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Original Article

Complete revascularization improves blood pressure control and clinical prognosis in patients with acute myocardial infarction by reducing oxidative stress

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Abstract: Objective: To investigate the effects of complete revascularization on oxidative stress, blood pressure control, and prognosis in patients with acute myocardial infarction (AMI). Methods: Clinical data from 80 AMI patients were retrospectively analyzed. Based on the SYNTAX Revascularization Index (SRI), patients were classified into the complete revascularization group (SRI=100%, n=31), partial revascularization group (SRI 50-99%, n=27), and low revascularization group (SRI<50%, n=22). Postoperative oxidative stress markers (malondialdehyde, MDA; angiotensin converting enzyme, ACE; and superoxide dismutase, SOD), myocardial injury marker (cardiac Troponin I, cTnI), and ambulatory blood pressure parameters were compared among groups. Their correlations were analyzed, and the predictive value of SRI for major adverse cardiovascular events (MACE) was evaluated. Results: One month after surgery, the complete revascularization group exhibited significantly lower MDA than the partial and low revascularization groups, higher SOD, and lower ACE and cTnI levels (all P<0.001). Six months postoperatively, the complete revascularization group showed improvements in 24 h SBP, 24 h SBP-SD and nocturnal blood pressure decline rate (all P<0.01). The incidence of MACE was significantly lower in the complete revascularization group (6.45% vs. 36.73%, P=0.002). Logistic regression showed that SRI was an independent protective factor for MACE (OR=0.119, 95% CI: 0.025-0.557, P=0.007). ROC curve analysis indicated its predictive value for MACE with an AUC of 0.835 and an optimal cut-off value of 69.360%. Conclusion: Complete revascularization improves blood pressure stability and reduces MACE risk in AMI patients by alleviating oxidative stress. Achieving an SRI above 69.360% can be considered a clinical target.

Keywords: Revascularization, acute myocardial infarction, oxidative stress, blood pressure, prognosis

Introduction

Acute myocardial infarction (AMI) is a major cardiovascular emergency associated with high morbidity and mortality. Rapid restoration of blood flow in the infarct-related artery remains the cornerstone of AMI management [1]. Current clinical guidelines emphasize that, once coronary anatomy is delineated, early revascularization should be performed to improve outcomes in AMI patients [2]. However, myocardial reperfusion after AMI can induce oxidative stress, leading to excessive production of reactive oxygen species (ROS). These ROS can damage cellular membranes, trigger cardiomyocyte necrosis and apoptosis, and ultimately aggravate myocardial injury. Thus, oxidative stress is recognized as a key pathophysiological mechanism in AMI progression [3]. Moreover, oxidative stress plays a critical role in blood pressure regulation. ROS can lead to abnormal blood pressure fluctuations after AMI through multiple pathways, such as activating the renin-angiotensin-aldosterone system, damaging vascular endothelial function, and interfering with central nervous system regulation; blood pressure variability itself has been confirmed as an independent risk factor for cardiovascular events [4].

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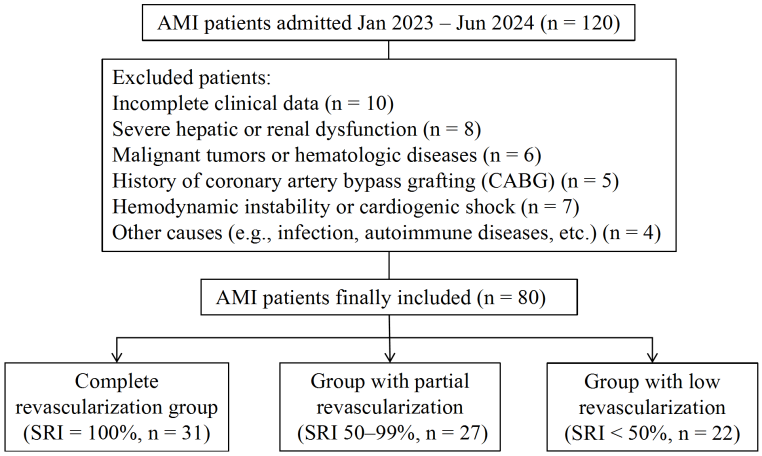

Figure 1. Flowchart of patient screening and inclusion. AMI, acute myocardial infarction.

Theoretically, more complete revascularization may reduce oxidative stress load from the source by improving myocardial oxygen supply, alleviating myocardial ischemia, protecting myocardial cell function, and promoting repair. However, the dose-effect relationship and its specific impact on subsequent blood pressure stability remain underexplored. Although previous studies have confirmed the overall benefits of revascularization for AMI patients, the intrinsic protective mechanism, particularly how different extents of revascularization affect the oxidative stress state and consequently influence long-term blood pressure stability and myocardial repair, lacks in-depth investigation and multi-indicator combined analysis [5-7]. In view of this, this study innovatively integrates oxidative stress biomarkers, myocardial injury markers, and 24-hour ambulatory blood pressure parameters to systematically evaluate the differential effects of varying SYNTAX Revascularization Index (SRI) levels on oxidative stress status, blood pressure regulation stability, and myocardial protection in AMI patients, and further assess the predictive value of SRI for long-term major adverse cardiovascular events (MACE), aiming to provide a reference for optimizing treatment strategies in AMI patients.

Materials and methods

Patient selection

This was a single-center retrospective observational study. The study protocol was submitted to the institutional ethics committee of the First Hospital of ShanXi Medical University for review and was approved, with a waiver of patient informed consent. Through querying the hospital electronic medical record system, 80 AMI patients admitted to our hospital between January 2023 and June 2024 were screened. According to the SRI, patients were divided into the complete revascularization group (SRI=100%, n=31), a partial revascularization group (SRI 50-99%, n=27), and a low revascularization group (SRI<50%, n=22). A retrospective analysis of data from these 80 AMI patients was conducted to externally validate the SRI-predicted MACE model. The patient screening process is shown in **Figure 1**. In this retrospective study, a prospective sample size estimation was not conducted prior to the study. Cases were continuously screened and included through the hospital electronic medical record system. The study included a total of 80 AMI patients, of whom 20 experienced MACE events, meeting the empirical requirement of at least 10 events per predictor variable (EPV ≥10) for multivariate logistic regression analysis. The sample size was sufficient to meet the basic stability requirements of the statistical analysis.

Inclusion and exclusion criteria

Inclusion criteria: Age ≥18 years; first diagnosis of acute ST-segment elevation myocardial infarction (STEMI) or high-risk non-ST-segment elevation myocardial infarction (NSTEMI); undergoing emergency percutaneous coronary intervention (PCI); coronary angiography confirming multivessel disease (MVD); complete clinical data, including baseline characteristics, surgical records, laboratory tests, and follow-up data.

Exclusion criteria: presence of cardiogenic shock or hemodynamic instability upon admission; severe hepatic or renal dysfunction; active infection, autoimmune disease, malignancy, or hematologic disorders; history of coronary

artery bypass grafting (CABG); severe valvular heart disease, cardiomyopathy, etc.

The establishment of the above inclusion and exclusion criteria aims to ensure the completeness and comparability of study subject data and to reduce the interference of potential confounding factors. Only AMI patients confirmed by coronary angiography as having multivessel disease and undergoing emergency PCI were enrolled to ensure the study population had a consistent pathological basis and treatment method. Exclusion of cardiogenic shock, severe hepatic or renal dysfunction, malignancy, infection, and other systemic diseases can prevent these factors from affecting oxidative stress levels and blood pressure variability, thereby improving the scientific validity and reliability of the study results.

Data extraction

Baseline data collection: (1) Demographic and clinical characteristics: Patient demographics, including age, sex, and body mass index (BMI), were obtained from the hospital's electronic medical record system. The type of AMI (STEMI or NSTEMI) and cardiovascular risk factors-including hypertension, diabetes, smoking history, and dyslipidemia-were determined based on medical history, electrocardiographic features, serum biomarkers, and coronary angiography findings. (2) Coronary lesion characteristics and interventional procedure data: All coronary angiograms were independently evaluated by two cardiologists in a blinded manner, with no access to group allocation or clinical outcomes. The SRI was calculated using the formula: SRI = [(baseline SYNTAX score - residual SYNTAX score)/baseline SYNTAX score] × 100%. The extent of coronary artery disease was recorded as either two-vessel or three-vessel involvement.

Outcome indicators: (1) Primary outcome indicators: a. MACE after myocardial revascularization was assessed based on the "2018 ESC/EACTS Guidelines on Myocardial Revascularization" [8]. The composite endpoint events occurring within 1 year after discharge were recorded via a combination of electronic medical record review and telephone follow-up, including cardiac death, recurrent myocardial infarction, readmission due to heart failure, and unplanned target vessel revascularization. b. Levels of malondialdehyde (MDA), superoxide dismutase (SOD), angiotensin-converting enzyme (ACE), and myeloperoxidase (MPO) were measured using an automated biochemical analyzer. (2) Secondary outcome indicators: a. Cardiac structure and function: Echocardiographic examinations were performed within 24 hours of admission and at 1 month postoperatively. All measurements were obtained and analyzed by two experienced sonographers using a standard echocardiography system. b. Myocardial injury indicators: Venous blood samples were obtained at admission, immediately before emergency PCI, and 1 month postoperatively. Cardiac troponin I (cTnI) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels were measured using the Abbott ARCHITECT i2000SR chemiluminescent immunoassay system. c. 24-hour ambulatory blood pressure monitoring (ABPM): At 6 months postoperatively, 24-hour ABPM was conducted using a noninvasive portable ABPM device (Spacelabs 90217). Blood pressure was automatically recorded every 30 minutes during the daytime (6:00-22:00) and every 60 minutes at night (22:00-6:00). Data were exported using specialized software by trained personnel, with valid readings exceeding 90%. Parameters included 24-hour mean systolic blood pressure (24 h SBP) and 24-hour systolic blood pressure standard deviation (24 h SBP-SD).

Statistical analysis

SPSS 27.0 was used for statistical analysis in this study. After normality testing, normally distributed data were expressed as mean ± standard deviation, and comparisons between groups were performed using t-tests or one-way ANOVA. Non-normally distributed data were analyzed using non-parametric tests. Categorical data were analyzed using χ^2 tests.

With MACE (0 = no event, 1 = event) as the dependent variable, univariate analysis (including parametric and non-parametric tests) was first performed on clinically significant candidate variables to preliminarily screen variables associated with MACE. Subsequently, their independent association with MACE was jointly assessed in a multivariate logistic regression model.

The multivariate model used a stepwise backward regression approach, with inclusion and exclusion criteria of $P<0.05$ and $P>0.10$,

Table 1. Comparison of baseline data and clinical indicators among the three groups

Index	Complete revascularization group (n=31)	Partial revascularization group (n=27)	Low revascularization group (n=22)	χ^2/F	*P*
Age ($\bar{x}\pm s$)	61.23 ± 8.73	63.70 ± 9.26	65.86 ± 10.13	*F*=1.628	0.203
Gender				χ^2=0.054	0.973
Male [n, (%)]	20 (64.52%)	18 (66.67%)	14 (63.64%)		
Female [n, (%)]	11 (35.48%)	9 (33.33%)	8 (36.36%)		
Body mass index (kg/m^2, $\bar{x}\pm s$)	24.58 ± 3.12	25.63 ± 3.45	26.01 ± 3.78	*F*=1.277	0.285
Smoking history [n, (%)]	15 (48.39%)	14 (51.85%)	10 (45.45%)	χ^2=0.201	0.904
Hypertension [n, (%)]	19 (61.29%)	16 (59.26%)	15 (68.18%)	χ^2=0.443	0.801
Diabetes [n, (%)]	10 (32.26%)	11 (40.74%)	12 (54.55%)	χ^2=2.642	0.267
Dyslipidemia [n, (%)	22 (70.97%)	18 (66.67%)	17 (77.27%)	χ^2=1.668	0.716
Admission systolic blood pressure (mmHg, $\bar{x}\pm s$)	138.52 ± 18.64	142.33 ± 20.15	145.91 ± 22.47	*F*=0.869	0.423
Admission diastolic blood pressure (mmHg, $\bar{x}\pm s$)	82.45 ± 11.23	85.17 ± 12.58	86.72 ± 13.41	*F*=0.825	0.442
Heart rate (beats/min, $\bar{x}\pm s$)	76.58 ± 13.42	78.89 ± 14.10	81.36 ± 15.80	*F*=0.721	0.489
Infarction type [n, (%)]				χ^2=0.054	0.973
STEMI	20 (64.52%)	18 (66.67%)	14 (63.64%)		
NSTEMI	11 (35.48%)	9 (33.33%)	8 (36.36%)		
Number of diseased blood vessels [n, (%)]				χ^2=2.467	0.291
Two-vessel disease	15 (48.39%)	10 (37.04%)	6 (27.27%)		
Three-vessel disease	16 (51.61%)	17 (62.96%)	16 (72.73%)		

Note: STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction.

respectively. Given that SRI is the core exposure variable in this study, used to quantify the degree of revascularization completion, it was mandatory to include it in the multivariate model as a continuous variable, unaffected by the stepwise selection process.

SRI was included in the logistic regression model as a proportional variable (SRI/100), and its regression coefficient (β) represents the impact of the degree of revascularization completion on the risk of MACE across the entire range (i.e., from 0% to 100%). Because SRI is the core exposure variable in this study, used to quantify the degree of revascularization completion, it was included in the multivariate logistic regression model, unaffected by the stepwise selection process, to avoid model bias caused by the removal of key exposure variables. Model performance was evaluated using ROC curves, calibration curves, and decision curves (DCA).

Results

Comparison of baseline characteristics and clinical indicators among groups

There were no significant differences among the three groups in terms of age, sex, distribution of cardiovascular risk factors, or baseline cardiac function (left ventricular ejection fraction, LVEF) (P>0.05) (**Table 1**).

Comparison of oxidative stress, myocardial injury markers, and ambulatory blood pressure parameters among groups

At 1 month postoperatively, the complete revascularization group exhibited significantly lower levels of oxidative stress markers, including MDA and ACE, higher levels of the antioxidant enzyme SOD, and lower cardiac injury marker cTnI compared with the partial and low revascularization groups (all P<0.05). At 6 months postoperatively, the complete revascularization group demonstrated significantly reduced 24-hour mean systolic blood pressure (24 h SBP), 24 h SBP-SD, and nocturnal blood pressure decline relative to the other groups, with statistically significant differences (P<0.05) (**Tables 2** and **3**).

Relationship between oxidative stress, myocardial injury, and blood pressure variability

Pearson correlation analysis demonstrated that oxidative stress markers MDA and ACE were positively correlated with both the cardiac

Table 2. Comparison of oxidative stress and myocardial injury marker levels one month after surgery ($\bar{x}\pm s$)

Index	Complete revascularization group (n=31)	Partial revascularization group (n=27)	Low revascularization group (n=22)	*F*	*P*
MDA (nmol/mL)	3.25 ± 0.78	4.10 ± 0.85	5.32 ± 1.15	32.441	<0.001
SOD (U/mL)	125.36 ± 15.42	108.75 ± 14.68	95.83 ± 13.27	27.078	<0.001
ACE (ng/mL)	35.21 ± 6.54	41.89 ± 7.80	48.76 ± 8.95	20.213	<0.001
cTnI (ng/mL)	0.15 ± 0.08	0.28 ± 0.08	0.45 ± 0.18	70.619	<0.001

Note: MDA, malondialdehyde; SOD, Superoxide dismutase; ACE, angiotensin-converting enzyme; cTnI, cardiac Troponin I.

Table 3. Comparison of ambulatory blood pressure parameters at 6 months after surgery ($\bar{x}\pm s$)

Index	Complete revascularization group (n=31)	Partial revascularization group (n=27)	Low revascularization group (n=22)	*F*	*P*
24 h mean SBP (mmHg)	122.35 ± 7.82	126.94 ± 8.71	133.68 ± 10.25	10.599	<0.001
24 h SBP-SD (mmHg)	9.87 ± 1.65	11.52 ± 2.03	13.86 ± 2.44	25.113	<0.001
Nocturnal blood pressure decline (%)	11.25 ± 3.21	9.68 ± 2.45	7.12 ± 1.88	15.617	0.002

Note: 24 h mean SBP, 24-hour mean systolic blood pressure; 24 h SBP-SD, 24-hour systolic blood pressure standard deviation.

Table 4. Correlation analysis of oxidative stress markers with myocardial injury and blood pressure variability

Index	Statistical value	MDA	SOD	ACE
cTnI	*r*	0.510	-0.565	0.379
	P	<0.001	<0.001	<0.001
24 h SBP-SD (mmHg)	*r*	0.445	-0.397	0.407
	P	<0.001	<0.001	<0.001

Note: cTnI, cardiac Troponin I; 24 h SBP-SD, 24-hour systolic blood pressure standard deviation; MDA, malondialdehyde; SOD, superoxide dismutase; ACE, angiotensin-converting enzyme.

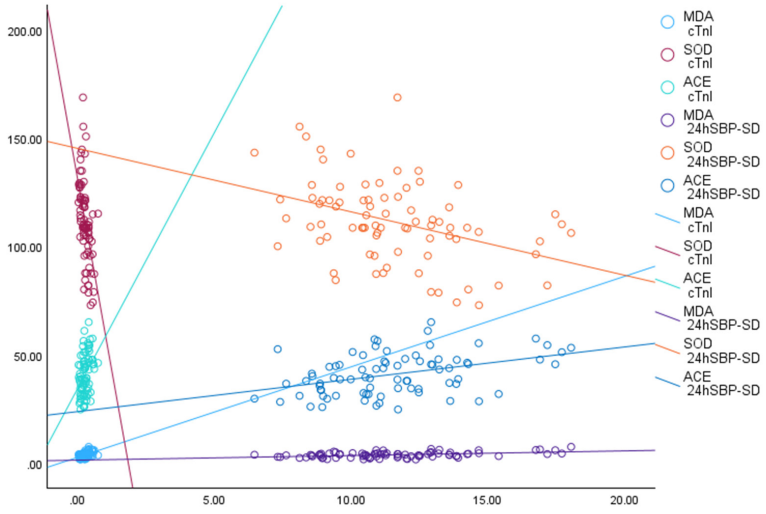

Figure 2. Scatter plots showing the correlations between oxidative stress markers and myocardial injury markers, as well as blood pressure variability indices.

injury marker cTnI and 24 h SBP-SD (r>0, P<0.05). In contrast, the antioxidant marker SOD was negatively correlated with cTnI and 24 h SBP-SD (r<0, P<0.05) (**Table 4**; **Figure 2**).

Effect of revascularization on cTnI and 24 h SBP-SD

Linear regression analysis indicated that the SRI was a negative predictor of both cTnI levels and 24 h SBP-SD (P<0.05) (**Table 5**; **Figure 3**).

Effect of revascularization on MACE

Within one-year post-discharge, the incidence of MACE was significantly lower in the complete revascularization group compared with the non-complete revascularization group (χ^2=9.287, P=0.002) (**Table 6**). Logistic regression analysis identified the SRI as an independent protective factor for MACE (P<0.05) (**Table 7**).

Table 5. The effect of revascularization on cTnI elevation and 24 h SBP-SD variability

Factor		*B*	*SE*	*β*	*t*	*P*	95% CI
cTnI elevation	Constant	0.580	0.028		20.660	<0.001	0.524~0.636
	SRI	-0.004	0.000	-0.800	-11.785	<0.001	-0.005~-0.003
24 h SBP-SD variability	Constant	15.632	0.619		25.247	<0.001	14.400~16.865
	SRI	-0.056	0.008	-0.628	-7.119	<0.001	-0.072~-0.040

Note: cTnI, cardiac Troponin I; 24 h SBP-SD, 24-hour systolic blood pressure standard deviation.

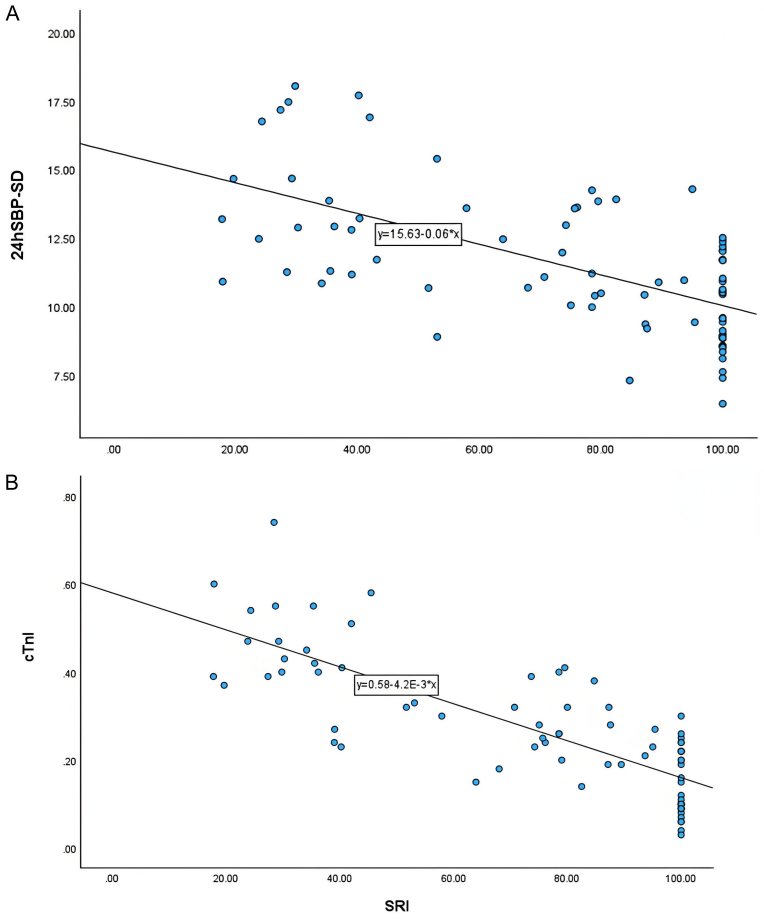

Figure 3. Effect of revascularization on 24-hour SBP-SD and cTnI elevation. Note: (A) Effect of revascularization on 24-hour SBP-SD; (B) Effect of revascularization on cTnI elevation. 24-hour SBP-SD, 24-hour systolic blood pressure standard deviation; cTnI, cardiac Troponin I.

Univariate analysis and logistic regression results between non-MACE and MACE groups

During the 1-year follow-up period, some patients experienced MACE. Patients were divided into the MACE group and the non-MACE group based on whether MACE occurred. The results of the univariate analysis showed significant differences between the two groups in oxidative stress-related indicators, myocardial injury indicators, and blood pressure variability parameters (all P<0.05). While age, body mass index, and some basic clinical indicators showed no statistically significant differences (all P>0.05) (**Table 8**).

Furthermore, the degree of revascularization was included as an independent variable in the univariate Logistic regression analysis. The results showed that a lower degree of revascularization was significantly associated with an increased risk of MACE, as shown in **Table 9**.

On this basis, combining the univariate analysis results with clinical significance, the degree of revascularization and relevant clinical indicators were included in the multivariate Logistic regression model. The multivariate analysis results revealed that after adjusting for potential confounding factors, the degree of revascularization remained independently associated with the risk of MACE, as shown in **Table 10**.

ROC curve analysis and validation of SRI for predicting MACE

ROC curve analysis demonstrated that the SRI had a high predictive value for MACE, with an AUC>0.8 and an optimal cutoff of 69.36% (**Figure 4** and **Table 11**). Calibration curves (**Figure 5**) indicated that the observed incidence of MACE was generally consistent with the predicted probabilities, although some

Table 6. Incidence of major adverse cardiovascular events (MACE) during 1-year follow-up [n, (%)]

Groups	n	Total MACE	Cardiogenic death	Reinfarction	Readmission due to heart failure	Unplanned revascularization
Complete revascularization group	31	2 (6.45%)	0 (0.00%)	1 (3.23%)	1 (3.23%)	0 (0.00%)
Non-complete revascularization group	49	18 (36.73%)	1(2.04%)	6 (12.24%)	6 (12.24%)	5 (10.20%)
Partial revascularization group	27	6 (22.22%)	0 (0.00%)	2 (7.41%)	2 (7.41%)	2 (7.41%)
Low revascularization group	22	12 (54.55%)	1 (3.70%)	4 (18.18%)	4 (18.18%)	3 (13.64%)

Table 7. Logistic Regression analysis of the effect of SRI on MACE

Factor	β	Standard error	Wald	*P*	*OR*	95% CI
Constant	-0.544	0.296	3.365	0.067	0.581	-
SRI	-2.131	0.789	7.294	0.007	0.119	0.025-0.557

Note: SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events; OR, odds ratio; CI, confidence interval.

Table 8. Comparison of clinical indicators between the MACE and the non-MACE groups

Variables	Non-MACE (n=59)	MACE (n=21)		P
MDA (nmol/mL)	3.96 ± 1.12	4.51 ± 1.45	t=-1.764	0.082
SOD (U/mL)	114.26 ± 18.98	104.24 ± 16.66	t=2.140	0.035
ACE (U/L)	40.50 ± 9.60	43.13 ± 8.61	t=-1.107	0.272
CTnI (ng/mL)	0.22 (0.13-0.31)	0.38 (0.26-0.47)	U=315.500	<0.001
24 h mean SBP (mmHg)	124.54 (118.54-133.68)	131.54 (121.52-133.68)	U=495.000	0.175
24 h SBP-SD (mmHg)	11.05 ± 2.38	12.86 ± 2.62	t=-2.924	0.005
Nocturnal blood pressure decline (%)	10.15 ± 3.16	8.00 ± 2.33	t=2.861	0.005
Age (year)	62.95 ± 8.91	64.43 ± 10.76	t=-0.618	0.538
BMI (kg/m^2)	25.48 ± 3.35	24.91 ± 3.72	t=0.642	0.523
DBP (mmHg)	139.72 ± 19.81	147.80 ± 20.65	t=-1.588	0.116
SBP (mmHg)	83.01 ± 11.44	88.86 ± 13.79	t=-1.907	0.060
HR (time/min)	77.27 ± 14.27	82.62 ± 13.89	t=-1.485	0.142

Note: Normally distributed variables are expressed as mean ± standard deviation, and non-normally distributed variables are expressed as median (interquartile range). MACE, major adverse cardiovascular events; MDA, malondialdehyde; SOD, Superoxide dismutase; ACE, angiotensin-converting enzyme; cTnI, cardiac Troponin I; 24 h SBP-SD, 24-hour systolic blood pressure standard deviation; BMI, body mass index; DBP, diastolic blood pressure (mmHg); SBP, systolic blood pressure (mmHg); HR, heart rate.

Table 9. Univariate logistic regression analysis of SRI sorting variables on MACE

Variables	OR	95% CI	P
SRI=100%	1.000	-	-
SRI<50%	9.333	(2.179-39.976)	0.0026
50%≤SRI<100%	3.267	(0.752-14.196)	0.1143

Note: SRI=100% was defined as the reference. SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

deviations suggested that calibration requires further evaluation. External validation using a separate cohort of 80 contemporaneous AMI patients yielded an SRI AUC of 0.912 (**Figure 6**), indicating strong predictive performance. The calibration curve, however, revealed overestimation of risk in the low-probability range and underestimation in the high-probability range (**Figure 7**), indicating that caution is needed when interpreting predicted probabilities at both extremes.

DCA of SRI for predicting MACE

DCA analysis (**Figure 8**) demonstrated that, across a high-risk threshold range of 0.1 to 0.8, the net benefit of the SRI-based model exceeded that of the null strategies. This indi-

Table 10. Multivariate logistic regression analysis of factors influencing MACE

Variables	OR	95% CI	P
SRI<50%	8.794	(0.327-236.139)	0.1953
50%≤SRI<100%	3.506	(0.468-26.251)	0.2219
Age	0.990	(0.929-1.056)	0.7693
BMI	0.873	(0.727-1.049)	0.1476
MDA	0.820	(0.438-1.534)	0.5345
SOD	1.011	(0.970-1.054)	0.5936
ACE	0.977	(0.905-1.054)	0.5436
cTnI	162.010	(0.163-160952.586)	0.1485

Note: SRI=100% was defined as the reference. SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events; BMI, body mass index; MDA, malondialdehyde; SOD, Superoxide dismutase; ACE, angiotensin-converting enzyme; cTnI, cardiac Troponin I.

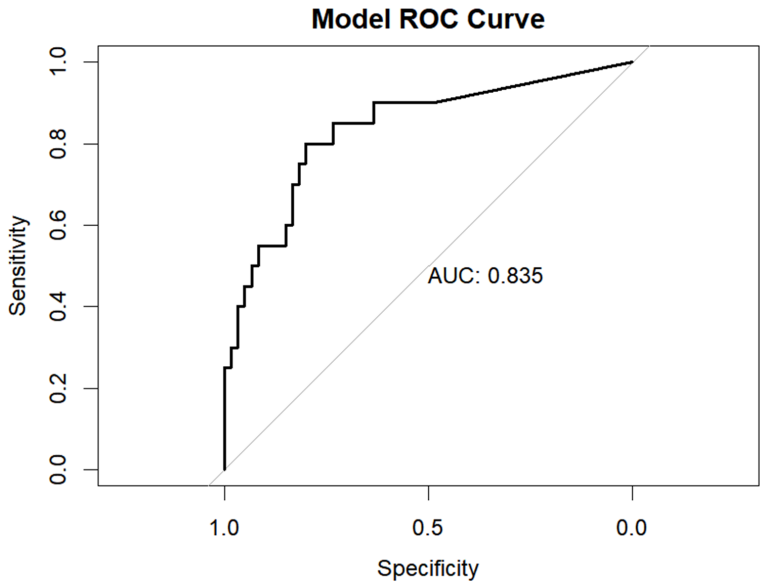

Figure 4. Receiver operating characteristic (ROC) curve of SRI for predicting MACE. Note: SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

cates that this predictive model provides a substantial net clinical benefit in guiding patient management.

Discussion

AMI is a common critical cardiovascular condition, with its pathogenesis primarily driven by myocardial hypoxia, interruption of coronary blood flow, and a complex interplay of additional mechanisms [9]. Percutaneous coronary intervention (PCI) and other reperfusion strategies remain the cornerstone of AMI treatment and have been shown to effectively improve patient prognosis. Nonetheless, a subset of patients continues to experience persistent myocardial injury and impaired cardiac function after reperfusion, which may have long-term consequences for quality of life [10, 11]. During the development of AMI, oxidative stress is a key mechanism underlying myocardial ischemia-reperfusion injury. Excessive generation of reactive oxygen species (ROS) can exacerbate inflammatory responses, promote lipid peroxidation of cardiomyocyte membranes, and impair mitochondrial function, collectively driving progressive myocardial damage and deterioration of cardiac function [12, 13]. Additionally, abnormal regulation of blood pressure may be associated with oxidative stress and endothelial dysfunction. Studies have demonstrated that such dysregulation can adversely affect myocardial perfusion and repair, potentially creating a vicious cycle that further exacerbates AMI prognosis [14, 15].

Revascularization improves myocardial perfusion and reduces residual ischemia, and it has been demonstrated to play a crucial role in the prognosis of AMI [16]. Moreover, revascularization may partially alleviate oxidative stress, thereby contributing to improved blood pressure control, enhanced endothelial function, and reduced myocardial injury [17]. In this study, at 1 month postoperatively, patients in the complete revascularization group exhibited lower levels of oxidative stress markers, including MDA and ACE, higher levels of the antioxidant enzyme SOD, and reduced cardiac injury marker cTnI compared with the partial and low revascularization groups. At 6 months postoperatively, the complete revascularization group also showed reduced 24-hour mean systolic blood pressure,

Table 11. ROC Curve analysis of SRI for predicting MACE

Index	AUC	*P*	95% CI	Optimal cutoff value	Sensitivity	Specificity
SRI	0.835	0.056	0.725-0.945	69.360%	0.800	0.800

Note: ROC curve, receiver operating characteristic curve; SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events; AUC, area under the curve; CI, confidence interval.

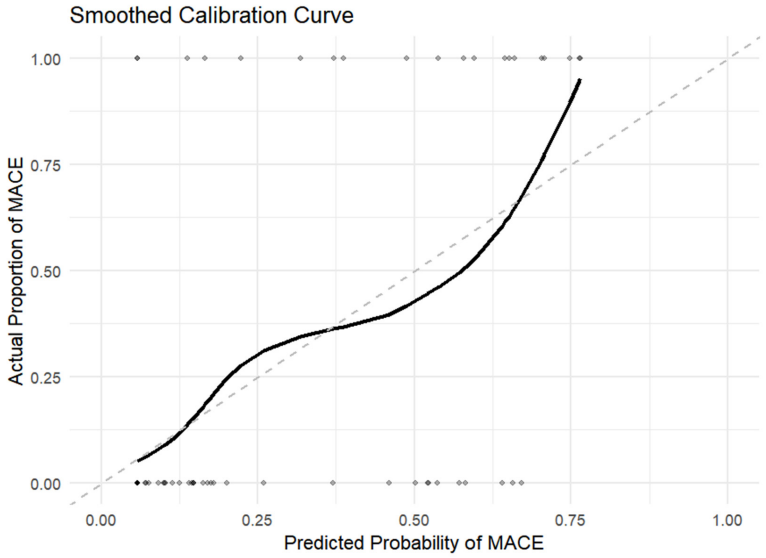

Figure 5. Calibration curve of the SRI for predicting MACE. Note: SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

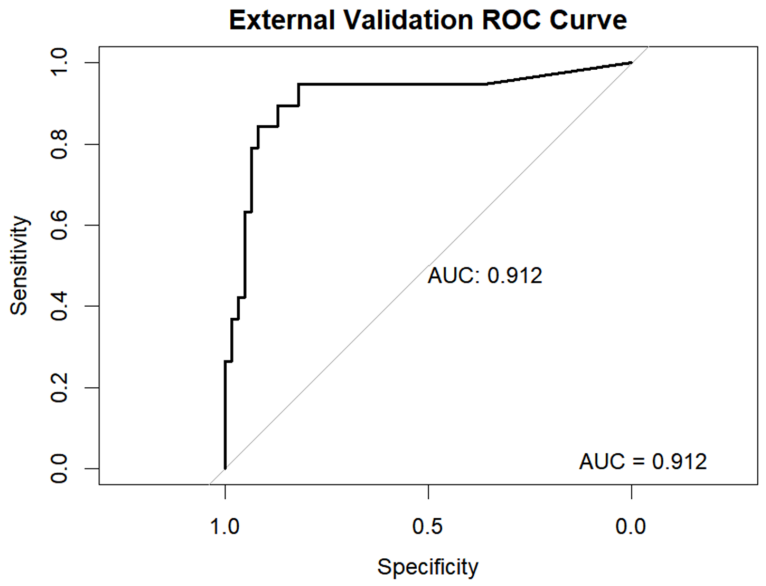

Figure 6. External validation receiver operating characteristic (ROC) curve of the SRI for predicting MACE. Note: SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

24 h SBP-SD, and nocturnal blood pressure decline relative to the other groups. The underlying mechanism may be that complete revascularization restores patency of all culprit vessels and addresses major stenotic lesions, thereby achieving more comprehensive myocardial perfusion and more effectively correcting ischemia. This process suppresses excessive ROS production, preventing amplification of oxidative stress and reducing accumulation of lipid peroxidation products such as MDA [18]. Concurrently, complete revascularization restores overall coronary blood flow, inhibits overactivation of the renin-angiotensin-aldosterone system (RAAS), and reduces the activity of key RAAS enzymes, including ACE [19]. SOD, a key endogenous antioxidant enzyme, helps eliminate ROS and mitigate oxidative damage. By alleviating myocardial metabolic disturbances, complete revascularization promotes SOD generation and restores its enzymatic activity. In terms of myocardial protection, more comprehensive revascularization facilitates the restoration of myocardial microcirculation, thereby reducing the release of the cardiac injury marker cTnI. Furthermore, at 6 months postoperatively, the complete revascularization group exhibited lower 24 h SBP, 24 h SBP-SD, and nocturnal blood pressure decline, indicating that complete revascularization not only enhances myocardial perfusion and oxygenation but also contributes to more stable blood pressure regulation. In this study,

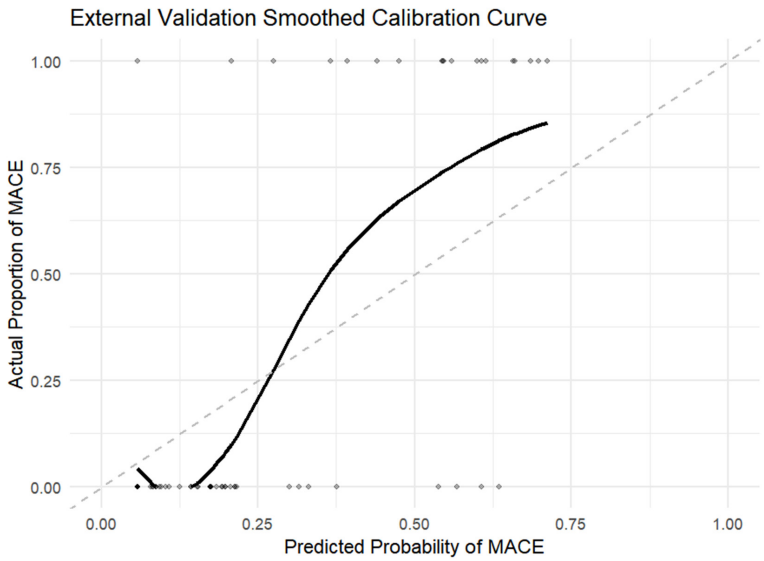

Figure 7. External validation calibration curve of the SRI for predicting MACE. Note: SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

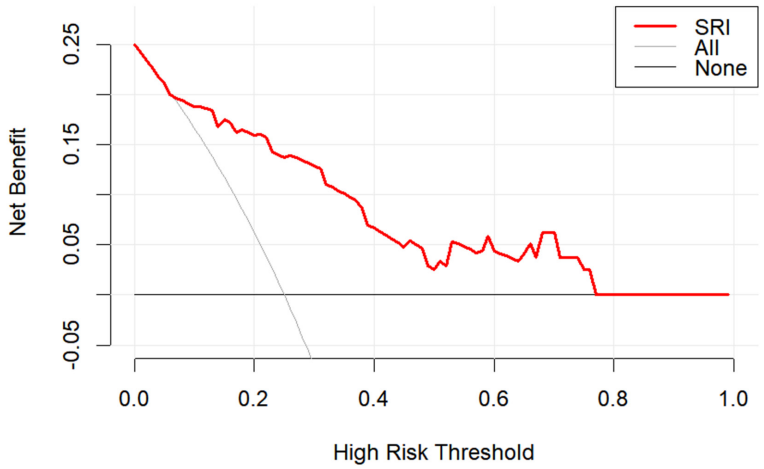

Figure 8. DCA curve of SRI for predicting MACE. Note: DCA, decision curve analysis; SRI, SYNTAX Revascularization Index; MACE, major adverse cardiovascular events.

Pearson correlation analysis demonstrated that oxidative stress markers MDA and ACE were positively correlated with cTnI and 24 h SBP-SD, whereas SOD was negatively correlated with these parameters. Linear regression analysis further indicated that SRI was a negative predictor of both cTnI levels and 24 h SBP-SD. These findings are basically consistent with those in the literature, and suggest that higher SRI values are associated with reduced myocardial injury and lower blood pressure variability, supporting the potential utility of SRI as an effective predictor of myocardial damage and blood pressure stability [20]. The underlying mechanism may be that more complete revascularization ensures more adequate myocardial perfusion, mitigating ischemia-reperfusion injury and preserving myocardial contractile function and reserve capacity. Consequently, higher SRI levels exert a stronger inhibitory effect on cTnI elevation and blood pressure variability.

Further results of this study demonstrated that the incidence of MACE within 1 year was lower in the complete revascularization group compared with the non-complete revascularization group. In the multivariate logistic regression model of this study, SRI was analyzed as a continuous predictive variable to assess the independent influence of the degree of revascularization on MACE risk, rather than a simple binary classification. The results indicated that SRI is an independent protective factor for MACE. The underlying mechanism may be that, compared with non-complete revascularization, complete revascularization restores patency of all culprit vessels, more thoroughly corrects coronary stenosis, and more effectively enhances myocardial perfusion. This directly reduces cardiomyocyte necrosis and apoptosis, facilitates restoration of coronary microcirculation, and improves myocardial reserve capacity and ischemic tolerance, thereby effectively lowering the risk of severe arrhythmias, heart failure, and other MACE [21]. Additionally, ROC curve analysis in this study demonstrated that SRI had a high predictive value for MACE, with an AUC>0.8 and an optimal cutoff of 69.36%. Calibration curves indicated that the observed incidence of MACE was generally consistent with the pre-

dicted probability, although some deviations were noted, suggesting that calibration requires further refinement. External validation yielded an SRI AUC of 0.912, indicating strong predictive performance; however, the calibration curve showed overestimation of risk in the low-probability range and underestimation in the high-probability range, highlighting the need for cautious interpretation at both extremes. DCA analysis demonstrated that across a high-risk threshold range of 0.1 to 0.8, the net benefit of the SRI-based curve exceeded that of the two null lines, indicating substantial clinical utility. Future studies should focus on improving model calibration and further validating its generalizability in larger cohorts.

This study has several limitations. First, its retrospective design and relatively small sample size may introduce uncontrolled confounding bias and limit the generalizability of the findings. Second, markers of oxidative stress and ambulatory blood pressure parameters were measured at a single postoperative time point, which precluded characterization of their dynamic trajectories and limited in-depth analysis of their temporal relationships. In addition, unmeasured confounding factors may have influenced the accuracy of the SRI's predictive value. Future studies should adopt prospective, multicenter designs and systematically collect data at multiple time points to validate these findings and elucidate the underlying mechanisms.

Furthermore, a systematic univariable logistic regression screening of all potential variables was not performed in this study. This is because the primary aim of our research was to investigate the mechanistic relationship between the completeness of revascularization and specific physiological parameters (oxidative stress, myocardial injury, and blood pressure stability), rather than to construct a comprehensive predictive model for MACE. Consequently, the original database was not pre-specified or grouped based on MACE outcomes. This design limitation precludes a direct comparison of baseline characteristics between the MACE and non-MACE groups. Future prospective studies with prognosis prediction as a primary objective should incorporate such prespecified groupings and systematic variable screening through univariable and multivariable analyses to validate our findings.

In summary, complete revascularization improves oxidative stress, stabilizes blood pressure, and enhances long-term cardiovascular prognosis in patients with AMI. Even when complete revascularization is not feasible, achieving an SRI of at least 69.36% should be targeted to optimize myocardial protection and blood pressure control.

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Disclosure of conflict of interest

None.

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