RESEARCH PAPER

Association and Predictive Value of the Systemic Immune-Inflammation Index and Systemic Inflammation Response Index for White Matter Hyperintensity Severity in Cerebral Small Vessel Disease

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ABSTRACT

Background and Objective: Chronic systemic inflammation plays a pivotal role in the pathogenesis of cerebral small vessel disease (CSVD) and the development of white matter hyperintensities (WMH). The Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) have emerged as readily available inflammatory biomarkers; however, evidence comparing their association and predictive performance for WMH severity remains limited. This study aimed to evaluate the association and predictive value of SII and SIRI for WMH severity based on the Fazekas score in patients with CSVD.

Methods: This retrospective cross-sectional study included 210 patients with CSVD who underwent brain magnetic resonance imaging and complete blood count testing at Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia. SII was calculated as platelet × neutrophil/lymphocyte, whereas SIRI was calculated as neutrophil × monocyte/lymphocyte. WMH severity was assessed using the Fazekas scale. Spearman's correlation was performed to evaluate the associations between inflammatory indices and WMH severity, while receiver operating characteristic (ROC) analysis assessed their predictive performance.

Results: Both SII and SIRI were significantly associated with WMH severity. SII demonstrated a moderate positive correlation ($r = 0.488$, $p < 0.001$), whereas SIRI showed a stronger positive correlation ($r = 0.687$, $p < 0.001$). ROC analysis demonstrated that SII yielded an area under the curve (AUC) of 0.780 (95% CI, 0.715–0.846), while SIRI showed superior predictive performance with an AUC of 0.897 (95% CI, 0.854–0.941). The optimal cutoff values were 666.7 for SII and 1.056 for SIRI.

Conclusions: Both SII and SIRI were significantly associated with WMH severity in patients with CSVD. SIRI demonstrated stronger correlations and superior predictive accuracy, suggesting its potential as a practical, non-invasive biomarker for clinical risk stratification.

Keywords: Cerebral Small Vessel Diseases; White Matter; Magnetic Resonance Imaging; Systemic Immune-Inflammation Index; Systemic Inflammation Response Index.

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INTRODUCTION

Stroke remains one of the leading causes of mortality and long-term disability worldwide, with cerebral small vessel disease (CSVD) recognized as a major cause of ischemic stroke, cognitive decline, gait impairment, and vascular dementia [1,2]. Among the neuroimaging markers of CSVD, white matter hyperintensities (WMH) are the most common manifestation and are strongly associated with adverse neurological outcomes [1,3]. WMH severity is routinely assessed using the Fazekas scale, a simple and reproducible visual grading system widely applied in clinical and research settings [3,4].

Chronic systemic inflammation has emerged as a key mechanism underlying CSVD progression through endothelial dysfunction, blood–brain barrier disruption, and persistent white matter injury [5,6]. Consequently, inflammation-based biomarkers derived from routine

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blood tests have gained increasing interest as practical tools for assessing disease severity [7].

The Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) are composite inflammatory indices calculated from peripheral blood cell counts that reflect the interaction between innate immunity, adaptive immunity, thrombosis, and vascular inflammation [7,8]. Previous studies have shown that elevated SII is associated with increased WMH burden, greater overall CSVD severity, and WMH progression, while higher SIRI has been linked to more severe white matter injury and poorer neurological outcomes [9–13].

However, evidence directly comparing the association and predictive performance of SII and SIRI for WMH severity based on the Fazekas score remains limited, particularly in Asian populations. Therefore, this study aimed to investigate the association and predictive value of SII and SIRI for WMH severity in patients with CSVD, with the hypothesis that higher inflammatory indices are associated with more severe WMH and may serve as accessible biomarkers for clinical risk stratification.

MATERIALS AND METHODS

Study Design and Setting

This retrospective cross-sectional analytical study was conducted at Dr. Wahidin Sudirohusodo Hospital in Makassar, Indonesia. Medical records of patients diagnosed with CSVD were reviewed during the 2026 study period to investigate the association and predictive value of the SII and SIRI for the severity of WMH.

Study Population

Adult patients (≥18 years) diagnosed with CSVD based on brain magnetic resonance imaging (MRI) were consecutively screened. Eligible participants had at least one vascular risk factor (hypertension, diabetes mellitus, or dyslipidemia), underwent brain MRI demonstrating WMH, and had complete blood count results available within seven days before or after the MRI examination. Patients were excluded if they had a history of large territorial ischemic stroke, massive intracerebral hemorrhage, severe traumatic brain injury, other neurological disorders affecting white matter (including multiple sclerosis, hereditary leukoencephalopathy, encephalitis, brain tumors, or chronic hydrocephalus), acute infections, active autoimmune diseases, hematologic disorders, or active malignancy. Records with incomplete MRI findings, missing hematological data, or an interval exceeding seven days between blood sampling and MRI were excluded from the final analysis.

Sample Size

The sample size was calculated for correlation analysis because no previous study had specifically evaluated the relationship between SII, SIRI, and WMH severity in patients with CSVD. Assuming a moderate correlation coefficient ($r = 0.30$), a two-sided significance level of 0.05, and 80% statistical power, the minimum required sample size was 85 participants. After accounting for an anticipated 15% rate of incomplete data, at least 100 patients were targeted for inclusion.

Data Collection and Variables

Demographic characteristics, vascular risk factors, laboratory findings, and brain MRI data were retrospectively extracted from electronic medical records using a standardized data collection form. Demographic variables included age and sex, while vascular risk factors comprised hypertension, diabetes mellitus, and dyslipidemia. Laboratory data were obtained from routine complete blood count examinations performed within seven days of the MRI examination and included absolute neutrophil, lymphocyte, monocyte, and platelet counts.

The SII was calculated by multiplying the absolute platelet count by the neutrophil count and dividing the result by the lymphocyte count, whereas the SIRI was calculated by multiplying the neutrophil count by the monocyte count and dividing the result by the lymphocyte count. The primary outcome was the severity of WMH on brain MRI. WMH was evaluated on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences using the Fazekas visual rating scale, in which periventricular and deep white matter lesions were graded separately on a scale of 0 to 3. The combined Fazekas score was subsequently categorized into mild (scores 1–2), moderate (scores 3–4), and severe (scores 5–6) WMH for subsequent analyses.

Statistical Analysis

Continuous variables were presented as mean ± standard deviation or median (interquartile range), depending on data distribution, while categorical variables were summarized as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. The associations between SII, SIRI, and WMH severity were evaluated using Spearman's rank correlation coefficient because WMH severity was treated as an ordinal variable. Receiver operating characteristic (ROC) curve analysis was subsequently performed to evaluate the discriminatory performance of SII and SIRI for predicting moderate-to-severe WMH. The optimal cutoff values were determined using the Youden index, and diagnostic performance was reported as the area under the ROC curve (AUC), sensitivity, specificity, positive predictive value, and negative predictive value. Statistical analyses were performed using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA), with statistical significance defined as a two-sided $p < 0.05$.

Ethical Considerations

The study protocol was approved by the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (Approval No. 361A/UN4.6.4.5.31/PP36/2026). As this study retrospectively analyzed anonymized medical records without direct patient involvement, the ethics committee waived the requirement for informed consent.

RESULTS

Baseline Characteristics of the Study Population

A total of 210 patients with CSVD were included in the analysis. The baseline demographic, clinical, and laboratory characteristics are presented in Table 1. The mean age of the participants was 57.45 ± 11.46 years. Male patients accounted for 58.1% (122/210) of the study population, while females comprised 41.9% (88/210). Hypertension was the most prevalent vascular risk factor, affecting 68.6% (144/210) of patients, followed by diabetes mellitus in 23.8% (50/210) and dyslipidemia in 10.5% (22/210).

Regarding white matter hyperintensity (WMH) severity, 47.6% (100/210) of patients had a Fazekas score of 1, 36.7% (77/210) had a score of 2, and 15.7% (33/210) had a score of 3. The median SII was 758.68 (range, 51.18–9223.37), whereas the median SIRI was 1.19 (range, 0.13–89.37), indicating substantial inter-individual variability in systemic inflammatory status. Overall, the study population consisted predominantly of middle-aged to older adults with a high prevalence of hypertension and predominantly mild-to-moderate WMH severity.

Association of SII and SIRI with White Matter Hyperintensity Severity

The associations between the SII, SIRI, and WMH severity were evaluated using Spearman's rank correlation because SII and SIRI were non-normally distributed continuous variables, and WMH severity, based on the Fazekas score, was analyzed as an ordinal variable. As shown in Table 2, both inflammatory indices were significantly associated with WMH severity. SII demonstrated a moderate positive correlation with WMH severity (Spearman's $r = 0.488$, $p < 0.001$), whereas SIRI showed a strong positive correlation (Spearman's $r = 0.687$, $p < 0.001$). These findings indicate that higher SII and SIRI values were associated with greater WMH severity, with SIRI exhibiting a stronger correlation than SII.

Receiver Operating Characteristic Analysis of SII and SIRI for Predicting WMH Severity

ROC curve analysis was conducted to assess the discriminatory performance of the SII and SIRI in predicting severe WMH. The results are summarized in Table 3, and the ROC curves are shown in Figure 1. SII demonstrated a significant ability to discriminate severe WMH, with an AUC of 0.780 (95% CI, 0.715–0.846; $p < 0.001$). The optimal cutoff value was 666.7, yielding a sensitivity of 82.7% and a specificity of 72.0%. SIRI exhibited superior diagnostic performance, with an AUC of 0.897 (95% CI, 0.854–0.941; $p < 0.001$). The optimal cutoff value was 1.056, corresponding to a sensitivity of 87.3% and a specificity of 84.0%.

As illustrated in Figure 1, both ROC curves were positioned above the reference line, indicating significant discriminatory ability for identifying severe WMH. The ROC curve for SIRI was consistently farther from the diagonal line and closer to the upper-left corner than that of SII, reflecting its superior predictive performance. Overall, although both inflammatory indices demonstrated good diagnostic accuracy, SIRI exhibited better discrimination than SII for predicting WMH severity.

DISCUSSION

The present study demonstrated that both the SII and the SIRI are significantly associated with the severity of WMH in patients with CSVD. Notably, SIRI exhibited a stronger correlation and superior predictive performance than SII. These findings support the growing evidence that systemic inflammation plays a central role in the pathogenesis and progression of CSVD.

The study population consisted predominantly of middle-aged and older adults, aligning with the established epidemiology of CSVD. Aging is a major determinant of CSVD because progressive endothelial dysfunction, arterial stiffening, impaired cerebrovascular autoregulation, and chronic cerebral hypoperfusion promote white matter injury and the development of WMH [1,3]. These findings are consistent with those reported by Debette and Markus (2010), who demonstrated that both the prevalence and severity of WMH increase substantially with advancing age [14]. Additionally, hypertension was the most prevalent vascular risk factor in our cohort, reinforcing its well-recognized role in CSVD pathogenesis. Chronic hypertension induces lipohyalinosis, arteriolar wall thickening, and luminal narrowing, resulting in endothelial dysfunction and chronic ischemic injury of the cerebral white matter [3,15]. Similarly, van den Liu et al. (2023) reported that poor blood pressure control was independently associated with accelerated WMH progression [16]. Although diabetes mellitus and dyslipidemia were less common in the present study, previous evidence suggests that their contribution to CSVD depends on disease duration and metabolic control rather than their mere presence [17].

Our findings demonstrated significant positive correlations between both inflammatory indices and WMH severity, indicating that greater systemic inflammatory activity is associated with more advanced white matter injury. These findings align with those of Nam et al. (2022), who reported that elevated SII was significantly associated with increased WMH burden [9]. Similarly, Xiao et al. (2024) found that higher SII correlated with a greater overall CSVD burden, while Del Brutto et al. (2023) showed that increased SII independently predicted longitudinal WMH progression [10,11]. Collectively, these studies support the concept that persistent systemic inflammation contributes to progressive cerebral microvascular injury and white matter degeneration.

The stronger association observed for SIRI compared with SII is biologically plausible. Unlike SII, which reflects the interaction between neutrophils, platelets, and lymphocytes, SIRI additionally incorporates circulating monocytes, an important mediator of chronic vascular inflammation. Activated monocytes infiltrate the vascular

wall, differentiate into macrophages, and release pro-inflammatory cytokines and reactive oxygen species, thereby promoting endothelial dysfunction, blood-brain barrier disruption, and chronic white matter damage [1,15]. This mechanism may explain why SIRI showed a stronger relationship with WMH severity in the present study. Similar findings have been reported by Zhang et al. (2021), Zhou et al. (2022), and Zhuang et al. (2025), who demonstrated that elevated SIRI was associated with more severe cerebrovascular injury and poorer neurological outcomes [12,13,18].

ROC analysis further demonstrated that both SII and SIRI possessed significant discriminatory ability for identifying severe WMH, although SIRI consistently outperformed SII. To our knowledge, few previous studies have directly compared the predictive performance of these two inflammatory indices for WMH severity. Nevertheless, our findings are consistent with previous investigations showing that composite inflammatory biomarkers exhibit good diagnostic performance in cerebrovascular diseases and may help in clinical risk stratification [19]. The superior predictive accuracy of SIRI observed in this study further supports its potential clinical utility as a readily available biomarker for identifying patients with advanced CSVD.

The association between systemic inflammation and WMH is supported by several biological mechanisms. Chronic inflammation contributes to endothelial activation, oxidative stress, microvascular remodeling, and increased permeability of the blood-brain barrier, leading to persistent hypoperfusion, demyelination, and axonal injury within the cerebral white matter [3,5,6]. Since SII and SIRI are derived from routine complete blood counts, they provide practical and inexpensive measures of systemic inflammatory status and may thus serve as useful adjunctive biomarkers in routine clinical practice.

Despite these promising findings, CSVD remains a multifactorial disease influenced by aging, hypertension, diabetes mellitus, dyslipidemia, and other vascular risk factors, in addition to inflammation [3,15]. Therefore, SII and SIRI should not be considered standalone diagnostic markers but rather complementary biomarkers that may improve clinical risk assessment when interpreted alongside neuroimaging findings and conventional vascular risk factors. Future prospective multicenter studies with longitudinal follow-up are warranted to validate the optimal cutoff values identified in this study and to determine whether these inflammatory indices can predict WMH progression and long-term clinical outcomes.

CONCLUSIONS

In conclusion, both the SII and the SIRI were significantly associated with the severity of WMH in patients with CSVD. Although both inflammatory indices demonstrated good discriminatory performance for predicting severe WMH, SIRI consistently showed stronger correlations and superior predictive accuracy compared with SII. These findings suggest that SII, and particularly SIRI, may serve as accessible, non-invasive biomarkers for assessing WMH severity and facilitating clinical risk stratification in patients with CSVD. Nevertheless, given the multifactorial nature of CSVD, these biomarkers should be interpreted in conjunction with neuroimaging findings and established vascular risk factors rather than as standalone diagnostic tools. Further prospective multicenter studies are warranted to validate these findings and determine their prognostic value for disease progression and clinical outcomes.

DECLARATIONS

Consent for Publication

Not applicable.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

SA conceived and designed the study, collected the data, performed the statistical analysis, interpreted the results, and drafted the manuscript. **AKB**, **GVS**, **YAF**, **UA**, and **CR** contributed to study design, data interpretation, critical revision of the manuscript, and approved the final version for publication. All authors read and approved the final manuscript.

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TABLES AND FIGURE

Table 1. Baseline Characteristics of the Study Population

Variable	Total (n = 210)
Age (years), mean ± SD	57.45 ± 11.46
Sex, n (%)	
Male	122 (58.1)
Female	88 (41.9)
Hypertension, n (%)	
Yes	144 (68.6)
No	66 (31.4)

Diabetes mellitus, n (%)	
Yes	50 (23.8)
No	160 (76.2)
Dyslipidemia, n (%)	
Yes	22 (10.5)
No	188 (89.5)
White matter hyperintensity (WMH) severity, n (%)	
Fazekas score 1	100 (47.6)
Fazekas score 2	77 (36.7)
Fazekas score 3	33 (15.7)
Systemic Immune-Inflammation Index (SII), median (range)	758.68 (51.18–9223.37)
Systemic Inflammation Response Index (SIRI), median (range)	1.19 (0.13–89.37)

Table 2. Correlation Between the Systemic Immune-Inflammation Index (SII), Systemic Inflammation Response Index (SIRI), and White Matter Hyperintensity (WMH) Severity

Variable	WMH Severity
SII	Spearman's r = 0.488p < 0.001n = 210
SIRI	Spearman's r = 0.687p < 0.001n = 210

Table 3. Receiver Operating Characteristic (ROC) Analysis of the Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) for Predicting White Matter Hyperintensity (WMH) Severity

Variable	AUC	Standard Error	p Value	95% Confidence Interval	
				Lower Limit	Upper Limit
SII	0.780	0.033	< 0.001	0.715	0.846
SIRI	0.897	0.022	< 0.001	0.854	0.941

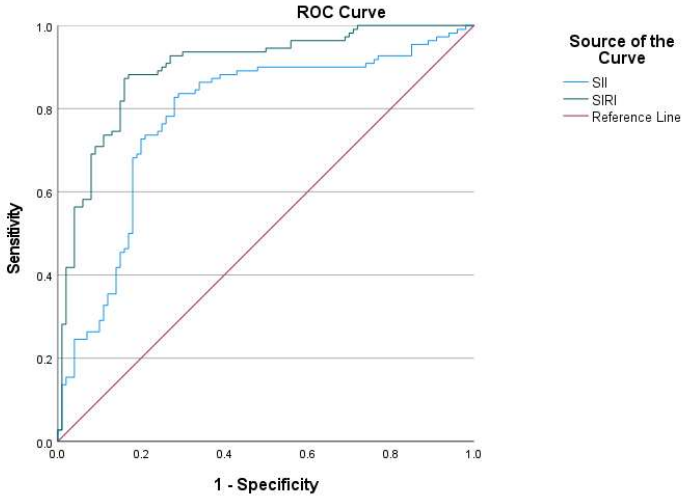

Figure 1. Receiver Operating Characteristic (ROC) Curve of the Systemic Immune-Inflammation Index (SII) for Predicting White Matter Hyperintensity (WMH) Severity

Supplementary Material

Table 4. Sensitivity and Specificity at Various Cut-off Values for SII and SIRI

Cut-Off	Value	Sensitivity	Specificity	Youden Index
SII	638.27	0.836	0.70	0.536
	650.91	0.836	0.71	0.546
	666.73	0.827	0.72	0.547
	678.14	0.818	0.72	0.538
	684.37	0.800	0.72	0.520
	691.56	0.791	0.72	0.511
SIRI	0.959	0.882	0.800	0.682
	0.987	0.882	0.820	0.702
	0.993	0.882	0.830	0.712
	1.056	0.873	0.840	0.713

	1.078 1.101	0.864 0.855	0.840 0.840	0.704 0.695

The optimal cut-off value was identified using the Youden index, calculated as sensitivity + specificity − 1.