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Association of the systemic immune-inflammation index with clinical outcomes in acute myocardial infarction patients with hypertension

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Abstract

Background A new indicator of immunological and inflammatory condition, the Systemic Immunoinflammatory Index (SII), has been linked to a bad prognosis in a number of disorders.

Methods Two thousand three hundred seventeen ICU patients were admitted with hypertension and acute myocardial infarction (AMI). Patients were grouped according to their baseline SII tertile number into Q1, Q2, and Q3 groups. The main outcomes were death from all causes at 30 days, 365 days, cardiogenic shock, and congestive heart failure.

Results The case fatality rate increases with increasing SII. The correlation between SII and 30-day all-cause mortality [hazard ratio (HR) 1.765, 95% confidence interval (CI) 1.330–2.343 (Q3 versus Q1 group)], 365-day all-cause mortality [HR 2.713, 95% CI 2.250–3.272 (Q3 versus Q1 group), HR 1.603, 95% CI 1.312–1.959 (Q3 vs. Q1 group)], congestive heart failure [odds ratio (OR) 1.255, 95% CI 1.006–1.565 (Q2 vs. Q1 group), OR 1.565, 95% CI 1.220–2.009 (Q3 vs. Q1 group)] and cardiogenic shock [OR 1.930. 95% CI 1.271–2.974 (Q2 vs. Q1 group)] were all validated. According to subgroup analysis, individuals who had chosen to have CABG surgery had a stronger correlation between SII and a worse outcome. According to Kaplan–Meier (K-M) survival curves, patients in the Q3 group with SII had the highest rates of morbidity and death. The RCS curves demonstrated an essentially linear connection between SII and 30 days, 365 days, and congestive heart failure even after controlling for covariates.

Conclusions SII was substantially correlated with 30-day all-cause mortality, 365-day all-cause mortality, in-hospital congestive heart failure, and cardiogenic shock in patients who had both hypertension and acute myocardial infarction. In individuals with acute myocardial infarction and hypertension, a greater SII would be regarded as an independent risk factor for a higher death rate.

Keywords Acute myocardial infarction, Hypertension, Inflammation, Prognosis

Introduction

Acute myocardial infarction (AMI) has been globally acknowledged as the primary cause of morbidity and mortality of cardiovascular diseases (CVDs). Patients diagnosed with AMI have a mortality rate of nearly 10% within a year, while the incidence of death during hospitalization range from 4 to 12% [1]. Hypertension, according to relevant research on myocardial infarction in

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China, contributes to approximately 51.2% of AMI cases [2]. Therefore, it is vital to promptly identify risk factors to improve clinical care and reduce cardiovascular disease in the future.

Low-grade inflammation plays a significant role in the initiation and persistence of high blood pressure despite effective control. This may be due to underlying immune cell activation and chronic inflammation [3–5]. Previous studies indicated that a high systemic immune-inflammation index (SII) is related to increased carotid intima-media thickness and left ventricular hypertrophy in hypertension patients [6, 7]. SII has the potential to predict the development of contrast induced nephropathy in patients with non-ST-segment elevation myocardial infarction [8]. The SII (neutrophil × platelet/lymphocyte) serves as an inflammation indicator taking into the counts of neutrophil, platelet, and lymphocyte, which has been introduced as a serum immune and inflammation marker to assess the remaining cardiovascular risk [9, 10].

Research have shown that AMI patients are often followed by major adverse cardiovascular events (MACE), including acute heart failure, malignant arrhythmias, cardiogenic shock, and sudden cardiac death [11–14]. Acute inflammation response and stress has been crucial to the pathogenesis of AMI [15]. Moreover, an increasing body of research has demonstrated that SII outperforms conventional risk factors in predicting the risk associated with MACE of in-hospital mortality and long-term adverse cardiovascular outcomes in AMI patients [7, 16]. Nevertheless, limited studies have been conducted on the relationship between SII and long-term clinical events, particularly mortality, heart failure and cardiogenic shock in AMI patients with hypertension. The present study aimed to evaluate the predictive impact of SII on clinical outcomes in AMI patients with hypertension.

Methods

Patients

Patients were recruited from the MIMIC-IV database from 2008 to 2019. Inclusion criteria were as follows: diagnosis of acute myocardial infarction combined with hypertension; 18 years of age or older. Exclusion criteria were as follows: patients with severe hepatic dysfunction; patients with malignant tumors; and patients lacking platelet counts, neutrophil counts, and lymphocyte counts.

Source of data and ethics approval

The Medical Information Mart for Intensive Care IV is a sizable critical care database that served as the foundation for our retrospective analysis [17]. This database has been authorized by the Institutional Review Board and is an updated version of MIMIC-III. Numerous enhancements have been made, such as the structure's simplification, the addition of new data components, and the enhancement of earlier data items' usability. The intensive care unit (ICU) patient population at Beth Israel Deaconess Medical Center is now fully covered by complete, high-quality data in MIMIC-IV, spanning the years 2008 through 2019, inclusive. After gaining access to the database, one author (LCD) was in charge of extracting the data.

Study design

The SII was calculated according to the following formula: SII = Platelet count × Neutrophil count / Lymphocyte count. Based on the tertiles of SII, it was further divided into three groups: Q1 (SII < 923.667,n = 773), Q2 (923.667 ≤ SII < 2287.17,n = 772), and Q3 (SII ≥ 2287.17,69,593,n = 772). The outcome of our research was defined as follows: 30-day mortality, 365-day mortality, congestive heart failure, and cardiogenic shock from the date of admission to the hospital.

Demographic data (age, gender, race), vital signs (systolic blood pressure, diastolic blood pressure, heart rate), and past medical history (hypertension, diabetes mellitus, chronic obstructive pulmonary disease (COPD)] were all recorded. Within the first 24 h of admission, multiple laboratory marker measurements were made using the first measurement including oxygen saturation (SpO2), lactate, hemoglobin, platelet count, neutrophil count, albumin, blood urea nitrogen (BUN), blood creatinine (Scr), glucose, prothrombin time (PT), prothrombin time activity (PTA), and white blood cell count, albumin, blood urea nitrogen (BUN), blood creatinine (Scr), alanine aminotransferase (ALT), azelotransferase (AST), glucose, prothrombin time (PT), prothrombin time activity (PTA), sodium, potassium, high-density lipoproteins (HDL), low-density lipoproteins (LDL), total cholesterol (TC), triglycerides (TG), coronary artery revascularization (PCI, CABG), acute physiology score (SAPS II) and sequential organ failure score (SOFA).

Data analysis

The formula for continuous variables with a normal distribution is mean ± standard deviation. The interquartile range, or median, is used to express continuous variables that are not regularly distributed. Numbers are used to express categorical variables (percentages).

Independent samples t-test or Mann–Whitney U-test was used for continuous variables and chi-square test was used for categorical variables. Univariate and multivariate Cox proportional risk models and univariate and multivariate logitic regression models were then constructed to test the correlation between SII quartiles and clinical outcomes (the reference group was the first quartile).

Variables with differences and common cardiovascular risk factors were included in multivariate cox and regression models for 30-day, 365-day all-cause mortality, congestive heart failure, and cardiogenic shock: model 1, uncorrected; model 2, corrected for age, gender and race; model 3, involved variables in model 2 and diabetes mellitus, COPD; and model 4, involved variables in model 3 and lactate, albumin, urea nitrogen, creatinine, AST, monocyte count, hemoglobin, white blood cell count, HDL, LDL, TC, TG and SpO2. At the same time, when SII was considered as a continuous variable, we used restricted cubic spline (RCS) curves in order to more flexibly model and visualize the relationship between SII on admission and the risk of congestive heart failure, cardiogenic shock, 30-day, and 365-day mortality. The cumulative incidence of all-cause mortality at 30 and 365 days was calculated by Kaplan–Meier survival curves. Subgroup analyses were also performed and presented as a forest sample. A two-sided $P < 0.05$ was considered statistically significant. All analyses were performed using R (R Foundation for Statistical Computing, Vienna, Austria).

Results

Baseline characteristics

A total of 2,317 patients were included in our analysis. The mean age was 72.71 ± 12.58 years and 60.6% were male. Of all individuals, 438 (18.9%) died of all-cause deaths within 30 days of admission to the intensive care unit (ICU), 756 (32.6%) died of all-cause deaths within 365 days, 194 (8.4%) were combined with cardiogenic shock while in the hospital, and 1,289 (55. 6%) were combined with congestive heart failure while in the hospital. Group analysis based on SII tertiles is shown in Table 1. The Tertile 3 (Q3) group of patients included more elderly patients, a higher percentage of male patients, a higher percentage of White patients, a higher percentage of COPD patients, and a higher percentage of PCI recipients. Heart rate, lactate, white blood cells (WBC), hemoglobin, blood creatinine, blood urea nitrogen (BUN), glucose, high-density lipoproteins (HDL), partially activated prothrombin time (APTT), prothrombin time (PT), and SpO2 and albumin decreased as the SPII ratio increased. Sequential Organ Failure Assessment (SOFA) and Simplified Acute Physiology Score II (SAPS II) scores rose when SII increased. Cardiogenic shock, cardiac arrest, and 30-day and 365-day all-cause mortality were all substantially correlated with increased SII.

Clinical outcomes in different groups stratified by SII

The Cox proportional risk model outcomes for 30-day all-cause mortality and 365-day all-cause mortality are shown in Table 2. In the uncorrected Cox model, 30-day all-cause mortality was higher in the Q2 and Q3 groups compared with the Q1 group [hazard ratio (HR) 1.644, 95% confidence interval (CI) 1.246–2.170 for Q2; HR 3.094 for Q3, 95% CI 2.400–3.987]. After adjusted by confounders, the Q3 group was 1.765 times higher than the Q1 group (HR 1.765, 95% CI 1.330–2.343). Similarly, in the uncorrected Cox model, 365-day all-cause mortality was higher in the Q2 and Q3 groups compared with the Q1 group [hazard ratio (HR) 1.603, 95% confidence interval (CI) 1.312–1.959 for Q2; HR 2.713, 95% CI 2.250–3.272 for Q3]. After adjusted by confounders, the Q2 group was 1.304 times higher than the Q1 group (HR 1.304, 95% CI 1.060–1.605). The Q3 group was 1.695 times higher than the Q1 group (HR 1.765, 95% CI 1.373–2.094). It is concluded that high SII is an independent risk factor for 30- and 365-day all-cause mortality in patients with hypertension and AMI.

The logistic proportional risk model outcomes for congestive heart failure and cardiogenic shock are shown in Table 3. In the uncorrected logistic model, the prevalence of congestive heart failure was higher in the Q2 and Q3 groups compared with the Q1 group [odds ratio (OR) 1.446 for Q2, 95% confidence interval (CI) 1.184–1.767; OR 2.057 for Q3, 95% CI 1.678–3.987]. 95% CI 1.678–2.524]. After adjusted by confounders, the Q2 group was 1.255 times as large as the Q1 group (OR 1.255, 95% CI 1.006–1.565) and the Q3 group was 1.565 times as large as the Q1 group (OR 1.565, 95% CI 1.220–2.009). Similarly, in the uncorrected Cox model, the prevalence of cardiogenic shock was higher in the Q2 and Q3 groups compared with the Q1 group [odds ratio (OR) 2.183, 95% confidence interval (CI) 1.473–3.290 for Q2; OR 2.128, 95% CI 1.433–3.211 for Q3]. After adjusted by confounders, the Q2 group was 1.930 times larger than the Q1 group (OR 1.930, 95% CI 1.271–2.974). Consequently, we draw the conclusion that in individuals with hypertension and AMI, a high SII is a separate risk factor for congestive heart failure and cardiogenic shock during hospitalization.

The Kaplan–Meier (K-M) survival curve analysis revealed statistical differences in 30-day and 365-day mortality among the three groups by SII tertiles. The 30-day and 365-day all-cause mortality rates were significantly higher ($p < 0.001$) in group Q3 compared with groups Q2 and Q1 (Fig. 1). The risk of cardiogenic shock, congestive heart failure, 30-day all-cause mortality, and 365-day all-cause mortality all rose linearly ($p < 0.05$) with increasing SII in the unadjusted model, according to the restricted triple spline technique of analysis (Supplementary Fig. 1). Following the model's correction for age, sex, race, heart rate, systolic and diastolic blood pressure,

Table 1 Baseline characteristics across SII

Variables	Total ($n=2317$)	Q1 ($n=773$)	Q2 ($n=772$)	Q3 ($n=772$)	*P* value
Age, Mean ± SD	72.71 ± 12.58	71.21 ± 12.26	72.35 ± 13.02	74.58 ± 12.21	< 0.001
Gender, n (%)					< 0.001
Female	912 (39.4)	272 (35.2)	293 (38)	347 (44.9)	
Male	1405 (60.6)	501 (64.8)	479 (62)	425 (55.1)	
Race, n (%)					< 0.001
American	5 (0.2)	2 (0.3)	1 (0.1)	2 (0.3)	
Asian	53 (2.3)	23 (3)	22 (2.8)	8 (1)	
Black	230 (9.9)	104 (13.5)	75 (9.7)	51 (6.6)	
Hispanic	63 (2.7)	27 (3.5)	22 (2.8)	14 (1.8)	
Other	73 (3.2)	34 (4.4)	13 (1.7)	26 (3.4)	
Unknown	445 (19.2)	164 (21.2)	145 (18.8)	136 (17.6)	
White	1448 (62.5)	419 (54.2)	494 (64)	535 (69.3)	
Diabetes, n (%)					0.745
No	1216 (52.5)	397 (51.4)	410 (53.1)	409 (53)	
Yes	1101 (47.5)	376 (48.6)	362 (46.9)	363 (47)	
COPD, n (%)					< 0.001
No	1709 (73.8)	621 (80.3)	567 (73.4)	521 (67.5)	
Yes	608 (26.2)	152 (19.7)	205 (26.6)	251 (32.5)	
CABG, n (%)					< 0.001
No	1751 (75.6)	448 (58)	589 (76.3)	714 (92.5)	
Yes	566 (24.4)	325 (42)	183 (23.7)	58 (7.5)	
PCI, n (%)					0.007
No	1680 (72.5)	580 (75)	528 (68.4)	572 (74.1)	
Yes	637 (27.5)	193 (25)	244 (31.6)	200 (25.9)	
SOFA	5.77 ± 3.88	5.36 ± 3.57	5.31 ± 3.83	6.65 ± 4.06	< 0.001
SAPSII	39.14 ± 13.69	36.73 ± 12.61	37.70 ± 13.59	43.00 ± 14.01	< 0.001
Lactate, mmol/L	2.32 ± 2.04	1.97 ± 1.59	2.19 ± 1.76	2.81 ± 2.54	< 0.001
Monocyte (109 /L)	0.69 ± 0.53	0.57 ± 0.37	0.68 ± 0.44	0.81 ± 0.70	< 0.001
Lymphocyte (109 /L)	1.57 ± 1.24	2.29 ± 1.65	1.50 ± 0.80	0.92 ± 0.56	< 0.001
Neutrophil (109 /L)	10.74 ± 6.17	6.99 ± 3.61	10.23 ± 4.55	14.99 ± 6.93	< 0.001
Hemoglobin (g/L)	10.97 ± 2.60	10.64 ± 2.65	11.16 ± 2.65	11.10 ± 2.47	< 0.001
Platelet (109 /L)	223.91 ± 110.55	168.92 ± 75.23	217.88 ± 87.04	285.00 ± 129.09	< 0.001
WBC, Mean ± SD	13.31 ± 6.83	10.19 ± 5.24	12.71 ± 5.40	17.03 ± 7.71	< 0.001
Albumin (g/dL)	3.50 ± 0.65	3.53 ± 0.68	3.57 ± 0.65	3.41 ± 0.62	< 0.001
BUN, mg/dL	34.62 ± 26.26	28.90 ± 22.75	34.24 ± 26.48	40.72 ± 27.95	< 0.001
Scr, mg/dL	1.93 ± 2.10	1.71 ± 2.03	1.93 ± 2.22	2.16 ± 2.04	< 0.001
Sodium, mEq/L	138.10 ± 5.15	138.71 ± 4.53	137.99 ± 5.06	137.60 ± 5.73	< 0.001
Potassium, mEq/L	4.47 ± 0.97	4.22 ± 0.83	4.46 ± 0.94	4.72 ± 1.06	< 0.001
Glucose, g/dL	186.77 ± 122.97	169.51 ± 88.56	177.83 ± 102.92	212.98 ± 160.98	< 0.001
PT, s	16.39 ± 9.94	15.89 ± 9.02	15.78 ± 7.67	17.50 ± 12.44	< 0.001
APTT, s	44.80 ± 32.46	42.30 ± 29.13	45.36 ± 33.44	46.74 ± 34.44	0.023
ALT, U/L	84.87 ± 372.94	62.93 ± 304.61	84.24 ± 436.52	107.47 ± 365.18	0.064
AST, U/L	158.99 ± 929.57	132.63 ± 1085.75	158.47 ± 1053.70	185.92 ± 550.97	0.530
HbA1c, %	6.61 ± 1.60	6.65 ± 1.57	6.57 ± 1.60	6.62 ± 1.65	0.660
HDL, mg/dl	47.48 ± 17.30	46.09 ± 15.18	47.21 ± 17.16	49.13 ± 19.22	0.002
LDL, mg/dl	87.45 ± 38.96	90.08 ± 41.22	87.73 ± 38.77	84.53 ± 36.60	0.019
TC, mg/dl	162.76 ± 48.35	164.65 ± 48.93	163.45 ± 49.56	160.16 ± 46.46	0.167
TG, mg/dl	153.92 ± 129.50	155.79 ± 119.01	157.24 ± 151.54	148.72 ± 114.83	0.384
HR, beats/min	87.00 ± 18.26	82.47 ± 16.48	86.96 ± 18.13	91.58 ± 18.97	< 0.001

Table 1 (continued)

Variables	Total ($n=2317$)	Q1 ($n=773$)	Q2 ($n=772$)	Q3 ($n=772$)	*P* value
SBP, mmHg	122.88 ± 23.80	123.57 ± 23.23	122.47 ± 23.43	122.58 ± 24.73	0.608
DBP, mmHg	67.72 ± 17.98	67.30 ± 17.12	67.86 ± 17.66	68.01 ± 19.11	0.714
SPO2, %	96.83 ± 4.24	97.73 ± 3.18	96.53 ± 4.56	96.24 ± 4.67	< 0.001
SII	2599.31 ± 3991.94	539.65 ± 218.07	1489.72 ± 386.71	5771.22 ± 5666.70	< 0.001
30 days all-cause mortality, n (%)					< 0.001
Yes	1879 (81.1)	692 (89.5)	642 (83.2)	545 (70.6)	
No	438 (18.9)	81 (10.5)	130 (16.8)	227 (29.4)	
365 days all-cause mortality, n (%)					< 0.001
Yes	1561 (67.4)	613 (79.3)	533 (69)	415 (53.8)	
No	756 (32.6)	160 (20.7)	239 (31)	357 (46.2)	
Cardiogenic shock, n (%)					< 0.001
Yes	2123 (91.6)	734 (95)	694 (89.9)	695 (90)	
No	194 (8.4)	39 (5)	78 (10.1)	77 (10)	
CHF, n (%)					< 0.001
Yes	1028 (44.4)	412 (53.3)	341 (44.2)	275 (35.6)	
No	1289 (55.6)	361 (46.7)	431 (55.8)	497 (64.4)	

Continuous variables that conform to the normal distribution are expressed as mean ± standard deviation. Continuous variables that are not normally distributed are expressed as median (interquartile range). Categorical variables are expressed as numbers (percentages)

COPD chronic obstructive pulmonary disease, *CHF* congestive heart failure, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *SpO2* saturation of hemoglobin with oxygen, *WBC* white blood cell, *BUN* blood urea nitrogen, *SCr* serum creatinine, *PT* prothrombin time, *PCI* percutaneous coronary intervention *CABG* coronary artery bypass graft, *SOFA* sequential organ failure assessment, *SAPS II* the Simplified Acute Physiology Score II

diabetes mellitus, chronic obstructive pulmonary disease, lactate, albumin, urea nitrogen, creatinine, AST, white blood cell count, hemoglobin, monocyte count, HDL, LDL, TC, TG, and SpO2. While there was no significant correlation between the SII and cardiogenic shock within hours of linearity, it was still linearly associated with the risk of all-cause mortality at 30 days, the risk of all-cause mortality at 365 days, and congestive heart failure (Fig. 2).

Subgroup and multivariate analysis of clinical outcomes in AMI with hypertension patients

We conducted subgroup analyses for SpO2, COPD, PCI, CABG, diabetes, sex, age, and COPD in order to investigate further if this association would vary under different settings (Fig. 3). Within the 30-day all-cause mortality subgroup analysis, we discovered that SII was more predictive of 30-day all-cause death among patients with SpO2 ≥ 95%, among women, among diabetic patients, among patients who did not choose PCI, and among patients aged 60–80 years. On the other hand, high levels of SII were linked to higher 365-day all-cause mortality in all subgroups of age, sex, diabetes mellitus, and COPD in the subgroup analysis of 365-day all-cause mortality. Additionally, in the subgroup analyses of SpO2 and surgical modality, SII was associated with a more predictive value of 365-day all-cause mortality in patients with SpO2.

Discussion

To our knowledge, this is the first study to investigate whether the inflammatory marker SII is an independent risk factor for poor clinical prognosis in patients with hypertension and acute myocardial infarction. Our study found that SII remained an independent risk factor for short-term mortality, long-term mortality, cardiogenic shock, and congestive heart failure in patients with hypertension combined with acute myocardial infarction after controlling for age, race, and cardiovascular risk factors. A subgroup analysis of this trial revealed that SII was more predictive of short-term mortality for patients with SpO2 ≥ 95% who were between the ages of 60 and 80, were female, had diabetes, and had not had PCI. In terms of 365-day mortality, patients with high SII had increased mortality in subgroups related to age, sex, diabetes, and COPD; however, SII had a higher predictive value in the subgroups related to SpO2 and surgical modality, namely in those who underwent CABG and had SpO2 ≥ 95%. Therefore, SII may be a reliable and convenient indicator to better identify high-risk patients with hypertension and acute myocardial infarction.

It is well recognized that hypertension is a separate risk factor for acute myocardial infarction, and those who are experiencing this condition require proper blood pressure management [18]. The prognosis is worse for patients with acute myocardial infarction combined with hypertension than it is for those

Table 2 Cox proportional hazard models for 30 days all-cause death and 365 days all-cause

30 days all-cause mortality			**365 days all-cause mortality**		
Variable	**HR(95%CI)**	***P* value**	**Variable**	**HR(95%CI)**	***p*.value**
Model 1			Model 1		
Q1	Ref		Q1	Ref	
Q2	1.644 (1.246, 2.170)	< 0.001	Q2	1.603 (1.312, 1.959)	< 0.001
Q3	3.094 (2.400, 3.987)	< 0.001	Q3	2.713 (2.250, 3.272)	< 0.001
P for trend		< 0.001	p for trend		< 0.001
Model 2			Model 2		
Q1	Ref		Q1	Ref	
Q2	1.591 (1.203, 2.103)	0.001	Q2	1.542 (1.260, 1.887)	< 0.001
Q3	2.880 (2.226, 3.727)	< 0.001	Q3	2.489 (2.057, 3.010)	< 0.001
P for trend		< 0.001	p for trend		< 0.001
Model 3			Model 3		
Q1	Ref		Q1	Ref	
Q2	1.564 (1.182, 2.069)	0.002	Q2	1.506 (1.231, 1.844)	< 0.001
Q3	2.812 (2.170, 3.643)	< 0.001	Q3	2.414 (1.994, 2.922)	< 0.001
P for trend		< 0.001	p for trend		< 0.001
Model 4			Model 4		
Q1	Ref		Q1	Ref	
Q2	1.312 (0.986, 1.746)	0.062	Q2	1.304 (1.060, 1.605)	0.012
Q3	1.765 (1.330, 2.343)	< 0.001	Q3	1.695 (1.373, 2.094)	< 0.001
P for trend		< 0.001	p for trend		< 0.001

Model 1 Univariate model. Model 2adjusted for age, gender and race. Model 3 adjusted for model 2 plus diabetes mellitus, COPD.Model 4 adjusted for model 3 plus lactate, albumin, urea nitrogen, creatinine, AST, monocyte count, hemoglobin, white blood cell count, HDL, LDL, TC, TG and SpO2

Table 3 Logistic models for cardiogenic shock and congestive heart failure

cardiogenic shock			**Congestive heart failure**		
Variable	**OR(95%CI)**	***P* value**	**Variable**	**OR(95%CI)**	***P* value**
Model 1			Model 1		
Q1	Ref		Q1	Ref	
Q2	2.183 (1.473, 3.290)	< 0.001	Q2	1.446 (1.184, 1.767)	< 0.001
Q3	2.128 (1.433, 3.211)	< 0.001	Q3	2.057 (1.678, 2.524)	< 0.001
P for trend		< 0.001	P for trend		< 0.001
Model 2			Model 2		
Q1	Ref		Q1	Ref	
Q2	2.221 (1.493, 3.357)	< 0.001	Q2	1.420 (1.156, 1.745)	0.001
Q3	2.193 (1.466, 3.333)	< 0.001	Q3	1.949 (1.579, 2.409)	< 0.001
P for trend		< 0.001	P for trend		< 0.001
Model 3			Model 3		
Q1	Ref		Q1	Ref	
Q2	2.223 (1.494, 3.363)	< 0.001	Q2	1.393 (1.129, 1.719)	0.002
Q3	2.198 (1.465, 3.348)	< 0.001	Q3	1.843 (1.486, 2.288)	< 0.001
P for trend		< 0.001	P for trend		< 0.001
Model 4			Model 4		
Q1	Ref		Q1	Ref	
Q2	1.930 (1.271, 2.974)	0.002	Q2	1.255 (1.006, 1.565)	0.045
Q3	1.491 (0.939, 2.393)	0.093	Q3	1.565 (1.220, 2.009)	< 0.001
P for trend		0.172	P for trend		< 0.001

Model 1 Univariate model. Model 2adjusted for age, gender and race. Model 3 adjusted for model 2 plus diabetes mellitus, COPD.Model 4 adjusted for model 3 plus lactate, albumin, urea nitrogen, creatinine, AST, monocyte count, hemoglobin, white blood cell count, HDL, LDL, TC, TG and SpO2

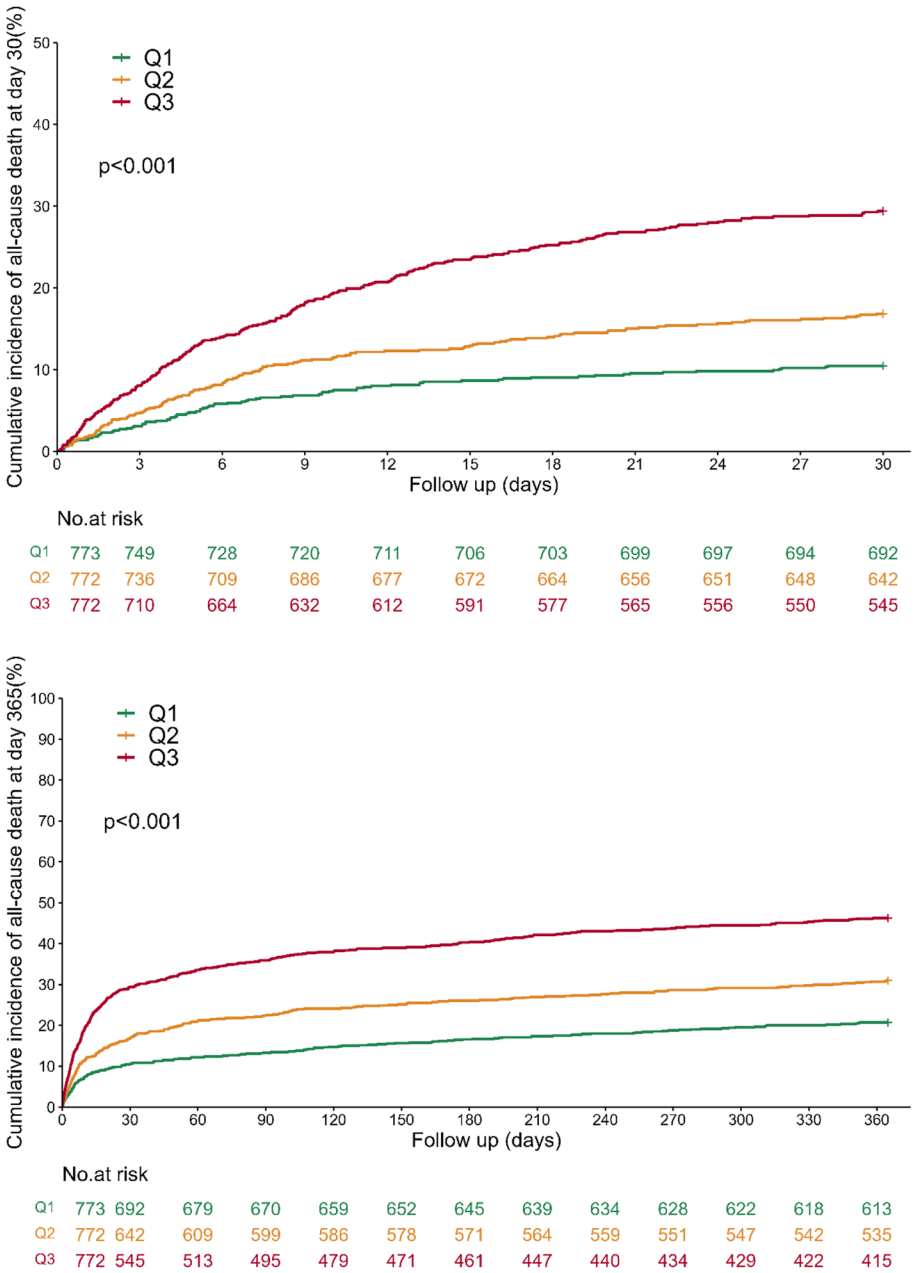


Fig. 1 Kaplan–Meier survival curve of 30-day all-cause mortality and 365-day all-cause mortality stratified by SII

without hypertension [19]. Inflammation is linked to a poor prognosis for acute myocardial infarction and is a crucial mechanism of hypertension-induced vascular endothelial cell damage [9]. It has been demonstrated that the pathophysiological mechanisms connected to inflammation persist long after blood pressure returns to normal [20]. This suggests that in individuals who have both hypertension and acute myocardial infarction, these mechanisms may play a significant role in determining the ongoing risk of disease development. Several indicators of inflammation, such as the C-reactive protein-to-albumin ratio, have been shown to have predictive value for acute and chronic cardiovascular disease. It has been shown that in heart failure patients with reduced ejection fraction and implantable cardiac defibrillators, an elevated C-reactive protein-to-albumin ratio predicts a poorer prognosis [21]. CRP/Albumin Ratio Has Higher Predictive Value Than SII in Predicting Atrial Fibrillation Recurrence After Cryoablation [22]. In the present study, the number of

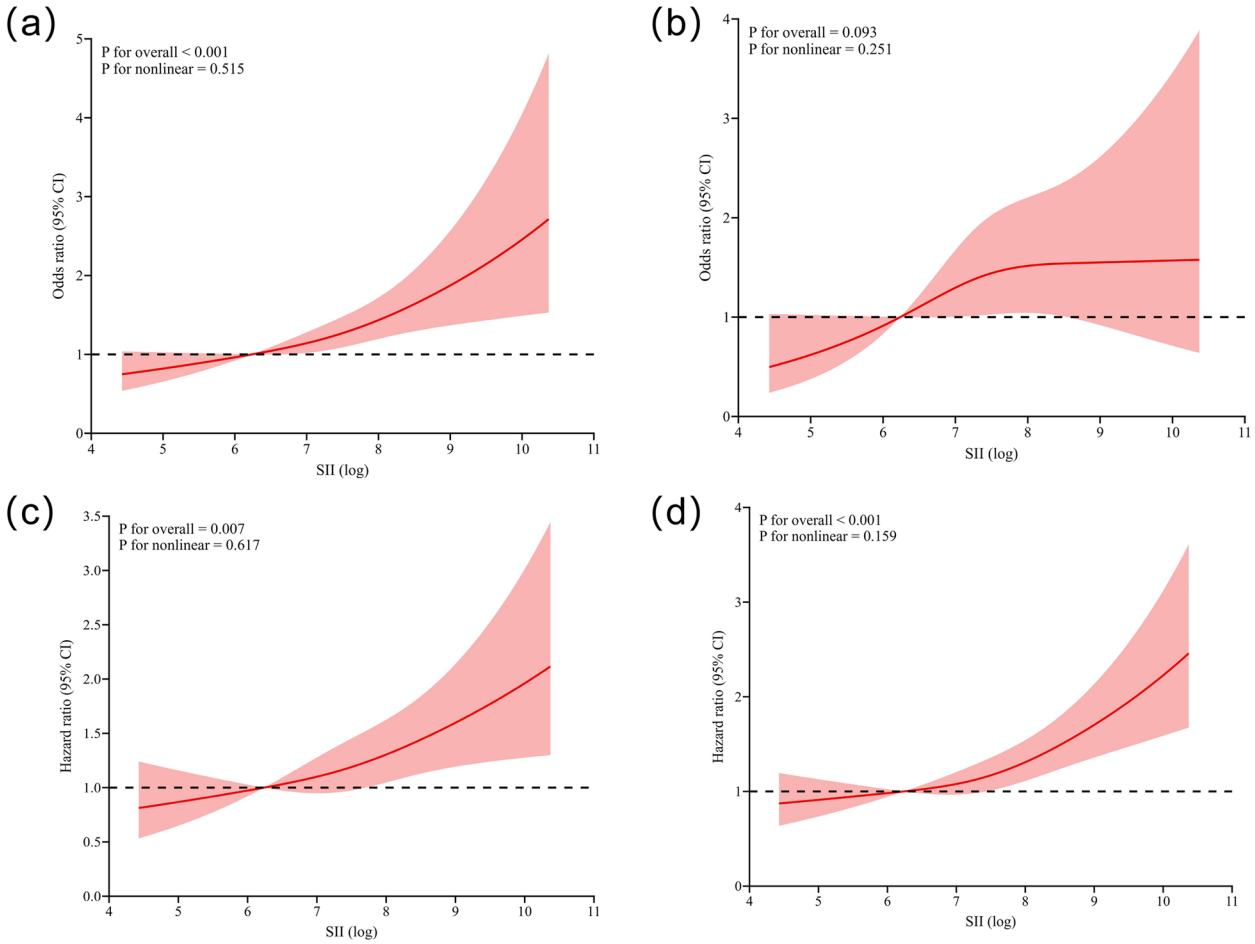
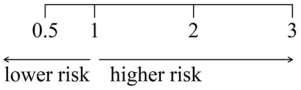


Fig. 2 The adjusted cubic spline model on the association between SII on a continuous scale and adjusted risk of Congestive heart failure (**a**),cardiogenic shock (**b**), 30-day all-cause mortality (**c**) and 365-day all-cause mortality (**d**) in patients with AMI and hypertension

cases with albumin and CRP indexes was too small to be included in the inclusion statistics with a large bias, and therefore were not included for comparison with SII, which is a shortcoming of this study. SII is a new inflammatory marker that has been linked to a number of illnesses, including cancer, heart disease, thickening of the carotid intima-media, elevated urine albumin, stroke, and more [23–27]. SII integrates data from platelet, lymphocyte, and neutrophil counts, largely representing the three immune response pathways—inflammatory, thrombotic, and adaptive. It has been demonstrated that SII is an independent predictor of the prevalence of hypertension and a more effective early warning indicator of systemic inflammation in hypertension. The current study demonstrated that SII was an independent risk factor for 30- and 365-day mortality in patients who had both hypertension and acute myocardial infarction (AMI). This finding suggests that inflammation is a significant factor in determining the short- and long-term prognosis of these patients. The possible pathophysiologic processes are: 1.Thrombotic tendencies and increased leukocyte recruitment are caused by an elevated platelet count and activated platelets, which may result in endothelial cell injury. Hypertension may arise as a result of increased systemic vascular resistance brought on by further endothelial dysfunction [28]. In the meantime, a range of prothrombotic events, including platelet adhesion, activation, and aggregation, are brought on by neutrophil extracellular traps, indicating that neutrophil-platelet interactions might be crucial in the proinflammatory process [29]. More significantly, the renin–angiotensin–aldosterone system in hypertension is intimately linked to low-grade inflammation or autonomous T cell activation [30]. Lastly, oxidative stress in the kidney and artery wall is increased by autoimmune inflammatory infiltrates, which can result in several typical mechanisms of hypertension [31]. 2. The rupture of atherosclerotic plaque depends on both innate and adaptive immunity. In both the development

(a)

Subgroups	N/event	HR (95%CI)	P value	P for interaction
Age				0.232
<60	372/31	1.489 (0.976 - 2.273)	0.065	
60~80	1209/185	1.265 (1.073 - 1.492)	0.005	
>80	736/222	1.127 (0.966 - 1.314)	0.127	
Gender				0.934
Male	1405/239	1.138 (0.985 - 1.314)	0.079	
Female	912/199	1.248 (1.063 - 1.465)	0.007	
Diabetes				0.379
no	1216/219	1.068 (0.922 - 1.237)	0.383	
yes	1101/219	1.268 (1.081 - 1.486)	0.003	
COPD				0.297
no	1709/301	1.229 (1.073 - 1.408)	0.003	
yes	608/137	1.130 (0.945 - 1.352)	0.180	
CABG				0.050
no	1751/418	1.047 (0.940 - 1.166)	0.405	
yes	566/20	1.677 (0.851 - 3.302)	0.135	
PCI				0.971
no	1680/350	1.187 (1.056 - 1.335)	0.004	
yes	637/88	1.056 (0.805 - 1.385)	0.695	
SPO2				0.180
<95%	464/127	1.068 (0.879 - 1.297)	0.507	
≥95%	1853/311	1.252 (1.100 - 1.424)	0.001	



(b)

Subgroups	N/event	HR (95%CI)	P value	P for interaction
Age				0.328
<60	372/61	1.456 (1.057 - 2.007)	0.022	
60~80	1209/321	1.292 (1.136 - 1.470)	<0.001	
>80	736/374	1.131 (1.002 - 1.275)	0.046	
Gender				0.312
Male	1405/397	1.198 (1.066 - 1.348)	0.003	
Female	912/359	1.191 (1.055 - 1.346)	0.005	
Diabetes				0.928
no	1216/369	1.146 (1.020 - 1.288)	0.021	
yes	1101/387	1.232 (1.090 - 1.392)	0.001	
COPD				0.749
no	1709/515	1.186 (1.067 - 1.317)	0.002	
yes	608/241	1.225 (1.061 - 1.413)	0.005	
CABG				0.002
no	1751/712	1.046 (0.961 - 1.138)	0.302	
yes	566/44	1.862 (1.162 - 2.982)	0.010	
PCI				0.881
no	1680/589	1.205 (1.098 - 1.322)	<0.001	
yes	637/167	1.097 (0.902 - 1.333)	0.355	
SPO2				0.006
<95%	464/212	1.024 (0.880 - 1.191)	0.760	
≥95%	1853/544	1.298 (1.174 - 1.436)	<0.001	

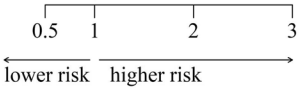


Fig. 3 Association between SII and risk of 30-day all-cause mortality (**a**) and 365-day all-cause mortality (**b**) in subgroups

of acute thrombotic episodes and the pathophysiology of atherosclerosis, platelets are crucial [32]. The start and progression of the atherosclerosis process depend on inflammatory cells. Acute coronary syndromes are directly linked to the development of coronary atherosclerotic plaque and thrombus, which block blood flow in the vicinity of the infarct-related artery. Both the start and development of this process as well as its

negative outcomes depend on inflammation. All things considered, patients who have both hypertension and AMI have a bad prognosis [33]. AMI occurs on the basis of atherosclerosis, which has been shown to be a systemic inflammatory disease, and inflammatory bowel disease and steatohepatitis, which are systemic inflammatory diseases, are strongly associated with cardiovascular events [34, 35].

This study shows that SII is an independent risk factor for congestive heart failure and cardiogenic shock during hospitalization in this cohort, in addition to being linked to short- and long-term prognoses in patients with hypertension and AMI. Patients with AMI frequently experience congestive heart failure, cardiogenic shock, and an elevated risk of long-term mortality. It has been demonstrated that in individuals with AMI, a greater baseline platelet count is a significant and reliable indicator of a bad prognosis. Furthermore, in cardiovascular disease, the platelet-to-lymphocyte ratio (PLR) is a powerful predictor of a bad prognosis [36]. Our analysis yielded data in a similar pattern. According to Lütfi et al. [37], SII is a reliable indicator of hospitalization and long-term death for STEMI patients. SII levels were linked to the no-reflow phenomenon (NRP) in patients with STEMI who received direct PCI, according to research by Kerim et al. Saban et al. claimed that increased SII score was independently associated with contrast nephropathy (CIN) formation in NSTEMI patients undergoing PCI [38, 39]. Both illustrate the important role of inflammation in the development of the disease. It provides new ideas to enhance anti-inflammatory therapy and develop targeted anti-inflammatory drugs in this patient population.

Furthermore, in the current investigation, SII was found to be more strongly related with a poor prognosis in the mortality subgroup analyses conducted on 30-day and 365-day patients who received CABG and had a SpO2 greater than 95%. The idea that SII is linked to the postoperative no-reflow phenomenon in AMI patients undergoing CABG may stem from the fact that patients who underwent CABG for AMI had more complex coronary artery pathology. This, in addition to surgical procedures and other factors that result in a SII, is more strongly associated with a poor prognosis in those whose SpO2 exceeds 95%. This finding contradicts previous research on the relationship between SII and oxygen saturation, perhaps because the SpO2 in this study was derived from the initial post-hospitalization SpO2.

Limitations

This study has certain shortcomings that should be acknowledged and resolved. First, selection bias may exist because this was an observational, retrospective study. Second, during the follow-up period, we did not investigate the impact of dynamic changes in SII levels on cardiovascular events; instead, we only evaluated SII levels at baseline. Therefore, to validate these results, larger sample sizes and extended follow-up are required.

Conclusions

Compared to patients with low SII, the study's findings indicate that patients with high SII in AMI with hypertension had a higher risk of developing congestive heart failure, cardiogenic shock, and 30- and 365-day all-cause death. In individuals with AMI and hypertension, elevated SII levels may be a reliable indicator of worse cardiac outcomes. Crucially, investigation into anti-inflammatory pathways as putative therapeutic targets is necessary, and SII, a readily available, reasonably priced biomarker, might offer fresh approaches to treatment.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12865-025-00690-y.

Supplementary Material 1: Supplementary Fig. 1: The unadjusted cubic spline model on the association between SII on a continuous scale and adjusted risk of Congestive heart failure,cardiogenic shock, 30-day all-cause mortalityand 365-day all-cause mortalityin patients with AMI and hypertension.

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Clinical trial number
Not applicable.

Authors' contributions
TZ and GT designed the study. TZ, CL analyzed and interpreted the data. TZ and SX drafted the manuscript. GT and XL revised the manuscript. All authors gave final approval of the final version to be published.

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Data availability
This study analyzed publicly accessible datasets. This data can be found here: https://mimic.mit.edu/docs/.

Declarations

Ethics approval and consent to participate
The study was performed according to the guidelines of the Helsinki Declaration. The use of the MIMIC-IV database was approved by the review committee of Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center. The data is publicly available (in the MIMIC-IV database), therefore, the ethical approval statement and the requirement for informed consent were waived for this study.

Consent for publication
This manuscript has not been published or presented elsewhere in part or in entirety, and is not under consideration by another journal. All the authors have approved the manuscript and agree with submission to your esteemed journal.

Competing interests
The authors declare no competing interests.

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