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Prognostic Value of the Systemic Immune–Inflammation Index for Major Cardiovascular Events in Patients with Stable Coronary Artery Disease

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ABSTRACT

Background: Chronic inflammation promotes the advancement of coronary atherosclerosis and increases poor cardiovascular outcomes, even in individuals with stable coronary artery disease. The Systemic Immune-Inflammation Index (SII), derived from basic hematological parameters, is a useful measure of systemic inflammatory and thrombotic activity; however, its effectiveness in stable coronary artery disease (CAD) has not been fully verified.

Objective: To determine the Systemic Immune-Inflammation Index's predictive value in predicting major adverse cardiovascular events in individuals with stable coronary artery disease.

Methods: This prospective observational study, was conducted at Al-Aleem Medical College, Lahore, Pakistan, between January 2024 and January 2026, included 180 individuals with stable coronary artery disease. The SII index was computed as the product of the platelet and neutrophil counts divided by the lymphocyte count after the patients' baseline clinical and laboratory data were gathered. Over the course of a year, patients' incidence of notable adverse cardiovascular events was tracked. Multivariable Cox proportional hazards regression, Kaplan-Meier survival analysis, and receiver operating characteristic (ROC) analysis were carried out.

Results: During the follow-up period, 38 patients had serious cardiovascular events. The SII levels were markedly elevated in those experiencing events compared to those not enduring events. The ideal cut-off value for SII was found to be 780, which had a sensitivity of 76.3%, a specificity of 74.6%, and an area under the curve of 0.82. Patients with SII ≥780 had markedly increased rates of cardiovascular events and reduced rates of event-free survival. SII served as an independent predictor of MACE after adjusting for traditional cardiovascular risk variables.

Conclusion: The SII functions as an autonomous and easily obtainable prognostic indicator for predicting MACE in patients with stable CAD. It may be used in conventional clinical evaluations and may improve risk classification and tailored secondary prevention.

Keywords: Stable coronary artery disease; Systemic Immune–Inflammation Index; major adverse cardiovascular events; inflammation; prognosis.

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INTRODUCTION

Stable coronary artery disease continues to be a significant contributor to long-term cardiovascular morbidity and death, despite advancements in medical treatment, risk factor management, and coronary revascularization [1]. Major adverse cardiovascular events (MACE), such as myocardial infarction, ischemic stroke, hospitalization for heart failure, repeat revascularization, or cardiovascular mortality, continue to occur in a considerable proportion of clinically stable patients [2]. Standard risk assessment is often influenced by age, gender, hypertension, diabetes mellitus, smoking history, lipid profile, left ventricular function, the extent of illness shown on angiography, and any previous cardiovascular history. Despite the therapeutic

use of the aforementioned criteria, they fail to address the additional risk present in several individuals with stable coronary artery disease [3].

Atherosclerosis is more than just a lipid-storage disease; it is a chronic inflammatory and immune-mediated vascular disease [4]. Plaque progression and clinical cardiovascular events are related to endothelial dysfunction, oxidative stress, activation of leukocytes, aggregation of platelets, and low-grade, chronic inflammation [5]. Even if the disease remains stable, inflammatory activity can still occur within the vascular wall and the systemic circulation, which can render the plaques vulnerable, lead to thrombosis, and trigger recurrent ischemic events. Thus, simple inflammatory markers might be useful in further refining risk stratification in patients who look well but stay vulnerable [6].

The Systemic Immune-Inflammation Index is a new hematological index based on platelet, neutrophil and lymphocyte counts. The calculation requires the platelet count to be multiplied by the neutrophil count and then divided by the lymphocyte count [7].

This index is a combination of three relevant biological pathways for cardiovascular disease, representing neutrophil-mediated inflammation, platelet-mediated thrombosis, and lymphocyte-mediated immune regulation, respectively. Hence, the SII could be linked to increased inflammation and hypercoagulability and perturbed immunity [8,9].

The Systemic Immune–Inflammation Index might offer a more comprehensive measurement of the inflammatory and thrombotic profile of coronary atherosclerosis than individual blood cell parameters or more basic inflammatory ratios [10]. Neutrophils contribute to the damage of endothelial cells and oxidative stress and destabilize plaques. Platelets play a role in thrombosis, vascular inflammation and activation of leukocytes. Lymphopenia is a possible indicator of physiological stress and deregulated immunity. These parameters, when taken together, might provide valuable prognosis information in addition to standard clinical parameters [11].

The advantage of the Systemic Immune–Inflammation Index is that it is available in standard complete blood count, which is easily available and is routinely ordered to evaluate the cardiovascular system, and it is cheap [12]. This makes it attractive to its use in clinical settings where more advanced biomarkers and imaging risk-assessment tools are not readily available. If reliable, the index could help clinicians select patients with stable coronary artery disease who are at greater risk of suffering an adverse event, and so should be monitored more closely, treated to better control risk factors and optimised with secondary prevention therapy and followed up promptly [13].

Although the study of Systemic Immune–Inflammation Index in different cardiovascular diseases has been studied, there is a need to study this index in chronic stable coronary artery disease [14]. Most of the literature has focused on prognosis in acute coronary syndrome, heart failure or patients undergoing coronary intervention, and few studies have examined its prognostic utility in clinically stable coronary artery disease populations. Association with future cardiovascular events in stable patients could be additional evidence for its role as a simple and practical prognostic marker. The aim of this study was to evaluate the predictive significance of SI-I in individuals with stable CAD and to determine whether SI-I alone correlates with worse cardiovascular outcomes [15].

MATERIALS AND METHODS

The study was prospective observational research conducted at Al-Aleem Medical College, Lahore, Pakistan, from January 2024 to January 2026. A total of 180 individuals diagnosed with stable coronary artery disease were recruited by a non-probability, sequential selection method. Stable coronary artery disease was characterized by coronary artery stenosis confirmed via angiography, a history of coronary artery bypass grafting and/or percutaneous coronary intervention, or stable angina managed by medical treatment without acute aggravation in the three months before the research.

Participants aged 35 to 80 years, irrespective of gender, were selected after the procurement of their written informed consent. To reduce variables that may influence inflammatory cell counts or cardiovascular outcomes, patients with acute coronary syndrome in the past three months, active inflammatory diseases, autoimmune disorders, malignancies, hematological conditions, severe renal impairment, severe liver disease, recent surgical interventions or trauma, corticosteroid administration, immunosuppressive treatment, or incomplete follow-up data were excluded. Baseline clinical and demographic data were obtained using a proforma. Age, gender, body mass index, smoking status, hypertension, diabetes mellitus, dyslipidemia, history of myocardial infarction, history of coronary revascularization, medication history, systolic and diastolic blood pressure, left ventricular ejection fraction, and, if relevant, angiographic disease profile were among the variables gathered.

Clinical data was collected from the relevant hospital records, out-patient records, laboratory and angiographic data. Venous blood samples were taken using standard aseptic precautions at the time of recruitment before the outcome assessment. A full blood count was performed in an automated hematology analyzer, which measured the number of platelets, neutrophils, and lymphocytes. Systemic Immune-Inflammation Index (SII) = PLT x NEU / LYM. Biochemical data were obtained when available, which included fasting blood glucose level, serum creatinine, and lipid profile. A serious adverse cardiovascular event throughout the follow-up period was the trial's main endpoint. Significant adverse cardiovascular events included cardiovascular death, non-fatal myocardial infarction, non-fatal ischemic stroke, hospitalization for

unstable angina, hospitalization for heart failure and repeat coronary revascularization.

Post-enrollment follow-up was conducted by outpatient visits, hospital record assessments, and telephonic communication for all patients over a 12-month duration. Medical data were used to validate reported occurrences, where possible.

To predict major adverse cardiovascular events, patients were classified into low and high Systemic Immune-Inflammation Index groups according to a cut-off value established using receiver operating characteristic curve analysis. The institutional ethics review board evaluated and sanctioned the study procedure. All individuals provided written informed consent prior to participation. Data confidentiality was maintained throughout the experiment, and all data were used only for research purposes.

The data were analyzed using SPSS version 26. For qualitative data, frequency and percentage were utilized; for regularly distributed quantitative variables, mean ± standard deviation was used; and for non-normally distributed data, median with interquartile range was employed. The chi-square test or Fisher's exact test for categorical variables and the independent-samples t-test or Mann-Whitney U test for continuous data were used to compare patients with and without significant adverse cardiovascular events.

The ideal cut-off value for the Systemic Immune–Inflammation Index was determined using receiver operating characteristic curve analysis. The low index and high index groups' event-free survival was compared using Kaplan-Meier survival analysis. Cox proportional hazards regression analysis was used to find independent predictors of serious adverse cardiovascular events. A p-value of less than 0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board (IRB) of Al-Aleem Medical College, Lahore, Pakistan (Approval No. IRB/AMC/2023/198). Written informed consent was obtained from all participants before enrolment.

RESULTS

The current study included 180 participants with stable coronary artery disease. The average age of the study population was 58.9 ± 9.6 years. The number of male and female patients was 116 and 64, respectively, yielding a ratio of 1.8:1. In the 12-month follow-up period, 142 people had no significant adverse cardiovascular events, whereas 38 patients did.

Individuals who had significant adverse cardiovascular events tended to be older than those who did not. The MACE group's average age was 63.8 ± 9.7 years, whereas the non-MACE group's average age was 57.6 ± 9.1 years. Although there were more male patients in both groups according to the gender distribution, the difference was not statistically significant. This implies that the events in the current investigation were not primarily influenced by gender imbalance.

Traditional cardiovascular risk factors were more prevalent in patients who had events. While diabetes mellitus was significantly more prevalent in the MACE group, hypertension, smoking, and dyslipidemia were more common, but not statistically significant. Multivessel coronary artery disease and a history of previous myocardial infarction were significant predictors of recurrent cardiovascular events. At baseline, patients with MACE exhibited a lower left ventricular ejection fraction, suggesting more severe cardiac dysfunction. Hematological inflammatory indicators showed significant differences between the two groups. People with MACE reported significantly lower lymphocyte levels and significantly higher neutrophil and platelet levels. The MACE group's SII level was much higher than the non-MACE groups. The findings demonstrate that people with catastrophic cardiovascular outcomes had an increased inflammatory and thrombotic burden (Table 1).

A receiver operating characteristic curve analysis was used to assess the Systemic Immune-Inflammation Index's predictive value for serious adverse cardiovascular events. The optimal cut-off value for the SII was 780. The area under the curve at this level, which was 0.82, demonstrated strong discriminative efficacy. At this threshold, SII's sensitivity was 76.3%, and its specificity was 74.6%. The positive predictive value was 44.6%, while the negative predictive value was 92.2%. The elevated negative predictive value suggests that patients with SII under the threshold were unlikely to have significant adverse cardiovascular events throughout the follow-up interval. The diagnostic accuracy was 75.0%, and the correlation was statistically significant (see Table 2).

Patients were separated into low-SII and high-SII groups based on the cut-off value of the ROC curve. The low-SII group included 115 patients, whereas the high-SII group had 65. The low-SII group consisted of 71 men and 44 women, whereas the high-SII group consisted of 45 men and 20 women. The gender distribution of the two SII groups did not significantly vary. Major adverse cardiovascular events were significantly more common in those with a SII ≥780. Nine patients in the low-SII group and 29 in the high-SII group had at least one MACE.

This was a very significant difference, indicating a robust association between cardiovascular events and elevated SII. Similar trends were seen with different cardiovascular outcomes. A higher risk of cardiovascular death, non-fatal myocardial infarction, non-fatal ischemic stroke, hospitalization for unstable angina, hospitalization for heart failure, and recurrent coronary revascularization is linked to elevated SII. The results show a correlation between elevated SII levels and the total incidence of MACE and other clinically significant cardiovascular events (Table 3).

Cox proportional hazards regression analysis was utilized to find independent predictors of significant adverse cardiovascular events. The revised model included SII group, age, gender, diabetes mellitus, previous myocardial infarction, multivessel coronary artery disease, and left ventricular ejection fraction.

Even after accounting for these clinical factors, SII ≥780 was still an independent predictor of major adverse cardiovascular events. The adjusted risk of MACE was 3.41-fold higher in patients with higher vs. lower SII. This validates that the prognostic relevance of SII could not be attributed solely to age, diabetes, angiographic disease burden, or left ventricular function.

Adverse cardiovascular outcomes were also independently associated with diabetes mellitus, multivessel coronary artery disease, and lower left ventricular ejection fraction. Neither male gender nor previous myocardial infarction were independent predictors after multivariable adjustment. Overall, the regression model demonstrated that SII provided additional prognostic information beyond conventional cardiovascular risk factors (Table 4).A 12-month follow-up Kaplan–Meier analysis showed that patients with SII ≥780 had substantially reduced event-free survival compared to those with SII <780. The survival curves began to diverge early and remained markedly divergent, indicating that the high-SII group faced an elevated risk of serious adverse cardiovascular events (Figure 1).

Overall, the findings showed that a high Systemic Immune–Inflammation Index was significantly linked to future major adverse cardiovascular events among stable coronary artery disease patients. High SII was associated with significantly increased event burden, clinical outcome, and adjusted cardiovascular risk.

The results also suggest a potential application of SII as a cost-effective and easy-to-obtain prognostic factor in stable coronary artery disease. It may have potential in identifying patients who may need more close scrutiny, more effective control of risk factors, and more aggressive secondary prevention, due to its incorporation of routine complete blood count parameters.

Table 1: Baseline characteristics according to major adverse cardiovascular events

Variable	No MACE (n=142)	MACE (n=38)	p-value
Age, years	57.6 ± 9.1	63.8 ± 9.7	0.001
Male gender	89 (62.7%)	27 (71.1%)	0.338
Female gender	53 (37.3%)	11 (28.9%)	0.338
BMI, kg/m²	27.1 ± 3.8	28.2 ± 4.1	0.118
Hypertension	88 (62.0%)	29 (76.3%)	0.102
Diabetes mellitus	56 (39.4%)	25 (65.8%)	0.004
Current smoking	42 (29.6%)	15 (39.5%)	0.241
Dyslipidemia	79 (55.6%)	26 (68.4%)	0.154
Previous myocardial infarction	31 (21.8%)	17 (44.7%)	0.005
Previous PCI/CABG	39 (27.5%)	16 (42.1%)	0.081
Multivessel CAD	48 (33.8%)	23 (60.5%)	0.003
LVEF, %	53.8 ± 7.4	47.9 ± 8.6	<0.001
Neutrophil count, ×10⁹/L	4.3 ± 1.1	5.7 ± 1.4	<0.001
Lymphocyte count, ×10⁹/L	2.1 ± 0.6	1.5 ± 0.4	<0.001
Platelet count, ×10⁹/L	244.6 ± 55.2	289.4 ± 61.7	<0.001
SII	523.7 ± 214.8	1098.5 ± 386.2	<0.001

Table 2: Predictive efficacy of the Systemic Immune–Inflammation Index for significant adverse cardiovascular events

Parameter	Value
Optimal SII cut-off	780
Area under ROC curve	0.82
95% confidence interval	0.75–0.89
Sensitivity	76.3%
Specificity	74.6%
Positive predictive value	44.6%
Negative predictive value	92.2%
Overall accuracy	75.0%
p-value	<0.001

Table 3: Cardiovascular outcomes according to Systemic Immune–Inflammation Index group

Outcome	SII <780 (n=115)	SII ≥780 (n=65)	p-value
Male gender	71 (61.7%)	45 (69.2%)	0.306
Female gender	44 (38.3%)	20 (30.8%)	0.306
Any MACE	9 (7.8%)	29 (44.6%)	<0.001
Cardiovascular death	2 (1.7%)	8 (12.3%)	0.003
Non-fatal myocardial infarction	3 (2.6%)	10 (15.4%)	0.001
Non-fatal ischemic stroke	1 (0.9%)	4 (6.2%)	0.042
Hospitalization for unstable angina	4 (3.5%)	11 (16.9%)	0.001
Heart failure hospitalization	3 (2.6%)	9 (13.8%)	0.004
Repeat coronary revascularization	5 (4.3%)	13 (20.0%)	0.001

Table 4: Cox regression study for determinants of significant adverse cardiovascular events

Variable	Adjusted HR	95% CI	p-value

SII ≥780	3.41	1.68–6.92	0.001
Age	1.04	1.00–1.08	0.041
Male gender	1.18	0.62–2.25	0.612
Diabetes mellitus	1.92	1.02–3.61	0.043
Previous myocardial infarction	1.54	0.82–2.91	0.178
Multivessel CAD	2.18	1.16–4.11	0.016
LVEF	0.94	0.90–0.98	0.005

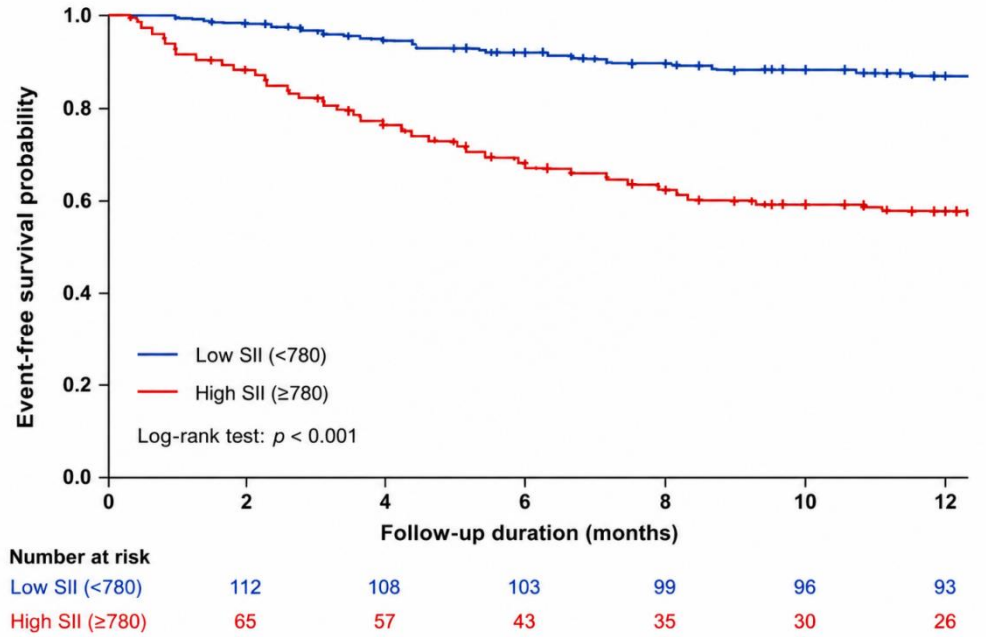

Figure 1: Kaplan-Meier event-free survival curves using the Systemic Immune-Inflammation Index. Patients with SII ≥780 showed substantially worse event-free survival compared to those with SII <780 (log-rank p < 0.001).

DISCUSSION

This prospective observational study showed that high SII was significantly correlated with MACE among patients with stable CAD [1]. The number of neutrophils, platelets, lymphocytes and the SII value were significantly higher in the patients who developed events than in the patients who didn't develop events [2]. These data indicate that chronic systemic inflammation and/or persistent systemic thromboimmune activation can result in poor cardiovascular outcome even in clinically stable patients [3]. The study showed that SII with a cut-off of 780 had an excellent capacity to predict MACEs, with an area under the curve of 0.82 [4]. Patients with SII ≥780 had substantially increased hospitalization rates for unstable angina, heart failure, recurrent coronary revascularization, ischemic stroke, and cardiovascular mortality. Patients with low SII were less likely to have events during follow-up, according to a high NPV [5].

These findings were confirmed by Kaplan–Meier analysis, which revealed that event-free survival was significantly worse for patients with high SII [6]. The early and sustained separation of the survival curves suggest that SII might also be used to identify high-risk patients before clinical deterioration. Importantly, SII was an independent predictor of MACEs even after adjusting for age, gender, diabetes mellitus, prior MI, multivessel coronary artery disease, and LVEF [7].

A biological explanation for this association is feasible. Neutrophils contribute to oxidative stress, endothelial dysfunction and destabilization of plaques [8]. Platelets are involved in thrombosis, vascular inflammation and interaction with activated leukocytes [9]. Low lymphocyte levels may be indicative of poor immune regulation and/or physiological stress. These 3 parameters when analyzed together give SII a more comprehensive view of inflammatory and thrombotic activity than any single blood cell count [10].

The undeniable advantage of SII is that it is simple and cost effective. It can be determined from common blood tests without extra blood tests [11]. This can be useful for daily clinical practice particularly in resource poor areas. In patients with stable coronary artery disease, SII could be useful for the clinical follow-up of the patients who deserve to be followed-up more closely, better control of risk factors, optimization of secondary prevention therapy and careful monitoring for recurrent ischemic events [12].

This study has some limitations. It was performed at one center and had a moderate sample size, thus potentially limiting generalizability [13,14]. No evaluations for changes over time were made as SII was measured only at baseline. Unmeasured inflammatory, nutritional or

treatment-related factors cannot be fully ruled out as residual confounding. Large multicenter studies are required to validate the best SII cut-off and to prove its usefulness in cardiovascular risk stratification in the clinical setting [15].

CONCLUSION

A higher SII was a significant predictor of major adverse cardiovascular events in patients with stable coronary artery disease. Independent predictors of poorer EFS and greater CV risk at 12 months were an SII value ≥780. Given its simplicity, low cost, and its being based on commonly used CBP values, SII might be a convenient prognostic marker to identify high-risk stable CAD patients who need more careful monitoring and more intensive secondary prevention strategies.

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Authors' Contributions: M.M.H. conceived and designed the study. H.A. contributed to data collection and patient follow-up. A.S. performed data analysis and interpretation. A.M. contributed to manuscript drafting and revision. All authors reviewed and approved the final manuscript.

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Data Availability: Data are available from the corresponding author upon reasonable request.

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