Cardiovascular Innovations and Applications

Vol. 7 (2023) 16
ISSN 2009-8618
DOI 10.15212/CVIA.2023.0005

RESEARCH ARTICLE

The Systemic Immune Inflammatory Index Predicts No-Reflow Phenomenon after Primary Percutaneous Coronary Intervention in Older Patients with STEMI

Jiaqi Wang[1], Feifei Zhang[2], Man Gao[2], Yudan Wang[3], Xuelian Song[2], Yingxiao Li[2], Yi Dang[2] and Xiaoyong Qi[2]

[1]*Hebei North University, Zhangjiakou, Hebei Province, China*
[2]*Department of Cardiology, Hebei General Hospital, Shijiazhuang 050051, Hebei Province, China*
[3]*Department of Cardiology, Tianjin Chest Hospital, Tianjin, 300222, China*

Received: 26 October 2022; Revised: 1 December 2022; Accepted: 10 January 2023

Abstract

Purpose: Coronary no-reflow phenomenon (NRP), a common adverse complication in patients with ST-segment elevation myocardial infarction (STEMI) treated by percutaneous coronary intervention (PCI), is associated with poor patient prognosis. In this study, the correlation between the systemic immune-inflammation index (SII) and NRP in older patients with STEMI was studied, to provide a basis for early identification of high-risk patients and improve their prognosis.

Materials and methods: Between January 2017 and June 2020, 578 older patients with acute STEMI admitted to the Department of Cardiology of Hebei General Hospital for direct PCI treatment were selected for this retrospective study. Patients were divided into an NRP group and normal-flow group according to whether NRP occurred during the operation. Clinical data and the examination indexes of the two groups were collected. Logistic regression was used to analyze the independent predictors of NRP, and the receiver operating characteristic curve was used to further analyze the ability of SII to predict NRP in older patients with STEMI.

Results: Multivariate logistic analysis indicated that hypertension (OR=2.048, 95% CI:1.252–3.352, P=0.004), lymphocyte count (OR=0.571, 95% CI:0.368–0.885, P=0.012), platelet count (OR=1.009, 95% CI:1.005–1.013, P<0.001), hemoglobin (OR=1.015, 95% CI:1.003–1.028, P=0.018), multivessel disease (OR=2.237, 95% CI:1.407–3.558, P=0.001), and SII≥1814 (OR=3.799, 95% CI:2.190–6.593, P<0.001) were independent predictors of NRP after primary PCI in older patients with STEMI. Receiver operating characteristic curve analysis demonstrated that SII had a high predictive value for NRP (AUC=0.738; 95% CI:0.686–0.790), with the best cut-off value of 1814, a sensitivity of 52.85% and a specificity of 85.71%.

Conclusion: For older patients with STEMI undergoing primary PCI, SII is a valid predictor of NRP.

Keywords: no-reflow; systemic immune-inflammation index; older patients; ST-segment elevation myocardial infarction

Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is a cardiovascular emergency that threatens patients' lives and health. Timely PCI

Correspondence: **Xiaoyong Qi**, Department of Cardiology, Hebei General Hospital, 348 Heping West Road, Xinhua District, Shijiazhuang, China, E-mail: hbghqxy126.com

©2023 Cardiovascular Innovations and Applications. Creative Commons Attribution-NonCommercial 4.0 International License

to restore the coronary blood supply is critical to improve the prognosis of these patients [1]. PCI reperfusion therapy is the preferred treatment strategy for patients with STEMI, because it can open acutely blocked coronary arteries and save ischemic cardiomyocytes; it also significantly decreases the risk of death in patients with acute myocardial infarction (AMI) and improves prognosis [2]. The incidence rate of NRP in patients with AMI is as high as 20% to 30% [3]. In addition, NRP is associated with adverse cardiovascular events in patients with STEMI [4, 5]. The pathophysiological mechanism of NRP remains unclear but is currently believed to be associated with factors such as distal embolism of diseased vessels, reperfusion injury, microcirculation disturbance, vasospasm, and oxidative stress [6]. SII, a new inflammatory index determined from the counts of neutrophils, lymphocytes, and platelets, can be used to simultaneously evaluate the immune thrombosis state and inflammatory state of the body. Previous studies have studied the relationship between SII and NRP. However, owing to the many complications and poor vascular condition in older patients with STEMI, identifying high-risk patients as soon as possible is essential to improve the prognosis of older patients [7]. Higher SII levels are significantly associated with short-term mortality and NRP in patients with STEMI undergoing PCI [8]. However, no clinical NRP risk prediction method is currently available, and the relationship between the SII level and NRP in older patients with STEMI is unclear. Therefore, this study focused on examining the correlation between SII and NRP in older patients with STEMI, to provide a basis for the early identification of high-risk patients and improvement of their prognosis.

Materials and Methods

Study Sample

This retrospective study included 578 older patients (≥60 years of age) who underwent primary PCI for STEMI at Hebei General Hospital between January 2017 and June 2020. STEMI was defined by chest discomfort or other ischemic symptoms, and development of new ST-segment elevations in two contiguous leads or new bundle branch blocks with an ischemic repolarization pattern. The exclusion criteria were treatment with fibrinolytic drugs, previous coronary artery bypass surgery, inflammatory disease or active infection, autoimmune disease, hematologic diseases, kidney or liver failure, and malignancy. A total of 578 patients were enrolled, and we collected relevant clinical and demographic data through the hospital's electronic medical record system.

This study was approved by the Ethics Committee of Hebei General Hospital. Because this study was retrospective, the requirement to obtain patients' informed consent was waived. This study strictly followed the Declaration of Helsinki.

Coronary Procedures

All enrolled patients were immediately administered a single dose of ticagrelor 180 mg or clopidogrel 300 mg and aspirin 300 mg after being diagnosed with STEMI. Before coronary intervention, patients were administered 100 μg/kg unfractionated heparin intravenously. The Judkins technique was used for coronary angiography. The radial approach was used for most patients, and the femoral approach was used for several patients. TIMI flow rating grades were evaluated by three interventional cardiologists who were blinded to the patients' relevant clinical data. NRP was defined by TIMI blood flow grade ≤2, and no dissection, distal flushing, or spasm occurring during angiography [9]. The patients were divided into an NRP group and normal-flow group according to whether NRP occurred during operation.

Laboratory Measurements

Before coronary intervention, blood samples were collected from patients through the anterior cubital vein. Laboratory examinations included white blood cell count, monocyte count, neutrophil count, lymphocyte count, platelet count, hemoglobin, troponin T level, fasting blood glucose, creatine kinase-MB, total cholesterol, low-density

lipoprotein cholesterol, triglyceride, serum creatinine, high-density lipoprotein, glomerular filtration rate, and other biochemical tests. SII was calculated as neutrophil count × platelet count/lymphocyte count [10]. NLR was calculated as neutrophil count/lymphocyte count. The medications administered during hospitalization followed the relevant clinical guidelines [11]. A cardiologist determined the regimens for angiotensin converting enzyme inhibitors, statins, beta-blockers, and other drugs. A family history of coronary heart disease, diabetes, smoking, hypertension, hyperlipidemia, and medical history of taking related drugs were determined based on patient self-reporting. Experienced echocardiographic physicians used Simpson's method to measure the left ventricular ejection fraction by transthoracic echocardiography.

Statistics

SPSS 26.0 was used for statistical analysis. Kolmogorov-Smirnov test was used to determine the normality of continuous variables. Continuous variables with a normal distribution were analyzed with *t* tests and are expressed as mean ± standard deviation. Continuous variables with non-normal distribution were analyzed with Mann-Whitney U tests. Categorical variables were statistically analyzed with X^2 tests and are expressed as numbers and percentages. Variables with $P<0.05$ were selected for inclusion in univariate and multivariate logistic regression analyses to determine the independent factors influencing NRP. The ability of SII and NLR to predict NRP after PCI in older patients with STEMI was evaluated with receiver operating characteristic (ROC) curve analysis, and the best cut-off value was determined. Multiple linear regression was used to analyze the correlations of independent factors with NRP and SII. Both tests were two sided. When $P<0.05$, differences were considered statistically significant.

Results

Baseline characteristics are listed in Table 1. The study population included 578 patients (mean age 69.95 ± 7.52 years, 183 women [31.7%]), 123 (21.3%) of whom were in the NRP group. Compared with the normal-flow group, the NRP group had a higher incidence of hypertension and smoking ($P<0.05$). The age, body mass index (BMI), sex, diastolic blood pressure, systolic blood pressure, heart rate, previous drug use, hospitalization drug use, door-to-balloon time, left ventricular ejection fraction, Killip grade at admission ≥2, and previous medical history showed no significant differences between groups.

The laboratory data and angiography results for the two groups of patients are shown in Table 2. The median SII was significantly higher in the NRP group than the normal flow group [1909.29 (1010.61–2854.94) vs 1088.76 (679.45–1546.71), $P<0.001$, Figure 1]. The white blood cell count, neutrophil count, platelet count, hemoglobin level, platelet to lymphocyte ratio, neutrophil-to-lymphocyte ratio, incidence of multivessel disease, and IABP use were significantly higher in the NRP group than the normal flow group, whereas the NRP group had lower lymphocyte counts. No significant differences were observed in lesion vessel, stent length, and stent diameter between groups.

ROC curve analysis indicated that SII had good predictive value for NRP in older patients with STEMI (AUC, 0.738; 95% CI: 0.686–0.790), with an optimal cut-off of 1814, a sensitivity of 52.85%, and a specificity of 85.71%. As shown in Figure 2 and Table 3, the predictive power of SII was greater than those of NLR (AUC: 0.688; 95% CI: 0.634–0.742) and PLR (AUC: 0.722; 95% CI: 0.667–0.776).

The univariate and multivariate logistic regression results are shown in Table 4. Collinearity was observed for NLR, PLR, white blood cell count, and neutrophil count; therefore, these variables were not included in the regression analysis. Multivariate analysis showed that SII ≥ 1814 (OR = 3.799, 95% CI: 2.190–6.593, $P<0.001$), hypertension (OR = 2.048, 95% CI: 1.252–3.352, $P=0.004$), lymphocyte count (OR = 0.571, 95% CI:0.368–0.885, $P=0.012$), platelet count (OR = 1.009, 95% CI:1.005–1.013, $P<0.001$), hemoglobin (OR = 1.015, 95% CI:1.003–1.028, $P=0.018$), and multivessel disease (OR = 2.237, 95% CI: 1.407–3.558, $P=0.001$) were independent predictors of NRP after primary PCI in older patients with STEMI.

Multiple linear regression analysis indicated no correlations of SII with hypertension, smoking, and multivessel disease ($P>0.05$), as shown in Table 5.

Table 1 Baseline Information Between Groups.

Characteristics	Normal-flow (n = 455)	No reflow (n = 123)	P value
Age (year)	70.02 ± 7.60	69.67 ± 7.21	0.650
BMI (kg/m^2)	25.35 (23.44–27.68)	25.53 (23.40–27.42)	0.802
Male, gender (n, %)	308 (67.7%)	87 (70.7%)	0.520
Systolic blood pressure (mmHg)	129.62 ± 25.80	127.07 ± 25.65	0.332
Diastolic blood pressure (mmHg)	78.66 ± 15.24	76.35 ± 15.33	0.137
Heart rate (bpm)	74.29 ± 19.26	75.85 ± 19.25	0.428
Family history of coronary heart disease (n, %)	41 (9.0%)	12 (9.8%)	0.799
Diabetes mellitus (n, %)	128 (28.1%)	35 (28.5%)	0.944
Smoking (n, %)	199 (43.7%)	67 (54.5%)	0.034
Hypertension (n, %)	258 (56.7%)	87 (70.7%)	0.005
History of hyperlipidemia (n, %)	43 (9.5%)	10 (8.1%)	0.653
Time from pain to intervention (≤6 h)	359 (78.9%)	98 (79.7%)	0.852
Door-to-balloon time (min)	60.00 (46.00–83.00)	61.00 (46.00–86.75)	0.606
Left ventricular ejection fraction (n, %)	54 (46–60)	56 (48–60)	0.134
Killip class ≥2 at admission (n, %)	171 (37.6%)	47 (38.2%)	0.898
Previous drug used			
β-blocker (n, %)	92 (20.2%)	23 (18.7%)	0.708
Statins (n, %)	35 (7.7%)	9 (9.4)	0.889
Clopidogrel (n, %)	49 (10.8%)	12 (9.8%)	0.746
Aspirin (n, %)	98 (21.5%)	21 (17.1%)	0.277
ACEI/ARB (n, %)	51 (11.2%)	14 (11.4%)	0.957
In-hospital ACEI therapy (n, %)	317 (69.7%)	84 (68.3%)	0.769
In-hospital statin therapy (n, %)	449 (98.7%)	120 (97.6%)	0.631
In-hospital diuretics therapy (n, %)	286 (62.9%)	66 (53.7%)	0.064
In-hospital β-blocker therapy (n, %)	375 (82.4%)	104 (84.6%)	0.577

ACEI/ARB, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; BMI, body mass index.

Discussion

Inflammation plays a central role in the occurrence and development of AMI through the complex interactions between inflammatory cells and inflammatory mediators [12]. Some studies have shown that the inflammatory state after STEMI is associated with left ventricular thrombus [13].

This study investigated the relationship between SII level and NRP in older patients with STEMI undergoing percutaneous coronary intervention. The baseline SII of older patients with STEMI with NRP after coronary intervention was significantly higher than that of patients with normal flow. Furthermore, in older patients with STEMI, SII levels were independently associated with NRP.

NRP is a serious complication of acute STEMI that can lead to adverse cardiovascular events. The pathophysiological mechanism of NRP has not yet been elucidated but is currently believed to be associated with factors such as ischemia-reperfusion injury, distal embolism of diseased vessels, microcirculation disturbance, oxidative stress, and vasospasm [14]. Because no effective treatment for NRP is currently available, predicting the occurrence of no-reflow, early assessment of high-risk patients, and the use of appropriate interventions to decrease the occurrence of no-reflow would benefit patients [15, 16]. In recent years, many studies have evaluated various indicators for predicting RNP after primary PCI in patients with STEMI, but no general consensus has been reached regarding widely accepted reliable predictors. Elevated NLR levels before interventional therapy appear to predict the risk of NRP after primary PCI in patients with AMI [17]. Another study has found that PLR is a reliable

Table 2 Laboratory Examinations and Angiography Results for the Two Groups.

Variable	Normal-flow (n = 455)	No reflow (n = 123)	P value
White blood cell count ($\times 10^9$/L)	9.12 (7.31–11.03)	10.37 (8.63–12.46)	<0.001
Monocyte count ($\times 10^9$/L)	0.41 (0.31–0.54)	0.44 (0.33–0.59)	0.173
Neutrophil count ($\times 10^9$/L)	7.15 (5.21–9.04)	8.62 (6.55–10.49)	<0.001
Lymphocyte count ($\times 10^9$/L)	1.41 (1.09–1.83)	1.07 (0.92–1.51)	<0.001
Platelet count ($\times 10^9$/L)	213 (181–249)	252 (225–278)	<0.001
Hemoglobin (g/dL)	132 (121–144)	137 (125.25–149)	0.011
Serum glucose (mg/dL)	113.22 (94.32–150.12)	122.58 (94.14–187.20)	0.190
Peak cardiac troponin T (ng/mL)	978 (870–1247.75)	958.50 (869.75–1229.50)	0.820
Peak creatine kinase-MB (U/L)	130.55 (60.74–240.89)	139.67 (79.17–270.51)	0.063
NT-pro BNP peak (pg/mL)	1771.18 ± 2703.04	1612.97 ± 2171.23	0.550
Total cholesterol (mg/dL)	76.5 (65.7–88.92)	80.64 (67.14–94.86)	0.117
Triglyceride (mg/dL)	20.7 (15.84–28.8)	22.68 (17.28–32.94)	0.139
High-density lipoprotein cholesterol (mg/dL)	18.54 (16.02–21.24)	18.54 (16.38–21.78)	0.687
Low-density lipoprotein cholesterol (mg/dL)	50.58 (41.4–59.76)	54 (42.48–63.18)	0.054
Serum creatinine (mg/dL)	77.25 (66.93–88.90)	78.48 (65.76–92.88)	0.568
Glomerular filtration rate (mL/min/1.73 m^2)	81.60 (67.04–90.53)	82.28 (69.75–90.99)	0.326
Uric acid (mg/dL)	310.24 (257.96–381.80)	329.20 (263.28–407.49)	0.211
Systemic immune-inflammation index	1088.76 (679.45–1546.71)	1909.29 (1010.61–2854.94)	<0.001
Monocyte to high-density lipoprotein cholesterol ratio	0.41 (0.30–0.56)	0.44 (0.32–0.56)	0.459
Platelet to lymphocyte ratio	147.69 (117.89–196.25)	235.09 (154.61–286.27)	<0.001
Neutrophil-lymphocyte ratio	5.04 (3.28–7.13)	7.71 (4.80–10.83)	<0.001
Culprit vessel (n, %)			0.057
LAD	193 (42.4%)	67 (54.5%)	
LCX	84 (18.5%)	17 (13.8%)	
RCA	178 (39.1%)	39 (31.7%)	
Multivessel disease	173 (38.0%)	71 (57.7%)	0.001
Thrombus aspiration (n, %)	124 (27.3%)	33 (26.8%)	0.925
Initial TIMI flow grade (n, %)			0.266
0–1	267 (58.7%)	79 (64.2%)	
≥2	188 (41.3%)	44 (35.8%)	
Number of stent	1.13 ± 0.421	1.17 ± 0.474	0.326
Stent diameter (mm)	3.00 (2.75–3.50)	3.00 (2.75–3.50)	0.168
Stent length (mm)	24 (18–32)	24 (18–31.50)	0.654
IABP used in procedure	28 (6.2%)	15 (12.2%)	0.023

SII, systemic immune-inflammation index; NT-proBNP, N-terminal pro brain natriuretic peptide; PLR, platelet to lymphocyte ratio; NLR, neutrophil-lymphocyte ratio; LCX, left circumflex; LAD, left anterior descending; RCA, right coronary artery; IABP, intra-aortic balloon pump; TIMI, thrombolysis in myocardial infarction.

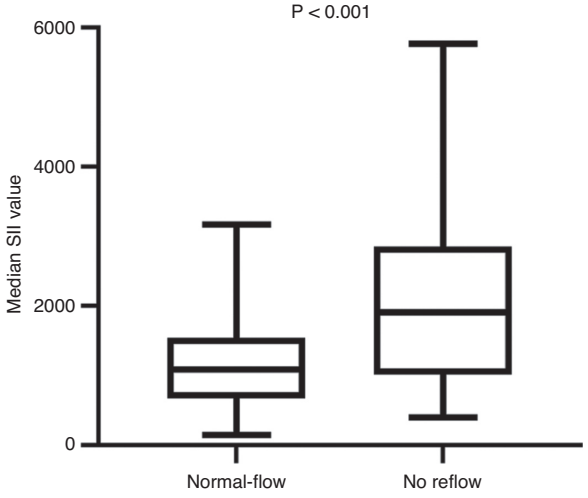

Figure 1 Median Systemic Immune-Inflammation Index Levels Between Groups.

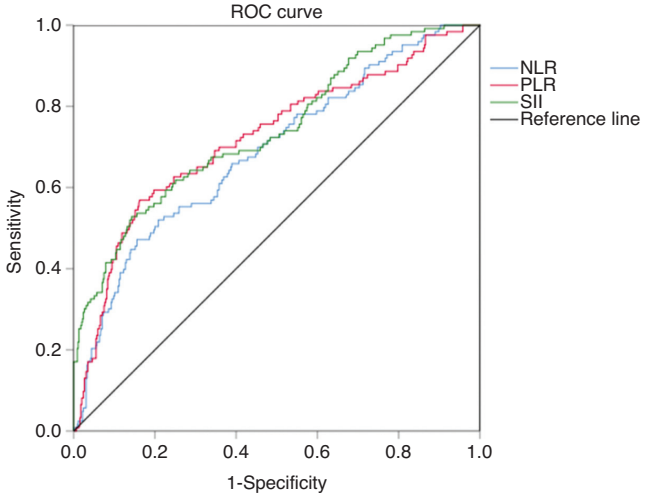

Figure 2 ROC Curve Analysis.

predictor of no-reflow after thrombus aspiration in patients with STEMI [18]. Zhang et al. [19] have compared the relationships of NLR, mean platelet volume, and platelet distribution width with NRP in patients with STEMI. Their findings indicated that NLR, mean platelet volume, and platelet distribution width were independent predictors of NRP in patients with STEMI; although NLR had the highest predictive value, the predictive value of these metrics did not differ significantly. Admission vitamin D levels have been associated with NRP and may predict NRP after PCI in patients with STEMI [20]. The model for end-stage liver disease-XI (MELD-XI) score can be used to predict NRP and short-term prognosis in patients with STEMI [21]. In addition, Rashed et al. [22] have shown that the preoperative CHA2DS2-VASc score can be used to predict NRP in patients with STEMI.

Cells of the innate immune system, such as lymphocytes and neutrophils, promote inflammatory responses in the vascular endothelium, and directly influence oxidative stress and cytokine production [23]. Platelet activation has been reported to play a key role in the coagulation system, which has a complex association with the innate immune system [24]. The SII is calculated from neutrophils, lymphocytes, and platelets, and it reflects the body's immune thrombotic status and inflammatory status simultaneously [7]. SII has been reported to predict new-onset atrial fibrillation after STEMI [25]. As a new inflammatory index, SII can predict the in-hospital and long-term outcomes of patients with STEMI after PCI [26].

This study showed that the baseline SII level of NRP was significantly higher in the group of older patients with STEMI after coronary intervention than in the normal-flow group; in addition, previous hypertension, neutrophils, platelets, hemoglobin, and multivessel disease were independent predictors of NRP in elderly STEMI patients. Baseline SII levels of NRP were significantly higher in older patients with STEMI after PCI than in the normal-flow group. In addition, platelets, hemoglobin, multivessel disease, previous hypertension, and lower

Table 3 ROC Analysis of the Indicated Variables in Predicting the No-Reflow Phenomenon.

Variable	AUC	95% CI	Cut-off	Sen.	Spe.	Youden index	P value
SII	0.738	0.686–0.790	1814	52.85	85.71	0.386	<0.001
PLR	0.722	0.667–0.776	222.5	56.91	83.74	0.407	<0.001
NLR	0.688	0.634–0.742	8.264	47.15	84.40	0.316	<0.001

PLR, platelet to lymphocyte ratio; SII, systemic immune-inflammation index; CI, confidence interval; NLR, neutrophil-lymphocyte ratio.

Table 4 Effects of the Indicated Variables on No Reflow in Univariate and Multivariate Regression Analyses.

Variable	Univariate analysis			Multivariate analysis		
	OR	95% CI	P value	OR	95% CI	P value
Smoking	1.539	1.031–2.297	0.035	1.560	0.979–2.487	0.061
Hypertension	1.845	1.200–2.838	0.005	2.048	1.252–3.352	0.004
Lymphocyte count	0.464	0.314–0.686	<0.001	0.571	0.368–0.885	0.012
Hemoglobin	1.015	1.004–1.027	0.008	1.015	1.003–1.028	0.018
Platelet count	1.010	1.006–1.013	<0.001	1.009	1.005–1.013	<0.001
SII ≥ 1814	6.605	4.255–10.254	<0.001	3.799	2.190–6.593	<0.001
Multivessel disease	2.226	1.485–3.336	<0.001	2.237	1.407–3.558	0.001
IABP used in procedure	2.118	1.093–4.105	0.026	1.907	0.854–4.258	0.115

IABP, intra-aortic balloon pump; OR, odds ratio; CI, confidence interval; SII, systemic immune-inflammation index.

Table 5 Multivariate Linear Regression Analysis of Hypertension, Smoking, Multivessel Disease, and SII.

Variable	B	SE	β	t	P value
Hypertension	13.038	225.236	0.005	0.058	0.954
Multivessel disease	200.208	207.414	0.088	0.965	0.336
Smoking	19.915	205.792	0.009	0.097	0.923

lymphocyte levels were independent predictors of NRP in these patients. These findings are similar to the results of previous studies [27, 28]. However, the average SII in this study was higher, possibly because of the chronic low-grade sterile inflammation caused by mitochondrial dysfunction, cellular senescence, autophagy deficiency, and dysbiosis of gut flora in older patients [27]. Our findings, including the results of ROC curve analysis, indicated that the predictive ability of SII for NRP in older patients with STEMI was higher than that of NLR and PLR. Therefore, SII may serve as a risk stratification index for older patients with STEMI and NRP.

Limitations

This study has several limitations. 1. This study was a single center retrospective study with a relatively inadequate sample size. 2. Only angiography was used to determine the presence or absence of reflow, and intracavitary imaging techniques, such as optical coherence tomography and intravascular ultrasound, were not used to further evaluate vascular lesions. 3. The patients included in this study were typical older patients with STEMI. 4. Data on clinical indicators such as pre-expansion, post-expansion, and thrombus load were not available. 5. Because of the lack of follow-up data, we were unable to further determine the relationship between SII and the poor long-term prognosis of older patients with acute myocardial infarction; continue to follow up the occurrence of poor prognosis in patients.

Conclusion

For older patients with STEMI undergoing primary PCI, SII was significantly correlated with NRP. SII has the potential to serve as a risk stratification index of these patients.

Data Availability Statement

If necessary, the original data supporting the conclusions of this article are available with the consent of the corresponding author.

Conflict of Interest

No potential conflicts of interest are reported by the authors.

Author Contributions

Conceptualization, Jiaqi Wang and Feifei Zhang; Data curation, Feifei Zhang and Yi Dang; Formal analysis, Xuelian Song and Yi Dang; Funding acquisition, Xiaoyong Qi; Investigation, Man Gao and Yingxiao Li; Resources, Yingxiao Li; Software, Man Gao and Xuelian Song; Supervision, Xiaoyong Qi; Writing – original draft, Jiaqi Wang and Yudan Wang; Writing – review & editing, Xiaoyong Qi.

REFERENCES

1. Ashoori A, Pourhosseini H, Ghodsi S, Salarifar M, Nematipour E, Alidoosti M, et al. CHA2DS2-VASc score as an independent predictor of suboptimal reperfusion and short-term mortality after primary PCI in patients with acute ST segment elevation myocardial infarction. Medicina (Kaunas) 2019;55(2):35.
2. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, et al. 2015 ACC/AHA/SCAI focused update on primary percutaneous coronary intervention for patients with ST-elevation myocardial infarction: an update of the 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention and the 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. J Am Coll Cardiol 2016;67(10):1235–50.
3. Wang Q, Shen H, Mao H, Yu F, Wang H, Zheng J. Shock index on admission is associated with coronary slow/no reflow in patients with acute myocardial infarction undergoing emergent percutaneous coronary intervention. J Interv Cardiol 2019;2019:7873468.
4. Kumar J, O'Connor CT, Kumar R, Arnous SK, Kiernan TJ. Coronary no-reflow in the modern era: a review of advances in diagnostic techniques and contemporary management. Expert Rev Cardiovasc Ther 2019;17(8):605–23.
5. Kim MC, Cho JY, Jeong HC, Lee KH, Park KH, Sim DS, et al. Long-term clinical outcomes of transient and persistent no reflow phenomena following percutaneous coronary intervention in patients with acute myocardial infarction. Korean Circ J 2016;46(4):490–8.
6. Hu X, Yang X, Li X, Li G, Zhou Y, Dong H. Elevated uric acid is related to the no-/slow-reflow phenomenon in STEMI undergoing primary PCI. Eur J Clin Invest 2022;52(4):e13719.
7. Esenboğa K, Kurtul A, Yamantürk YY, Tan TS, Tutar DE. Systemic immune-inflammation index predicts no-reflow phenomenon after primary percutaneous coronary intervention. Acta Cardiol 2022;77(1):59–65.
8. Vatan MB, Çakmak AC, Ağaç S, Eynel E, Erkan H. The systemic immune-inflammation index predicts impaired myocardial perfusion and short-term mortality in ST-segment elevation myocardial infarction patients. Angiology 2022;0(0):1–9.
9. Niccoli G, Burzotta F, Galiuto L, Crea F. Myocardial no-reflow in humans. J Am Coll Cardiol 2009;54(4):281–92.
10. Hu B, Yang XR, Xu Y, Sun YF, Sun C, Guo W, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res 2014;20(23):6212–22.
11. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39(2):119–77.
12. Kraemer BF, Martinovic I, Lindemann S. Human platelets release TGFBIp in acute myocardial infarction. Heart Vessels 2022;37:1962–70.
13. Lechner I, Reindl M, Tiller C, Holzknecht M, Fink P, Plangger J, et al. Association between inflammation and left ventricular thrombus formation following ST-elevation myocardial infarction. Int J Cardiol 2022;361:1–6.
14. Allencherril J, Jneid H, Atar D, Alam M, Levine G, Kloner RA, et al. Pathophysiology, diagnosis, and management of the no-reflow phenomenon. Cardiovasc Drugs Ther 2019;33(5):589–97.
15. Zorlu Ç, Köseoğlu C. Comparison of RCHA2DS2-VASc score and CHA2DS2-VASc score prediction of no-reflow phenomenon in patients with ST-segment elevation myocardial infarction. Turk Kardiyol Dern Ars 2020;48(7):664–72. English.
16. Aggarwal P, Rekwal L, Sinha SK, Nath RK, Khanra D, Singh AP. Predictors of no-reflow phenomenon following percutaneous coronary intervention for ST-segment elevation myocardial infarction. Ann Cardiol Angeiol (Paris) 2021;70(3):136–42.
17. Namazi M, Mahmoudi E, Safi M, Jenab Y, Vakili H, Saadat H, et al. The no-reflow phenomenon: is it predictable by demographic factors and routine laboratory data? Acta Biomed 2021;92(5):e2021297.
18. Şenöz O, Emren SV, Erseçgin A, Yapan Emren Z, Gül İ. Platelet-Lymphocyte ratio is a predictor for the development of no-reflow phenomenon in patients with ST-segment elevation myocardial infarction after thrombus aspiration. J Clin Lab Anal 2021;35(6):e23795.
19. Zhang Q, Hu M, Sun J, Ma S. The combination of neutrophil-to-lymphocyte ratio and platelet

correlation parameters in predicting the no-reflow phenomenon after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. Scand Cardiovasc J 2020;54(6):352–7.
20. Şen Ö, Şen SB, Topuz AN, Topuz M. Vitamin D level predicts angiographic no-reflow phenomenon after percutaneous coronary intervention in patients with ST segment elevation myocardial infarction. Biomark Med 2021;15(15):1357–66.
21. Zhang XT, Lin ZR, Zhang L, Zhao ZW, Chen LL. MELD-XI score predict no-reflow phenomenon and short-term mortality in patient with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. BMC Cardiovasc Disord 2022;22(1):113.
22. Rashed MI, Saleh MA, Elfekky EM, Elmahmoudy AM. CHA2DS2 VASc score and brachial artery flow-mediated dilation as predictors for no-reflow phenomenon in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Egypt Heart J 2022;74(1):13.
23. Striz I, Brabcova E, Kolesar L, Sekerkova A. Cytokine networking of innate immunity cells: a potential target of therapy. Clin Sci (Lond) 2014;126(9):593–612.
24. Lentz BR. Exposure of platelet membrane phosphatidylserine regulates blood coagulation. Prog Lipid Res 2003;42(5):423–38.
25. Bağcı A, Aksoy F. Systemic immune-inflammation index predicts new-onset atrial fibrillation after ST elevation myocardial infarction. Biomark Med 2021;15(10):731–9.
26. Orhan AL, Şaylık F, Çiçek V, Akbulut T, Selçuk M, Çınar T. Evaluating the systemic immune-inflammation index for in-hospital and long-term mortality in elderly non-ST-elevation myocardial infarction patients. Aging Clin Exp Res 2022;34(7):1687–95.
27. Bochaton T, Leboube S, Paccalet A, Crola Da Silva C, Buisson M, Mewton N, et al. Impact of age on systemic inflammatory profile of patients with ST-segment-elevation myocardial infarction and acute ischemic stroke. Stroke 2022;53(7):2249–59.
28. Anh DT, Minh HV, Binh HA, Bao TQ, Hai NTT, Nam LX, et al. Age related differences in acute coronary syndrome: an observation at a central hospital in Vietnam. J Transl Int Med 2021;9(1):32–7.