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SII AND SIRI AS PREDICTORS OF IN-HOSPITAL MACE IN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT
Major adverse cardiac events (MACE) are a leading cause of morbidity and mortality in acute myocardial infarction (AMI). Systemic inflammation plays a key role in atherosclerosis, plaque rupture, and thrombosis. The Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) are novel biomarkers that may support early risk stratification, but data from Indonesia are limited. Objective to assess the association between SII, SIRI, and MACE in AMI patients at Adam Malik Hospital, Medan. Methods: This cross-sectional study used a total sampling technique, including 224 AMI patients hospitalized from January–December 2023. SII and SIRI were calculated from neutrophil, lymphocyte, monocyte, and platelet counts. MACE during hospitalization was recorded. Mann–Whitney U and logistic regression were used ($p < 0.05$). Of 224 patients, 79.5% were male with a mean age of 56.49 years. STEMI accounted for 63.8% of cases, and hypertriglyceridemia was the most common comorbidity (27.2%). MACE occurred in 57 patients (25.4%), including 15 deaths (6.7%). Mean SII was 1090.52 and mean SIRI was 3.83. Both SII ($p = 0.002$) and SIRI ($p = 0.001$) were significantly associated with MACE. SIRI predicted MACE with an OR of 1.233 (95% CI: 1.151–1.320). SII and SIRI are significantly associated with MACE in AMI, but only SIRI shows predictive value for adverse outcomes.

Keywords: acute myocardial infarction; MACE; SII; SIRI

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INTRODUCTION

Cardiovascular disease (CVD) remains the foremost cause of global morbidity and mortality, posing a significant burden on public health systems worldwide. According to World Health Organization (WHO), approximately 17.9 million people died from CVD in 2019, with 85% of these deaths attributable to acute myocardial infarction (AMI) and stroke. (World Health Organization, 2021) AMI affects an estimated 126 million individuals globally (1.72% of the population), with a notably higher prevalence among men, particularly those over the age of 40. Over the past two decades, the epidemiological burden of CVD has shifted from high-income to low- and middle-income countries, where more than three-quarters of all cardiovascular deaths now occur.(Martin et al., 2024)

In Indonesia, the prevalence of heart disease increased from 0.5% in 2013 to 1.5% in 2018 based on the Riset Kesehatan Dasar 2018. Cardiovascular mortality also rose from 356.05 to 412.46 deaths per 100,000 men between 2000 and 2019. (Harmadha et al., 2023) In Sumatera Utara, the prevalence of heart disease was 1.3%, with a higher prevalence among women. At RSUP Haji

Adam Malik Medan, 378 cases of AMI were recorded in 2023, underscoring the growing burden of this condition at the regional level. (Riskesd, 2019).

Inflammation is central to the pathophysiology of atherosclerosis, plaque rupture, and thrombosis—the core mechanisms underlying AMI. Numerous studies have demonstrated that leukocytes and their components, including neutrophils, lymphocytes, and monocytes, play pivotal roles in the development and progression of coronary artery disease (CAD). Furthermore, these hematological parameters are inexpensive and easily accessible, making them valuable as potential biomarkers for risk prediction in cardiovascular events.(Jin et al., 2021). Recently, novel inflammatory markers such as the Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) have emerged as promising prognostic indicators. Unlike single-ratio markers such as the neutrophil-to-lymphocyte ratio (NLR) or platelet-to-lymphocyte ratio (PLR). (Jin et al., 2021) SII and SIRI integrate neutrophil, lymphocyte, monocyte, and platelet counts, providing a more comprehensive representation of both the inflammatory and immune response. Elevated SII and SIRI levels have been associated with poor clinical outcomes in various diseases, including malignancies and cardiovascular disorders.(Geng et al., 2018)

In the cardiovascular context, increased SII has been linked to more severe coronary stenosis, with previous studies identifying a cut-off value of 652.83 that yielded a sensitivity of 71.0% and specificity of 86.0%. (Liu et al., 2021) Elevated SIRI has also been shown to independently predict new-onset atrial fibrillation in patients with ST-elevation myocardial infarction (STEMI) undergoing percutaneous coronary intervention. (Wang et al., 2022) Furthermore, patients with STEMI exhibit higher SII and SIRI levels than those with stable CAD. A cohort study by Wei et al. demonstrated that SII and SIRI showed strong predictive ability for major adverse cardiac events (MACE), with good sensitivity and specificity at defined cut-off values.(Wei et al., 2023).

MACE—including outcomes such as recurrent infarction, arrhythmia, heart failure, cardiogenic shock, and death—remains a critical determinant of short- and long-term prognosis in AMI patients. Previous research has reported MACE incidence rates ranging from 4.2% to 51% in patients with STEMI, with events occurring from the first day of hospitalization up to 10 years post-infarction. (Widyastuti W, 2023) Early identification of high-risk patients is therefore essential for guiding timely clinical decision-making and improving patient outcomes. Despite the availability of various risk assessment tools, their clinical application is often limited by complexity, cost, or the need for advanced laboratory testing—constraints that are especially relevant in resource-limited settings.

In this context, SII and SIRI offer potential advantages as prognostic tools. These indices can be rapidly derived from routine complete blood counts, which are widely available, cost-effective, and non-invasive. However, evidence regarding their predictive value for MACE in the Indonesian population remains limited. Therefore, this study aims to investigate the association between SII and SIRI values and the occurrence of in-hospital MACE in patients with acute myocardial infarction at Adam Malik Hospital. Establishing the prognostic utility of these biomarkers in a local clinical setting could provide a simple and accessible approach for risk prediction, complementing existing diagnostic and prognostic models in AMI management.

METHOD

This study was designed as an observational cross-sectional analysis and was conducted at RS Adam Malik Medan. The research included patients admitted with a diagnosis of acute myocardial infarction (AMI) between January and December 2023. A total of 224 patients who met the inclusion criteria were enrolled using a total sampling technique. Eligible participants were adults with a confirmed AMI diagnosis based on typical clinical symptoms, electrocardiographic findings, and elevated cardiac biomarkers. Patients with hematological malignancies, autoimmune diseases, or incomplete medical records were excluded to minimize confounding factors. Data were collected

retrospectively from medical records.

Demographic characteristics, comorbidities, and clinical presentations were recorded at the time of admission. Laboratory examinations, including complete blood count and troponin I measurements, were performed routinely upon hospital admission. The hematological parameters were analyzed using an automated hematology analyzer (Sysmex 1000). From these parameters, the Systemic Immune-Inflammation Index (SII) and the Systemic Inflammation Response Index (SIRI) were calculated. The SII was obtained by multiplying the platelet count by the neutrophil count and dividing the result by the lymphocyte count, while the SIRI was calculated by multiplying the neutrophil count by the monocyte count and dividing by the lymphocyte count. All laboratory values were expressed in $\times 10^9$/L.

Major Adverse Cardiac Events (MACE) were defined as a composite of in-hospital mortality, heart failure, cardiogenic shock, reinfarction, and clinically significant arrhythmias. These events were identified through medical records and verified by the attending cardiologist to ensure diagnostic accuracy. Statistical analysis was performed using standard statistical software Numerical data were presented as mean ± standard deviation or median with interquartile range, depending on data distribution, whereas categorical variables were presented as frequencies and percentages. Comparisons between patients with and without MACE were analyzed using the Mann–Whitney for continuous variables. To determine independent predictors of MACE, binary logistic regression analysis was applied. A p-value < 0.05 was considered statistically significant.

RESULT

A total of 224 patients with acute myocardial infarction (AMI) were included in this study. The majority of patients were male (79.5%) with a mean age of 56.49 ± 10.39 years. Most patients were diagnosed with STEMI (63.8%), while NSTEMI accounted for 36.2% of cases. The most common comorbidity was hypertriglyceridemia (27.2%), followed by hypertension and diabetes mellitus.

Table 1.
Respondent Characteristics with Acute Myocardial Infarction

Characteristics	n = 224
Gender, n (%)	
Male	178 (79,5)
Female	46 (20,5)
Age, (mean ± SD)	56,49 ± 10,39
18 – 38	4
39 – 59	138
60 – 80	82
Length of Hospital Stay, days	4 (1 – 23)
Comorbidities, n (%)	
Diabetes Mellitus	51 (22,8)
Hypertension	50 (22,3)
Diabetes and Hypertension	52 (23,2)
Hypercholesterolemia, n (%)	40 (17,9)
Hypertriglyceridemia, n (%)	61 (27,2)
None	71 (31,7)
AMI Type, n (%)	
NSTEMI	81 (36,2)
STEMI	143 (63,8)

Based on hematological analysis (Table 4.2), the mean platelet count was 246,948 ± 88,745/µL, neutrophils 8,47 (2,26 – 26,03) 10^3/µL, lymphocytes 1,88 (0,22 – 7,45) 10^3/µL, and monocytes 0,88 (0,12 – 2,86)10^3/µL. The mean SII value was 1,090.52 (range: 214.04–9,298.8) and the mean SIRI value was 3.83 (range: 0.35–31.19).

Table 2.
Laboratory Characteristics in Patients with Acute Myocardial Infarction

Laboratory Characteristics	n = 224
Platelet countt, 10^9/L	246,5 (123 – 556)
Leukocyte count, 10^3/ µL	11,66 (4,58 – 29,85)
Absolute neutrophil count, 10^3/ µL	8,47 (2,26 – 26,03)
Absolute lymphocyte count, 10^3/ µL	1,88 (0,22 – 7,45)
Absolute monocyte count, 10^3/ µL	0,88 (0,12 – 2,86)
Troponin I, ng/mL	11,56 (0,01 – 15)
Total cholesterol, mg/dL	166,77 ± 44,56
Triglycerides, mg/dL	148,24 (41 – 559)
LDL cholesterol, mg/dL	108 (15 – 276,4)
HDL cholesterol, mg/dL	38 (12 – 84)
SII	1090,52 (214,04 – 9298,8)
SIRI	3,83 (0,35 – 31,19)

Data presented as mean±SD or median (min-max)

During hospitalization, 57 patients (25.4%) experienced major adverse cardiovascular events (MACE). The most frequent event was in-hospital death (6.7%), followed by heart failure (6.3%), ventricular tachycardia (4.9%), stroke (4.5%), and cardiogenic shock (3.1%). (Table 3)

Table 3.
Distribution of Major Adverse Cardiac Events (MACE)

MACE Event	n = 57
Heart Failure	14 (6,3)
Stroke	10 (4,5)
Cardiogenic Shock	7 (3,1)
Ventricular tachycardia	11 (4,9)
Mortality	15 (6,7)

Table 4.
Association of SII and SIRI with in-hospital MACE

Variable	MACE		*p-value*
	Yes (n=57)	No (n=167)	
SII	1634,67 (130,44-5596,67)	1046,34 (104,89-9298,8)	0,002
SIRI	6,06 (0,35 – 31,19)	3,28 (0,71 – 25,51)	<0,001

Elevated SII and SIRI values were significantly associated with the occurrence of MACE (p = 0.002 and p = 0.001, respectively).(Table 4) Logistic regression analysis identified SIRI as an independent predictor of MACE, with an odds ratio of 1.233 (95% CI: 1.151–1.320). This indicates that each unit increase in SIRI increases the likelihood of adverse events during hospitalization by approximately 23%. (Table 5).

Table 5.
Binary Logistic Regression Analysis of SIRI and SII with in-hospital MACE

Variable	Outcome	OR	95% CI	*p-value*
SIRI	MACE	1,233	1,151 – 1,320	0,047
SII		1,000	1,000 – 1,000	0,551

DISCUSSION

This study is, to the best of the authors' knowledge, the first to evaluate the relationship between major adverse cardiac events (MACE) and novel inflammatory markers derived from a simple complete blood count—namely the Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI)—in patients with acute myocardial infarction (AMI) in Indonesia. Unlike most previous studies that rely on high-cost or advanced biomarkers, this study highlights the potential of routine hematological parameters as rapid, inexpensive, and widely available tools for risk stratification in resource-limited clinical settings.

These findings are consistent with the well-established epidemiology of AMI, which shows a higher prevalence in men, particularly in middle-aged groups, due to differences in hormonal protection, risk factor exposure, and lifestyle behaviors. (International Diabetes Federation, 2025) . Estrogen has been shown to have vasoprotective effects, which partly explains the lower incidence in premenopausal women, while male patients tend to develop atherosclerotic disease earlier. (Purba et al., 2023) The most frequent comorbidity was hypertriglyceridemia (27.2%), followed by hypertension and diabetes mellitus. These conditions are well-recognized cardiovascular risk factors, contributing to endothelial dysfunction, atherosclerosis, and plaque instability. (Zeller et al., 2024).

A predominance of ST-elevation myocardial infarction (STEMI) was observed in 63.8% of cases, while 36.2% presented with NSTEMI. This predominance of STEMI is similar to epidemiological patterns observed in many low- and middle-income countries, where delayed hospital presentation and limited access to preventive cardiovascular care contribute to more severe clinical manifestations at diagnosis (World Health Organization, 2021) National data also reflect a similar pattern in Indonesia, where late presentation increases the likelihood of complete coronary occlusion/ STEMI and leads to worse in-hospital outcomes. Persistent delays in patient presentation and limited access to timely reperfusion therapy remain major healthcare challenges in Indonesia.(Riskesdas, 2019).

The laboratory profile of the study population demonstrated a median platelet count of 246.5 × 10^9/L and a median leukocyte count of 11.66 × 10^3/µL. Elevated leukocyte and neutrophil levels are common in AMI and reflect the acute inflammatory response triggered by ischemic injury. Myocardial necrosis activates the innate immune system, leading to rapid mobilization of neutrophils, monocytes, and other inflammatory mediators. This cellular infiltration contributes to further tissue injury, infarct expansion, and adverse remodeling. (Yuan et al., 2023) The median absolute neutrophil count was 8.47 × 10^3/µL, while the median lymphocyte count was lower (1.88 × 10^3/µL). This pattern is expected, as AMI is often associated with neutrophilia and relative lymphopenia. Neutrophils play a major role in plaque destabilization, the release of proteolytic enzymes, and amplification of the inflammatory cascade. (Dziedzic et al., 2023) Lymphopenia, on the other hand, reflects physiological stress and cortisol-mediated suppression of adaptive immunity, which is often linked to worse outcomes (Nunez et al., 2011) Monocyte levels were also elevated (median 0.88 × 10^3/µL), consistent with their role in post-infarction inflammation and tissue repair. (Chistiakov et al., 2016).

Troponin I levels were significantly increased (median 11.56 ng/mL), reflecting myocardial injury. Troponin is a sensitive and specific biomarker of cardiomyocyte necrosis and is routinely used for diagnosis and risk stratification in AMI. (Perhimpunan Dokter Spesialis Kardiovaskular Indonesia, 2018). Dyslipidemia is a well-established risk factor for atherosclerosis, promoting plaque formation and instability.Low HDL cholesterol and elevated LDL cholesterol have been strongly associated with coronary plaque progression and increased risk of major adverse cardiovascular events.(Li et al., 2022).

Inflammatory indices were also measured in this study. The median SII was 1090.52 (214.04–9298.8), and the SIRI was 3.83 (0.35–31.19). This aligns with the findings of Sun H. et al, who reported significantly higher SII levels in AMI patients compared to controls. (Sun et al., 2024) Similarly, a Chinese study demonstrated that patients with MACE had higher SII than those without MACE, even after adjustment for confounders (1.255,89 [681,18 – 2.190,82]; p< 0.005). (Wei et al., 2023) Pre-procedural SII has also been identified as an independent predictor of mortality and the no-reflow phenomenon in patients with ST-elevation myocardial infarction (STEMI), emphasizing its prognostic value. (Esenboğa et al., 2022). The mean SIRI was consistent with the Kailuan study. This study showed that SIRI ≥ 1.07 was associated with an increased risk of AMI and all-cause

mortality, even after adjusting for inflammatory markers. (Jin et al., 2021) Supporting this, Ozilhan et al, reported a significantly higher incidence of MACE in patients with SIRI > 2.3 compared to those with lower values (19.1% vs. 4.0%, $p < 0.001$). (Ozilhan MO et al., 2023).

MACE occurred in 25.4% of patients (Table 3). These findings are consistent with previous reports indicating that MACE can occur in 4.2–51% of STEMI patients depending on patient risk profile and hospital resources. Heart failure remains the most common complication after AMI, caused by loss of viable myocardium and impaired contractility. Cardiogenic shock is associated with high mortality and typically reflects extensive myocardial damage. Stroke and arrhythmias are common due to persistent coronary instability and electrical disturbance in ischemic myocardium.

Bivariate analysis (Table 4) using the Mann–Whitney U test showed that both SII and SIRI values were significantly higher in patients with MACE compared to those without. These results are consistent with prior studies demonstrating the prognostic value of SIRI and SII in AMI. Liu et al. and Marchi et al. (2021) reported that SIRI is independently associated with in-hospital mortality and heart failure in STEMI patients undergoing percutaneous coronary intervention. (Marchi et al., 2024) Elevated SII has also been linked to severe coronary stenosis and worse outcomes, although its predictive power may be influenced by patient heterogeneity. (Liu et al., 2021).

In this study, we also observed a significant association between elevated SIRI levels and the occurrence of in-hospital MACE in patients with AMI, aligning with previous research in this area. This result is consistent with the findings of Wei et al., who showed that higher SIRI levels (Q2: 1.72–3.68; Q3: >3.68) were associated with increased MACE risk (OR 2.153 and 1.251, respectively). (Wei et al., 2023) Zhou et al., also demonstrated that high SIRI was a significant MACE predictor (OR 2.015; 95% CI: 1.352–3.002). (Zhou et al., 2024) Conversely, Liu et al. (2023) found that admission SIRI was not independently predictive, while SIRI measured 12 hours after PCI significantly correlated with MACE (HR 1.079; 95% CI: 1.050–1.108). (Liu et al., 2023) These results suggest that SIRI may provide stronger and more consistent prognostic information than SII, and that measurement timing may play a key role in improving its predictive accuracy.

SIRI, which is derived from neutrophil, monocyte, and lymphocyte counts, reflects the interplay between inflammatory activation and immune regulation, both of which play a central role in atherosclerotic plaque formation and rupture. This can be explained by the platelet component of SII, which is more susceptible to external influences such as prior antiplatelet therapy, hemodynamic changes, or individual variability. Platelet count alone may not adequately represent the underlying atherosclerotic inflammatory process, making SII less sensitive as a predictor compared to SIRI in the acute phase of AMI.

This study has several strengths, including a representative patient population from a tertiary referral hospital and the use of simple, widely available biomarkers. However, several limitations should be acknowledged. The study was conducted at a single center using a cross-sectional design, which limits causal inference. The sample size, while adequate for initial evaluation, may not fully capture all confounding variables. Future studies should consider evaluating SIRI and SII dynamically at multiple time points. Multicenter and prospective studies are required to validate these findings and to determine optimal cut-off values for SIRI and SII in predicting MACE in different populations.

CONCLUSION

This study demonstrates a significant association between Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI) with the occurrence of major adverse cardiac events (MACE) during hospitalization in patients with acute myocardial infarction. While both indices were associated with MACE, only SIRI showed an independent predictive value,

indicating its potential utility as a simple, inexpensive, and accessible prognostic biomarker for early risk stratification in clinical settings. These findings support the concept that inflammatory and immune responses play a crucial role in the pathophysiology and short-term outcomes of AMI. The integration of SIRI into clinical risk assessment may enhance early identification of high-risk patients and guide more intensive monitoring and management strategies, particularly in resource-limited hospitals. Further prospective, multicenter studies with larger sample sizes are needed to validate these results and explore their prognostic value in different populations.

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