ORIGINAL ARTICLE

Association between SYNTAX II score and electrocardiographic evidence of no-reflow in patients with ST-segment elevation myocardial infarction

ST-segment yükselmeli miyokart enfarktüsü bulunan hastalarda SYNTAX Skoru II ile elektrokardiyografik no-reflow arasındaki ilişki

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ABSTRACT

Objective: This study was performed to examine the association between the SYNTAX II score (SS-II) and no-reflow observed on electrocardiography and examine their use in the evaluation of risk of an in-hospital major adverse cardiovascular event (MACE) in patients with ST-segment elevation myocardial infarction (STEMI).

Methods: A total of 126 consecutive STEMI patients who underwent primary percutaneous coronary intervention (pPCI) were recruited. The SS-II was derived using angiographic and basic patient clinical features. The difference in the sum of ST-segment elevations measured between before the pPCI and the assessment determined approximately 60 minutes after the pPCI was interpreted as the sum of ST-segment resolution (ΣSTR). MACE is a composite endpoint frequently used in cardiovascular research and usually includes endpoints reflecting safety and effectiveness. ΣSTR <50% was defined as incomplete ΣSTR (no-reflow group; n=44), while ΣSTR ≥50% was defined as complete ΣSTR (normal-flow group, n=82).

Results: The SS-II was significantly higher in the no-reflow group (p<0.001). SS-II and no-reflow findings were associated with MACE. Logistic regression analysis demonstrated significant predictive values of SS-II (Odds ratio [OR]: 1.169; 95% confidence interval [CI]: 1.084–1.260; p<0.001) and ΣSTR (OR: 0.764; 95% CI: 0.632–0.924; p=0.006) for in-hospital MACE.

Conclusion: SS-II was significantly associated with no-reflow as assessed by electrocardiography. In patients with STEMI, SS-II and no-reflow (incomplete ΣSTR) may be important predictive factors for in-hospital MACE.

ÖZET

Amaç: Bu çalışma, ST-segment yükselmeli miyokart enfarktüsü (STEMİ) bulunan hastalarda SYNTAX skoru II (SS-II) ile elektrokardiyografide no-reflow arasındaki ilişkiyi ve iki skorun hastane içi ciddi istenmeyen kardiyovasküler olayların (MACE) belirlenmesinde doğruluğunun incelenmesi amacıyla yapıldı.

Yöntemler: Bu çalışmaya primer perkütan koroner girişim (pPCI) uygulanmış toplam 126 ardışık STEMİ'li hasta alındı. SS-II anjiyografik ve temel hasta klinik özellikleri kullanılarak elde edildi. PPCİ'den önce ölçülen ve pPCİ sonrası yaklaşık 60 dakika sonra saptanan ST segment yüksekliklerinin toplamındaki fark ST-segment çözünürlüğü (ΣSTR) toplamı olarak yorumlanmıştır. MACE, kardiyovasküler araştırmalarda sıklıkla kullanılan bir kompozit son noktadır ve genellikle güvenilirliği ve etkinliği yansıtan uç noktaları içerir. ΣSTR <%50 inkomplet ΣSTR (no-reflow, n=44) olarak tanımlanırken, ΣSTR ≥%50 tam ΣSTR (normal-flow, n=82) olarak tanımlandı.

Bulgular: SS-II, no-reflow grubunda anlamlı derecede yüksekti (p<0.001). SS-II ve no-reflow bulguları MACE ile ilişkiliydi. Lojistik regresyon analizinde hastane içi MACE için SS-II (Odds oranı [OO]: 1.169, %95 Güven aralığı [GA]: 1.084–1.260, p<0.001) ve ΣSTR'nin (OO: 0.764, %95 GA: 0.632–0.924, p=0.006) anlamlı öngördürücü değerleri gösterildi.

Sonuç: Elektrokardiyografi ile değerlendirilen no-reflow, SS-II ile anlamlı şekilde ilişkiliydi. STEMİ'li hastalarda, SS-II ve no-reflow (inkomplet ΣSTR), hastane içi MACE için önemli öngörücü faktörler olabilir.

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Patients admitted to the hospital for ST-segment elevation myocardial infarction (STEMI) exhibit a wide variety of clinical and laboratory characteristics, as well as varying degrees of disease severity. In recent years, cardiologists have begun using risk scoring methods to determine the severity and degree of complications of cardiovascular disease in patients with STEMI.[1] The absence of a mechanism to incorporate clinical variables into a SYNTAX score was a significant limitation in patients with complex coronary artery disease (CAD). As a result of these limitations, the SYNTAX II score (SS-II) was developed.[2] The SS-II is associated with both angiographic (anatomical SYNTAX score) and clinical variables, such as age, sex, left ventricular ejection fraction (LVEF), creatinine clearance (CrCl), chronic obstructive pulmonary disease (COPD), and peripheral vascular complications. The SS-II provides a more accurate and individualized estimate of mortality, and it is therefore a more clinically useful tool in the management of complex CAD.[3]

Abbreviations:

ΣSTR	*Sum of ST-segment resolution*
BMI	*Body mass index*
CAD	*Coronary artery disease*
CI	*Confidence interval*
COPD	*Chronic obstructive pulmonary disease*
CrCl	*Creatinine clearance*
DM	*Diabetes mellitus*
HL	*Hyperlipidemia*
HT	*Hypertension*
LVEF	*Left ventricular ejection fraction*
MACE	*Major adverse cardiovascular events*
MI	*Myocardial infarction*
NOAF	*New onset atrial fibrillation*
OR	*Odds ratio*
PAD	*Pulmonary artery disease*
pPCI	*Primary percutaneous coronary intervention*
ROC	*Receiver operating characteristic*
SS-II	*SYNTAX score II*
STEMI	*ST-segment elevation myocardial infarction*
TIMI	*Thrombolysis in Myocardial Infarction*

Reperfusion through primary percutaneous coronary intervention (pPCI) prevents ischemic myocardial dysfunction. However, some studies have shown no functional improvement in the left ventricle despite normal flow in the epicardial coronary artery. A "no-reflow" phenomenon has been defined as myocardial perfusion failure without a cause for impeded flow, such as dissection, mechanical obstruction, or distal embolism, despite opening the responsible artery through percutaneous intervention.[4,5] Inadequate microvascular perfusion has been associated with a poor prognosis.[6,7] Despite normal epicardial coronary flow, reduced myocardial reperfusion has been associated with distal embolization, vasoconstriction, neutrophil activation, intravascular thrombus formation, platelet aggregation, tissue edema, and myocardial contracture.[8–10] Electrocardiography is the preferred method for tissue-level reperfusion assessment, as it is reliable, simple, and accessible.[11,12]

The present study was performed to examine the relationship between SS-II and no-reflow in patients with STEMI who are undergoing pPCI, and the sum of ST-segment resolution (ΣSTR) for risk stratification of an in-hospital major adverse cardiovascular event (MACE).

METHODS

Study design

This observational, cross-sectional study was performed at a training and research hospital in compliance with the Declaration of Helsinki. Upon receiving approval from the local ethics committee, informed consent was requested and obtained from all of the participating patients.

Study population

A total of 156 consecutive patients with STEMI who were admitted to the coronary care unit of the institution between December 2016 and June 2017 were selected. Six patients were excluded from the research due to renal failure (serum creatinine level >2.5 mg/dL), 13 due to ΣSTR-related confounders (newly developed left branch block, left ventricular hypertrophy), and 3 for inadequate qualitative and quantitative analyzes of ΣSTR. In addition to these exclusionary criteria, 6 patients with a history of coronary artery bypass graft and 2 patients with other severe diseases with <1 year expected survival were excluded from the study. In all, remaining 126 eligible patients were enrolled after undergoing pPCI.

Study protocol

STEMI was defined as follows: presentation with symptoms of ischemia that increased or occurred at rest within 12 to 24 hours of symptom onset; typical rise or fall in cardiac biomarker levels; ST-segment elevation >2 mm in ≥2 contiguous leads, with leads V1, V2, and V3 measuring at least 0.2 mV or remaining leads measuring at least 0.1 mV; and new or presumed left-bundle branch block observed on electrocardiography.[13] A diagnosis of hypertension (HT) was made if patients were currently on therapy and/or had arterial blood pressure >140/90. Diabetes

mellitus (DM) was diagnosed when patients were currently on diabetic medication and/or when the fasting blood glucose level was ≥126 mg/dL. Hyperlipidemia (HL) was defined as a cholesterol level >200 mg/dL, triglyceride level >150 mg/dL, history of dyslipidemia, and/or using anti-lipidemic therapy. Active smokers or patients with a smoking history of at least 1 pack/year until 1 month before inclusion in the study were considered to have a smoking history. Family history of sudden cardiac death was defined as cardiac death in a first-degree male or female family member at <55 or <65 years of age, respectively. Multivessel disease was diagnosed when there was evidence of 2 or more major epicardial coronary arteries with >50% narrowing. Body mass index (BMI) was measured as body weight (kg) divided by height squared (m).

Coronary angiography and primary percutaneous coronary intervention

Selective coronary angiography was performed using the Judkins technique at 30 frames/second in multiple angulated views. Angiograms were measured by 2 independent, blinded cardiologists. In cases of disagreement, the final decision was made by consensus. Aspirin (300 mg), clopidogrel (loading dose of 600 mg and a maintenance dose of 75 mg), and unfractionated heparin (70 IU/kg bolus) were administered during the procedure. Tirofiban was administered during the pPCI according to the operator's preference. Dual antiplatelet therapy, beta-blocker, angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, and statin were administered as a maintenance therapy for all participants.[14]

SYTAX II score

The SS-II for PCI was derived from the basic clinical features of the patients, as described previously.[2] Briefly, the SS-II was calculated and points were added to the score according to a predefined algorithm, taking 6 other clinical variables (age, sex, LVEF, CrCl, COPD, and peripheral artery disease [PAD]) into account. The Cockcroft and Gault formula was used to calculate CrCl.[15] M-mode echocardiography was performed to assess the LVEF in the 2-dimensional echocardiography. In accordance with the EuroSCORE definition, COPD was defined as the long-term use of bronchodilators or steroids due to lung disorders.[16] Peripheral vascular disease was identified in accordance with the definition in the Arterial Revascularization Therapies Study Part I (ARTS I).[17]

Laboratory analysis and electrocardiography

Blood sample was drawn from the antecubital vein in the emergency department and used for laboratory analyses. A routine laboratory blood work-up was conducted for all of the patients. The first electrocardiographic measurements were performed immediately before pPCI. Sixty minutes after the pPCI, the ST-segment elevation was measured at 20 milliseconds from point J on the electrocardiogram. DI, aVL, and V1 via V6 leads were used to measure total ST-segment elevation for anterior infarctions, and DII, DIII, and aVF for inferior infarctions.[18] The difference between the pre-pPCI and post-pPCI ST-segment elevation sums was used as the ΣSTR. The sum of the pre-pPCI and post-pPCI ST-segment elevation measurements was compared with ΣSTR, and a no-reflow group assignment was made if the ΣSTR was <50%, while it was categorized as normal flow if the ΣSTR was ≥50%.

Major adverse cardiovascular events

In-hospital MACE included non-fatal myocardial infarction (MI), in-hospital mortality, and stent thrombosis before discharge. Non-fatal MI was defined as persistent in-hospital chest pain upon follow-up, or electrocardiographic changes occurring concurrent with a new measurement of ≥20% elevated cardiac biomarkers following the recurrent pain. Stent thrombosis was defined as described previously.[19] In-hospital mortality was defined as death during hospitalization due to cardiac arrest, MI, or other cardiac cause.

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). The normality of the distribution of the data was determined with the Kolmogorov–Smirnov test. Continuous variables are presented as mean±SD and/or median, while categorical variables are shown as numbers and proportions. Student's t-test was performed for the comparison of continuous parametric variables, while the Mann-Whitney U test and a chi-square test were used to compare continuous and categorical nonparametric variables, respectively. Univariate regression analysis was performed to examine associations between MACE and

other variables, while multivariate logistic regression analysis identified clinical predictors of MACE. In univariate logistic regression analysis, variables with a p-value <0.25 were identified as potential risk markers and included as covariates in multivariate analysis The final model was generated by determining the most discriminating factors between the groups using the backward logistic regression method. Odds ratio (OR) and confidence interval (CI) were determined. A receiver operating characteristic (ROC) curve was used to assess the predictive accuracy of SS-II for PCI and ΣSTR with respect to MACE.

Table 1. Baseline demographic and clinical parameters of the study population

Variables	No-reflow group (n=44) incomplete ΣSTR			Normal-flow group (n=82) complete ΣSTR			*p*
	n	%	Mean±SD	n	%	Mean±SD	
Age (years)			68.7±7.6			63.4±8.3	0.001
Gender (male)	41	50.0		23	52.5		0.808
Diabetes mellitus	10	22.7		6	7.3		0.013
Smoking	31	70.5		19	23.2		<0.001
Hypertension	42	95.0		35	42.7		<0.001
Hyperlipidemia	20	45.5		12	14.6		<0.001
Peripheral arterial disease	12	27.3		8	9.8		0.010
Chronic obstructive pulmonary disease (%)	13	29.5		6	7.0		0.001
Previous stroke	12	27.3		7	8.5		0.005
Family history	20	45.5		12	14.6		<0.001
Previous medications							
Acetylsalicylic acid	7	15.9		16	19.5		0.618
Beta blockers	7	15.9		11	13.4		0.703
ACEI/ARB	10	22.7		17	20.7		0.795
Statin	6	13.6		8	9.8		0.509
Calcium channel blockers	7	15.9		10	12.2		0.561
Body mass index (kg/m²)			26.9±3.8			24.8±2.7	0.002
Syntax score			19.9±5.0			15.6±5.2	<0.001
Syntax II score			46.4±10.6			31.1±9.1	<0.001
Multivessel disease	33	75.0		20	24.4		<0.001
Tirofiban	20	45		61	74		<0.001
Angiographic success of the procedure	35	27.8		67	53.2		0.768
Left ventricular ejection fraction (%)			53.2±3.7			58.0±3.3	<0.001
Glucose (mg/dL)			127.3±60.9			116.9±53.0	0.103
Creatinine clearance (ml/min)			72.5±43.0			75.9±43.6	0.677
Total cholestrol (mg/dL)			175.9±48.2			174.4±44.4	0.864
Triglyceride (mg/dL)			177.9±34.1			173.7±44.2	0.648
HDL-cholesterol (mg/dL)			30.7±8.8			33.6±9.7	0.092
LDL-cholesterol (mg/dL)			109.8±31.1			109.5±27.7	0.955
White blood cell ($10^3 \times \mu L$)			10.9±7.3			10.4±3.8	0.678
Hemoglobin (g/dL)			13.4±2.2			14.9±4.3	0.474
Platelet ($10^3 \times \mu L$)			242.5±76.8			237.6±62.8	0.699

ACEI: Angiotensin-converting enzyme inhibitors; ARB: Angiotensin receptor blockers; ΣSTR: ST-segment resolution; HDL: high density lipoprotein; LDL: low density lipoprotein.

RESULTS

The study population consisted of 126 patients with STEMI: 44 patients were assigned to the no-reflow group (28.6%; mean age: 68.7±7.6 years) and 82 patients assigned to the normal-flow group (22.2%; mean age: 63.4±8.3 years). The baseline demographic and clinical parameters of the study population are provided in Table 1. The incidence rate of HT, HL, DM, smoking, PVD, COPD, family history of CAD, multivessel disease, and previous stroke was greater in the no-reflow group than in the normal-flow group ($p<0.05$ for all). All of the patient groups had similar previous medication usage and angiographic procedure success ($p>0.05$). Tirofiban usage (45% vs 74%; $p<0.001$) was significantly lower in the no-reflow group. SS, SS-II

Table 2. In-hospital adverse events

Variables	Overall (n=126)		No-reflow group (n=44) incomplete ΣSTR		Normal-flow group (n=82) complete ΣSTR	
	n	%	n	%	n	%
Major adverse cardiac event	29	23	18	40.9	11	37.9
In hospital mortality	9	7.1	7	15.9	2	2.4
Non-fatal myocardial infarction	15	11.9	9	20.5	6	7.3
Stent thrombosis	13	10.3	9	7.1	4	4.9

Table 3. Univariate logistic regression analysis showing the predictors of major adverse cardiac events

Variables	Odds ratio*	95% Confidence interval*	*p*
Gender (male), (%)	0.012	-0.140–0.160	0.896
Age (years)	-0.044	-0.001–0.013	0.079
Hypertension (%)	-0.030	-0.217–0.165	0.786
Hyperlipidemia (%)	-0.153	-0.342–0.036	0.112
Diabetes (%)	0.079	-0.121–0.322	0.373
Smoking (%)	-0.002	-0.192–0.189	0.822
SYNTAX II score	0.481	0.394–1.364	<0.001
Left ventricular ejection fraction (%)	0.084	-0.012–-0.151	0.420
Body mass index	-0.035	-0.058–0.011	0.004
Incomplete ΣSTR (no-reflow)	-0.424	-0.668–-0.181	0.001

*Values were obtained by the univariate logistic regression analysis. ΣSTR: ST-segment resolution.

Table 4. Multivariate logistic regression analysis showing the predictors of major adverse cardiac events

	Odds ratio*	95% Confidence interval*	*p*
Step 1			
SYNTAX II score percutaneous coronary intervention	5.679	1.935–16.669	0.002
Body mass index	1.041	0.970–1.118	0.265
Incomplete ΣSTR (no-reflow)	0.784	0.658–0.933	0.006
Step 2 (final step)			
SYNTAX II score percutaneous coronary intervention	7.417	2.783–19.767	<0.001
Incomplete ΣSTR (no-reflow)	0.792	0.667–0.941	0.008

Final steps of the backward logistic regression method are shown. MACE: Major adverse cardiovascular events; ΣSTR: ST-segment resolution.

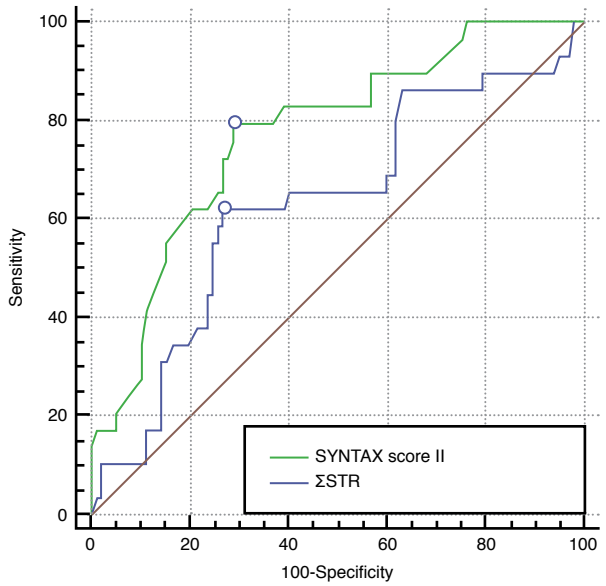

Figure 1. Comparison of receiver operating characteristic curves for the SYNTAX score II for primary percutaneous coronary intervention and sum of ST-segment resolution in the risk stratification of in-hospital major adverse cardiac event in ST-segment elevated myocardial infarction patients.

for PCI, BMI, and age were significantly greater in the no-reflow group than in the normal-flow group ($p<0.05$ for all). There were no significant differences in laboratory values between the groups ($p>0.05$) (Table 1).

In-hospital adverse events are reported in Table 2. The overall rate of in-hospital MACE ($p<0.001$), in-hospital mortality ($p=0.005$), non-fatal MI ($p=0.030$), and stent thrombosis ($p=0.006$) was greater in the no-reflow group compared with the normal-flow group.

The OR and 95% CI for each parameter using a univariate logistic regression model are listed in Table 3. According to these results, high SS-II for PCI, no-reflow, and BMI were associated with MACE. However, only SS-II for PCI (OR: 1.169; 95% CI: 1.084–1.260; $p<0.001$) and no-reflow (OR: 0.764; 95% CI: 0.632–0.924; $p=0.006$) were independent predictors of MACE in multivariate analysis (Table 4).

Comparison of the diagnostic accuracy of SS-II for PCI for the determination of MACE and ΣSTR using pairwise ROC curve analysis yielded no significant differences (Fig. 1).

DISCUSSION

In this study, the relationship between SS-II and no-reflow observed on electrocardiography (incomplete ΣSTR) was evaluated in STEMI patients treated with pPCI. The main findings of this study were as follows: *(i)* SS-II was significantly associated with no-reflow as assessed by electrocardiography, and *(ii)* SS-II is an independent predictor of in-hospital MACE.

The SS-II was developed to add to the decision-making power of the SYNTAX score related to PCI.[2] The new scoring system, created by combining anatomical and clinical variables, has demonstrated an absolute superiority over the original, purely anatomical SS.[20] In the SIRTAX (Paclitaxel-Eluting Stents for Coronary Revascularization) study, Girasis et al.[21] analyzed the anatomical SS in conjunction with age, creatinine level, and ejection fraction, and found it to be useful for predicting events predominantly in STEMI patients with pPCI. Compared with the anatomical SS, the clinical SS has demonstrated stronger discriminative potency in predicting 5-year all-cause mortality.[3] Similarly, in the ARTS II study (Study II of Arterial Revascularization Treatments), clinical SS significantly predicted both mortality and composite ischemic endpoints over a 5-year period.[22]

In a multicenter study conducted by Rencuzogullari et al.,[23] patients with contrast-induced nephropathy were older, had a reduced estimated glomerular filtration rate on admission, lower LVEF, and increased incidence of PAD. Therefore, it is plausible that patients with a higher SS-II would also have an increased incidence of contrast-induced nephropathy. Farooq et al.[24] reported that better and more specific patient categorization results can be obtained with the addition of significant clinical variables to the SS. The combination of the EuroSCORE and SS showed an enhanced risk prediction ability for adverse events in CAD patients compared with SS alone.[25,26] SS-II is a superior tool to predict MACE in patients with STEMI undergoing pPCI.[27]

Rencuzogullari et al.[28] conducted a comprehensive evaluation of the relationship between CAD severity and new onset atrial fibrillation (NOAF) using SS and SS-II scores. There was a strong correlation between a high SS-II and NOAF, and patients with NOAF had a poorer prognosis in the long-term follow-up in their study. In another study, Rencuzogullari et al.[29] evaluated the relationship between SS-II and cardiac rupture in patients with STEMI treated with pPCI. SS-II was shown to be an independent factor related to the

development of cardiac rupture in STEMI patients. Karakoyun et al.[30] reported that the relationship between hemoglobin A1c and SSII was stronger than that with the original SS.

No-reflow is an important complication in patients (50%) following pPCI, because it is associated with a poor long-term outcome.[31] Consistent with previous studies, no-reflow was seen in 34.9% of our patients. Microvascular plaques are important in the pathogenesis of no-reflow and, as mentioned in previous studies, the formation of emboli with plaque fragmentation can occur either as a result of plaque characteristics, or as a result of platelet-platelet or platelet-leukocyte clustering.[32] Rapid ST-segment resolution within 30 to 60 minutes of successful pPCI has been shown to predict greater improvement in ejection fraction, reduced infarct size, and improved survival compared with delayed ST-segment resolution.[33] In a multicenter study conducted by Chan et al.,[34] patients with no-reflow had poorer in-hospital and 30-day clinical outcomes and a higher overall incidence of MACE at the beginning and end of the PCI procedure compared to normal reflow patients. Our findings were also consistent with the literature.[35–37]

Many studies, such as the research conducted by Abdi et al.,[9] have investigated the relationship between no-reflow and increased inflammation with platelet reactivity. The authors showed that the white blood cell count and thrombus grade after pPCI were independent factors associated with no-flow. To estimate the incidence of no-reflow, Niccoli et al.[38] assessed the role of plasma TXA2 levels. It is clear that TXA2 plays an important role in platelet activation and aggregation, and in platelet-derived coronary vasoconstriction. Although a number of studies have addressed no-reflow and cardiovascular events in relation to each other, the correlation between no-reflow and long-term prognosis remains unclear. Thus, in the early period, no-reflow has greater prognostic significance.[39]

In clinical practice, a variety of methods have been used to identify reperfusion success in the setting of STEMI, including the Thrombolysis in Myocardial Infarction (TIMI) grade, corrected TIMI frame count, myocardial blush grade, and ΣSTR.[40–42] ST-segment changes reflect the myocardial flow more than epicardial flow, which provides better prognostic information than coronary angiography. Many studies have shown strong associations between ST-segment recovery of ≥70% (complete resolution), infarct size, and morbidity and mortality.[43,44] Similar to previous studies, we found that older age, history of DM, smoking, HT, HL, peak creatine kinase MB, decreased LVEF, lower rate of preprocedural TIMI grade 3, and a higher rate of multivessel disease were significantly associated with incomplete ST-segment recovery.[45,46]

Limitations

The main limitations of this study are its single-center design and small patient population. In addition, compared with ΣSTR alone, additional information on tissue perfusion to detect myocardial perfusion could be obtained by other means, such as quantitative myocardial contrast echocardiography. Furthermore, the myocardial blush grade was not assessed. More detailed results could be obtained using a digital electrocardiography system with high-resolution display. Finally, although many studies have demonstrated relationships between increased oxidative stress, thrombosis, and no-reflow, we did not assess elevated oxidative stress markers or platelet reactivity in the present study.

Conclusions

To the best of our knowledge, this is the first study to focus on the association between SS-II, no-reflow (incomplete ΣSTR), and in-hospital MACE in patients with STEMI undergoing pPCI. The results suggested that the SS-II and no-reflow may be important predictors of in-hospital MACE in patients with STEMI.

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