

Prognostic Value of Systemic Immune-Inflammation Index and NT-proBNP in Patients with Acute ST-Elevation Myocardial Infarction

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Objective: Our aim was to assess systemic immune-inflammation index (SII) and NT-proBNP value either in singly or in combination to predict acute ST-elevation myocardial infarction (STEMI) patient prognosis.

Methods: Analyzed retrospectively the clinical features and laboratory data of STEMI confirmed patients in our hospital from January to December 2020. The levels of SII and NT-proBNP were detected. The Kaplan-Meier approach and Spearman's rank correlation coefficient were used to construct the overall major adverse cardiac event (MACE) curve. Multivariate Cox regression analysis was applied to detect MACE predictors. In addition, the DeLong test and receiver operating characteristic (ROC) curve analyzed each factor performance on its own and composite multivariate index to predict MACEs.

Results: The MACE group showed statistically significant differences in SII, NT-proBNP in comparison to the non-MACE group ($P=0.003$, $P<0.001$). Based on Kaplan-Meier analysis, SII and NT-proBNP showed positive correlation with MACE (log-rank $P<0.001$). SII and NT-proBNP were independent predicting factors for long-term MACEs in multivariate Cox regression analysis ($P<0.001$, HR: 2.952, 95% CI 1.565–5.566; $P<0.001$, HR: 2.112, 95% CI 1.662–2.683). SII and NT-proBNP exhibited a positive correlation ($R=0.187$, $P<0.001$) in correlation analysis. According to the ROC statistical analysis, the combination exhibited 78.0% sensitivity and 88.0% specificity in the prediction of MACE. According to the results of the AUC and DeLong test, the combined SII and NT-proBNP performed better as a prognostic index than each of the individual factor indexes separately ($Z=2.622$, $P=0.009$; $Z=3.173$, $P<0.001$).

Conclusion: SII and NT-proBNP were independent indicators of clinical prognosis in acute STEMI patients, and they correlated positively. These factors could be combined to improve clinical prognosis.

Keywords: systemic immune-inflammation index, NT-proBNP, STEMI, MACEs, risk assessment

Introduction

ST-segment elevation myocardial infarction (STEMI) has been recognized as the disease with the highest mortality and morbidity in the world for many years; ruptured atherosclerotic plaque is the most common cause of STEMI.¹ As emergency intervene technology matures and gains acceptance in clinical practices, emergency intervention therapy has become the most appropriate treatment for acute STEMI. Percutaneous coronary intervention (PCI) showed clinical prognosis improvement by increasing of myocardial blood flow perfusion.² However, STEMI patients are still at risk for long-term adverse cardiovascular events (MACEs).³ Studies have shown that current smoking, history of heart failure, and diabetes mellitus are independent predictors of MACEs after STEMI.^{4,5} SII and NT-proBNP levels are rarely reported to be associated with MACEs.

Several studies have demonstrated that inflammation contributes to the progress of atherosclerotic ischemic heart disease. Research showed that the systemic immune-inflammation index (SII), a combination of lymphocytes, neutrophils, and platelets, can predict the development of cancer.⁶ According to several recent studies, the SII is correlated with adverse consequences in patients with cardiovascular disease, including atrial fibrillation, chronic heart failure and cardiomyopathy.^{7,8}

NT-proBNP is a prohormone released by cardiomyocytes, mainly in response to increased wall-filling stress in cardiac chambers, which then leaks into the peripheral circulation.^{9,10} NT-proBNP has also been identified as a strong independent prognostic marker in different cardiac patients. As a result of local stretch mechanisms in the area surrounding the ischemic region, NT-proBNP production increases after acute myocardial ischemia.¹¹ It has been shown in recent years that elevated NT-proBNP is linked to a better outcome, and the most studied outcome is in-hospital/30-day mortality.^{12,13}

Both SII and NT-proBNP may independently predict the severity and outcome of cardiovascular disease. This is conducive to early stratification and preventing the occurrence of MACEs. The objective of current study is to assess predictive valuableness of SII, NT-proBNP, alone and combined for the clinical outcomes of STEMI patients.

Materials and Methods

Design and Study Population

This is a retrospective observational cohort study of patients with STEMI diagnosed at a single center at Xuzhou Medical University's Affiliated Hospital between January and December 2020.

The criteria for the early diagnosis of STEMI include the following: (I) typical angina and/or signs of ischemia at rest lasting more than 20 minutes; (ii) the occurrence of ST-segment elevation aligns with Myocardial Infarction of 2 mm or higher in adjacent chest leads and/or ST-segment elevation of ≥ 1 mm in $2 \geq$ standard leads or the existence of a new (or probably new) left bundle branch block on electrocardiogram at admission; and (iii) positive indicators of myocardial injury.¹⁴

Patients excluded were those with chronic heart failure (New York Heart Association (NYHA) class 3 or higher), severe chronic renal failure or severe hepatic impairment, inflammation (including ongoing infection, systemic inflammation, or autoimmune disease), cancer, stroke, valvular heart disease, hematologic disorders, pregnant.

The study initially enrolled 389 patients. However, a sum of 34 patients were exempted from follow up and a sum of 355 patients were enrolled as a result, including 262 males (78.0%), with an average age of 63.40 ± 12.72 years. The study protocol was accepted by the Affiliate of Xuzhou Medical University's Ethics Committee in compliance with the Helsinki Declaration. Participants provided written/oral informed consent. A combination of medical records and telephone interviews was used in the study to collect data. The flow chart for the study displayed in Figure 1.

Clinical and Demographic Characteristics

In the first 24 hours following admission, patient's blood samples were collected. Medical records were reviewed for demographic and clinical characteristics, including gender, age, diabetes and hypertension histories, smoking status, and medication histories. Left ventricular ejection fraction (LVEF) values and Lab results were collected includes lipid profile, complete blood count, NT-proBNP, and fibrinogen, as well as fasting blood-glucose (FBG), alcohol consumption, glycated hemoglobin, uric acid, and urea nitrogen. The SII was calculated based on the absolute peripheral neutrophil count \times platelet count/lymphocyte count.

Division of Patient Groups

From the medians of SII and NT-proBNP, patients were grouped into four groups (Group Q1, $SII \leq 1240.61$ or $NT\text{-}proBNP \leq 280.53$; Group Q2, $SII > 1240.61$ and $NT\text{-}proBNP > 280.53$). In Group Q1 (low SII/low NT-proBNP), $n = 263$; in Group Q2 (high SII + high NT-proBNP), $n = 92$. Subsequently, two groups of patients were formed ($n = 100$) and ($n = 255$) based on whether they experienced MACEs throughout follow up.

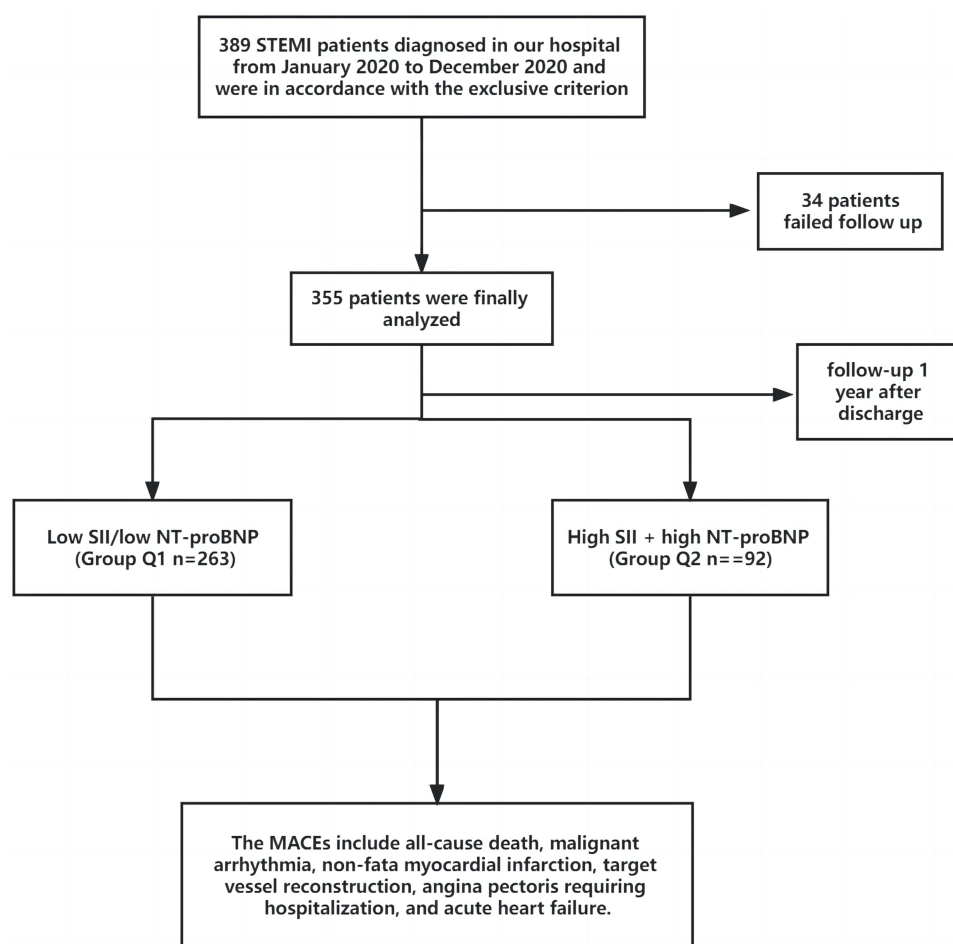


Figure 1 The flow chart of participants inclusion.

Clinical Endpoint and Follow Up

The MACEs occurred during the hospitalization or within 1 year after discharge were recorded, including deaths from all causes, not fatal MI, malignant arrhythmia, target vessel reconstruction, unstable angina, acute heart failure, and non-fatal stroke.

Statistical Analysis

For the analysis, SPSS 26.0 for Windows (SPSS Inc., Chicago, IL, USA) was used. Counts and percentages (%) are expressed for categorical variables, while mean \pm standard deviations or medians and quartile intervals (%) are expressed for continuous variables. Independent sample *t*-tests were employed for comparison between parameter values among groups, Mann–Whitney *U*-tests were applied for comparison between non-parameter values across groups, and for comparing classified variables, the chi-square (χ^2) test was carried out. Pearson correlation coefficient analysis was used to investigate correlation between SII and NT-proBNP. The prognosis predictive value throughout follow-up examined with Cox regression analysis. Kaplan-Meier analysis was used to calculate the cumulative incidence of long-term outcomes, and the Log rank test was used to compare between groups. $P < 0.05$ was regarded as significant. Receivers operating characteristic (ROC) curves were created to examine the predictive value of risk variables. It was determined that the diagnostic effect was better when ROC curves were compared. The Delong test was applied to analyze the SII, NT-proBNP and combined indexes in predicting the efficacy of MACE.

Results

Baseline Clinical Characteristics of Patients

A sum of 355 individuals were enrolled in the research who were clinically diagnosed with STEMI and were followed for one year. Table 1 displays the clinical, ultrasonic cardiogram, and lab results for the study participants. Except for diuretics, there were no statistically significant differences between the two groups in terms of drug administration. Patients in Group Q2 were markedly older than those in Group Q1, and in Group Q2, SII, NT-proBNP, WBC count, neutrophil count, platelet count, CKMB were higher. In Group Q2, lymphocyte count, total cholesterol, and creatinine were lower than those of Group Q1.

Table 1 Clinical and Laboratory Characteristics of Enrolled Patients According to SII and NT-proBNP

Variable	Low SII/Low NT-proBNP (Group Q1 n=263)	High SII+ High NT-proBNP (Group Q2 n=92)	P
Age (years, $\bar{x} \pm s$)	62.24 \pm 12.97	66.49 \pm 11.51	0.005
Gender			
Male, n (%)	196 (74.5)	66 (71.7)	0.178
Laboratory parameters			
WBC, ($10^9/L$)	9.833 \pm 3.084	11.076 \pm 2.813	0.001
Neut, ($10^9/L$)	7.69 (5.67–9.81)	9.86 (8.41–11.83)	<0.001
Lymph, ($10^9/L$)	1.70 (1.20–2.40)	0.90 (0.80–1.20)	<0.001
RBC, ($10^9/L$)	4.719 \pm 0.544	4.592 \pm 0.463	0.069
HB, (g/L)	143.99 \pm 16.979	138.75 \pm 15.449	0.008
PLT, ($10^9/L$)	199.00 (166.75–239.25)	228.00 (194.00–262.00)	<0.001
Total cholesterol (mmol/L)	4.501 \pm 1.161	4.181 \pm 0.998	0.044
Triglycerides (mmol/L)	1.697 \pm 1.962	1.278 \pm 0.738	0.084
HDL-C (mmol/L)	1.001 \pm 0.262	1.005 \pm 0.226	0.904
LDL-C (mmol/L)	2.60 (2.05–3.14)	2.85 (2.24–3.16)	0.497
CKMB (ng/mL)	5.94 (2.44–31.03)	12.86 (5.41–55.68)	<0.001
Creatinine (μ mol/L)	62 (55–74)	55.50 (46.00–71.00)	0.006
Fibrinogen (g/L)	2.902 \pm 1.06	3.135 \pm 1.161	0.107
LVEF (%)	51 (50–55)	51 (49.75–56.25)	0.363
Daily smokers, n (%)	82 (31.2)	56 (60.9)	<0.001
Use of therapeutic agents			
Aspirin, n (%)	263 (100)	92 (100)	1
Clopidogrel/Ticagrelor, n (%)	263 (100)	92 (100)	1
Beta-blocker, n (%)	198 (75.3)	76 (80.9)	0.894
ACEI/ARB, n (%)	156 (59.3)	52 (56.5)	0.196

(Continued)

Table 1 (Continued).

Variable	Low SII/Low NT-proBNP (Group Q1 n=263)	High SII+ High NT-proBNP (Group Q2 n=92)	P
CCB, n (%)	58 (22.1)	16 (17.4)	0.202
Diuretics, n (%)	78 (29.7)	59 (64.1)	<0.001
Statins, n (%)	231 (87.8)	89 (96.7)	0.508
Nitrates, n (%)	175 (66.5)	68 (73.9)	0.728
Hypertension, n (%)	82 (31.2)	40 (43.5)	0.098
Diabetes mellitus, n (%)	66 (25.1)	18 (19.6)	0.152
Heart rate (bpm)	83.08 ± 15.513	87.13 ± 13.380	0.074
Systolic pressure (mmHg, $\bar{x} \pm s$)	125.59 ± 20.531	123.73 ± 22.362	0.470
Diastolic pressure (mmHg, $\bar{x} \pm s$)	78.92 ± 13.202	76.24 ± 15.517	0.116
SII	798.07 (458.41–1134.34)	2091.68 (1775.56–2642.77)	<0.001
NT-proBNP (pg/mL)	935.581 ± 2432.152	2657.958 ± 4665.864	<0.001

Abbreviations: WBC, white blood cell; Neut, neutrophils; Lymph, lymphocyte; RBC, red blood cell; HB, hemoglobin; PLT, platelet; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor inhibitor; CCB, calcium channel blockers; SII, systemic immune-inflammation index.

Clinical Characteristics of Patients with and without MACEs

In this research, as a composite endpoint, 100 patients (28.2%) developed MACEs, with 39 cases (11.0%) of all-cause mortality and 22 cases (6.2%) of re-hospitalization for angina. 14 cases (3.9%) of nonfatal myocardial infarction; 7 cases (2.0%) of target vessel reconstruction; of which 16 cases (4.5%) of acute heart failure; and 2 cases (0.6%) of non-fatal stroke. MACE patients were noted to be older and diabetes was more prevalent. Furthermore, the neutrophil count, hemoglobin, CK-MB, creatinine, fibrinogen, SII, and NT-proBNP levels in the MACE group were greater than in the non-MACE group. Additionally, the lymphocyte count and the proportion of male and the history of diuretics and nitrates in MACE-free group was higher (Table 2).

Table 2 Comparison of Clinical and Laboratory Characteristics of Enrolled Patients According to the Occurrence of MACEs

Variable	MACE Group (n=100)	MACE-Free Group (n=255)	P
Age (years, $\bar{x} \pm s$)	65.80 ± 11.92	62.46 ± 12.92	0.026
Gender			
Male, n (%)	69 (69.0)	193 (75.7)	<0.001
Laboratory parameters			
WBC, ($10^9/L$)	10.618 ± 3.066	9.998 ± 3.045	0.086
Neut, ($10^9/L$)	8.53 (6.85–10.81)	7.47 (5.51–9.60)	0.002
Lymph, ($10^9/L$)	1.15 (0.80–1.50)	1.40 (1.00–2.20)	0.004

(Continued)

Table 2 (Continued).

Variable	MACE Group (n=100)	MACE-Free Group (n=255)	P
RBC, (10 ⁹ /L)	4.574 ± 0.513	4.731 ± 0.528	0.019
HB, (g/L)	138.61 ± 17.284	144.11 ± 16.264	0.005
PLT, (10 ⁹ /L)	216.50 (180.50–259.75)	207.00 (173.00–245.00)	0.035
Total cholesterol (mmol/L)	4.379 ± 1.156	4.424 ± 1.115	0.775
Triglycerides (mmol/L)	1.445 ± 0.910	1.634 ± 1.957	0.43
HDL-C (mmol/L)	1.006 ± 0.227	1.001 ± 0.262	0.86
LDL-C (mmol/L)	2.58 (2.20–3.06)	2.62 (2.09–3.14)	0.801
CKMB (ng/mL)	22.27 (6.00–82.99)	8.09 (2.65–38.15)	0.001
Creatinine (μmol/L)	64.50 (52.00–76.75)	61.00 (52.00–72.00)	0.17
Fibrinogen (g/L)	3.318 ± 1.233	2.825 ± 0.999	<0.001
LVEF (%)	50 (47–53)	51 (50.00–56.00)	0.105
Daily smokers, n (%)	40 (40.0)	122 (47.8)	0.182
Use of therapeutic agents			
Aspirin, n (%)	100 (100)	255 (100)	1
Clopidogrel/Ticagrelor, n (%)	100 (100)	255 (100)	1
Beta-blocker, n (%)	84 (84.0)	190 (74.5)	0.055
ACEI/ARB, n (%)	67 (67.0)	141 (55.3)	0.044
CCB, n (%)	21 (21.0)	53 (20.8)	0.964
Diuretics, n (%)	52 (52.0)	85 (33.3)	0.001
Statins, n (%)	74 (74.0)	246 (96.5)	<0.001
Nitrates, n (%)	72 (72.0)	171 (67.1)	0.368
Hypertension, n (%)	42 (42.0)	80 (31.4)	0.058
Diabetes mellitus, n (%)	38 (38.0)	46 (18.0)	<0.001
Heart rate (bpm)	86.05 ± 14.507	83.49 ± 15.224	0.264
Systolic pressure (mmHg, $\bar{x} \pm s$)	123.19 ± 19.729	125.83 ± 21.520	0.301
Diastolic pressure (mmHg, $\bar{x} \pm s$)	77.21 ± 14.771	78.57 ± 13.558	0.422
SII	1649.08 (912.04–2447.79)	1122.94 (562.24–1830.82)	<0.001
NT-proBNP (pg/mL)	3544.303 ± 5420.347	566.283 ± 1029.638	<0.001

Abbreviations: WBC, white blood cell; Neut, neutrophils; Lymph, lymphocyte; RBC, red blood cell; HB, hemoglobin; PLT, platelet; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; LVEF, left ventricular ejection fractions; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor inhibitor; CCB, calcium channel blockers; SII, systemic immune-inflammation index.

Risk Factor Analysis for MACEs Development

Based on MACE occurrence as an endpoint indicator, under univariate Cox survival analysis, lymphocyte count, hemoglobin, red blood Cell distribution width (RDW), hypersensitive C-reactive protein (hs-CRP), fibrinogen (FIB), The multivariate Cox survival analysis included Ln(SII) and Ln(NT-proBNP) (P <0.01) and showed that Ln(SII) (HR =

Table 3 Univariate and Multivariate Cox Regression Analysis Results for MACEs

Variables	HR	Univariate 95% CI	P value	HR	Multivariate 95% CI	P value
Lymph	0.604	0.426–0.857	0.005	1.149	0.698–1.891	0.586
Hemoglobin	0.982	0.970–0.994	0.004	0.973	0.903–1.048	0.472
HCT	0.943	0.903–0.985	0.009	1.114	0.866–1.433	0.400
Fibrinogen	1.276	1.067–1.525	0.007	1.208	0.924–1.578	0.166
RDW	2.21	1.569–3.114	<0.001	0.975	0.869–1.095	0.673
hs-CRP	1.018	1.004–1.012	<0.001	0.994	0.984–1.005	0.285
Ln(SII)	2.262	1.591–3.217	<0.001	2.952	1.565–5.566	<0.001
Ln(NT-proBNP)	2.277	1.828–2.836	<0.001	2.112	1.662–2.683	<0.001

Abbreviations: Ln(SII), natural -logarithm of systemic immune-inflammation index; Ln(NT-proBNP); natural-logarithm of NT-proBNP.

2.952 $P < 0.001$, 95% CI 1.565–5.566) and Ln(NT-proBNP) (HR = 2.112 $P < 0.001$, 95% CI 1.662–2.683) independently predicted the development of MACE in STEMI patients (Table 3).

Long-term patient survival rates were assessed using the Kaplan-Meier survival analysis. As displayed in Figure 2, the MACEs at 1 year were significantly different between the two groups. In the group with high SII and high NT-proBNP, MACE incidence was greater (log rank: 29.67, $P < 0.001$).

Correlation Between the SII and NT-proBNP

The Pearson correlation analysis was utilized to measure the correlation counts between SII and NT-proBNP. The findings revealed a positive correlation between SII and NT-proBNP ($R = 0.187$, $P < 0.001$).

Effect of Combined SII and NT-proBNP on Development of MACE

ROC analysis was done to determine if the SII and NT-proBNP could enhance the prediction of long-term MACEs. The ROC curve analysis indicated that for the SII, the AUC was 0.649 (95% CI: 0.587–0.712, $P < 0.001$), for the NT-proBNP, the AUC was 0.774 (95% CI: 0.710–0.835, $P < 0.001$), and for the SII and NT-proBNP combined, the AUC was 0.840 (95% CI: 0.791–0.887, $P < 0.001$) (Figure 3). Compared with the SII and NT-proBNP, and the SII combined with NT-proBNP, the

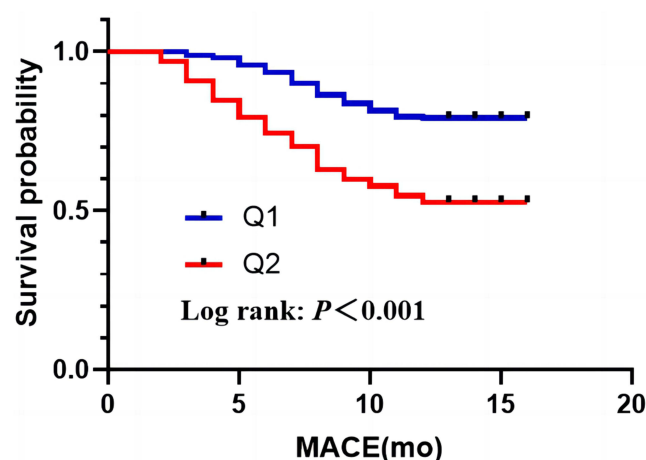


Figure 2 Kaplan-Meier survival curves of MACEs in STEMI patients according to the level of SII and NT-proBNP. Based on the median SII and NT-proBNP (Group Q1, SII ≤ 1240.61 or NT-proBNP ≤ 280.53 ; Group Q2, SII > 1240.61 and NT-proBNP > 280.53).

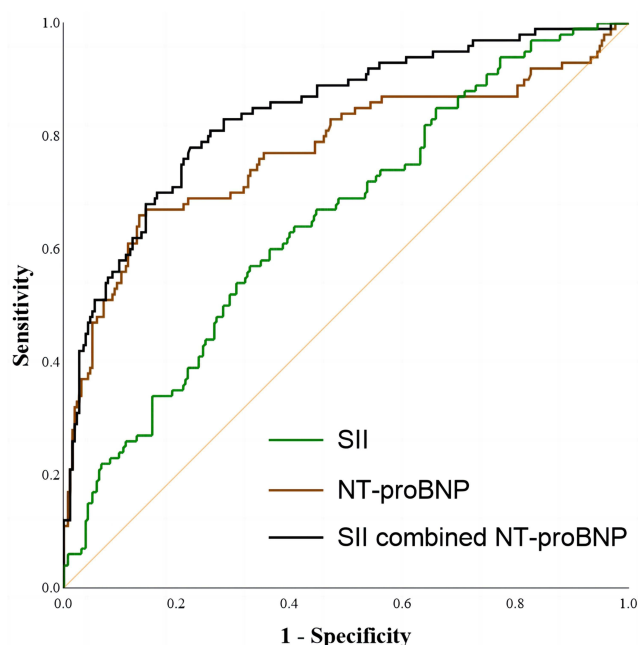


Figure 3 Receiver operating characteristic curves of SII, NT-proBNP, SII combined with NT-proBNP for 1-year MACEs prediction.

combination predicted MACEs better than SII or NT-proBNP alone, and the variance between them was statistically significant. ($Z = 2.622$, $P = 0.009$; $Z = 3.173$, $P < 0.001$).

Discussion

STEMI is a type of coronary heart disease that is seriously life-threatening. It refers to the plaque rupture in these coronary arteries that contain unstable atherosclerotic plaques due to lack of blood supply and oxygen, thereby leading to coronary micro thrombosis, making myocardial tissue cell swelling, degeneration and necrosis. Clinical manifestations consist of persistent chest tightness and chest pain, gastrointestinal symptoms, palpitations caused by arrhythmia.¹⁵ Once myocardial infarction occurs, cardiomyocytes cannot regenerate at the infarct site and the necrotic myocardial tissue becomes thinning. It may cause heart rupture, papillary muscle rupture, abnormal heart conduction pathway, heart failure and other complications. These complications seriously affect the prognosis of patients.¹⁶ STEMI is one of the most prevalent types of myocardial infarction. Moreover, it is common among the middle-aged and elderly individuals.¹⁷ In recent years, STEMI incidence and mortality have been increasing year by year. Recently, it is not unusual for many young patients to have an acute coronary syndrome.¹⁸ Therefore, it is extremely important to pay clinical attention to the follow-ups on the treatment of patients with STEMI whilst also effectively assessing poor outcomes risk of in STEMI patients, as well as timely formulating the corresponding individualized intervention measures, so as to reduce the mortality of STEMI patients and provide better patients prognostic quality of life.

SII is a systemic immune inflammation index, which integrates more inflammation and immune indicators that reflect the body state. It can better reflect the inflammation and immune system status of patients.¹⁹ After the inflammation occurs in the human body, it may promote the abnormal rise of platelets. Abnormally aggregated platelets also adhere to the surface of the endothelial cells of the body, thus inducing local ischemia, hypoxia, micro thrombosis and then blocking blood vessels, causing ischemic MI, stroke, peripheral vascular diseases and other malignant clinical events.²⁰ Serum levels of SII are linked to a greater chance of developing ischemic heart disease, ACS, cardiovascular events, and stroke.^{21,22} An abnormal decrease in lymphocyte count indicates excessive lymphocyte apoptosis, which reduces the immune system capacity and immune dysfunction. It is then easy to promote the endothelial dysfunction, resulting in abnormal aggregation and thrombosis after platelet activation.²³ While neutrophils can also activate and induce inflammatory reactions in the body, promote the abnormal coronary plaques, induce the atherosclerotic plaque rupture

and thrombosis,^{24,25} and then increase cardiovascular complications risk. Dziedzic et al have reported²⁶ that after SII is applied to patients with STEMI. In the short-term prognosis, SII levels are significantly higher in the poor prognosis group than in the good prognosis group.

NT-proBNP is the remaining other half of the product when the BNP precursor (pro BNP) cleaves into BNP. It is mainly a hormone secreted by ventricular cells that has many biological effects, such as vasodilation, balancing water and sodium metabolism, inhibiting renin-angiotensin and sympathetic nervous system, etc. When the ventricular volume and pressure load increase, or the left heart function is impaired, it can be greatly expressed in a short time and reflect the cardiac function.^{27,28} Some studies report that NT-proBNP can be used as an early warning and diagnostic indicator for STEMI.²⁹

The current study assessed the ability of SII and NT-proBNP alone and jointly to predict MACE in a cohort of patients with STEMI. Compared to other biomarkers, SII and NT-proBNP can better respond to inflammatory conditions and ventricular load and wall pressure in STEMI patients.³⁰ The outcomes of univariate and multivariate Cox survival analysis show that SII and NT-proBNP are both independent predictors of MACE in STEMI patients. MACE incidence is significantly higher in the patients with higher values of SII and NT-proBNP. Moreover, SII is also correlated with NT-proBNP to some extent. SII combined with NT-proBNP may improve the predictive power of cardiovascular complications in STEMI patients.

Limitations

This research has some limitations. Firstly, this is a single-center retrospective research, and the inseparable limitations of these investigations will invariably result in selection bias. Secondly, the follow-up period is short and the sample size is small; therefore, multicenter and larger sample sizes are required to establish clinical relevance.

Conclusion

This study demonstrated that SII and NT-proBNP in STEMI patients may be used for prediction of MACEs development, and SII had a positive correlation with NT-proBNP. A combination of the SII and NT-proBNP might improve clinical outcome predictions in these patients.

Data Sharing Statement

The corresponding author can provide the Data upon appropriate request.

Ethics Approval and Consent Participate

The study was approved by the Medical Ethics Committee of Xuzhou Medical University's Affiliated Hospital in compliance with the Helsinki Declaration. Due to the retroactive nature of the investigation, the review committee relaxed the necessity for written informed consent. We eliminated all private patient data before examining the data.

Consent for Publication

All participants and/or their legal guardians given permission for the publishing of their information.

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Disclosure

The authors declare no conflicts of interest in this work.

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