Effect of Distal Embolization on Myocardial Perfusion Reserve After Percutaneous Coronary Intervention
A Quantitative Magnetic Resonance Perfusion Study

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Background—Studies have shown that a subset of patients demonstrate persistent impairment in microcirculatory function after percutaneous coronary intervention (PCI). Distal embolization of plaque contents has been postulated as the main mechanism for this. We sought to investigate this further by evaluating PCI-induced changes in myocardial perfusion reserve index (MPRI) over time in segments with “distal-type” procedure-related myonecrosis using high-resolution quantitative cardiovascular magnetic resonance imaging.

Methods and Results—Forty patients undergoing PCI were studied with pre-PCI and 24-hour post-PCI delayed-enhancement magnetic resonance imaging and first-pass perfusion magnetic resonance imaging at rest and stress. Twenty patients underwent a third magnetic resonance imaging scan at 6 months. For perfusion imaging, 3 short-axis images were acquired during every heartbeat with a T1-weighted turboFLASH sequence. MPRI was calculated as the ratio of hyperemic to resting myocardial blood flow and subdivided according to the presence and location of new delayed hyperenhancement. Twenty-one patients demonstrated new distal hyperenhancement after PCI. Mean MPRI in revascularized myocardial segments not demonstrating new HE was significantly increased after the procedure (2.06 [95% CI, 1.99 to 2.13] before PCI and 2.52 [95% CI, 2.42 to 2.62] after PCI; P<0.001). In contrast, MPRI in segments with distal hyperenhancement was reduced after PCI (2.16 [95% CI, 1.95 to 2.37] before PCI; 2.00 [95% CI, 1.82 to 2.19] after PCI; mixed-model \( z = -4.82; P<0.001 \)). Changes in mean MPRI 24 hours after PCI in segments upstream to new injury were not significantly different compared with perfusion changes in remote myocardium (\( z = -0.68; P=0.50 \)). At 6 months after the procedure, mean MPRI in segments with new injury improved significantly compared with MPRI measured in these segments at 24 hours after PCI.

Conclusions—MPRI is reduced in myocardial segments that demonstrate new distal irreversible injury at 24 hours after PCI. These reductions are confined to the segments with injury and do not affect the entire supply territory of the culprit vessel. (Circulation. 2007;116:1458-1464.)

Key Words: coronary disease ■ embolism ■ magnetic resonance imaging ■ percutaneous coronary intervention ■ perfusion ■ stents

Percutaneous coronary intervention (PCI) results in enlarged luminal cross-sectional area and improved myocardial blood flow (MBF). However, despite most patients demonstrating improvement in coronary vasodilatory reserve (as assessed by intracoronary Doppler\(^1\)) and myocardial perfusion reserve index (MPRI; assessed by cardiovascular magnetic resonance imaging\(^2\)) after coronary stenting, a minority demonstrate persistent impairment in microcirculatory function after PCI, even after substantial conduit area enlargement. Several potential mechanisms have been suggested for this observation, including persistently elevated basal blood flow after transient ischemia, microvascular stunning resulting from particulate embolization, or acute/chronic impairment of the microvascular circulatory response.

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Cardiovascular magnetic resonance imaging (CMR) permits assessment of both myocardial scar and myocardial...
perfusion concurrently with high spatial resolution. CMR during first pass of an injected tracer permits assessment of myocardial perfusion both at rest and during pharmacological stress and gives superior spatial resolution compared with nuclear imaging methods. CMR perfusion results from animal experiments have shown a strong correlation with microspheres for the assessment of blood flow, and we have recently used this technique to report resting MBF in patients with hibernating myocardium. Delayed-enhancement MRI (DE-MRI), initially validated in large-animal models, allows assessment of the transmural extent of irreversible injury and is superior to single-photon–emission computed tomography for the identification of subendocardial myocardial infarction. It permits precise quantification of even small areas of myocardial necrosis after surgical or percutaneous revascularization. In the setting of PCI, 2 distinct patterns of new myocardial necrosis have been observed (distal and adjacent-to-stent injury), with distal embolization and ischemia caused by side-branch occlusion speculated to be possible mechanisms.

In the present study, we evaluated PCI-induced changes in MPRI and procedure-related myonecrosis using high-resolution quantitative CMR. We prospectively quantified resting and stress MBF before and after PCI using perfusion CMR and compared it with MBF in remote normal myocardium. We then compared the MPRI changes in myocardial segments with and without PCI-induced myonecrosis. We hypothesized that MPRI would be impaired in myocardial segments with new “distal” PCI-induced injury (ie, new hyperenhancement (HE) occurring in a distal distribution). We also speculated that myocardial segments “upstream” to the injury would not demonstrate evidence of microvascular dysfunction after the procedure.

Methods

Ethics

The study was approved by our institutional ethics committee. Informed written consent was obtained from each patient.

Patient Population and CMR Imaging Time Points

Sixty-eight consecutive patients with either 1- or 2-vessel coronary artery disease who were scheduled for complex PCI (30 mm of stent to a single vessel or treatment of a segment that involved at least 1 major side branch >2.0 mm and patients undergoing planned 2-vessel PCI) were enrolled in the study. We excluded patients with a clinical history of myocardial infarction, chronic total occlusion, and typical MRI or adenosine contraindications. In the present study, we analyzed 44 consecutive patients who underwent both the initial CMR scan (consisting of cine, rest/stress perfusion, and DE-MRI) 24 hours before PCI and repeat CMR (cine, rest/stress perfusion, and DE-MRI) imaging 24 hours after PCI who did not have preexisting HE in the initial CMR scan (Figure 1). Twenty patients in this study cohort were included in previous studies comparing troponin I release after PCI and new myocardial HE. A randomly selected group of 20 patients (45%) of the study cohort were reimaged at 6 months.

CMR Protocol

Patients were studied in a 1.5-T clinical MR scanner (Siemens Sonata, Erlangen, Germany), and steady-state free-precession cine images were acquired in 2 long-axis and 7 to 9 short-axis views, as previously described. A gadolinium-based contrast agent (Gadodiamide, Omniscan, Nycomed Amersham, Little Chalfont, Buckinghamshire, UK) was then administered intravenously at a dose of 0.04 mmol/kg body weight (injection rate, 6 mL/s), followed by a saline flush of 15 mL at the same rate, for both stress and rest imaging. Perfusion imaging was performed every heartbeat during first pass using a T1-weighted fast (spoiled) gradient-echo sequence in 3 short-axis imaging planes representing the basal, midventricular, and apical myocardial segments according to recommended guidelines. Perfusion pulse sequence parameters were as follows: image acquisition time, 180 ms; echo time, 0.99 ms; matrix, 128×80; and slice thickness, 8 mm. The typical voxel size was 2.9×2.3×8 mm. A nonselective saturation recovery pulse was used with a delay of 10 ms. All slices were imaged during each heartbeat for a total of 50 heartbeats. An initial perfusion scan was performed during maximal vasodilation, followed by a second scan ~15 minutes later during rest. Vasodilation was induced by intravenous infusion of adenosine 0.14 mg · kg⁻¹ · min⁻¹ for 3 minutes before start of the scan. The adenosine infusion was continued until acquisition of the first 10 to 15 images. After rest perfusion imaging, we gave a “top-up” dose of 0.045 mmol/kg for a total gadolinium-DTPA dose of 0.125 mmol/kg before DE imaging. The DE images were acquired after an 8-minute delay with the use of an inversion-recovery segmented gradient-echo sequence as previously described.
CMR Postprocessing and Data Analysis

For perfusion analysis, the endocardial and epicardial contours were traced by an examiner blinded to angiography (MRI-MASS, Medical Imaging Solutions, Leiden, the Netherlands) and corrected manually for displacements (eg, breathing). The myocardium was divided into 24 corresponding segments; in each segment, the MBF was determined (in mL · min⁻¹ · g⁻¹) by deconvolution of the signal intensity curves with an arterial input function measured in the left ventricular blood pool, with explicit accounting for any delay in the arrival of the tracer. Because basal MBF is closely related to the rate-pressure product, an index of left ventricular oxygen consumption, values for rest flow in each patient also were corrected for the respective rate-pressure product. MPRI was calculated as the ratio of MBF during hyperemia to rest. DE after processing has been described previously. Hyperenhanced pixels were defined as those with image intensities ≥2 SD above the mean of image intensities in a remote myocardial region in the same image. Furthermore, the transmural extent of infarction in each myocardial segment was calculated by dividing the hyperenhanced area by the total area of the segment and scored with the following system: no HE, grade 0; 1% to 25% HE, grade 1; 26% to 50% HE, grade 2; 51% to 75% HE, grade 3; and 76% to 100% HE, grade 4. The diagnostic coronary angiogram that segment and scored with the following system: no HE, grade 0; 1% to 25% HE, grade 1; 26% to 50% HE, grade 2; 51% to 75% HE, grade 3; and 76% to 100% HE, grade 4. The diagnostic coronary angiogram was used as the gold standard in defining affected myocardial segments. Each of the 3 (basal, midventricular, and apical) short-axis slices was ascribed a coronary artery territory according to standard criteria.

Statistical Analysis

Values are expressed as mean (95% CI or SD) or median (interquartile range) as appropriate. Using recorded MPRI or MBF in each segment as the outcome, we fit a linear mixed model with fixed effects for slice and segment as categorical variables and random intercepts, with an independent covariance structure. In all models looking at the change in MPRI or MBF at different time points as the outcome, we adjusted further for the baseline value. Models were fit using the maximum likelihood estimation to account for the missing values, assuming that these data are missing at random. Segment was not added as a second random level because there was only a single observation per segment nested within slice nested within patient; thus, there was no within-segment (nested within slice/patient) variation. We decided that slice would be fitted as a fixed effect as opposed to a random effect because we were interested only in making inference to the 3 slices used in this study. In fact, we repeated the analysis with patient and slice as random effects, which did not affect the results. The normal error distribution of the predicted random intercept models was assessed graphically (graphs not shown). Statistical significance was taken throughout at the 5% level (P<0.05). All analyses were carried out with Stata Version 9.0 (Stata Corp, College Station, Tex).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Patient Characteristics and Follow-Up

The mean age of the study patients was 62±11 years; 31 (70%) were male. Diagnostic cardiac MRI data were available for 40 of the 44 patients (91%) who underwent stress/rest perfusion imaging both before and 24 hours after PCI (Figure 1). Regarding the cases of incomplete imaging data, 2 patients were unwilling to undergo adenosine infusion, and in 2 patients, the section orientations at rest and during stress did not match sufficiently for comparison because of patient movement. During adenosine-induced stress, the subjects’ mean heart rate increased from 61±10 to 78±9 bpm. Mean systolic blood pressure was 129±22 mm Hg at rest and 121±17 mm Hg during adenosine-induced stress. No arrhythmias or other marked side effects were observed during stress.

Comparison of Changes in MPRI 24 Hours After Intervention Between Affected and Unaffected Segments

Across all 40 patients (848 segments), 769 segments (91%) could be analyzed. Of the 769 total myocardial segments analyzed, 404 segments (53%) were in territories subtended by a severely diseased coronary vessel (called affected), and 365 (48%) were supplied by nondiseased coronary arteries (called unaffected). When all patients and all segments were considered, the mean MPRI was 2.08 (95% CI, 2.01 to 2.15) in segments with and 2.49 (95% CI, 2.39 to 2.58) in segments without significant coronary stenosis before PCI (mixed-model z=-0.26; P<0.001). After the procedure, the mean MPRI was 2.41 (95% CI, 2.32 to 2.50) in revascularized segments and 2.58 (95% CI, 2.48 to 2.69) in unaffected segments (z=-3.35; P=0.001).

Comparison of Changes in MPRI Between Myocardial Segments With and Without Post-PCI Injury

When preprocedural and postprocedural DE-MRIs were compared, 25 patients (63%) had evidence of new myocardial HE. Of these, 21 patients (84%) had distal HE (as previously defined) and are included in the final analysis. In the distal HE group, the mean mass of HE per patient was 5.1±3.0 g. We evaluated the levels of MBF before and early after intervention in only those segments that exhibited new distal HE after the procedure (HE group). Eighty-two of 404 (20%) intervened myocardial segments demonstrated new irreversible injury after revascularization. Mean MPRI in such segments was 2.16 (95% CI, 1.95 to 2.37) before PCI and dropped to 2.00 (95% CI, 1.82 to 2.19) after PCI (z=-2.07; P=0.039). Figure 2 shows an example of MPRI changes in a patient with new distal HE. In contrast, mean MPRI across all patients in revascularized myocardial segments not demonstrating new irreversible injury (no HE) was significantly increased after the procedure (2.06 [95% CI, 1.99 to 2.13] before PCI and 2.52 [95% CI, 2.42 to 2.62] after PCI; z=8.73; P<0.001). When the 2 groups (HE and no HE) were compared, the absolute change in MPRI before and after the procedure was significantly different in the non-HE segments (0.46; 95% CI, 0.36 to 0.55) from the HE segments (−0.16; 95% CI, −0.29 to 0.02; z=-4.82; P<0.001; Table 1, first section).

This relationship also held true when the MPRI changes were compared in only the 21 patients with new distal HE (Table 1, second section). After the procedure, there was a drop in MPRI in segments with HE (−0.16; 95% CI, −0.29...
Comparison of MPRI Changes 24 Hours After PCI Between Segments With New Distal Injury and Subgroups of Segments Without Injury

We assessed MPRI in myocardial segments upstream to procedural injury (HE) and compared them with MPRI in remote segments (segments that underwent PCI in a second vessel in the same patient but not displaying new injury) and segments with no PCI (segments that were subtended by arteries that did not undergo PCI) (Table 1, third section). Changes in mean MPRI after PCI in segments upstream to new injury were not significantly different compared with perfusion changes in remote myocardium ($z = 0.68$; $P = 0.50$), indicating that there was no significant reduction in perfusion reserve in upstream segments after the procedure. When these MPRI changes were compared with the myocardial segments (in the same patient and vessel) that exhibited new HE after PCI, there was a significant difference in the change in MPRI ($z = 4.88$; $P < 0.001$).

Comparison of MPRI Changes 6 Months After PCI Between Segments With New Distal Injury and Subgroups of Segments Without Injury

Of 40 patients in the study cohort, 20 (50%) were rescanned at 6 months after the procedure. At 6 months, mean MPRI in segments with new injury was 2.52 (95% CI, 2.24 to 2.79) compared with 2.04 (95% CI, 1.86 to 2.23) 24 hours after PCI and 2.64 (95% CI, 2.32 to 2.96) before PCI. The initial deterioration of MPRI in segments with new injury observed early after PCI was no longer evident after 6 months, and the change in MPRI for segments with new injury was no longer statistically significant ($z = 1.21$; $P = 0.23$). Overall, the

to 0.02) in contrast to an increase in MPRI in segments without evidence of new HE (0.17; 95% CI, 0.04 to 0.29), and the difference between the 2 groups was significant ($z = 6.85$; $P < 0.001$).

Table 1. MPRI Changes Before and Early After PCI in Segments With and Without New Distal HE Across All Patients, Only in Patients With New Distal HE, and Subdivided According to Culprit Vessel Territory

<table>
<thead>
<tr>
<th></th>
<th>Segments, n</th>
<th>Pre-PCI MPRI</th>
<th>Post-PCI MPRI</th>
<th>Change After 24 h</th>
<th>$z$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected segments in all 40 patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No HE</td>
<td>322</td>
<td>2.06 (1.99–2.13)</td>
<td>2.52 (2.42–2.62)</td>
<td>0.46 (0.36–0.56)</td>
<td>4.82</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HE (distal)</td>
<td>82</td>
<td>2.16 (1.95–2.37)</td>
<td>2.00 (1.82–2.1)</td>
<td>–0.16 (–0.29––0.02)</td>
<td>4.88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Affected segments in 21 patients with distal new injury</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No HE</td>
<td>170</td>
<td>2.18 (2.07–2.30)</td>
<td>2.35 (2.22–2.47)</td>
<td>0.17 (0.04–0.29)</td>
<td>6.85</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HE (distal)</td>
<td>82</td>
<td>2.16 (1.95–2.37)</td>
<td>2.00 (1.82–2.19)</td>
<td>–0.16 (–0.29––0.02)</td>
<td>4.88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>All segments in 21 patients with distal new injury</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upstream (reference group)</td>
<td>141</td>
<td>2.18 (2.05–2.30)</td>
<td>2.31 (2.17–2.44)</td>
<td>0.13 (0.03–0.23)</td>
<td>…</td>
<td>…</td>
</tr>
<tr>
<td>Distal</td>
<td>70</td>
<td>2.22 (1.98–2.45)</td>
<td>2.05 (1.84–2.26)</td>
<td>–0.17 (–0.32––0.02)</td>
<td>4.88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Remote</td>
<td>90</td>
<td>2.22 (2.08–2.37)</td>
<td>2.43 (2.25–2.60)</td>
<td>0.20 (0.02–0.39)</td>
<td>0.68</td>
<td>0.50</td>
</tr>
<tr>
<td>No PCI</td>
<td>148</td>
<td>2.24 (2.08–2.39)</td>
<td>2.42 (2.31–2.54)</td>
<td>0.19 (0.04–0.33)</td>
<td>0.70</td>
<td>0.48</td>
</tr>
</tbody>
</table>

Upstream indicates myocardial segments supplied by the culprit vessel proximal to the distal HE; remote, segments that underwent PCI in a second vessel in the same patients but not displaying new injury; distal HE, segments demonstrating new distal injury; and no PCI, segments that are subtended by arteries that did not undergo PCI.
changes in MPRI between the segments with new distal HE and the corresponding upstream segments were not significantly different after 6 months ($z = -1.15; P = 0.25$; Figure 3).

Correlation of Magnitude of New Injury to MPRI
We excluded all segments with no myocardial injury and all segments with adjacent injury. In the remaining segments (distal HE group), the absolute percentages of new injury were measured for each segment. Values were categorized into the 4 predefined levels with the following boundaries: 0% to 25% (grade 1), 26% to 50% (grade 2), 51% to 75% (grade 3), and 76% to 100% (grade 4). Early after PCI, there were no segments in grade 4 (>76% necrosis); 53% of segments were in grade 1; 35% were in grade 2; and 12% were in grade 3. The new variable with the 3 valid categories was entered into the mixed model as a fixed effect with MPRI as the outcome. We could not measure any significant trend correlation between the absolute amount of necrosis and the difference in MPRI after 6 months ($r^2 = 0.12, P = 0.16$).

Changes in Rest and Stress MBF Immediately and 6 Months After PCI
Mean resting MBF did not differ across any of the groups before or after PCI (Table 2). Mean hyperemic MBF was reduced significantly in the segments with new HE immediately after PCI and increased at 6 months (Table 2).

Discussion
This study demonstrates that MPRI is reduced in myocardial segments that demonstrate new myonecrosis at 24 hours after PCI. Furthermore, although MPRI is reduced in such segments, it is not reduced in segments upstream from the injury (in the territory of the culprit vessel) compared with remote myocardial segments. Late after the procedure, we found a normalization of MPRI in myocardial segments that demonstrated new irreversible injury. Our findings have important implications for our understanding of the changes in the coronary microvasculature both early and late after percutaneous coronary intervention.

To the best of our knowledge, this is the first study to concurrently examine myocardial perfusion and necrosis serially after PCI with a validated, quantitative CMR technique. Previous studies using intracoronary Doppler in the setting of PCI have shown a residual reduction of relative coronary flow velocity reserve immediately after PCI (related to elevated baseline flow velocity),1,2,20–22 and a persistent reduction in relative coronary vasodilatory reserve has been associated with cardiac biomarker elevation after the procedure.2 Gibson et al23 demonstrated that patients with impaired Thrombolysis In Myocardial Infarction (TIMI) perfusion grade had a 10-fold-higher incidence of postprocedural creatine kinase-MB elevation. Bolognese et al24 found, using a combination of TIMI flow grade, corrected TIMI frame count, TIMI perfusion grade, and myocardial contrast echocardiography, that post-PCI cardiac troponin I elevation in high-risk patients with acute coronary syndrome is associated with an abnormal tissue-level perfusion. We and others have recently demonstrated that areas of new myonecrosis visualized with DE-MRI are linked to impaired TIMI perfusion grade on angiographic analysis immediately after the procedure.14,25 In the present study, we have extended this by finding that areas of myocardium injured during PCI exhibit reduced myocardial perfusion for at least 24 hours after the interventional procedure.

We found that myocardial MPRI at 24 hours in the segments with new HE was reduced as a result of a lack of increase in hyperemic blood flow rather than an increase in baseline (resting) blood flow. In contrast to experimental models of coronary embolization in which the particulate size is small26 (≈42 μm), coronary embolization after PCI of native arteries has been found to result in thrombotic and nonthrombotic embolic material ranging from 100 to 550 μm.27 It is likely that the blunted hyperemic flow response persisting at 24 hours seen in our study is a feature of both myocardial necrosis and extensive macrovascular and microvascular plugging of the distal vascular bed. Our findings are in agreement with those of Dupouy et al,28 who found 2

### Table 2. Rest and Stress MBF 24 Hours and 6 Months After PCI

<table>
<thead>
<tr>
<th>Segment Description</th>
<th>Rest MBF, mL · min⁻¹ · g⁻¹</th>
<th>Stress MBF, mL · min⁻¹ · g⁻¹</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before PCI (HE negative)</td>
<td>1.0 (0.9–1.1)</td>
<td>2.2 (2.0–2.4)</td>
<td>...</td>
</tr>
<tr>
<td>After PCI (HE positive)</td>
<td>1.0 (0.9–1.1)</td>
<td>2.2 (1.9–2.5)</td>
<td>...</td>
</tr>
<tr>
<td>Early after PCI (HE negative)</td>
<td>1.0 (0.9–1.2)</td>
<td>2.8 (2.3–3.3)</td>
<td>0.01*</td>
</tr>
<tr>
<td>Early after PCI (HE positive)</td>
<td>1.0 (0.9–1.2)</td>
<td>1.9 (1.7–2.1)</td>
<td>0.03*</td>
</tr>
<tr>
<td>Late after PCI (HE negative)</td>
<td>1.1 (0.9–1.3)</td>
<td>2.7 (2.3–3.0)</td>
<td>0.8†</td>
</tr>
<tr>
<td>Late after PCI (HE positive)</td>
<td>1.2 (0.9–1.4)</td>
<td>2.8 (2.3–3.3)</td>
<td>0.03†</td>
</tr>
</tbody>
</table>

*Compared with pre-PCI value; †compared with 24-hour post-PCI value.
patterns of postprocedural coronary flow velocity reserve impairment: The first related to a significantly higher baseline average peak velocities within 10 minutes after the last balloon inflation that normalized within 24 hours after PCI, and the second related to a lack of increase in hyperemic average peak velocity in the setting of a normal postprocedural baseline average peak velocity that persisted over the next 24 hours. In the present study, MPRI improved in the segments with new myonecrosis at 6 months. This was a result of improvement in hyperemic blood flow in these myocardial segments that, in turn, likely was due to a resolution in the microcirculatory impairment from distal embolization. Although all patients with new injury had evidence of scarred myocardium in the 6-month scan (data not shown), this did not adversely affect the MPRI in these segments because the total volume of scar (per segment) was small.

Using a sensitive, high-spatial-resolution technique, we did not find a reduction in MPRI in myocardial segments upstream of the new HE. This implies that detection of impaired antegrade flow is likely to be local to the territory affected by plaque embolization and that the no-reflow phenomenon does not affect the whole territory of the epicardial vessel. This is particularly likely to be the case when the volume of myocardial injury is small, as was the case in most of our cohort. Our findings also suggest that pharmacological therapy administered in the catheter laboratory for the no-reflow phenomenon may be of limited value because perfusion remains abnormal >24 hours after the PCI procedure. Indeed, it is possible to speculate that the apparent benefits of vasodilator therapy for no reflow may reflect dilatation of predominantly unaffected vascular beds with minimal impact on territories plugged with atheroembolic debris.

Our findings are in contrast to the preliminary CMR perfusion study of Al-Saadi et al., who found that although MPRI in segments after balloon angioplasty alone did not completely normalize in a subset of patients, the stented group demonstrated a complete normalization of MPRI 24 hours after the procedure. There are, however, important differences between this early study and our present study. First, the stented group was small (only 13 of 35 patients [37%]) in the former study. Second, we enrolled high-risk PCI patients in the present study with at least a 30% rate of new irreversible injury. Although the exact rate of procedure-related myonecrosis is not known in the study by Al Saadi et al (because they did not report on CMR or biochemical markers of myocardial injury), it is likely to be low/negligible because it was in a low-risk PCI group. Hence, the MPRI changes in the stented group in their study might reflect the MPRI changes found in the non-HE segments in our study (ie, significant increase 24 hours after PCI). Third, the measures of perfusion reserve indexes also are not directly comparable between these studies because the former used semiquantitative assessment of MBF and we used a quantitative CMR perfusion method.

This study has a number of limitations. It is a highly selected PCI patient cohort that is not representative of many patients undergoing PCI in the current era. The extremely high rate of PCI-related myonecrosis in this study (60%), greater than seen in our previous consecutive series, is a further reflection of this limitation. We excluded patients with side-branch injury because they were too few for a meaningful statistical comparison. Future studies with more patients need to address this further. Although great care was taken to match the myocardial segments of HE with perfusion segments, some misregistration errors could have occurred. Perfusion measurements in 3 short-axis slices did not give complete coverage of the left ventricular myocardium in this study. However, we were still able to assess 16 of 17 myocardial segments in the American Heart Association model. Furthermore, Nagel and colleagues have shown that evaluating only the 3 inner (ie, excluding the most basal and apical) short-axis slices by CMR perfusion resulted in a higher diagnostic accuracy for the detection of significant CAD than evaluating 5 short-axis slices.

Conclusions

Using quantitative CMR perfusion and DE imaging, we have shown that MPRI is reduced in myocardial segments demonstrating new irreversible injury at 24 hours after PCI. Furthermore, these reductions are transitory, seem to be confined to the segments with injury, and do not affect the entire supply territory of the culprit vessel. The sensitivity of CMR in the assessment of MBF and necrosis makes it a powerful tool in the investigation of pathophysiological mechanism injury in the setting of coronary revascularization.

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Disclosures

None.

References


Studies have shown that some patients demonstrate impairment in microcirculatory function and new myocardial necrosis after percutaneous coronary intervention (PCI). Distal embolization of plaque contents has been postulated as a possible mechanism. However, the relationship between the extent of PCI-induced myonecrosis and the degree of microcirculatory impairment is less clear. We evaluated PCI-induced changes in myocardial perfusion reserve index over time in segments with “distal-type” procedure-related myonecrosis using high-resolution quantitative cardiovascular magnetic resonance imaging. Patients at relatively high risk for distal embolization during stenting were studied with pre-PCI and 24-hour and 6-month post-PCI delayed-enhancement cardiovascular magnetic resonance imaging and first-pass perfusion cardiovascular magnetic resonance imaging at rest and stress. We found that the myocardial perfusion reserve index is reduced in the presence of new myonecrosis early after PCI. At 6 months, there was normalization of myocardial perfusion reserve index in segments that demonstrated PCI-related myonecrosis. There was no relationship between the extent of myonecrosis on cardiovascular magnetic resonance imaging and the reduction in perfusion reserve either early or late after PCI. Microcirculatory impairment early after PCI may be due to both new myonecrosis and transitory macro/microvascular plugging of the distal vascular bed.

CLINICAL PERSPECTIVE
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