higher in patients with poor prognosis.[5]

It was found from the NIHSS score that moderate-mild severity strokes (NIHSS ≤ 10) were the most common in the study population, 70 patients (71.4%). NIHSS numbers could be used to describe the inflammatory severity of stroke through several mechanisms such as edema, inflammatory mediators, calcium excitotoxicity, and oxidative stress.[30] Measurement of NIHSS on admission of patients with acute ischemic stroke can also describe patients' prognosis.[31] This is proven by the fair correlation between NIHSS with mRS ($r = 0.37$ and $p < 0.01$) and BI ($r = -0.38$ and $p < 0.01$).

Logistic regression analysis of SII and NIHSS indices in mRS clinical outcome showed that patients with high SII Index (≥ 773.242) and with moderate-severe NIHSS (>10) had a higher risk of a poorer mRS (>2) with *OR* 22.44 ($p = 0.005$) and *OR* 10.45 ($p = 0.003$), respectively. Analysis of ordered logistic regression of SII and NIHSS Index in BI clinical outcome showed

that a high SII Index (≥773.242) caused an increase in the adjusted-*OR* of BI's base adjusted-*OR* of 4.95 and moderate-severe NIHSS (>10) led to an increase in the adjusted-*OR* of BI's base adjusted-*OR* of 12.37 from BI's base adjusted-*OR* (p<0.01 and p<0.01 respectively).

Regression tests of the input variables of the SII Index and NIHSS showed that the adjusted-*OR* is higher than the unadjusted-*OR*. This indicates the presence of a confounding variable that affects both input variables. When viewed from the concepts of the SII Index and NIHSS, these two variables describe the existence of an inflammatory process in different perspectives. This suggests the existence of another inflammatory mechanism that affects both variables, which were not studied or found in this study.[32]

Previous study results on the SII Index and NIHSS support this finding. Huang (2023) showed that patients with moderate to severe acute ischemic stroke had higher SII values compared to patients with mild stroke, with a statistically significant difference ($p < 0.001$).[33] Wu et al. (2022) also reported that the highest SII quartile was associated with an increased risk of mortality in patients with acute ischemic stroke, reinforcing the evidence that high SII values reflect more severe systemic inflammatory processes and have a direct impact on the clinical outcomes of patients.[34]

The limitations of our study were the alteration of the SII index before and after the medical treatment and time window after the onset of stroke until the whole blood test was taken. Those limitations might also be studied for future research.

Conclusion

The Systemic Immune Inflammation Index has proven useful as a predictor of disability prognosis in acute ischemic stroke patients.

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