Reperfusion Syndrome: Relationship of Coronary Blood Flow Reserve to Left Ventricular Function and Infarct Size

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OBJECTIVES

We tested the hypothesis that the reperfusion syndrome (RS), defined as an additional elevation of the ST segment upon reperfusion, may be a marker of microcirculatory reperfusion injury during acute myocardial infarction (AMI).

BACKGROUND

The pathophysiology of the RS is unknown, and its prognostic implications are controversial.

METHODS

Twenty-one patients with an anterior AMI treated ≤12 h after onset by primary coronary angioplasty (PTCA) were studied. Coronary velocity reserve (CVR), an index of microcirculatory function, was measured using a Doppler guidewire. Left ventricular (LV) ejection fraction, infarct size (percent defect) and LV end-systolic volume index (LVESVi) were evaluated by radionuclide ventriculography, 201T1 single-photon emission computed tomography and contrast ventriculography, respectively.

RESULTS

Baseline ST elevation and pain-to-TIMI 3 time were similar in patients with and without RS. Patients with RS (10/21) had a lower post-PTCA CVR than patients without RS (median [95% confidence interval]: 1.2 [1–1.3] vs. 1.6 [1.5–1.7], p < 0.005). Even though predischarge CVR was similar in the two groups, infarct size at six weeks (26 [21 to 37] vs. 14 [10–17] % 201T1 defect, p = 0.001) and predischarge LVESVi (45% [40 to 52] vs. 30% [29 to 38] mL/m², p = 0.001) were larger, and LV ejection fraction at six weeks (40% [37 to 46] vs. 55% [50 to 60], p = 0.004) was lower in patients with RS than in patients without RS.

CONCLUSIONS

Patients with RS during primary PTCA for an anterior AMI have a transiently lower CVR than patients without RS, but sustained LV dysfunction and larger infarct size, suggesting that RS is a marker of microcirculatory reperfusion injury. (J Am Coll Cardiol 2000;35: 1162–9) © 2000 by the American College of Cardiology

In a substantial subset of patients with acute myocardial infarction (AMI), timely revascularization fails to salvage ischemic myocardium (1). This unfavorable outcome stems from a discrepancy between an open epicardial infarct-related coronary artery and the absence of blood flow (no-reflow) in the damaged distal microvessels (2). The aim of this study was to determine whether a reperfusion syndrome (RS), defined as an acute additional elevation of the ST segment upon reperfusion (3–9), is a marker of impaired reperfusion. Coronary velocity reserve (CVR), an index of microcirculation integrity, was measured in patients with and without RS after primary percutaneous transluminal coronary angioplasty (PTCA) for an anterior AMI. We tested the hypotheses that patients with RS would have: 1) lower post-PTCA CVR, and 2) more extensive myocardial infarction and worse left ventricular (LV) function at follow-up.

METHODS

Patient population. The study population comprised 21 patients referred to the Coronary Care Unit ≤12 h after onset of an anterior AMI and who underwent primary PTCA. Only patients with total occlusion (i.e., Thrombolysis in Myocardial Infarction (TIMI) grade 0 or 1 flow) of the proximal or midleft anterior descending coronary artery (LAD) were included. Patients with prior AMI, cardiogenic shock or potential alteration of microcirculatory function due to a history of diabetes mellitus or severe LV hypertrophy were excluded. In order to prevent heterogeneous collection of flow velocity data, the same operator (L.J.F.) performed all the Doppler analyses, and, hence, patients were not consecutively included. The study protocol was

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approved by the Human Research Committee of the Institutional Review Board, and patients gave their informed consent before inclusion.

**Electrocardiographic analysis.** Two electrocardiograms (ECGs) were analyzed. The first ECG was obtained immediately before angioplasty (TIMI 0 flow) and the second ≤2 min after recanalization of the infarct-related artery (TIMI 3 flow). Patients were classified into two groups depending upon presence or absence of RS, defined as an isoelectric line was defined as the preceding PR segment. The isoelectric line was defined as the preceding PR segment. Patients with new right or left bundle branch block or persistent ventricular arrhythmia were excluded.

**Coronary balloon angioplasty.** Each patient received ≥250 mg aspirin and heparin (100 IU/kg IV) before PTCA. Percutaneous transluminal coronary angioplasty was performed using a 0.014-in Doppler-tipped guidewire (FloWire, Endosonics Rancho Cordova, California). Procedural success was defined as TIMI 3 flow and <50% residual stenosis. Coronary artery dissections were graded according to National Heart, Lung and Blood Institute classification (10). Stents were implanted in case of unsatisfactory result or preprocedural acute reocclusion. Stented patients received ticlopidine (250 mg twice a day) and aspirin (100 mg), whereas nonstented patients were given aspirin (250 mg) only. Each patient underwent predischarge coronary angiography.

Quantitative coronary angiography and measurement of LV volumes. Quantitative coronary angiography was performed using the Philips DCI system. Arterial diameter at the angioplasty site was measured on end-diastolic frames. Two orthogonal projections were used to compute the minimal lumen diameter (MLD) and the percent diameter stenosis. The guiding catheter was used as a scaling device. Collateral vessels were graded according to Rentrop’s classification (11). Left ventricular end-systolic volume index, an independent predictor of early and late mortality after AMI (12), was evaluated according to the area-length method, using LV angiograms obtained in the 30° right anterior oblique projection during predischarge angiography.

**Coronary flow velocity reserve measurements.** Blood flow velocity was measured after angioplasty and during predischarge angiography using the Doppler guidewire connected to a real-time spectrum analyzer (FloMap, Endosonics), as described (13). The Doppler tip was positioned at a distance ≥5 times the vessel diameter distal to the angioplasty site to avoid poststenotic turbulence. Velocity sampling was repeated in the same arterial segment at the predischarge study. Flow-dependent dilatation of epicardial coronary arteries was abolished by intracoronary injection of 1 mg linsidomine.

Time-averaged peak velocity (APV) was computed online and continuously recorded on videotape. Coronary velocity reserve was calculated as the ratio of hyperemic to resting APVs (APVpeak/APVbase) after intracoronary injection of 18 μg adenosine (14). Because postreperfusion hyperemic flow may result in underestimation of CVR, measurements were performed only when APV had reached a plateau for ≥10 min, without cyclic flow variations, as indicated by the recording of velocity trends (15). Measurements were repeated until 2 consecutive CVR determinations varied by ≤10%. In each patient, CVR was also measured in an angiographically normal coronary artery.

**Determination of myocardial infarct size and LV ejection fraction.** Measurements of total creatine kinase activity (normal value <295 IU/L) were performed every 6 h for 48 h after PTCA. Infarct size was first approximated by peak creatine kinase activity, then measured by the postinjection percentage 201T1 defect; on rest 201T1 single-photon

### Table 1. Baseline Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>RS</th>
<th>No RS</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>10 (47.6)</td>
<td>11 (52.3)</td>
<td></td>
</tr>
<tr>
<td>Male gender (n, [%])</td>
<td>7 (70)</td>
<td>7 (63.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>63.5 (52.1–71.3)</td>
<td>59 (47.4–67)</td>
<td>NS</td>
</tr>
<tr>
<td>Pain-to-CCU time (min)</td>
<td>198 (151–316)</td>
<td>250 (191–300)</td>
<td>NS</td>
</tr>
<tr>
<td>Exacerbation of pain at reperfusion (n, [%])</td>
<td>10 (100)</td>
<td>1 (9.1)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>ΣST pre-PTCA (mm)</td>
<td>14.5 (9.6–18.2)</td>
<td>13 (9.5–16.6)</td>
<td>NS</td>
</tr>
<tr>
<td>ΣST ≤2 min post-PTCA (mm)</td>
<td>25 (18.9–29.7)</td>
<td>10 (7.5–13.1)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CCU = coronary care unit; RS = reperfusion syndrome; ΣST = sum of ST-segment elevation.
emission computed tomography performed six weeks after AMI (16). Radionuclide LV ejection fraction was measured six weeks after AMI (17).

Statistical analysis. Continuous variables are expressed as medians (95% confidence interval). Intergroup comparisons of continuous variables at day 0 were performed by one-way analysis of variance. Inter- and intragroup comparisons of hemodynamic and blood flow velocity data required a two-way repeated measures analysis of variance, which tested the effects of group (RS vs. no RS), time (post-PTCA vs. predischarge) and the interaction of both on these variables. If the global test showed a significant effect or interaction, post hoc unpaired or paired Student t tests were performed for intergroup and intragroup comparisons, respectively. Categorical variables were analyzed by the chi-square test. Relation between Rentrop score and post-PTCA CVR was determined by regression analysis. All statistical analyses were performed with the SAS V6.12 software. A value of p < 0.05 was considered statistically significant.

RESULTS

Clinical characteristics. An ECG pattern suggestive of RS, which was always associated with an exacerbation of chest pain, was found in 10 of 21 patients. Clinical characteristics of patients with and without RS were similar (Table 1).

PTCA and angiographic data. All the patients had TIMI 0 or 1 flow before PTCA. Collateral flow to the ischemic myocardium was significantly lower in patients with RS (Table 2). Percutaneous transluminal coronary angioplasty

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### Table 2. Angiographic Data During Primary PTCA

<table>
<thead>
<tr>
<th></th>
<th>RS</th>
<th>no RS</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivessel disease</td>
<td>4 (40)</td>
<td>5 (45.4)</td>
<td>NS</td>
</tr>
<tr>
<td>Collateral flow</td>
<td>0 (0–0.8)</td>
<td>1 (0.8–1.7)</td>
<td>0.003</td>
</tr>
<tr>
<td>Reference segment</td>
<td>3.1 (2.9–3.3)</td>
<td>3 (2.8–3.1)</td>
<td>NS</td>
</tr>
<tr>
<td>MLD post-PTCA</td>
<td>2.6 (2.1–2.9)</td>
<td>2.5 (2.3–2.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Percent stenosis</td>
<td>14.5 (9.7–28.4)</td>
<td>14 (9.5–22)</td>
<td>NS</td>
</tr>
<tr>
<td>Stent implantation</td>
<td>6 (60)</td>
<td>6 (54.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Angiographic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>complications (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiographic no- or low-reflow</td>
<td>1 (10)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Distal embolism</td>
<td>2 (20)</td>
<td>2 (18.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Dissection (grade &gt; B*)</td>
<td>3 (30)</td>
<td>4 (36.4)</td>
<td>NS</td>
</tr>
<tr>
<td>Acute reocclusion</td>
<td>2 (20)</td>
<td>1 (9.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Time from pain to TIMI 3 flow (min)</td>
<td>290 (212–393)</td>
<td>320 (242–341)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Dissection protruding outside the lumen of the vessel and persisting after passage of the contrast material. MLD = minimal lumen diameter; RS = reperfusion syndrome.

### Table 3. Hemodynamic and Flow Velocity Data

<table>
<thead>
<tr>
<th></th>
<th>Heart Rate (/min)</th>
<th>Mean ABP (mm Hg)</th>
<th>APV&lt;sub&gt;base&lt;/sub&gt; (cm/s)</th>
<th>APV&lt;sub&gt;peak&lt;/sub&gt; (cm/s)</th>
<th>CVR Infarct-Related Artery</th>
<th>CVR Normal Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-PTCA</td>
<td>82.5 (80–91)</td>
<td>93.5 (89.3–104)</td>
<td>16.5 (13.8–20.8)</td>
<td>22.5 (17–26)</td>
<td>1.2 (1.1–1.3)</td>
<td>2 (1.7–2.2)</td>
</tr>
<tr>
<td>Predischarge</td>
<td>64 (60–74.7)</td>
<td>*</td>
<td>17 (14.3–21.3)</td>
<td>36 (28.6–40.3)</td>
<td>1.8 (1.7–2.2)</td>
<td>2.8 (2.2–3.4)</td>
</tr>
<tr>
<td>no RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-PTCA</td>
<td>85 (76–89)</td>
<td>90 (85.2–102.6)</td>
<td>16 (13.4–21.8)</td>
<td>26 (22.2–32.7)</td>
<td>1.6 (1.5–1.7)</td>
<td>2.2 (1.9–2.5)</td>
</tr>
<tr>
<td>Predischarge</td>
<td>60 (57.5–66)</td>
<td>*</td>
<td>18 (14–18.5)</td>
<td>32 (28–38.5)</td>
<td>2 (1.9–2.2)</td>
<td>3 (2.7–3.1)</td>
</tr>
</tbody>
</table>

2-Way ANOVA (p Values)

<table>
<thead>
<tr>
<th></th>
<th>Group effect</th>
<th>Time effect</th>
<th>Interaction</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>0.2</td>
<td>&lt;0.005</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>0.8</td>
<td>0.01</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>0.7</td>
<td>0.1</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>0.03</td>
<td>&lt;0.005</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>&lt;0.005</td>
<td>&lt;0.005</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>0.3</td>
<td>&lt;0.005</td>
<td>0.6</td>
</tr>
</tbody>
</table>

*p < 0.005 vs. post-PTCA; †p = 0.01 vs. post-PTCA; ‡p = 0.05 vs. RS; ¶p < 0.005 vs. RS; §p < 0.005 vs. infarct-related artery; ¶¶p = 0.02 vs. infarct-related artery. ABP = arterial blood pressure; APV = time-averaged peak velocity; CVR = coronary velocity reserve; RS = reperfusion syndrome.
was successful in each case, resulting in similar residual stenoses and MLDs in the two groups. Transient acute reocclusion occurred in two patients with RS and one without RS, in each case 10 min after initial reperfusion.

Coronary stents were implanted in a similar proportion of patients with and without RS. No antiglycoprotein IIb/IIIa agent was used. Predischarge coronary angiography was performed in each patient except one with RS, who died of LV rupture 24 h after PTCA. The median intervals between initial and predischarge angiographies were seven days in both groups. No patient had silent reocclusion or early restenosis (<50% stenosis at the target site). Residual stenoses at the target site were 14% (6 to 23) and 13% (10 to 19) in patients with and without RS, respectively.

**Hemodynamic and coronary flow velocity data.** Coronary flow velocity decreased progressively after reperfusion, then reached a plateau, without detectable cyclic flow variations. The hemodynamic and flow velocity variables, at steady state after PTCA and at predischarge follow-up, are shown in Table 3. At each time point, hemodynamic variables were similar in patients with and without RS. However, in each group, heart rate and mean arterial blood pressure significantly decreased over time.

Although post-PTCA CVR in the infarct-related coronary artery was severely depressed in both groups, CVR was significantly lower in patients with RS. Representative examples are depicted in Figures 1 and 2. The lower CVR in patients with RS was related to significantly lower hyperemic velocities, whereas resting velocities were similar in the two groups. Overall, post-PTCA CVR correlated positively with initial Rentrop score (p = 0.001, R = 0.6).

There was a marked effect of time (p < 0.005) and a significant group (RS vs. no RS) by time (post-PTCA vs. predischarge) interaction (p = 0.03) on CVR in the infarct-related artery. Indeed, in both groups, predischarge CVR was significantly higher than post-PTCA CVR. However, percent increase in CVR was higher in patients with RS (66.7% [54–87.2] vs. 31.6% [20.5–38.1], p < 0.005), and, hence, predischarge CVR was similar in the two groups. Improvement of CVR over time was related to a significant increase in hyperemic velocities in patients with RS (Fig. 3).

**Figure 1.** ECG and coronary flow patterns in a patient with reperfusion syndrome. Primary PTCA of the mid-LAD was performed 245 min after the onset of chest pain. **Panel A:** Electrocardiogram pattern. Thirty s after reperfusion, the patient complained of increased chest pain. The ECG obtained at that moment shows additional elevation of ST segment in leads V2 to V4; sum of ST segment elevation (ΣST) increased from 13 to 31 mm. **Panel B:** Flow velocity pattern in the distal LAD 10 min after successful PTCA. On-line flow velocity spectrum and time-averaged peak velocity (APV) are displayed on the top screen. Bottom left and right screens indicate base (BAPV = 19 cm/s) and adenosine-induced hyperemic (PAPV = 26 cm/s) APVs, respectively. Residual coronary velocity reserve (RATIO) is 1.3. ECG = electrocardiogram; LAD = left anterior descending coronary artery; PTCA = percutaneous transluminal coronary angioplasty.
Control CVR, measured in an angiographically normal coronary artery, was not different in the two groups. However, control CVR was higher than CVR in the infarct-related artery at each time point and in both groups. In addition, there was a significant effect of time (p < 0.005) on control CVR, which was higher at predischarge than post-PTCA (Table 3).

Infarct size and LV function. Peak creatine kinase activity was higher in patients with RS than in patients without RS (5,550 [4,653–6,452] vs. 3,500 [2,021–4,127] IU/L, p = 0.001). Similarly, the actual infarct size, measured six weeks after AMI by the postinjection percentage $^{201}$T1 defect, was larger in patients with RS (Fig. 4). In addition, radionuclide LV ejection fraction six weeks after AMI was significantly lower, and predischarge LV end-systolic volume index was markedly larger in patients with RS.

In-hospital complications. One patient with RS died from LV rupture 24 h after PTCA. Six of 10 patients with RS, but only 2 of 11 without RS, experienced ≥1 episode of in-hospital congestive heart failure (Killip grade ≥2, p = 0.05).

DISCUSSION

In this study: 1) a reperfusion syndrome was noted in 48% of patients with an anterior AMI who were revascularized using primary PTCA, 2) post-PTCA CVR was more severely depressed in patients with RS than in patients without RS, 3) although transient, the more severe alteration of CVR in patients with RS was associated with more in-hospital episodes of heart failure, larger myocardial infarcts and greater depression of LV function.

ST changes during myocardial reperfusion. The poor prognosis associated with persistent ST segment elevation after primary angioplasty has been recently emphasized (18,19). In contrast, the prognostic value of acute exacerbation of ST segment elevation upon reperfusion (i.e., RS) is unclear. Even though patients with RS during successful thrombolysis (4–6,20) as well as PTCA (7–9) may be at

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**Figure 2.** Electrocardiogram and coronary flow patterns in a patient without reperfusion syndrome. Primary PTCA of the mid-LAD was performed 255 min after the onset of chest pain. Panel A: Electrocardiogram pattern. The patient experienced a gradual decline of chest pain. The ECG obtained 1 min after reperfusion shows a partial resolution of ST segment elevation in leads V1 to V4, sum of ST segment elevation (ΣST) decreased from 14 to 4 mm. Panel B: Flow velocity pattern in the distal LAD 10 min after successful PTCA. On-line flow velocity spectrum and time-averaged peak velocity (APV) are displayed on the top screen. Bottom left and right screens indicate base (BAPV = 17 cm/s) and adenosine-induced hyperemic (PAPV = 35 cm/s) APVs, respectively. Residual coronary velocity reserve (RATIO) is 2.0. ECG = electrocardiogram; LAD = left anterior descending coronary artery; PTCA = percutaneous transluminal coronary artery.
particularly high risk of developing large infarcts and severe LV dysfunction, this issue is still debated (3).

**Pathophysiology of the reperfusion syndrome.** It is unlikely that acute reocclusion or distal emboli account for RS, because, in our study, these angiographic complications were infrequent and occurred with a similar incidence in patients with and without RS. Moreover, cyclic variations of flow velocity, an indicator of transient episodes of reocclusion (15), were not observed in patients with RS. Kern et al. (21) have recently described a broad spectrum of coronary flow velocities in AMI patients with stable TIMI 3 flow after primary PTCA, with a higher incidence of recurrent ischemia in patients with velocities ≤20 cm/s. In this study, however, post-PTCA flow velocity was similar in patients with and without RS. Hence, the heterogeneity flow velocity after reperfusion is not a major determinant of RS.

**Reperfusion syndrome and microcirculatory dysfunction.** One of the main findings of this study was that patients with RS had a lower post-PTCA CVR than patients without RS. Coronary flow reserve decreased after myocardial ischemia/reperfusion (22). Recently, several investigators using intracoronary Doppler catheters (23,24), Doppler guidewires (21,23,25,26), videodensitometry (27) or positron emission tomography (28) have consistently reported in AMI patients a markedly depressed flow reserve in the infarct zone, despite an angiographically successful revascularization.

The lower post-PTCA CVR observed in patients with RS may be the consequence of larger myocardial infarction and more extensive destruction of resistive arterioles. However, the fact that, in these patients, CVR improved by >70% a few days after reperfusion and reached a level similar to that found in patients without RS does not support this hypothesis.

Alternatively, the transiently lower CVR in patients with RS may reflect microvascular reperfusion injury (29). Microvascular types of reperfusion injury include the no-reflow phenomenon (22) as well as an impairment of the microcirculatory function without obstruction to blood flow (30). It is unknown which of these two mechanisms is predominantly involved in the dramatic reduction of CVR observed in patients with RS. However, the specific flow patterns of no-reflow after primary PTCA (31) were not observed in patients with RS, suggesting that transient microcirculatory dysfunction prevails.

What may predispose patients to RS is unclear. Baseline characteristics were similar in the two groups, except for collateral flow, which was more preserved in patients without RS. Whether collateral flow to the infarct zone prevents reperfusion-related microcirculatory dysfunction and related RS is unknown; however, the positive correlation between Rentrop grade and post-PTCA CVR favors this hypothesis.
Clinical relevance of the reperfusion syndrome. Although the severe impairment of CVR in patients with RS was transient, it was associated with larger infarct size and poor recovery of LV function. Previous studies using myocardial contrast echocardiography (32), positon emission tomography (33) and videodensitometry (27) have demonstrated that the preservation of an intact microcirculation in the reperfused myocardium is a prerequisite for myocardial salvage (34). More recently, Neumann et al. (26) reported serial measurements of coronary flow reserve using a Doppler guidewire in 19 AMI patients who underwent primary PTCA and stent implantation (26). They found in 13 patients an improvement of flow reserve in the first hour after reperfusion, whereas six patients showed an early decrease in flow reserve and tended to have larger enzymatic infarct size. Whether this early decrease of flow reserve can be related to the RS remains to be determined.

Study limitations. The main limitation of this study was the lower CVR found in these patients, but also by several baseline clinical characteristics known to determine infarct size. However, only patients with an occluded proximal or mid-LAD were included in this study, and baseline ST elevation was similar in patients with and without RS, suggesting that the areas at risk were in the same range. The lower collateral flow grade in patients with RS certainly contributed to the severity of myocardial infarction. Paucity of collateral flow has been related to the amplitude of baseline ST segment elevation (35). Whether it may also account for acute ST changes upon reperfusion is unknown. Finally, pain-to-TIMI 3 time was similar in the two groups.

Another limitation is that CVR, the parameter chosen to relate RS to microcirculatory dysfunction, is dependent upon several factors other than microcirculation, such as hemodynamic variables (36) and residual stenosis of the conduit artery (25). However, there was no difference in heart rate, mean arterial blood pressure and residual stenosis among groups, both at initial and predischarge angiography.

In summary, this study demonstrated that the RS, which frequently accompanies the brisk reopening of occluded LADs during primary PTCA for AMI, is associated with severe, transient and, at least in part, reperfusion-related alteration of microcirculatory function. Patients with RS may benefit from adjuvant therapies aimed at improving myocardial reperfusion, e.g., adenosine (37) or abciximab (38).

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