Association of SYNTAX score with myocardial injury in STEMI patients: a cardiac magnetic resonance study

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Abstract

SYNTAX score (SS) is positively correlated with postprocedural myocardial injury identified by high sensitivity cardiac troponin in patients undergone elective coronary artery intervention, evidences about the association of SS with myocardial injury in STEMI patients were still scarce. A total of 149 consecutive patients within 24 h of STEMI were enrolled in the study. Both angiography and cardiac magnetic resonance (CMR) were performed during hospitalization. The time was 7.05 h (4.44,95.91, IQR) from symptom to angiography and 7.31 ± 2.60 days from symptom to CMR. The total median SS was 17(9–25, IQR). In terms of myocardial injury parameters, there was a positive correlation between SS and infarct size (IS) ($p < 0.001$, Spearman $r = 0.292$), and negative correlation between SS and myocardial salvage index (MSI) ($p < 0.001$, Spearman $r = -0.314$). There was no significant correlation between SS and area at risk, microvascular obstruction or intramyocardial hemorrhage. According to SS, patients were divided into low SS (< 22) (LSS)($n = 96$) or mediate-high SS (≥ 22) (MHSS)($n = 53$) group. In the multivariable model, after adjustment for important known predictors of IS, MHSS was significantly associated with high IS ($\geq$ mean 35.43) (odd ratio = 2.245, 95% confidence interval [1.002–5.053], $p = 0.048$), as a continuous variate, SS was also significant associated with high IS (odd ratio = 1.053, 95% confidence interval [1.014–1.095], $p = 0.008$). The areas under the receiver operating characteristic curves of SS for high IS and low MSI were 0.664 and 0.610. Conclusion: of STEMI patients who presented to hospital within 24h from symptom onset, SS was positively related with IS and negatively with MSI. SS was an independent predictor of IS after adjusting for important covariates.

Introduction

It was well established that in acute myocardial infarction patients, high infarct size (IS) and intramyocardial hemorrhage (IMH) were closely related to bad prognosis[1, 2]. Microvascular obstruction (MVO) is the underlying cause for the no-reflow phenomenon in ST-segment elevation myocardial infarction (STEMI). The presence and extent of MVO measured by cardiovascular magnetic resonance imaging (CMR) after primary percutaneous coronary intervention (PCI) in STEMI are strongly associated with mortality and hospitalization for heart failure within 1 year and might be a better predictor of long-term major cardiovascular adverse events than IS[3, 4]. All these parameters, reflecting severity of myocardial injury, were typical indexes of CMR, which has emerged as a robust imaging modality for assessing patients after acute myocardial injury[5]. It is important to identify patients with severe myocardial injury so as to initiate intense care at early phase.

The anatomical Synergy between PCI with Taxus and Cardiac Surgery (SYNTAX) score is an important instrument that can help clinicians to establish the optimum revascularization approach in patients with complex coronary artery disease (CAD)[6–9]. Previous studies found that SYNTAX score (SS) correlated significantly with cardiac troponin releases after elective PCI and could predicts peri-procedural myocardial injury defined as elevated troponin I at 6–24 h post-PCI[10, 11]. Therefore, it may be reasonable to assume that the complexity of CAD represented by SS is the surrogate of myocardial injury.

The objective of this study was to investigate the association of SS with myocardial injury identified by CMR in STEMI patients.

Method

Participants

We conducted a single-center retrospective observational study at Cangzhou central hospital-a tertiary care hospital. We identified 276 STEMI patients undergone angiography between October 2018 and September 2020. The inclusion criteria were as follow: (1) electrocardiography (ECG) features consistent with acute STEMI (ST-segment elevation ≥ 1 mm in ≥ 2 limb leads or ≥ 2 mm in ≥ 2 contiguous precordial leads); (2) time duration within 24h from typical chest pain to hospitalization; (3) undergone CMR within 3~10 days from symptom onset. A total of 127 patients were excluded for the following reasons: declined CMR for personal reasons(n=16); old myocardial infarction history(n=8); unstable clinical station(n=39); claustrophobia(n=3); undergone CMR over10 days from symptom onset(n=10); hospitalization over 24h from symptom onset(n=51). Finally, 149 patients were enrolled for the analysis (**Fig.1**). The present study was approved by the ethical committee at Cangzhou Central Hospital. Requirement for informed consent was waived, because of the observational nature of the study.

SYNTAX score

The score calculator is available on the SS website (www.SYNTAXscore.com). Our analysis was performed by two independent operators. Every significant inter-observer difference required a recalculation.

Laboratory tests, echocardiography and CMR[12,13]

Blood samples were taken immediately after hospitalization, and ultrasound was performed 7 $\pm$ 3 days after PCI. CMR was performed using a 3.0-T scanner (GE Discovery MR750w; GE Healthcare, Milwaukee, WI, USA) with electrocardiographic-gated image acquisition. MRI parameters were measured on short-axis images covering the entire left ventricle (8-/0-mm slice thickness/slice gap) with the following sequences: a steady-state free precession (SSFP) cine sequence to determine the left ventricular (LV) function, mass and volume and a short-tau inversion recovery T2-weighted (T2-STIR) sequence to determine the area at risk (AAR) of myocardial infarction. Late gadolinium enhancement (LGE) images were acquired approximately 10~15 min after the intravenous administration of gadolinium-based contrast medium (0.2 mmol/kg, Magnevist, gadopentetate dimeglumine injection, Bayer) to determine the IS.

Analysis was performed using dedicated software (cmr42 version 5.11.3, Circle Cardiovascular Imaging, Calgary, Alberta, Canada). Images were anonymized, batched, and analyzed in a blinded fashion by two experienced operators. The AAR was defined as high-signal myocardial edema mass/LV mass ratio. The IS was defined as the hyperenhanced myocardium on the LGE images and is expressed as the infarcted

LV mass/LV mass ratio. MVO was defined as dark areas surrounded by hyperenhanced myocardium on the LGE images. The presence of intramyocardial hemorrhage (IMH) was defined as hypointense areas within the brighter edematous zone on T2-STIR images. The papillary muscles were included in the LV cavity volume. The regions of interest (ROIs) for the volumes of AAR, IMH, IS, and MVO were created by manually drawing the lesion contours, while the LV volume was calculated by the semiautomated drawing of endocardial and epicardial contours for the whole LV myocardium on each slice. Myocardial edema was described as areas with a signal intensity >5 SD that of remote normal myocardium. The IS was calculated using the >5 SD method. Discordant cases were reviewed and reconciled with superior imaging specialists.

Statistical methods

SPSS was used for statistical analysis. Categorical data are presented as numbers and percentages, and continuous data are presented as the mean (standard deviation) or median (interquartile range). Continuous variables with normal distributions were compared using the t-test. Continuous parameters that were not normally distributed were compared using the Mann-Whitney test. Categorical variables were compared using the chi-squared test (or Fisher's exact test when the expected value was <5). Independent predictors of high IS and low MSI were determined in a multivariate binary logistic regression model adjusted for all baseline variables found to be significant in the univariate analysis. Receiver operating characteristic (ROC) curves were generated to determine the usefulness of SS to discriminate high IS and low MSI. All statistical analyses were performed using SPSS version 20 (SPSS Inc, Chicago, IL, USA) and graphs produced using GraphPad Prism version 8.0 (GraphPad Software, La Jolla, CA, USA). A p value of < 0.05 was regarded as statistically significant.

Results

Patient characteristics

Of a total 149 patients, mean age was 59.89 ± 10.86 years, 72.5% were male. The overall median SS was 17(IQR:9-25). According to SS, patients were divided into low SS (LSS)(n=96) or mediate-high SS (MHSS) (n=53) group. Patient characteristics between two groups are shown in **Table 1**. Compared with LSS group, MHSS group had higher age and APOB, lower body weight index and creatinine clearance rate and a higher prevalence of multivessel disease, initial TIMI flow grade 0/1, and initial TIMI thrombus grade 4/5 (all $p < 0.05$).

TTE and CMR parameters

All TTE parameters were comparable (**Table 2**). Whereas CMR showed a lower LVEF (45.65 ± 12.01 VS 52.86 ± 13.45 , $p = 0.001$), MSI (24.52 [IQR:13.92-35.69] VS 30.30 [IQR:19.23-45.41], $p = 0.016$) and higher LVESV (63.96 [IQR:51.34-80.38], $p = 0.029$), IS (38.06 ± 9.40 VS 33.93 ± 11.94 , $p = 0.029$) in MHSS group patients when compared with those in LSS group. There was no significant difference between two groups in terms of MVO, IMH and AAR (**Table 2 and Fig.2**).

Correlations between SS and CMR parameters

Correlations between SS and CMR parameters are presented in **Fig.3**. SS had a positive correlation with IS(%LV) ($r=0.292$, $p<0.001$) and negative with MSI ($r=-0.314$, $p<0.001$). An example is illustrated in **Fig.4**. The ROC of SS was 66.4% at $IS(\%LV) \geq \text{mean } 35.43$ and 61.0% at $MSI \leq \text{median } 28.01$ (**Fig.5**).

Univariate and multivariate logistic regression for $IS \geq \text{mean } 35.43$ and myocardial salvage index $\leq \text{median } 28.01$

After screening of all clinical, procedural, laboratory and TTE parameters presented in both **table 1** and **table 2**, the odd ratio (OR) for $IS \geq \text{mean } 35.43$ was significant in 9 variables including initial heart rate, pro-BNP, CK, CKMB, LVEF-TTE, left ventricular end-diastolic diameter (LVEDD), infarct-related artery LAD, $SS \geq 22$ (as categorical variable) and SS (as continuous variable) (all $p<0.05$); the OR of LVEF-TTE, baseline TIMI flow 0/1 and SS (not as categorical variable) for lower MSI ($\leq \text{median } 28.01$) were significant (**Table 4**). In multivariate logistic regression model for high IS, LVEF-TTE (OR, %95CI, 0.913(0.865, 0.962), $p=0.001$) and $SS \geq 22$ (OR, %95CI, 2.245(1.002, 5.053), $p=0.048$) were significant in model 1 (**Fig.6**); in model 2, LVEF-TTE (OR, %95CI, 0.914(0.866, 0.965), $p=0.001$) and SS (as continuous variable) (OR, %95CI, 1.053(1.014, 1.095), $p=0.008$) were also the independent predictors of high IS; for low MSI, only LVEF-TTE (OR, %95CI, 0.924(0.881, 0.970), $p=0.001$) was the independent predictor (**Fig.6**).

Discussion

The present study is, to the best of our knowledge, the first report on the relationship between SS and myocardial injury in STEMI patients presenting to hospital within 24h from symptom onset. We identified three major findings: (1) there was a significant positive correlation between SS and IS and negative correlation between SS and MSI (**Fig. 3**). (2) compared with LSS group, MHSS group has lower LVEF deprived by CMR and higher LVESV, but LVEDV was comparable between groups. (3) according to the univariate and multivariate logistic models, SS (both as categorical variable and continuous variable) was the independent predictor of high IS after adjusted for confounders (**Fig. 6**).

In the CvLPRIT study[14], patients treated with a staged complete revascularization (CR) had higher SS (18.3, 15–26 vs. 16, 12–21.5, $p = 0.021$) than those treated with immediate CR. Interestingly, staged approach patients also had larger IS (%LV) (19.1, 10.2–37.1 vs. 11.6, 6.8–17.6, $p = 0.006$) and lower MSI (35.1, 5.9–66.4 vs. 61.7, 37.4–75.5, $p = 0.008$) compared with immediate CR. However, there was no direct relationship among them were stated. In another several studies[10, 11, 15, 16], in patients undergone CABG or PCI, SS was not only positively related to peri-procedural myocardial injury (deprived by elevated cardiac troponin and CKMB 6h after operation), but also an independent predictor of it. The increase in release of cardiac biomarkers after selective PCI was significantly associated with the extent of atherosclerosis identified by the SS [17]. These findings were consistent with those of our study. Furthermore, in our study, there was no significant relationship between SS and AAR was detected, thus,

the negative correlation between SS and MSI might be interpreted by the positive correlation between SS and IS.

In our study, the LVEF-TTE in LSS group was comparable with that in MHSS group (57.63 ± 8.87 VS 54.77 ± 9.55 , $p = 0.070$), however, LVEF-CMR in LSS group was significantly higher than that in MHSS group (52.86 ± 13.45 VS 45.65 ± 12.01 , $p = 0.001$). In our study population, LVEF-CMR was lower than LVEF-TTE, in parallel to another recent multicenter registry[18]. Compared with LVEF-TTE, LVEF-CMR significantly improved MACE prediction in the group of patients with echocardiography-LVEF < 50%, and had better prognostic meaning[18].

The influence of SS on myocardial injury after STEMI could have important clinical implication. In China, CMR was available in only few of large hospitals. Risk stratification for STEMI patients is very important. By evaluating the SS, we might find the patients who have high risk of myocardial injury. Despite numerous failures to date, the prevention and treatments for STEMI patients with high risk of myocardial injury should remain a focus of future cardiovascular research.

This study has a few limitations. First, the proportion of patients with specific SS group (< 22, $n = 96$; $22 \leq < 32$, $n = 36$; > 32 , $n = 17$) in our population was too small to allow conclusions in all 3 groups. Consequently, tests of interaction were underpowered, especially when adjusted for covariates. Therefore, bias from residual confounding factors could still be present. Second, although all patients were enrolled consecutively, part of them declined CMR for personal reasons or unstable clinical station, for example, severe heart failure; on the other hand, only patients presented to hospital within 24h from symptom onset were included in our study population, thus it should be very cautious to interpretate the conclusions in the specific patients.

In conclusion, of STEMI patients within 24h from symptom onset, SS was positively related with IS and negatively with MSI. SS was an independent predictor of IS after adjusting for important covariates.

Declarations

Funding None

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

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Tables

Table 1 Demographic and Clinical characteristics of patients

	Whole (n=149)	SS<22 (n=96)	SS ≥22(n=53)	p value
Baseline Characteristics				
Age	59.89±10.86	58.32±11.47	62.72±9.09	0.011
Male	108(72.5)	68(70.8)	40(75.5)	0.572
BMI	25.78±3.42	26.25±3.62	24.92±2.86	0.023
Hypertension	64(43.0)	45(46.9)	19(35.8)	0.228
Smoking	91(61.1)	56(58.3)	35(66.0)	0.385
Hyperlipidemia	17(11.4)	11(11.5)	6(11.3)	1.0
Diabetes	35(23.5)	21(21.9)	14(26.4)	0.550
Initial heart rate	76.79±13.35	76.53±11.69	77.27±16.07	0.645
GRACE score	109.97±22.73	107.32±22.81	114.75±22.06	0.089
CRUSADE score	18.0(9.0,24.0)	15.0(9.0,22.0)	18.5(12.8,29.3)	0.056
Procedural data for the patients				
Pain to door				
≤6h	99(66.4)	60(62.5)	39(73.6)	0.206
>6h	50(33.6)	36(37.5)	14(26.4)	
door to reperfusion/angiography only(h)	1.17(0.68,80.91)	1.23(0.69,10.10)	0.98(0.63,234.73)	0.830
Pain to reperfusion/ angiography only (h)	7.05(4.44,95.91)	7.33(4.68,23.39)	6.22(4.27,246.73)	0.629
Culprit lesion				
LAD	60(40.3)	37(38.5)	23(43.4)	0.603
Non-LAD	89(59.7)	59(61.5)	30(56.6)	
No. of diseased vessels				
0	4(2.7)	4(4.2)	0(0)	<0.001
1	54(36.2)	51(53.1)	3(5.7)	
2	45(30.2)	29(30.2)	16(30.2)	
3	46(30.9)	12(12.5)	34(64.2)	
Initial TIMI flow grade				
0/1	93(62.4)	52(54.2)	41(77.4)	0.008
2/3	56(37.6)	44(45.8)	12(22.6)	
Initial TIMI thrombus grade				

0~3	56(37.6)	45(46.9)	11(20.8)	0.002
4/5	93(62.4)	51(53.1)	42(79.2)	
Final TIMI flow grade				
0/1	9(6.0)	5(5.2)	4(7.5)	0.721
2/3	140(94.0)	91(94.8)	49(92.5)	
Post-dilatation in patients with stent implanting				
Yes	86(81.9)	57(81.4)	29(82.9)	0.858
No	19(18.1)	13(18.6)	6(17.1)	
Mode of reperfusion (n, %)				
Balloon angioplasty	29(19.5)	17(17.7)	12(22.6)	0.675
PCI with stent	105(70.5)	70(72.9)	35(66.0)	
Angiography only	15(10.1)	9(9.4)	6(11.3)	
Laboratory data				
Creatinine clearance rate	102.24(77.99,123.64)	111.12(79.81,127.97)	92.11(72.60,108.06)	0.016
cTnI	0.32(0.03,4.08)	0.35(0.06,5.81)	0.18(0.03,2.27)	0.207
WBC	10.51±3.62	10.64±3.69	10.27±3.53	0.566
AST	29.35(19.03,61.88)	31.70(19.85,67.0)	26.80(18.60,55.20)	0.378
CK	176.0(108.5,579.0)	218.0(110.75,616.0)	158.0(96.0,558.25)	0.337
CKMB	25.55(16.13,75.90)	25.85(16.33,61.68)	24.5(16.0,83.90)	0.821
LDL	2.96±0.85	2.89±0.76	3.07±0.99	0.272
APOA	1.06±0.22	1.06±0.22	1.05±0.21	0.704
APOB	0.99±0.26	0.96±0.22	1.06±0.30	0.038
Medication				
Aspirin	146(98.0%)	93(96.9)	53(100.0)	0.553
Clopidogrel	46(30.9)	33(34.4)	13(24.5)	0.358
Ticagrelor	101(67.8)	62(64.6)	39(73.6)	
No clopidogrel or ticagrelor	2(1.3)	1(1.0)	1(1.9)	
Statin	143(96.0)	92(95.8)	51(96.2)	1.0
Beta blocker	86(57.7)	56(58.3)	30(56.6)	0.864
ACEI	6(4.0)	3(3.1)	3(5.7)	0.666
ARB	29(19.5)	17(17.7)	12(22.6)	0.519

Categorical data is presented as absolute (percentage), normal distributed data as mean+ standard deviation, and other continuous data with medians including first and third quartiles in brackets

BMI body weight index, *GRACE* the Global Registry of Acute Coronary Events, *CRUSADE* Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the American College of Cardiology/American Heart Association Guidelines, *LAD* left anterior descending artery, *TIMI* thrombolysis in myocardial infarction, *WBC* white blood cell, *LDL* low density lipoprotein, *ACEI* Angiotensin-Converting Enzyme Inhibitors, *ARB* Angiotensin Receptor Blocker,

Table 2 TTE and CMR imaging parameters

Characteristic	Whole	SYNTAX score<22 (n=96)	SYNTAX score ≥22(n=53)	p value
TTE				
	56.60±9.19	57.63±8.87	54.77±9.55	0.070
LVEF, %				
LVEDD, mm	49.32±6.30	49.33±5.43	49.29±7.67	0.970
LAD, mm	37.75±4.96	37.55±4.68	38.12±5.48	0.512
CMR				
Symptom to CMR (days)	7.31±2.60	7.22±2.77	7.46±2.27	0.589
LVEF, %	50.29±13.37	52.86±13.45	45.65±12.01	0.001
LVEDV, mL	119.12(101.19,141.06)	117.31(102.89,136.79)	125.40(93.88,148.14)	0.262
LVESV, mL	55.01(43.99,75.05)	51.28(38.23,72.17)	63.96(51.34,80.38)	0.004
IS, % of LV	35.43±11.94	33.97±12.95	38.06±9.40	0.029
MVO				
No. (%)	54(36.2)	35(36.5)	19(35.8)	1.0
% of LV	1.35(0.40,3.20)	1.0(0.4,3.1)	1.4(0.4,4.7)	0.690
%IS	5.94(1.54,9.27)	6.01(1.48,9.25)	5.10(1.62,14.95)	0.821
ml	1.93(0.48,4.74)	1.72(0.45,4.55)	2.22(0.49,6.44)	0.765
AAR (% of LV)	51.05±15.83	50.83±16.18	51.46±15.32	0.821
IMH				
No. (%)	32(21.5)	22(22.9)	10(18.9)	0.678
ml	1.46(0.56,5.01)	1.75(0.68,4.88)	1.08(0.42,6.35)	0.535
% of (LV)	0.87(0.48,2.97)	1.13(0.47,2.93)	0.59(0.40,4.24)	0.675
%AAR	3.25(1.10,8.18)	3.70(1.62,7.31)	2.21(0.79,12.39)	0.589
MSI (%)	28.01(15.94,41.81)	30.30(19.23,45.41)	24.52(13.92,35.69)	0.016

Categorical data is presented as absolute (percentage), normal distributed data as mean+ standard deviation, and other continuous data with medians including first and third quartiles in brackets

TTE transthoracic echocardiography, *CMR* cardiac magnetic resonance imaging, *LVEF* left ventricular ejection fraction, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *IS* infarct size, *LV* left ventricular, *MVO* microvascular obstruction, *AAR* area at risk, *IMH* intramyocardial hemorrhage, *MSI* myocardial salvage index

Table 3 Correlations Among SYNTAX score and CMR parameters

	IMH (%LV)	IMH (%AAR)	MVO (%IS)	MVO (%LV)	MVO (ml)	IS (%LV)	AAR (%IS)	MSI
r (Spearman)		0.07	0.05	0.14	0.18	0.18	0.292	0.03
p		0.69	0.80	0.32	0.20	0.20	<0.001	0.76
N		32	32	54	54	54	149	149

CMR cardiac magnetic resonance imaging, *IMH* intramyocardial hemorrhage, *MVO* microvascular obstruction, *IS* infarct size, *AAR* area at risk, *MSI* myocardial salvage index

Table 4 univariable logistic regression model for IS $\geq$ mean 35.43 and myocardial salvage index $\leq$ median28.01

Predictors	Univariable model		
	p value	Odds ratio	95% confidence index
High IS ($\geq$ mean 35.43)			
Initial heart rate	0.043	1.027	1.001-1.055
Pro-BNP	0.037	1.0	1.0-1.001
CK	0.010	1.001	1.0-1.001
CKMB	0.020	1.005	1.001-1.010
LVEF-TTE	<0.001	0.892	0.847-0.939
LVEDD-TTE	0.045	1.062	1.001-1.127
IRA=LAD (ref. other arteries)	0.020	2.214	1.131-4.334
SYNTAX score			
≥ 22	0.010	2.500	1.245-5.019
as continuous variable	0.001	1.059	1.024-1.095
Lower MSI ($\leq$ median28.01)			
LVEF-TTE	<0.001	0.918	0.875-0.962
Baseline TIMI flow 0/1 (ref. TIMI flow 2/3)	0.036	2.119	1.049-4.280
SYNTAX score			
Continuous variable	0.020	1.039	1.006-1.073
≥ 22	0.214	1.561	0.773-3.152

IS infarct size, *LVEF* left ventricular ejection fraction, *TTE* transthoracic echocardiography, *LVEDD* left ventricular end-diastolic diameter, *IRA* infarct related artery, *LAD* left anterior descending artery, *MSI* myocardial salvage index, *TIMI* thrombolysis in myocardial infarction

Figures

Figure 1

Study flow chart of patient enrollment AMI acute myocardial infarction, CMR cardiac magnetic resonance imaging

Figure 2

CMR parameters comparison between MHSS group and LSS group MVO microvascular obstruction, IMH intramyocardial hemorrhage, LVEF left ventricular ejection fraction, CMR cardiac magnetic resonance imaging, IS infarct size, LV left ventricular, AAR area at risk, MSI myocardial salvage index

Image not available with this version

Figure 3

Correlations Among SYNTAX score, infarct size (%LV) and MSI LV left ventricular, MSI myocardial salvage index

Figure 4

Two case examples. Case one (a ~ e): A 59 years old man with anterior ST-elevation myocardial infarction was identified to have single total occluded LAD (STNTAX score 18.5 and infarct size 22.74); Case two (f ~ j): A 64 years old man also with anterior ST-elevation myocardial infarction was identified to have total occluded LAD and diffused diseased intermediate branch and RCA (STNTAX score 32.5 and infarct size 54.91) LAD left anterior descending artery, RCA right coronary artery

Figure 5

Receiver operating characteristic curves for SS to discriminate $IS \geq \text{mean } 35.43$ (a) and myocardial salvage index $\leq \text{median } 28.01$ (b)

Figure 6

multivariable logistic regression model for $IS \geq \text{mean } 35.43$ and myocardial salvage index $\leq \text{median } 28.01$
OR odd ratio, CI confidence interval, IS infarct size, LVEF left ventricular ejection fraction, TTE transthoracic echocardiography, MSI myocardial salvage index, TIMI thrombolysis in myocardial infarction