Predictive Factors and Impact of No Reflow After Primary Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction

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Background—The investigation of no-reflow phenomenon after percutaneous coronary intervention (PCI) in patients with acute ST-segment–elevation myocardial infarction has therapeutic implications. We investigated the predictive factors, persistence in time, and impact of no reflow on myocardial salvage, ventricular function, and mortality.

Methods and Results—The study included 1140 patients with ST-segment–elevation myocardial infarction undergoing primary PCI and paired scintigraphic examinations (before intervention and 7 to 14 days thereafter). After primary PCI, 108 patients had no reflow and 1032 patients had normal coronary flow. The median salvage index was 0.34 (interquartile range, 0.15, 0.49) in patients with no reflow versus 0.55 (interquartile range, 0.29, 0.81) in patients with normal flow (P<0.001). Left ventricular ejection fraction at 6 months after PCI was 47.7±13.1% in the no-reflow group versus 54.2±13.9% in the group with normal flow after PCI (P<0.001). In 80.3% of patients with no reflow, normalization of blood flow >6 months after PCI occurred and correlated with improvement in the left ventricular ejection fraction. Independent predictors of no reflow were residual flow in the infarct-related artery (P=0.03), C-reactive protein (P<0.001), and previous myocardial infarction (P=0.013). Kaplan–Meier estimates of 1-year mortality were 16.7% (n=18) in patients with no reflow versus 5.5% (n=56) in patients with normal flow (hazard ratio, 3.35; 95% CI, 1.97 to 5.69; P<0.001).

Conclusions—No reflow after primary PCI was associated with reduced myocardial salvage, larger infarct size, worse left ventricular ejection fraction at 6 months, and increased risk of 1-year mortality. In 4 of 5 patients with no reflow after PCI, restoration of normal flow occurred 6 months after reperfusion. (Circ Cardiovasc Interv. 2010;3:27-33.)

Key Words: mortality ■ myocardial infarction ■ no reflow ■ percutaneous coronary intervention ■ reperfusion

Although primary percutaneous coronary intervention (PCI) is the most advantageous and rewarding reperfusion strategy available in patients with acute ST-segment–elevation myocardial infarction (STEMI), it fails to restore optimal myocardial reperfusion in a sizeable portion of patients, mostly because of no-reflow phenomenon. According to Kloner et al., no reflow is defined as suboptimal myocardial reperfusion through a part of coronary circulation without angiographic evidence of mechanical vessel obstruction. During the last 3 decades, multiple experimental and clinical studies have identified a number of predisposing factors of no-reflow phenomenon and have proposed an array of explanatory mechanisms and strategies to overcome it in the clinical setting. The impact of no reflow on the clinical outcome has been well documented. In clinical setting, however, several aspects of no-reflow phenomenon remain poorly understood. First, the clinical predictors of no reflow after primary PCI are only partially known. Second, the impact of no reflow on myocardial salvage by primary PCI in patients with STEMI has not been investigated in clinical setting. Third, the persistence in time of the no reflow after primary PCI and whether its persistence or resolution impacts left ventricular function are largely unknown.

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The aim of this study was 4-fold: (1) to investigate the relationship between no reflow after primary PCI and myocardial salvage quantified by 2 scintigraphic studies before and after PCI procedure; (2) to identify predictive factors of no reflow after PCI by including among other baseline characteristics, the time-to-treatment interval, scintigraphic area at risk, baseline angiographic data, and C-reactive protein; (3) to investigate the persistence or resolution of no reflow in the 6-month angiography; and (4) to assess the impact of persistence or resolution of no reflow in the...
6-month angiography on the left ventricular function and the impact of no reflow on the 1-year mortality.

Methods

Patients

Between January 1998 and December 2007, 1518 patients with STEMI presenting within 24 hours from the symptom onset were treated with primary PCI in the Deutsches Herzcentrum Munich. Patients undergoing thrombolysis or coronary artery bypass surgery were not included. Patients with mechanical failures (n = 28 patients), patients with missing scintigraphic examinations (n = 316 patients; 174 patients missing baseline scintigraphic study and 142 patients missing scintigraphic study performed 7 to 14 days after intervention), and those with angiograms of inadequate quality (n = 34 patients) were excluded. Thus, this study included 1140 patients with STEMI who underwent successful primary PCI and had paired scintigraphic examinations (before intervention and 7 to 14 days thereafter). The diagnosis of STEMI was established in the presence of chest pain lasting for >20 minutes associated with electrocardiographic changes (ST-segment elevation of ≥1 mm in at least 2 extremities; electrocardiographic leads or ≥2 mm in at least 2 contiguous precordial leads or left bundle branch block of new onset). The diagnosis was confirmed by coronary angiography in all patients. Patients with a mechanical failure to open the occluded coronary arteries were not included in this study.

Angiographic Evaluation and Diagnosis of No Reflow

Angiographic criteria were used for the diagnosis of no reflow.

Coronary angiography was performed according to the standard criteria. Offline analysis of digital angiograms was performed in the core laboratory using an automated edge detection system (CMS, Medis Medical Imaging Systems, Neuen, The Netherlands). The initial and postprocedural blood flow in the infarct-related artery was graded according to the Thrombolysis in Myocardial Infarction (TIMI) grading system. Severity of heart failure was assessed according to the Killip classification. Global left ventricular ejection fraction (LVEF) was determined by using the area-length method. The diagnosis of no reflow required the following criteria: (1) angiographic evidence of reopening of occluded coronary artery and successful stent placement with no evidence of flow-limiting residual stenosis (<50%), dissection, spasm, or apparent thrombus and (2) angiographic documentation of a TIMI flow grade ≤2, at least 10 minutes after the end of PCI procedure.

Primary PCI (>80% with stent implantation) and periprocedural care were performed according to the standard criteria. Bare metal stents were mostly used. Postinterventional antplatelet therapy consisted of ticlopidine (500 mg/d) or clopidogrel (300 mg or 600 mg as a loading dose followed by 75 mg/d for at least 4 weeks to 6 months) and aspirin (200 mg/d administered orally and continued indefinitely). Other cardiac medications were left at the discretion of the treating physician.

Scintigraphic Study

Before initiation of the PCI procedure, patients received an intravenous injection of 27 mCi (1000 MBq) of 99mTc-sestamibi. Single-photon emission computed tomography was performed within 6 to 8 hours after the injection of radioactive agent. A follow-up myocardial scintigraphy was scheduled 7 to 14 days after the PCI procedure.

A multichannel camera system equipped with low-energy and high-resolution collimators was used for myocardial imaging. Images were acquired in a 64×64 matrix by an acquisition time of 40 seconds per image. With dedicated software (ICON, version 6.0.2) transaxial slices were reconstructed. A volumetric sampling tool was applied to create polar maps of relative distribution throughout the entire left ventricle. Each polar map was normalized to its individual maximum. The defect size was defined as <50% uptake area. Four parameters were obtained using paired scintographic examinations: initial perfusion defect (perfusion defect in the initial scintigraphy), final infarct size (perfusion defect in the follow-up scintigraphy), absolute salvage (initial perfusion defect minus final infarct size), and salvage index (initial perfusion defect minus final infarct size divided by initial perfusion defect). The first 3 parameters were expressed as percentage of the left ventricle. The fourth parameter denotes the proportion of the initial perfusion defect salvaged by reperfusion (therapeutic and spontaneously occurring). All measurements were performed in the scintigraphic core laboratory by investigators unaware of clinical diagnosis.

End Points and Follow-Up

The outcomes of this analysis were myocardial salvage, factors predisposing for no reflow, LVEF at 6 months, and 1-year mortality. Occurrence of myocardial infarction (MI), stroke, and the need for revascularization within the first year after primary PCI were also assessed. As a standard practice in our institution, all patients were scheduled to undergo coronary angiography 6 months after the procedure or whenever they showed symptoms or signs of myocardial ischemia. Furthermore, patients were advised to contact our outpatient clinic or the hospital for any new symptoms or other cardiac symptoms. The follow-up information was obtained by a phone call at 30 days, a visit at 6 months, and another phone call at 1 year; patients who had cardiac complaints underwent a complete clinical, electrocardiographic, and laboratory evaluation. Information about death was obtained from hospital records, death certificates, or phone contact with relatives of the patient or attending physician. The follow-up information was obtained by personnel blinded to the clinical characteristics of the patients.

Statistical Analysis

Data are presented as median (interquartile range), mean±SD, or n (%). The distribution of the data was analyzed with one-sample Kolmogorov-Smirnov test. Categorical data were compared with χ² test. Continuous data were compared with Kruskal-Wallis rank-sum test or 2-tailed unpaired t test. Univariable and multivariable logistic regression models were used to identify the correlates of the no reflow after primary PCI. All variables of Tables 1 and 2 were entered into the univariable logistic regression; based on the variables that resulted significant (P < 0.05) from univariable logistic regression, a multivariable logistic regression model was constructed using the backward variable selection method. A multiple linear regression model was used to test the association between the no reflow and infarct size on the 7 to 14 days single photon emission computed tomography. All variables of Tables 1 and 2 with no reflow were entered into the model. One-year mortality was estimated by applying the Kaplan–Meier method. Univariable and multivariable Cox proportional hazards models were used to assess the association between no reflow and 1-year mortality enabling calculation of unadjusted and adjusted hazard ratios. All variables of Tables 1 and 2 with no reflow were entered into the univariable model; based on the variables that resulted significant (P < 0.05) from univariable Cox proportional hazards model, a multivariable Cox proportional hazards model was constructed using the backward variable selection method. A multiple linear regression model was used to test the association between no reflow and infarct size on the 7 to 14 days single photon emission computed tomography. All variables of Tables 1 and 2 with no reflow were entered into the model. The follow-up information was obtained by personnel blinded to the clinical characteristics of the patients.

Results

Baseline Characteristics

At the end of primary PCI procedure, 1032 patients had a TIMI flow grade 3 (group with normal epicardial flow) and 108 patients had a TIMI flow grade ≤2 (group with no reflow). Baseline characteristics of the patients are shown in Table 1. Patients with no reflow were older, had more previous infarctions, and had higher Killip class, higher activity of creatine kinase myocardial band, higher concentrations of serum creatinine and C-reactive protein, and
longer time-to-treatment intervals than patients with normal flow after primary PCI. The proportion of current smokers was smaller among patients of the no-reflow group. Baseline angiographic characteristics are shown in Table 2. Patients of the no-reflow group had more reduced LVEF and less often residual anterograde epicardial flow in the infarct-related artery than the group with normal flow.

Predictors of No Reflow Phenomenon

Univariable and multivariable logistic regressions were used to identify predictors of no reflow after primary PCI (see Methods section for variables entered into the models). In univariable analysis, 10 variables (age, smoking, previous MI, Killip class, serum creatinine, C-reactive protein, time-to-treatment interval, LVEF, baseline TIMI flow grade, and initial perfusion defect) were identified as predictors of no-reflow phenomenon. After application of backward variable selection method, 4 variables (previous MI, C-reactive protein, baseline TIMI flow grade, and initial perfusion defect) remained as significant independent predictors of no reflow after primary PCI. Results are shown in Table 3.

Scintigraphic Infarct Size and Myocardial Salvage

Compared with patients with normal flow restoration, patients with no reflow had significantly larger initial perfusion defect (26.0% [18.5%, 52.5%] of the left ventricle versus 24.0% [14.0%, 40.0%] of the left ventricle; \( P = 0.008 \)) and perfusion defect at 7 to 14 days (18.9% [10.3%, 33.8%] of the...
left ventricle versus 9.1% [3.0%, 20.9%] of the left ventricle; \( P < 0.001 \). The absolute amount of salvaged myocardium (9.8% [5.3%, 16.3%] of the left ventricle versus 12.0% [5.0%, 21.0%] of the left ventricle; \( P = 0.04 \)) was significantly smaller in patients with no reflow than in patients with normal flow. Salvage index or proportion of initial area at risk salvaged was significantly lower in patients with no reflow versus those with normal flow after PCI (0.34 [0.15, 0.49] versus 0.55 [0.29, 0.81], \( P < 0.001 \); Figure 1).

Multiple linear regression model was used to test the association between no reflow and infarct size on the 7 to 14 days single photon emission computed tomography (see Methods section for variables entered into the model). The model identified initial perfusion defect (\( P < 0.001 \)), no reflow (\( P < 0.001 \)), arterial hypertension (\( P = 0.015 \)), C-reactive protein (\( P = 0.005 \), female sex (\( P = 0.007 \), inverse association), and baseline LVEF (\( P < 0.001 \), inverse association) as independent correlates of a larger infarct size.

### Flow Status and Left Ventricular Function in the Follow-Up Angiography

Follow-up angiography was performed in 76 patients (70.4%) of the no-reflow group and 867 patients (84.0%) of the normal flow group after 194.3 (171.2, 215.9) days. Among 76 patients of the group with no reflow, TIMI flow grade was improved to TIMI flow grade 3 in 61 of them (80.3%). Fifteen patients (19.7%) from this group continued to have suboptimal TIMI flow in the follow-up angiography. Among 867 patients of the group with normal blood flow who underwent follow-up angiography, TIMI flow grade 3 was found in 761 (87.8%). In 106 patients (12.2%), TIMI flow grade was found to be suboptimal (TIMI grade \( \leq 2 \)) in the follow-up angiography (Figure 2).

Patients with no reflow had worse LVEF compared with patients with normal flow after PCI in the follow-up angiography (47.7 \pm 13.1\% versus 54.2 \pm 13.9\%; \( P < 0.001 \)). \( \Delta \)LVEF or LVEF at follow-up angiography minus baseline LVEF was 0.6 \pm 10.8\% in the group with no reflow versus 4.3 \pm 12.4\% in the group with normal flow after primary PCI (\( P = 0.005 \)). Among patients in the group with no reflow, those who showed an improvement in the TIMI flow grade in the follow-up angiography (61 of 76 patients with no reflow and 6 months angiography) showed also an improvement in the LVEF (\( \Delta \)LVEF = 2.1 \pm 10.9\%). Conversely, patients with persistence of suboptimal blood in the follow-up angiography (15 of 76 patients with no reflow and 6 months angiographic data) showed a worsening of LVEF (\( \Delta \)LVEF = -8.9 \pm 10.8\%; \( P = 0.002 \)) compared with the subgroup with improved blood flow; Figure 3).

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**Table 3. Predictors of No Reflow Phenomenon Obtained From Univariable and Multivariable Logistic Regression Models**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Unadjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous MI</td>
<td>1.61 (1.02 to 2.73)</td>
<td>2.17 (1.18 to 3.99)</td>
</tr>
<tr>
<td>C-reactive protein (for 1-mg/L increase)</td>
<td>1.02 (1.01 to 1.03)</td>
<td>1.02 (1.01 to 1.04)</td>
</tr>
<tr>
<td>Baseline TIMI flow grade (for 1-grade decrease)</td>
<td>1.98 (1.52 to 2.57)</td>
<td>2.02 (1.47 to 2.76)</td>
</tr>
<tr>
<td>Initial perfusion defect (for 5% of the LV increase)</td>
<td>1.10 (1.04 to 1.16)</td>
<td>1.07 (1.01 to 1.13)</td>
</tr>
</tbody>
</table>

LV indicates left ventricle; OR, odds ratio.

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**Figure 1.** Salvage index or proportion of initial area at risk salvaged by primary PCI in patients with normal flow or no reflow after PCI. Median values with 25th to 75th and 5th to 95th percentiles are shown.

**Figure 2.** Blood flow status immediately (upper part) and 6 months after (lower part) primary PCI.

**Figure 3.** Difference between the LVEF at 6 months with the baseline LVEF (mean \pm SEM). A, Between patients with normal flow and no reflow after PCI. B, Between patients with no reflow after primary PCI who showed normalization of TIMI flow in the 6-month and those with persistence of suboptimal blood flow in the 6-month angiography.
Impact of No Reflow on 1-Year Clinical Outcome

Within the first year after primary PCI, 72 patients died (6.3%). Eighteen deaths occurred among patients of the no-reflow group and 56 deaths occurred among patients with normal after primary PCI (Kaplan–Meier estimates of 1-year mortality, 16.7% and 5.5%, respectively; hazard ratio, 3.35 [95% CI, 1.97 to 5.69], P<0.001; Figure 4). Recurrent MI occurred in 6 patients of the no-reflow group versus 34 patients of the normal flow group (3.3% versus 5.6%; P=0.22). Stroke occurred in 2 patients of no-reflow group versus 11 patients of normal flow group (1.8% versus 1.1%; P=0.46). Death, MI, or stroke occurred in 22 patients of the no-reflow group versus 89 patients of the normal flow group (20.4% versus 8.6%; P<0.001). Target lesion revascularization was required in 23 patients of the no-reflow group versus 202 patients of the normal flow group (21.3% versus 19.6%; P=0.67). Major adverse cardiac and cerebrovascular events occurred in 40 patients of the no-reflow group versus 265 patients of the normal flow group (37.0% versus 25.7%; P=0.011).

Univariable and multivariable Cox proportional hazards models were used to test the association between no reflow and 1-year mortality (see Methods section for variables entered into the models). The univariable correlates (P<0.05) of 1-year mortality were no reflow, age, diabetes, previous MI, systolic blood pressure, Killip class, creatinine level, C-reactive protein, LVEF, multivessel disease, infarct-related artery, and initial perfusion defect. After application of backward variable selection method, 5 variables (no reflow, age, diabetes, Killip class, and creatinine level) remained as independent predictors of mortality during the first year (Table 4).

### Table 4. Predictors of Mortality During the First Year Obtained From Univariable and Multivariable Cox Proportional Hazards Models

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Unadjusted HR (95% CI)</th>
<th>Adjusted HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No reflow</td>
<td>3.35 (1.97 to 5.69)</td>
<td>1.91 (1.11 to 3.30)</td>
</tr>
<tr>
<td>Age (for 10-year increase)</td>
<td>2.06 (1.67 to 2.55)</td>
<td>1.85 (1.49 to 2.28)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.53 (1.58 to 4.04)</td>
<td>1.81 (1.11 to 2.94)</td>
</tr>
<tr>
<td>Killip class (for 1-class increase)</td>
<td>2.76 (2.30 to 3.30)</td>
<td>2.38 (1.97 to 2.88)</td>
</tr>
<tr>
<td>Creatinine (for 1-mg/dL increase)</td>
<td>2.19 (1.77 to 2.71)</td>
<td>1.80 (1.38 to 2.35)</td>
</tr>
</tbody>
</table>

LV indicates left ventricle; HR, hazard ratio.

PCI, and the diagnosis of the no reflow was based on the coronary angiography which is considered as the gold standard for the detection of this condition in clinical setting. Moreover, myocardial salvage and LV function were assessed by using the most reliable tools of the estimation of the infarct size12 or left ventricular function11 in clinical realm.

The main findings of this study can be summarized as follows:

1. The development of no-reflow phenomenon was associated with considerable reduction of the myocardial salvage by primary PCI in patients with STEMI. Because reduced myocardial salvage results in larger myocardial necrosis,13,14 this could be the principal mechanism by which no reflow influences left ventricular function and subsequent mortality.
2. The lack of residual blood flow in the infarct-related artery, large infarct size, previous MI, and elevated level of C-reactive protein at baseline are independent predictors of the development of no reflow after primary PCI.
3. In 4 of 5 patients with no reflow, normalization of blood flow occurred 6 months after primary PCI.
4. Patients with no reflow after primary PCI who showed normalization of blood flow in the 6-month angiography showed also a significantly better left ventricular function than patients in whom suboptimal blood flow persisted 6 months after primary PCI.

The relationship between development of no reflow and the amount of myocardium to be salvaged by reperfusion or whether no reflow per se contributes to myocardial necrosis is still poorly understood. This study showed that the proportion of initial area at risk or amount of myocardium salvaged by primary PCI was markedly reduced in patients with no reflow compared with patients with normal flow restoration by primary PCI. Because the paired scintigraphic studies were performed with, at least, 1 week in between, the myocardial cell death was less curbed by reperfusion in patients with no reflow than in those with normal flow restoration by primary PCI. This resulted in less myocardial salvage and greater infarct size in patients with no reflow. Two putative mechanisms may be proposed to explain less stoppable continuation of myocardial necrosis in patients with no reflow. First, persistence of microvascular obstruction—the key mechanism of no-reflow phenomenon—ham-
pers tissue perfusion and enables continuation of tissue ischemia and progressive cell loss. Experimental studies in rats and clinical studies have demonstrated that compromised tissue perfusion persists 4 weeks after reperfusion. The finding of an association between the presence of no-reflow phenomenon and a larger infarct size on the 7 to 14 days single photon emission computed tomography, independent of other clinical variables, seems to provide support for the proposed mechanism. Second, restoration of less than optimal blood flow may favor reperfusion injury, supposed to play an important role in the genesis of no reflow itself, which may further promote cell death.

The finding that initial area at risk was an independent correlate of no reflow corroborates previous studies that have demonstrated a close correlation between infarct size and the size of anatomic no reflow. Because local necrosis is associated with tissue destruction, including vascular tissue, edema, and mechanical compression which constitute potential mechanisms of no reflow, the association between the infarct size and no reflow is explainable. Several mechanisms may explain the protective role of residual blood flow in the infarct-related artery before reperfusion with regard to development of no reflow. First, residual blood flow in the infarct-related artery is associated with reduced infarct size. Second, infarct-related arteries prone to spontaneous recanalization may have less thrombotic burden by meaning less distal embolization, which is considered a crucial factor for the development of no reflow after PCI. Third, early restoration of blood flow may alleviate tissue ischemia and prevent or attenuate the full-blown microvascular damage. Interestingly, we did not find an independent association between time-to-treatment and no reflow after primary PCI. Although the exact mechanism cannot be offered, there is a possibility that the association between time-to-treatment interval and no reflow may be overridden by the powerful association between infarct size and no reflow.

By showing an independent association between baseline C-reactive protein level and no reflow, our study suggests that baseline inflammation may increase the risk of no reflow after primary PCI. One mechanism by which higher levels of C-reactive protein promote development of no reflow may involve suggestion that elevated level of C-reactive protein may increase infarct size by activating the complement cascade in the ischemic/necrotic tissue. Additionally, elevated level of C-reactive protein or other inflammatory molecules may promote microvascular obstruction through an array of mechanisms. However, the association between baseline inflammation and development of no reflow remains controversial.

Finally, our study showed that 4 of 5 patients with no reflow showed normalization of the blood flow 6 months after PCI. This finding could be of clinical importance. Although patients with no reflow after PCI had worse left ventricular function at 6 months than patients with normal flow restoration, a marked deterioration in the left ventricular function occurred only in patients in whom suboptimal blood flow persisted. Otherwise, resolution of no reflow seems to protect against the negative remodeling of left ventricle within the 6-month period. These findings seem to concur with experimental data showing infarct expansion in animals with persistence of compromised tissue perfusion 4 weeks after reperfusion.

In conclusion, the development of no reflow in patients with STEMI after primary PCI was associated with significant reduction in myocardial salvage by primary PCI, larger scintigraphic infarct size, worse LVEF at 6 months, and increased risk of 1-year mortality. The extent of initial area at risk, the lack of residual blood flow in the infarct-related artery, previous MI, and elevated levels of C-reactive protein were independent correlates of no reflow. In 4 of 5 patients with no reflow after PCI, normalization of blood flow occurred within the 6 months after reperfusion. Persistence of compromised tissue perfusion was associated with worse left ventricular function compared with patients in whom normal blood flow was restored within the 6 months after PCI.

Disclosures
None.

References


**CLINICAL PERSPECTIVE**

No-reflow phenomenon affects 5% to 50% of the patients with acute ST-segment–elevation myocardial infarction undergoing reperfusion therapy. Evidence available suggests that no reflow negates the benefit of reperfusion therapy, promotes subsequent negative remodeling of the left ventricle, and worsens the prognosis of patients with ST-segment–elevation myocardial infarction. This study reported a frequency of no reflow of 9.5% after primary percutaneous coronary intervention (PCI). The lack of residual blood flow in the infarct-related artery, large initial area at risk (quantified with scintigraphy), elevated C-reactive protein level, and previous myocardial infarction were identified as independent correlates of no reflow after primary PCI. There was an intricate relationship between infarct size and no reflow in which larger initial perfusion defects (areas at risk) were associated with more frequent no reflow phenomenon which on its side further increased the infarct size by reducing the salvaging capacity of primary PCI. We found that for every 5% of the left ventricle increase in the initial perfusion defect, the adjusted risk of no reflow was increased by 7%. Patients with no reflow had significantly more reduced left ventricular ejection function 6 months after acute event. By 6 months, coronary angiography showed that normal flow has been restored in 80% of patients with no reflow. No-reflow resolution was associated with better left ventricular ejection fraction compared with patients in whom no reflow persisted by 6 months. No reflow was associated with an ∼3-fold increase in the adjusted risk of death within the first year after primary PCI. These findings provide mechanistic information on the harmful effects of no-reflow phenomenon and increase the awareness to better diagnose and treat it in patients with ST-segment–elevation myocardial infarction undergoing primary PCI.
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