

High Systemic Immune-Inflammation Index as a Predictor of In-Hospital and 30-Day Major Adverse Cardiovascular Events among Patients with STEMI and NSTEMI Undergoing Percutaneous Coronary Intervention: A Prospective Cohort Study

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Research Article

Keywords: acute coronary syndrome, major adverse cardiovascular events, non-ST-segment elevation myocardial infarction, percutaneous coronary intervention, ST-segment elevation myocardial infarction, systemic immune-inflammation index

Posted Date: July 15th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10356261/v1>

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Additional Declarations: No competing interests reported.

Abstract

Background

Systemic inflammation contributes to plaque instability, thrombosis, and adverse outcomes after acute coronary syndrome (ACS). The systemic immune-inflammation index (SII) integrates platelet, neutrophil, and lymphocyte counts, but prospective evidence after percutaneous coronary intervention (PCI) remains limited. This study evaluated admission SII as a predictor of in-hospital and 30-day major adverse cardiovascular events (MACE).

Methods

This prospective cohort study enrolled 120 consecutive adults with ST-segment elevation myocardial infarction (STEMI) or non-ST-segment elevation myocardial infarction (NSTEMI) who underwent PCI at a tertiary referral hospital in Indonesia. SII, neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) were calculated from admission complete blood counts. Receiver operating characteristic analysis determined optimal cut-offs. Cox proportional hazards regression evaluated independent predictors of MACE, and Kaplan-Meier analysis compared 30-day event-free survival.

Results

The optimal SII cut-off was 1101.32 (AUC 0.750, 95% CI 0.664–0.836; sensitivity 83%; specificity 60%). The corresponding AUCs were 0.768 for NLR and 0.738 for PLR. In-hospital MACE occurred in 62.0% of the high-SII group and 18.4% of the low-SII group (RR 3.37, 95% CI 1.819–6.258; $p < 0.001$). High SII independently predicted MACE (adjusted HR 3.96, 95% CI 1.907–8.242; $p < 0.001$). Mean event-free survival was 12.71 $\pm$ 1.62 days with high SII and 25.38 $\pm$ 1.48 days with low SII (log-rank $p < 0.001$). In the STEMI subgroup, high SII remained independently predictive (adjusted HR 3.89, 95% CI 1.796–8.429; $p < 0.001$). Thirty-day MACE after discharge did not differ significantly (4.2% vs 0%; $p = 0.145$).

Conclusion

High admission SII independently predicted earlier MACE and identified patients with a markedly higher incidence of in-hospital events after PCI. Its association with post-discharge 30-day MACE was not statistically significant. SII may provide a low-cost adjunct for early risk stratification in ACS.

Introduction

Acute coronary syndrome (ACS), encompassing ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), remains a major cause of cardiovascular morbidity and mortality despite advances in reperfusion and pharmacological treatment. Percutaneous

coronary intervention (PCI) improves epicardial reperfusion and survival, yet major adverse cardiovascular events (MACE) continue to occur during hospitalization and early after discharge. Early identification of high-risk patients is therefore essential to guide monitoring, treatment intensity, and allocation of critical-care resources [1–5].

The pathophysiology of ACS extends beyond coronary occlusion. Plaque rupture or erosion triggers a thrombo-inflammatory cascade involving platelet activation, neutrophil recruitment, endothelial dysfunction, oxidative stress, and coagulation. Inflammatory activation may persist after successful reperfusion and contribute to microvascular obstruction, reperfusion injury, adverse ventricular remodeling, recurrent ischemia, cardiogenic shock, malignant arrhythmias, heart failure, and mortality [6–10].

The systemic immune-inflammation index (SII), calculated as platelet count multiplied by neutrophil count and divided by lymphocyte count, integrates thrombotic activity, innate inflammation, and adaptive immune status. It may provide a broader representation of thrombo-inflammatory burden than the neutrophil-to-lymphocyte ratio (NLR) or platelet-to-lymphocyte ratio (PLR), although comparative performance varies across populations [11–14].

Observational studies and meta-analyses have associated elevated SII with mortality, heart failure, no-reflow, recurrent myocardial infarction, in-stent complications, and composite MACE after PCI. Reported cut-offs vary because of differences in ethnicity, case mix, sampling time, outcome definition, and follow-up duration [15–31]. Local validation is therefore necessary before SII can be incorporated into routine clinical risk assessment.

Evidence from Indonesia remains limited, particularly from prospective cohorts evaluating in-hospital and early post-discharge outcomes. This study aimed to determine the optimal admission SII cut-off, compare the discriminatory performance of SII with NLR and PLR, and assess whether high SII independently predicts MACE in patients with STEMI and NSTEMI undergoing PCI.

Methods

Study design and setting

This analytical prospective cohort study was conducted from December 2025 to April 2026 at a tertiary cardiovascular referral hospital in Denpasar, Bali, Indonesia. Consecutive sampling was used. Patients were followed during the index hospitalization and for 30 days after discharge.

Participants

Adults aged 18 years or older with STEMI undergoing PCI or NSTEMI undergoing PCI within 72 hours were eligible. The diagnosis was established using clinical, electrocardiographic, and cardiac biomarker

criteria consistent with the Fourth Universal Definition of Myocardial Infarction [3].

Exclusion criteria were acute infection, severe infection or sepsis; a history of malignancy; end-stage kidney disease requiring hemodialysis; chronic hepatitis or cirrhosis; autoimmune disease; coronary artery spasm or another secondary cause of angina or myocardial infarction; previous systemic corticosteroid use; refusal to participate; or incomplete data required for analysis.

Sample size

The minimum sample size was calculated for an observational cohort using expected in-hospital MACE proportions of 33.3% in the high-SII group and 10.4% in the low-SII group, with a two-sided alpha of 0.05 and 80% power. The calculation yielded 50 participants per group. After allowing 10% for attrition, the target sample was 110; 120 participants were ultimately enrolled.

Data collection and inflammatory indices

Demographic data, cardiovascular risk factors, clinical presentation, laboratory measurements, echocardiographic findings, angiographic characteristics, and procedural data were prospectively recorded. A complete blood count was obtained at admission before PCI using the hospital automated hematology analyzer. SII was calculated as platelet count x neutrophil count / lymphocyte count. NLR was calculated as neutrophil count / lymphocyte count, and PLR as platelet count / lymphocyte count.

Outcome measures

The primary outcome was in-hospital MACE, defined as the occurrence of at least one prespecified event during the index hospitalization: cardiovascular death, acute heart failure, malignant ventricular arrhythmia, stroke, or cardiogenic shock. The secondary outcome was MACE occurring during the 30 days after discharge. Outcomes were ascertained from hospital records and structured follow-up contact.

Statistical analysis

Analyses were performed using IBM SPSS Statistics version 26.0. Categorical data are presented as n (%). Continuous variables were assessed for distribution and summarized as mean +/- standard deviation or median (interquartile range), as appropriate. High- and low-SII groups were compared using the chi-square test or Fisher exact test for categorical variables and the independent-samples t test or Mann-Whitney U test for continuous variables.

Receiver operating characteristic (ROC) curves were used to determine optimal cut-offs for SII, NLR, and PLR and to estimate the area under the curve (AUC), 95% confidence interval, sensitivity, and specificity.

The AUCs were compared descriptively; no formal between-curve significance test was prespecified. The SII cut-off was 1101.32, defining high SII as ≥ 1101.32 and low SII as < 1101.32 .

Bivariable associations with MACE were reported as risk ratios (RRs) with 95% confidence intervals. Variables with $p < 0.25$ in bivariable analysis were entered into multivariable Cox proportional hazards regression; adjusted hazard ratios (aHRs) with 95% confidence intervals were reported. The final model included age > 60 years, high SII, smoking, and GRACE score > 140 . Kaplan-Meier curves and the log-rank test compared event-free survival by SII category. A prespecified sensitivity analysis repeated the Cox model in the STEMI subgroup because STEMI accounted for most participants. A two-sided $p < 0.05$ was considered statistically significant.

Ethics approval and consent to participate

The study was approved by the institutional health research ethics committee (institution and approval number blinded for peer review). Written informed consent was obtained from every participant before enrollment. The study complied with the Declaration of Helsinki.

Results

Participant characteristics

A total of 120 patients were included. Seventy-one (59.2%) had high admission SII and 49 (40.8%) had low SII. The groups were comparable in age, sex, hypertension, diabetes mellitus, smoking, obesity, kidney function, GRACE score, infarction type, left ventricular ejection fraction, culprit vessel, and extent of coronary disease (Table 1). STEMI accounted for 109 of 120 participants (90.8%).

Table 1. Baseline characteristics according to admission SII

Characteristic	High SII (n=71)	Low SII (n=49)	p value
Age >60 years	32 (45.1)	26 (53.1)	0.389
Male sex	61 (85.9)	41 (83.7)	0.735
Hypertension	30 (42.3)	24 (49.0)	0.467
Diabetes mellitus	13 (18.3)	11 (22.4)	0.577
Current smoking	41 (57.7)	36 (73.5)	0.077
Obesity	33 (46.5)	26 (53.1)	0.478
eGFR <90 mL/min/1.73 m ²	49 (69.0)	34 (69.4)	0.965
GRACE score >140	12 (16.9)	6 (12.2)	0.483
STEMI	64 (90.1)	45 (91.8)	0.752
LVEF <40%	19 (26.8)	11 (22.4)	0.592
LAD culprit lesion	40 (56.3)	25 (51.0)	0.729
Multivessel disease	46 (64.8)	34 (69.4)	0.599

Data are n (%). eGFR, estimated glomerular filtration rate; GRACE, Global Registry of Acute Coronary Events; LAD, left anterior descending artery; LVEF, left ventricular ejection fraction; STEMI, ST-segment elevation myocardial infarction.

Discriminatory performance of inflammatory indices

SII, NLR, and PLR each discriminated patients who developed MACE, with all AUCs exceeding 0.70 (Table 2). NLR had the numerically highest AUC (0.768), followed by SII (0.750) and PLR (0.738). SII had the highest sensitivity (83%). These differences were descriptive and were not tested using a formal comparison of correlated ROC curves.

Table 2. ROC performance of SII, NLR, and PLR for MACE

Index	AUC	95% CI	Cut-off	Sensitivity	Specificity	p value
SII	0.750	0.664-0.836	1101.32	83%	60%	<0.001
NLR	0.768	0.684-0.853	5.86	71%	71%	<0.001
PLR	0.738	0.650-0.825	143.53	81%	61%	<0.001

AUC, area under the curve; CI, confidence interval; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; ROC, receiver operating characteristic; SII, systemic immune-inflammation index.

[Insert Figure 1 near here]

Figure 1. ROC curves of SII, NLR, and PLR for MACE. Receiver operating characteristic curves compare the discriminatory performance of admission systemic immune-inflammation index, neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio for major adverse cardiovascular events.

Clinical outcomes

In-hospital MACE occurred in 44 patients (62.0%) with high SII and 9 (18.4%) with low SII ($p<0.001$). The bivariable RR was 3.37 (95% CI 1.819-6.258). No individual component differed significantly between SII groups. Cardiogenic shock and post-discharge 30-day MACE were numerically more frequent in the high-SII group, whereas both observed strokes occurred in the low-SII group (Table 3).

Table 3. Clinical outcomes according to admission SII

Outcome	High SII (n=71)	Low SII (n=49)	p value
In-hospital MACE	44 (62.0)	9 (18.4)	<0.001
Cardiovascular mortality	9 (12.7)	4 (8.2)	0.434
Acute heart failure	12 (16.9)	6 (12.2)	0.483
Malignant arrhythmia	6 (8.5)	1 (2.0)	0.238
Stroke	0 (0.0)	2 (4.1)	0.165
Cardiogenic shock	16 (22.5)	4 (8.2)	0.058
30-day MACE after discharge	3 (4.2)	0 (0.0)	0.145

Data are n (%). Components of in-hospital MACE were not mutually exclusive. MACE, major adverse cardiovascular events.

Cox regression and survival analysis

Age >60 years, high SII, smoking, and GRACE score >140 entered the multivariable Cox model. High SII was the only statistically significant independent predictor, with an aHR of 3.96 (95% CI 1.907-8.242; $p<0.001$). GRACE score >140 showed a non-significant trend (aHR 1.85; $p=0.075$).

Kaplan-Meier analysis showed shorter event-free survival in the high-SII group. Estimated mean event-free survival was 12.71 +/- 1.62 days (95% CI 9.543-15.894) with high SII and 25.38 +/- 1.48 days (95% CI 22.477-28.299) with low SII; the log-rank test was significant (chi-square=21.462; $p<0.001$).

[Insert Figure 2 near here]

Figure 2. Thirty-day event-free survival by admission SII category. Kaplan-Meier curves compare patients with high and low admission systemic immune-inflammation index; tick marks indicate censored

observations.

STEMI subgroup analysis

Because 109 of 120 participants had STEMI, the multivariable Cox model was repeated in this subgroup. High SII remained the only statistically significant independent predictor (aHR 3.89, 95% CI 1.796-8.429; $p<0.001$), with an effect estimate similar to that of the overall cohort (Table 4).

Table 4. Cox models in the overall cohort and STEMI subgroup

Variable	Overall cohort aHR (95% CI)	p value	STEMI subgroup aHR (95% CI)	p value
Age >60 years	1.10 (0.604-2.028)	0.742	0.96 (0.508-1.824)	0.907
High SII	3.96 (1.907-8.242)	<0.001	3.89 (1.796-8.429)	<0.001
Current smoking	1.00 (0.567-1.782)	0.985	1.19 (0.658-2.158)	0.563
GRACE score >140	1.85 (0.940-3.672)	0.075	1.90 (0.950-3.817)	0.069

aHR, adjusted hazard ratio; CI, confidence interval; GRACE, Global Registry of Acute Coronary Events; SII, systemic immune-inflammation index; STEMI, ST-segment elevation myocardial infarction.

Discussion

This prospective cohort found that high admission SII was associated with a markedly greater incidence of in-hospital MACE after PCI and independently predicted earlier MACE in Cox regression. Patients with $SII \geq 1101.32$ had an in-hospital MACE rate of 62.0%, compared with 18.4% in those with lower values, and a nearly fourfold adjusted hazard. The association remained consistent when the analysis was restricted to the STEMI subgroup.

The biological plausibility of this association is strong. Neutrophils contribute to endothelial injury, oxidative stress, proteolytic plaque destabilization, neutrophil extracellular trap formation, and thrombus propagation. Platelets participate in thrombosis and inflammatory signaling through interactions with leukocytes and endothelium. Relative lymphopenia reflects stress-related suppression of adaptive immune regulation. SII combines these three pathways and therefore provides a compact measure of acute thrombo-inflammatory burden [6-14].

The present findings are consistent with cohort studies and meta-analyses linking high SII to mortality, heart failure, impaired myocardial perfusion, no-reflow, recurrent ischemic events, and composite MACE after acute myocardial infarction and PCI [15-31]. The locally derived cut-off of 1101.32 lies within the broad range reported internationally. Variation across studies is expected because SII is influenced by

population characteristics, timing of blood sampling, laboratory methods, clinical severity, and outcome definitions.

Comparison with NLR and PLR requires a balanced interpretation. All three indices demonstrated acceptable discrimination. NLR had the highest numerical AUC (0.768), followed by SII (0.750) and PLR (0.738), whereas SII had the highest sensitivity (83%) and the largest bivariable RR for MACE. Because the AUCs were compared descriptively rather than with a formal correlated-curve test, the present data support comparable performance, not statistical superiority of one index. SII may nevertheless offer biological appeal by simultaneously integrating neutrophils, platelets, and lymphocytes.

Individual clinical outcomes did not differ significantly. Cardiogenic shock approached significance, but the number of events was limited. Importantly, stroke should not be interpreted as more frequent with high SII: both strokes occurred in the low-SII group, and the difference was non-significant ($p=0.165$). Only three post-discharge 30-day MACE events occurred, all in the high-SII group, but this difference was also non-significant. The study is therefore more informative for acute in-hospital risk than for isolated post-discharge events.

The STEMI sensitivity analysis strengthens the primary inference. STEMI comprised 90.8% of the cohort, and high SII remained independently predictive with an aHR of 3.89, closely matching the overall estimate of 3.96. This consistency suggests that the main result was not explained by combining distinct STEMI and NSTEMI treatment pathways, although the small NSTEMI sample prevents a reliable NSTEMI-specific estimate.

From a clinical perspective, SII is inexpensive, rapidly available, and calculated from a routine complete blood count. It may help identify patients who warrant closer hemodynamic monitoring, early recognition of heart failure or arrhythmia, and a lower threshold for intensive observation after PCI. SII should be viewed as an adjunct rather than a replacement for clinical assessment, electrocardiography, cardiac biomarkers, ventricular function, angiographic findings, and validated scores such as GRACE [32-37]. Future studies should test whether adding SII to established risk scores improves discrimination, calibration, and clinical decision-making.

This study has several strengths: prospective enrollment, standardized pre-PCI blood sampling, consecutive recruitment, reporting of both discrimination and time-to-event analyses, and a subgroup analysis addressing the predominance of STEMI. The principal limitations are its single-center design, modest sample size, small number of individual and post-discharge events, lack of serial SII measurements, absence of external validation, and limited ability to control all clinical and angiographic determinants such as ischemic time, thrombus burden, post-PCI TIMI flow, no-reflow, antiplatelet regimen, and post-discharge adherence. Larger multicenter cohorts with longer follow-up and prespecified incremental-value analyses are required.

Conclusion

High admission SII was independently associated with earlier MACE and a substantially greater incidence of in-hospital MACE among patients with STEMI and NSTEMI undergoing PCI. SII ≥ 1101.32 conferred an adjusted hazard ratio of 3.96, and the association remained consistent in the STEMI subgroup. Admission SII was not significantly associated with isolated post-discharge 30-day MACE in this cohort. As a low-cost index derived from routine blood counts, SII may complement established early risk-stratification methods, pending external validation.

Abbreviations

ACS: acute coronary syndrome; aHR: adjusted hazard ratio; AUC: area under the curve; CI: confidence interval; eGFR: estimated glomerular filtration rate; GRACE: Global Registry of Acute Coronary Events; LAD: left anterior descending artery; LVEF: left ventricular ejection fraction; MACE: major adverse cardiovascular events; NLR: neutrophil-to-lymphocyte ratio; NSTEMI: non-ST-segment elevation myocardial infarction; PCI: percutaneous coronary intervention; PLR: platelet-to-lymphocyte ratio; ROC: receiver operating characteristic; RR: risk ratio; SII: systemic immune-inflammation index; STEMI: ST-segment elevation myocardial infarction.

Declarations

Acknowledgements

The authors thank the Department of Cardiology and Vascular Medicine, Prof. Dr. I.G.N.G. Ngoerah Central General Hospital, the patients who participated, and the healthcare professionals involved in recruitment, data collection, and follow-up.

Authors' contributions

MCH: conceptualization, data collection, investigation, formal analysis, and writing - original draft. MCH and IKSSD: conceptualization and formal analysis. MCH, IKSSD, and HW: methodology. IKSSD and HW: supervision and writing - review and editing. All authors read and approved the final manuscript.

Funding

The authors received no external funding for this study.

Availability of data and materials

The datasets supporting the conclusions of this article are available from the corresponding author upon reasonable request, subject to institutional ethics and data-protection requirements.

Ethics approval and consent to participate

The study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Udayana University/Prof. Dr. I.G.N.G. Ngoerah Central General Hospital, Denpasar, Indonesia (No.

0388/UN14.2.2.VII.14/LT/2026). Written informed consent was obtained from all participants before enrollment. The study was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable. The manuscript contains no identifiable individual participant information.

Competing interests

The authors declare no competing interests.

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Figures

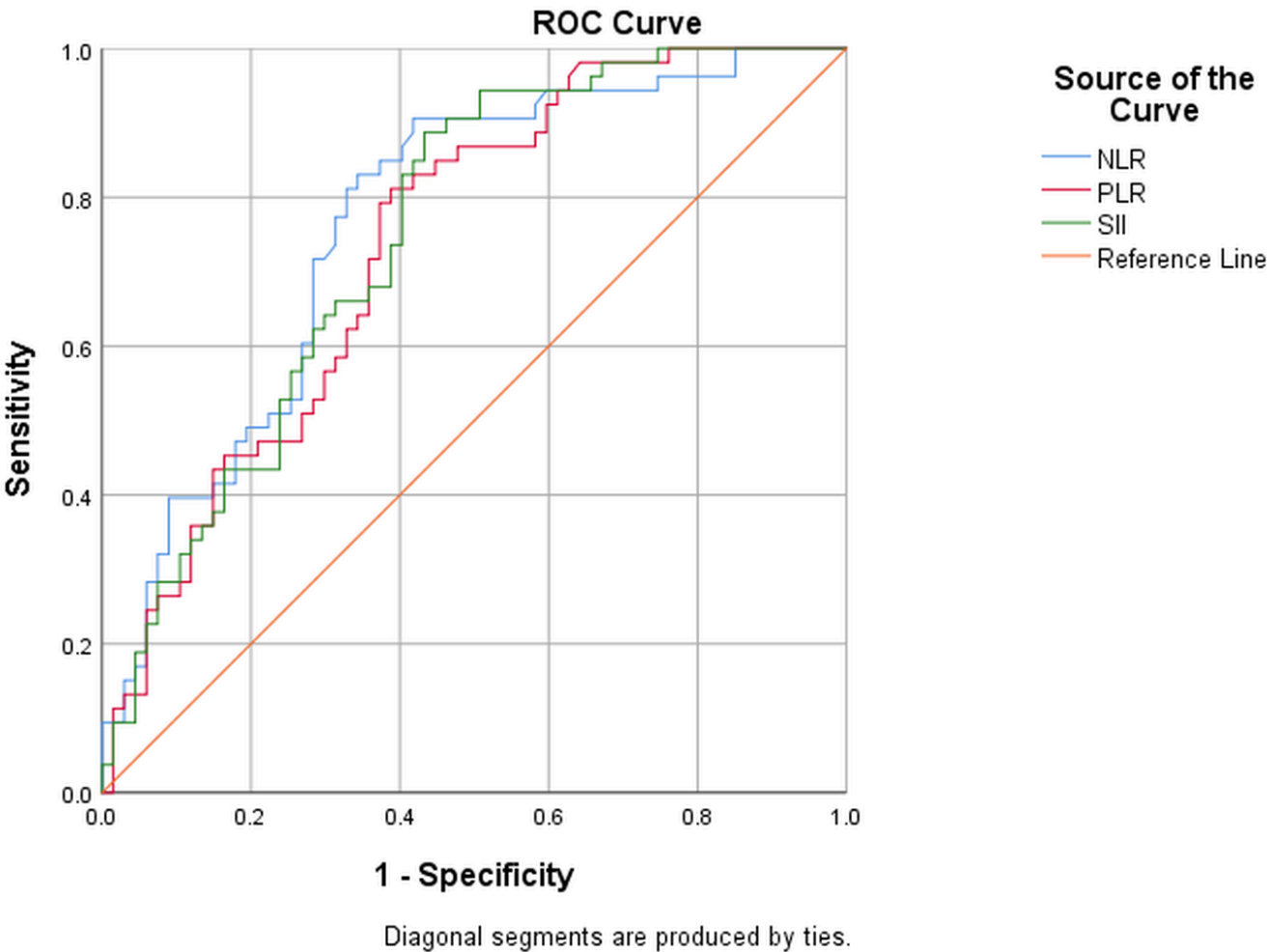


Figure 1

ROC curves of SII, NLR, and PLR for MACE. Receiver operating characteristic curves compare the discriminatory performance of admission systemic immune-inflammation index, neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio for major adverse cardiovascular events.

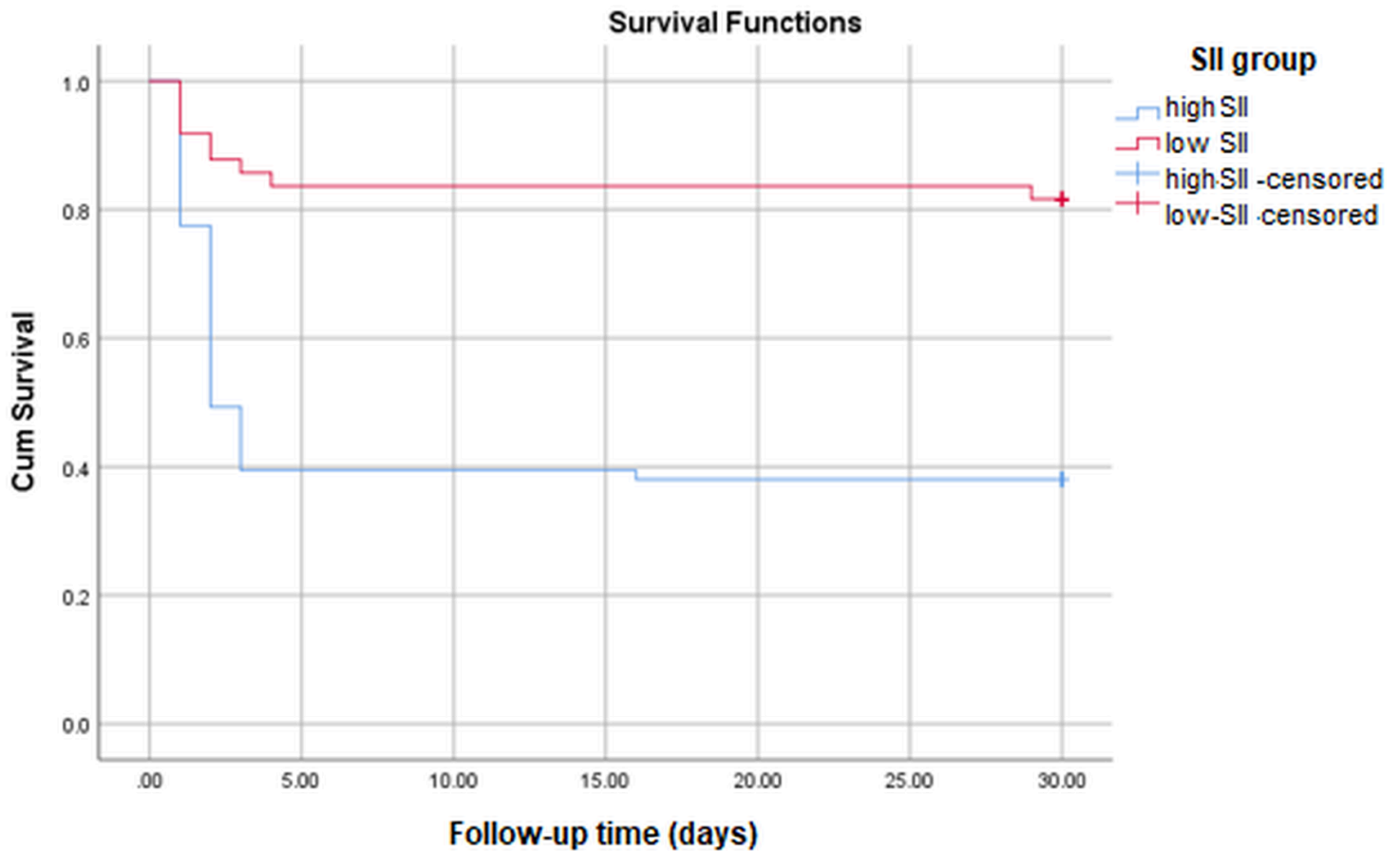


Figure 2

Thirty-day event-free survival by admission SII category. Kaplan-Meier curves compare patients with high and low admission systemic immune-inflammation index; tick marks indicate censored observations.

Supplementary Files

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