

High Admission Systemic Inflammation Response Index as an Independent Predictor of In-Hospital Adverse Events Among Patients with Acute Heart Failure: A Prospective Cohort Study

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Abstract

Background

Acute heart failure is associated with substantial in-hospital morbidity and mortality, and systemic inflammation contributes to myocardial injury, endothelial dysfunction, and multiorgan impairment. The systemic inflammation response index (SIRI), derived from neutrophil, monocyte, and lymphocyte counts, may provide a practical measure of inflammatory burden. This study evaluated whether admission SIRI independently predicts in-hospital adverse events in patients with acute heart failure.

Results

From January to March 2026, a prospective longitudinal observational study consecutively enrolled 102 adults hospitalized with acute heart failure at a tertiary referral hospital in Indonesia. Admission SIRI was calculated as absolute neutrophil count multiplied by absolute monocyte count and divided by absolute lymphocyte count. Participants were followed prospectively until discharge, in-hospital death, or 30 days of hospitalization, whichever occurred first. Forty-two patients (41.2%) experienced at least one adverse event. Median SIRI was higher in patients with adverse events than in those without events (7.08 [interquartile range 5.28–15.80] vs 2.52 [1.79–3.74]; $p < 0.001$). The area under the receiver operating characteristic curve was 0.944 (95% confidence interval 0.902–0.986; $p < 0.001$), with an optimal cut-off of 4.295. Adverse events occurred in 38 of 45 patients (84.4%) with $\text{SIRI} \geq 4.295$ and in 4 of 57 patients (7.0%) with $\text{SIRI} < 4.295$ (unadjusted odds ratio 71.93, 95% confidence interval 19.66–263.19; $p < 0.001$). After adjustment for clinical and laboratory covariates, high SIRI remained independently associated with adverse events (adjusted odds ratio 79.88, 95% confidence interval 14.68–434.64; $p < 0.001$). Analysis of SIRI as a continuous variable yielded a consistent association (adjusted odds ratio 3.03 per unit increase, 95% confidence interval 1.77–5.16; $p < 0.001$).

Conclusions

Admission SIRI independently predicted in-hospital adverse events and showed excellent discrimination in this cohort. As a low-cost index derived from routine blood counts, SIRI may complement early risk stratification in acute heart failure, pending external validation.

Introduction

Acute heart failure (AHF) is a life-threatening clinical syndrome characterized by the rapid onset or worsening of signs and symptoms of heart failure requiring urgent evaluation and frequently unplanned hospitalization. Despite advances in pharmacological and device-based therapy, patients admitted with AHF remain at risk for hemodynamic deterioration, arrhythmia, organ dysfunction, nosocomial infection,

respiratory failure, and death. These complications prolong hospitalization, increase resource use, and are associated with poor subsequent outcomes.¹⁻⁴

The pathophysiology of AHF extends beyond impaired cardiac pump function. Neurohormonal activation, venous congestion, tissue hypoperfusion, endothelial dysfunction, oxidative stress, and systemic inflammation interact to promote myocardial injury and multiorgan dysfunction. Pro-inflammatory cytokines and activation of innate immune cells may further aggravate ventricular dysfunction, vascular injury, renal impairment, and susceptibility to infection.⁵⁻⁷

Inflammatory biomarkers investigated in heart failure include C-reactive protein, interleukin-6, total leukocyte count, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, and systemic immune-inflammation index. The systemic inflammation response index (SIRI) combines absolute neutrophil, monocyte, and lymphocyte counts and may provide a more integrated measure of innate inflammatory activation and adaptive immune suppression. Elevated SIRI has been associated with adverse outcomes across cardiovascular conditions.⁸⁻¹¹

Evidence regarding admission SIRI as a predictor of a broad composite of in-hospital adverse events in AHF remains limited, particularly in Southeast Asian populations. This study aimed to determine the optimal admission SIRI cut-off, evaluate its association with in-hospital adverse events, and assess whether SIRI independently predicts these events after adjustment for relevant clinical and laboratory variables.¹²⁻¹⁶

Methods

Study design and setting

This prospective longitudinal observational study was conducted from January to March 2026 in the Emergency Department, Integrated Cardiovascular Care Unit, High Care Unit, and cardiovascular wards of a tertiary cardiovascular referral hospital in Bali, Indonesia. Consecutive adult patients admitted with AHF were enrolled and followed prospectively during hospitalization. The diagnosis of AHF was established by the attending cardiologist using clinical presentation, physical examination, electrocardiography, laboratory testing, chest imaging, and echocardiography in accordance with contemporary guideline criteria.¹

Participants

Patients were eligible when they were older than 18 years, had de novo or recurrent AHF requiring hospitalization, and provided written informed consent. Exclusion criteria were sepsis or septic shock; cardiogenic shock classified as Society for Cardiovascular Angiography and Interventions stage D or E; hematological disease or malignancy; autoimmune or chronic inflammatory disease;

immunosuppressive or systemic corticosteroid use within the preceding three months; immunosuppression unrelated to medication, including human immunodeficiency virus infection; chronic liver disease; end-stage kidney disease or routine hemodialysis; and refusal to participate.

Sample size and recruitment

The minimum sample size was calculated using an expected in-hospital adverse-event proportion of 33%, a two-sided alpha of 0.05, and an absolute precision of 10%. The calculated minimum was 85 participants; after adding 10% for potential attrition, the target sample was 94. Consecutive sampling continued until the target was exceeded. A total of 102 participants were included in the final analysis.

Data collection and SIRI calculation

Demographic characteristics, clinical findings, comorbidities, admission hemodynamics, laboratory measurements, echocardiographic variables, treatment-related variables, and outcomes were prospectively recorded using a standardized case-report form. A 3-mL peripheral venous blood sample was obtained at admission, before initial intervention and treatment whenever clinically feasible, and analyzed in the hospital clinical pathology laboratory. SIRI was calculated from the first complete blood count using the following formula:

$$\text{SIRI} = (\text{absolute neutrophil count} \times \text{absolute monocyte count}) / \text{absolute lymphocyte count}$$

SIRI was initially analyzed as a continuous variable. Receiver operating characteristic (ROC) analysis and the Youden index were used to identify the optimal cut-off for the composite outcome. Participants were then categorized as having high SIRI (≥ 4.295) or low SIRI (< 4.295).

Outcome assessment

Participants were followed prospectively from admission until discharge, in-hospital death, or 30 days of hospitalization, whichever occurred first; no post-discharge follow-up was performed. The primary outcome was a composite in-hospital adverse event, defined as the occurrence of at least one prespecified event after enrollment: clinically significant arrhythmia, hemodynamic deterioration, new requirement for invasive or non-invasive positive-pressure ventilation, acute kidney dysfunction, liver dysfunction, cerebral dysfunction, hospital-acquired pneumonia, or all-cause in-hospital death.

Acute kidney dysfunction was defined by an increase in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase to at least 1.5 times baseline within seven days, or urine output below 0.5 mL/kg/hour for at least six hours. Liver dysfunction was defined as a new or worsening increase in alanine or aspartate aminotransferase to at least three times the upper reference limit or total bilirubin to at least twice the upper reference limit. Cerebral dysfunction required a new decrease in Glasgow Coma Scale

score of at least two points lasting at least six hours or acute encephalopathy not explained by sedation, severe metabolic disturbance, stroke, or seizure. Hospital-acquired pneumonia was counted only when it developed at least 48 hours after admission and was supported by compatible clinical, radiological, and laboratory findings. Hemodynamic deterioration required new initiation or clinically meaningful escalation of inotropic, vasopressor, or mechanical circulatory support together with evidence of hypoperfusion. Baseline infection, baseline cardiogenic shock, and ventilation already in use at admission were recorded as baseline covariates and were not automatically counted as incident outcome events.

Statistical analysis

Continuous variables were assessed for normality using the Shapiro-Wilk test and are presented as mean $\pm$ standard deviation or median (interquartile range), as appropriate. Categorical variables are presented as number and percentage. Between-group comparisons used the independent-samples t test or Mann-Whitney U test for continuous variables and the chi-square test or Fisher exact test for categorical variables, as appropriate.

ROC analysis estimated the area under the curve (AUC), 95% confidence interval (CI), and optimal cut-off using the Youden index. Variables with $p < 0.25$ in bivariable analysis, together with clinically relevant variables identified a priori, were entered into a multivariable binary logistic regression model using the enter method. Adjusted odds ratios (aORs) with 95% CIs were reported. The primary model used dichotomized SIRS, and a sensitivity analysis repeated multivariable regression with SIRS as a continuous variable. Associations between high SIRS and individual outcome components were assessed using chi-square or Fisher exact testing. All tests were two-sided, and $p < 0.05$ was considered statistically significant. Analyses were performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA).

Ethical considerations

The study protocol was approved by the Health Research Ethics Committee of the Faculty of Medicine, Udayana University/Prof. Dr. I.G.N.G. Ngoerah General Hospital. Written informed consent was obtained from every participant before enrollment. The study was conducted in accordance with the Declaration of Helsinki.

Results

Participant characteristics

All 102 enrolled participants completed in-hospital observation and were included in the final analysis. The mean age was 58.69 ± 12.27 years, and 74 participants (72.5%) were male. Acute coronary

syndrome was the underlying AHF etiology in 57 participants (55.9%), cardiogenic shock was present at admission in 24 (23.5%), infection was present at admission in 30 (29.4%), and 68 (66.7%) were referred from another hospital. Median SIRI in the overall cohort was 3.97 (interquartile range 2.36-6.63). Baseline characteristics are summarized in Table 1.

In-hospital adverse events

During hospitalization, 42 participants (41.2%) experienced at least one adverse event, whereas 60 (58.8%) had no adverse event. Because the primary outcome was composite, a participant could experience more than one component. Hemodynamic deterioration was the most frequent component, followed by clinically significant arrhythmia, acute kidney dysfunction, hospital-acquired pneumonia, and all-cause in-hospital death (Table 2).

Admission SIRI and discriminative performance

Admission SIRI was higher among participants who experienced adverse events than among those without events (median 7.08 [5.28-15.80] vs. 2.52 [1.79-3.74]; $p<0.001$). ROC analysis identified 4.295 as the optimal cut-off. Admission SIRI showed excellent discrimination for the composite outcome, with an AUC of 0.944 (95% CI 0.902-0.986; $p<0.001$) (Figure 1).

Forty-five participants had high SIRI and 57 had low SIRI. Adverse events occurred in 38 of 45 participants (84.4%) with high SIRI and 4 of 57 (7.0%) with low SIRI (Pearson chi-square=62.24; $p<0.001$). High SIRI was associated with an unadjusted OR of 71.93 (95% CI 19.66-263.19) for the composite outcome (Table 3).

Multivariable and additional analyses

Variables with $p<0.25$ in bivariable analysis were body mass index, systolic blood pressure, diastolic blood pressure, serum creatinine, estimated glomerular filtration rate, aspartate aminotransferase, alanine aminotransferase, AHF etiology, cardiogenic shock at admission, referral status, and high SIRI. After simultaneous adjustment for these variables, high SIRI remained the only statistically significant independent predictor of the composite outcome (aOR 79.88, 95% CI 14.68-434.64; $p<0.001$) (Table 4).

When SIRI was modeled as a continuous variable with the same covariate set, each one-unit increase in admission SIRI remained independently associated with the composite outcome ($B=1.107$; aOR 3.03, 95% CI 1.77-5.16; $p<0.001$). This result was directionally consistent with the categorical analysis.

High SIRI was associated with clinically significant arrhythmia, hemodynamic deterioration, acute kidney dysfunction, hospital-acquired pneumonia, positive-pressure ventilation, and all-cause in-hospital death. Odds ratios for positive-pressure ventilation, liver dysfunction, and cerebral dysfunction could not be

estimated reliably because of sparse-event distributions; Fisher exact testing was used for these comparisons (Table 5).

[Insert Figure 1 near here]

Discussion

Principal findings

This prospective cohort study found a strong association between admission SIRI and clinically relevant adverse events during hospitalization for AHF. Participants who developed the composite outcome had substantially higher SIRI values at admission. SIRI demonstrated excellent discrimination, and the ROC-derived threshold of 4.295 identified a group in which more than four-fifths experienced an adverse event. The association persisted after adjustment for multiple clinical and laboratory covariates. Modeling SIRI continuously produced a concordant result, reducing concern that the finding depended solely on dichotomization at a data-derived threshold.

The magnitude of the adjusted association was large and the confidence interval was wide. This pattern reflects strong separation between high- and low-SIRI groups but limited statistical precision in a modest single-center cohort. The point estimate should therefore not be interpreted as a transportable causal effect. Rather, the categorical and continuous analyses jointly support SIRI as a prognostic marker that warrants validation in larger cohorts.

Biological plausibility

Systemic inflammation is increasingly recognized as an active contributor to AHF rather than a passive epiphenomenon. Congestion and hypoperfusion can promote endothelial injury, oxidative stress, cytokine release, and immune-cell recruitment. These pathways can worsen myocardial dysfunction, renal and hepatic impairment, microvascular perfusion, and susceptibility to secondary infection.^{5-7,17-18}

SIRI integrates three complementary leukocyte populations. Activated neutrophils generate reactive oxygen species, proteolytic enzymes, and neutrophil extracellular traps that may amplify endothelial and myocardial injury.²⁰⁻²¹ Monocytes migrate into injured tissue and differentiate into macrophages that regulate cytokine signaling, extracellular-matrix remodeling, and fibrosis.¹⁹ Lymphopenia may reflect neurohormonal stress and loss of adaptive immune regulation.²² A high SIRI therefore represents simultaneous innate immune activation and relative adaptive immune suppression, which may explain its association with a broad spectrum of complications rather than a single organ-specific outcome.

Comparison with previous studies

The present findings are consistent with studies linking elevated SIRI to mortality and adverse cardiovascular outcomes in acute decompensated heart failure, chronic heart failure, ischemic heart failure after percutaneous coronary intervention, coronary artery disease, and acute coronary syndrome.¹²⁻¹⁶ The current study extends this evidence by prospectively evaluating a composite that captures multiple in-hospital complications, including hemodynamic deterioration, arrhythmia, organ dysfunction, hospital-acquired pneumonia, ventilation, and death. These complications are clinically relevant in hospitalized patients with heart failure.^{4,23-27}

The AUC of 0.944 was higher than values commonly reported for single inflammatory markers. Nevertheless, direct claims of superiority over C-reactive protein, interleukin-6, natriuretic peptides, or established AHF risk scores cannot be made because these comparators were not formally evaluated in the present analysis. The data support strong discrimination within this cohort, not incremental value over existing models.

Clinical implications

SIRI is calculated from a routine complete blood count and requires no additional blood sampling, specialized assay, or laboratory infrastructure. Admission SIRI may therefore provide an accessible adjunct to the initial assessment of patients with AHF, particularly where access to advanced biomarkers is limited. A value at or above 4.295 may prompt closer observation, but the threshold should not yet be used as a stand-alone decision rule because it was derived and tested in the same cohort.

SIRI should complement, not replace, comprehensive assessment incorporating history, physical examination, hemodynamics, renal and hepatic function, cardiac biomarkers, electrocardiography, imaging, and established prognostic indicators. External validation and formal evaluation of calibration, reclassification, and incremental predictive value are required before routine clinical implementation.

Strengths and limitations

The study has several strengths. Participants were enrolled consecutively and followed prospectively using prespecified outcome definitions. Blood counts were obtained at admission, preserving the temporal relationship between the biomarker and subsequent events. The outcome captured a clinically meaningful spectrum of in-hospital complications. The consistency between categorical and continuous multivariable analyses strengthens the internal coherence of the findings.

Several limitations should be considered. The single-center design and local case mix may limit generalizability. The sample size was modest, several individual events were uncommon, and the resulting estimates were imprecise. The SIRI cut-off was derived and evaluated in the same dataset, creating a risk of optimism. SIRI was measured only once; serial changes during treatment were not assessed. Residual confounding remains possible despite multivariable adjustment, and no formal

comparison with natriuretic peptides, C-reactive protein, interleukin-6, or established AHF risk scores was performed. The study also did not assess post-discharge outcomes.

Conclusion

Admission SIRI independently predicted in-hospital adverse events in patients hospitalized with AHF and demonstrated excellent discrimination in this prospective cohort. Because SIRI is derived from routine complete blood count parameters, it may serve as an accessible adjunct for early risk stratification. The cut-off and effect estimates require external validation in larger multicenter studies before clinical implementation.

Abbreviations

AHF
acute heart failure
ALT
alanine aminotransferase
AST
aspartate aminotransferase
AUC
area under the curve
CI
confidence interval
eGFR
estimated glomerular filtration rate
HCU
High Care Unit
ICVCU
Integrated Cardiovascular Care Unit
LVEF
left ventricular ejection fraction
NE
not estimable
OR
odds ratio
ROC
receiver operating characteristic
SIRI
systemic inflammation response index
TAPSE
tricuspid annular plane systolic excursion.

Declarations

Ethics approval and consent to participate

The study was approved by the institutional health research ethics committee (institution and approval details withheld for anonymous peer review). Written informed consent was obtained from all participants before enrollment.

Consent for publication

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

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Author Contribution

HSS contributed to conceptualization, methodology, investigation, data curation, formal analysis, interpretation, and writing the original draft. HW and AAADY contributed to conceptualization, methodology, supervision, clinical interpretation, and critical revision of the manuscript. BAPDS and IKSSD contributed to investigation, data curation, clinical interpretation, and critical revision of the manuscript. IGPS contributed to methodology, statistical review and validation, interpretation, and critical revision of the manuscript. All authors read and approved the final manuscript and agree to be accountable for the work.

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Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available because they contain clinical participant data, but de-identified data may be available from the corresponding author on reasonable request and subject to institutional ethics and data-protection requirements.

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Tables

Tables are available in the Supplementary Files section.

Figures

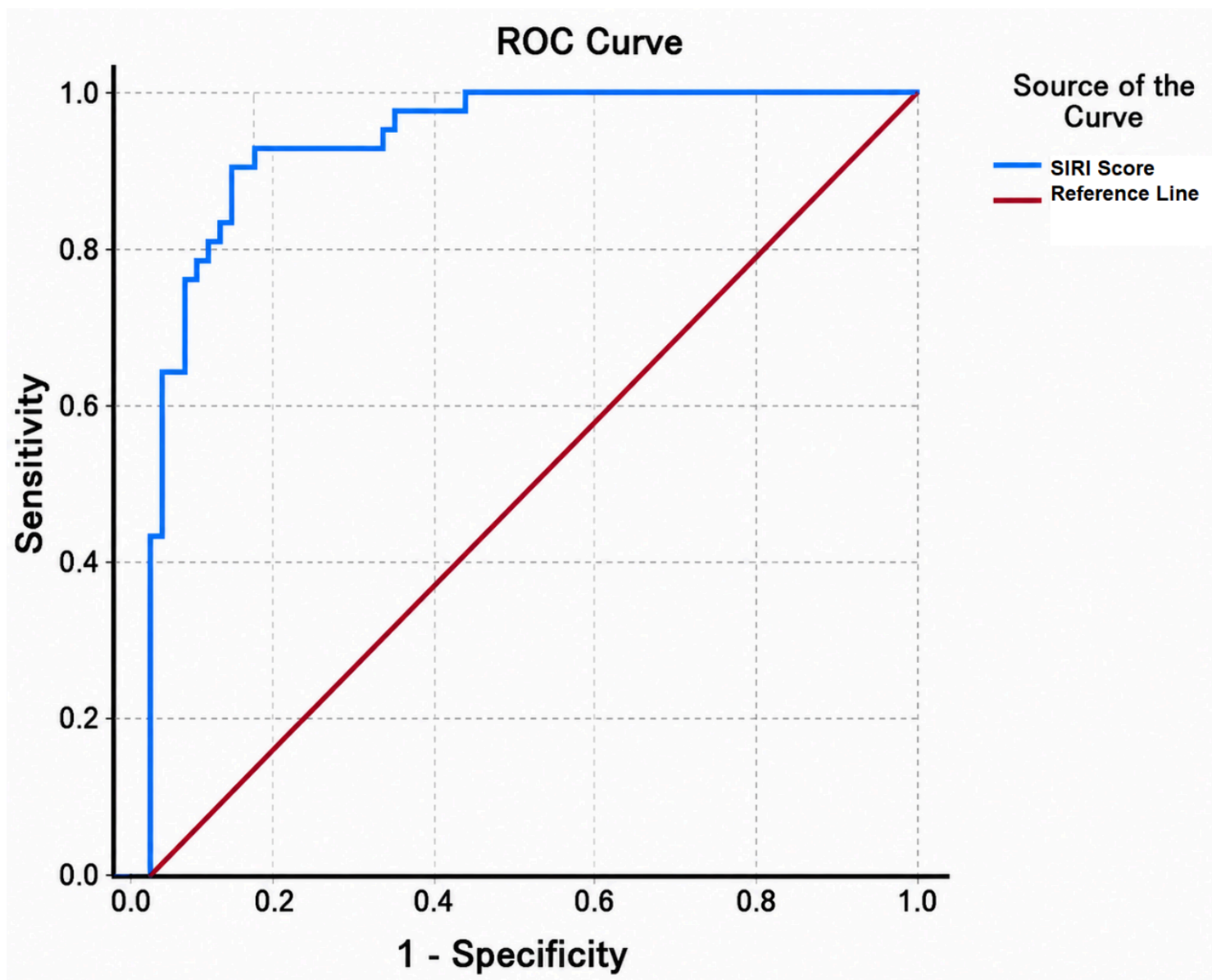


Figure 1

Receiver operating characteristic curve of admission SIRS for predicting the composite in-hospital adverse-event outcome. The diagonal line represents discrimination expected by chance. AUC, area under the curve; ROC, receiver operating characteristic; SIRS, systemic inflammation response index.

Supplementary Files

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