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ORIGINAL PAPER

Comparison of syntax score and syntax score II to predict "no reflow phenomenon" in patients with ST-segment elevation myocardial infarction

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Abstract Even though the relationship between syntax score (SS) and coronary no-reflow phenomenon has been studied, the relation between SS and syntax score II (SS II) in patients with no-reflow phenomenon is unknown. We aimed to define the relationship between coronary no-reflow phenomenon and SS II as compared with SS. This study enrolled 193 patients undergoing primary percutaneous coronary intervention for ST elevation myocardial infarction in whom 42 patients developed the no-reflow phenomenon. SS and SS II were calculated in all patients. Bland Altman analysis was used to compare receiver-operating characteristic (ROC) curve analysis results. SS and SS II values were significantly higher in the no-reflow group than the reflow group (28.3 ± 5.5 vs. 18.8 ± 10.1; $p < 0.001$ and 42.5 (22.1–58.5) vs. 26.1 (13–49.8); $p < 0.001$ respectively). SS II value >32.3 yielded an area under the curve value of 0.881 (95% CI 0.820–0.942; $p < 0.001$) and independently predicted no-reflow with a sensitivity of 88% and a specificity of 80% (OR 1.150, 95% CI 1.047–1.263, $p = 0.003$). Comparison of ROC curve results with Bland Altman analysis showed that area under curve of SS II was larger than that of SS (0.881 vs. 0.785, $p = 0.01$). SS II may be a more useful tool than SS for prediction no-reflow phenomenon after primary percutaneous coronary intervention in patients with ST elevation myocardial infarction.

Keywords No reflow · Coronary artery disease · ST-elevation myocardial infarction · Syntax score · Syntax score II

Introduction

Acute myocardial infarction (AMI) is one of the leading cause of death in modern era [1]. No-reflow phenomenon is not a rare complication of primary percutaneous coronary intervention (pPCI). It is described as inadequate myocardial perfusion without evidence of vessel obstruction. The frequency of no-reflow phenomenon is 5–50% after pPCI following myocardial infarction [2, 3].

The syntax score (SS) is an anatomic scoring system based on the coronary angiogram that quantitatively characterizes the coronary vasculature with respect to the number, location, complexity, and functional impact of angiographically obstructive lesions [4]. SS is measured to define the coronary artery complexity and allows prospective risk stratification of patients undergoing PCI. Farooq et al. developed SS II to predict mortality for patients with complex lesion following PCI and coronary artery by-pass graft (CABG) surgery [5]. The SS II briefly contain six clinical variables [age, sex, creatinine clearance, peripheral vascular disease, chronic obstructive pulmonary disease (COPD) and left ventricular ejection fraction] and

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two anatomical variables [anatomical SS and unprotected left main coronary artery (ULMCA) disease]. Although the relationship between coronary artery complexity and no-reflow phenomenon has been previously investigated [6, 7], the relation between SS and SS II in patients with no-reflow phenomenon has not been studied yet.

In the present study, we aimed to investigate the association between no-reflow phenomen and extent and complexity of coronary artery disease (CAD), calculated by SS and SS II, in patients who underwent pPCI for ST elevation myocardial infarction (STEMI).

Materials and methods

Study patients

A total of 193 consequtive STEMI patients (125 males, mean age, 61.5 ± 9 years) who underwent pPCI between July 2015 and September 2016 were enrolled in the study. The patients with end stage liver and renal disorders, coagulopathy, history of intolerance to dual anti-platelet drugs and malignancies were excluded from the study. Patients with non-STEMI and unstable angina and those treated with thrombolytics and conservative methods were also excluded.

All patients provided a written or oral-witnessed informed consent at emergency department and the protocol of study was approved by the local ethics committee of the hospital in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines.

Definitions

Admission blood samples were used for laboratory assesment. Cockroft–Gault formula was performed for calculation of glomerular filtration rate (GFR). The patients on treatment of lipid lowering therapy or those who had serum triglyceride level ≥150 mg/dL or high density lipoprotein (HDL) cholesterol <40 mg/dL or low density lipoprotein (LDL) cholesterol ≥140 mg/dL were recorded as dyslipidemic. Modified Simpson method was applied for left ventricular ejection fraction. STEMI was defined in the presence of following criteria; detection of rise and/or fall of cardiac biomarker values (preferably troponin) with at least one value above the 99th percentile of the upper reference limit and with at least one of the following: symptoms of ischaemia; new or presumably new significant ST–T changes or new left bundle branch block; development of pathological Q waves in the electrocardiography; imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality; identification of an intracoronary thrombus by angiography or autopsy [8].

Acute transient or persistent coronary flow reduction [final thrombolysis in myocardial infarction (TIMI) flow grade <3 or final myocardial blush grade (MBG) <2] at the target vessel lesion in the absence of spasm, trombus, dissection and/or significant residual stenosis was defined as epicardial no-reflow [9].

Any of major coronary vessels with a 50% or greater narrowing in diameter was defined as significant stenosis. Multivessel disease was defined if >50% diameter stenosis included ≥2 major epicardial coronary arteries. The restoration of TIMI III flow with <20% residual stenosis on visual assesment in major epicardial coronary arteries was defined as complete revascularization.

Angiographic analysis

Coronary angiography was performed via transfemoral approach to all patients. Seldinger method with a 7 french catheter was used. Patients received 600 mg clopidogrel (p.o) and 300 mg acetylsalicylic acid (p.o) and 70 IU/kg intravenous unfractionated heparin before pPCI.

Calculation syntax score and syntax score II

The SS and SS II was calculated by using the online calculator (http://www.syntaxscore.com). Two experienced interventional cardiologists who were familiar to the website and blinded to the study assessed and calculated SS and SS II. In the cases with conflicting results both cardiologists analyzed together and made a consensus. Patients were divided into two groups based on SS (Group I SS <22 and Group II SS ≥22) and SS II (Group I SS II <32 and Group II SS II ≥32).

Statistical analysis

Continuous variables are expressed as mean ± standard deviation, whereas categorical variables are expressed as percentage (%). Chi square or Fisher exact test were used for comparison of categorical data. The normality distribution of continuous variables was tested with Kolmogorov–Smirnov test. Correlation between continuous variables was assessed by Pearson correlation test and non-continuous variables was assesed by Spearman test. Student t test or Mann–Whitney U test was used to compare continuous variables between the two groups. In order to identify the independent predictors of no-reflow multivariate logistic regression analysis was performed. The cut-off values of SS and SS II for prediction of no-reflow were detected by using receiver-operating characteristic (ROC) curve analysis. A 2-sided p value of <0.05 was considered as significant. Data were analyzed by using SPSS 15.0 version (SPSS Inc, Chicago, Illinois). Bland Altman analysis

(MedCalc software for Windows) was used to compare ROC curve analysis results.

Results

A total of 193 patients [68 females (35.2%) and 125 males (64.8%)] with a mean age of 61.5 ± 9 years were included in the study. The baseline clinical and laboratory characteristics of the patients are shown in Table 1. Ninety-five patients had SS <22 and 98 had SS ≥22. Diabetes mellitus, hypertension and LMCA disease were more often present in patients with higher SS. The development of no-reflow was significantly different between the SS groups ($p < 0.001$). The clinical and laboratory characteristics of the patients according to SS are shown in Table 2.

The SS II of the patients ranged from 13 to 58.5 (median 28.3). One hundred twenty-five patients had SS II <32 and 68 had SS II ≥32. There were significant differences between two groups in terms of gender, presence of LMCA disease, peripheral artery disease, COPD and no-reflow phenomenon ($p < 0.001$ for all). The clinical and laboratory characteristics of the patients according to SS II are shown in Table 3.

No-reflow phenomenon was observed in 42 (21.8%) cases (mean age: 61.5 ± 9.1 years) and reflow group included 151 patients (mean age: 61.5 ± 9 years). Aspiration thrombectomy was performed in 53 (27.5%) patients and GP2b3a inhibitor (tirofiban) was used in 77 (39.9%) patients. The differences in the baseline clinical characteristics of patients with no-reflow and reflow are listed in Table 4.

SS, SS II, gender, hypertension, diabetes mellitus, prior stent implantation, ejection fraction, LMCA disease,

Table 1 The baseline clinical and laboratory characteristics of the patients

Variables	Values
Age (years)	61.5 ± 9.0
Sex, male (n, %)	125 (64.8)
HT (n, %)	89 (46.1)
Smoking (n, %)	81 (42)
DM (n, %)	41 (21.2)
DL (n, %)	58 (30.1)
LDL-C (mg/dL)	150 (131–178)
HDL-C (mg/dL)	34.3 ± 7.9
TG (mg/dL)	170 (138.5–196.5)
GFR (mL/min/1.73 m^2)	110 (93.5–117)
Glucose (mg/dL)	99 (88–117)
Platelet count	245 (191–333)
Hb (g/dL)	13.5 ± 1.4
ASA (n, %)	65 (33.7)
Klopidogrel (n, %)	11 (5.6)
Statin (n, %)	23 (24.7)
Beta blocker (n, %)	116 (60.1)
Insulin (n, %)	42 (21.7)
OAD (n, %)	42 (21.7)
LMCA disease (n, %)	31 (16.1)
PAD (n, %)	39 (20.2)
COPD (n, %)	58 (30.1)
Prior MI (n, %)	32 (16.6)
Prior PCI (n, %)	24 (12.4)
Multivessel disease (n, %)	89 (46.1)
No-reflow (n, %)	42 (21.8)
Syntax score	20.9 ± 10.1
Syntax score II	28.3 (23.3–35.9)

Continuous variables with normal distribution were expessed as mean ± standard deviation and continuous variables without normal distribution were expressed as median (25th–75th percentiles)

HT hypertension, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triglyceride, *GFR* glomerular filtration rate, *LMCA* left main coronary artery, *PAD* peripheral arterial disease, *COPD* chronic obstructive pulmonary disease, *DM* diabetes mellitus, *DL* dyslipidemiae, *EF* ejection fraction

Table 2 The baseline clinical and laboratory characteristics of the patients according to SS subgroups

Variables	SS ≤22 (n = 95)	SS >22 (n = 98)	p value
Age (years)	60.2 ± 8.6	62.7 ± 9.2	0.046
Sex, male (n, %)	69 (72.6)	56 (57.1)	0.024
HT (n, %)	37 (38.8)	52 (53.1)	0.049
Smoking (n, %)	40 (42.1)	41 (41.8)	0.970
LDL-C (mg/dL)	138 (71–224)	160 (108–388)	0.001
HDL-C (mg/dL)	37 ± 7.7	31.7 ± 7.3	0.000
TG (mg/dL)	167 (71–435)	176.5 (81–491)	0.386
GFR (mL/min/1.73 m^2)	110 (52–135)	105 (35–125)	0.019
LMCA disease (n, %)	6 (6.3)	25 (25.5)	0.000
PAD (n, %)	19 (20)	20 (20.4)	0.944
COPD (n, %)	32 (33.7)	26 (26.5)	0.279
DM (n, %)	10 (10.5)	31 (31.6)	0.000
DL (n, %)	28.4	31.6	0.627
No-reflow (n, %)	8 (8.4)	34 (34.7)	<0.001
EF (%)	50 (28–73)	50 (25–69)	0.647
Thrombectomy (n, %)	16 (16.8)	37 (37.8)	0.001
GP2b3a inhibitors (n, %)	32 (33.7)	35 (35.7)	0.767
TIMI frame count	19 (15–27)	21 (18–30)	0.003

Continuous variables with normal distribution were expessed as mean ± standard deviation and continuous variables without normal distribution were expressed as median (25th–75th percentiles)

HT hypertension, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triglyceride, *GFR* glomerular filtration rate, *LMCA* left main coronary artery, *PAD* peripheral arterial disease, *COPD* chronic obstructive pulmonary disease, *DM* diabetes mellitus, *DL* dyslipidemiae, *EF* ejection fraction, *SS* syntax score, *SS II* syntax score II

Table 3 The baseline clinical and laboratory characteristics of the patients according to SS II subgroups

Variables	SS II ≤32 (n = 125)	SS II >32 (n = 68)	p value
Age (years)	60.8 ± 8.9	62.7 ± 9.1	0.148
Sex, male (n, %)	98 (78.4)	27 (39.7)	<0.001
HT (n, %)	47 (37.6)	42 (61.8)	<0.001
Smoking (n, %)	52 (41.6)	29 (42.6)	0.888
LDL-C (mg/dL)	145 (71–375)	160 (99–388)	0.121
HDL-C (mg/dL)	34.4 ± 7.9	32.5 ± 8	0.803
TG (mg/dL)	168 (71–435)	179 (85–491)	0.331
GFR (mL/min/1.73 m^2)	112 (65–135)	95 (35–130)	<0.001
LMCA disease (n, %)	14 (11.2)	17 (25)	<0.001
PAD (n, %)	6 (4.8)	33 (48.5)	<0.001
COPD (n, %)	26 (20.8)	32 (47.1)	<0.001
DM (n, %)	24 (19.2)	17 (25)	0.347
Glucose (mg/dL)	98 (66–211)	99.5 (67–230)	0.404
No-reflow (n, %)	5 (4)	37 (54.4)	<0.001
Thrombectomy (n, %)	32 (25.6)	21 (30.9)	0.432
GP2b3a inhibitors (n, %)	34 (27.2)	33 (48.5)	0.003
TIMI frame count	18 (15–21.5)	28 (21.5–32)	<0.001

Continuous variables with normal distribution were expessed as mean ± standard deviation and continuous variables without normal distribution were expressed as median (25th–75th percentiles)

HT hypertension, *DM* diabetes mellitus, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triglyceride, *GFR* glomerular filtration rate, *LMCA* left main coronary artery, *PAD* peripheral arterial disease, *COPD* chronic obstructive pulmonary disease

peripheral artery disease, TIMI grade <3, blood glucose level and GFR were found to be predictors of no-reflow in univariate logistic regression analysis. Multiple logistic regression analysis provided that the SS II (OR 1.150, 95% CI 1.047–1.263, p = 0.003), LVEF (OR 0.842, 95% CI 0.763–0.930, p = 0.001), and LMCA disease (OR 15.309, 95% CI 3.086–75.942, p = 0.001) were the independent predictors of no-reflow (Table 5). The ROC curve analysis was performed to detect the best cut-off value of SS and SS II in the prediction of no-reflow. A SS value of >23.5 yielded an area under the curve (AUC) value of 0.785 (95% CI 0.721–0.850; p < 0.001). Furthermore, the SS value >23.5 demonstrated a sensitivity of 76% and a specificity of 70% for the prediction of no-reflow (Fig. 1a). Also, the ROC curve analysis was performed to detect the best cut-off value of SS II in the prediction of no-reflow. SS II value >32.3 yielded an AUC value of 0.881 (95% CI 0.820–0.942; p < 0.001). Furthermore, a SS II value of >32.3 demonstrated a sensitivity of 88% and a specificity of 80% for the prediction of no-reflow (Fig. 1b). The comparison of the ROC curves showed that AUC of SS II was larger than that of SS (0.881 vs. 0.785, p = 0.01) (Fig. 1c).

Discussion

In this study, we focused on the potential relationship between SS, SS II and the development of no-reflow in patients undergoing pPCI for STEMI. Our results indicate that higher SS II is independently associated with no-reflow and may be more useful than SS to predict no-reflow in patients undergoing pPCI for STEMI.

Several previous studies showed the relationship between no-reflow phenomenon and SS [6, 10]. No-reflow is associated with worse prognosis in patients with STEMI undergoing pPCI. There are several mechanisms that contribute to the development of no-reflow. The main mechanism is thought to be the distal embolization of intracoronary thrombus [11, 12]. In situ thrombosis is one of the proposed mechanisms that could be associated with impaired myocardial perfusion in STEMI patients undergoing pPCI. One of the no-reflow mechanisms is microvascular damage and individual susceptibility of coronary microcirculation to injury [13]. STEMI patients complicated with no-reflow have more extent coronary atherosclerosis than patients without no-reflow. Also it has been shown that SS is an independent predictor of no-reflow phenomenon [7, 10, 14]. This relationship could be explained with different mechanisms. Diffuse coronary artery disease is associated with impaired microcirculatory resistance and effects epicardial blood flow [15]. There is also a relationship between intensity and complexity of coronary artery lesion and oxidative stress in patients with acute coronary syndrome. As a result the vasodilator effect of nitric oxide, prostacyclin and adenosin is reduced [16].

Table 4 Comparison of the baseline clinical characteristics of the patients with no-reflow and reflow

Variable	No-reflow (n = 42, 21.8%)	Reflow (n = 51, 78.2%)	p value
Age (years)	61.5 ± 9.14	61.5 ± 9.03	0.950
Sex, male (n, %)	16 (8.1)	109 (72.2)	<0.001
Hypertension (n, %)	28 (66.7)	61 (40.4)	0.003
Diabetes mellitus (n, %)	14 (33.3)	27 (17.9)	0.030
Smoking status (n, %)	18 (42.9)	63 (41.7)	0.895
Prior stent (n, %)	9 (21.4)	15 (9.9)	0.046
DL (n, %)	14 (33.3)	44 (29.1)	0.6
Family history (n, %)	13 (31)	51 (33.8)	0.731
EF (%)	45 (25–60)	50 (28–73)	<0.001
LMCA disease (n, %)	20 (47.6)	11 (7.3)	<0.001
COPD (n, %)	15 (35.7)	43 (28.5)	0.366
PAD (n, %)	16 (38.1)	23 (15.2)	0.001
ASA (n, %)	13 (31)	52 (34.4)	0.673
Klopidogrel (n, %)	4 (9.5)	7 (4.6)	0.227
Statin (n, %)	11 (26.2)	12 (9.4)	0.685
Beta blocker (n, %)	24 (57.1)	92 (60.9)	0.658
Insulin (n, %)	7 (16.7)	35 (23.2)	0.667
OAD (n, %)	13 (31)	29 (19.2)	0.137
Blood glucose level (mg/dL)	110 (79–230)	96 (66–211)	0.002
Hb (g/dL) (mean ± SD)	12.5 ± 1.4	13.8 ± 1.3	<0.001
PLT count ($\times 10^3$ cells/dL)	239 (168–389)	245 (150–405)	0.717
LDL (mg/dL)	160 (106–388)	145 (71–375)	0.235
HDL (mg/dL)	32.4 ± 7.9	34.9 ± 7.9	0.083
Triglycerides (mg/dL)	178.5 (85–381)	169 (71–491)	0.986
TIMI grade <3 (n, %)	13 (31)	5 (3.3)	<0.001
GFR (mL/min/1.73 m^2)	77 (35–125)	111 (64–135)	<0.001
Infarcted related artery			
LAD (n, %)	11 (26.2)	68 (45)	
Cx (n, %)	9 (21.4)	21 (13.9)	
RCA (n, %)	16 (38.1)	54 (35.8)	
LMCA (n, %)	6 (14.3)	8 (5.3)	
Syntax score	28.3 ± 5.5	18.8 ± 10.1	<0.001
Syntax score II	42.5 (22.1–58.5)	26.1 (13–49.8)	<0.001
Thrombectomy (n, %)	17 (40.5)	36 (23.8)	0.033
GP2b3a inhibitors (n, %)	25 (59.5)	42 (27.8)	<0.001
TIMI frame count	30.5 (27–33)	18 (15–23)	<0.001

Continuous variables with normal distribution were expessed as mean ± standard deviation and continuous variables without normal distribution were expressed as median (25th–75th percentiles)

ASA acetylsalicylic acid, *COPD* chronic obstructive pulmonary disease, *Cx* Circumflex artery, *DL* dyslipidemia, *EF* ejection fraction, *DM* diabetes mellitus, *GFR* glomerular filtration rate, *Hb* haemoglobin, *HDL* high density lipoprotein, *LAD* left anterior descending artery, *LDL* low density lipoprotein, *LMCA* left main coronary artery, *MPV* mean platelet volume, *n* number of patients, *OAD* oral antidiabetics, *PAD* peripheral arterial disease, *PLT* platelet, *RCA* right coronary artery, *SD* standard deviation

Distal thromboembolism has been recognized as the predominant mechanism for no reflow. Several strategies to avoid thromboembolism are manual or mechanical aspiration thrombectomy, direct stenting without prior ballooning, embolic protection devices and intracoronary abciximab [17]. Pharmacological drug treatment of no reflow includes intracoronary administration of adenosine, calcium channel blockers (verapamil, diltiazem and nicardepine), nicorandil, sodium nitroprusside and glycoprotein 2b/3a inhibitors (abciximab, tirofiban and eptifibatide) [17]. In the present study, aspiration thrombectomy was performed in 53 (27.5%) patients and GP2b3a inhibitor (tirofiban) was used in 77 (39.9%) patients.

Table 5 Independent predictors of no-reflow phenomenon in multivariate logistic regression analysis

Variables	Univariate OR, 95% CI	Univariate p value	Multivariate OR, 95% CI	Multivariate p value
Syntax score	1.110 (1.065–1.157)	<0.001	1.085 (0.995–1.183)	0.064
Syntax score 2	1.201 (1.138–1.268)	<0.001	1.150 (1.047–1.263)	0.003
LVEF	0.829 (0.773–0.888)	<0.001	0.842 (0.763–0.930)	0.001
LMCA	11.570 (4.885–27.402)	<0.001	15.309 (3.086–75.942)	0.001
Prior PCI	2.473 (0.966–6.141)	0.046	0.981 (0.141–6.826)	0.985
Gender	4.217 (2.058–8.641)	<0.001	1.518 (0.440–5.231)	0.509
DM	2.296 (1.069–4.933)	0.030	0.876 (0.214–3.583)	0.853

DM diabetes mellitus, *LMCA* left main coronary artery, *LVEF* left ventricular ejection fraction, *PCI* primary percutaneous coronary intervention

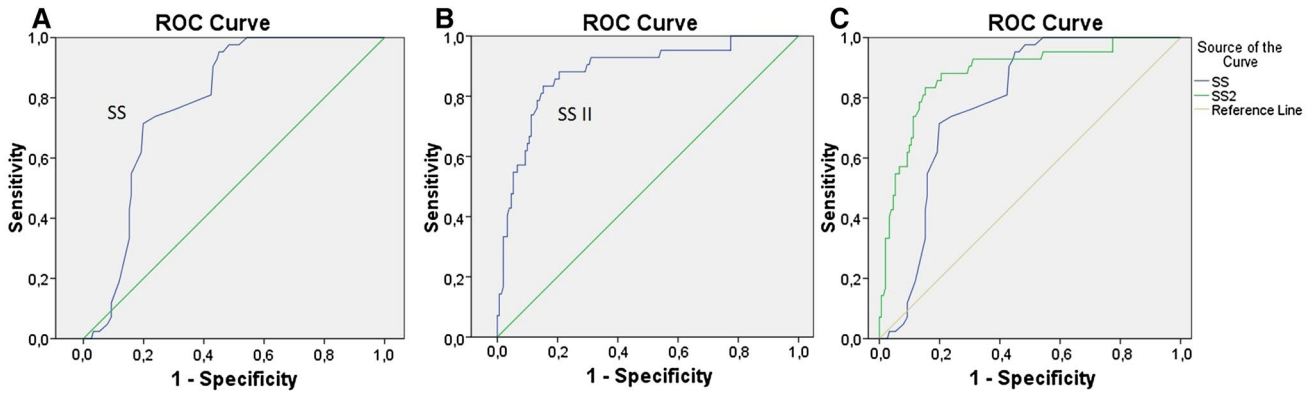

Fig. 1 ROC curve graphics to detect the best cutoff value of SS (**a**) and SS II (**b**) in the prediction of no-reflow phenomenon respectively. The comparison of the area under the curves (AUC) of ROC curve analyses (**c**) showed that AUC of SS II was larger than that of SS (0.881 vs. 0.785, p = 0.01) (*ROC* receiver-operating characteristic, *SS* syntax score, SS II syntax score II)

Patients with chronic renal disease have a hypercoagulable state and this condition is associated with increased risk of no-reflow in STEMI [18]. Sensoy et al. demonstrated that renal function disorders at admission is an independent predictor of no-reflow in STEMI [19]. Iwakura et al. showed the significant relation between admission blood glucose and no-reflow phenomenon [20].

In a study by Liang et al. it was reported that patients with COPD were more likely to have multi-vessel disease than patients without COPD [21]. Konecny et al. reported that patients with COPD undergoing PCI had a significantly higher incidence of cardiac mortality and myocardial infarction than patients without COPD [22]. In a differently designed study, Koseoglu et al. reported that COPD is a risk factor for complex coronary artery disease, and forced expiratory volume in 1 s (FEV1) is negatively correlated with SS [23].

SS II contain clinical variables apart from SS like GFR, age, COPD. These clinical variables could partially explain why SS II is associated with microvascular obstruction and intracoronary thrombus formation.

Recently undertaken study by Wang et al. divided patients into three groups based on SS II. The frequency of MBG 0/1 was significantly more common in SS II high tertile. They showed that high SS II score predicted no reflow with a 66% sensitivity and 54% specificity. Also the same study demonstrated that higher SS II was significantly and independently associated with MBG 0/1 [24].

There are many factors that are associated with development of no reflow phenomenon. To the best of our knowledge, there are no previous study that compared SS and SS II and development of no reflow. In the present study we found that SS II >32.3 independently predicts no reflow with a 88% sensitivity and 80% specificity in patients undergoing pPCI for STEMI.

The present study has some limitations. Firstly the sample size is quite small. Furthermore, it is a non-randomized retrospective study which has been conducted in a single center.

Conclusion

The major finding of the present study is that SS II predicts no-reflow phenomenon in patients undergoing pPCI for STEMI with an acceptable sensitivity and specificity values. Higher SS II is independently associated with no-reflow and may be more useful than SS to predict no-reflow in patients undergoing pPCI for STEMI. Future studies with larger sample size will be needed to confirm the results of the present study.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interests regarding the publication of this paper.

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