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Predicting mortality within 28 days using SIICI and SOFA in a retrospective cohort of invasively ventilated patients with severe nosocomial pneumonia

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Research Article

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Abstract

Background

Early prognostic assessment is essential in patients with severe hospital-acquired pneumonia (SHAP) requiring invasive mechanical ventilation. We evaluated the association between the systemic immune-inflammatory complex index (SIICI) and 28-day mortality and its incremental predictive value beyond the Sequential Organ Failure Assessment (SOFA) score.

Methods

This retrospective cohort study included 159 adults with SHAP who received invasive mechanical ventilation between January 2019 and December 2025. SIICI was calculated from the first complete blood count within 24 h after diagnosis; SOFA was assessed using the worst values during this period. Independent predictors were identified using multivariable logistic regression. A model combining SIICI and SOFA was compared with SOFA alone using the area under the receiver operating characteristic curve (AUC), net reclassification improvement (NRI), and integrated discrimination improvement (IDI). Calibration was assessed with 1000 bootstrap resamples. Decision curve analysis and Kaplan–Meier analysis assessed potential clinical utility and survival differences, respectively.

Results

Of 159 patients, 57 died and 102 survived within 28 days. SIICI (odds ratio [OR] 1.008, 95% confidence interval [CI] 1.001–1.016; P = 0.031), SOFA (OR 1.526, 95% CI 1.184–1.966; P = 0.001), and the Acute Physiology and Chronic Health Evaluation II score (OR 1.172, 95% CI 1.040–1.321; P = 0.009) were independently associated with mortality. The combined model had an AUC of 0.887 (95% CI 0.837–0.937), compared with 0.850 for SOFA alone (ΔAUC 0.037; P = 0.008) and 0.781 for SIICI alone. NRI was 0.162 (P = 0.004), and IDI was 0.067 (P = 0.003). Calibration showed agreement between predicted and observed outcomes (Hosmer–Lemeshow χ^2 = 11.251; P = 0.188). Decision curve analysis suggested a net benefit over a range of threshold probabilities. Mortality was 75.4% in the high-risk group and 13.7% in the low-risk group defined by the combined model (log-rank P < 0.001).

Conclusions

Higher SIICI was independently associated with 28-day mortality in invasively ventilated patients with SHAP. Adding SIICI to SOFA improved discrimination and risk stratification in this cohort. External validation is needed before clinical implementation.

Background

Hospital-acquired pneumonia (HAP) is a common nosocomial infection, accounting for approximately 25% of infectious episodes in intensive care units (ICUs) [1]. Severe HAP (SHAP) requiring ICU admission carries a particularly poor prognosis; a prospective observational study reported an ICU mortality rate of up to 53% [2]. Patients requiring invasive mechanical ventilation represent a high-risk population in whom early prognostic assessment is especially important. Prediction models have also been developed for ventilator-associated pneumonia (VAP) in mechanically ventilated patients [3]. Accurate risk stratification using readily available clinical indicators may help identify patients at increased risk and inform treatment planning. However, conventional tools such as the Sequential Organ Failure Assessment (SOFA) score primarily quantify established organ dysfunction and may incompletely capture early immune and inflammatory dysregulation [4].

Composite inflammatory indices derived from complete blood counts have attracted increasing attention because they are inexpensive and readily available. A higher neutrophil-to-lymphocyte ratio (NLR) has been associated with increased mortality in sepsis [5], and the systemic inflammation response index (SIRI) has shown prognostic value in patients with sepsis [6]. Nevertheless, these indices capture only selected components of the host response. The recently proposed systemic immune-inflammatory complex index (SIICI) incorporates neutrophils, monocytes, platelets, and lymphocytes and has shown greater discrimination for sepsis mortality than other composite inflammatory indices [7].

As in sepsis, the pathophysiology of SHAP involves dysregulated proinflammatory and anti-inflammatory responses. Excessive neutrophil and monocyte activation can amplify inflammation and contribute to multiorgan dysfunction, whereas compensatory anti-inflammatory responses may promote immunosuppression and increase susceptibility to secondary infections [8, 9]. These mechanisms provide a rationale for evaluating SIICI in SHAP. However, evidence regarding its prognostic value in patients receiving invasive mechanical ventilation remains limited. Because SOFA quantifies organ dysfunction whereas SIICI reflects aspects of systemic immune and inflammatory status, the two measures may provide complementary prognostic information.

This study therefore assessed the association between SIICI and 28-day mortality in invasively ventilated patients with SHAP and examined whether adding SIICI to SOFA improved mortality discrimination and risk stratification.

Methods

Study design and participants

This retrospective cohort study included patients with SHAP admitted to the respiratory intensive care unit (RICU) of Zhengzhou Central Hospital Affiliated to Zhengzhou University between January 2019 and December 2025. Patients were eligible if they were aged at least 18 years, met the diagnostic criteria for HAP in the Chinese guidelines for the diagnosis and treatment of hospital-acquired pneumonia and

ventilator-associated pneumonia in adults (2018 edition) [1], and met the criteria for severe disease, defined as requiring invasive mechanical ventilation or having septic shock requiring vasoactive support despite adequate fluid resuscitation. All included patients received invasive mechanical ventilation and had complete clinical records.

Patients were excluded if they had an immunodeficiency disorder or were receiving long-term immunosuppressive therapy, died or left hospital against medical advice within 24 h after SHAP diagnosis, or were pregnant or breastfeeding. A total of 159 patients met the eligibility criteria.

The study was approved by the Medical Ethics Committee of Zhengzhou Central Hospital Affiliated to Zhengzhou University (approval number 2026020). The requirement for informed consent was waived because of the retrospective study design.

Data collection and outcome assessment

Clinical information was collected retrospectively from electronic medical records and nursing records. Data included age, sex, the presence of septic shock, laboratory measurements, and disease severity scores. The first complete blood count obtained within 24 h after SHAP diagnosis provided white blood cell, neutrophil, lymphocyte, monocyte, and platelet counts. Biochemical measurements included alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum creatinine, and blood urea nitrogen (BUN). SOFA and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores were calculated using the worst values recorded within 24 h after diagnosis.

The primary outcome was all-cause mortality within 28 days after SHAP diagnosis, with the date of diagnosis serving as the start of follow-up. In-hospital deaths were confirmed using electronic medical records and death records. For patients discharged before day 28, survival status was verified using readmission or outpatient records together with telephone follow-up. Patients for whom follow-up information could not be obtained were excluded.

Calculation of SIICI

SIICI was calculated from the first complete blood count obtained within 24 h after diagnosis using the following formula [7]:

SIICI = (neutrophil count × monocyte count × 10^3) / (platelet count × lymphocyte count).

Statistical analysis

Statistical analyses were performed using R version 4.4.1. Normally distributed continuous variables are presented as means ± standard deviations and were compared using independent-samples t tests. Non-normally distributed variables are presented as medians (25th percentile, 75th percentile) and were

compared using Mann–Whitney U tests. Categorical variables are presented as numbers (percentages) and were compared using χ^2 tests or Fisher's exact tests, as appropriate.

Univariable analyses were used to screen candidate predictors. Variables meeting the $P < 0.05$ criterion and showing no multicollinearity were entered into multivariable logistic regression to identify independent predictors of 28-day mortality. To assess the incremental value of SIICI beyond SOFA, a base model containing SOFA alone was compared with a logistic regression model containing both SOFA and SIICI. Predicted probabilities were obtained from the combined model.

Receiver operating characteristic (ROC) curves were generated, and areas under the curves (AUCs) with 95% confidence intervals (CIs) were calculated. Optimal cutoffs were selected using the maximum Youden index, and AUCs were compared using DeLong's test. Net reclassification improvement (NRI) and integrated discrimination improvement (IDI) were used to evaluate the incremental predictive value of adding SIICI to SOFA. For categorical reclassification, predicted mortality probabilities were classified as low (< 0.10), intermediate (0.10–0.90), or high (> 0.90).

Calibration was assessed using 1000 bootstrap resamples and the Hosmer–Lemeshow test. Decision curve analysis (DCA) was used to assess the potential clinical net benefit. Patients were also classified into high- and low-risk groups using the optimal ROC-derived cutoff for each predictor or model. Survival over 28 days was estimated using the Kaplan–Meier method, and differences were compared using the log-rank test.The date of SHAP diagnosis served as the time origin; patients who died contributed their length of ICU stay as the event time, whereas survivors were censored at ICU discharge or at day 28, whichever occurred first. All tests were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Clinical characteristics

Of the 159 patients included, 102 survived and 57 died within 28 days after SHAP diagnosis. Clinical characteristics are summarized in Table 1. Compared with survivors, non-survivors more frequently had septic shock and had higher white blood cell counts, procalcitonin (PCT), SIICI, BUN, lactate, activated partial thromboplastin time (APTT), international normalized ratio (INR), SOFA scores, and APACHE II scores, together with lower albumin concentrations (all $P < 0.05$). Age, sex, and the other characteristics listed in Table 1 did not differ significantly between groups (all $P > 0.05$).

Table 1
Clinical characteristics of survivors and non-survivors at 28 days

Variable	Survivors (n = 102)	Non-survivors (n = 57)	Statistic (Z/t/χ^2)	P value
Age (years)[]	74.50 (63.25–83.00)	79.00 (70.00–84.00)	1.632	0.103
Male sex[]	69 (67.6%)	40 (70.2%)	0.023	0.857
Septic shock[]	36 (35.3%)	35 (61.4%)	9.057	0.004
CRO infection[]	26 (25.5%)	13 (22.8%)	0.034	0.836
Bloodstream infection[]	5 (4.9%)	8 (14.0%)	2.937	0.080
Duration of invasive ventilation (h)[]	322.5 (208.5–523.5)	327.0 (215.0–453.0)	0.281	0.779
Temperature (°C)[]	36.8 (36.7–37.0)	36.8 (36.7–37.0)	0.035	0.972
Heart rate (beats/min)[]	98 (89–115)	108 (85–121)	1.715	0.086
Respiratory rate (breaths/min)[]	25.5 (20–30)	25 (19–29)	0.422	0.673
Mean arterial pressure (mmHg)[]	85.61 ± 18.48	83.02 ± 21.2	0.795	0.428
White blood cell count (10^9/L)[]	9.74 (7.52–14.27)	13.29 (9.44–18.78)	3.168	0.002
CRP (mg/L)[]	76.56 (35.89–115.00)	88.99 (57.47–132.74)	1.514	0.130
PCT (ng/mL)[]	0.50 (0.21–1.30)	1.80 (0.52–5.15)	3.955	< 0.001
SIICI[]	24.94 (11.25–48.81)	78.46 (36.82–158.49)	5.874	< 0.001
Albumin (g/L)[]	29.05 (27.22–32.5)	28 (25.35–30.35)	1.970	0.049
ALT (U/L)[]	23 (14–48.5)	30 (19.5–49)	1.244	0.214
AST (U/L)[]	35 (24–61.25)	45 (30–73.5)	1.890	0.059
Serum creatinine (μmol/L)[]	75.1 (48.55–107.93)	99.3 (57.98–185.52)	1.807	0.071

[] n (%); [] mean ± standard deviation; [] median (25th–75th percentile). Continuous variables were compared using independent-samples t tests or Mann–Whitney U tests, as appropriate; categorical variables were compared using χ^2 tests or Fisher's exact tests. CRO infection refers to infection with carbapenem-resistant Gram-negative bacilli in the source definition. Abbreviations are defined in the List of abbreviations.

Variable	Survivors (n = 102)	Non-survivors (n = 57)	Statistic (Z/t/χ^2)	P value
BUN (mmol/L)□	10.2 (6.29–14.7)	12.7 (8.25–22.78)	2.614	0.009
PaO_2 (mmHg)□	95.3 (79.42–118.75)	98.2 (81.8–124)	0.334	0.738
$PaCO_2$ (mmHg)□	38.2 (33.9–45.48)	39.7 (30.6–47.2)	0.390	0.697
PaO_2/FiO_2 ratio□	161.3 (111.53–221.25)	147.0 (102.7–232)	-0.831	0.406
Lactate (mmol/L)□	1.3 (0.83–1.95)	1.9 (1.3–2.8)	3.602	< 0.001
APTT (s)□	33.05 (27.4–37.38)	35.9 (31.2–44.2)	2.600	0.009
INR□	1.12 (1.03–1.23)	1.21 (1.1–1.41)	3.595	< 0.001
D-dimer (mg/L)□	2.83 (1.72–5.73)	3.18 (1.79–8.47)	0.693	0.488
SOFA score□	5 (4–7)	8 (8–10)	7.389	< 0.001
APACHE II score□	12.0 (9.2–15.0)	18.0 (15.0–22.0)	6.821	< 0.001

□ n (%); □ mean ± standard deviation; □ median (25th–75th percentile). Continuous variables were compared using independent-samples t tests or Mann–Whitney U tests, as appropriate; categorical variables were compared using χ^2 tests or Fisher's exact tests. CRO infection refers to infection with carbapenem-resistant Gram-negative bacilli in the source definition. Abbreviations are defined in the List of abbreviations.

Logistic regression analysis

Multicollinearity diagnostics showed variance inflation factors of 1.04–1.38, all below 5, indicating no substantial multicollinearity among the candidate variables [10]. The univariable and multivariable logistic regression results are presented in Table 2. SIICI (OR 1.008, 95% CI 1.001–1.016; P = 0.031), SOFA score (OR 1.526, 95% CI 1.184–1.966; P = 0.001), and APACHE II score (OR 1.172, 95% CI 1.040–1.321; P = 0.009) were independently associated with 28-day mortality. Some variables that differed significantly between outcome groups were not significant in univariable logistic regression; skewed distributions or nonlinear associations may have contributed to this difference.

Table 2
Univariable and multivariable logistic regression analyses of 28-day mortality

Variable	Univariable analysis		Multivariable analysis			
	OR (95% CI)	P	OR (95% CI)	P	β	SE
Septic shock	2.917 (1.492–5.701)	0.002	0.881 (0.322–2.408)	0.805	-0.127	0.513
White blood cell count	1.086 (1.026–1.150)	0.005	1.090 (0.992–1.199)	0.073	0.087	0.048
PCT	1.048 (0.993–1.106)	0.087				
SIICI	1.012 (1.006–1.019)	< 0.001	1.008 (1.001–1.016)	0.031	0.008	0.004
Albumin	0.948 (0.884–1.017)	0.137				
BUN	1.056 (1.014–1.099)	0.008	1.014 (0.974–1.056)	0.495	0.014	0.021
Lactate	1.131 (0.992–1.290)	0.066				
APTT	1.021 (0.996–1.047)	0.104				
INR	2.830 (0.883–9.070)	0.080				
SOFA score	1.840 (1.509–2.245)	< 0.001	1.526 (1.184–1.966)	0.001	0.422	0.129
APACHE II score	1.328 (1.207–1.461)	< 0.001	1.172 (1.040–1.321)	0.009	0.159	0.061

OR, odds ratio; CI, confidence interval; β, regression coefficient; SE, standard error. A dash indicates that a multivariable estimate was not reported. The multivariable model in this table is distinct from the two-predictor SIICI–SOFA model. Other abbreviations are defined in the List of abbreviations.

Discrimination and incremental predictive value

The AUCs for SIICI and SOFA alone were 0.781 and 0.850, respectively; the difference was not statistically significant (P = 0.146). Adding SIICI to SOFA increased the AUC to 0.887 (95% CI 0.837–0.937), significantly exceeding that of SOFA alone (ΔAUC 0.037; DeLong P = 0.008). The combined model also improved reclassification and discrimination, with an NRI of 0.162 (P = 0.004) and an IDI of 0.067 (P = 0.003). Detailed performance measures are provided in Table 3, and the ROC curves are shown in Fig. 1.

Table 3
Predictive performance of SIICI, SOFA, and the combined model

Performance measure	SIICI	SOFA	SIICI–SOFA model
AUC	0.781	0.850	0.887
95% CI	0.705–0.858	0.792–0.908	0.837–0.937
P value for AUC	< 0.001	< 0.001	< 0.001
Optimal cutoff	45.05	7.5	0.397
Sensitivity	73.7%	75.4%	75.4%
Specificity	72.5%	79.4%	86.3%
ΔAUC (P value)			0.037 (0.008)
NRI (P value)			0.162 (0.004)
IDI (P value)			0.067 (0.003)

AUC, area under the receiver operating characteristic curve; CI, confidence interval; NRI, net reclassification improvement; IDI, integrated discrimination improvement. Cutoffs were selected using the maximum Youden index. ΔAUC, NRI, and IDI were calculated with SOFA as the reference model. The combined-model cutoff is a predicted probability between 0 and 1. A dash indicates that the corresponding result was not reported or was not applicable.

The logistic regression equation for the combined model was:

$$\text{logit}(P) = -5.182 + 0.009 \times \text{SIICI} + 0.565 \times \text{SOFA},$$

where P denotes the predicted probability of death within 28 days.

Calibration and potential clinical utility

Calibration analysis using 1000 bootstrap resamples showed that the bias-corrected calibration curve was close to the ideal reference line, with a mean absolute error of 0.013. The Hosmer–Lemeshow test did not indicate a statistically significant lack of fit ($\chi^2 = 11.251$; $P = 0.188$; Fig. 2). DCA suggested that the combined model provided a net benefit over a range of threshold probabilities, supporting its potential use in identifying patients at increased mortality risk (Fig. 3).

Survival analysis

Kaplan–Meier curves were generated using the optimal ROC-derived cutoffs for SIICI, SOFA, and the combined model. For SIICI, 28-day mortality was 60.0% in the high-risk group and 16.9% in the low-risk group (log-rank $P < 0.001$; Fig. 4A). For SOFA, the corresponding mortality rates were 67.2% and 14.7% (log-rank $P < 0.001$; Fig. 4B). For the combined model, mortality was 75.4% in the high-risk group and 13.7% in the low-risk group (log-rank $P < 0.001$; Fig. 4C).

Discussion

In this cohort of invasively ventilated patients with SHAP, higher SIICI was independently associated with 28-day mortality. Combining SIICI with SOFA improved the AUC from 0.850 to 0.887 and yielded an NRI of 0.162 and an IDI of 0.067. These findings suggest that a complete-blood-count-derived index may complement an established organ dysfunction score for prognostic assessment in this population.

HAP remains an important cause of poor outcomes in critically ill patients [1]. Dysregulated host responses and the resulting progression of organ dysfunction contribute to its clinical severity. SIICI integrates four circulating cell populations and may therefore reflect several aspects of inflammatory activation and immune regulation. Previous studies have evaluated SIICI for sepsis prognosis and postoperative sepsis prediction [7, 11], but its prognostic role in invasively ventilated patients with SHAP has received limited attention.

Wang et al. first proposed SIICI for prognostic assessment in sepsis [7]. Its biological rationale relates to the involvement of myeloid cell activation, lymphocyte dysfunction, and platelet abnormalities in severe infection. Excessive neutrophil and monocyte activation can promote inflammatory mediator release, whereas impaired lymphocyte function contributes to immunosuppression [12]. Platelet abnormalities also participate in immunothrombosis. SIICI places neutrophils and monocytes in the numerator and lymphocytes and platelets in the denominator, combining these related processes in a single measure. Severe pneumonia also involves dysregulated inflammation and immune suppression [8, 9, 13, 14], providing a plausible explanation for the association observed in the present study.

The adjusted OR of 1.008 per unit of SIICI should be interpreted in relation to its distribution. Median SIICI was 78.46 in non-survivors and 24.94 in survivors, a difference of approximately 53.5 units. Based on the reported per-unit OR, a 10-unit increase corresponds to approximately 8% higher odds of 28-day mortality. Wang et al. studied 3944 patients with sepsis in the Medical Information Mart for Intensive Care IV (MIMIC-IV) database and reported an AUC of 0.640 for 30-day mortality [7]. Zhou et al. found that adding SIICI to a base model improved prediction of sepsis after ureteroscopic lithotripsy, increasing the AUC from 0.807 to 0.863 ($P < 0.001$) [11]. Although the populations and outcomes differ, these findings support further investigation of SIICI in infection-related risk assessment.

SOFA is widely used to quantify organ dysfunction and is central to the clinical operationalization of Sepsis-3 [15, 16]. Its AUC of 0.850 (95% CI 0.792–0.908) in the present study is consistent with the AUC of 0.856 reported by Wen et al. for 30-day mortality in HAP [17]. Raith et al. also demonstrated its prognostic value in ICU patients with suspected infection [18]. However, SOFA requires assessment of respiratory, cardiovascular, hepatic, renal, coagulation, and neurological function. Some components, including bilirubin and creatinine, may not become markedly abnormal early in the disease course, and measurements may be influenced by interventions such as renal replacement therapy [19]. An additional indicator of immune and inflammatory status may therefore improve risk stratification.

The incremental value of SIICI was supported by several performance measures. The increase in AUC of 0.037 was statistically significant, and an NRI of 0.162 indicated a net improvement in reclassification across the specified risk categories [20]. The IDI of 0.067 corresponded to a 6.7-percentage-point increase in the difference in mean predicted probabilities between non-survivors and survivors. Calibration analysis showed agreement between predicted and observed outcomes, and DCA suggested potential clinical net benefit. At the optimal predicted-probability cutoff of 0.397, mortality in the high-risk group was approximately 5.5 times that in the low-risk group. The separation of the survival curves suggests that the model may help distinguish prognostic trajectories during the 28-day follow-up period. These observations support further evaluation of the model as an aid to early clinical assessment.

The improvement may reflect the different physiological processes captured by SIICI and SOFA. Immune and inflammatory dysregulation can contribute to immunothrombosis, impaired microcirculatory perfusion, and subsequent organ dysfunction [21, 22]. SOFA measures established dysfunction across six organ systems, whereas SIICI reflects circulating cellular components of the host response. In some patients, pronounced inflammation may precede overt organ dysfunction, potentially limiting early risk assessment using SOFA alone [23, 24]. Conversely, when organ failure predominates, an inflammatory index alone may not adequately capture prognosis. These complementary perspectives may explain the improvement observed when the measures were combined. Related evidence comes from Mangalesh et al., who found that adding the systemic immune-inflammation index (SII) to SOFA improved sepsis mortality prediction (NRI 0.084, P = 0.025; IDI 0.056, P = 0.001) [25].

APACHE II was also independently associated with 28-day mortality (OR 1.172, 95% CI 1.040–1.321; P = 0.009). However, it requires 12 physiological measurements, including arterial blood gas and creatinine measurements, and its calculation can be time-consuming in routine ICU practice. SIICI requires only four complete blood count components, and SOFA summarizes six organ systems. We therefore evaluated SIICI with SOFA as a relatively simple model intended to balance prognostic performance with practical feasibility. This choice does not establish superiority over a model incorporating APACHE II.

Several limitations should be considered. First, the study was retrospective and conducted at a single center, with a limited sample size. Detailed pathogen and antimicrobial resistance profiles, antibiotic treatment strategies, and ventilation parameters were not comprehensively captured. These factors may confound the associations and affect model performance, and multicenter prospective validation is required. Second, SIICI was calculated only from the first complete blood count within 24 h after diagnosis. We did not evaluate longitudinal changes, which may provide additional prognostic information and warrant investigation in future studies.

Conclusions

Higher SIICI was independently associated with 28-day mortality in patients with SHAP receiving invasive mechanical ventilation. Adding SIICI to SOFA improved discrimination and risk stratification in this retrospective cohort. Because SIICI can be calculated from routinely available blood counts, the

combined model may offer a practical approach to prognostic assessment. External validation is required before it can guide clinical management.

Abbreviations

ALT
alanine aminotransferase
APACHE II
Acute Physiology and Chronic Health Evaluation II
APTT
activated partial thromboplastin time
AST
aspartate aminotransferase
AUC
area under the receiver operating characteristic curve
BUN
blood urea nitrogen
CI
confidence interval
CRO
carbapenem-resistant organism
CRP
C-reactive protein
DCA
decision curve analysis
HAP
hospital-acquired pneumonia
ICU
intensive care unit
IDI
integrated discrimination improvement
INR
international normalized ratio
MIMIC-IV
Medical Information Mart for Intensive Care IV
NLR
neutrophil-to-lymphocyte ratio
NRI
net reclassification improvement
OR

odds ratio
$PaCO_2$
arterial partial pressure of carbon dioxide
PaO_2
arterial partial pressure of oxygen
PCT
procalcitonin
RICU
respiratory intensive care unit
ROC
receiver operating characteristic
SE
standard error
SHAP
severe hospital-acquired pneumonia
SII
systemic immune-inflammation index
SIICI
systemic immune-inflammatory complex index
SIRI
systemic inflammation response index
SOFA
Sequential Organ Failure Assessment
VAP
ventilator-associated pneumonia
VIF
variance inflation factor.

Declarations

Ethics approval and consent to participate

The study was approved by the Medical Ethics Committee of Zhengzhou Central Hospital Affiliated to Zhengzhou University (approval number 2026020). The requirement for informed consent was waived because of the retrospective design.

Clinical trial number

Not applicable. This study is a retrospective observational cohort study based on routinely collected clinical data; no interventional clinical trial was conducted, and trial registration is therefore not required.

Consent for publication

Not applicable. This study did not contain any individual person's data in any form, including individual details, images, or videos.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Author contributions

TH conceived and designed the study, collected and curated the clinical data, performed the statistical analysis, and drafted the manuscript. RXZ, XYL, SFL and RD contributed to patient management and clinical data collection, and assisted with data verification. YQG supervised the study and provided critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Figures

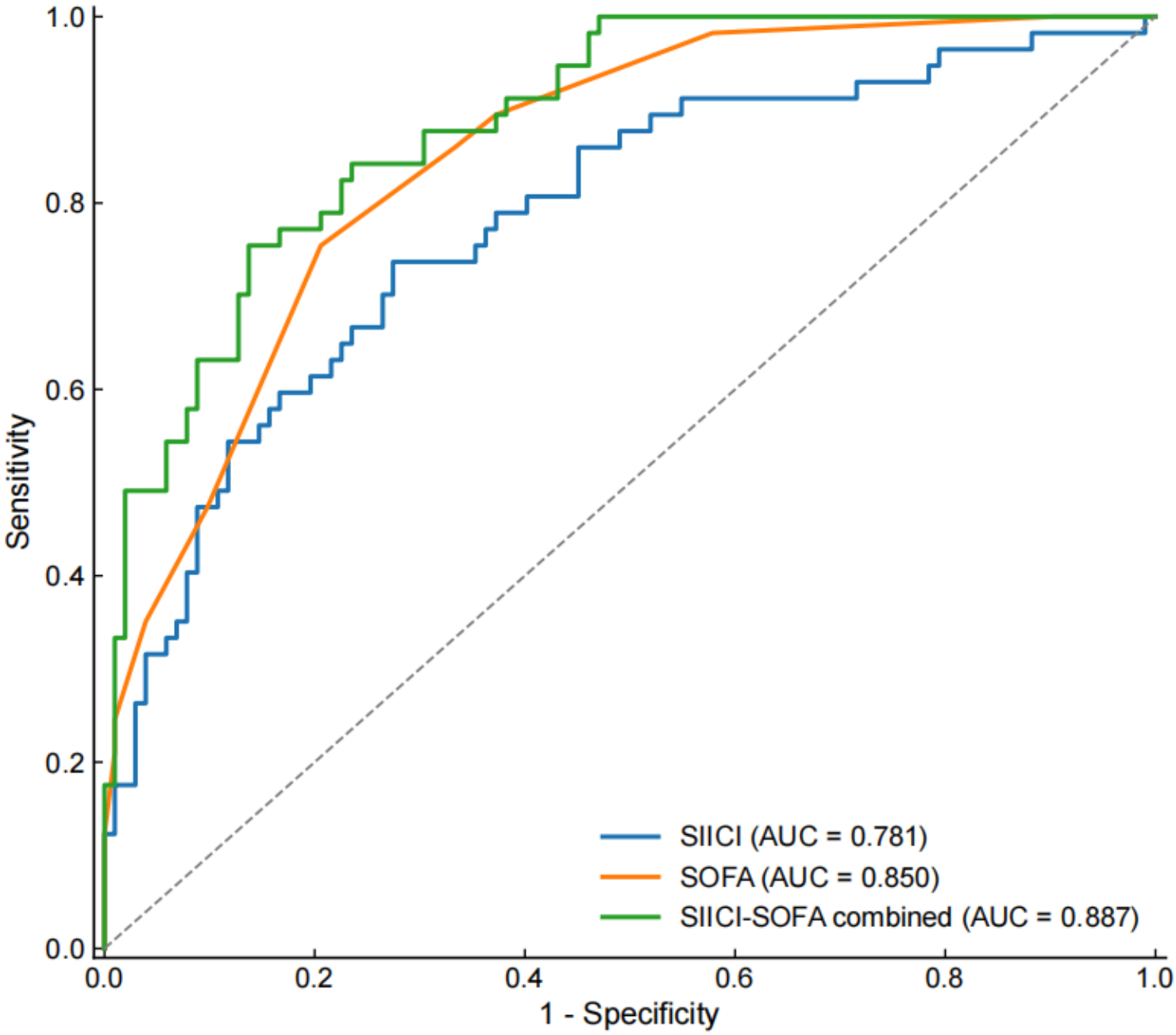


Figure 1

Receiver operating characteristic curves for predicting 28-day mortality. Curves are shown for SIICI alone, SOFA alone, and the combined SIICI–SOFA model. The corresponding AUCs were 0.781, 0.850, and 0.887, respectively.

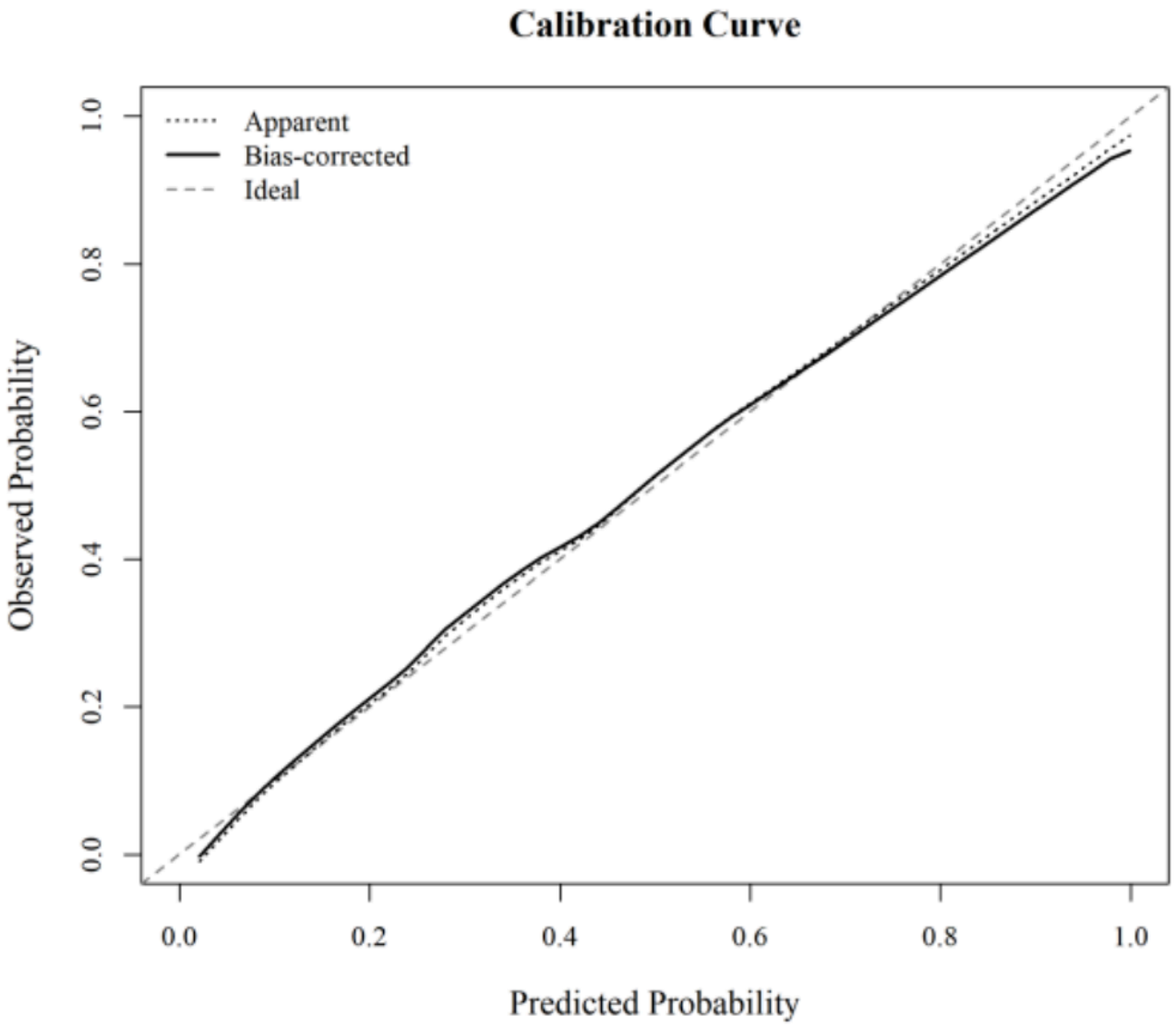


Figure 2

Calibration of the combined SIICI–SOFA model. The apparent and bias-corrected calibration curves are compared with the ideal reference line. Internal calibration assessment used 1000 bootstrap resamples; the mean absolute error was 0.013.

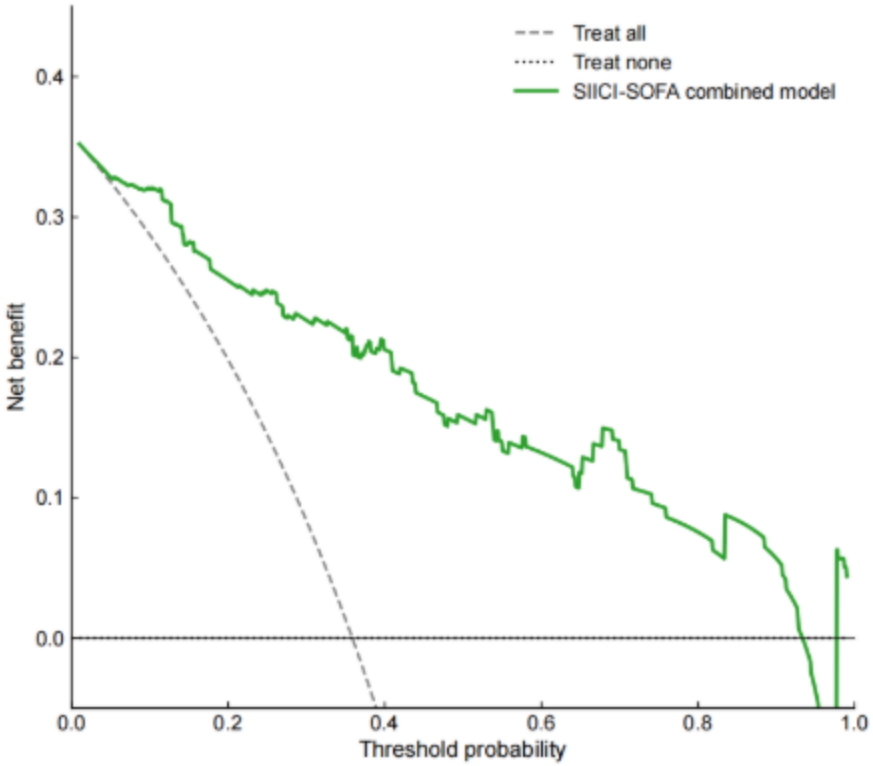


Figure 3

Decision curve analysis of the combined SIICI–SOFA model. Net benefit is plotted against threshold probability. The model is compared with the strategies of treating all patients and treating none.

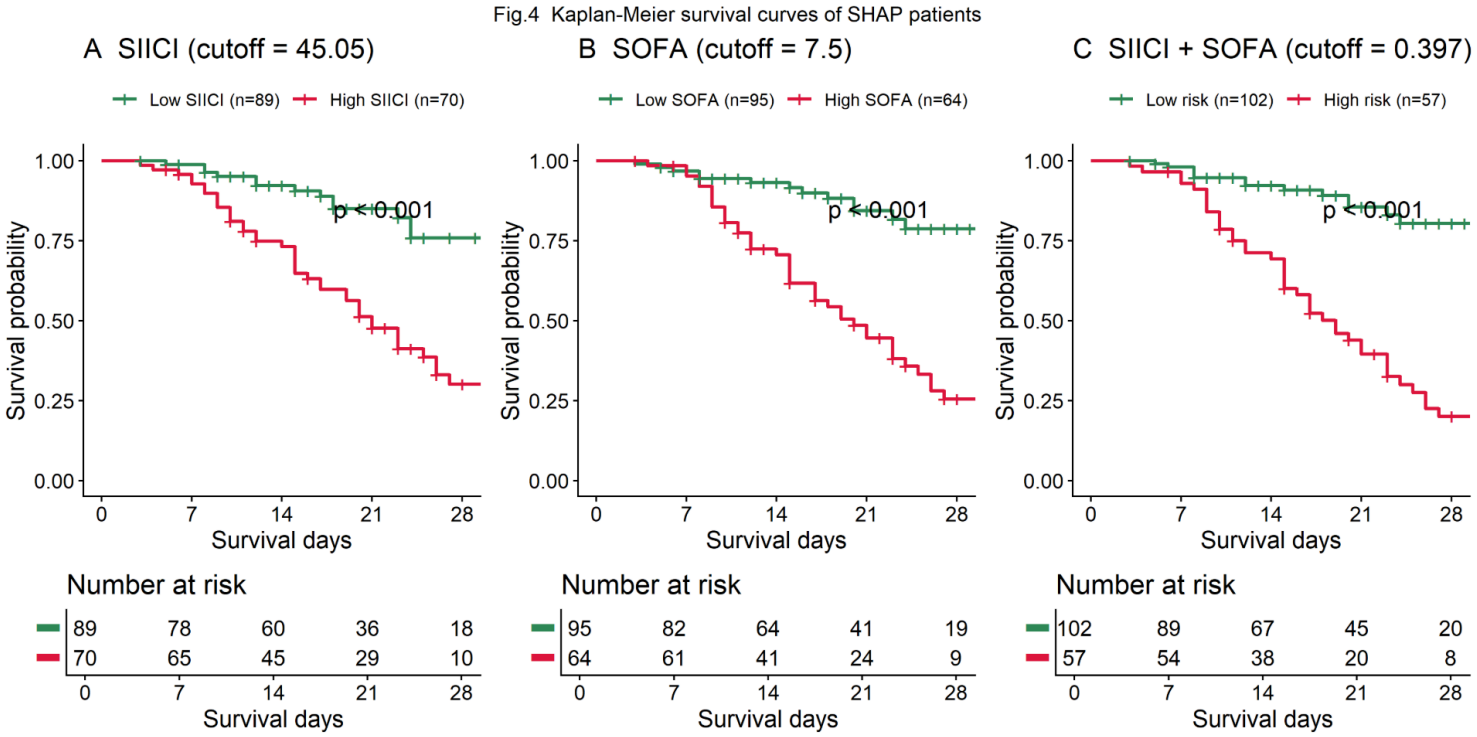


Figure 4

Kaplan–Meier survival curves stratified by prognostic risk. Patients were stratified using the optimal ROC-derived cutoffs for (A) SIICI, 45.05; (B) SOFA, 7.5; and (C) the predicted probability from the combined SIICI–SOFA model, 0.397. Survival differences were assessed using the log-rank test (all $P < 0.001$). Group-specific mortality rates reported in the text were calculated from the events within each risk group.