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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The relationship between Gensini score and ST-segment resolution in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Abstract

Background: Clinical outcomes of patients with myocardial infarction are primarily determined by the successful restoration of myocardial reperfusion and the severity of coronary atherosclerosis.

Aim: To investigate the predictive value of Gensini score on ST-segment resolution (STR) in patients undergoing primary percutaneous coronary intervention (pPCI) for acute ST-elevation myocardial infarction (STEMI).

Methods: The present study prospectively included 114 consecutive patients (mean age 54 ± 10 years, 15 women) with STEMI who underwent successful pPCI. Sum of ST-segment elevation amount in millimetres was obtained before angioplasty and 60 min after pPCI. ∑STR < 50% was accepted as a ECG sign of no-reflow phenomenon. Thrombus grading was calculated according to the results of coronary angiography, and Gensini score (GS-pPCI) was calculated after pPCI without incorporating culprit lesion. Patients were divided into two groups according to STR: those with STR(–), and those with STR(+). Patients were also analysed according to the infarct-related artery.

Results: GS-pPCI was significantly higher in patients with STR(–) (10.1 ± 11.8 vs. 22 ± 18.6, p = 0.005). GS-pPCI was inversely correlated with STR (r = –0.287, p = 0.002). In subgroup analysis, patients in the STR(–) group with culprit lesion in left anterior descending artery and left circumflex artery also showed higher GS-pPCI (10.9 ± 13.5 vs. 23.5 ± 21.3, p = 0.03 and 9.6 ± 8.7 vs. 24.1 ± 21, p = 0.04, respectively). High thrombus burden was also observed more frequently in patients with STR(–) (68% vs. 43%, p = 0.03). Multivariate logistic regression analysis demonstrated that GS-pPCI and high thrombus burden independently predicted inadequate STR (OR 1.07, 95% CI 1.03–1.12, p = 0.001 and OR 3.28, 95% CI 1.11–9.72, p = 0.03, respectively).

Conclusions: GS-pPCI and high thrombus burden play an important role in predicting inadequate STR in patients with STEMI treated with pPCI.

Key words: acute myocardial infarction, Gensini score, ST-segment resolution, thrombus burden

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INTRODUCTION

Despite enormous developments in the diagnosis and management of coronary atherosclerosis, ST-elevation myocardial infarction (STEMI) still remains a significant health concern worldwide. In the current treatment modalities, primary percutaneous coronary interventions (pPCI) are of paramount importance for the treatment of STEMI. However, the beneficial effect of the successful restoration of epicardial coronary flow on myocardial salvage may be overshadowed by inadequate myocardial perfusion, a condition referred to as 'no-reflow phenomenon' [1]. In about one third of patients undergoing pPCI for STEMI, adequate myocardial perfusion was not observed despite Thrombolysis In Myocardial Infarction (TIMI) 3 flow [2].

Although several factors are identified in the pathogenesis of the no-reflow phenomenon, their contribution in the development of the no-reflow phenomenon and strategies to overcome it in the clinical setting still remain undetermined. Reperfusion injury, microembolisation of atherothrombotic debris, and microvessel spasm, all causing microvascular damage, are some of the postulated mechanisms [3, 4].

It has been shown that myocardial reperfusion is critical for a positive clinical outcome in patients with STEMI [5]. Therefore, early recognition of no-reflow phenomenon and improving myocardial perfusion are now the focus of reperfusion therapy in acute STEMI besides restoring early epicardial patency. Unfortunately, accurate prediction of reperfusion cannot be made solely by clinical assessment. Various techniques have been used to evaluate the level of myocardial reperfusion. Inadequate ST-segment resolution (STR) is accepted as an electrocardiography (ECG) sign of no-reflow phenomenon. STR that reflects myocardial flow rather than epicardial flow is the most frequently used practical method due to its ready availability and simplicity. Furthermore, resolution of ST-segment elevation after reperfusion therapy has also been found to be correlated with positive clinical outcomes [6].

Based on well-established coronary angiography findings, Gensini score as an objective method can assess the severity of coronary artery disease (CAD) and also provide some prognostic information [7]. Compared to using the number of diseased coronary vessels to judge the severity of CAD, Gensini score reflects the severity of coronary stenoses more effectively, because it is based on more detailed information. It has been shown to be clearly associated with cardiogenic death and short- and long-term major adverse cardiac events [8]. The severity of CAD may also predict the extent of myocardial salvage to be gained after pPCI. However, to the best of our knowledge, an association between the severity of CAD using Gensini score and STR in patients with STEMI has not been revealed until now.

The aim of this study was to investigate the predictive value of the severity of CAD using Gensini score on the STR that reflects myocardial perfusion in patients with acute STEMI undergoing pPCI.

METHODS

Study population

All consecutive patients admitted to the coronary care unit with a diagnosis of first acute STEMI who underwent pPCI were considered eligible and enrolled if they fulfilled the following criteria: (1) symptoms lasting for ≥ 30 min, consistent with acute STEMI within 12 h of symptom onset; (2) new ST elevation at the J point in at least two contiguous leads with the cut-points: ≥ 0.2 mV; (3) > 3-fold increase in serum creatine kinase (CK) levels; (4) angiographic evidence of total occlusion, i.e. TIMI-0 or -1 grades; (5) Patients with successful angioplasty (stable TIMI 3 flow and < 30% residual stenosis at the occlusion site); and (6) No additional > 50% stenosis on the infarct-related coronary artery (IRA) distal to culprit lesion.

Six patients were excluded because of left ventricular hypertrophy criteria on baseline ECG, defined as Sokolow-Lyon voltages > 35 mV (n = 3); and missing (n = 1) or not interpretable ECG because of the development of a left bundle-branch block (n = 1) and permanent pacemaker (n = 1).

The final study population therefore consisted of 114 patients. The study protocol was approved by the local ethical committee, and informed consent was obtained from each patient.

Study protocol

A 12-lead ECG was recorded in each patient just after hospital admission. All patients received aspirin (300 mg) and clopidogrel (300 mg) prior to patient transfer to the cath lab. This study was perfomed before prasugrel and ticagrelor were clinically in use. Emergency coronary angiography was performed using the percutaneous femoral approach. IRA was graded according to TIMI classification, and collateral vessels were graded according to the classification of Rentrop. Good collateral flow was defined as Rentrop collateral flow of 2–3. Heparin (100 U/kg) was administered in all patients after the coronary anatomy had been defined. Occlusion of the IRA was crossed by using a 0.014 in guide wire and if necessary balloon angioplasty was performed. Routine bare-metal stenting was attempted directly or following balloon angioplasty. Primary coronary angioplasty was defined as successful if restored blood flow was TIMI 3 in the IRA and residual stenosis following stent deployment was < 30%. Additional stents were implanted as required. A repeat 12-lead ECG was obtained 60 min after successful primary coronary angioplasty. All the patients were treated according to the recommendations of the then ACC/AHA guidelines for the management of patients with STEMI [9]. The use of glycoprotein IIb/IIIa receptor blocker was left to the decision of the operator.

Electrocardiographic analysis

Measurements were obtained from first ECG, recorded on admission and second ECG recorded 60 min after successful primary angioplasty. ECGs were analysed in a blinded fashion

by two cardiologists. The sum of ST-segment elevation amount in [mm] was obtained from the first ECG and the second ECG. ST-segment elevation in [mm] was measured 20 ms after the J point. The sum of ST-segment elevations were measured in leads I, aVL, and V_1 through V_6 for anterior infarctions and in leads II, III, aVF, V_5, and V_6 for inferior infarctions. The difference between two measurements was accepted as resolution of the sum of ST-segment elevation and expressed as ∑STR. Patients were separated into two groups according to the classification of Schroder et al. [10]. Patients with ∑STR ≥ 50% were accepted as the STR(+) group, and patients with ∑STR < 50% were accepted as the STR(–) group. Patients were also subgrouped according to the IRA.

Echocardiographic analysis

Transthoracic echocardiography was performed using a ESAOTE 2.5 MHz probe (ESAOTE, Genoa, Italy) within 72 h of the hospitalisation. The left ventricular ejection fraction (LVEF) was determined using modified Simpson's method.

Coronary angiographic findings

Multiple views were obtained in all patients, with visualisation of the left anterior descending (LAD) and left circumflex coronary (LCX) arteries in at least four views, and the right coronary artery (RCA) in at least two views. Based on the results of coronary angiography, all scores was calculated in a blinded fashion by two cardiologists.

Gensini score equals the sum of all segment scores (each segment score equals a segment weighting factor multiplied by a severity score), as previously described [7]. Segment weighting factors range from 0.5 to 5.0. Severity scores reflecting the specific percentage luminal diameter reduction of the coronary artery segment are 32 for 100%, 16 for 99%, 8 for 90%, 4 for 75%, 2 for 50%, and 1 for 25%. Thus, segments supplying a larger area of myocardium are more heavily weighted and multiple severe proximal lesions gain the highest score. We calculated Gensini score after pPCI (GS-pPCI) without incorporating culprit lesion.

Each coronary lesion that produced a luminal narrowing ≥ 50% in vessels ≥ 1.5 mm was separately scored using the SYNTAX score calculator, and this was then added up to provide an overall SYNTAX score. The online latest updated version (2.11) was used for the calculation of the SYNTAX scores (http://www.SYNTAXscore.com) [11].

Angiographic coronary thrombus burden was scored based on the TIMI thrombus grading scale ranging from grade 0 (no thrombus), to grade 5 (very large thrombus content that completely occludes vessel flow) [12]. TIMI thrombus grading was based on the initial diagnostic coronary angiogram. After restoring antegrade flow through guidewiring or small balloon dilatation in patients with TIMI thrombus grade 5, a coronary angiogram enabled restratification of an underlying residual thrombus defined by final TIMI thrombus grade [12, 13]. We used a simple bi-level categorisation [14] whereby only two thrombus grades were used: a low thrombus burden is assigned to final TIMI grades 1–3, and a high thrombus burden corresponds to final TIMI grades 4–5.

Multivessel disease was defined as the presence of a stenosis greater than 50% in three major epicardial coronary arteries based on coronary angiography.

Analysis of patient data

A clinical history of risk factors such as age, gender, diabetes mellitus, hypertension, hyperlipidaemia and smoking were determined for each study patient. Cardiac symptoms lasting < 30 min were defined as a sign of angina pectoris, and angina occurring within 48 h before the onset of infarction was defined as preinfarction angina. After treatments for acute STEMI, symptom-onset-to-balloon time and door-to-balloon time have also been recorded. Venous blood samples were obtained during the first 24 h for routine biochemical analyses.

Statistical analysis

Quantitative variables were expressed as mean ± standard deviation, and qualitative variables were expressed as percentages. Data was tested for normal distribution using the Kolmogorov-Smirnov test. A comparison of parametric values between two groups was made using a two-tailed Student t-test, and for nonparametric values a Mann-Whitney U test was used. Categorical variables were compared using the χ^2 test or Fisher's test. Spearman rho and Pearson tests were used for correlation analysis. Binary logistic regression analysis was used to evaluate the independent association between inadequate STR and clinical parameters. A p value of < 0.05 was considered statistically significant. All statistical analyses were carried out using SPSS version 18.0 (SPSS, Chicago, IL, USA).

RESULTS

The study population consisted of 114 patients (mean age 54 ± 10 years, range 35–83, 99 men and 15 women) with STEMI who underwent pPCI. Patients with STEMI were divided into two groups according to STR: those with STR(–) (n = 25 [21.9%], and those with STR(+) (n = 89 [78.1%]). The baseline characteristics are presented in Table 1.

Although metabolic syndrome was not significantly higher, body mass index (BMI) (p = 0.03) and waist circumference (p = 0.03) were significantly higher in the STR(–) group. BMI and waist circumference were also significantly correlated with STR(–) group (r = 0.218, p = 0.02 and r_s = 0.202, p = 0.03, respectively).

In patients with an inadequate STR peak, CK-MB levels were higher (p = 0.04) and LVEF was significantly reduced (p = 0.001). There was also a significant reverse correlation between LVEF and inadequate STR (r_s = –0.408, p = 0.001).

The baseline characteristics of patients subgrouped according to the IRA are set out in Table 2.

Table 1. Clinical characteristics of the patients grouped according to ST-segment resolution

	Electrocardiography findings		P
	STR(+)	STR(–)	
Age [years]	53.3 ± 10.3	55.8 ± 13.7	0.32
Sex (male/female)	79/10	20/5	0.25
Symptom-onset-to-balloon time [min]	194.4 ± 116.2	185 ± 96.4	0.71
Door-to-balloon time [min]	28.8 ± 8.1	30.8 ± 7.6	0.28
Pre-infarction angina	37%	24%	0.22
Admission Killip class ≥ 2	5%	4%	0.99
Body mass index [kg/m^2]	27.1 ± 3.8	28.9 ± 2.7	**0.03**
Diabetes mellitus	19%	24%	0.59
Smoking	54%	60%	0.59
Total cholesterol [mg/dL]	208.5 ± 32.6	213.7 ± 28.1	0.49
LDL-C [mg/dL]	124.3 ± 23.8	128.3 ± 20.5	0.49
Metabolic syndrome components:			
Fasting glucose [mg/dL]	119.2 ± 38.5	132.9 ± 37.1	0.12
HDL-C [mg/dL]	41.6 ± 7.9	40.1 ± 7.1	0.44
Triglyceride [mg/dL]	199.8 ± 156.8	215.3 ± 126.8	0.66
Waist circumference [cm]	96.6 ± 9.1	101.4 ± 10.2	**0.03**
Hypertension	33%	44%	0.29
Metabolic syndrome	38%	52%	0.22
Anterior wall infarction	34%	48%	0.19
Peak CK-MB [IU/L]	142.4 ± 88.1	203.1 ± 129.4	**0.04**
LVEF [%]	50.1 ± 7.9	41.8 ± 7.1	**0.001**
Medication after infarction:			
ACE-I	92%	100%	0.15
Beta-blocker	92%	96%	0.50
GP IIb/IIIa RB	64%	60%	0.71
Statin	98%	100%	0.45
Nitrate	56%	64%	0.48
Stent implantation	85%	96%	0.15
Predilatation	68%	83%	0.16
First stent diameter [mm]	3.2 ± 0.4	3.2 ± 0.5	0.79
First stent length [mm]	19.3 ± 4.4	19.9 ± 4.9	0.51
Further stent implantation	10%	4%	0.34
Culprit lesion:			
LAD proximal	16%	28%	0.16
LCX proximal	1%	4%	0.39
RCA proximal	24%	12%	0.21
Good collateral circulation	44%	32%	0.29
Triple-vessel disease	15%	28%	0.12
High thrombus burden	43%	68%	**0.03**
SYNTAX score	15.8 ± 8.3	18.8 ± 9.3	0.12
GS-pPCI	10.1 ± 11.8	22 ± 18.6	**0.005**
ST-segment resolution [%]	74.2 ± 13.2	19.6 ± 14.8	**0.001**

ACE-I — angiotensin converting enzyme inhibitor; CK-MB — creatine kinase-MB fraction; GP IIb/IIIa RB — glycoprotein IIb/IIIa receptor blocker; GS-pPCI — Gensini score after primary percutaneous coronary intervention; HDL-C — high density lipoprotein-cholesterol; LAD — left anterior descending artery; LCX — left circumflex artery; LDL-C — low density lipoprotein-cholesterol; LVEF — left ventricular ejection fraction; RCA — right coronary artery

Table 2. Clinical characteristics of patients grouped by infarct-related artery

Infarct-related artery	LAD		P	LCX		P	RCA		P
	STR(+) (n = 30)	STR(–) (n = 12)		STR(+) (n = 14)	STR(–) (n = 5)		STR(+) (n = 45)	STR(–) (n = 8)	
Age [years]	50.7 ± 8.9	53.6 ± 14.3	0.44	57.4 ± 13.8	60.8 ± 14.7	0.64	53.8 ± 9.7	56.1 ± 13.4	0.56
Sex (male/female)	27/3	9/3	0.33	13/1	5/0	0.99	39/6	6/2	0.59
Symptom-onset-to-balloon time [min]	222.5 ± 148.4	195.8 ± 89.1	0.57	193.9 ± 109.5	176 ± 99.9		175.8 ± 89.8	174.4 ± 115.4	0.97
Door-to-balloon time [min]	26.5 ± 7.7	29.6 ± 8.9	0.26	31.5 ± 6.4	33.4 ± 6.8	0.58	29.6±8.6	31 ± 6.2	0.66
Pre-infarction angina	30%	33%	0.83	50%	20%	0.24	38%	13%	0.16
Admission Killip class ≥ 2	13%	8%	0.99	0%	0%	–	0%	0%	–
Body mass index [kg/m^2]	27.1 ± 3.2	28.8 ± 2	0.11	27.5 ± 4.3	27.4 ± 3.3	0.97	26.9 ± 4.1	30.2 ± 3.1	**0.04**
Diabetes	13%	27%	0.36	21%	20%	0.99	22%	25%	0.99
Smoking	63%	67%	0.84	57%	60%	0.91	47%	50%	0.86
Total cholesterol [mg/dL]	209.2 ± 33.5	211.8 ± 28.3	0.83	207.2 ± 23.6	216 ± 29.3	0.56	208.4 ± 35.3	215.6 ± 31.3	0.62
LDL-C [mg/dL]	123.6 ± 26.2	128.3 ± 21.4	0.63	126.7 ± 28.7	131 ± 16.8	0.79	123.9 ± 20.8	126.9 ± 23.9	0.74
Metabolic syndrome components:									
Fasting glucose [mg/dL]	122.7 ± 45.9	141 ± 40.5	0.24	110.9 ± 19.1	124.4 ± 34.1	0.28	119.5 ± 38	126.3 ± 35.6	0.64
HDL-C [mg/dL]	41.2 ± 8.3	40.6 ± 5.8	0.83	45.2 ± 4.8	41.3 ± 10.2	0.3	40.6 ± 8.2	38.7 ± 8.2	0.59
Triglyceride [mg/dL]	208.9 ± 161.4	223.5 ± 152.3	0.79	203.1 ± 159.9	205.1 ± 122.3	0.98	192.6 ± 156.1	207.1 ± 93.1	0.81
Waist circumference [cm]	96.9 ± 8.2	99.5 ± 9.9	0.38	97.3 ± 13.1	101 ± 10.4	0.58	96.3 ± 8.4	104.4 ± 11.3	**0.02**
Hypertension	20%	42%	0.15	21%	40%	0.57	44%	50%	0.77
Metabolic syndrome	40%	50%	0.55	36%	40%	0.99	38%	63%	0.19
Peak CK-MB [IU/L]	154.3 ± 87.8	221.9 ± 112.3	0.05	148.5 ± 109.7	193.8 ± 225.9	0.57	132.8 ± 82.3	180 ± 70.7	0.16
LVEF [%]	43.9 ± 7.6	36.3 ± 4.1	**0.002**	51 ± 5.4	44.4 ± 5.4	**0.03**	53.9 ± 6.1	48.6 ± 3.9	0.07
Medication after infarction:									
ACE-I	98%	100%	0.99	93%	100%	0.99	89%	100%	0.99
Beta-blocker	100%	100%		100%	100%		84%	88%	0.99
GP IIb/IIIa RB	70%	67%	0.83	43%	60%	0.51	67%	50%	0.37
Statin	100%	100%		100%	100%		96%	100%	0.99
Nitrate	70%	75%	0.75	64%	40%	0.35	44%	63%	0.35
Stent implantation	83%	100%	0.29	89%	100%	0.99	79%	80%	0.99
Predilatation	85%	100%	0.29	50%	67%	0.99	61%	63%	0.94
First stent diameter [mm]	3.1 ± 0.3	3.1 ± 0.3	0.94	3.1 ± 0.4	3.4 ± 0.9	0.42	3.3 ± 0.4	3.1 ± 0.5	0.46

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Table 2. (cont.) Clinical characteristics of patients grouped by infarct-related artery

Infarct-related artery	LAD		P	LCX		P	RCA		P
	STR(+) (n = 30)	STR(–) (n = 12)		STR(+) (n = 14)	STR(–) (n = 5)		STR(+) (n = 45)	STR(–) (n = 8)	
First stent length [mm]	19.4 ± 4.7	20.4 ± 5.6	0.57	17.5 ± 3.8	18.7 ± 2.3	0.63	19.6 ± 4.3	19.8 ± 5	0.91
Further stent implantation	10%	0%	0.55	0%	0%		13%	13%	0.99
Proximal culprit lesion	47%	58%	0.49	7%	20%	0.47	44%	38%	0.72
Good collateral	53%	33%	0.24	57%	40%	0.51	33%	25%	0.64
Triple-vessel disease	7%	25%	0.13	7%	20%	0.47	22%	38%	0.39
High thrombus burden	37%	75%	**0.03**	29%	60%	0.31	51%	63%	0.55
SYNTAX score	20.2 ± 8.9	24.1 ± 6.4	0.17	12.3 ± 6.4	12.6 ± 5.1	0.92	14 ± 7.3	14.9 ± 11.3	0.84
GS-pPCI	10.9 ± 13.5	23.5 ± 21.3	**0.03**	9.6 ± 8.7	24.1 ± 21	**0.04**	9.8 ± 11.6	18.6 ± 14.1	0.06
ST-segment resolution [%]	68.1 ± 10.8	20.8 ± 15.7	**0.001**	76.1 ± 13.4	19.4 ± 16.6	**0.001**	77.7 ± 13.3	17.9 ± 14.1	**0.001**

ACE-I — angiotensin converting enzyme inhibitor; CK-MB — creatine kinase-MB fraction; GP IIb/IIIa RB — glycoprotein IIb/IIIa receptor blocker; GS-pPCI — Gensini score after primary percutaneous coronary intervention; HDL-C — high density lipoprotein-cholesterol; LDL-C — low density lipoprotein-cholesterol; LVEF — left ventricular ejection fraction

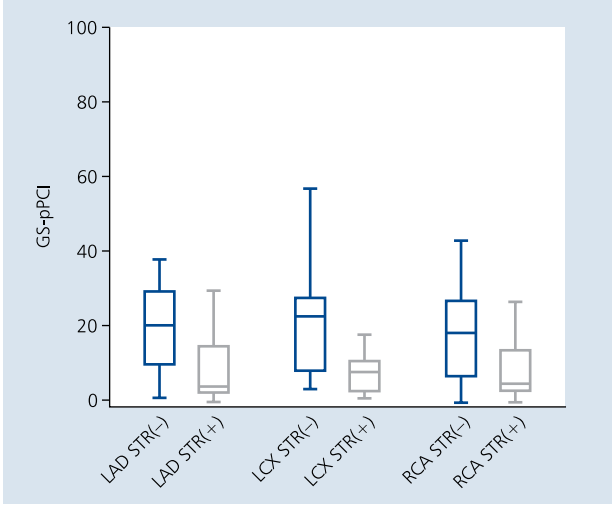

Figure 1. Comparison of Gensini score after primary percutaneous coronary intervention (GS-pPCI) according to infarct-related artery. ST-segment resolution (STR)(–) patients with culprit lesion in left anterior descending artery (LAD) and left circumflex artery (LCX) showed higher GS-pPCI compared to STR(+) patients; RCA — right coronary artery

Waist circumference and BMI were also higher in STR(–) patients with culprit lesion in RCA ($p = 0.04$ and $p = 0.02$, respectively). LVEF was significantly reduced in STR(–) patients with culprit lesion in LAD and LCX ($p = 0.002$ and $p = 0.03$, respectively). In subgroup analysis, although peak CK-MB levels were higher in all STR(–) patients, it was found to be insignificant.

Coronary angiographic findings and STR

Patients with inadequate STR demonstrated higher GS-pPCI (10.1 ± 11.8 vs. 22 ± 18.6, $p = 0.005$) and a high coronary thrombus burden more frequently (43% vs. 68%, $p = 0.03$). In subgroup analysis, patients in the STR(–) group with culprit lesion in LAD and LCX also showed higher GS-pPCI (10.9 ± 13.5 vs. 23.5 ± 21.3, $p = 0.03$ and 9.6 ± 8.7 vs. 24.1 ± 21, $p = 0.04$, respectively) (Fig. 1). Figure 2 illustrates that low GS-pPCI could determine adequate STR ($r = –0.287$, $p = 0.002$). Patients in the STR(–) group with culprit lesion in LAD also revealed a high thrombus burden more frequently (37 vs. 75, $p = 0.03$). Although SYNTAX score and GS-pPCI were positively correlated ($r = 0.425$, $p = 0.001$), SYNTAX score did not differ between patients with STR(+) and STR(–).

There was no significant difference between the two groups with respect to anterior myocardial infarction, good collateral circulation, triple-vessel disease, proximal culprit lesion, predilatation, stent implantation, stent size, stent diameter or further stent implantation ($p \geq 0.05$).

The effects of different variables on inadequate STR were calculated in univariate analysis for each. The variables for which the unadjusted p value was < 0.10 in logistic

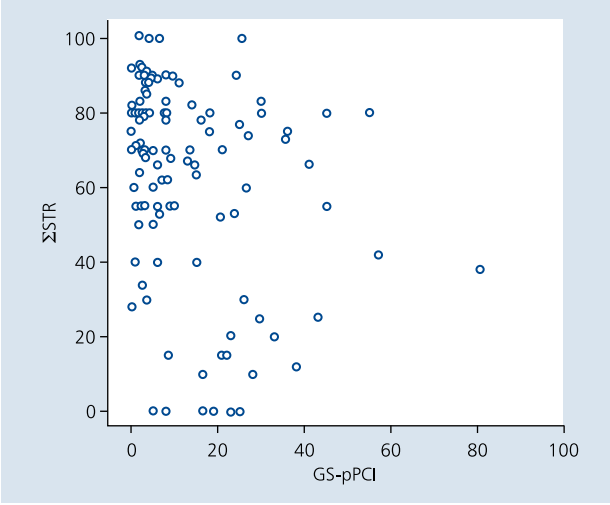

Figure 2. Correlation between ratio of ∑ST-segment resolution (STR) and Gensini score after primary percutaneous coronary intervention (GS-pPCI). There is an inverse correlation between ratio of ∑STR and GS-pPCI

regression analysis were identified as potential risk markers and included in the full model. Metabolic syndrome, BMI, anterior wall infarction, presence of good collateral channels, high thrombus burden and Gensini score were analysed with the multivariate logistic regression model. Multivariate logistic regression analysis demonstrated that the GS-pPCI and a high thrombus burden were associated with inadequate STR (OR 1.07, 95% CI 1.03–1.12, p = 0.001 and OR 3.28, 95% CI 1.11–9.72, p = 0.03, respectively) (Table 3).

Observer variabilities

Intra- and interobserver variability for the measurements of ∑STR and GS-pPCI were 3.5% and 4.5%, and 3% and 3.9%, respectively.

DISCUSSION

In the present study, patients with STR(–) had higher GS-pPCI and a high thrombus burden more frequently than those with STR(+). Also, patients with inadeqaute STR demonstrated a higher peak CK-MB level along with lower LVEF. Multivariate analysis revealed that GS-pPCI and a high thrombus burden were independent predictive factors for inadequate STR.

The investigation of no-reflow phenomenon after pPCI in patients with STEMI has therapeutic implications and prognostic importance. Previous studies have shown that those patients having STEMI with inadequate ST-segment resolution are more likely to have reduced myocardial salvage and a poor follow-up outcome [15, 16].

Reduced myocardial salvage results in larger myocardial necrosis, and this is the principal mechanism by which no reflow impairs left ventricular function. Our results also showed reduced LVEF in patients with inadequate STR.

Table 3. Univariate and multivariable analysis of predictors of inadequate ST-segment resolution

	Odds ratio (95% CI)	P
Univariate analysis		
Age	1.64 (0.89–2.55)	0.19
Male gender	0.51 (0.16–1.65)	0.26
Symptom-onset-to-balloon time	1.21 (0.95–1.54)	0.12
Waist circumference	1.05 (0.92–1.11)	0.11
Body mass index	1.13 (1.01–1.27)	0.04
Fasting glucose	2.72 (0.67–1.77)	0.46
LDL-cholesterol	1.01 (0.99–1.03)	0.57
Smoking	1.28 (0.52–3.15)	0.59
Diabetes	1.34 (0.46–3.86)	0.59
Hypertension	1.63 (0.66–4.02)	0.29
Metabolic syndrome	4.66 (1.04–18.78)	0.04
Pre-infarction angina	0.49 (0.12–3.47)	0.21
Anterior wall infarction	6.89 (0.96–29.22)	0.07
Triple-vessel disease	2.27 (0.79–6.52)	0.13
LAD proximal	2.08 (0.73–5.91)	0.17
LCX proximal	3.67 (0.22–60.8)	0.37
RCA proximal	0.44 (0.12–1.62)	0.22
Good collateral	1.81 (0.98–3.1)	0.05
GP IIb/IIIa receptor blocker	0.86 (0.44–1.79)	0.24
Predilatation	1.63 (0.77–4.93)	0.22
Stent implantation	2.99 (0.52–6.67)	0.34
High thrombus burden	2.85 (1.32–7.30)	0.03
SYNTAX score	1.04 (0.94–1.09)	0.14
GS-pPCI	1.05 (1.02–1.09)	0.001
Multivariate analysis		
High thrombus burden	3.28 (1.11–9.72)	0.03
GS-pPCI	1.07 (1.03–1.12)	0.001

CI — confidence interval; GS-pPCI — Gensini score after primary percutaneous coronary intervention; GP — glycoprotein; LAD — left anterior descending artery; LCX — left circumflex artery; LDL — low density lipoprotein; RCA — right coronary artery

The mechanisms underlying no-reflow phenomenon are complex, multifactorial and incompletely elucidated. Initial ischaemic injury and distal microembolisation of plaque and/or thrombus from the lesion site are the proven mechanisms that play pivotal roles in no-reflow phenomenon [3, 17]. Our findings also showed a high thrombus burden in patients with inadequate STR.

The initial ischaemic injury is primarily determined by the severity of atherosclerosis and location of the culprit lesion. The complexity of the culprit lesion in pPCI has also been associated with no-reflow [18]. In a previous study, more atheromatous plaque debris was demonstrated in the IRA lumen after pPCI in patients with no-reflow [19]. In addition,

it was shown that atheromatous plaque and thrombus burden were associated with pre-interventional angiographic morphology [20]. Gensini score as an index of severity of CAD has been shown to be correlated significantly with average plaque burden and plaque area determined by intravavascular ultrasound [21]. So, it may be assumed that some of the no-reflow mechanisms are primarily determined by the severity of CAD and this could be predicted by the Gensini score, as our findings demonstrated. Although Gensini score was found not to be associated with angiographic no-reflow in some of the previous studies [22], our results showed a significant association between GS-pPCI and inadequate STR reflecting myocardial no-reflow.

Peak serum levels of myocardial enzymes are closely related to the severity of CAD in patients with acute coronary syndromes. Peak CK-MB levels in patients with STR(–) were also found to be higher in our study. It is obvious that higher myocardial enzyme peak serum levels were a consequence of the no-reflow.

In previous studies, it was indicated that SYNTAX score was associated with no-reflow [23, 24]. However, our results showed that it was not significantly high in patients with STR(–) despite SYNTAX score and GS-pPCI being positively correlated. Although both scores were grading the severity of CAD, there are many differences between them. In SYNTAX score, the percent diameter stenosis is not considered in the algorithm, and a distinction has been made only between occlusive (100% diameter stenosis) and non occlusive (50–99% diameter stenosis) disease. Also, a lesion is defined as significant when it causes ≥ 50% reduction in luminal diameter by visual assessment in vessels ≥ 1.5 mm. In Gensini score, lesions causing < 50% reduction in luminal diameter and vessels < 1.5 mm diameter are considered in the algorithm So, these differences or patient selection may explain the association between inadequate STR and Gensini score rather than SYNTAX score.

Limitations of the study

The major limitation of this study is the small sample size. Therefore the ability to generalise this correlation might be limited. Second, although quantitative myocardial contrast echocardiography can give more detailed information about microvascular circulation, an indirect method ∑STR was used for detecting microvascular function in a blinded manual conventional manner. Third, the use of coronary angiography to visually quantify atherosclerosis is limited because remodelling may obscure substantial disease burden in arterial walls that can be detected by intravascular ultrasound.

CONCLUSIONS

Our results suggest that GS-pPCI together with a high thrombus burden might be useful predictors of inadequate STR, independent of other risk factors. So, information provided by GS-pPCI on the risk of inadequate STR in patients with STEMI may encourage more aggressive medical treatment and risk modification. However, additional large-scale studies are needed to clarify the clinical utility of GS-pPCI in the prediction of poor myocardial reperfusion.

Conflict of interest: *none declared*

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Zależność między punktacją w skali Gensiniego a rezolucją uniesienia odcinka ST u chorych z ostrym zawałem serca z uniesieniem odcinka ST poddanych pierwotnej przezskórnej interwencji wieńcowej

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Streszczenie

Wstęp: Stan kliniczny chorych, którzy przebyli zawał serca, zależy głównie od skutecznego przywrócenia perfuzji mięśnia sercowego i nasilenia zmian miażdżycowych w naczyniach wieńcowych.

Cel: Celem pracy była ocena wartości predykcyjnej punktacji w skali Gensiniego w odniesieniu do rezolucji uniesienia odcinka ST (STR) u osób poddanych pierwotnej przezskórnej interwencji wieńcowej (pPCI) z powodu ostrego zawału serca z uniesieniem odcinka ST (STEMI).

Metody: Do badania włączono 114 kolejnych pacjentów (średnia wieku 54 ± 10 lat, 15 kobiet) ze STEMI, u których wykonano — zakończony powodzeniem — zabieg pPCI. Obliczono łączne uniesienie odcinka ST w milimetrach przed angioplastyką i 60 min po pPCI. ∑STR < 50% uznano za elektrokardiograficzny wskaźnik zjawiska *no-reflow*. Stopień obciążenia skrzeplinami określono na podstawie koronarografii, a punktację w skali Gensiniego (GS-pPCI) obliczono po przeprowadzeniu pPCI, nie uwzględniając zmiany odpowiedzialnej za powstanie zawału. Pacjentów podzielono na dwie grupy w zależności od STR: STR(–) i STR(+). Chorych analizowano również w zależności od tętnicy odpowiedzialnej za zawał.

Wyniki: U pacjentów z STR(–) wartość GS-pPCI była istotnie wyższa (10,1 ± 11,8 *vs*. 22 ± 18,6; p = 0,005). Stwierdzono ujemną korelację między GS-pPCI i STR (r = –0,287; p = 0,002). W analizie podgrup wykazano ponadto, że u chorych z grupy STR(–), u których zmiana będąca przyczyną zawału (*culprit lesion*) znajdowała się w gałęzi międzykomorowej przedniej lub gałęzi okalającej, wartości GS-pPCI były wyższe (odpowiednio 10,9 ± 13,5 *vs*. 23,5 ± 21,3; p = 0,03 i 9,6 ± 8,7 *vs*. 24,1 ± 21; p = 0,04). U pacjentów z STR(–) częściej stwierdzano również duże obciążenie skrzeplinami (68% *vs*. 43%, p = 0,03). W wieloczynnikowej analizie regresji logistycznej wykazano, że GS-pPCI i duże obciążenie skrzeplinami były niezależnymi czynnikami predykcyjnymi niedostatecznej STR (odpowiednio, OR 1,07; 95% CI 1,03–1,12; p = 0,001 i OR 3,28; 95% CI 1,11–9,72; p = 0,03).

Wnioski: Wartość GS-pPCI i duże obciążenie skrzeplinami są ważnymi czynnikami predykcyjnymi niedostatecznej STR u chorych ze STEMI poddanych pPCI.

Słowa kluczowe: ostry zawał serca, skala Gensiniego, rezolucja uniesienia odcinka ST, obciążenie skrzeplinami

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