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The Relationship Between Elevated Systemic Immune-Inflammatory Index (SII) and the Severity of Coronary Lesions in Chronic Coronary Syndrome (CCS) Patients Undergoing Coronary Angiography at Haji Adam Malik Hospital Medan

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## ABSTRACT

Introduction: This study examines the correlation between Systemic Immune Inflammation Index (SII) and coronary lesion severity in Chronic Coronary Syndrome (CCS) patients using the SYNTAX score. Inflammatory markers, including neutrophillymphocyte ratio (NLR), monocyte-lymphocyte ratio (MLR), and platelet-lymphocyte ratio (PLR), are associated with atherosclerosis and CAD.

Methods: This retrospective observational study analyzed correlation between SII values and coronary lesion severity in CCS patients who underwent coronary angiography. Data were collected between June and August 2024. Patients were categorized based on the SYNTAX score, which evaluates coronary lesion severity. The study used statistical tests including mean tests, multivariate logistic regression, Spearman correlation, and ROC curve analysis to assess SII's sensitivity and specificity in predicting lesion severity. A pvalue < 0.05 was considered significant.

**Results**: The study subjects were 115 samples with an average age of  $59.41 \pm 9.45$ , with 78 male samples (67,8%). It was found that the SII value has a strong correlation with the SYNTAX score, specifically with a mild SYNTAX score of 365.7 (183.45 - 853.91), a moderate SYNTAX score of 695.52 (534.83 - 1838.57), and a severe SYNTAX score of 1026.7 (413.57 - 3813.83; p value < 0.001).

Conclusion: The study found a significant relationship between SII value and coronary lesion severity, measured by SYNTAX score. Higher SII values were associated with more severe coronary lesions, suggesting SII can serve as an effective predictor of lesion severity in CCS patients.

## Keywords

Systemic Immune-Inflammatory Index, Chronic Coronary Syndrome, Angiography, CCS, SII

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## INTRODUCTION

Cardiovascular diseases (CVDs) collectively remain the leading cause of death worldwide and significantly contribute to health complications and excessive healthcare costs. In 2019, it was estimated that 17.9 million people died from CVDs, representing 32% of global deaths [1]. In Indonesia, data from the 2013 Basic Health Research (Riskesdas) show that the prevalence of heart disease increases with age, with the highest occurrence in the 65-74 age group, accounting for about 2% of the population aged 15 years and older [2].

Coronary artery disease (CAD) is a chronic process, as atherosclerotic lesions in the coronary arteries are dynamic, broad in spectrum, and exhibit changes in their natural history, involving various parts of the coronary circulation [3,4]. Chronic coronary syndrome (CCS) is a heterogeneous group of conditions that includes both obstructive and non-obstructive coronary artery disease, with or without myocardial infarction

or prior revascularization, ischemic heart disease diagnosed through non-invasive tests, and chronic angina syndromes with various underlying causes [5,6].

The pathological process of CAD includes atherosclerosis and coronary artery spasm. tis known that the inflammatory process plays a crucial role in the formation and progression of atherosclerosis. Inflammatory markers have been found to correlate with the severity of CAD and the prognosis of cardiovascular diseases in a negative direction [7,8]. Metabolic disturbances, endothelial damage, inflammation, and immune dysfunction can drive the occurrence and progression of coronary atherosclerosis, leading to coronary heart disease (CHD). Inflammation plays a significant role in the vulnerability of plaque formation and rupture, which can trigger the formation of blood clots, ultimately resulting in myocardial infarction [9].

Several inflammatory markers, such as C-reactive protein (CRP), tumor necrosis factor- $\alpha$ , and various interleukins, are associated with adverse outcomes in CAD. Simple hematological indices, such as the neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR), are also useful inflammatory indicators and promising prognostic factors in cardiovascular diseases [10]. The main ratios used include NLR, monocyte-lymphocyte ratio (MLR), and PLR. These ratios have been shown to be markers of atherosclerosis and are correlated with the prevalence of coronary heart disease and poor cardiovascular outcomes [9].

Hu et al. developed a novel marker known as the systemic immune-inflammation index (SII). The SII is calculated by multiplying the platelet count by the NLR value, incorporating three types of cells to provide valuable information about inflammation [9]. SII is a relatively new inflammatory marker obtained from the combination of neutrophil, platelet, and lymphocyte counts. Initially developed to predict clinical outcomes and prognosis in hepatocellular carcinoma, SII has since been applied to other solid tumors, including colorectal, esophageal, and cervical cancers [10].

The relationship between SII values as a predictor of coronary lesion severity in CCS patients remains insufficiently understood, and there is limited literature discussing CCS or the relationship between SII and coronary lesion severity in CCS patients undergoing coronary angiography. In Indonesia, there has been no study to evaluate this relationship. Therefore, this study aims to assess the role of SII in predicting the severity of coronary lesions in CCS patients undergoing coronary angiography.

## **METHOD**

This study was designed as a retrospective, observational, descriptive research aimed at analyzing the relationship between the Systemic Immune Inflammation Index (SII) and the severity of coronary lesions in patients with Chronic Coronary Syndrome (CCS) through the SYNTAX score. SII was calculated using the formula for platelet count multiplied by the Neutrophil-Lymphocyte Ratio (NLR) multiplied by 10<sup>3</sup>, while the severity of coronary lesions was assessed based on the SYNTAX score, which was derived from coronary angiography. This approach allowed for a comprehensive understanding of the inflammatory indices and their correlation with coronary artery disease (CAD) severity in CCS patients.

The research was conducted at the Haji Adam Malik General Hospital's Integrated Heart Center in Medan, Indonesia. Data collection occurred from June to August 2024, following the approval from the ethics committee. The sample was obtained retrospectively through the review of electronic medical records within this period. The target population included all patients diagnosed with CCS who had undergone coronary angiography. The accessible population was defined as patients who met the inclusion criteria and had records from the specified timeframe. A total of 115 samples were selected using a total sampling technique from the eligible patient pool.

Inclusion criteria required that patients be diagnosed with CCS, had undergone coronary angiography, and had complete laboratory results as per the study's requirements. Exclusion criteria included diagnoses of Acute Coronary Syndrome (ACS), those with a SYNTAX score of 0, a history of Peripheral Artery Disease (PAD), severe valvular heart disease, acute or chronic infections, and other significant chronic diseases such as malignancies, autoimmune disorders, or kidney disease.

Data collection followed a systematic process. After obtaining ethical clearance, samples were selected based on the inclusion and exclusion criteria. Laboratory tests, including blood sampling, were performed prior

to coronary angiography. Subsequently, coronary angiography was conducted on each participant to assess the severity of CAD using the SYNTAX score, which was calculated independently by two cardiologists, blinded to clinical data, ensuring the objectivity and accuracy of the assessment. The severity of CAD was classified according to the SYNTAX score: mild (0-22), moderate (22-32), and severe (>32). Lesions were considered significant if stenosis was  $\geq$ 50% in lesions with a diameter  $\geq$ 1.5 mm. Once all data were collected, statistical analysis was performed to evaluate the correlation between SII values and SYNTAX scores using appropriate statistical methods.

The study adhered to rigorous ethical guidelines. Approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara, and the research permission was granted by the Research and Development Unit of Haji Adam Malik Hospital Medan.

## **RESULTS**

A total of 115 patients diagnosed with Chronic Coronary Syndrome (CCS) who underwent coronary angiography at Haji Adam Malik Hospital from June 2024 to August 2024 were included in this study. These patients met the inclusion and exclusion criteria and were categorized based on the severity of CAD, which was classified as mild, moderate, or severe, according to the SYNTAX score. A SYNTAX score of less than 22 indicated mild severity, a score between 22-32 indicated moderate severity, and a score above 32 indicated severe CAD. Two interventional consultants at Adam Malik Hospital validated the lesion severity based on the SYNTAX score, and the results were consistent across all severity categories.

The baseline characteristics of the study participants are shown in Table 4.1. The average age of the study participants was  $59.41 \pm 9.45$  years. Among the 115 participants, 78 were male (67.8%). The average monocyte count was  $8.34 \pm 2.59$ , neutrophils averaged  $58.12 \pm 9.82$ , and lymphocytes averaged  $29.29 \pm 8.19$ . Several variables showed non-normal distributions, with medians reported for platelet count (286,000), monocyte-lymphocyte ratio (MLR) (0.28%), neutrophil-lymphocyte ratio (NLR) (1.98%), platelet-lymphocyte ratio (PLR) (10.1%), Systemic Immune Inflammation Index (SII) (563.19 x  $10^3$ ), and the SYNTAX score (20.5).

Table 1. Baseline Characteristics of the Study Sample

Variable	Description	
Age (years)	$59.41 \pm 9.45$	
Gender (Male) (%)	78 (67.8%)	
Monocyte	$8.34 \pm 2.59$	
Neutrophil	$58.12 \pm 9.82$	
Lymphocyte	$29.29 \pm 8.19$	
Platelet (x1,000)	286 (105 - 613)	
MLR (%)	0.28 (0.02 - 1.86)	
NLR (%)	1.98 (0.76 - 17.51)	
PLR (%)	10.1 (4.22 - 43.14)	
SII (x1,000)	563.19 (183.45 - 3813.83)	
SYNTAX Score	20.5 (2 - 67)	

Further analysis based on CAD severity, as defined by the SYNTAX score, is shown in Table 4.2. The results indicated that patients with severe (SYNTAX >32) and moderate (SYNTAX 22-32) scores were significantly older ( $62.72 \pm 7.97$  years; p < 0.001), had higher neutrophil counts ( $63.85 \pm 10.21$ ; p < 0.001), lower lymphocyte counts ( $24.92 \pm 7.34$ ; p < 0.001), higher platelet counts (median 417; p < 0.001), higher NLR (median 2.52; p < 0.001), higher PLR (median 16.3; p < 0.001), and higher SII values (median 1026.7 x  $10^3$ ; p < 0.001) compared to patients with mild SYNTAX scores (SYNTAX <22).

## **Data Characteristics Based on SYNTAX Score**

The study found that the SYNTAX score distribution was as follows: 59 samples with a mild SYNTAX score (<22), 24 samples with a moderate SYNTAX score (22-32), and 32 samples with a severe SYNTAX score (>32). Significant age differences were observed across the SYNTAX score groups. Patients in the severe and moderate SYNTAX score groups were older than those in the mild group ( $62.72 \pm 7.97$  years; p < 0.001). The male gender predominated in all groups, with 71% of the mild group, 62.5% of the moderate group, and 65.6% of the severe group being male.

Table 2. Baseline Characteristics of the Study Sample Based on SYNTAX Score

Variable	SYNTAX <22	SYNTAX 22-32	SYNTAX >32	p-
v ai iable	(n = 59)	(n = 24)	(n = 32)	value
Age (years)	$56.25 \pm 9.45$	$62.75 \pm 8.92$	$62.72 \pm 7.97$	< 0.001
Gender (Male) (%)	42 (71%)	15 (62.5%)	21 (65.6%)	0.856
Monocyte	$8.77 \pm 2.29$	$8.09 \pm 2.29$	$7.75 \pm 3.19$	0.194
Neutrophil	$54.17 \pm 8.43$	$60.2 \pm 8.24$	$63.85 \pm 10.21$	< 0.001
Lymphocyte	$32.65 \pm 7.60$	$26.83 \pm 7.27$	$24.92 \pm 7.34$	< 0.001
Platelet (x1,000)	231 (115 - 387)	342.5 (105 - 433)	417 (289 - 613)	< 0.001
MLR (%)	0.27 (0.12 - 0.51)	0.28 (0.17 - 1.86)	0.3 (0.02 - 0.67)	0.246
NLR (%)	1.60 (0.76 - 6.35)	2.12 (1.45 - 17.51)	2.52 (0.84 - 12.63)	< 0.001
PLR (%)	6.86 (4.22 - 13.66)	11.89 (10.16 - 22.61)	16.3 (10.71 - 43.14)	< 0.001
SII (x1,000)	365.7 (183.45 - 853.91)	695.52 (534.83 - 1838.57)	1026.7 (413.57 - 3813.83)	< 0.001

Among the blood parameters, no significant difference was found in monocyte counts across the groups, with a p-value of 0.194. However, significant differences were observed for neutrophils, lymphocytes, and platelets. The severe SYNTAX group showed significantly higher neutrophil counts ( $63.85 \pm 10.21$ ; p < 0.001), lower lymphocyte counts ( $24.92 \pm 7.34$ ; p < 0.001), and higher platelet counts (median 417 x  $10^3$ ; p < 0.001). The inflammatory ratios, including NLR, PLR, and SII, also showed significant differences, with the severe group having the highest values for NLR (median 2.52; p < 0.001), PLR (median 16.3; p < 0.001), and SII (median  $1026.7 \times 10^3$ ; p < 0.001).

### **Correlation Between Inflammatory Ratios and SYNTAX Score**

Further analysis using Spearman's correlation (Table 4.4) showed strong positive correlations between NLR, PLR, and SII with the SYNTAX score. The correlation coefficients were 0.460 for NLR (p < 0.001), 0.871 for PLR (p < 0.001), and 0.835 for SII (p < 0.001). However, MLR showed no significant correlation with the SYNTAX score (Spearman's rho 0.147; p value 0.118).

Table 4. Spearman Correlation Test for SYNTAX Score

Variable	Spearman's rho	p-value
MLR	0.147	0.118
NLR	0.460	< 0.001
PLR	0.871	< 0.001
SII	0.835	< 0.001

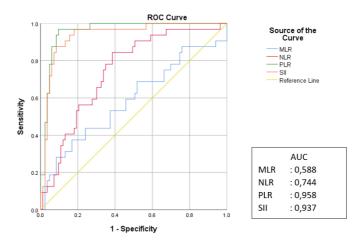


Figure 1. ROC Curve Analysis of Inflammatory Ratios as Predictors for Severe SYNTAX Score

# ROC Curve Analysis of Inflammatory Ratios as Predictors for Severe SYNTAX Score

To identify which inflammatory ratio parameter was most effective in predicting a severe SYNTAX score (>32), ROC curve analysis was performed. As shown in Figure 4.1, an SII value of >742.7  $\times$  10<sup>3</sup> demonstrated the highest sensitivity (90.6%) and specificity (86.7%) for predicting patients with severe SYNTAX scores. This analysis highlights SII as the most significant inflammatory marker in predicting severe coronary lesions, as it exhibits both high sensitivity and specificity.

## **DISCUSSION**

This study aimed to evaluate the relationship between inflammatory cell ratios—specifically the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII)—with the severity of coronary artery disease as indicated by the SYNTAX score in patients with acute ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI). The findings demonstrate significant correlations between these inflammatory markers and the severity of coronary artery lesions, emphasizing their potential as predictive biomarkers for cardiovascular events.

Neutrophils play a key role in the inflammatory process, contributing to endothelial dysfunction and progression of atherosclerosis. Neutrophils are involved in tissue damage and the propagation of inflammation in advanced stages of atherosclerosis, where they induce smooth muscle cell death and plaque rupture [11]. The study's finding of a positive correlation between the neutrophil count and the size of the necrotic core, lesion size, and plaque vulnerability corroborates previous research showing that neutrophils exacerbate atherosclerotic plaque progression and instability. Additionally, this study supports the view that neutrophils, along with other inflammatory cells, can serve as reliable indicators of cardiovascular risk.

Lymphocytes are central to modulating the immune response, and their reduced numbers have been linked with poor outcomes in various cardiovascular diseases, including coronary artery disease and heart failure [5,11]. The results of this study, which found that low lymphocyte counts are associated with higher SYNTAX scores, align with previous findings that lymphopenia predicts adverse clinical outcomes, including cardiovascular events. The apoptosis of lymphocytes within atherosclerotic plaques facilitates plaque growth, lipid core expansion, and subsequent rupture, all of which contribute to thrombus formation [11].

Monocytes, another critical cell type in the progression of atherosclerosis, contribute to plaque formation through the secretion of pro-inflammatory cytokines and enzymes. In this study, monocyte count was found to be an independent predictor of cardiovascular events, aligning with previous research that has established monocytes as key players in the initiation and progression of atherosclerosis. Monocyte activation and migration into the intima, where they differentiate into macrophages, further accelerate plaque development and thrombus formation [5,11]. Elevated monocyte counts have been linked to increased cardiovascular risk and poor outcomes in myocardial infarction and stroke, as evidenced by studies involving both high-risk populations and general cohorts.

Platelets have long been recognized as critical in thrombosis and atherogenesis. Activated platelets contribute to both the inflammatory response and the formation of atherothrombotic lesions. This study found that higher platelet counts were associated with more severe coronary artery lesions, supporting the role of platelets in disease progression and suggesting their potential as predictive biomarkers for adverse cardiovascular outcomes. Platelet activation enhances the inflammatory process by interacting with leukocytes and endothelial cells, which can further accelerate the development of coronary artery disease [8,11].

The primary objective of this study was to assess the utility of inflammatory ratios in predicting the severity of coronary artery disease. The findings indicate that the NLR, PLR, and SII are strongly associated with SYNTAX scores, which are widely used to assess the complexity of coronary lesions. Elevated NLR, PLR, and SII were observed in patients with severe SYNTAX scores, indicating that these inflammatory ratios may serve as useful adjuncts to traditional clinical markers in predicting the severity of coronary artery disease.

The SII, in particular, emerged as a significant marker in this study, showing a higher predictive value for severe coronary lesions compared to other inflammatory ratios. Previous studies have demonstrated that SII, which incorporates neutrophils, platelets, and lymphocytes, provides a more balanced and comprehensive assessment of the inflammatory response compared to other individual markers [12]. The high sensitivity (90.6%) and specificity (86.7%) of SII for predicting severe SYNTAX scores (>32) further support its potential as a valuable biomarker in clinical practice.

The results of this study align with the findings of previous research also reported strong correlations between inflammatory markers and SYNTAX scores, reinforcing the idea that inflammatory responses play a significant role in determining the complexity of coronary artery disease. The present study's findings also corroborate previous studies on the predictive value of SII, which has been shown to outperform traditional cardiovascular risk factors in predicting adverse events such as myocardial infarction, stroke, and heart failure [13].

However, it is noteworthy that the mean lymphocyte-to-monocyte ratio (MLR) did not show a significant difference among the three groups with different SYNTAX scores. This contrasts with the findings who reported a strong association between MLR and coronary artery disease severity. This discrepancy may reflect differences in study populations, methodology, or the specific inflammatory pathways targeted by these ratios [14,15].

The findings from this study suggest that inflammatory cell ratios, particularly NLR, PLR, and SII, could serve as practical, non-invasive biomarkers for assessing the severity of coronary artery disease and predicting major cardiovascular events [16]. These markers could help clinicians stratify patients based on their risk of adverse outcomes and guide treatment decisions, especially in the setting of acute myocardial infarction with ST-segment elevation. Furthermore, SII, with its high sensitivity and specificity, may offer significant promise in clinical applications for risk stratification in this patient population [17-20].

While the study provides valuable insights into the role of inflammatory markers in coronary artery disease, there are several limitations to consider. First, this was a single-center study, and the results may not be generalizable to other populations. Additionally, the study focused on a specific cohort of patients undergoing PCI, which may limit the applicability of these findings to other cardiovascular conditions. Future research should involve larger, multi-center cohorts and explore the longitudinal predictive value of these inflammatory ratios in broader patient populations.

## **CONCLUSION**

This study demonstrates a significant relationship between the systemic immune-inflammation index (SII) and severe SYNTAX scores in patients with chronic coronary syndrome. In addition, markers such as NLR and PLR were found to correlate with the severity of coronary artery disease. Multivariate logistic regression analysis further confirmed that SII, along with lymphocytes, platelets, and PLR, plays a significant role in predicting severe SYNTAX scores. Given its high sensitivity and specificity, SII emerges as a cost-effective, readily available, and useful biomarker for predicting coronary artery disease severity and aiding in risk stratification.

### **DECLARATIONS**

Ethics approval and consent to participate were obtained. Permission for this study was obtained from the Ethics Committee of the Universitas Sumatera Utara and Haji Adam Malik General Hospital.

### **CONSENT FOR PUBLICATION**

The Authors agree to publication in the Journal of Society Medicine.

### **FUNDING**

None

### **COMPETING INTERESTS**

The authors declare no conflicts of interest in this study.

## **AUTHORS' CONTRIBUTIONS**

All authors significantly contributed to the work in terms of execution, data acquisition, analysis, interpretation, or all these areas. Contributed to drafting, revising, and critically reviewing the manuscript. Approved the final version for publication, agreed to the journal submission, and agreed to be accountable for all aspects of the work.

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