Journal of Inflammation Research

Dovepress
Taylor & Francis Group

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ORIGINAL RESEARCH

Combined Systemic Immune-Inflammation Index-Prognostic Nutritional Index Score in Evaluating the Prognosis of Patients with Severe Community-Acquired Pneumonia

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Background: While both the systemic immune-inflammation index (SII) and prognostic nutritional index (PNI) have demonstrated prognostic value in various diseases, the clinical utility of their combined score (SII-PNI) for predicting outcomes in patients with severe community-acquired pneumonia (SCAP) remains incompletely understood. The aim of this study is to explore the predictive value of SII-PNI score in patients with SCAP.

Methods: We conducted a retrospective analysis of the clinical data of 138 patients diagnosed with SCAP. The SII, PNI, and the SNII-PNI score were calculated. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal threshold of SII-PNI. Multivariable logistic regression models were used to assess the association between the SII-PNI score and 28-day mortality.

Results: The cutoff values for predicting 28-day mortality were >4689.82 for SII and <32.18 for PNI, respectively, with sensitivities of 59.1% and 60.3% and specificities of 85.3% and 68.2%. Multivariate analysis reveals that a SII-PNI score of 2 (OR, 14.11; 95% CI, 3.18–62.66; p = 0.001) was independently associated with a high risk of 28-day mortality.

Conclusion: Our results indicate that a higher SII-PNI score at admission was linked to poor prognosis in SCAP patients. The combined SII-PNI score can effectively help clinicians assess disease progression and optimize risk assessment and clinical management for SCAP patients.

Keywords: severe community-acquired pneumonia, systemic immune-inflammation index, SII, prognostic nutritional index, PNI, SII-PNI score, prognosis

Introduction

Severe community-acquired pneumonia (SCAP) represents a critical global health challenge,[1] associated with significant morbidity and mortality even with modern antimicrobial therapies and critical care support.[2,3] Clinical deterioration typically manifests within 24–72 hours post-admission,[4] which underscores the importance of early prognostic stratification to guide intensive monitoring and targeted interventions.[5] Existing severity assessment tools such as CURB-65 (confusion, urea, respiratory rate, blood pressure and 65 years of age), Pneumonia Severity Index (PSI), and the Quick Sepsis-related Organ Failure Assessment (qSOFA), while clinically useful, demonstrate limited discriminative capacity for predicting SCAP-specific outcomes.[6,7] Biomarkers such as lactate dehydrogenase (LDH), C-reactive protein (CRP), procalcitonin (PCT), neutrophil-to-lymphocyte ratio (NLR), and specific cytokines exhibit similar limitations.[8–12] Notably, up to 40% of hospitalized CAP patients present with nutritional deficits,[13] which are modifiable risk factors strongly linked to immune dysfunction and adverse clinical outcomes. The Prognostic Nutritional Index (PNI) combines serum albumin levels and lymphocyte counts, is a validated tool for assessing immune and nutritional status. By

Received: 21 February 2025
Accepted: 24 May 2025
Published: 31 May 2025

integrating nutritional and inflammatory biomarkers, PNI has demonstrated prognostic value in COVID-19,[14] AECOPD,[15] cancer,[16] and heart failure.[17] Similarly, the Systemic Immune-Inflammation Index (SII), derived from neutrophil, lymphocyte, and platelet counts, serves as a systemic inflammation marker and may predict outcomes in malignancies[18] and COVID-19.[19–21] Both indices utilize routine laboratory parameters and provide cost-effective clinical risk stratification.

Emerging evidence suggests synergistic effects from combining SII and PNI.[22–24] In oncology, the composite SII-PNI score outperforms individual indices in predicting chemotherapy response and survival outcomes.[25] In gastric cancer, the pre-treatment SII-PNI score serves as a key tool for identifying high-risk individuals and predicting chemotherapy sensitivity.[26–28] Notably, in advanced non-small cell lung cancer, while neither baseline SII nor PNI shows significant correlation with chemotherapy response ($p > 0.05$), a SII-PNI score of 2 emerges as an independent risk factor for reduced progression-free survival (PFS) and overall survival (OS).[24] Mechanistically, this composite measure captures both the systemic inflammation (via SII) and nutritional status (via PNI) – dual axes critically impaired in SCAP pathophysiology.

Despite these advances, no studies have systematically investigated the SII-PNI score's prognostic utility in SCAP populations. Given the pathophysiological parallels between cancer-associated inflammation and SCAP's hyperinflammatory phenotype, we hypothesize that this composite index may enhance early identification of high-risk patients. Our study aims to validate the SII-PNI score's predictive efficacy for 28-day mortality in SCAP, potentially establishing a novel clinician-friendly decision-support tool.

Materials and Methods

Study Design and Participants

One hundred and thirty-eight patients with SCAP admitted to the Affiliated People's Hospital of Ningbo University between January 2022 and December 2023 were enrolled in this study, and their clinical data were retrospectively analyzed. All participants provided signed informed consent, and the study was approved by the Ethics Committee of the Affiliated People's Hospital of Ningbo University [2024–006], adhering to the ethical guidelines of the Declaration of Helsinki.

The diagnosis of SCAP was based on the criteria outlined in the guidelines of the American Thoracic Society (ATS) and Infectious Diseases Society of America (IDSA)[29] at admission and were of age ≥ 18 years. Further, patients diagnosed with hospital acquired pneumonia, or aspiration pneumonia, were excluded from the study. Additionally, we did not include individuals with known HIV positivity, autoimmune connective tissue diseases, leukemia, myelodysplastic syndrome, lymphoma, or a history of immunosuppression (ie, recent use of immunosuppressive medications within 90 days, undergoing solid organ transplantation, and receiving ≥10 mg/day prednisolone or equivalent for at least 14 days).

Data Collection

A senior medical resident manually extracted the following data from the electronic medical record system: demographic characteristics, clinical characteristics and comorbidities, and results of routine laboratory tests. The laboratory tests included: lymphocyte, neutrophil, and platelet (PLT) counts, PCT, brain natriuretic peptide (BNP), CRP, blood urea nitrogen (BUN), creatinine (Cr), albumin, pH, partial pressure of oxygen in arterial blood (PaO_2), and partial pressure of carbon dioxide ($PaCO_2$). These data were used to determine the PNI and SII. The PNI was calculated using: PNI = serum albumin (g/L) + 5 × lymphocyte count (10^9/L).[14,15] On the other hand, the SII was defined as platelet counts (10^9/L) × neutrophil-to-lymphocyte ratio (SII = P×N/L ratio).[20]

All patients received standard care and antibiotic treatment as prescribed by the attending physician in accordance with guidelines.[29] Additionally, they were closely monitored and followed up for 28 days and their treatment outcomes were recorded. The all-cause 28-day mortality rate was documented for the study population. Those who survived for 28 days or more were classified as "survivors", while those who died within the 28-day follow-up were categorized as "non-survivors".

Statistical Analysis

All data analysis was performed using IBM SPSS Statistics 25.0 software. The normality of variables was assessed using the Shapiro–Wilk test. Continuous variables with a normal distribution were expressed as means ± standard deviations, while non-normally distributed variables were presented as medians with interquartile ranges (IQR). Categorical variables were analyzed using Chi-square test, along with the independent-samples t-test and Mann–Whitney U-test. Receiver operating characteristic (ROC) curve analysis was performed to establish the optimal cutoff value. Survival data were assessed by examining Kaplan–Meier plots. Factors associated with the 28-day mortality were identified through univariate and multivariate logistic regression analyses. A p value of less than 0.05 was considered statistically significant.

Results

Demographic Characteristics

After applying the inclusion and exclusion criteria, 138 patients with SCAP were included in the study. The demographic characteristics of these 138 patients are summarized in Table 1; 28 (20.29%) of the patients were females, and the median

Table 1 Baseline Characteristics of the Study Cohort Stratified by Their Final 28-Day Survival Status

	Survivors (n = 116)	Non-Survivors (n = 22)	Total (n = 138)	p
Median age, year	76.00 (69.25–83.00)	80.00 (69.00–88.00)	76.50 (69.75–84.00)	0.417
Female, (%)	24 (20.69%)	4 (18.18%)	28 (20.29%)	0.789
Respiratory rate	20.00 (19.00–22.00)	21.50 (20.00–25.50)	20.00 (19.75–23.00)	0.012
Body temperature, °C	37.05 (36.80–37.60)	37.50 (37.00–37.93)	37.10 (36.80–37.73)	0.097
SBP, mmHg	122.42 ± 22.96	118.50 ± 28.75	121.80 ± 23.90	0.482
DBP, mmHg	70.52 ± 11.55	67.86 ± 13.51	70.09 ± 11.87	0.338
Heart rate, per minute	93.22 ± 16.56	97.00 ± 17.86	90.82 ± 16.77	0.334
Comorbidities, (%)				
Coronary artery disease	15 (12.93%)	4 (18.18%)	19 (13.77%)	0.512
Heart failure	43 (37.07%)	15 (68.18%)	58 (42.03%)	0.007
Arrhythmia	15 (12.93%)	4 (18.18%)	19 (13.77%)	0.512
Diabetes	29 (25.00%)	6 (27.27%)	34 (24.64%)	0.822
Hypertension	48 (41.38%)	5 (22.73%)	53 (38.41%)	0.099
Renal diseases	20 (17.24%)	3 (13.64%)	23(16.67%)	0.677
Biochemistry				
Lymphocytes, $\times 10^9$ /L	0.73 (0.42–0.98)	0.35 (0.24–0.50)	0.66 (0.35–0.92)	<0.001
Neutrophils, $\times 10^9$ /L	6.75 (4.60–10.45)	10.21 (6.72–15.40)	7.16 (4.76–11.28)	0.005
Platelet, $\times 10^9$ /L	187.50 (139.75–251.00)	171.00 (125.50–318.75)	184.00 (138.25–257.25)	0.827
TP, g/L	59.10 ± 7.11	59.73 ± 7.85	59.20 ± 7.21	0.708
Albumin, g/L	30.65 ± 4.22	28.98 ± 3.03	30.38 ± 4.09	0.079
ALT, U/L	16.50 (11.00–32.75)	23.00 (13.75–41.00)	17.00 (11.00–33.25)	0.226
AST, U/L	24.00 (17.00–37.75)	38.00 (16.50–67.75)	24.50 (17.00–44.00)	0.175
Cr, μmol/L	69.50 (54.00–90.75)	86.50 (52.50–154.50)	70.00 (53.75–103.25)	0.123
BUN, mmol/L	7.44 (5.12–9.81)	9.56 (7.21–18.93)	7.87 (5.39–10.74)	0.006
Glucose, mmol/L	7.31 (5.63–9.23)	6.90 (5.26–13.04)	7.31 (5.59–9.50)	0.805
CRP, mg/L	115.60 (77.72–169.28)	144.72 (59.13–308.83)	116.00 (77.10–181.37)	0.286
PCT, ng/mL	0.40 (0.12–1.62)	1.09 (0.35–11.79)	0.52 (0.14–2.26)	0.006
BNP, pg/mL	143.50 (82.25–293.00)	264.50 (136.84–734.50)	152.00 (83.75–308.25)	0.006
Fibrinogen, g/L	7.32 (5.70–9.00)	8.03 (6.94–9.52)	7.51 (5.76–9.20)	0.307
D-dimer, mg/L	501.50 (330.00–1012.50)	703.00 (482.75–1156.00)	532.50 (340.00–1024.75)	0.222
pH	7.44 (7.40–7.47)	7.44 (7.35–7.47)	7.44 (7.40–7.50)	0.328
PaO2, mmHg	75.00 (62.00–92.50)	74.50 (64.00–91.00)	75.00 (62.00–91.00)	0.625

(*Continued*)

https://doi.org/10.2147/JIR.S521440

Table 1 (Continued).

	Survivors (n = 116)	Non-Survivors (n = 22)	Total (n = 138)	p
PaCO2, mmHg	37.50 (33.00–44.75)	36.50 (32.00–52.50)	37.00 (33.00)45.00	0.852
RFI	237.93 (180.69–300.00)	211.43 (180.50–227.50)	227.54 (182.07–281.40)	0.012
PNI	34.17 ± 4.82	31.01 ± 3.38	33.67 ± 4.75	0.004
SII	1808.23 (999.82–3671.82)	5553.56 (3020.22–8558.05)	2256.98 (1149.41–4369.38)	<0.001

Notes: Data following a normal distribution are shown as the mean ± SD, while those not normally distributed are depicted as median (IQR) and categorical variables are displayed as count (%).

Abbreviations: SBP, Systolic blood pressure; DBP, Diastole blood pressure; TP, Total protein; ALT, Alanine transaminase; AST, Aspartate transaminase; Cr, Creatinine; BUN, Blood urea nitrogen; CRP, C-reactive protein; PCT, Procalcitonin; BNP, Brain natriuretic peptide; PaO2, Partial pressure of oxygen in arterial blood; PaCO2, Partial pressure of carbon dioxide in arterial blood; RFI, Respiratory failure index; PNI, Prognostic nutritional index; SII, Systemic immune- inflammation index.

age of the patients was 76.5 years (range: 60.75–84.00). The 28-day mortality rate was 15.9% (22/138); accordingly, the patients were divided into 2 groups depending on their survival status after 28 days: "survivors" (n = 116) and "non-survivors" (n = 22). Statistically significant differences were observed between the two groups in respiratory rate, heart failure, respiratory failure index (RFI), neutrophil and lymphocyte counts, as well as levels of BUN, PCT, BNP, PNI, and SII ($p < 0.05$) (Table 1). However, there are no significant differences in body temperature, systolic blood pressure (SBP), diastolic blood pressure (DBP), CRP, D-dimer, PaO_2, or $PaCO_2$ (Table 1).

Associations Between PNI or SII and Prognosis

ROC curve analysis demonstrated that the area under curve (AUC) for SII and PNI in predicting to 28-day mortality were 0.822 (95% CI: 0.727–0.918; $p < 0.001$) and 0.684 (95% Cl: 0.575–0.792; $p = 0.006$), respectively (Figure 1). Optimal cutoff values for SII and PNI were determined to evaluate their predictive performance. A cutoff value of 4689.82 (Yoden Index: 0.61) in SII was associated with a sensitivity of 59.1% and a specificity of 85.3% in predicting 28-day mortality. Similarly, for PNI, a cutoff value of 32.18 (Yoden Index: 0.4) corresponded to a sensitivity of 60.3% and specificity of 68.2%, respectively (Figure 1).

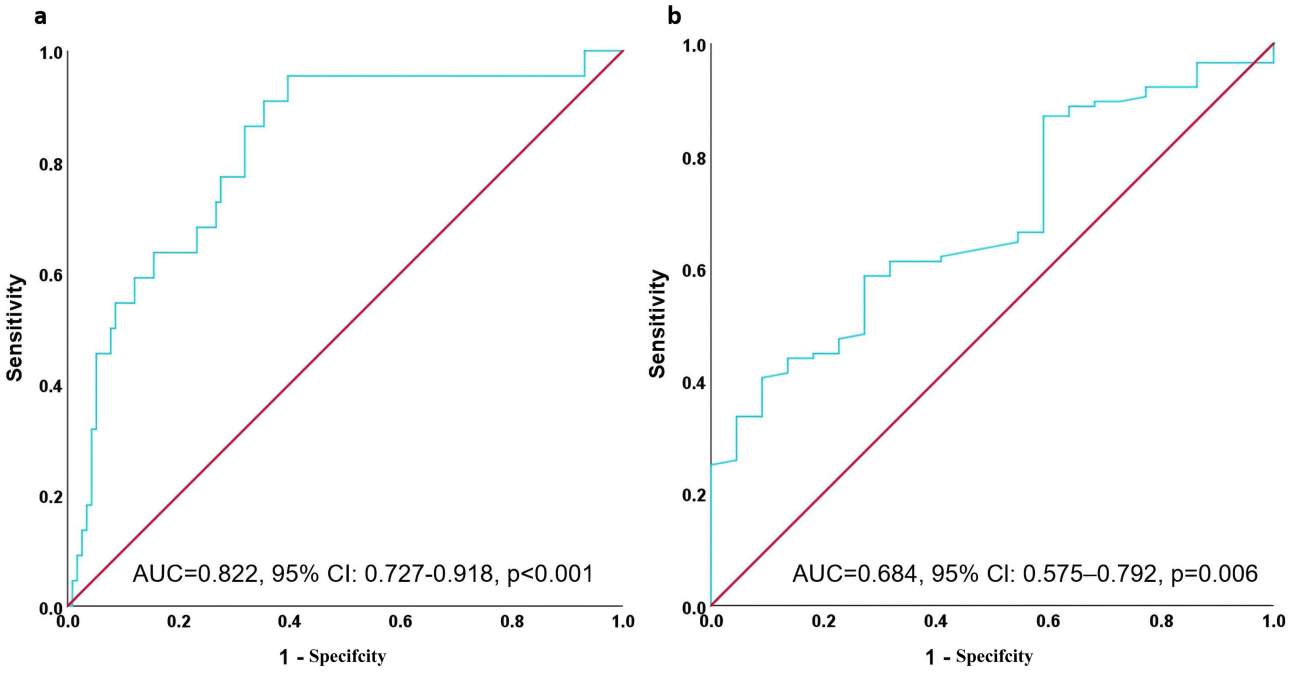

Figure 1 ROC analysis demonstrated the association between (**a**) SII or (**b**) PNI and 28-day mortality.

Survival Outcomes with Different Levels of the SII-PNI Score

Patients were stratified into three groups based on their SII-PNI score: a score of 2 (n = 24) indicating high SII (≥4689.82) and low PNI (≤32.18); a score of 1 (n = 43) indicating either low SII (<4689.82) or high PNI (>32.18); and a score of 0 (n = 71) indicating low SII (<4689.82) and high PNI (>32.18). Table 2 summarizes the baseline characteristics of the cohort stratified by the SII-PNI scores. During the 28-day follow-up, 22 patients died. The 28-day mortality rates for groups with SII-PNI scores of 0, 1, and 2 were 7.04%, 13.95%, and 45.83%, respectively ($p < 0.001$) (Figure 2).

Univariate and Multivariate Analysis for 28-Day Mortality

Univariate and multivariate logistic regression analyses were performed to evaluate factors associated with 28-day mortality. In the univariate analysis, the following factors showed significant associations with 28-day mortality: RFI (OR, 0.06; 95% CI, 0.01–0.47; p = 0.007), CRP (OR, 1.01; 95% CI, 1.00–1.01; p = 0.030), PCT (OR, 1.05; 95% CI, 1.013–1.08; p = 0.005), heart failure (OR, 3.64; 95% CI, 1.38–9.63; p = 0.009), and an SII-PNI score of 2 (OR, 11.17; 95% CI, 3.32–37.57; p < 0.001). Furthermore, the multivariate analysis showed that RFI (OR, 0.07; 95% CI, 0.01–0.55; p = 0.012), heart failure (OR, 4.06; 95% CI, 1.18–13.91; p < 0.05), and SII-PNI score of 2 (OR, 14.11; 95% CI, 3.18–62.66; p = 0.001) were independently associated with a higher risk of 28-day mortality (Table 3).

Table 2 Baseline Characteristics of the Study Cohort Stratified by SII-PNI Group

	SII-PNI = 0 (n = 71)	SII-PNI=1 (n = 43)	SII-PNI=2 (n = 24)	p
Median age (Range), year	79.00 (70.00–84.00)	75.00 (71.00–86.00)	73.00 (61.00–78.25)	0.053
Female, n (%)	18(25.35%)	6(13.95%)	4(16.57%)	0.303
Respiratory rate, per minute	20.97 ± 3.02	21.58 ± 2.90	23.08 ± 4.69	0.078
Body temperature, °C	37.00 (36.70–37.5)	37.20(36.80–38.10)	37.50(37.03–38.00)	0.019
SBP, mmHg	124.00(106.00–144.00)	117.00(105.00–139.00)	124.50(103.00–135.75)	0.728
DBP, mmHg	72.00(61.00–80.00)	67.00(60.00–77.00)	71.50(60.25–81.25)	0.583
Heart rate, per minute	90.00(81.00–100.00)	93.00(80.00–111.00)	97.00(89.25–109.00)	0.170
Comorbidities, n (%)				
Coronary artery disease	8(11.27%)	9(20.93%)	2(8.33%)	0.243
Heart failure	24(33.80%)	24(55.81%)	10(41.67%)	0.070
Arrhythmia	10(14.08%)	7(16.28%)	2(8.33%)	0.660
Diabetes	20(28.17%)	6(13.95%)	8(33.33%)	0.129
Hypertension	35(49.30%)	13(30.23%)	5(20.83%)	0.019
Renal diseases	12(16.90%)	7(16.28%)	4(16.67%)	0.996
Biochemistry				
BUN, mmol/L	8.45 ± 4.86	9.79 ± 6.35	9.35 ± 4.35	0.401
CRP, mg/L	107.00(76.60–153.63)	125.00(63.42–202.70)	153.79(77.83–258.54)	0.136
PCT, ng/mL	2.78 ± 9.92	6.59 ± 13.43	5.93 ± 14.70	0.012
BNP, pg/mL	224.15 ± 309.33	360.37 ± 383.81	402.04 ± 700.11	0.012
pH	7.44(7.40–7.47)	7.44(7.40–7.47)	7.44(7.38–7.47)	0.851
PaO2, mmHg	73.00(63.00–89.00)	76.00(61.00–91.00)	78.00(62.75–92.75)	0.826
PaCO2, mmHg	40.63 ± 13.36	40.53 ± 16.01	47.54 ± 19.90	0.289
RFI	235.00(193.10–280.95)	214.29(160.00–310.34)	221.53(184.21–264.36)	0.518

Notes: Data following a normal distribution are shown as the mean ± SD, while those not normally distributed are depicted as median (IQR) and categorical variables are displayed as count (%).

Abbreviations: SBP, Systolic blood pressure; DBP, Diastole blood pressure; TP, Total protein; BUN, Blood urea nitrogen; CRP, C-reactive protein; PCT, Procalcitonin; BNP, Brain natriuretic peptide; PaO2, Partial pressure of oxygen in arterial blood; PaCO2, Partial pressure of carbon dioxide in arterial blood; RFI, Respiratory failure index.

https://doi.org/10.2147/JIR.S521440

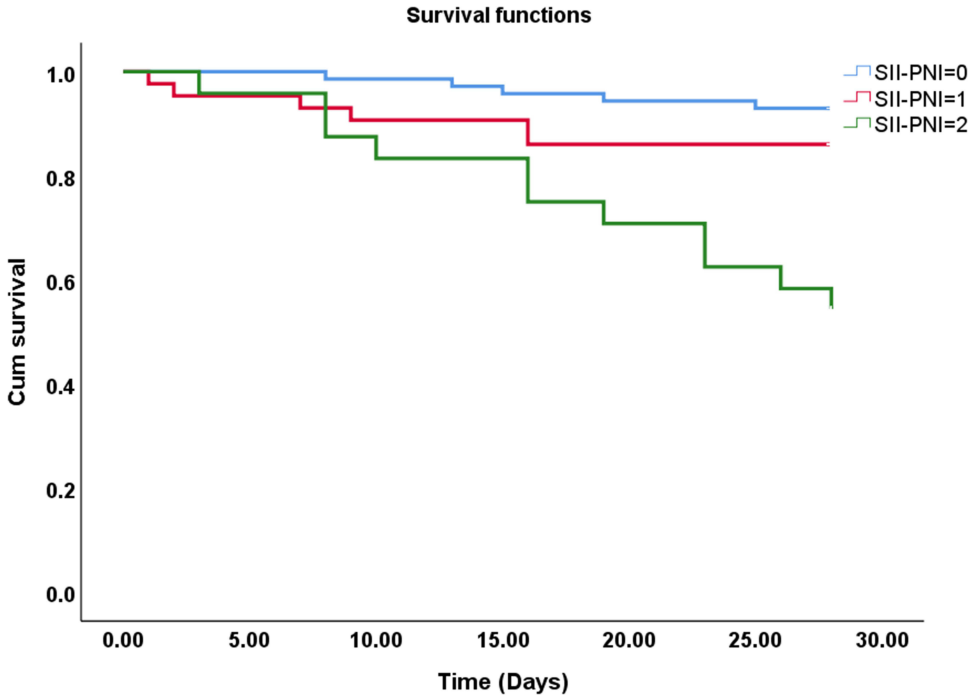

Figure 2 Kaplan-Meier survival curve according to SII-PNI score for 28-days.

Discussion

Traditional scoring systems, such as PSI and CURB-65, rely on static parameters like age and comorbidities, which cannot dynamically reflect patients' immune-inflammatory status or nutritional reserves. This limitation results in insufficient early identification of high-risk patients. SII and PNI, reflecting immune-nutritional status, show prognostic potential in a range of diseases.[21,30,31] The combined SII-PNI score improves cancer prognosis prediction, but its role in infections like SCAP remains unclear. This study evaluates SII-PNI's prognostic value in SCAP, integrating current evidence.

We determined optimal cutoff values for SII and PNI independently, then combined these parameters to create the novel SII-PNI score. Patients with a SII-PNI score of 2 exhibited a 28-day mortality rate of 45.83%, which was significantly higher than the rates observed in patients with scores of 0 or 1 ($p < 0.05$), indicating the prognostic validity

Table 3 Univariate and Multivariate Logistic Regression Analysis for the Prediction of 28-Day Mortality

Variable	Univariate			Multivariate		
	HR	95 CI %	p-value	HR	95 CI %	p-value
RFI						
>250	1.00	Reference		1.00	Reference	
≤250	0.06	0.01–0.47	0.007	0.07	0.01–0.55	0.012
CRP	1.01	1.00–1.01	0.030	1.00	1.00–1.01	0.374
PCT	1.05	1.01–1.08	0.005	1.04	1.00–1.09	0.041
Heart failure	3.64	1.38–9.63	0.009	4.06	1.18–13.91	0.026
SII-PNI score						
0	1.00	Reference		1.00	Reference	
1	2.14	0.61–7.50	0.234	1.65	0.40–6.76	0.484
2	11.17	3.32–37.57	<0.001	14.11	3.18–62.66	0.001

Abbreviations: RFI, Respiratory failure index; CRP, C-reactive protein; PCT, Procalcitonin; PNI, Prognostic nutritional index; SII, Systemic immune- inflammation index.

of SII-PNI in SCAP. To our knowledge, this is the first study to demonstrate that a higher SII-PNI score can serve as a reliable prognostic marker for SCAP. Further research in this regard may help identify targeted interventions aimed at reducing the mortality rates among patients with SCAP.

Hematological indices derived from white blood cell counts are useful in assessing inflammatory activity.[14,32] Prognostic markers based on multiple parameters such as lymphocyte, neutrophil, and platelet counts exhibit greater reliability than those relying on a single factor.[33] Elevated platelets and neutrophils indicate an excessive inflammatory response, whereas decreased lymphocytes reflect immune depletion; their combined measurement quantifies a state of "inflammatory-immunological imbalance".[34] Given that lymphocytes, neutrophils, and platelets play distinct roles in immune response, SII may better identify patients at higher risk of severe infections and appears to be a more comprehensive biomarker compared to NLR or platelet-lymphocyte ratio.[34,35] Furthermore, previous studies have demonstrated its utility as a biomarker in various inflammatory diseases, including coronary artery disease,[36] malignant tumors,[37,38] kidney stones,[39] and hypertension.[40] In patients with intracerebral hemorrhage, SII is correlated with stroke-associated pneumonia and has prognostic value.[41,42]

Nutritional status, immunity, and inflammation are closely interrelated.[43] Malnutrition in CAP patients will aggravate infection,[44] prolong hospitalization,[45] and increase mortality risk.[46–48] Adequate nutrition is essential for strengthening pulmonary infection defense[49] and has been linked to improved survival rates.[50] Appropriate nutritional interventions can mitigate oxidative stress and inflammation, thereby enhancing immune response and improving prognosis.[51,52] The PNI, first introduced by Buzby et al in 1980, was initially used to assess surgical risk and guide preoperative nutritional support in gastrointestinal surgery.[53] This index, calculated from serum albumin and total lymphocyte count, reflects both nutritional status and immune function. Studies suggest that hypoalbuminemia may result from malnutrition, malabsorption, comorbidities, aging, and pro-inflammatory cytokine-mediated suppression of albumin synthesis.[54] These factors may synergistically contribute to hypoalbuminemia in SCAP patients. Lymphocytes are involved in immune surveillance and immune modulation. Therefore, a decrease in lymphocyte count indicates impaired immune defense. Given the roles of lymphocytes and albumin in immune function and nutritional status, a low PNI is associated with poor prognosis.[16,23,54,55]

In our study, the AUC values for SII and PNI in predicting 28-day mortality were 0.822 (95% CI: 0.727–0.918; $p < 0.001$) and 0.684 (95% Cl: 0.575–0.792; $p = 0.006$), respectively. The overall mortality rate of SCAP patients was 15.94%, consistent with previous reports.[56] The SII-PNI score further stratified mortality risk: patients with an SII-PNI score of 0 had a mortality rate of 7.04%, whereas rates in those scoring 1 or 2 were significantly higher (13.95% and 45.83%, respectively; $p < 0.001$). These findings emphasize the potential utility of the combination of SII and PNI as a prognostic indicator in SCAP patients and may have implications for clinical decision-making and patient management strategies.

Our findings demonstrate that the SII-PNI score is a simple, cost-effective, and widely applicable prognostic biomarker for SCAP. Higher SII-PNI scores reflect elevated neutrophil-to-lymphocyte or platelet-to-lymphocyte ratios, both established predictors of poor CAP outcomes.[57] In addition, platelets are involved in regulating inflammatory responses and driving the activation of neutrophils, monocytes, and vascular endothelium. The elevated responsiveness of platelets is linked to an increased occurrence of myocardial damage and associated acute cardiovascular incidents in individuals with SCAP.[58] Second, an elevated SII-PNI score reflects a decreased lymphocyte count, suggesting a poor prognosis in CAP patients.[59] Third, low serum albumin at admission indicates malnutrition and impaired protein synthesis,[60] independently predicting 30-day mortality.[61] Early identification of high SII-PNI scores enables targeted interventions, including intensive monitoring, immunomodulatory therapies, and personalized nutritional support to improve cellular immunity and recovery. Future studies will validate this score in broader populations.

There are certain limitations to our study. First is the study design; since this is a single-center, retrospective study with a limited sample size. Multi-center, prospective studies with larger cohorts are required to validate the prognostic value of the SII-PNI score in SCAP patients. Secondly, the exclusion criteria might have omitted patient subgroups that could benefit from SII-PNI score assessment. Future research should enroll more diverse populations to explore additional clinical applications of this scoring system.

Conclusion

This study demonstrates that integrating SII and PNI provides a comprehensive evaluation of systemic inflammation and nutritional status in SCAP patients. Those with a SII-PNI score of 2 exhibit significantly higher 28-day mortality than scores 0–1. This scoring system may help clinicians rapidly assess patient severity at admission and initiate timely interventions.

Data Sharing Statement

The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported by the Health Technology Planning Project of Ningbo (Grant numbers No.2023Y59); and the Project of NINGBO Leading Medical & Health Discipline (Project Number: 2022-B19).

Disclosure

The authors declare no competing interests in this work.

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