Section: General Medicine

Original Research Article

ASSOCIATION BETWEEN THE SYSTEMIC IMMUNE INFLAMMATION INDEX AND DISEASE SEVERITY IN PATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA

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Received *: 17/09/2026*
Received in revised form *: 15/10/2026*
Accepted *: 27/10/2026*

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DOI: 10.70034/ijmedph.2026.4.224

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 1461-1468

ABSTRACT

Background: Community-acquired pneumonia (CAP) is a major cause of morbidity and mortality worldwide. Early identification of severe disease is essential for appropriate clinical management. The Systemic Immune-Inflammation Index (SII), derived from peripheral neutrophil, lymphocyte, and platelet counts, has emerged as a potential inflammatory marker for disease severity assessment. The aim is to evaluate the association between SII and disease severity in patients with community-acquired pneumonia and assess its diagnostic and prognostic significance.

Materials and Methods: This hospital-based, prospective observational analytical study included 116 adult patients with CAP, categorized into non-severe (n=72) and severe (n=44) groups according to ATS/IDSA criteria. Admission SII, inflammatory biomarkers, CURB-65 scores, clinical characteristics, and hospital outcomes were evaluated. Statistical analysis was performed using SPSS version 28.0. Correlation analysis, receiver operating characteristic (ROC) analysis, and multivariable logistic regression were performed, considering $p<0.05$ statistically significant.

Results: Patients with severe CAP were significantly older than those with non-severe CAP (66.48 ± 12.09 vs. 54.61 ± 13.14 years; $p<0.001$). Median SII was significantly higher in severe CAP (2,119 vs. 1,133; $p<0.001$). Higher SII correlated positively with CURB-65 score ($\rho=0.391$), C-reactive protein ($\rho=0.427$), and hospitalization duration ($\rho=0.312$), while showing a negative correlation with oxygen saturation ($\rho=-0.382$). ICU admission (61.36% vs. 11.11%) and in-hospital mortality (18.18% vs. 2.78%) were significantly higher in severe CAP. SII demonstrated good discriminatory performance (AUC=0.844), with 72.73% sensitivity and 84.72% specificity at a cut-off of 1,774.32. SII remained independently associated with severe CAP (aOR=3.18; $p<0.001$).

Conclusion: Elevated SII was significantly associated with CAP severity and adverse clinical outcomes. It may serve as a simple, inexpensive, and readily available supplementary marker for early risk stratification in patients with CAP.

Keywords: Community-acquired pneumonia, Systemic Immune-Inflammation Index, Disease severity, Inflammatory biomarkers, CURB-65.

INTRODUCTION

Community-acquired pneumonia (CAP) is an acute infection of the pulmonary parenchyma acquired outside healthcare facilities and remains an important cause of preventable illness, hospitalization, and death worldwide. Older adults and individuals with chronic medical conditions are particularly vulnerable. Its burden extends beyond respiratory illness to functional impairment and systemic complications. Host factors, pathogen exposure, and healthcare access influence prognosis. Symptoms may be initially nonspecific, complicating recognition of patients at greater risk of early clinical

deterioration. Reliable early risk assessment is therefore essential for appropriate triage and timely treatment in hospital settings.[1] The pathogenesis of CAP involves interactions among invading microorganisms, pulmonary host defenses, and systemic immune responses. Bacteria, respiratory viruses, and atypical pathogens may reach the lungs through aspiration or inhalation. Alveolar macrophages, neutrophils, epithelial cells, and circulating immune mediators cooperate to eliminate pathogens, but an excessive or poorly regulated inflammatory response can injure the alveolar-capillary membrane. Increased vascular permeability, inflammatory exudation, and impaired gas exchange may produce hypoxaemia and respiratory distress. In susceptible patients, local pulmonary inflammation can progress to widespread immune dysregulation, endothelial dysfunction, circulatory disturbances, and organ injury. These processes make blood-based inflammatory measurements potentially relevant to severity assessment.[2] Assessment of CAP severity is a fundamental component of initial clinical evaluation because treatment requirements vary considerably among affected patients. Individuals with less extensive disease may respond to conventional antimicrobial therapy and supportive care, whereas severe cases can require intensive monitoring, respiratory support, and management of septic shock. CURB-65 and the Pneumonia Severity Index estimate risk, while ATS/IDSA criteria identify severe CAP using major and minor clinical features. Nevertheless, clinical scoring systems depend on several physiological and laboratory variables and should be interpreted alongside bedside assessment. Accessible indicators may support timely escalation decisions.[3] Laboratory biomarkers provide a complementary approach to evaluating the inflammatory response in CAP. Commonly assessed parameters include total leukocyte count, differential leukocyte counts, C-reactive protein, and procalcitonin. Their interpretation is affected by pathogen, illness duration, comorbidities, and treatment. Their concentrations may also overlap between different levels of disease severity, limiting the usefulness of any single marker. An ideal supplementary marker should be inexpensive, rapidly available, reproducible, and obtainable during routine admission investigations. Such indices should complement clinical assessment, imaging findings, and existing prognostic tools rather than replace them. Blood count-derived indices are therefore attractive because they utilize information already generated through standard hematological testing without requiring additional specialized assays.[4] The inflammatory response in pneumonia involves dynamic changes in neutrophils, lymphocytes, and platelets. Neutrophils contribute to antimicrobial defense through phagocytosis and inflammatory mediator release, but excessive activation may intensify pulmonary tissue damage. Lymphocytes participate in adaptive immune regulation, and a reduction in circulating lymphocyte counts may reflect physiological stress or immune dysfunction during serious infection. Platelets interact with leukocytes and endothelial cells in inflammatory and thrombotic pathways. Accordingly, combined cellular measures may capture complementary aspects of infection-associated inflammation. Lymphocyte abnormalities therefore support considering composite indices rather than total leukocyte counts alone.[5] The Systemic Immune-Inflammation Index (SII) integrates three readily available hematological components and is calculated by multiplying the platelet count by the absolute neutrophil count and dividing the product by the absolute lymphocyte count. A higher SII may reflect neutrophil-predominant inflammation, platelet involvement, and relative lymphocyte depletion. Unlike isolated cell counts, this composite index is designed to represent the balance between inflammatory activation and immune regulation. Other related measures, including the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio, have also been explored as adjuncts to conventional pneumonia assessment. Because SII requires only a routine blood count, it may support risk stratification where specialized testing is unavailable.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, prospective observational analytical study to evaluate the association between the Systemic Immune-Inflammation Index (SII) and disease severity among patients diagnosed with community-acquired pneumonia (CAP). The study aimed to assess the relationship of SII with clinical presentation, inflammatory biomarkers, pneumonia severity scores, respiratory dysfunction, complications, and clinical outcomes. Additionally, the diagnostic performance of SII in identifying severe CAP was evaluated. The study was conducted at a tertiary care hospital in collaboration with the Departments of General Medicine, Pulmonary Medicine, Emergency Medicine, and Biochemistry. Patients presenting to the outpatient department, emergency department, or requiring hospital admission with a diagnosis of community-acquired pneumonia were assessed for eligibility. A total of 116 adult patients diagnosed with community-acquired pneumonia were included using a consecutive sampling technique. Patients were categorized into two groups according to disease severity: Group A comprised patients with non-severe CAP, while Group B comprised patients with severe CAP. Disease severity was classified according to the American Thoracic Society/Infectious Diseases Society of America (ATS/IDSA) criteria. The Systemic Immune-Inflammation Index was calculated and compared between the groups to determine its association with pneumonia severity.

Inclusion Criteria

Patients aged 18 years and above with clinically and radiologically confirmed community-acquired pneumonia were eligible for inclusion. CAP was diagnosed based on compatible respiratory symptoms, including fever, cough, sputum production, dyspnoea, or pleuritic chest pain, along with newly developed pulmonary infiltrates on chest radiography or computed tomography. Patients requiring hospitalization for CAP and providing informed consent were included.

Exclusion Criteria

Patients with hospital-acquired or ventilator-associated pneumonia, active pulmonary tuberculosis, active malignancy, haematological disorders, autoimmune diseases, or chronic inflammatory conditions were excluded. Patients receiving immunosuppressive therapy, chemotherapy, or prolonged systemic corticosteroid treatment were also excluded. Additionally, patients with recent major surgery, significant trauma, or conditions substantially affecting peripheral blood cell counts were excluded to minimize interference with SII measurements.

Methodology

Detailed demographic and clinical information was recorded using a structured proforma. Parameters included age, gender, body mass index, smoking history, alcohol consumption, duration of respiratory symptoms, and associated comorbidities such as diabetes mellitus, hypertension, chronic obstructive pulmonary disease, cardiovascular disease, and chronic kidney disease. Clinical parameters included body temperature, heart rate, respiratory rate, systolic and diastolic blood pressure, peripheral oxygen saturation (SpO_2), altered mental status, and requirement for supplemental oxygen. Relevant chest radiographic findings, including unilateral or bilateral infiltrates, multilobar involvement, and pleural effusion, were documented.

Laboratory Investigations

Peripheral venous blood samples were collected at admission, preferably before initiation of systemic corticosteroids. Complete blood count was performed using an automated haematology analyser to determine total leukocyte count, absolute neutrophil count, absolute lymphocyte count, platelet count, haemoglobin concentration, and neutrophil-to-lymphocyte ratio (NLR). Additional investigations included C-reactive protein (CRP), procalcitonin (where available), serum electrolytes, blood urea nitrogen, serum creatinine, blood glucose, serum albumin, and liver function tests. Arterial blood gas analysis and PaO_2 /FiO_2 ratio were assessed when clinically indicated. Sputum examination, sputum culture, and blood cultures were performed according to clinical requirements.

Calculation of Systemic Immune-Inflammation Index

The Systemic Immune-Inflammation Index was calculated from absolute peripheral blood cell counts using the following formula:

SII = Platelet Count × Absolute Neutrophil Count / Absolute Lymphocyte Count

All three cell counts were expressed in $\times 10^9$ /L. The neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio were also calculated for comparative assessment. Admission SII values were analysed as continuous variables, and their relationship with clinical severity, inflammatory biomarkers, and hospital outcomes was assessed.

Assessment of Pneumonia Severity

Disease severity was evaluated using CURB-65 and ATS/IDSA severity criteria. CURB-65 included confusion, blood urea nitrogen, respiratory rate, blood pressure, and age ≥65 years. Severe CAP was defined by the presence of at least one major ATS/IDSA criterion or three or more minor criteria. Major criteria included invasive mechanical ventilation or septic shock requiring vasopressors. Patients not fulfilling the severe CAP criteria were classified as having non-severe CAP. The association of SII with severity scores, hypoxaemia, and radiological extent of pneumonia was evaluated.

Assessment of Clinical Outcomes

Patients were assessed for intensive care unit admission, requirement for non-invasive or invasive mechanical ventilation, development of acute respiratory failure, sepsis, septic shock, acute kidney injury, length of hospital stay, and in-hospital mortality. Relationships between admission SII values and these clinical outcomes were examined to evaluate its potential prognostic significance.

Statistical Analysis

Statistical analysis was performed using **IBM SPSS Statistics version 28.0**. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution assessed using the Shapiro–Wilk test. Categorical variables were presented as frequencies and percentages. The independent samples t-test or Mann–Whitney U test was used for continuous variables, while the Chi-square test or Fisher's exact test was applied for categorical variables. Spearman's correlation analysis assessed associations between SII and clinical severity parameters. Receiver operating characteristic (ROC) curve analysis determined the area under the curve (AUC), optimal SII cut-off, sensitivity, and specificity for identifying severe CAP. Multivariable logistic regression was used to assess the independent association of SII with severe CAP after adjustment for potential confounders. A **p-value <0.05** was considered statistically significant.

RESULTS

[Table 1] Baseline Demographic and Clinical Characteristics According to CAP Severity (N=116)

A total of 116 patients with community-acquired pneumonia were included in the study, of whom 72 (62.07%) had non-severe CAP and 44 (37.93%) had

severe CAP. The mean age was significantly higher among patients with severe CAP (66.48 ± 12.09 years) compared with non-severe CAP (54.61 ± 13.14 years; p<0.001). Hypertension was significantly more prevalent in the severe group (54.55% vs. 25.00%; p=0.003), while diabetes mellitus was also more frequent (43.18% vs. 25.00%; p=0.064), although the difference was not statistically significant. Patients with severe CAP had a longer duration of symptoms (5.64 ± 1.51 vs. 4.62 ± 1.89 days; p=0.002), higher respiratory rates (30.40 ± 4.30 vs. 23.80 ± 3.60 breaths/min; p<0.001), and lower admission SpO_2 (86.10 ± 5.70% vs. 93.40 ± 2.80%; p<0.001). Dyspnoea (90.91% vs. 75.00%; p=0.049), altered mental status (36.36% vs. 6.94%; p<0.001), and bilateral or multilobar infiltrates (56.82% vs. 22.22%; p<0.001) were significantly more frequent among severe CAP patients.

[Table 2] Admission Inflammatory and Laboratory Markers in Non-Severe and Severe CAP

Laboratory investigations demonstrated significantly greater inflammatory activity among patients with severe CAP compared with those having non-severe CAP. The median total leukocyte count (12.55 vs. 10.30 $\times 10^9$ /L; p<0.001) and absolute neutrophil count (10.22 vs. 7.83 $\times 10^9$ /L; p<0.001) were significantly higher in severe CAP, whereas the absolute lymphocyte count was significantly lower (1.42 vs. 1.80 $\times 10^9$ /L; p=0.014). The median Systemic Immune-Inflammation Index was markedly elevated in severe CAP patients (2,119; IQR: 1,606–2,841) compared with non-severe patients (1,133; IQR: 878–1,533; p<0.001). Similarly, the neutrophil-to-lymphocyte ratio (7.27 vs. 4.45; p<0.001), platelet-to-lymphocyte ratio (200.87 vs. 151.69; p=0.002), and platelet count (302.67 vs. 271.37 $\times 10^9$ /L; p=0.029) were significantly higher in severe cases. Inflammatory biomarkers, including C-reactive protein (110.60 vs. 40.76 mg/L; p<0.001) and procalcitonin (0.98 vs. 0.37 ng/mL; p<0.001), also showed substantial elevations in severe CAP. Furthermore, blood urea nitrogen was significantly increased in severe patients (49.66 vs. 27.90 mg/dL; p<0.001), indicating greater renal or systemic physiological disturbance associated with disease severity.

[Table 3] Clinical Severity Categories and In-Hospital Outcomes by CAP Group

Analysis of clinical severity and hospital outcomes revealed significantly greater morbidity and mortality among patients with severe CAP compared with non-severe CAP. A CURB-65 score of 0–1 was observed in 66.67% of non-severe patients compared with only 6.82% of severe patients, whereas a score ≥3 was considerably more frequent in severe CAP (65.91% vs. 5.56%; overall p<0.001). Intensive care unit admission was required in 61.36% of severe cases compared with 11.11% of non-severe cases (p<0.001). Similarly, non-invasive ventilation was required in 38.64% versus 9.72% (p<0.001), while invasive mechanical ventilation (20.45%) and septic shock requiring vasopressors (15.91%) occurred exclusively in the severe CAP group. Acute kidney injury developed significantly more frequently among severe patients (29.55% vs. 4.17%; p<0.001). In-hospital mortality was substantially higher in severe CAP (18.18%) compared with non-severe CAP (2.78%; p=0.006), while the mean duration of hospitalization was significantly prolonged (9.86 ± 3.05 vs. 6.40 ± 2.10 days; p<0.001), demonstrating the greater clinical burden associated with severe pneumonia.

[Table 4] Spearman Correlation Between Admission SII and Clinical or Biochemical Parameters (N=116)

Spearman correlation analysis demonstrated significant associations between admission Systemic Immune-Inflammation Index and multiple indicators of pneumonia severity. A statistically significant positive correlation was observed between SII and CURB-65 score (ρ=+0.391; p<0.001), indicating that higher SII values were associated with greater clinical severity. SII also showed a significant positive correlation with respiratory rate (ρ=+0.376; p<0.001), whereas a significant negative correlation was observed with admission SpO_2 (ρ=−0.382; p<0.001), suggesting an association between elevated systemic inflammation and impaired oxygenation. Among biochemical parameters, C-reactive protein demonstrated the strongest positive correlation with SII (ρ=+0.427; p<0.001), while blood urea nitrogen showed a weaker but statistically significant positive correlation (ρ=+0.221; p=0.017). Additionally, SII was positively correlated with the duration of hospital stay (ρ=+0.312; p<0.001), indicating that patients with elevated admission SII tended to require longer hospitalization.

[Table 5] ROC Diagnostic Performance and Multivariable Logistic Regression for Severe CAP

Receiver operating characteristic curve analysis demonstrated good discriminatory performance of the Systemic Immune-Inflammation Index for identifying severe community-acquired pneumonia, with an area under the curve (AUC) of 0.844 (95% CI: 0.768–0.908; p<0.001). At an optimal cut-off value of 1,774.32, SII demonstrated a sensitivity of 72.73% and specificity of 84.72%. The neutrophil-to-lymphocyte ratio showed comparable diagnostic performance, with an AUC of 0.843 (95% CI: 0.771–0.907), sensitivity of 84.09%, and specificity of 72.22% at a cut-off of 5.19. However, C-reactive protein exhibited the highest numerical discriminatory ability, with an AUC of 0.937 (95% CI: 0.887–0.975), sensitivity of 84.09%, and specificity of 91.67% at a threshold of 73.26 mg/L. Multivariable logistic regression demonstrated that SII remained significantly associated with severe CAP after adjustment for age and hypertension, with each 500-unit increase in SII corresponding to an adjusted odds ratio of 3.18 (95% CI: 2.01–5.02; p<0.001). Increasing age was also independently associated with severe CAP (aOR=1.09 per year; 95% CI: 1.04–1.14; p<0.001), whereas hypertension

showed a positive but statistically non-significant association (aOR=2.71; 95% CI: 0.90–8.14; p=0.076).

Table 1: Baseline demographic and clinical characteristics according to CAP severity (N = 116)

Parameter	Non-severe CAP (n=72)	Severe CAP (n=44)	p-value
Age (years), mean ± SD	54.61 ± 13.14	66.48 ± 12.09	<0.001
Male sex	44 (61.11%)	29 (65.91%)	0.693
Current/former smoking	26 (36.11%)	20 (45.45%)	0.335
Diabetes mellitus	18 (25.00%)	19 (43.18%)	0.064
Hypertension	18 (25.00%)	24 (54.55%)	0.003
Chronic obstructive pulmonary disease	14 (19.44%)	14 (31.82%)	0.179
Duration of symptoms (days), mean ± SD	4.62 ± 1.89	5.64 ± 1.51	0.002
Dyspnoea at presentation	54 (75.00%)	40 (90.91%)	0.049
Altered mental status	5 (6.94%)	16 (36.36%)	<0.001
Respiratory rate (breaths/min), mean ± SD	23.80 ± 3.60	30.40 ± 4.30	<0.001
SpO_2 at admission (%), mean ± SD	93.40 ± 2.80	86.10 ± 5.70	<0.001
Bilateral/multilobar infiltrates	16 (22.22%)	25 (56.82%)	<0.001

Table 2: Admission inflammatory and laboratory markers in non-severe and severe CAP

Laboratory parameter	Non-severe CAP (n=72)	Severe CAP (n=44)	p-value
Total leukocyte count ($\times 10^9$ /L)	10.30 (9.32–11.89)	12.55 (10.69–14.40)	<0.001
Absolute neutrophil count ($\times 10^9$ /L)	7.83 (6.81–8.70)	10.22 (8.60–12.02)	<0.001
Absolute lymphocyte count ($\times 10^9$ /L)	1.80 (1.29–2.36)	1.42 (1.04–1.92)	0.014
Platelet count ($\times 10^9$ /L)	271.37 (224.36–303.90)	302.67 (254.19–335.44)	0.029
SII (platelets × neutrophils / lymphocytes)	1,133 (878–1,533)	2,119 (1,606–2,841)	<0.001
Neutrophil-to-lymphocyte ratio	4.45 (3.51–5.48)	7.27 (6.03–9.01)	<0.001
Platelet-to-lymphocyte ratio	151.69 (106.42–205.97)	200.87 (146.05–295.42)	0.002
C-reactive protein (mg/L)	40.76 (32.12–50.39)	110.60 (80.07–152.45)	<0.001
Procalcitonin (ng/mL)	0.37 (0.26–0.67)	0.98 (0.71–1.40)	<0.001
Blood urea nitrogen (mg/dL)	27.90 (23.04–33.42)	49.66 (35.69–57.35)	<0.001

Table 3: Clinical severity categories and in-hospital outcomes by CAP group

Clinical parameter / outcome	Non-severe CAP (n=72)	Severe CAP (n=44)	p-value
CURB-65 score 0–1	48 (66.67%)	3 (6.82%)	<0.001
CURB-65 score 2	20 (27.78%)	12 (27.27%)	—
CURB-65 score ≥3	4 (5.56%)	29 (65.91%)	—
ICU admission	8 (11.11%)	27 (61.36%)	<0.001
Non-invasive ventilation	7 (9.72%)	17 (38.64%)	<0.001
Invasive mechanical ventilation	0 (0.00%)	9 (20.45%)	<0.001
Septic shock requiring vasopressors	0 (0.00%)	7 (15.91%)	<0.001
Acute kidney injury	3 (4.17%)	13 (29.55%)	<0.001
In-hospital mortality	2 (2.78%)	8 (18.18%)	0.006
Hospital stay (days), mean ± SD	6.40 ± 2.10	9.86 ± 3.05	<0.001

Table 4: Spearman correlation between admission SII and clinical or biochemical parameters (N = 116)

Variable	Spearman's ρ	p-value
CURB-65 score	+0.391	<0.001
Respiratory rate (breaths/min)	+0.376	<0.001
Admission SpO_2 (%)	-0.382	<0.001
C-reactive protein (mg/L)	+0.427	<0.001
Blood urea nitrogen (mg/dL)	+0.221	0.017
Duration of hospital stay (days)	+0.312	<0.001

Table 5: ROC diagnostic performance and multivariable logistic regression for severe CAP

Analysis	Marker / predictor	Estimate (95% CI)	Additional details	p-value
ROC analysis	SII	AUC 0.844 (0.768–0.908)	Threshold 1,774.32; sensitivity 72.73%; specificity 84.72%	<0.001
ROC analysis	NLR	AUC 0.843 (0.771–0.907)	Threshold 5.19; sensitivity 84.09%; specificity 72.22%	<0.001
ROC analysis	CRP	AUC 0.937 (0.887–0.975)	Threshold 73.26; sensitivity 84.09%; specificity 91.67%	<0.001
Adjusted logistic model	SII (per +500 units)	aOR 3.18 (2.01–5.02)	Adjusted for other listed predictors	<0.001
Adjusted logistic model	Age (per year)	aOR 1.09 (1.04–1.14)	Adjusted for other listed predictors	<0.001
Adjusted logistic model	Hypertension (yes vs no)	aOR 2.71 (0.90–8.14)	Adjusted for other listed predictors	0.076

DISCUSSION

The present study evaluated 116 patients with community-acquired pneumonia (CAP), comprising 72 (62.07%) patients with non-severe CAP and 44 (37.93%) with severe CAP. The mean age was significantly higher in severe CAP patients (66.48 ± 12.09 years) compared with non-severe patients (54.61 ± 13.14 years; p<0.001). Hypertension was also significantly more frequent in severe CAP (54.55% vs. 25.00%; p=0.003), while diabetes mellitus showed a higher prevalence without statistical significance (43.18% vs. 25.00%; p=0.064). These findings are consistent with Kothe et al. (2008), who conducted a prospective multicentre study involving 2,647 CAP patients, including 1,349 patients aged ≥65 years and 1,298 younger patients. They reported an overall 30-day mortality of 6.3%, with mortality increasing to 10.3% among elderly patients. Advanced age remained an independent predictor of mortality even after adjustment for comorbidities and disease severity. Their observations support the present findings that increasing age and underlying clinical conditions are important factors associated with adverse pneumonia severity and prognosis.[7] The present study demonstrated significantly greater respiratory compromise among patients with severe CAP, with respiratory rates of 30.40 ± 4.30 versus 23.80 ± 3.60 breaths/min (p<0.001) and admission SpO_2 values of 86.10 ± 5.70% versus 93.40 ± 2.80% (p<0.001). Altered mental status was observed in 36.36% of severe cases compared with 6.94% of non-severe cases, while bilateral or multilobar infiltrates were present in 56.82% versus 22.22%, respectively (both p<0.001). Similar observations were reported by España et al. (2006), who developed a clinical prediction rule using 1,057 CAP patients and validated it in two additional cohorts comprising 719 and 1,121 patients. Their analysis identified eight independent predictors of severe pneumonia, including respiratory rate greater than 30 breaths/min, altered mental status, blood urea nitrogen greater than 30 mg/dL, severe hypoxaemia, advanced age, and bilateral or multilobar pulmonary involvement. The prediction model achieved an AUC of 0.92, demonstrating excellent discriminatory performance. These findings correspond with the present observations and emphasize the importance of respiratory dysfunction, neurological abnormalities, and radiological involvement in identifying severe CAP.[8] The present study demonstrated significant alterations in peripheral blood inflammatory indices among patients with severe CAP, particularly increased neutrophil counts and reduced lymphocyte counts. The median neutrophil count was significantly higher in severe CAP (10.22 vs. 7.83 $\times 10^9$ /L; p<0.001), whereas the lymphocyte count was lower (1.42 vs. 1.80 $\times 10^9$ /L; p=0.014). Consequently, the median neutrophil-to-lymphocyte ratio was significantly elevated in severe CAP (7.27 vs. 4.45; p<0.001), accompanied by a markedly increased SII (2,119 vs. 1,133; p<0.001). Comparable findings were reported by de Jager et al. (2012) in a prospective study involving 395 patients with CAP. Their study demonstrated significantly higher neutrophil-to-lymphocyte count ratios among non-survivors (23.3 ± 16.8) compared with survivors (13.0 ± 11.4). The NLR also increased progressively with CURB-65 severity scores and demonstrated an AUC of 0.701 for predicting mortality, compared with 0.565 for CRP and 0.672 for total leukocyte count. Although their study evaluated mortality rather than severe CAP classification, these observations support the prognostic relevance of neutrophil predominance and relative lymphopenia. The present findings further suggest that incorporating platelet counts into the SII may provide additional information regarding systemic inflammatory activity.[9] The present study also demonstrated significantly elevated inflammatory biomarkers among patients with severe CAP, including median C-reactive protein concentrations of 110.60 mg/L compared with 40.76 mg/L in non-severe CAP (p<0.001). Similarly, median procalcitonin levels were significantly higher in severe patients (0.98 vs. 0.37 ng/mL; p<0.001), while blood urea nitrogen was markedly increased (49.66 vs. 27.90 mg/dL; p<0.001). These findings are supported by Curbelo et al. (2017), who prospectively evaluated 154 hospitalized CAP patients and reported 30-day and 90-day mortality rates of 7.79% and 12.99%, respectively. They observed higher admission CRP concentrations among patients who died within 30 days (18.8 vs. 13.4 mg/dL), along with higher procalcitonin levels (10.8 vs. 1.4 ng/mL) and NLR values (16.8 vs. 10.5). Their ROC analysis demonstrated AUC values of 0.70 for CRP, 0.70 for procalcitonin, and 0.76 for NLR in predicting 30-day mortality. These results are broadly consistent with the elevated inflammatory markers observed in the severe CAP group of the present study, emphasizing the association between systemic inflammatory responses and adverse pneumonia outcomes.[10] The present study revealed a strong association between clinical severity classification and adverse hospital outcomes. A CURB-65 score ≥3 was observed in 65.91% of severe CAP patients compared with only 5.56% of non-severe patients (p<0.001). Intensive care unit admission was considerably more frequent in severe CAP (61.36% vs. 11.11%), while non-invasive ventilation was required in 38.64% versus 9.72%, respectively (both p<0.001). In-hospital mortality was also substantially higher among severe CAP patients (18.18% vs. 2.78%; p=0.006), accompanied by a longer mean hospital stay (9.86 ± 3.05 vs. 6.40 ± 2.10 days; p<0.001). These findings are comparable with those reported by Capelastegui et al. (2006), who evaluated 1,776 CAP patients, comprising 1,100 inpatients and 676 outpatients. Their study demonstrated a progressive increase in 30-day mortality with increasing CURB-65 scores,

with mortality rates of 0%, 1.1%, 7.6%, 21.0%, 41.9%, and 60.0% for scores of 0, 1, 2, 3, 4, and 5, respectively. They also reported significant associations between higher CURB-65 scores, mechanical ventilation requirements, and hospitalization duration. These observations support the present findings that increasing pneumonia severity is associated with greater resource utilization, prolonged hospitalization, and increased mortality.[11] The present study demonstrated a significantly greater frequency of major complications in patients with severe CAP, including acute kidney injury in 29.55% compared with 4.17% of non-severe patients ($p<0.001$). Invasive mechanical ventilation was required in 20.45% of severe CAP patients, while septic shock requiring vasopressors occurred in 15.91%. Furthermore, the severe CAP group exhibited an in-hospital mortality rate of 18.18%, indicating a considerable burden of organ dysfunction and adverse outcomes. Comparable observations were reported by Li et al. (2025) in a retrospective study involving 613 elderly patients with CAP. Their investigation reported an overall in-hospital mortality rate of approximately 48%, with mortality increasing from 35.6% among patients aged 60–70 years to 65.3% among those older than 80 years. Multivariable analysis identified acute kidney injury (OR=2.98; 95% CI: 1.77–5.01), acute respiratory distress syndrome (OR=4.00; 95% CI: 2.17–7.37), and increasing age (OR=1.05; 95% CI: 1.02–1.07) as significant predictors of in-hospital mortality. Although mortality was higher in their elderly cohort, their findings reinforce the present observations that renal impairment, respiratory failure, and advanced age are associated with unfavourable CAP outcomes.[12] The present study identified significant correlations between admission SII and multiple clinical and biochemical indicators of CAP severity. SII demonstrated positive correlations with CURB-65 score ($\rho=+0.391$; $p<0.001$), respiratory rate ($\rho=+0.376$; $p<0.001$), CRP ($\rho=+0.427$; $p<0.001$), and blood urea nitrogen ($\rho=+0.221$; $p=0.017$). Conversely, admission SpO_2 exhibited a significant negative correlation with SII ($\rho=-0.382$; $p<0.001$), while hospitalization duration showed a positive association ($\rho=+0.312$; $p<0.001$). Similar findings were reported by Terzi et al. (2025), who retrospectively evaluated 240 hospitalized patients with CAP and investigated systemic inflammatory indices in relation to conventional biomarkers and pneumonia severity scores. Their study demonstrated significant positive correlations between SII and CRP ($\rho=0.275$; $p<0.001$), procalcitonin ($\rho=0.209$; $p=0.001$), CURB-65 score ($\rho=0.216$), and Pneumonia Severity Index ($\rho=0.199$). The reported 30-day mortality rate was 15.4%, with significantly higher SII values among non-survivors ($p=0.043$). Compared with their study, the present findings demonstrate stronger numerical correlations between SII and both CRP and CURB-65 score. These observations suggest that SII reflects systemic inflammatory activity and may complement established clinical severity assessment tools.[13] The present study demonstrated good diagnostic performance of SII for identifying severe CAP, with an AUC of 0.844 (95% CI: 0.768–0.908; $p<0.001$). At a cut-off value of 1,774.32, SII achieved a sensitivity of 72.73% and specificity of 84.72%. The diagnostic performance of SII was comparable to that of NLR (AUC=0.843), whereas CRP demonstrated a numerically higher AUC of 0.937, with sensitivity and specificity of 84.09% and 91.67%, respectively. Multivariable logistic regression further demonstrated a significant association between SII and severe CAP (aOR=3.18 per 500-unit increase; 95% CI: 2.01–5.02; $p<0.001$), after adjustment for age and hypertension. These findings can be compared with Chen et al. (2025), who evaluated 138 patients with severe CAP and investigated the prognostic performance of SII combined with the Prognostic Nutritional Index. They reported an SII AUC of 0.822 (95% CI: 0.727–0.918) for predicting 28-day mortality, with an optimal SII threshold greater than 4,689.82, sensitivity of 59.1%, and specificity of 85.3%. Their combined SII-PNI score of 2 was independently associated with increased mortality (OR=14.11; 95% CI: 3.18–62.66; $p=0.001$), with 28-day mortality reaching 45.83% in the highest-risk group compared with 7.04% in the lowest-risk group. Although the two studies assessed different outcomes, their findings collectively support the potential utility of SII as an accessible supplementary marker for CAP risk stratification.[14]

CONCLUSION

The present study demonstrated a significant association between elevated Systemic Immune-Inflammation Index (SII) and increased disease severity in patients with community-acquired pneumonia. Higher SII values were associated with greater inflammatory activity, respiratory dysfunction, prolonged hospitalization, and adverse clinical outcomes. SII demonstrated good diagnostic performance in identifying severe CAP and remained significantly associated with disease severity after adjustment for age and hypertension. Being a simple, inexpensive, and readily available hematological marker, SII may serve as a useful supplementary tool for early risk stratification and clinical assessment of patients with CAP. Further prospective studies are required to validate its prognostic utility and optimal cut-off values.

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