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Research article

Prognostic value of the systemic immune-inflammation index in patients with acute respiratory distress syndrome: A retrospective study

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ABSTRACT

Background: Inflammation is critical in the etiology and progression of acute respiratory distress syndrome (ARDS). This study aims to rigorously assess the predictive capacity of systemic immune-inflammation index (SII) in determining the outcomes of patients with ARDS.
Methods: Patient data were extracted from version 2.2 of the Medical Information Mart for Intensive Care IV (MIMIC-IV). The Receiver Operating Characteristic (ROC) curve was deployed to determine the optimal cutoff value for the SII, facilitating the stratification of participants into distinct cohorts based on SII levels. The relationship between SII and survival outcomes was rigorously evaluated using Cox proportional hazards models. The association between SII and patient survival was rigorously examined using Cox proportional-hazard models. The impact of varying SII levels on mortality was quantitatively assessed through these models, with the results articulated as hazard ratios (HRs) and 95% confidence intervals (CIs). Three distinct models were formulated for this analysis: Model 1 employed univariate Cox regression to relate SII with mortality; Model 2 introduced adjustments for age and sex; and Model 3 extended these adjustments to include age, sex, race, SAPS II, APSIII, Hemoglobin, Albumin, Pneumonia, SpO2, and SBP.
Results: Post-application of the inclusion criteria, a cohort of 976 eligible patients was delineated for detailed examination. Univariate analysis focusing on 30-day mortality within the SII $\geq$1694, the hazard ratio (HR) was 1.42 (95% confidence interval (CI): 1.11, 1.81). However, after adjusting for confounding factors such as age, sex, race, Simplified Acute Physiology Score II (SAPS II), Acute Physiology Score (APS) III, Hemoglobin, Albumin, Pneumonia, SpO2, and Systolic Blood Pressure (SBP), an SII value of $\geq$1694 was identified as an independent and significant risk factor for mortality in patients with ARDS, with an HR of 1.38 (95% CI: 1.08–1.77, P = 0.0016). This trend was consistent for 90-day and one-year mortality rates.
Conclusions: SII surfaced as an autonomous determinant of mortality in ARDS patients, affirming its status as an accessible and dependable prognostic indicator for individuals newly diagnosed with this critical condition. Additional research is imperative to further elucidate the prognostic implications of SII in the therapeutic management of patients with ARDS.

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1. Introduction

Acute respiratory distress syndrome (ARDS) is a serious complication characterized by non-cardiogenic pulmonary edema and refractory hypoxemia [1–4]. Epidemiological studies show a 10.4% prevalence of ARDS during ICU admission; the overall in-hospital mortality is 40.0% [5,6]. ARDS manifests a spectrum of complications that impact patients over both short-term and long-term periods [7]. In its acute phase, individuals frequently confront ongoing respiratory failure, which necessitates a prolonged dependence on mechanical ventilation. Following the acute phase, long-term repercussions are prevalent, encompassing impairments in physical and cognitive functions, along with mental health challenges and other consequential health issues [8]. Despite progress in ICU therapies, such as lung-protective ventilation strategies and the use of intravenous steroids, the mortality rate among ARDS patients continues to be alarmingly high [9]. Due to the high mortality, early prognosis prediction in ARDS has always been a topic under exploration [10, 11]. Although the measures of Sequential Organ Failure Assessment (SOFA) [12] and the Simplified Acute Physiological Score II (SAPS II) [13] are routinely used for the prognostic assessment of critically ill patients, these are not specific to ARDS. Risk prediction models for ARDS require the application of many variables and complex formulas [14,15]. Therefore, a simple predictive biomarker is expected to play an important role in guiding treatment regimens [16].

Inflammation is a pivotal factor in the advancement of critical illnesses and is frequently linked with ARDS, consequently elevating the mortality risk [17,18]. ystemic immune-inflammatory index (SII) was initially characterized as a potential biomarker or the assessment of tumor proliferation and cardiovascular disease pathogenesis [19–21].SII was calculated as platelets count x neutrophils count/lymphocytes count, and it is now considered to reflect inflammation status better [22,23].The SII has been effectively used to evaluate prognosis in patients suffering from various conditions, including stroke [24] and cancer [25]. However, the correlation between SII and the prognosis of patients with ARDS is still not well-established and warrants further exploration. The study was meticulously structured to investigate the association between the SII and the prognostic outcomes in patients afflicted with ARDS.

2. Methods

2.1. Data source

This study was conducted as a single-center, retrospective analysis. This investigation harnessed the publicly accessible MIMIC-IV, version 2.2. This extensive database encompasses health records of over 190,000 ICU patients between 2008 and 2016. The architectural framework and use of the database were sanctioned by the Institutional Review Boards of both the Massachusetts Institute of Technology, Cambridge, MA, USA, and the Beth Israel Deaconess Medical Center.

2.2. Inclusion criteria

From the 190,000 patients in the MIMIC-IV database, the study focused exclusively on those admitted to the ICU. Eligibility criteria included patients aged over 18 years at the time of first admission and an ICU stay exceeding two days, aligning with the Berlin criteria [26,27]. Exclusion criteria were meticulously defined as follows: (1) ICU stays shorter than 48 h; (2) patient records with more than 5% missing data; and (3) absence of SII in the records.

2.3. Extraction of data

The dataset encompassed demographic information (sex, age, race) and vital sign parameters (temperature, respiratory rate, heart rate, mean arterial pressure (MAP), diastolic blood pressure (DBP), systolic blood pressure (SBP), and transcutaneous oxygen saturation (SpO2)). Laboratory profiles included counts of lymphocytes, neutrophils, white blood cells, and platelets, alongside measurements of hemoglobin, anion gap, serum creatinine, chloride, and sodium levels. Data capture was initiated at the point of admission and continued until the occurrence of death or completion of one year of follow-up, incorporating information from the initial 24 h of hospitalization at Beth Israel Deaconess Medical Center. The study designated 30-day mortality as the primary clinical endpoint, with 90-day and one-year mortality rates as secondary endpoints.

2.4. Statistical analysis

The Receiver Operating Characteristic (ROC) curve was deployed to determine the optimal cutoff value for the SII, facilitating the stratification of participants into distinct cohorts based on SII levels. Baseline demographic characteristics of the study population were stratified by these SII levels. Continuous variables were expressed as mean ± SD, while categorical variables were presented as interquartile range (IQR) and frequency. Variations in continuous variables among different SII cohorts were evaluated using the Kruskal-Wallis test. In contrast, categorical variables were analyzed using the chi-square test ($\chi 2$).

The association between SII and patient survival was rigorously examined using Cox proportional-hazard models. The impact of varying SII levels on mortality was quantitatively assessed through these models, with the results articulated as hazard ratios (HRs) and 95% confidence intervals (CIs). Three distinct models were formulated for this analysis: Model 1 employed univariate Cox regression to relate SII with mortality; Model 2 introduced adjustments for age and sex; and Model 3 extended these adjustments to include age, sex, race, SAPS II, APSIII, Hemoglobin, Albumin, Pneumonia, SpO2, and SBP.

Acknowledging disparities in baseline characteristics, a propensity score matched (PSM) analysis was conducted, pairing subjects at a 1:1 ratio using a caliper width of 0.01 of the SD of the logit of the propensity score.

A p-value of less than 0.05 was deemed indicative of statistical significance. Comprehensive statistical analyses were performed utilizing R software (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Patient characteristics

Initially, records from the MIMIC-IV database for 19,000 ICU admissions at Beth Israel Deaconess Medical Center were accessed. Post-application of the inclusion criteria, a cohort of 976 eligible patients was delineated for detailed examination. This cohort comprised 539 males and 437 females, with a mean age of 63.7 years.

Patients were stratified into two cohorts based on the SII: one cohort included 488 patients with SII <1694, while the other encompassed 488 patients with SII ≥1694. Comprehensive data pertaining to vital signs, laboratory parameters, and the history of comorbidities for each SII cohort are delineated in Table 1. It was observed that patients with elevated SII levels frequently presented with concomitant increases in creatinine levels, decreases in chloride concentrations, and diminished levels of transcutaneous oxygen saturation (SpO2).

Table 1
Baseline characteristics of the study population.

Characteristics	SII		*P* value
	< 1694(n = 488)	≥1694 (n = 488)	
Clinical parameters			
Age, years	63.1 ± 16.8	64.4 ± 16.7	0.112
Sex, n (%)			0.949
Male	218 (44.7%)	219 (44.9%)	
Female	270 (55.3%)	269 (55.1%)	
Ethnicity, n(%)			0.031
White	291 (59.6%)	326 (66.8%)	
Black	63 (12.9%)	42 (8.6%)	
Other	134 (27.5%)	120 (24.6%)	
Vital signs			
SBP, mmHg	122.3 ± 26.1	123.8 ± 24.5	0.146
DBP, mmHg	68.9 ± 17.4	68.7 ± 17.8	0.692
MAP, mmHg	83.5 ± 18.7	84.2 ± 17.7	0.465
Heart rate, beats/minute	91.0 ± 20.2	94.2 ± 21.5	0.018
Respiratory rate, times/minute	20.6 ± 6.4	21.4 ± 6.8	0.078
Temperature, °C	36.7 ± 1.1	36.8 ± 0.9	0.077
SpO2, %	97.1 ± 4.1	96.6 ± 4.4	0.172
Comorbidities			
Pneumonia, n (%)	163 (33.4%)	203 (41.6%)	0.008
Renal failure, n (%)	105 (21.5%)	95 (19.5%)	0.428
CHD, n (%)	128 (26.2%)	107 (21.9%)	0.116
Laboratory parameters			
SII	822.1 ± 451.2	5318.6 ± 5234.7	<0.001
Albumin, g/dL	3.1 ± 0.7	3.0 ± 0.8	0.940
N,%	71.4 ± 13.7	85.4 ± 7.1	<0.001
L,%	17.2 ± 9.9	6.0 ± 3.0	<0.001
Platelet, 10^9/L	162.7 ± 78.3	275.3 ± 136.8	<0.001
Hemoglobin, g/dL	9.7 ± 2.3	9.9 ± 2.3	0.248
Hematocrit, %	35.1 ± 6.6	35.6 ± 6.9	0.500
Serum creatinine, mg/dl	1.2 ± 0.9	1.4 ± 1.5	0.119
Serum chloride, mg/dl	102.7 ± 5.6	101.2 ± 6.6	<0.001
Serum sodium, mg/dl	137.6 ± 4.6	136.7 ± 5.0	<0.001
Anion gap, mg/dl	17.6 ± 4.9	17.6 ± 4.9	0.880
Scoring systems			
SAPSII	45.1 ± 15.6	46.8 ± 15.2	0.185
APSIII	50.3 ± 22.8	52.1 ± 24.8	0.320
Mortality			
30-day	110 (22.5%)	153 (31.4%)	<0.001
90-day	127 (26.0%)	175 (35.9%)	<0.001
One year	166 (34.0%)	213 (43.6%)	<0.001

Abbreviations: SII: Systemic immuno-inflammatory index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; SAPSII: simplified acute physiology score II.

3.2. Association of SII with 30-day,90-day, and one-year mortality

In the univariate analysis, several factors were identified as significant risk factors for mortality in ARDS patients (Table 2). Varied models were constructed to assess the association between the SII and mortality in ARDS patients. The outcomes, encompassing effect sizes (HR) and 95% CIs, are elaborated in Table 3.

In the SII ≥1694 cohort, the univariate analysis for 30-day mortality yielded an HR of 1.42 (95% CI: 1.11–1.81). Nonetheless, upon adjustment for confounders such as age, sex, race, SAPS II, APSIII, Hemoglobin, Albumin, Pneumonia, SpO2, and SBP, an SII ≥1694 emerged as an independent, significant mortality predictor in ARDS patients, with an HR of 1.38 (95% CI: 1.08–1.77; p = 0.0109).

For 90-day mortality, the univariate analysis indicated an HR of 1.43 (95% CI: 1.14–1.79) for the SII ≥1694 cohort. Post-adjustment for the stated confounders, an SII ≥1694 remained a substantial independent predictor of mortality, with an HR of 1.39 (95% CI: 1.10–1.75; p = 0.0057).

Likewise, in the one-year mortality analysis, the SII ≥1694 cohort demonstrated an HR of 1.36 (95% CI: 1.11–1.67) in the univariate analysis. After factoring in the same confounders, an SII ≥1694 consistently proved to be a significant independent determinant of mortality, with an HR of 1.31 (95% CI: 1.07–1.61; p = 0.0103).

3.3. Propensity score matching analysis

A PSM was executed to refine the understanding of the relationship between the SII and prognosis in ARDS patients. This analysis ensured equitable distribution of baseline patient characteristics across different SII cohorts, effectively minimizing potential biases, as detailed in Table 4. In analyzing 30-day mortality using Cox regression, a high SI was distinctly identified as an independent determinant associated with an elevated risk of mortality.

Table 2
Cox regression associating variables with 30-day mortality.

Characteristics	HR (95% CIs)	*P* value
Clinical parameters		
Age, years	1.01 (1.00, 1.02)	0.0027
Sex		
Male	1.0	
Female	1.16 (0.90, 1.48)	0.6544
Ethnicity		
White	1.0	
Black	1.07 (0.71, 1.62)	0.1252
Other	1.48 (1.14, 1.93)	0.0035
Vital signs		
SBP, mmHg	1.00 (0.99, 1.00)	0.4535
DBP, mmHg	1.00 (1.00, 1.01)	0.3512
MAP, mmHg	1.00 (0.99, 1.01)	<0.0001
Heart rate, beats/minute	1.00 (1.00, 1.01)	0.8697
Respiratory rate, times/minute	1.03 (1.01, 1.04)	0.0008
Temperature, °C	0.84 (0.77, 0.92)	0.0002
SpO2, %	0.96 (0.94, 0.98)	0.0009
Comorbidities		
Pneumonia, n (%)	1.23 (0.96, 1.57)	0.1009
Renal failure, n (%)	1.35 (1.02, 1.78)	0.0361
CHD, n (%)	1.01 (0.76, 1.34)	0.9267
Laboratory parameters		
SII	1.00 (1.00, 1.00)	0.0113
Albumin, g/dL	0.78 (0.63, 0.96)	0.0064
N,%	1.02 (1.00, 1.03)	0.0179
L,%	0.96 (0.95, 0.98)	<0.0001
Platelet, 10^9/L	1.00 (1.00, 1.00)	0.5611
Hemoglobin, g/dL	1.00 (0.95, 1.05)	0.9334
Hematocrit, %	1.01 (0.99, 1.03)	0.4208
Serum creatinine, mg/dl	1.07 (1.03, 1.10)	<0.0001
Serum chloride, mg/dl	0.99 (0.97, 1.00)	0.1285
Serum sodium, mg/dl	1.16 (0.99, 1.36)	0.0588
Anion gap, mg/dl	1.14 (1.07, 1.22)	<0.0001
Scoring systems		
SAPSII	1.05 (1.04, 1.06)	<0.0001
APSIII	1.02 (1.02, 1.02)	<0.0001

Abbreviations: HR: hazard ratio, CI: confidence interval; SII: Systemic immuno-inflammatory index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; SAPSII: simplified acute physiology score II.

Table 3
HR (95% CIs) for all-cause mortality across groups of SII level.

SII	Model 1[a]		Model 2[b]		Model 3[c]	
	HR (95% CIs)	*P* value	HR (95% CIs)	*P* value	HR (95% CIs)	*P* value
30-Day all-cause mortality						
<1694	1.0		1.0		1.0	
≥1694	1.42 (1.11, 1.81)	0.0052	1.43 (1.12, 1.83)	0.0044	1.38 (1.08, 1.77)	0.0109
90-Day all-cause mortality						
<1694	1.0		1.0		1.0	
≥1694	1.43 (1.14, 1.79)	0.0023	1.43 (1.14, 1.80)	0.0022	1.39 (1.10, 1.75)	0.0057
One-Year all-cause mortality						
<1694	1.0		1.0		1.0	
≥1694	1.36 (1.11, 1.67)	0.0031	1.37 (1.12, 1.68)	0.0026	1.31 (1.07, 1.61)	0.0103

Abbreviations: HR: hazard ratio, CI: confidence interva.
Notes: Models 1, 2 and 3 were derived from Cox proportional hazards regression models.
[a] model 1 covariates were adjusted for nothing.
[b] model 2 covariates were adjusted for age, sex and race.
[c] model 3 covariates were adjusted for age, sex, race, SAPSII，APSIII，Hemoglobin,Albumin，Pneumonia,SpO2,and SBP.

Table 4
Characteristics of patients before and after PSM.

Characteristics	SII		*P* value
	< 1694	≥1694	
Clinical parameters			
Age, years	63.07 ± 16.73	64.24 ± 17.01	0.3049
Sex, n (%)			1
Male	241 (55.7)	240 (55.4)	
Ethnicity, n(%)			0.883
White	262 (60.5)	289 (66.7)	
Black	59 (13.6)	37 (8.5)	
Other	112 (25.9)	107 (24.7)	
Vital signs			
SBP, mmHg	122.61 ± 25.85	123.60 ± 24.52	0.411
DBP, mmHg	69.16 ± 17.48	68.36 ± 17.96	0.893
MAP, mmHg	83.74 ± 18.82	83.67 ± 17.79	0.891
Respiratory rate, times/minute	21.4 ± 4.6	22.1 ± 4.9	0.084
Temperature, °C	37.0 ± 0.8	37.0 ± 0.8	0.466
SpO2, %	96.4 ± 3.7	96.1 ± 2.9	0.073
Comorbidities			
Pneumonia, n (%)	113 (55.1)	124 (60.5)	0.317
Renal failure, n (%)	26 (12.7)	32 (15.6)	0.478
CHD, n (%)	36 (17.6)	39 (19.1)	0.769
Scoring systems			
SAPSII	45.21 ± 16.36	46.72 ± 15.01	0.246
APSIII	50.40 ± 22.89	51.86 ± 24.29	0.726
Mortality			
30-day	95 (21.9)	131 (30.3)	0.0068
90-day	110 (25.4)	148 (34.2)	0.006
One year	147 (33.9)	182 (42)	0.017

Abbreviations: SII: Systemic immuno-inflammatory index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; SAPSII: simplified acute physiology score II; and PSM, Propensity score matching analysis.

3.4. Association of SII with 30-day mortality

We conducted a detailed analysis to explore the dose-response relationship between the SII and 30-day mortality in ARDS patients. Utilizing the Cox regression model, our analysis revealed a clear dose-response correlation. As depicted in Fig. 1, SII was identified as an independent and significant risk factor for mortality among these patients.

4. Discussion

Inflammation is pivotal in the evolution, pathogenesis, and prognosis of ARDS [28]. his comprehensive study successfully established the SII as an independent predictor of all-cause mortality in ARDS patients. Significantly, the data highlighted that elevated SII levels are robustly associated with increased all-cause mortality in ARDS patients, even when a wide array of variables is taken into consideration. Given its integration into routine diagnostics, the SII serves as a cost-efficient and easily accessible biomarker, thereby

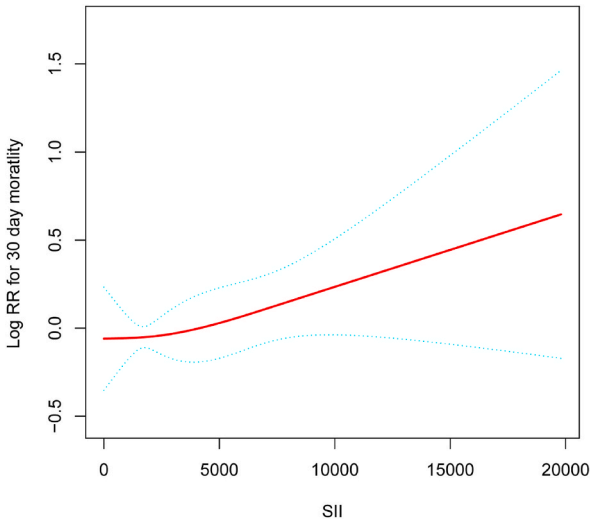

Fig. 1. The dose-response association of SII with 30-day mortality.

enhancing its utility in clinical practice.

ARDS is linked with multi-organ diseases and systemic inflammation . In some previous studies [29–32], multiple biomarkers were used for prognostic prediction in ARDS. However, these predictors are limited, and a single mechanism cannot accurately predict the outcomes of a complex syndrome, such as ARDS.

SII is acknowledged as a powerful predictor in a spectrum of diseases and organ dysfunctions [33,34]. Its relevance spans across various malignancies such as small-cell lung cancer [35], colorectal cancer, hepatocellular carcinoma [23], and gastric cancer [36], where in elevated SII correlates with diminished survival rates. Fundamentally, SII is calculated from the counts of three pivotal circulating immune cells: lymphocytes, neutrophils, and platelets, thus reflecting the systemic inflammatory state and serving as a marker routinely used to gauge systemic inflammation.

Study suggests that the pathogenesis of ARDS may be influenced by increased secretion of pro-inflammatory cytokines, aggregation of neutrophils, and compromised alveolar-capillary barriers. We observed that patients exhibiting elevated neutrophil activity also demonstrated higher levels of reactive oxygen species. Lymphocytes, a key component of circulating leukocytes, play a vital role in regulating adaptive immune responses and are intricately linked with innate immunity. They act as specific mediators of inflammation and perform regulatory and protective functions. The activation of platelets, another essential element, can be instigated by diverse inflammatory mediators including epinephrine, cytokines, serotonin, and dopamine.

To our knowledge, the principal strength of this study resides in its comprehensive analysis of the correlation between the SII and mortality among ARDS patients in the ICU setting. Moreover, we strategically selected mortality as the primary outcome to underscore its clinical relevance. Nonetheless, our investigation is subject to certain constraints. Initially, the retrospective nature of the study and its confinement to a single center may introduce selection bias. Furthermore, the measurement of SII was conducted solely at the point of ICU admission, precluding the evaluation of SII fluctuations during the ICU stay. Additionally, the size of the patient cohort was relatively restricted. Lastly, inherent limitations associated with the MIMIC database could not be circumvented, including the absence of key clinical decision-making factors and the presence of some inaccuracies within the data.

5. Conclusion

In summary, the SII has emerged as an independent predictor of mortality among patients with ARDS, consolidating its role as a readily available and reliable prognostic tool for those recently diagnosed with this severe ailment. It is paramount to conduct further investigations to comprehensively decipher the prognostic significance of SII in the nuanced therapeutic management of severe ARDS cases.

Data availability statement

The dataset supporting the findings of this study is available from the corresponding author upon reasonable and justified request.

Disclosure

None.

CRediT authorship contribution statement

Xiaodang Pan: Investigation. **Junnan Xu:** Investigation. **He Wu:** Data curation, Conceptualization. **Jie Wang:** Data curation. **Quanwan Kong:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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