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Predictive factors of myocardial reperfusion in patients with anterior wall acute myocardial infarction

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Abstract

Background: *The no-reflow phenomenon due to microvasculature damage is sometimes observed in patients despite patency of the infarct-related artery. The study aimed to assess the predictive value of clinical, hemodynamic and electrocardiographic parameters for the development of the no-reflow phenomenon in patients after successful coronary reperfusion.*

Methods: *Eighty-six patients, mean age 58.4 ± 11.2, underwent primary percutaneous coronary intervention (PCI) for acute anterior myocardial infarction (AMI). Angiographic parameters, i.e. TIMI grade flow, cTFC, TMPG, wall motion score index (WMSI), ST-segment resolution and segmental perfusion, were estimated by myocardial contrast echocardiography (MCE).*

Results: *As evidenced by MCE, 54 patients were classified as the reflow ones and 32 as no-reflow. Patients from the no-reflow group showed a higher creatine kinase peak ($p = 0.0034$), higher kinase-MB ($p = 0.0033$) and higher troponin level ($p = 0.062$), longer time span between the onset of pain and reperfusion ($p = 0.0003$), worse baseline WMSI ($p = 0.0022$), inferior flow in the infarct-related artery and ST-segment resolution. Univariate analysis revealed that age, time span between the onset of chest pain to PCI, all angiographic parameters, WMSI and ST-segment resolution were related to the no-reflow phenomenon. Multivariate logistic regression analysis revealed that lack of preservation of normal or near-normal flow before PCI and significant impairment of left ventricle contractility were independent predictive factors of the no-reflow phenomenon.*

Conclusions: *MCE yields vital information about the outcome of coronary intervention in patients with AMI. Development of a no-reflow phenomenon is correlated with the severity of myocardial damage and poor flow through the infarct-related artery before PCI.* (Cardiol J 2008; 15: 57–62)

Key words: contrast echocardiography, myocardial perfusion, coronary angioplasty no-reflow phenomenon

Editorial p. 1

Introduction

The treatment of patients with acute myocardial infarction (AMI) is aimed at the restoration of patency in the infarct-related artery. Successful coronary recanalisation is not always associated, however, with the actual restoration of myocardial perfusion. In about one third of patients with acute myocardial infarction, no adequate perfusion was observed, due to microvascular damage sustained [1, 2].

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In these patients, left ventricle remodelling, cardiac events and poor follow-up outcome occurred more frequently. Regretfully, clinical assessment does not facilitate accurate identification of reperfusion [3].

Myocardial contrast echocardiography (MCE) has recently emerged as a potentially useful method of studying myocardial perfusion in coronary artery disease and reperfusion after acute myocardial infarction. Experimental studies in animals have confirmed that MCE accurately delineates myocardium during coronary occlusion, as well as the ultimate size of an infarct [4–7].

Several factors are reported to impact microvascular damage, clinical condition and ultimate outcome of patients with AMI, although their contribution in the development of the no-reflow phenomenon as yet remains undetermined [8].

The present study aimed to compare clinical, hemodynamic and electrocardiographic parameters in patients characterised by reflow and those featuring no-reflow phenomenon, after a reperfused anterior wall AMI, with a view to establishing their predictive value in relation to the no-reflow phenomenon.

Methods

Eighty-six patients, 68 men and 18 women (mean age 58.4 ± 11.2 years; range 29–78), underwent coronary angioplasty (PCI, percutaneous coronary intervention) following diagnosis of anterior wall acute myocardial infarction, within 12 hours of the onset of symptoms. The diagnosis was made on the basis of prolonged chest pain ≥ 30 min, ST-segment elevation ≥ 2 mm in two contiguous electrocardiographic leads and an increase in serum creatinine kinase, or troponin levels. Hemodynamically unstable patients were excluded from the study.

Two-dimensional echocardiography and MCE was performed in all patients before and immediately after PCI. Two-dimensional images of the parasternal long and short-axis view and apical long axis 4- and 2-chamber view were obtained using the Aloka™ system and an ultrasound harmonic transducer transmitting 1.88 MHz and receiving 3.75 MHz. The dynamic range was approximately 60 dB. Wall motion (WM) was interpreted in line with the established criteria. Regions were scored as normal or abnormal: hypokinetic, akinetic or dyskinetic, in compliance with the American Society of Echocardiography guidelines [9]. Wall motion score index (WMSI) was calculated as the sum of individual scores of the respective segments divided by the actual number of segments.

Myocardial perfusion was assessed in real time, using Optison™ second generation contrast agent which was injected as a bolus (0.3–0.5 ml) at a low mechanical index (0.3), through harmonic imaging, and with a 16-segment model of the left ventricle. Segmental myocardial contrast was graded semiquantitatively in line with the following score: 0 — lack of perfusion (no visible contrast effect), 0.5 — partial perfusion (patchy myocardial contrast enhancement), 1 — normal perfusion (homogenous contrast effect). The risk area was defined as the number of segments with no perfusion prior to angioplasty.

We defined the no-reflow zone on the end--diastolic images as a contrast perfusion defect after PCI. We quantified the area of no-reflow as its ratio to the risk area — lack of perfusion before PCI. When the ratio exceeded 25%, myocardial reperfusion in the corresponding segments was considered incomplete (MCE no-reflow). If this ratio was ≤ 25%, we regarded myocardial reperfusion as adequate (MCE reflow).

ST-segment resolution was analysed in all patients. The sum of ST elevation was assessed in three contiguous leads in the infarct zone, 60 ms from the J point. The extent of ST-segment resolution was assessed 90 and 180 minutes after reperfusion and expressed as the percentage of the ST-segment elevation shown on the baseline presenting ECG [10].

Coronary angiography was performed in multiple projections, using the standard Judkins technique. The patency of the infarct-related artery was classified in compliance with the thrombolysis in myocardial infarction (TIMI) criteria; whereas the corrected TIMI frame count (cTFC) and TIMI Myocardial Perfusion Grade (TMPG) were assessed using the techniques described in detail in previous reports [11–13].

Primary PCI was performed in all patients. Twenty-two patients, whose estimated transfer time was in excess of 90 minutes, were administered with a reduced dose of a fibrinolytic drug — alteplase (bolus of 15 mg, followed by an infusion of 35 mg/60 min) and glycoprotein IIb/IIIa inhibitor — abciximab (bolus of 0.25 mg/kg followed by an infusion of 0.125 μg/kg/min, max. 10) before the actual transportation to our cat-lab. These infusions were continued during the actual transfer to the cat-lab, with a view to performing coronary angioplasty, hence named the facilitated angioplasty.

The study protocol was approved by the local ethical committee and informed consent was obtained from each patient.

Statistical analysis

Continuous data were expressed as mean ± ± SD standard deviation and were compared using Student's t-test. We made comparisons by using one-way analysis of variance for continuous variable, and the significance of the difference was calculated by using the Scheffé F-test for factor analysis. Categorical variables were compared by using the chi-square test or Fisher's test. Univariate and multivariate logistic regression analyses were used to identify independent predictors for development of the no-reflow phenomenon. Differences were considered significant at $p < 0.05$.

Results

Out of 86 study subjects, 68 (79%) had no history of coronary artery disease or pre-infarction angina, 18 (21%) had previously experienced chest pains, 52 (60%) had suffered from hypertension, 66 (77%) from hyperlipidemia, 22 (26%) from diabetes mellitus, and 38 (44%) had a history of smoking.

Significant stenosis of the left anterior descending (LAD) coronary artery in the 6^{th} or 7^{th} segment (mean 92.3 ± 12.6%) was confirmed in all patients. Forty-five patients had single-vessel and 23 had double-vessel disease with a non-significant coronary stenosis (< 50% luminal diameter stenosis) in the second coronary artery, whereas in 18 patients significant coronary stenosis (50–70% luminal diameter stenosis) was observed in other arteries than the infarct-related one.

Total occlusion of the infarct-related coronary artery (TIMI 0) was observed in 34 patients, and in 38 patients (mainly from the facilitated angioplasty group) coronary flow appeared (TIMI 2 or 3).

Using MCE, the no-reflow phenomenon was observed within the risk area in 32 patients (37%) (MCE no-reflow), and the remaining 54 (63%) of patients were classified as the reflow ones (MCE reflow). Table 1 shows the characteristics of the total of patients as well as both groups of patients: with MCE reflow and with MCE no-reflow.

The patients with MCE no-reflow were characterised by longer time elapsed from the onset of chest pain to coronary angioplasty ($p = 0.0003$), higher level of creatine kinase peak (CPK) ($p = = 0.0034$) and CPK/MB ($p = 0.0033$) and troponin ($p = 0.062$), smaller flow in epicardial artery, as determined by TIMI and cTFC, and smaller myocardial perfusion, as determined by TMPG before PCI.

Among 34 patients with complete occlusion (TIMI flow grade 0) of the LAD, higher incidence of TIMI flow grade 0 was observed before PCI in patients with MCE no-reflow than in patients with MCE-reflow, i.e. 23 *vs.* 11 patients, respectively ($p = 0.0026$). After PCI, TIMI 0 or 1 was observed only in patients with MCE no-reflow. Among 36 patients with TMPG 0 before PCI, 28 patients were classified to the MCE no-reflow group. After PCI, TMPG 0 was observed only in patients with MCE no-reflow ($p = 0.0001$).

WMSI was greater and ST-segment resolution was smaller in patients with MCE no-reflow than in those with MCE reflow. In 21 patients with MCE no-reflow, ST-segment resolution was < 30%, whereas this value was noted only in one patient within the MCE reflow group. Complete resolution (ST-segment resolution > 70%) was observed only in patients in the MCE reflow group (Table 1).

We did not find any differences between the respective groups of patients treated with either primary or facilitated angioplasty. As evidenced by MCE in 39 (61%) patients treated with primary PCI and 15 (68%) treated with the facilitated one, there was a significant improvement of myocardial perfusion after PCI — reflow group ($p = 0.461$).

Multivariate logistic regression analysis revealed that lack of preservation of normal or near-normal flow before PCI (OR = 2.6455; $p = 0.0663$) and significant impairment of left ventricle contractility (OR = 0.0001; $p = 0.0303$) were independent predictive factors of the no-reflow phenomenon.

Univariate analysis indicated that age, time elapsed from the onset of chest pain to coronary angioplasty, complete occlusion (TIMI 0) or poor flow in the LAD, as assessed by cTFC, poor myocardial perfusion, as assessed by TMPG, and poor ST-segment resolution, were independent predictors of the no-reflow phenomenon (Table 2).

Discussion

Microvascular damage due to AMI remains an important prognostic factor. In about one third of patients with AMI, despite angiographically established patency of the infarct-related artery, there was no adequate perfusion, due to microvascular damage sustained. The patients with no improvement of myocardial perfusion after successful PCI made up the high-risk group, hence having rather poor follow-up outcome, and a lack of recovery of left ventricular function might reasonably be anticipated [14, 15].

In the present study we investigated the factors related to the no-reflow phenomenon in patients with anterior wall acute myocardial infarction. Much in keeping with the reports of other investi-

Table 1. Characteristics of patients with myocardial contrast echocardiography (MCE) reflow and MCE no-reflow immediately after coronary angioplasty.

	Total (n = 86)	MCE reflow (n = 54)	MCE no-reflow (n = 32)	p
Age (years)	58.4 ± 11.2	55.7 ± 11.9	63.04 ± 9.7	0.0114
Women/men	18/68	11/43	7/25	0.5758
Time to PCI	3.9 ± 1.3	3.1 ± 1.25	4.7 ± 1.4	0.003
Peak creatine kinase [U/L]	3973.32 ± 3887.6	2940.4 ± 3298.4	5694.7 ± 4253.1	0.0034
Peak creatine kinase-MB [U/L]	388.16 ± 343.4	306.5 ± 335.1	524.1 ± 319.9	0.0033
Troponin	57.5 ± 85.7	37.9 ± 74.1	87.2 ± 94.9	0.0629
Diameter stenosis before PCI (%)	92.3 ± 12.6	90.1 ± 13.8	95.9 ± 9.0	0.0032
TIMI before PCI				
TIMI 0	34 (39%)	11 (20%)	23 (72%)	0.0026
TIMI 1	14 (16%)	8 (15%)	6 (19%)	0.6864
TIMI 2	23 (27%)	21 (39%)	2 (6%)	0.0087
TIMI 3	15 (18%)	14 (26%)	1 (3%)	0.02
TIMI after PCI				
TIMI 0	1 (1%)	0	1 (3%)	0.1982
TIMI 1	5 (6%)	0	5 (16%)	0.0055
TIMI 2	16 (19%)	5 (9%)	11 (34%)	0.019
TIMI 3	64 (74%)	49 (91%)	15 (47%)	0.0721
cTFC before PCI	62.45 ± 39.1	42.2 ± 23.4	92.2 ± 38.9	0.00009
cTFC after PCI	39.3 ± 31.1	27.1 ± 14.3	57.3 ± 40.8	0.0028
TMPG before PCI				
TMPG 0	36 (42%)	8 (15%)	28 (88%)	0.001
TMPG 1	9 (10%)	8 (15%)	1 (3%)	0.1177
TMPG 2	25 (29%)	22 (40%)	3 (9%)	0.017
TMPG 3	16 (19%)	16 (30%)	0	0.0032
TMPG after PCI				
TMPG 0	11 (13%)	0	11 (34%)	0.0001
TMPG 1	14 (16%)	0	14 (44%)	< 0.0001
TMPG 2	25 (29%)	19 (35%)	6 (19%)	0.2205
TMPG 3	36 (42%)	35 (65%)	1 (3%)	0.0001
ST-segment resolution < 30%	22 (26%)	1 (2%)	21 (66%)	< 0.0001
ST-segment resolution 30–70%	30 (35%)	19 (35%)	11 (34%)	0.9577
ST-segment resolution > 70%	34 (39%)	34 (63%)	0	< 0.0001
Pre-infarction angina	18 (21%)	11 (20%)	7 (22%)	0.8935
No pre-infarction angina	68 (79%)	43 (80%)	25 (78%)	0.9548
Diabetes mellitus	22 (26%)	12 (22%)	10 (31%)	0.4790
Hypertension	52 (60%)	29 (54%)	23 (72%)	0.4143
Hyperlipidemia	66 (77%)	44 (81%)	22 (69%)	0.6207
Smoking	38 (44%)	14 (26%)	24 (75%)	0.0074
Family history of CAD	31 (36%)	21 (39%)	10 (31%)	0.6222
WMSI before PCI	1.6 ± 0.17	1.55 ± 0.18	1.7 ± 0.1	0.0022
WMSI after PCI	1.48 ± 0.22	1.37 ± 0.18	1.66 ± 0.15	0.00004
WMSI 3 day	1.36 ± 0.21	1.25 ± 0.18	1.55 ± 0.12	0.00009
EF before PCI (%)	43.5 ± 9.7	47.9 ± 7.4	36.8 ± 8.9	0.001
EF after PCI (%)	44.1 ± 9.5	48.4 ± 7.3	37.6 ± 9.1	0.001
EF 3 day (%)	49.8 ± 8.9	54.2 ± 7.1	42.8 ± 6.8	0.001

CAD — coronary artery disease; TIMI — thrombolysis in myocardial infarction trial flow grade; cTFC — corrected TIMI frame count; TMPG — TIMI myocardial perfusion grade; PCI — percutaneous coronary intervention; WMSI — wall motion score index; EF — ejection fraction

Table 2. Univariate predictors of the no-reflow phenomenon.

Parameter	Univariate analysis (p)
Age	0.009
Time to PCI	< 0.001
Peak creatine kinase [U/L]	0.009
Peak creatine kinase-MB [U/L]	0.02
Troponin	0.031
Diameter stenosis before PCI (%)	0.042
TIMI before PCI	< 0.001
TIMI 0 before PCI	< 0.001
cTFC before PCI	< 0.001
TMPG before PCI	< 0.001
ST-segment resolution	0.001
Pre-infarction angina	0.916
No pre-infarction angina	0.631
Diabetes mellitus	0.507
Hypertension	0.35
Hyperlipidemia	0.004
Smoking	0.55
Family history of CAD	0.907
WMSI before PCI	0.002

CAD — coronary artery disease; TIMI — thrombolysis in myocardial infarction trial flow grade; cTFC — corrected TIMI frame count; TMPG — TIMI myocardial perfusion grade; PCI — percutaneous coronary intervention; WMSI — wall motion score index

gators, univariate analysis indicated that the age of patients, segmental left ventricular function (as assessed by WMSI) and the initial flow through the infarct-related artery were the factors actually instrumental in the development of a no-reflow phenomenon [16, 17].

Several studies pointed to the protective role of residual coronary flow, which also happens to be very much in line with our own findings. Patients with totally occluded coronary arteries tended to have poor final TIMI flow compared to those with partially occluded vessels [18–21]. Complete occlusion of the infarct-related artery (TIMI 0), poor flow of LAD (as assessed by cTFC) and poor myocardial perfusion (as assessed by TMPG) visibly affected the actual development of the no-reflow phenomenon. Seventy two per cent of patients characterised by initial TIMI 0 and 88% of patients with TMPG 0 were therefore allocated to the MCE no-reflow group.

Combination fibrinolytic therapy restored infarct-related artery potency in 18 (81%) patients of the facilitated angioplasty group before PCI but did not effect clinical outcomes.

Our study indicated that the severity of myocardial damage before coronary reperfusion impacted the final results [22–26]. All parameters which attested to myocardial damage, i.e. longer time to reperfusion, higher level of myocardial necrotic factors, smaller ST-segment resolution, were closely related to the no-reflow phenomenon.

Some authors have argued that pre-infarction angina might actually attenuate the development of the no-reflow phenomenon [27, 28]. However, our study found no evidence to support this assertion. Neither did we observe any differences between patients with or without pre-infarction angina in either group, nor indeed any impact on the actual development of the no-reflow phenomenon.

From several clinical risk factors, only hyperlipidemia and smoking habit prove to be significant predictors of the no-reflow phenomenon [29].

Conclusions

1. Myocardial contrast echocardiography yields useful information on the outcome of coronary intervention in patients with anterior wall acute myocardial infarction.
2. Development of the no-reflow phenomenon frequently appears to be correlated with the severity of myocardial damage (higher level of myocardial necrotic markers, longer duration of ischemia).
3. Preservation of normal or near-normal coronary flow before intervention correlates significantly with good PCI outcome in patients with anterior wall acute myocardial infarction.

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