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Prognostic Value of Systemic Immune-Inflammation Index (SIII) in Predicting Major Adverse Cardiac Events (MACE) in Patients Undergoing Percutaneous Coronary Intervention (PCI): An Observational Study

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Abstract

Background

Major adverse cardiovascular events (MACE) continue after percutaneous coronary intervention (PCI) despite advances in revascularisation. Systemic inflammation contributes to atherosclerosis and plaque instability. The Systemic Immune-Inflammation Index (SII) is an inexpensive, easily calculated biomarker derived from routine blood counts that integrates inflammatory and immune pathways. This study evaluated its prognostic value for MACE after PCI.

Methods

This single-center retrospective observational study included 400 consecutive adults undergoing PCI for stable angina or acute coronary syndromes over 1 year. Pre-procedural complete blood counts were used to calculate SII [(platelet × neutrophil)/lymphocyte]. The primary outcome was in-hospital MACE, comprising all-cause mortality, non-fatal myocardial infarction, and ischemic stroke. Patients were stratified by SII quartiles. ROC analysis and multivariable logistic regression assessed predictive performance and independent associations.

Results

MACE occurred in 6.8% (n = 27). SII was significantly higher in patients with MACE than without (1127.19 ± 335.99 vs. 599.92 ± 324.55 $\times 10^9$/L; $p < 0.001$). High SYNTAX scores (> 27) occurred in 72% of the highest versus 1% of the lowest SII quartile ($p < 0.001$). Increasing SII was associated with higher troponin positivity and lower discharge left ventricular ejection fraction ($p < 0.01$). ROC analysis showed excellent predictive ability (AUC 0.886; 95% CI 0.827–0.945), with an optimal cut-off ≥ 776 $\times 10^9$/L. Multivariable analysis identified SII (adjusted OR 5.36 per quartile increase; $p = 0.001$) and diabetes mellitus (adjusted OR 4.41; $p = 0.024$) as independent predictors of MACE.

Conclusion

Elevated SII independently predicts MACE after PCI and may serve as a simple, cost-effective risk-stratification tool in routine clinical practice.

Introduction

Cardiovascular disease still remains the leading cause of mortality worldwide, with coronary artery disease (CAD) and acute coronary syndrome (ACS) accounting for a substantial proportion of cardiovascular deaths.[1,2] Despite all major advances in interventional cardiology—particularly the widespread use of drug-eluting stents, improved revascularization techniques, and optimized

antithrombotic therapy—the incidence of major adverse cardiac events (MACE), including myocardial infarction, stroke, and death, remains significant following percutaneous coronary intervention (PCI).[3–5] These residual adverse outcomes highlight the need for improved risk stratification beyond conventional clinical and angiographic parameters.

Atherosclerosis is now recognized as a chronic inflammatory disease of the arterial wall and forms the fundamental pathological substrate of cardiovascular disease.[6,7] Inflammation contributes not only to plaque formation and progression but also to plaque destabilization and thrombosis, ultimately precipitating the acute ischemic events.[8,9] Inflammatory cells and mediators play a pivotal role throughout the atherosclerotic process, influencing endothelial dysfunction, lipid accumulation, and vascular remodelling. Consequently, the assessment of systemic inflammation has gained increasing importance in cardiovascular risk prediction.

Various hematologic markers derived from routine blood counts have been explored as surrogate indicators of systemic inflammation. Among these, the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have emerged as simple and cost-effective prognostic tools associated with disease severity and adverse cardiovascular outcomes.

In 2014, Hu et al. introduced the Systemic Immune-Inflammation Index (SII), which integrates neutrophil, platelet, and lymphocyte counts to provide a comprehensive assessment of inflammatory and immune status.[10] SII may offer superior prognostic value compared with single inflammatory markers, by reflecting innate immunity, thrombosis, and adaptive immune response. Emerging evidence suggests that SIII, calculated as (platelet count × neutrophil count) / lymphocyte count, is a reliable predictor of adverse outcomes in cardiovascular disease.[11]

Given the persistent burden of MACE after PCI and the central role of inflammation in atherosclerosis, further evaluation of SIII as a prognostic marker in PCI patients is warranted.

Methods

Study Design and Setting

This observational, retrospective cohort study was conducted at a single tertiary care center over a 12-month period. This study complies with the Declaration of Helsinki and was approved by the Institutional Ethics Committee, with chairman head of the committee Dr.Ganesh Dhakale, Government Medical College, Nagpur with No. 3825 dated 28/08/2025. The study included adult patients who underwent percutaneous coronary intervention (PCI) for either stable angina or acute coronary syndromes (ACS), including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina.

Study Population

All consecutive patients aged ≥ 18 years undergoing PCI during the study period were screened for eligibility. Patients were included if they had a confirmed diagnosis of ACS or stable angina and had complete pre-procedural complete blood count (CBC) data available. Patients with active infection or chronic inflammatory disease, known malignancy or hematologic disorders, and use of immunosuppressive therapy within the preceding three months were excluded from study.

Diagnosis of Clinical Diseases

The diagnosis of ACS was established based on current ESC/AHA guidelines, incorporating clinical presentation, electrocardiographic findings, and cardiac biomarker elevation. Stable angina was diagnosed in patients with exertional chest pain or equivalent symptoms relieved by rest or nitroglycerin and supported by objective evidence of ischemia on stress testing or coronary angiography. Hypertension, diabetes mellitus, and smoking history were defined as per standard clinical criteria and documented in the patient records.

Data Collection and Variables

Data was obtained from institutional medical records and angiographic databases. Demographic variables like Age, sex, body mass index (BMI) noted. Clinical variables like Hypertension, diabetes mellitus, smoking status added in database. Laboratory variables like Neutrophil count, lymphocyte count, platelet count obtained from CBC reports and systemic immune-inflammation index (SII), calculated as (platelet count × neutrophil count) / lymphocyte count. Procedural variables also noted like Lesion location, baseline and residual SYNTAX scores, and type of PCI performed. Outcome variables like inhospital Major adverse cardiovascular events (MACE) and left ventricular ejection fraction (LVEF) at baseline and during discharge were collected.

Outcome Definition

The primary outcome was the occurrence of MACE, defined as a composite of all-cause mortality, non-fatal myocardial infarction and ischemic stroke.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was assessed using the Shapiro–Wilk test. Data presented as mean ± SD or median (IQR) as appropriate, and categorical variables as frequencies and percentages. Comparisons between patients with and without MACE were made using the Student's t-test or Chi-square test. Patients were stratified into SII quartiles, with comparisons performed using ANOVA or Chi-square test for trend. ROC analysis assessed predictive performance. Logistic regression identified independent predictors of MACE. All tests were two-tailed, with $p < 0.05$ considered significant.

Results

Study Population and Incidence of MACE

A total of 400 patients undergoing percutaneous coronary intervention (PCI) were included in the analysis. During the study period, Major Adverse Cardiovascular Events (MACE) occurred in 6.8% (n = 27) of patients. The individual components of MACE included all-cause mortality in 3.3%, reinfarction in 2.0%, and stroke in 1.5% of the study population.

Table 1
Baseline Clinical Characteristics and Biomarkers

Variable	Category	n (%)
Sex	Female	97 (24.3%)
	Male	303 (75.8%)
Chest pain duration	< 2 hours	37 (9.3%)
	2–24 hours	164 (41.0%)
	24–72 hours	87 (21.8%)
	> 72 hours	112 (28.0%)
Hypertension	Present	305 (76.3%)
	Absent	95 (23.8%)
Diabetes Mellitus	Present	296 (74.0%)
	Absent	104 (26.0%)
Smoking Status	Smoker	266 (66.5%)
	Non-smoker	134 (33.5%)
Trop T	Positive	272 (68.0%)
	Negative	128 (32.0%)
Clinical Diagnosis	STEMI	200 (50.0%)
	NSTEMI	55 (13.8%)
	Unstable Angina	145 (36.3%)
Type of PTCA	Elective	343 (85.8%)
	Primary	57 (14.2%)
MACE (overall)	Yes	27 (6.8%)
	No	373 (93.3%)
MACE components	Death	13 (3.3%)
	Reinfarction	8 (2.0%)
	Stroke	6(1.5%)

Baseline Clinical Characteristics and Association with MACE

Patients who experienced MACE demonstrated a significantly higher burden of coronary artery disease. A high SYNTAX score (> 27) was present in 85.2% of patients with MACE, compared to 18.2% in those without MACE (p < 0.001).

Table 2
Baseline Clinical Characteristics and Biomarkers by Occurrence of MACE

Variable	Category	No MACE, n (%)	MACE, n (%)	p value*
Sex	Female	90 (24.1)	7 (25.9)	0.833
	Male	283 (75.9)	20 (74.1)	
Hypertension (HTN)	No	89 (23.9)	6 (22.2)	0.847
	Yes	284 (76.1)	21 (77.8)	
Smoking status	No	124 (33.2)	10 (37.0)	0.687
	Yes	249 (66.8)	17 (63.0)	
Diabetes mellitus (DM)	No	94 (25.2)	10 (37.0)	0.176
	Yes	279 (74.8)	17 (63.0)	
Syntax group	> 27	68 (18.2)	23 (85.2)	**< 0.001**
	18–27	43 (11.5)	2 (7.4)	
	< 18	262 (70.2)	2 (7.4)	
Troponin T (Trop T)	Negative	122 (32.7)	6 (22.2)	0.259
	Positive	251 (67.3)	21 (77.8)	

The Systemic Immune-Inflammation Index (SIII) was markedly elevated in patients who developed MACE. Mean SIII values were significantly higher in the MACE group compared to the no-MACE group (1127.19 ± 335.99 ×10⁹/L vs. 599.92 ± 324.55 ×10⁹/L; p < 0.001). Additionally, patients with MACE exhibited significantly lower lymphocyte counts(2.10 ± 0.51 ×10⁹/L vs. 2.59 ± 0.77 ×10⁹/L; p < 0.001).

Although patients with MACE tended to be older and had higher neutrophil counts and lower ejection fraction (EF) at discharge, these differences did not reach statistical significance (age: $p = 0.099$; neutrophils: $p = 0.085$; EF: $p = 0.196$).

Table 3
Continuous Baseline Variables by Occurrence of (MACE) Markers of myocardial injury and systemic inflammation also showed significant trends. Troponin T positivity increased from 53.0% in Q1 to 74.0% in Q4 (P for trend = 0.003).

Variable	MACE, Mean ± SD	No MACE, Mean ± SD	Mean diff	95% CI (diff)	p value*
Age (years)	52.63 ± 10.17	55.97 ± 10.15	-3.34	-7.36 to 0.68	0.099
BMI (kg/m²)	27.56 ± 2.47	27.50 ± 1.77	0.06	-0.89 to 1.01	0.912
Neutrophils (10×10⁹/L)	7.05 ± 2.03	6.19 ± 2.53	0.86	-0.00 to 1.72	0.085
Lymphocytes (10×10⁹/L)	2.10 ± 0.51	2.59 ± 0.77	-0.5	-0.69 to -0.30	**< 0.001**
SIII (10×10⁹/L)	1127.19 ± 335.99	599.92 ± 324.55	527.27	393.74 to 660.80	**< 0.001**
EF on discharge (%)	43.21 ± 12.027 (n = 14)	46.85 ± 10.249 (n = 373)	-3.636	-9.156 to 1.885	0.196

Table 4
Baseline Clinical Characteristics by Systemic Immune-Inflammation Index (SII) Quartiles

Variable	Category	Q1 (</= 389.41), Lowest	Q2 (389.42-534.12–534.12)	Q3 (534.13–803.77)	Q4 (> 803.78), Highest	Total	P trend
Sex	Female	13 (13.0)	28 (28.3)	30 (29.7)	26 (26.0)	97	0.023
	Male	87 (87.0)	71 (71.7)	71 (70.3)	74 (74.0)	303	
Hypertension (HTN)	No	24 (24.0)	18 (18.2)	26 (25.7)	27 (27.0)	95	0.475
	Yes	76 (76.0)	81 (81.8)	75 (74.3)	73 (73.0)	305	
Diabetes mellitus (DM)	No	26 (26.0)	24 (24.2)	29 (28.7)	25 (25.0)	104	0.897
	Yes	74 (74.0)	75 (75.8)	72 (71.3)	75 (75.0)	296	
Smoking status	No	31 (31.0)	36 (36.4)	35 (34.7)	32 (32.0)	134	0.848
	Yes	69 (69.0)	63 (63.6)	66 (65.3)	68 (68.0)	266	
Troponin T (Trop T)	Negative	47 (47.0)	28 (28.3)	27 (26.7)	26 (26.0)	128	**0.003**
	Positive	53 (53.0)	71 (71.7)	74 (73.3)	74 (74.0)	272	
Syntax group	> 27	1 (1.0)	5 (5.1)	13 (12.9)	72 (72.0)	91	**< 0.001**
	18–27	0 (0.0)	8 (8.1)	21 (20.8)	16 (16.0)	45	
	< 18	99 (99.0)	86 (86.9)	67 (66.3)	12 (12.0)	264	

Mean neutrophil counts increased significantly across quartiles, from 4.37 ± 1.41 ×10⁹/L in Q1 to 7.80 ± 3.02 ×10⁹/L in Q4 ($p < 0.001$), while lymphocyte counts decreased progressively, reaching the lowest values in Q4 (2.25 ± 0.64 ×10⁹/L; P for trend < 0.001).

Left ventricular systolic function also deteriorated with increasing SIII. Ejection fraction at discharge declined significantly from 49.85 ± 8.72% in Q1 to 45.00 ± 10.34% in Q4 ($p = 0.003$).

Table 5
Continuous Baseline Variables by Systemic Immune-Inflammation Index (SII) Quartiles

Variable	SII quartile	n	Mean ± SD	ANOVA F (df)	ANOVA p value
Age (years)	Q1 (< 389.41)	100	54.86 ± 9.91	1.493 (3, 396)	0.216
	Q2 (390–534.12)	99	55.02 ± 10.05		
	Q3 (534.13–803.77)	101	57.54 ± 10.75		
	Q4 (> 803.78)	100	55.54 ± 9.86		
BMI (kg/m²)	Q1 (< 389.41)	100	27.75 ± 1.27	1.924 (3, 396)	0.125
	Q2 (390–534.12)	99	27.52 ± 1.70		
	Q3 (534.13–803.77)	101	27.16 ± 2.08		
	Q4 (> 803.78)	100	27.60 ± 2.09		
Neutrophils (10×10⁹/L)	Q1 (< 389.41)	100	4.37 ± 1.41	44.368 (3, 396)	**< 0.001**
	Q2 (390–534.12)	99	6.02 ± 1.77		
	Q3 (534.13–803.77)	101	6.80 ± 2.16		
	Q4 (> 803.78)	100	7.80 ± 3.02		
Lymphocytes (10×10⁹/L)	Q1 (< 389.41)	100	2.66 ± 0.75	10.874 (3, 396)	**< 0.001**
	Q2 (390–534.12)	99	2.82 ± 0.75		
	Q3 (534.13–803.77)	101	2.51 ± 0.82		
	Q4 (> 803.78)	100	2.25 ± 0.64		
EF on discharge (%)	Q1 (< 389.41)	100	49.85 ± 8.72	4.619 (3, 383)	**0.003**
	Q2 (390–534.12)	98	46.48 ± 10.38		
	Q3 (534.13–803.77)	101	45.35 ± 11.14		

Variable	SII quartile	n	Mean ± SD	ANOVA F (df)	ANOVA p value
	Q4 (> 803.78)	88	45.00 ± 10.34		

Clinical and Laboratory Characteristics According to SIII Quartiles

Patients were stratified into four quartiles based on SIII distribution, with Q1 (≤ 389.41 ×10⁹/L) representing the lowest and Q4 (≥ 803.78 ×10⁹/L) the highest quartile.

A strong and significant association was observed between increasing SIII quartiles and disease severity. The prevalence of a high SYNTAX score (> 27) increased progressively across quartiles (P for trend < 0.001), with 72.0% of patients in Q4 exhibiting a high SYNTAX score compared to only 1.0% in Q1.

Predictive Performance of SIII for MACE

Receiver operating characteristic (ROC) curve analysis demonstrated that SIII had excellent discriminatory power for predicting MACE, with an area under the curve (AUC) of 0.886 (95% CI: 0.827–0.945; p < 0.001).

The optimal SIII cut-off value for predicting MACE was identified as ≥ 776 ×10⁹/L, yielding a sensitivity of 85.2%, specificity of 77.5%, and a Youden index of 0.63.

Multivariable Predictors of MACE

On multivariable logistic regression analysis adjusted for age, sex, body mass index, hypertension, smoking status, and ejection fraction at discharge, both SIII and diabetes mellitus emerged as independent predictors of MACE.

Each one-quartile increase in SIII was associated with a 5.36-fold increase in the odds of MACE (adjusted OR 5.36, 95% CI 2.06–13.98; p = 0.001). Diabetes mellitus was also independently associated with adverse outcomes (adjusted OR 4.41, 95% CI 1.22–16.00; p = 0.024).

Table 6
Univariable and multivariable logistic regression analyses for the association of clinical variables with major adverse cardiovascular events (MACE).

Variable	Univariable OR (95% CI)	P-value	Multivariable OR (95% CI)	P-value
Age (per year)	0.98 (0.95–1.02)	0.126	0.96 (0.90–1.03)	0.237
Male sex	1.15 (0.55–2.41)	0.704	0.44 (0.06–3.04)	0.401
BMI (per kg/m²)	1.03 (0.97–1.10)	0.316	1.09 (0.84–1.41)	0.503
Hypertension	1.14 (0.62–2.09)	0.685	1.83 (0.45–7.47)	0.401
Diabetes mellitus	2.21 (1.04–4.70)	0.038	4.41 (1.22–16.00)	**0.024**
Current/past smoking	1.28 (0.67–2.45)	0.455	2.91 (0.47–17.91)	0.25
Ejection fraction on discharge (per %)	0.98 (0.96–1.01)	0.195	0.99 (0.94–1.05)	0.709
SII (per 1-quartile increase)	**6.33 (2.90–13.81)**	**< 0.001**	**5.36 (2.06–13.98)**	**0.001**

Discussion

In this observational study involving patients undergoing percutaneous coronary intervention, we demonstrated that an elevated Systemic Immune-Inflammation Index (SIII) is a strong and independent predictor of Major Adverse Cardiovascular Events (MACE). Our findings reinforce and extend existing evidence highlighting the pivotal role of systemic inflammation in determining cardiovascular risk across the spectrum of coronary artery disease.

Our observation that higher SIII values are significantly associated with increased MACE incidence is consistent with the findings of Saylik and Akbulut, who reported that elevated SIII independently predicted major adverse cardiovascular events in patients with ST-segment elevation myocardial infarction (STEMI).[12] While their study focused exclusively on STEMI patients, our study extends these findings to a broader PCI population, including elective and non-ST elevation presentations, underscoring the generalizability of SIII as a prognostic biomarker.

Similarly, Öcal et al. demonstrated that SIII predicted both in-hospital and long-term outcomes in STEMI patients.[13] The strength of association observed in our study—where each one-quartile increase in SIII was associated with a more than five-fold increase in MACE risk—appears comparable or even stronger, likely reflecting differences in patient characteristics and outcome definitions.

Our study revealed a strong association between increasing SIII quartiles and higher SYNTAX scores, indicating greater coronary disease complexity. This finding closely parallels the results of Gur et al., who demonstrated that SIII is a determinant of atherosclerotic burden and identifies high-risk patients with

acute coronary syndromes.[14] Similar associations between SIII and angiographic severity have also been reported by Liu et al. and Dziedzic et al., further supporting the role of systemic inflammation in diffuse and complex coronary artery disease.[15,16]

The progressive increase in troponin positivity with rising SIII levels in our cohort suggests greater myocardial injury among patients with heightened inflammatory burden. This observation aligns with findings by Chen et al., who demonstrated that inflammation-based indices such as the neutrophil-to-lymphocyte ratio correlate with myocardial damage and impaired cardiac function in acute coronary syndrome patients.[17]

Furthermore, Demirci et al. reported a significant relationship between elevated SIII and the no-reflow phenomenon during PCI for saphenous vein graft lesions.[18] Although no-reflow was not specifically assessed in our study, the association between higher SIII, increased myocardial injury, and worse outcomes suggests overlapping inflammatory mechanisms contributing to microvascular dysfunction.

An important and relatively underreported finding of our study is the progressive decline in left ventricular ejection fraction with increasing SIII quartiles. This observation supports earlier work by Chen et al. and Fan et al., who reported that inflammatory indices were associated with myocardial damage and subsequent ventricular dysfunction in acute coronary syndrome patients undergoing PCI.[17,19] Our data add to this evidence by demonstrating that SIII correlates not only with ischemic burden but also with early post-PCI ventricular performance.

The excellent discriminative performance of SIII in our study (AUC 0.886) compares favourably with previously published literature. Huang et al. reported that SIII effectively predicted clinical outcomes in elderly patients with acute myocardial infarction undergoing PCI, although with slightly lower predictive accuracy.[20] Differences in population demographics, age distribution, and inflammatory profiles may explain the higher AUC observed in our cohort.

Our findings are further supported by large population-based and mechanistic studies. Wei et al. demonstrated that both SIII and systemic inflammatory response indices are significantly associated with the clinical risk of acute myocardial infarction,[21] while Ma and Li, using NHANES data, confirmed a strong association between SIII and coronary heart disease prevalence.[22] These data collectively emphasize that SIII reflects a systemic inflammatory milieu that promotes plaque instability, thrombosis, and adverse cardiovascular outcomes.

In multivariable analysis, both SIII and diabetes mellitus independently predicted MACE in our study. The independent prognostic value of SIII after adjustment for conventional risk factors mirrors findings from multiple prior investigations,[23–25] and highlights the incremental utility of inflammation-based indices beyond traditional clinical parameters.

Given its simplicity, low cost, and availability from routine blood tests, SIII may serve as a practical risk stratification tool to identify high-risk PCI patients who may benefit from intensified surveillance and aggressive secondary prevention strategies.

Limitations

As with previous studies, the observational, single-center design limits causal inference. SIII was assessed at a single time point, and serial measurements may provide additional prognostic information. Nevertheless, the consistency of our findings with prior literature strengthens their validity.

Conclusion

In agreement with and extending prior studies, our results demonstrate that elevated SIII is a powerful and independent predictor of MACE following PCI. Its strong association with coronary complexity, myocardial injury, ventricular dysfunction, and adverse clinical outcomes supports the role of SIII as an integrated biomarker of inflammatory and immune dysregulation in contemporary interventional cardiology practice.

Highlights

Systemic Immune-Inflammation Index (SII) is a simple, inexpensive biomarker derived from routine blood counts that integrates inflammatory and immune status.

Elevated SII is strongly associated with increased risk of in-hospital major adverse cardiovascular events (MACE) in patients undergoing PCI.

SII shows excellent predictive performance for MACE (AUC 0.886) with an optimal cut-off value ≥ 776 $\times 10^9$/L.

Higher SII levels correlate with greater coronary artery disease complexity, higher troponin positivity, and reduced left ventricular ejection fraction.

SII and diabetes mellitus are independent predictors of MACE, supporting the role of SII in routine risk stratification after PCI.

Declarations

Ethics Approval and Consent to Participate

This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was reviewed and approved by the **Institutional Ethics Committee, with chairman head of the committee Dr.Ganesh Dhakale, Government Medical College, Nagpur**, with Ethics Committee approval number **3825**, dated **28/08/2025**. As this was a retrospective observational study based on existing

medical records and angiographic databases, individual informed consent was not required, as per the decision of the Ethics Committee.

Consent for Publication

Not Applicable. This manuscript does not contain identifying images or other personal or clinical details that could compromise the anonymity of individual participants.

Competing Interests

The authors declare that they have no competing interests.

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Author Contribution

Conceptualization: Sonali Gambhire, Rupesh Agrawal, Pradeep Deshmukh; Methodology: Sonali Gambhire, Rupesh Agrawal, Sunil Washimkar, Pradeep Deshmukh; Formal analysis: Sonali Gambhire, Rupesh Agrawal; Investigation: Sonali Gambhire, Rupesh Agrawal, Sunil Washimkar; Data curation: Sonali Gambhire, Rupesh Agrawal, Sunil Washimkar; Writing – original draft: Sonali Gambhire; Writing – review & editing: Sonali Gambhire, Rupesh Agrawal, Sunil Washimkar, Pradeep Deshmukh; Validation: Sunil Washimkar, Pradeep Deshmukh; Supervision: Rupesh Agrawal, Sunil Washimkar, Pradeep Deshmukh; Project administration: Sonali Gambhire, Pradeep Deshmukh.All authors have read and approved the final manuscript

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available because they contain information derived from patient medical records and may contain potentially identifiable patient information. Access to the data may be provided subject to appropriate ethical and institutional approvals.

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Figures

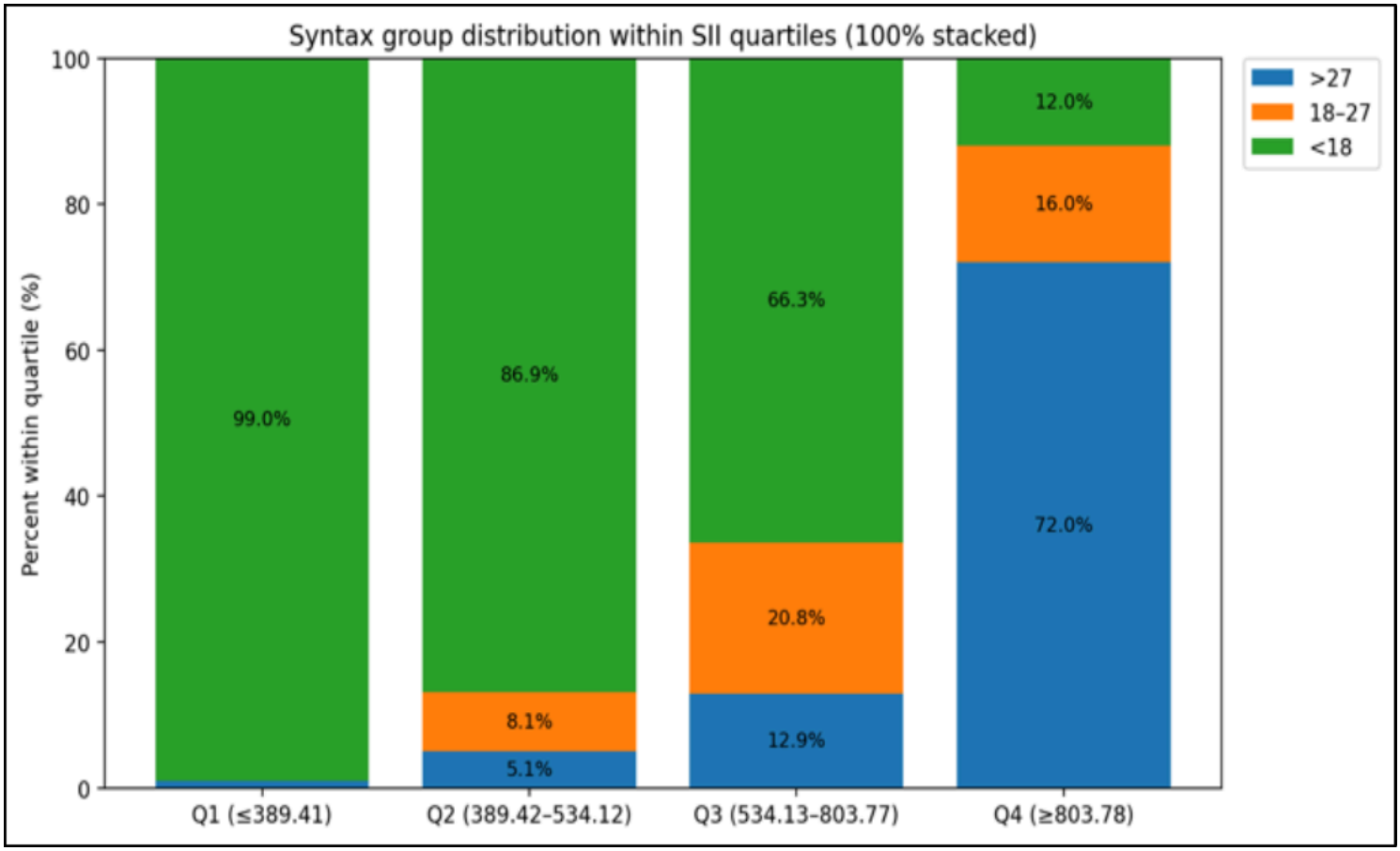


Figure 1

SYNTAX Score Distribution Within SII Quartiles.

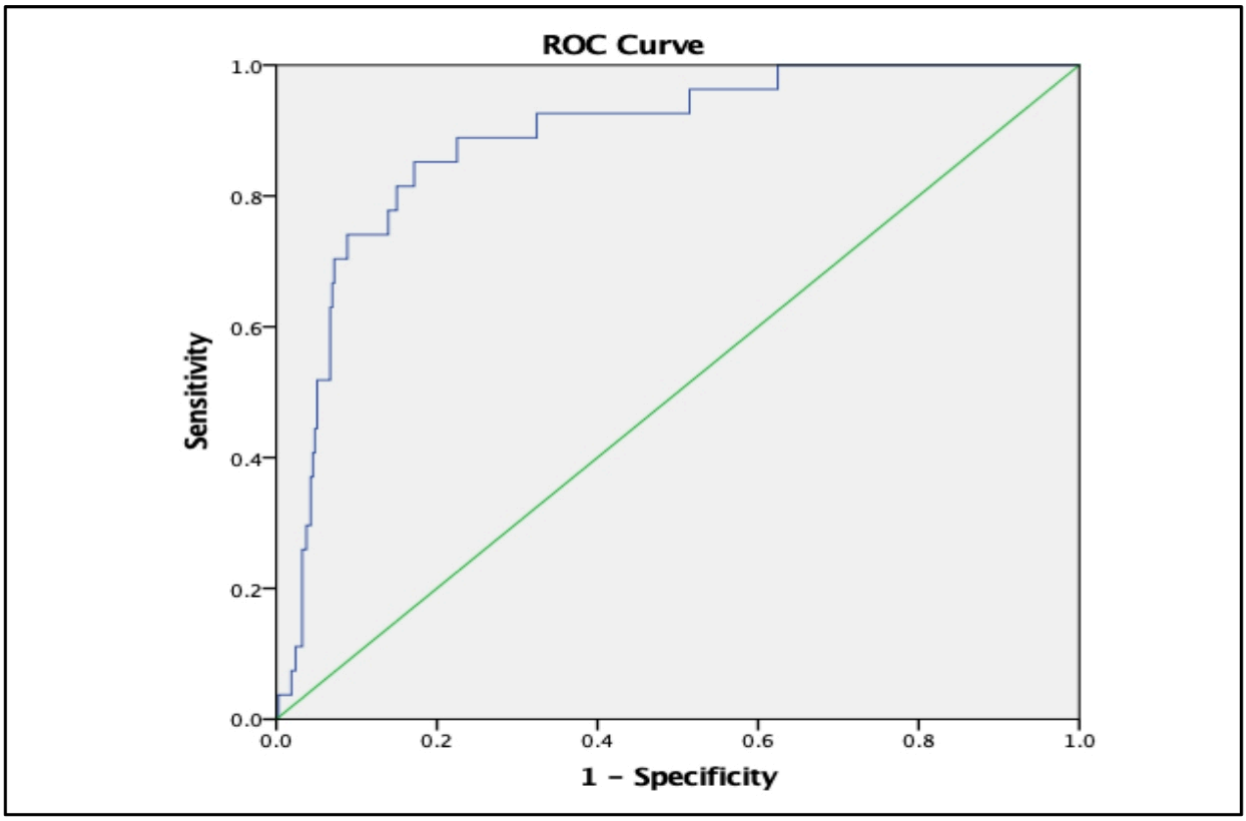


Figure 2

ROC Curve for Predictive Performance of SIII for MACE.

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