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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Does SYNTAX score predict in-hospital outcomes in patients with ST elevation myocardial infarction undergoing primary percutaneous coronary intervention?

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Abstract

Background: SYNTAX score (SxS) has been demonstrated to predict long-term outcomes in stable patients with coronary artery disease. But its prognostic value for patients with acute coronary syndrome remains unknown.

Aim: To evaluate whether SxS could predict in-hospital outcomes for patients admitted with ST elevation myocardial infarction (STEMI) who undergo primary percutaneous coronary intervention (pPCI).

Methods: The study included 538 patients with STEMI who underwent pPCI between January 2010 and December 2012. The patients were divided into two groups: low SxS (< 22) and high SxS (> 22). The SxS of all patients was calculated from an initial angiogram and TIMI flow grade of infarct related artery was calculated after pPCI. Left ventricular systolic functions of the patients were evaluated with an echocardiogram in the following week. The rates of reinfarction and mortality during hospitalisation were obtained from the medical records of our hospital.

Results: The high SxS group had more no-reflow (41% and 25.1%, $p < 0.001$, respectively), lower ejection fraction (38.2 ± 7.5% and 44.6 ± 8.8%, $p < 0.001$, respectively), and greater rates of re-infarction (9.5% and 7.3%, $p = 0.037$, respectively) and mortality (0.9% and 0.2%, $p = 0.021$, respectively) during hospitalisation compared to the low SxS group. On multivariate logistic regression analysis including clinical variables, SxS was an independent predictor of no-reflow (OR 1.081, 95% CI 1.032–1.133, $p = 0.001$).

Conclusions: SxS is a useful tool that can predict in-hospital outcomes of patients with STEMI undergoing pPCI.

Key words: SYNTAX score, coronary flow, systolic function, re-infarction, mortality

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INTRODUCTION

The SYNTAX (Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery) score (SxS) has been developed as a comprehensive angiographic scoring system for the prospective quantification of coronary lesions with respect to their number, location, and complexity [1–3]. In addition, the SxS is a good predictor of adverse cardiovascular events including cardiac death, myocardial infarction (MI) and target lesion revascularisation in multi-vessel diseases treated with percutaneous coronary intervention (PCI) or surgery [4–6].

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Therefore, SxS is a useful tool in choosing an intervention strategy. Although the SxS is proposed to predict adverse cardiac events in patients with stable coronary disease, its prognostic value remains unknown for patients presenting with acute coronary syndrome (ACS) and ST elevation MI (STEMI).

Most scoring systems that predict adverse events in unstable patients contain only clinical features such as ST segment changes, Killip class and serum creatin levels, while not taking into account the characteristics of coronary lesions [1–3]. In contrast, the SxS includes only angiographic features and the complexity of coronary lesions, while not containing any clinical features.

Here, we assessed the ability of SxS to predict in-hospital outcomes in patients undergoing primary PCI (pPCI). To address this question, we analysed patients with STEMI undergoing pPCI in our hospital, and we evaluated the relationship between SxS and coronary flow, systolic function, rate of reinfarction and in-hospital mortality.

METHODS

Our observational study included 538 patients admitted for STEMI who underwent pPCI from January 2010 to December 2012. All the patients included in our study were either patients with STEMI admitted with chest pain to our emergency department or referred from another hospital for pPCI. STEMI was diagnosed when the patients had symptoms of acute MI (AMI) lasting at least 30 min and accompanied by at least 1 mm (0.1 mV) ST segment elevation in two or more contiguous leads. The patients, all admitted in the first 12 h of onset of chest pain and ongoing chest pain or having Killip II, Killip III or cardiogenic shock, had urgent diagnostic angiography. None of the patients had been treated with fibrinolytic therapy. All patients received aspirin plus clopidogrel (loading dose 300 or 600 mg) before or during coronary intervention. Unfractionated heparin 100 U/kg was administered at the beginning of the procedure to keep the activated clotting time > 200 s. Femoral access was mostly preferred for pPCI. If the bilateral femoral artery was occluded, radial or brachial artery was used for access according to the physician's preference. If the infarct related artery (IRA) had much thrombus burden, a thrombectomy catheter was used after wiring IRA. After stent implantation on the culprit lesion, if there was still thrombus in IRA or no-reflow developed, tirofiban threapy was administered. Predilatation or postdilatation after stent implantation of the lesion was at the operator's discretion. Only bare metal stents were used for pPCI. After stent placement, clopidogrel was used for at least one year and aspirin was used indefinitely.

The only exclusion criterion was any patient with a previous coronary artery bypass grafting in whom the SxS could not be calculated. Baseline clinical and procedural characteristics were retrospectively recorded in a dedicated electronic database of the hospital. The SxS of patients were calculated by three interventional cardiologists from initial diagnostic angiography of AMI. All coronary lesions with a diameter stenosis ≥ 50% in vessels ≥ 1.5 mm were scored, using the SxS algorithm, which is available on the website www.syntaxscore.com. By scoring initial angiography, IRA with Thrombolysis in Myocardial Infarction (TIMI) flow of 0 or 1 were accepted as total occlusion with thrombus.

Primary end-points were the TIMI grade of coronary flow after pPCI, systolic function, re-infarction and in-hospital mortality.

TIMI flow grade 0 was defined as the absence of any antegrade flow, TIMI flow grade 1 as faint antegrade coronary flow with incomplete filling of the distal coronary bed, TIMI flow grade 2 as delayed or sluggish antegrade flow with complete filling of the distal territory, and TIMI flow grade 3 as normal flow which fills the distal coronary bed completely. The no-reflow phenomenon was defined by final TIMI flow grade < 3.

All the patients were assessed with an echocardiogram (Philips IE33 Matrix, Philips Healthcare, the Netherlands) within a week after AMI. Ejection fraction (EF) was received as as indicator of systolic function and calculated by the modified Simpson method.

Repeated MI in the acute post-PCI phase was defined as clinical signs of reinfarction with recurrent or persistent symptoms and ST-segment changes, or a second peak in the creatinine kinase-MB mass or troponin-T/troponin-I increase to ≥ 3 times the upper limit of normal but not related to an interventional procedure and new pathological Q waves in two or more contiguous electrocardiograph leads.

The in-hospital mortality rate for all patients was obtained from the medical records of the hospital.

Statistical analysis

All analyses were performed using SPSS V 16.0 for Windows (version 16.0, SPSS, Chicago, IL, USA). All data is presented as mean ± standard deviation unless otherwise stated. Continuous variables were checked for the normal distribution using the Kolmogorov-Smirnov statistics, and those that did not satisfy the criteria were log-transformed to obtain normal distribution. Comparison of parametric values between the two groups was performed by means of independent samples t test. Comparisons of nonparametric values between the two groups were performed by Mann-Whitney U test. Categorical variables were compared by the χ^2 test. In addition, we used multiple regression and logistic regression to observe the association between no-reflow groups and the impact of the principal variables (SxS) and of other variables that probably acted as confounders (diabetes mellitus, arterial hypertension, hyperlipidaemia, family history, gender, EF, creatinine, and Killip class). Receiver operating characteristic (ROC) analyses were used to detect the cutoff value of SxS in the prediction of no-reflow. A two-sided $p < 0.05$ for the final model was considered as significant.

RESULTS

Among 538 patients, 481 of them (90.3%) were admitted with STEMI due to total occlusion (TIMI flow grade 0 or 1) with thrombus. Stent implantation was performed in 510 (94.7%) patients. Thrombectomy catheter was used in 108 (20%) patients. In addition, tirofiban therapy was administered to 247 (45.9%) patients.

The mean SxS of the patients was 19.4 (range 7–42.5). The patients were classified into two groups, namely low SxS (SxS < 22) and high SxS (SxS > 22). While 338 (62.8%) patients had low SxS, 200 (37.1%) patients had high SxS. While 278 (51.6%) patients were admitted with anterior MI, 260 (48.3%) patients were admitted with non-anterior AMI. Post PCI no-reflow was detected in 167 (31.0%) patients.

Baseline clinical and angiographic characteristics of patients in relation to SxS are presented in Table 1. Hypertension, hyperlipidaemia, current smoking, family history, gender and mean age were similar between the two groups. However, the frequency of diabetes mellitus was higher in the high SxS group. The rates of anterior MI, having left anterior descending artery (LAD) occlusion as IRA, left main coronary artery disease, the number of diseased vessels, bifurcation lesions in IRA, and chronic total occlusions were also higher in the high SxS group. There was no significant difference in the number of stents implanted to IRA and stent diameter between the two groups. However, the total length of stent implanted to IRA was significantly longer in the high SxS group. Patients with high SxS presented with higher pulse rates, lower systolic and diastolic blood pressures, and higher Killip 2–3 classes and shock. There was no significant difference in terms of in-hospital medications between the two groups. There was no significant difference between the two groups in terms of stent placement, usage of thrombectomy catheter, and tirofiban therapy.

Considering the rates of post PCI no-reflow, there was a significant difference between the two groups (Table 2). Patients with high SxS had higher no-reflow rates. ROC analysis identified SxS > 19.75 as the best cut-off value predicting no-reflow (sensitivity of 66%, specificity of 54%, ROC area under curve: 0.650, 95% CI 0.59–0.70, $p < 0.001$; Fig. 1). In addition, the SxS was shown to be an independent predictor for no-reflow on multivariate logistic regression analysis (Table 3). Smoking, Killip class and EF were other independent predictors for no-reflow on multivariate logistic regression analysis (Table 3).

Considering left ventricular functions of the patients, while mean EF of patients in the low SxS group was 44.6%, mean EF of patients in the high SxS group was 38.2% ($p < 0.001$) (Table 2).

There was a significant difference in re-infarction rates during hospitalisation between the two groups. Forty-four patients were diagnosed as having re-infarction according to criteria stated in the methods section. Fourteen of them presented with stent thrombosis and two of them died due to cardiogenic shock. Patients with high SxS had a higher rate of re-infarction during hospitalisation ($p = 0.037$) (Table 2).

Twenty-five patients died during hospitalisation. Two of them had acute renal failure due to contrast induced nephropathy. Sixteen of them had cardiogenic shock due to AMI before or during pPCI. Six of them were caused by either cardiogenic shock or acute pulmonary oedema in the coronary intensive care unit after pPCI. One patient had cardiac tamponade owing to rupture of coronary artery during PCI. The rate of in-hospital mortality was higher in patients with high SxS (0.9% and 0.2%, $p = 0.021$, respectively; Table 2).

Fourteen patients had unsuccessful pPCI, four of whom underwent urgent surgery. While eight of these 14 patients were in the high SxS group, six were in the low SxS group ($p = 0.67$).

Evaluating the patients according to post-PCI TIMI flow grade, patients with no-reflow had a higher SxS ($p < 0.001$) (Table 4). The EF of the patients with post-PCI TMI 3 flow was more favourable than the EF of the patients with no-reflow (44.8% and 38.8%, $p < 0.001$, respectively; Table 4). Considering the rate of mortality during hospitalisation, patients with no-reflow had a greater mortality than patients with TIMI flow grade 3 (0.5% and 13.7%, $p < 0.001$, respectively; Table 4).

DISCUSSION

SxS is validated to predict the outcomes in stable patients with multivessel or left main disease undergoing elective PCI or bypass surgery. However, whether SxS has prognostic value in unstable patients is still controversial, because SxS considers only the coronary anatomy of patients, while all the scoring systems related to ACS consider the clinical characteristics of patients [7–10].

The present study is one of a few in the literature evaluating whether SxS could predict outcomes in patients admitted with STEMI. We examined the relationship between SxS and post-PCI TIMI flow grade, systolic function, rate of reinfarction and in-hospital mortality.

Myocardial no-reflow after pPCI is associated with increased clinical events and a poor survival rate [11, 12]. According to our results, high SxS is a risk factor in developing no-reflow in pPCI. SxS > 19.75 is an independent predictor of TIMI flow grade 0. In our study, the incidence of no-reflow was 31%, although the incidence of no-reflow during PCI has ranged from 12% to 25% in some studies that have used the criterion of TIMI flow grade ≤ 2 [13, 14]. In some studies, the incidence has been 29% using the TIMI myocardial perfusion grade [15], and 34% to 39% using myocardial contrast echocardiography [16, 17]. In our study, most patients were referred to our hospital from another hospital from remote regions for pPCI with inadequate antithrombotic and anticoagulant therapy. The transfer of these patients could cause a high rate of no-reflow due to prolonged door-to-balloon time and inadequate platelet inhibition.

Table 1. Baseline characteristics of patients according to SYNTAX score (SxS)

	Low SxS (< 22); n = 338	High SxS (> 22); n = 200	P
Age	56 ± 8	60 ± 10	0.03
Gender (male)	263 (77.8%)	148 (74%)	0.1
Diabetus mellitus	84 (24.8%)	119 (59.5%)	< 0.01
Hyperlipidaemia	107 (31.6%)	65 (32.5%)	0.91
Family history	63 (18.6%)	42 (21%)	0.7
Current smoker	128 (37.8%)	101 (50.5%)	0.079
Hypertension	138 (40.8%)	105 (52.5%)	0.2
Creatinine clearance [mL/min]	75 ± 2	72 ± 9	0.29
Renal failure (GFR < 30)	6 (1.7%)	8 (4.0%)	0.02
Previous PCI	32 (9.4%)	26 (13%)	0.72
Previous MI	21 (6.2%)	24 (12%)	0.39
Anterior infarction	123 (36.3%)	155 (77.5%)	< 0.01
Non-anterior infarction	215 (63.6%)	45 (22.5%)	< 0.01
IRA:			
Left anterior descending artery	123 (36.3%)	155 (77.5%)	< 0.01
Circumflex artery	56 (16.5%)	16 (8.0%)	< 0.01
Right artery	159 (47.0%)	29 (14.5%)	< 0.01
Left main disease	4 (1.2%)	12 (6.0%)	0.012
Number of diseased vessels	1.3 ± 0.5	1.8 ± 0.7	< 0.01
Total occlusion with thrombus	301 (89.0%)	185 (92.5%)	0.59
Thrombectomy	64 (18.9%)	44 (22%)	0.82
Tirofiban therapy	158 (46.7%)	89 (44.5%)	0.78
Chronic total occlusion	12 (3.5%)	28 (14%)	< 0.01
Bifurcation treatment in IRA	33 (9.7%)	41 (20.5%)	< 0.01
Complete revascularisation	299 (88.4%)	122 (61%)	< 0.01
Balloon predilatation	114 (33.7%)	68 (34%)	0.94
Balloon postdilatation	64 (18.9%)	38 (19%)	0.90
Stent implantation	322 (95.2%)	188 (94%)	0.1
Total length of stent in the culprit lesion [mm]	23 ± 6	32 ± 4	< 0.01
Number of stents implanted in the culprit lesion	1	1	0.6
Stent diameter [mm]	3.0 ± 0.5	3.2 ± 0.6	0.24
Time from onset of symptoms to hospital presentation [min]	122	117	0.35
Time from hospital presentation to angioplasty [min]	65	63	0.41
Pulse rate [bpm]	75 ± 22	84 ± 15	0.028
Systolic BP [mm Hg]	138 ± 20	118 ± 24	0.038
Diastolic BP [mm Hg]	78 ± 16	72 ± 18	0.25
Killip classes 2–3	31 (9.1%)	26 (13%)	< 0.01
Shock	27 (7.9%)	27 (13.5%)	< 0.01
Cardiac arrest	16 (4.7%)	11 (5.5%)	0.48
Atrioventricular block complete	18 (5.3%)	16 (8.0%)	0.06
Medication:			
Aspirin	330 (97.6%)	188 (94%)	0.69
Clopidogrel	322 (95.2%)	186 (93%)	0.71
ACE inhibitor	303 (89.6%)	178 (89%)	0.90
Beta-blocker	299 (88.4%)	174 (87%)	0.87
Statin	322 (95.2%)	191 (95.5%)	0.89

Data is expressed in numbers (percentages) and mean ± 1 standard deviation; ACE — angiotensin converting enzyme; BP — blood pressure; GFR — glomerular filtration rate; IRA — infarct related artery; MI — myocardial infarction; PCI — percutaneous coronary intervention

Table 2. The relationship between SYNTAX score (SxS) and no-reflow, ejection fraction, re-infraction, in-hospital mortality

	Low SxS (< 22); n = 338	High SxS (> 22); n = 200	P
No-reflow*	85 (25.1%)	82 (41.0%)	0.001
Ejection fraction	44.6 ± 8.8	38.2 ± 7.5	< 0.001
Re-infarction	25 (7.3%)	19 (9.5%)	0.037
In-hospital mortality	7 (2.0%)	18 (9.0%)	0.021

*TIMI flow grade 2, 1 or 0; Data is expressed in numbers (percentages) and mean ± 1 standard deviation.

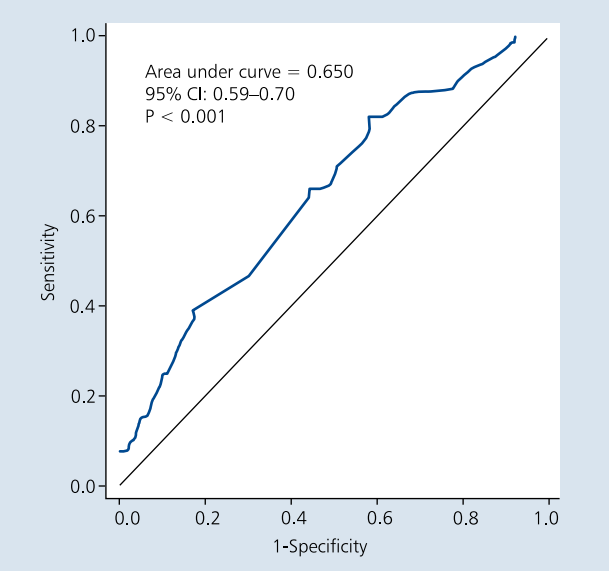

Figure 1. The receiver-operating characteristic curve analysis of SYNTAX score for predicting no-reflow; CI — confidence interval

Table 3. Multivariate logistic regression analysis showing relationship betwen clinical characteristics, ejection fraction and SYNTAX score and no-reflow in STEMI

Variable	Odds ratio (95% CI)	P
Age	0.993 (0.948–1.039)	0.759
Diabetus mellitus	0.601 (0.288–1.255)	0.176
Smoking	3.665 (1.831–7.337)	< 0.001
Hypertension	1.332 (0.681–2.606)	0.402
Hyperlipidaemia	1.092 (0.533–2.237)	0.810
Family history	0.390 (0.151–1.007)	0.52
Gender (male)	0.849 (0.379–1.903)	0.691
Creatinine	0.911 (0.442–1.965)	0.72
Killip class	1.122 (1.073–1.174)	0.001
Ejection fraction	0.826 (0.356–1.887)	0.03
SYNTAX score	1.081 (1.032–1.133)	0.001

CI — confidence interval; STEMI — ST elevation myocardial infarction

Patients with a high SxS had a more complex anatomy of coronary arteries including multivessel disease, diffuse disease, bifurcation lesions, chronic total occlusion and left main disease. These features could make the procedure of pPCI more difficult and complicated. Therefore, myocardial no-reflow could be expected in patients with complex coronary anatomy. Magro et al. [18] found similar results in their study. They examined 669 patients admitted with STEMI and found that post-PCI no-reflow rate of patients with high SxS (SxS > 16) was significantly high.

In addition, patients with no-reflow had a lower EF and a high in-hospital mortality rate. Besides, many studies have shown that developing no-reflow after pPCI is associated with a poor prognosis and poor long-term outcomes [11, 12]. Therefore, measures including pharmacologic agents such as glycoprotein IIb/IIIa inhibitors, adenosine, nitroprusside and nicorandil, as well as mechanical ones such as thrombus aspiration, to prevent no-reflow should be taken into consideration in high SxS patients with STEMI.

Systolic function of the left ventricle after AMI is one of the most important predictors of long-term outcomes [19, 20]. In our study, patients with high SxS had lower EF. Because more

Table 4. The relationship between TIMI flow grade and ejection fraction, re-infarction, in-hospital mortality

	TIMI flow grade 3 (n = 371)	No-reflow* (n = 167)	P
SYNTAX score	16.7 ± 8	22.1 ± 9	< 0.001
Ejection fraction	44.8 ± 8	38.8 ± 9	< 0.001
Re-infarction	28 (7.5%)	16 (9.5%)	0.54
In-hospital mortality	2 (0.5%)	23 (13.7%)	< 0.001

*TIMI flow grade 2, 1 or 0; Data is expressed in numbers (percentages) and mean ± 1 standard deviation.

no-reflow developed in patients with high SxS, inadequate reperfusion occurred in the myocardium despite patent IRA. In addition, patients with high SxS had more multivessel disease, chronic total occlusion and LAD occlusion, so the myocardium of these patients had a greater ischaemic area.

We evaluated the rate of re-infarction of patients during hospitalisation. When analysing the data of the patients, we found that only 14 presented with stent thrombosis. The rest of the patients were accepted as repeated infarction because of either a second peak in cardiac enzyme or ongoing or recurrent symptoms and ST segment changes after stent placement. After control coronary angiogram, it was determined that these symptoms developed due to occlusion of branch of IRA or distal embolisation of plaque and thrombus during stent placement. Acute stent thrombosis is related mostly to technical reasons. Interestingly, the rate of acute stent thrombosis was higher in patients with high SxS. As patients with high SxS have a complex coronary anatomy, these patients also had undergone complex pPCI procedure. Consequently, patients with high SxS tend to have early stent thrombosis. Prior studies showed that patients with high SxS had increased haematological parameters including mean platelet volume, red distribution width and neutrophil to lymphocyte ratio, all associated with high atherothrombotic events [21–23]. Therefore, patients with high SxS could have a haematologic predisposition to stent thrombosis. In addition, the increased rate of stent thrombosis was shown in the highest SxS tertile (SxS > 16) in a substudy of the STRATEGY trial [10].

Our study has shown that SxS predicts the in-hospital mortality rate of patients with STEMI. Presentation with STEMI and having SxS > 22 at the same time was associated with a high in-hospital mortality rate. In the high SxS group, the presence of complex coronary anatomy was abundant and this had been associated with more no-reflow, systolic dysfunction and higher rates of re-infarction. In addition, more diabetic patients were in the high SxS group. However, whether the SxS predicts long-term outcomes in patients with STEMI undergoing pPCI is controversial. Scherff et al. [24] evaluated whether the SxS could predict the mortality rate of elderly patients with STEMI in 30 days and one year. They found that the SxS predicted adverse clinical events in 30 days, but it did not predict clinical outcomes in one year. They concluded that with the complexity of coronary lesions, only periprocedural risk could be predicted in contrast to the long-term outcomes after discharge. In another study, Magro et al. [25] examined the data of 736 patients with STEMI who had undergone pPCI. They found that the SxS derived from angiography after pPCI predicted major adverse coronary events and long-term mortality in patients with STEMI [25]. Thus, studies including a greater number of patients and longer follow-ups are needed for more decisive results in order to evaluate the relationship between the SxS and long-term mortality in unstable patients.

Owing to the low number of failed pPCI in our study, we cannot make any comment about the relation between high SxS and failed pPCI. If a higher number of patients could be included in the study, we would achieve more informative results.

Limitations of the study

Our study was retrospective, and data on enzymatic or other imaging-derived infarct size quantification was not available in all patients. Also, myocardial blush grade and ST-segment resolution as markers of reperfusion could not be determined in all patients, and we used corrected TIMI frame count instead. All patients were followed only during the hospitalisation period.

CONCLUSIONS

The SxS can be used for risk classification in patients undergoing pPCI. In addition, the characteristics of the coronary anatomy carry prognostic value in patients undergoing pPCI.

Conflict of interest: *none declared*

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Czy skala SYNTAX umożliwia prognozowanie powikłań wewnątrzszpitalnych u chorych z zawałem serca z uniesieniem odcinka ST poddanych pierwotnej przezskórnej interwencji wieńcowej?

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Streszczenie

Wstęp: Wykazano, że punktacja w skali SYNTAX (SxS) jest czynnikiem predykcyjnym odległych powikłań u pacjentów ze stabilną chorobą wieńcową. Jednak jej wartość prognostyczna u chorych z ostrym zespołem wieńcowym nadal nie jest znana.

Cel: Niniejsze badanie przeprowadzono, aby ustalić, czy punktacja SxS umożliwia prognozowanie powikłań wewnątrzszpitalnych u pacjentów hospitalizowanych z powodu zawału serca z uniesieniem odcinka ST (STEMI) poddanych pierwotnej przezskórnej angioplastyce wieńcowej (pPCI).

Metody: Do badania włączono 538 chorych z STEMI, u których wykonano pPCI w okresie od stycznia 2010 do grudnia 2012 r. Pacjentów podzielono na dwie grupy: z niską punktacją SxS (< 22) i z wysoką punktacją SxS (> 22). Punktację SxS dla wszystkich badanych obliczono na podstawie wykonanych początkowo angiogramów, a po przeprowadzeniu pPCI określono stopień przepływu w skali TIMI w tętnicy odpowiedzialnej za zawał. Czynność skurczową lewej komory oceniano w badaniu echokardiograficznym w następnym tygodniu. Odsetek pacjentów, u których nastąpił dorzut zawału lub zgon w trakcie hospitalizacji, uzyskano z dokumentacji medycznej szpitala.

Wyniki: W grupie z wysoką punktacją SxS stwierdzono częstsze występowanie zjawiska *no-reflow* (odpowiednio 41% i 25,1%; p < 0,001), mniejszą wartość frakcji wyrzutowej (odpowiednio 38,2 ± 7,5% i 44,6 ± 8,8%; p < 0,001) oraz większy odsetek dorzutów zawału (odpowiednio 9,5% i 7,3%; p = 0,037) i zgonów (odpowiednio 0,9% i 0,2%; p = 0,021) w trakcie hospitalizacji w porównaniu z grupą charakteryzującą się niską punktacją SxS. W wieloczynnikowej analizie regresji logistycznej uwzględniającej zmienne kliniczne punktacja SxS była niezależnym czynnikiem predykcyjnym zjawiska *no-reflow* (OR 1,081; 95% CI 1,032–1,133; p = 0,001).

Wnioski: Skala SYNTAX jest przydatnym narzędziem umożliwiającym prognozowanie powikłań wewnątrzszpitalnych u chorych z STEMI poddanych pPCI.

Słowa kluczowe: skala SYNTAX, przepływ wieńcowy, czynność skurczowa, dorzut zawału serca, śmiertelność

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