

Systemic Immune-Inflammation Index and Prognosis in Patients with Heart Failure

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Abstract

Background: Inflammation and immune dysregulation play critical roles in the progression and prognosis of heart failure (HF). This study aimed to investigate the prognostic value of the systemic immune-inflammation index (SII) in patients with HF.

Methods: The SII index (platelet count $\times$ neutrophil-to-lymphocyte ratio) was evaluated in two independent HF cohorts: Zigong Fourth People's Hospital (ZIG-HF, N = 1,937) and the Medical Information Mart for Intensive Care HF database (MIMIC-HF, N = 1,177). Associations between SII and clinical outcomes, including all-cause mortality and hospitalization, were assessed using Cox regression analysis. Predictive performance was evaluated using the concordance index (C-index), net reclassification improvement (NRI), and integrated discrimination improvement (IDI) in the ZIG-HF cohort and externally validated in the MIMIC-HF cohort.

Results: In the ZIG-HF cohort, high SII (≥ 700) was significantly associated with increased all-cause mortality at 28 days (HR = 2.78, 95% CI: 1.19-6.46), 3 months (HR = 2.56, 95% CI: 1.15-5.69), and 6 months (HR = 2.26, 95% CI: 1.15-5.69). SII alone showed modest predictive ability for mortality in both cohorts (C-index: 0.654 in ZIG-HF; 0.680 in MIMIC-HF). Adding SII to the least absolute shrinkage and selection operator model significantly improved prediction in the ZIG-HF (C-index: 0.738, NRI: 0.285), with external validation in the MIMIC-HF cohort (C-index: 0.732, NRI: 0.087, IDI: 0.087).

Conclusions: In patients with HF, elevated SII levels are independently associated with increased mortality. Incorporating this index into predictive models improves risk stratification for death.

Introduction

Heart failure (HF) is a syndrome characterized by impaired cardiac pumping function and inadequate cardiac output to meet the body's basic metabolic needs, with high incidence and mortality rates [1, 2]. Currently, more than 64 million individuals worldwide are living with HF [3]. It is projected that the total medical expenses for HF patients will increase by \$32.2 billion between 2012 and 2030 [4]. There is an urgent need to develop prognostic biomarkers that can enable clinical doctors to identify high-risk patients promptly. Immune-inflammatory dysregulation, marked by sustained cytokine overproduction (e.g., TNF- α , IL-6), aberrant leukocyte activation, and unresolved systemic inflammation, is increasingly recognized as a central driver of chronic disease progression across multiple organ systems [5]. Currently, research has provided evidence linking chronic inflammation and abnormal immune activation to the onset and progression of HF [6].

Studies have substantiated the significant roles of lymphocytes, neutrophils, and monocytes/macrophages in myocardial remodeling and the development of HF [7]. Certain cytokines, including tumor necrosis factor (TNF)- α , Interleukin (IL)-1, and other factors, are linked to diminished cardiac function, inducing cardiomyocyte hypertrophy and fibrosis, thereby indirectly fostering the progression of HF. Emerging evidence suggests that inflammatory factors can function as biomarkers to predict poor prognosis in HF, offering novel insights into its treatment [8]. The systemic immune-inflammation (SII) index, calculated by multiplying the platelet count by the neutrophil count and dividing it by the lymphocyte count, serves as a novel inflammatory marker [9] and has shown promise as a valuable tool for predicting survival outcomes in patients with diverse cardiovascular diseases, such as coronary heart disease [10] and aortic valve stenosis [11]. However, evidence regarding the association between the SII index and prognosis in patients with HF remains limited. Accordingly, this study aimed to evaluate the prognostic value of SII and its incremental predictive utility for mortality in HF populations.

Materials and methods

Data sources

The study involved two independent HF datasets. The Zigong-HF study included data from electronic medical records of HF patients at Zigong Fourth People's Hospital, spanning from December 2016 to June 2019 [12] (hereafter referred to as the ZIG-HF cohort). The MIMIC-HF cohort was derived from a retrospective analysis of the MIMIC-HF database in Dryad Dataset (<https://doi.org/10.5061/dryad.0p2ngf1zd>), including 1,177 HF patients hospitalized in the intensive care units (ICU) extracted from the MIMIC-HF database (V.1.4, 2016) which is a publicly available critical-care database containing de-identified data on 46,520 patients and 58,976 admissions to the ICU of the Beth Israel Deaconess Medical Center, Boston, USA. The details of the study characteristics of the MIMIC-HF cohort can be found in the previous articles by Li et al. [13].

Study design, population, and outcomes

All patients included in the ZIG-HF cohort met the diagnostic criteria for HF according to the guidelines of the European Society of Cardiology 2016 [14]. All reported data were collected at the time of initial hospitalization, with follow-up commencing immediately after discharge. Data were extracted from electronic records, reviewed by specialists for inconsistencies, and followed up at 28 days, 3 months, and 6 months via in-person visits or telephone calls. In the present study, patients with missing exposure values (platelet, neutrophil, and lymphocyte), and patients with acquired immune deficiency syndrome (AIDS), leukemia, malignant lymphoma, and solid tumors were excluded. Ultimately, 1,937 patients out of 2,008 patients were included for analysis, as depicted in **Figure 1**. The clinical endpoints in ZIG-HF included 28-day mortality, 3-month mortality,

and 6-month mortality [12]. The outcomes were obtained from medical records or through mandatory follow-up visits at 28 days, 3 months, and 6 months via telephone calls.

The MIMIC-HF dataset was obtained from a public database of Dryad from Li et al. [13], which included 1,177 patients with a diagnosis of HF, identified ICD-9 codes at the time of ICU admission, with available left ventricular ejection fraction (LVEF) or brain natriuretic peptide (BNP). The outcome of the MIMIC-HF cohort was in-hospital mortality, defined as the vital status at the time of hospital discharge in survivors and non-survivors.

Patients and/or the public were not directly involved in this study. All the data presented in this study were open-accessed from online databases, which were approved by the review boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center, and the People's Hospital of Zigong.

Exposure and co-variables

The baseline SII index was calculated as the total peripheral platelet count (p) multiplied by the neutrophil-to-lymphocyte ratio (N/L) ($SII = p \times N/L$ ratio). Co-variables such as age, gender, pulse, blood pressure, body mass index (BMI), New York Heart Association (NYHA) classification, and laboratory values including hemoglobin, potassium, chloride, BNP, platelet count, neutrophil count, lymphocyte count, estimated glomerular filtration rate (eGFR), albumin, low-density lipoprotein cholesterol (LDL-C), left ventricular ejection fraction (LVEF), high sensitivity troponin (Hs-cTNT), as well as a record of co-morbidities including peripheral vascular disease, cerebrovascular disease, moderate to severe chronic kidney disease, diabetes, connective tissue diseases, chronic obstructive pulmonary disease, congestive heart failure, and the use of medications such as aspirin, atorvastatin, β -blockers, angiotensin-converting enzyme inhibitors (ACEI)/angiotensin receptor blockers (ARBs), diuretics and mineralocorticoid receptor antagonists (MRA) were extracted for analysis.

Statistical analysis

The SII index was categorized based on the median value of 700. Cox hazard regression expressed as hazard ratios (HRs) and their 95% confidence intervals (CIs) was used to analyze the association between SII index and outcomes in the ZIG-HF cohort, either category variable and continuous analysis (Z-scores). The Cox proportional hazards assumption was tested through Schoenfeld residual testing. Adjustments for the Cox models were selected based on clinical experience and established prognostic relevance (e.g., age, comorbidities). Two nested models were constructed. The age, sex, and BMI were adjusted in the first model, the final model included age, sex, pulse rate, potassium levels, hemoglobin, estimated glomerular filtration rate, presence of moderate to severe chronic kidney disease, diabetes, chronic obstructive pulmonary disease, cerebrovascular disease, peripheral vascular disease, diastolic and systolic blood pressure, low-density lipoprotein cholesterol, use of β -blockers, ACEI/ARB, diuretics, and BNP. Moreover, to adjust for the potential bias of confounding, an inverse probability of treatment weighting (IPTW) analysis including all covariates of all baseline characteristics. After IPTW adjustment, all standardized differences were < 0.1 for all baseline characteristics among high and low SII index. (data not shown)

Then the predictive ability of the SII index was tested in ZIG-HF and validated in the MIMIC-HF cohort, expressed as C-index. Finally, the C-index, continuous net reclassification improvement (NRI) index, and integrated discrimination improvement (IDI) index were used to assess whether the addition of the SII index to a baseline model would enhance the predictive performance for mortality. CIs of C-index, NRI scores, and IDI scores were computed by bootstrap resampling. The baseline predictive model was established using the Least Absolute Shrinkage and Selection Operator (LASSO) method, a data-driven approach that enhances predictive accuracy by penalizing model complexity, thereby mitigating overfitting risks. Specifically, baseline models included two predictor sets: 3 risk factors (eGFR, serum K⁺, BNP) and 6 risk factors (eGFR, serum K⁺, BNP, SBP, sex, heart rate). The comparison of the C-index after SII addition was computed by the methods of DeLong et al. [15]. Statistical analyses were conducted using R software (Version 4.2.2, R Foundation for Statistical Computing). A p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

A total of 1,937 patients with HF in the ZIG-HF were included in the association analysis (**Figure 1**). The baseline characteristics of these patients, stratified by median SII values (700), are presented in **Table 1**. The baseline characteristics indicated that patients with a high SII index ($SII \geq 700$) tended to be older, had a higher class of NYHA, and exhibited elevated levels of BNP, had a higher proportion of moderate to severe chronic kidney disease, lower usage rates of diuretics, β -blockers, MRA, and ACE inhibitors or ARBs ($P < 0.05$).

Association between SII index and outcomes

During the 6-month follow-up period, the incidence rates of all-cause mortality were as follows: 1.86% ($N = 36$) for 28-day mortality, 2.01% ($N = 39$) for mortality at 3 months, and 2.79% ($N = 54$) for mortality at 6 months (**Table 2**). The survival curve showed that patients with high SII index values experienced more deaths (log-rank $P = 0.001$) than those with lower SII index values (**Figure 2A**). The cumulative incidence of hospitalization crossed at approximately 30 days: the high-SII group showed a higher incidence in the early period, while the low-SII group exhibited a higher

incidence thereafter (log-rank $P = 0.026$) (**Figure 2B**). In unadjusted analysis, high SII was associated with a lower risk of hospitalization at 3 months (HR = 0.879, 95% CI: 0.736-1.051, $P = 0.016$) (**Supplemental Table S1**). However, this association did not remain significant after multivariable adjustment, suggesting that the observed difference was largely driven by baseline confounders. The higher mortality in the high-SII group (**Table 2**) may also represent a competing risk that limits the occurrence of subsequent hospitalizations.

Unadjusted analysis showed that patients with high SII (SII ≥ 700) were associated with an increased risk of 28-day mortality (HR = 4.20, 95% CI: 1.84 - 9.59), 3-month mortality (HR = 3.80, 95% CI: 1.74 - 8.30), and 6-month mortality (HR = 3.81, 95% CI: 1.75 - 8.31), all of their P -value < 0.01 . With adjustments for age, sex, pulse rate, potassium, hemoglobin, estimated glomerular filtration rate, moderate to severe chronic kidney disease, diabetes, chronic obstructed pulmonary disease, cerebrovascular disease, peripheral vascular disease, diastolic blood pressure, systolic blood pressure, low density lipoprotein cholesterol, β -blockers, ACEI/ARB, diuretics, and BNP, high SII index were associated with 28-days mortality (HR = 2.78, 95% CI: 1.19 - 6.46), 3-months (HR = 2.56, 95% CI: 1.15 - 5.69) and 6-months mortality (HR = 2.26, 95% CI: 1.15 - 5.69), all of their P -value < 0.05 (**Table 2**).

Additional predictive ability of the SII index in predicting mortality

We assessed the predictive ability for mortality of the SII index in two ZIG-HF and MIMIC-HF cohorts. SII index showed a low predictive ability for 6-month mortality in ZIG-HF (C-index: 0.654, 95% CI: 0.571 - 0.736) and MIMIC-HF cohort (C-index: 0.680, 95% CI: 0.632 - 0.725). A traditional predictive model was developed with model1 (eGFR, serum K+, BNP) and model2 (eGFR, serum K+, BNP, SBP, sex, heart rate) by the LASSO method (**Figure 3A&B**). Addition of the SII index to the LASSO models significantly improved the C-index for 6-month mortality in the ZIG-HF cohort (C-index increased from 0.708 to 0.736, $P = 0.031$ for model with 3 risk factors; from 0.711 to 0.738, $P = 0.023$ for model with 6 risk factors), along with significant improvements in continuous NRI (both $P < 0.05$), although the IDI did not reach statistical significance (**Figure 3C&D and Table 3**). These improvements in predictive ability were validated by C-index ($P < 0.001$), NRI ($P < 0.02$), and IDI ($P < 0.02$) in the MIMIC-HF cohort (**Table 4 and Figure 4**).

Discussion

Major findings

This HF cohort study showed that the SII index was independently associated with death in patients with HF. Although the SII index alone showed modest discriminative ability for mortality, its addition to conventional prognostic factors significantly improved the predictive performance in two independent HF cohorts. These results showed that the SII index may be a new predictor for mortality in patients with HF.

A variety of pro-inflammatory cytokines and inflammatory mediators have been identified as factors for the incidence and prognosis of HF [16]. These cytokines can affect various aspects of heart function, including heart contractility, hypertrophy, apoptosis, and fibrosis. This leads to ongoing changes in the structure and function of the heart, known as myocardial remodeling [17,18]. The SII index, calculated from platelet counts and the neutrophil-to-lymphocyte ratio, serves as a clinical marker of a patient's inflammatory and immune status in various diseases [19]. The link between elevated SII index and various adverse clinical outcomes has been established. For instance, the interplay between systemic inflammation and local immune response is recognized as the seventh hallmark of cancer, playing a significant role in the initiation, progression, and metastasis of various cancer types [20, 21]. Meanwhile, the SII index provided additional predictive ability of major cardiovascular events than traditional risk factors in coronary artery disease following coronary intervention [10]. However, the studies on the SII index and HF are relatively sparse. Our study revealed that higher SII index values upon admission were independently associated with mortality in patients with HF, as determined by NRI and IDI in two independent cohorts. In the ZIG-HF cohort, the significant NRI indicated that the SII index improved risk reclassification, particularly in identifying high-risk subgroups, whereas the non-significant IDI suggested limited improvement in overall risk discrimination across the entire population (**Table 3**). Notably, both NRI and IDI were significant in the external validation cohort (**Table 4**), supporting the incremental prognostic value of SII when combined with conventional risk factors. Our findings may indicate that systemic inflammation, as measured by SII, predominantly modifies risk trajectories in patients with HF rather than uniformly shifting risk probabilities for all patients. Therefore, we suggest that the SII may be a new predictor of prognosis in patients with HF.

Since infections are a common cause of HF hospitalizations and have an impact on the SII index, this study did not exclude participants who had acute infections. Nevertheless, it is questionable whether the SII index responds to the status of the infection or to the baseline inflammatory immune level of the patient. Combined with the fact that there was also a difference in the number of leukocytes at our baseline level (although they were in the normal range). However, the relationship between the SII index and prognosis cannot be analyzed by infection stratification due to the lack of pathogenetic indicators, such as CRP, blood cultures, chest CT scans, and other indicators of lung infection. This needs to be discussed in light of the currently available literature.

The HR for mortality gradually decreased from 28 days to 6 months, suggesting that the prognostic strength of baseline SII may attenuate over time. This trend is consistent with the interpretation of SII as an acute-phase inflammatory marker, whose predictive utility might be more pronounced for short-term outcomes. Nevertheless, the association remained statistically significant at 6 months, and the addition of SII significantly improved the predictive performance of conventional models for 6-month mortality. These findings indicate that SII retains meaningful

prognostic value beyond the acute phase. Whether its predictive utility extends beyond 6 months warrants further investigation in future prospective studies. Notably, the divergent trends between all-cause mortality and hospitalization events (**Figure 2**) may reflect distinct underlying mechanisms. While our study assessed all-cause hospitalizations as secondary outcomes, emerging evidence suggests heart failure-specific endpoints (e.g., HF-related rehospitalization or worsening HF episodes) may better reflect disease progression and therapeutic response [22, 23]. The stronger association of SII with mortality could be attributed to its role in capturing systemic inflammatory burden leading to irreversible organ damage (e.g., myocardial fibrosis or renal dysfunction) [24], whereas the weaker correlation with hospitalization events might be amplified by the heterogeneity of non-HF admissions in our composite endpoint.

Potential mechanism

The potential mechanism of the SII index may be an indicator of inflammation. Significant increases in circulating pro-inflammatory cytokines are commonly observed in patients with HF [25, 26]. Moreover, various studies using experimental animal models of HF, induced by diverse pathophysiological disturbances, have shown that inhibiting pro-inflammatory cytokine signaling can yield beneficial effects [27-29]. Extensive clinical and experimental research data suggest that inflammatory cytokines and chemokines are crucial in the development of myocardial dysfunction and adverse cardiac remodeling [30]. The potential mechanism may involve fibrogenic response, proliferation of activated fibroblasts, and deposition of extracellular matrix activated by pro-inflammatory cytokines.

The differential predictive power of SII for mortality versus hospitalization may stem from their distinct pathophysiological drivers. For HF-specific hospitalizations driven by volume overload or hemodynamic compromise, inflammatory activation might play a secondary role compared to neurohormonal mechanisms [31]. Elevated SII reflects sustained systemic inflammation that directly contributes to terminal organ damage through multiple pathways: neutrophil extracellular traps (NETs) promote myocardial apoptosis [32], platelet activation accelerates microvascular thrombosis [33], and lymphopenia indicates impaired adaptive immunity against persistent inflammatory stimuli [34, 35]. These processes culminate in irreversible outcomes like sudden cardiac death or multiorgan failure, which dominate mortality endpoints [36, 37]. In contrast, hospitalization events are frequently driven by acute triggers (e.g., pneumonia, arrhythmias) where transient SII elevations may be masked by therapeutic interventions or lack temporal alignment with biomarker measurement timing [38, 39]. This dichotomy aligns with prior observations that inflammatory markers like CRP show stronger mortality prediction than readmission risk in heart failure populations [40, 41].

Comparisons with previous studies

A retrospective cohort study involving 4606 subjects revealed that elevated SII effectively predicted heightened mortality rates at 30 days, 90 days, and during hospitalization, along with an increased risk of major adverse cardiovascular events in critically ill patients with HF [42]. Zheng et al. demonstrated a significant positive correlation between SII levels and the prevalence of HF [43]. Additionally, a cross-sectional study indicated a positive association between SII levels and HF prevalence, revealing a non-linear relationship between SII and HF incidence [44]. Notably, there is a scarcity of studies on the association between the SII index and HF, particularly in China. Our study encompassed two independent cohorts of HF patients, revealing an independent association between the SII index and mortality post-adjustment. Furthermore, the inclusion of the SII index in our baseline prognostic model enhanced the predictive accuracy of mortality in HF patients.

Limitations

Nevertheless, it is important to recognize certain limitations. Firstly, due to the nature of retrospective design, a causal relationship between the SII and HF cannot be established. Second, the number of studies investigating the SII in relation to HF is limited. Consequently, there is a need for large-scale clinical studies in the future to better understand this relationship. Third, given that the SII fluctuates throughout a patient's hospitalization, utilizing the average SII during admission may be a reasonable approach. Fourthly, database limitations precluded the inclusion of critical variables such as the use of other statins, C-reactive protein (CRP) levels, prior HF hospitalization history, and detailed hospitalization records (e.g., admission reasons and cause of mortality), which may confound outcome interpretation. Fifth, the relatively low 6-month mortality in our cohort (2.79%) compared to national Chinese HF data (30-day: 2.4%; 1-year: 13.7%) [45] likely reflects selection bias and cultural factors affecting mortality documentation. This discrepancy underscores the need for caution when extrapolating our findings to broader HF populations. Future studies should focus on: (1) implementing more robust mortality tracking mechanisms, particularly for terminally ill patients who return home; (2) incorporating multicenter data to improve population representativeness; (3) adopting prospective designs to systematically document end-of-life care decisions. Sixth, the use of all-cause hospitalization as an endpoint rather than HF-specific events may have diluted the observed associations, as non-cardiac admissions (e.g., infections) with distinct inflammatory profiles were included [46]. Seventh, our findings are derived solely from hospitalized HF patients and may not extrapolate to outpatient populations, warranting future studies to address this gap. Notably, the limited events relative to adjusted variables (particularly in Model III's 18 covariates) and baseline imbalances in key factors (e.g., age) may risk overfitting, despite mitigation via inverse probability treatment weighting and multi-model validation. Finally, insufficient phenotypic data precluded assessment of SII effects across HF subtypes.

Conclusion

In this study involving two independent cohorts of patients with heart failure, the SII index was independently associated with all-cause mortality, with consistent findings across short-term (28 days) and intermediate-term (3 months and 6 months) follow-up. Although SII alone exhibited modest discriminative ability for mortality, its addition to conventional risk models significantly improved risk reclassification and overall predictive performance, as demonstrated by significant improvements in C-index and NRI, with external validation in the MIMIC-HF cohort supporting the robustness of these findings. Notably, the association between SII and all-cause hospitalization was less consistent and largely attenuated after multivariable adjustment, likely reflecting competing risk from the substantially higher mortality in the high-SII group and the heterogeneity of hospitalization events. These results suggest that SII, as an easily obtainable and cost-effective inflammatory marker, may serve as a complementary tool for identifying heart failure patients at elevated risk of death, particularly when integrated into existing risk stratification models. However, given the retrospective design and the lack of detailed infection-related and HF-specific outcome data, the causal relationship and the optimal time window for SII assessment remain to be clarified. Future prospective studies with longer follow-up periods, standardized serial SII measurements, and HF-specific endpoints are warranted to further validate the clinical utility of SII in guiding individualized management of HF.

Non-standard Abbreviations and Acronyms

Abbreviations	Full name
ACEI	Angiotensin-converting enzyme inhibitors
AIDS	Acquired Immune Deficiency Syndrome
ARBs	Angiotensin receptor blockers
CIs	confidence intervals
eGFR	estimated glomerular filtration rate
HF	heart failure
HRs	hazard ratios
ICU	intensive care units
IDI index	Integrated Discrimination Improvement Index
IL-1	Interleukin-1
IPTW	inverse probability of treatment weighting
LDL-C	low-density lipoprotein cholesterol
LVEF	left ventricular ejection fraction
NRI index	net reclassification improvement index
BNP	Brain natriuretic peptide
NYHA	New York Heart Association
SII	systemic immune-inflammation
TNF-α	tumor necrosis factor-α

Declarations

Ethics approval and informed consent

Patients and/or the public were not directly involved in this study. All the data presented in this study were open-accessed from online databases, which was approved by the review boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center and the People’s Hospital of Zigong.

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

Data availability

The MIMIC-HF dataset is publicly available via Dryad (<https://doi.org/10.5061/dryad.0p2ngf1zd>). The ZIG-HF cohort data have been deposited in Figshare (Machine-accessible metadata file describing the reported data: 10.6084/m9.figshare.13064054) and are fully described in the following publication: Zhang Z, Cao L, Chen R, et al. Electronic healthcare records and external outcome data for hospitalized patients with heart failure. *Scientific Data* 2021;8(1):46. Please note that the ZIG-HF data can also be accessed via PhysioNet (<https://physionet.org/content/heart-failure-zigong/1.3/>). The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Competing interests

All authors declare that they have no conflicts of interest.

Authors' contributors

Wenting Wang and Xiao Liu were responsible for the entire project and revised the draft. Xiao Liu, Menglu Liu, and Jiang Cheng conducted the research selection, data extraction, statistical analysis, and data interpretation. Jing Zhang, Chenming Guo, Yujie Zhao, and Ayiguli Abudukeremu drafted the first edition of the manuscript. Peng Yu, Jie Chen, Wengen Zhu, and Zenghui Zhang plotted the graphs. All authors participated in the interpretation of the results and prepared the final version of the manuscript.

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Tables

Table 1. Baseline characteristics of the study population by systemic immune-inflammation index

	SII <700 (N=968)	SII ≥700 (N=969)	p -value
SII index	410.45 (278.71-546.66)	1295.00 (909.58-2126.92)	<0.001
Length of hospital stays, days	10 (9)	10 (8)	0.266
Age, years			0.005
<70	288 (29.75%)	233 (24.05%)	
≥70	680 (70.25%)	736 (75.95%)	
Female	415 (42.87%)	393 (40.56%)	0.302
Pulse rate, bpm	82.48 (21.10)	87.73 (21.60)	<0.001
SBP, mmHg	129.30 (23.40)	132.88 (25.89)	0.002
DBP, mmHg	76.13 (14.01)	76.81 (14.97)	0.625
BMI, kg/m2	22.01 (13.45)	21.60 (14.29)	0.004
NYHA class			0.002
II	175 (18.08%)	157 (16.20%)	
III	527 (54.44%)	475 (49.02%)	
IV	266 (27.48%)	337 (34.78%)	
Laboratory values			
Hemoglobin, g/L	117.09 (24.02)	113.23 (24.91)	<0.001
Potassium, mmol/L	3.95 (0.63)	4.01 (0.77)	0.468
Chloride, mmol/L	103.65 (99.90-106.63)	101.30 (97.60-104.90)	<0.001
BNP, ng/ml	731.08 (293.93-1589.19)	791.86 (324.67-1896.77)	0.027
WBC, 10 ⁹ /L	5.92 (2.17)	8.66 (3.92)	<0.001
Platelet, 10 ⁹ /L	118.78 (44.66)	172.01 (70.72)	<0.001
Neutrophil, 10 ⁹ /L	4.06 (1.64)	7.20 (3.63)	<0.001
Lymphocyte, 10 ⁹ /L	1.27 (0.65)	0.80 (0.44)	<0.001
eGFR, ml/min*1.73m2	72.36 (36.42)	65.04 (36.52)	<0.001
Albumin	37.03 (4.71)	36.14 (5.11)	<0.001
LDL-C	1.81 (0.75)	1.90 (0.75)	0.011
Hs-cTNT	0.04 (0.02-0.09)	0.07 (0.03-0.15)	<0.001
Echocardiographic			
LVEF	51 (40-62)	51 (42-60)	0.984
LVEDD	53.63 (11.11)	52.68 (10.80)	0.123
E/A	0.98 (0.64-1.59)	1.06 (0.68-1.65)	0.593
Co-morbidities			
Peripheral vascular disease	48 (4.96%)	46 (4.75%)	0.829
Cerebrovascular disease	80 (8.26%)	65 (6.71%)	0.193
Diabetes	211 (21.80%)	240 (24.77%)	0.122
COPD	112 (11.60%)	113 (11.7%)	0.944
Congestive Heart Failure	895 (92.4%)	908 (93.8%)	0.244
Moderate to severe chronic kidney disease	198 (20.48%)	255 (26.34%)	0.002
Medications			

Aspirin	455 (47.00%)	478 (49.38%)	0.295
Atorvastatin	380 (39.26%)	416 (42.98%)	0.096
β -blockers	288 (29.75%)	220 (22.73%)	<0.001
ACEI/ARB	405 (41.84%)	336 (34.71%)	0.001
Diuretics	944 (97.52%)	912 (94.21%)	<0.001
MRA	905 (93.40%)	868 (89.76%)	0.004

Note: Data are expressed as mean (SD), median (interquartile range) and numbers (percentage) as appropriate, and are derived from admission.

SBP = systolic blood pressure; DBP= Diastolic blood pressure; BMI = body mass index; DM = diabetes mellitus; NYHA = New York Heart Association; BNP = brain natriuretic peptide; eGFR = estimated Glomerular Filtration Rate; LDL-C = low density lipoprotein cholesterol; Hs-cTNT = high sensitivity troponin; LVEF = left ventricular ejection fraction; LVEDD = left ventricular end diastolic diameter; E/A = early to late diastolic mitral inflow velocity ratio; COPD = chronic obstructive pulmonary disease; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin II receptor blockers; MRA = mineralocorticoid receptor antagonists.

Table 2. Association of the systemic immune-inflammation index (SII index) with all-cause mortality in patients with heart failure

Outcomes	Case/N	Model I HR (95%CI) <i>p</i>	Model II HR (95%CI) <i>p</i>	Model III HR (95%CI) <i>p</i>	IPTW matched
28 days mortality	36(1.86%)				
SII index <700	7 (0.72%)	Reference			
SII index $\geq$700	29(2.99%)	4.20(1.84-9.59) 0.0006	4.22(1.84-9.63) 0.0006	2.78(1.19-6.46) 0.0018	3.19(1.18-8.59) 0.022
Per 1 SD		1.43(1.26-1.62) <0.0001	1.46(1.28-1.66) <0.0001	1.40(1.20-1.63) <0.0001	
3 months mortality	39 (2.01%)				
SII index <700	8 (0.83%)	Reference			
SII index $\geq$700	31 (3.20%)	3.80(1.74-8.30) 0.0007	3.83(1.76-8.38) 0.0007	2.56(1.15-5.69) 0.021	2.94(1.18-7.37) 0.020
Per 1 SD		1.41(1.25-1.61) <0.0001	1.44(1.27-1.64) <0.0001	1.40(1.20-1.63) <0.0001	
6 months mortality	54(2.79%)				
SII index <700	15(1.55%)	Reference			
SII index $\geq$700	39(4.03%)	3.81(1.75-8.31) 0.0008	3.83(1.75-8.37) 0.0007	2.26(1.15-5.69) 0.0208	1.97(1.02-3.78) 0.042
Per 1 SD		1.44(1.27-1.64) 0.0001	1.42(1.25-1.61) <0.0001	1.40(1.20-1.63) <0.0001	

Note: Model I was an unadjusted model.

Model II was adjusted for age, sex, and BMI.

Model III was adjusted for age, sex, pulse rate, potassium, hemoglobin, estimated glomerular filtration rate, moderate to severe chronic kidney disease, diabetes, chronic obstructive pulmonary disease, cerebrovascular disease, peripheral vascular disease, diastolic blood pressure, systolic blood pressure, low Density lipoprotein cholesterol, β -blockers, ACEI/ARB, diuretics, and BNP.

Table 3. C-index, NRI, and IDI for evaluating improvement to predict 6-month mortality after adding SII to the baseline model in patients with heart failure, testing cohort (ZIG-HF cohort)

	C-index (95%CI)	P value	Sensitivity	Specificity	Accuracy	Positive- LR	Negative- LR	NRI (%) (95%CI)	P value	IDI (%) (95%CI)	P value
SII index	0.654 (0.571- 0.736)	-	0.519	0.773	0.7666	2.2919	0.6223	-	-	-	-
LASSO model (3 risk factors)	0.708 (0.631- 0.785)	Ref	0.629	0.716	0.5527	1.7904	0.3399	Ref	Ref	Ref	Ref
SII index + LASSO model (3 risk factors)	0.736 (0.662- 0.802)	0.031	0.611	0.766	0.7922	2.9369	0.5104	0.280 (0.260- 0.3970)	0.033	0.001 (-0.006- 0.011)	0.571
LASSO model (6 risk factors)	0.711 (0.636- 0.782)	Ref	0.695	0.629	0.5172	1.6572	0.3643	Ref	Ref	Ref	Ref
SII index + LASSO model (6 risk factors)	0.738 (0.676- 0.802)	0.023	0.779	0.609	0.6722	2.1408	0.4414	0.285(0.001- 0.016)	0.040	-0.001 (-0.016- 0.013)	0.990

Note: 3 risk factors: eGFR, potassium, BNP, 6 risk factors: eGFR, potassium, BNP, heart rate, SBP, gender.

CI = confidence interval; NRI = net reclassification improvement; IDI = the integrated discrimination improvement.

Table 4. C-index, NRI and IDI for evaluating improvement to predict 6-months mortality after adding SII to baseline model in patients with heart failure, validated cohort (MIMIC-HF cohort)

	C-index (95% CI)	P value	Sensitivity	Specificity	Accuracy	Positive- LR	Negative- LR	NRI (%) (95%CI)	P value	IDI (%) (95%CI)	P value
SII index	0.680 (0.632- 0.725)	-	0.556	0.757	0.729	2.288	0.586	-	-	-	-
LASSO model (3 risk factor)	0.619 (0.569- 0.668)	Ref	0.409	0.783	0.731	1.883	0.755	Ref	Ref	Ref	Ref
LASSO model (3 risk factors) + SII index	0.703 (0.659- 0.750)	<0.001	0.521	0.809	0.769	2.734	0.592	0.139 (0.052- 0.226)	0.002	0.139 (0.052- 0.226)	0.002
LASSO model (6 risk factors)	0.682 (0.634- 0.733)	Ref	0.497	0.776	0.736	2.211	0.649	Ref	Ref	Ref	Ref
LASSO model (6 risk factors) + SII index	0.732 (0.689- 0.769)	<0.001	0.653	0.706	0.699	2.220	0.492	0.087 (0.017- 0.156)	0.015	0.087 (0.017- 0.157)	0.015

Note: 3 risk factors: eGFR, potassium, BNP, 6 risk factor: eGFR, potassium, BNP, heart rate, SBP, gender.
CI = confidence interval; NRI = net reclassification improvement; IDI = the integrated discrimination improvement.

Figures

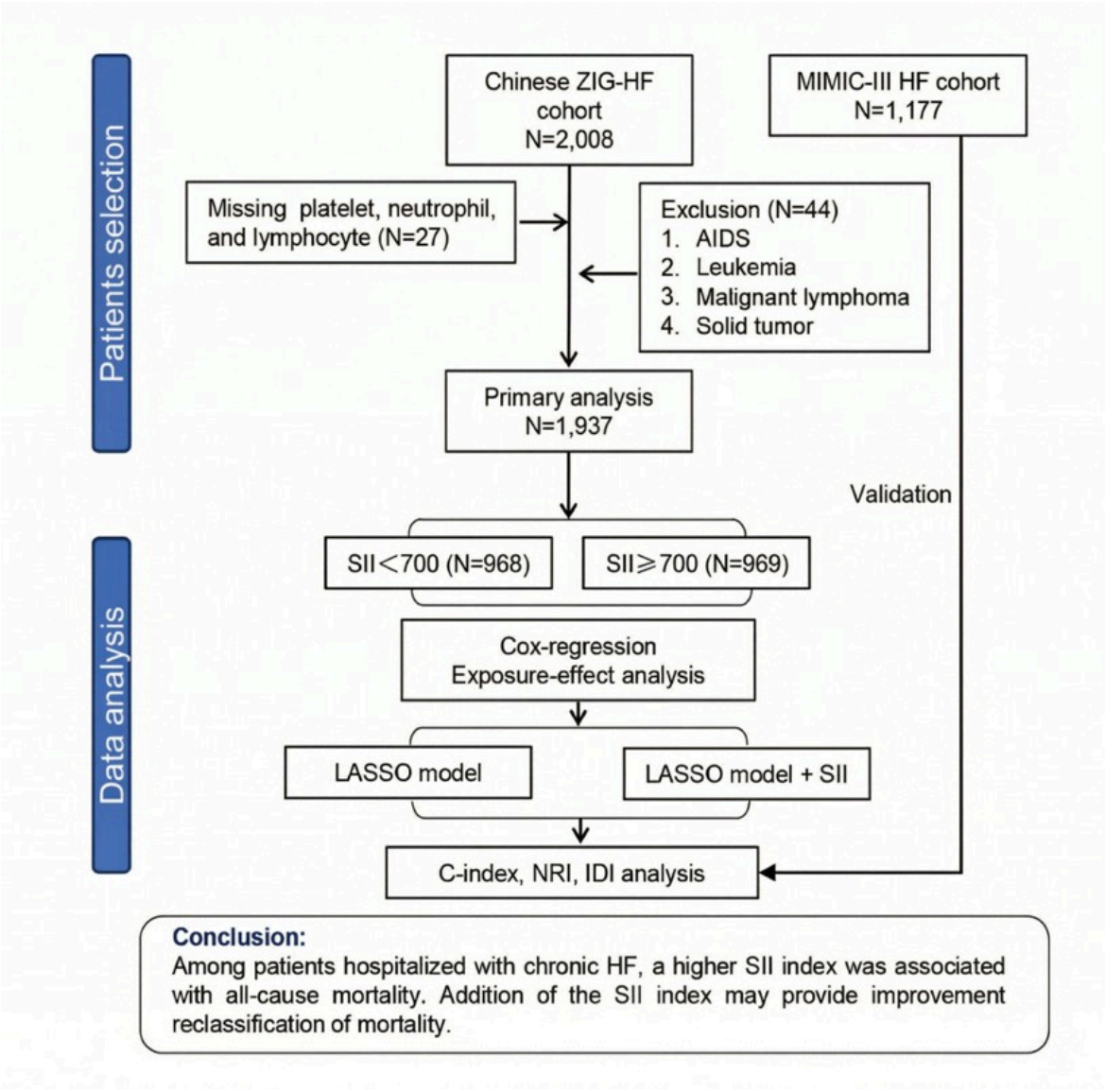


Figure 1

Workflow and major finding of this study.

Note: HF = heart failure; AIDS = acquired immune deficiency syndrome; SII = systemic immune-inflammation index; ZIG-HF = HF patients at Zigong Fourth People's Hospital; MIMIC = medical information mart for intensive care; LASSO = least absolute shrinkage and selection operator; NRI = net reclassification improvement; IDI = integrated discrimination improvement.

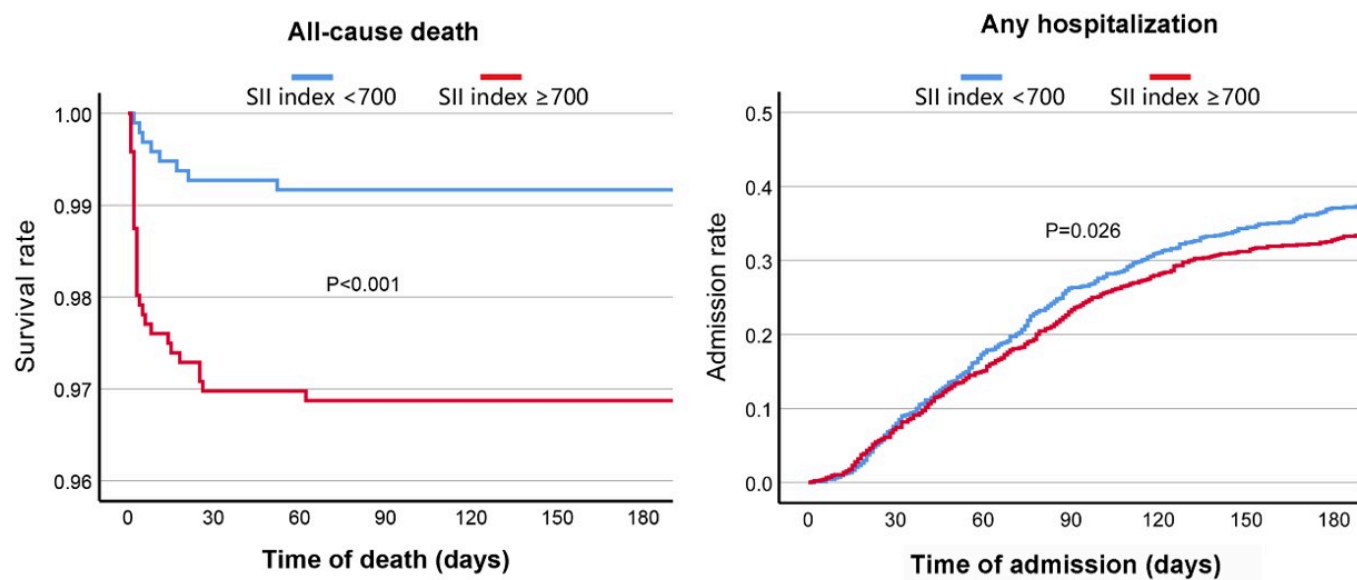


Figure 2

Kaplan-Meier survival curve for chronic heart failure across the dichotomization of the SII.

Note: SII = systemic immune-inflammation index.

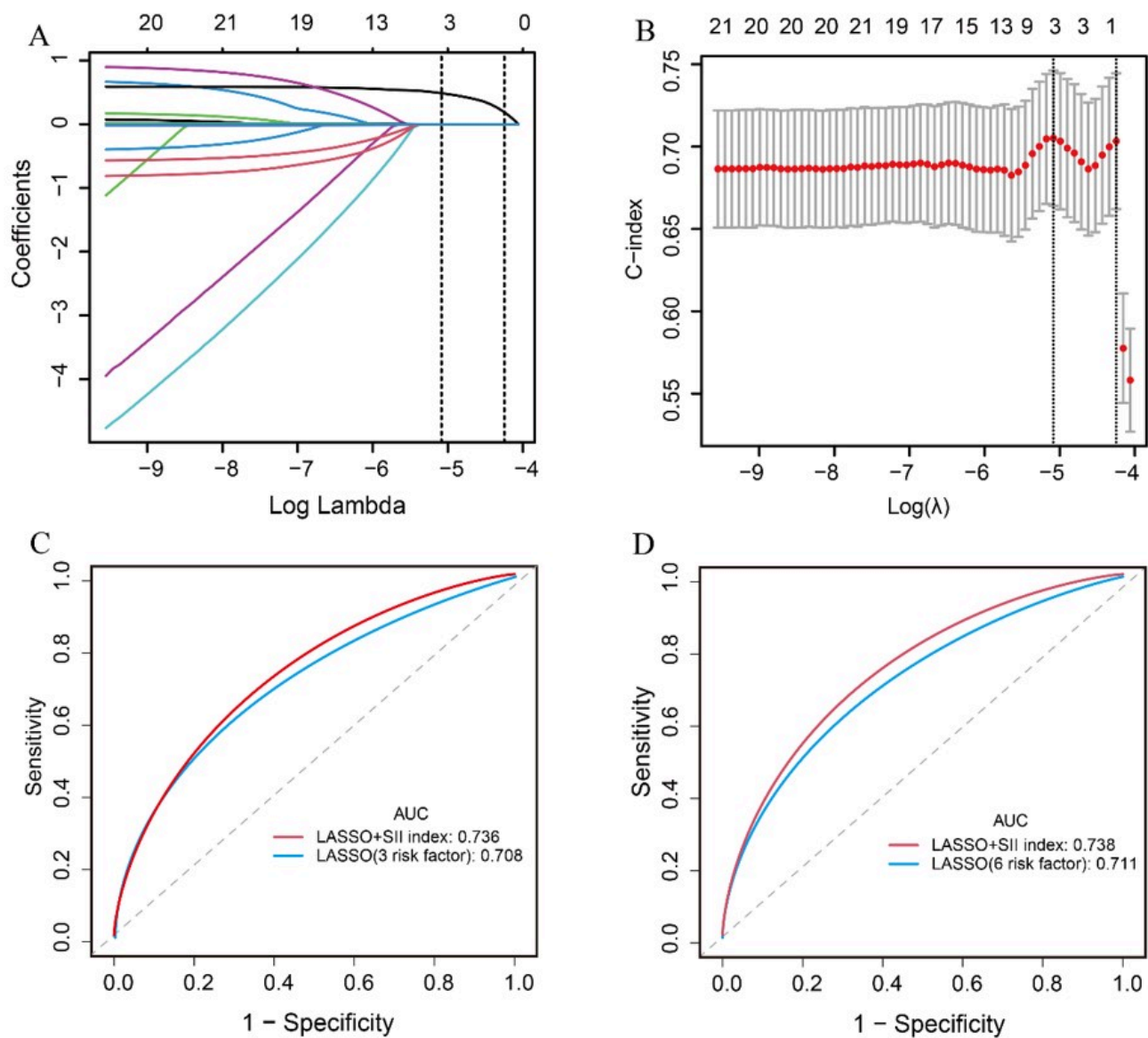


Figure 3

Prediction comparison of the LASSO model and the LASSO+SII model for 6-month mortality in patients with chronic HF in the ZIGHF cohort. A: Association between the coefficient of variables and λ . Each line corresponded to one distinct variable. Along with the increasing $\log(\lambda)$, the coefficient of the variable tended to be close to 0. B: The selection of an applicable model. We plotted the misclassification versus $\log(\lambda)$. C: Prediction comparison of the LASSO model (3 risk factors) and the LASSO+SII model via C-index. D: Prediction comparison of the LASSO model (6 risk factors) and the LASSO+SII model via C-index.

Note: LASSO = least absolute shrinkage and selection operator; SII = systemic immune-inflammation index; HF = heart failure.

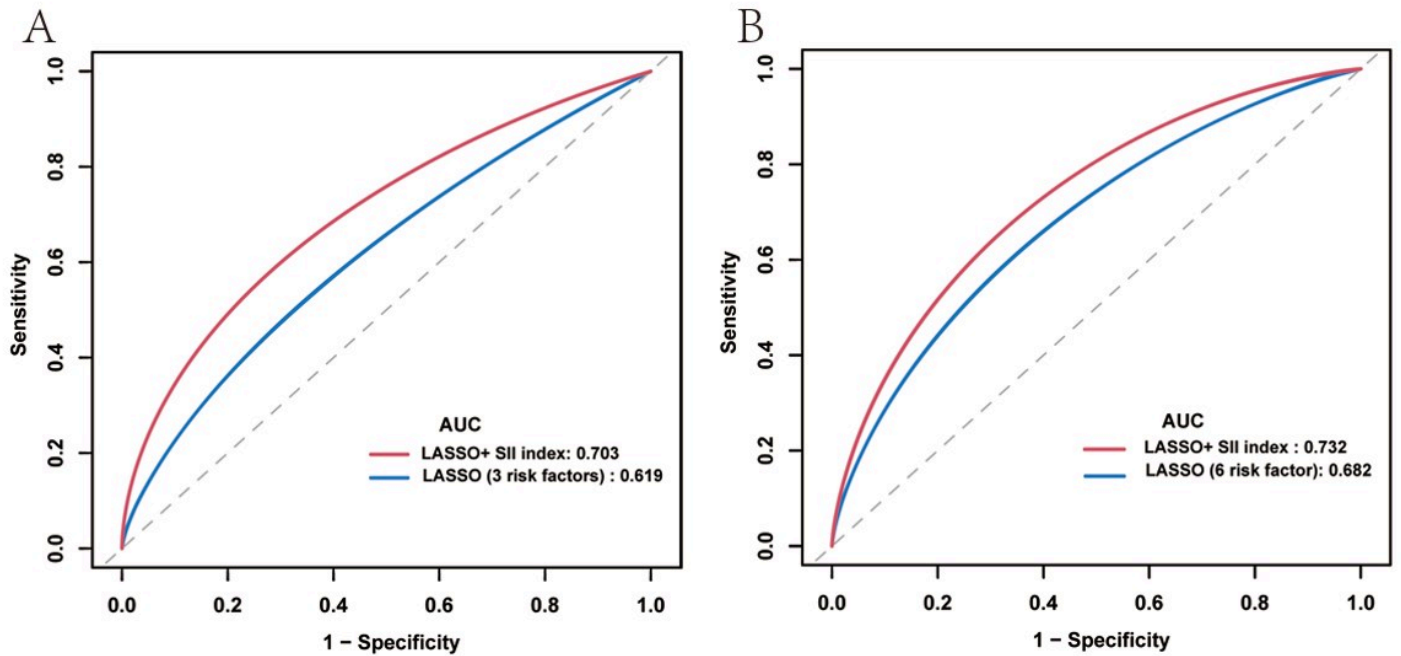


Figure 4

Validation of prediction ability for death by comprising the LASSO+SII model and the LASSO model in patients with chronic HF in the MIMIC-HF cohort.

A: Prediction comparison of the LASSO model (3 risk factors) and the LASSO+SII model via the C-index. B: Prediction comparison of the LASSO model (6 risk factors model) and the LASSO+SII model via C-index.

Note: LASSO = least absolute shrinkage and selection operator; SII = systemic immune-inflammation index; HF = heart failure.

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