Detection of Embolic Particles With the Doppler Guide Wire During Coronary Intervention in Patients With Acute Myocardial Infarction

Efficacy of Distal Protection Device

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OBJECTIVES
We investigated whether embolic particles could be detected as high-intensity transient signals (HITS) with a Doppler guide wire during percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI). We also assessed whether these signals could be reduced using a distal protection (DP) device.

BACKGROUND
Embolicization of thrombi and plaque components to the microcirculation is a major complication of PCI in patients with AMI. Embolic particles running in the cerebral artery are detected as HITS by transcranial Doppler ultrasound.

METHODS
We prospectively studied 16 consecutive patients with AMI who underwent direct PCI within 24 h after the onset of symptoms. A PercuSurge GuardWire (Medtronic AVE, Santa Rosa, California) was used as the DP device. Eight patients were randomly assigned to the non-DP group, and the remaining eight were assigned to the DP group. Coronary flow velocity was recorded continuously from before the first balloon inflation to after balloon deflation.

RESULTS
All patients in the non-DP group had HITS detected (12 ± 9 counts) within five consecutive beats (4 ± 1 beat) after balloon deflation, but none were detected in any of the patients in the DP group.

CONCLUSIONS
The Doppler guide wire can be used to visually detect and count emboli as HITS, and the DP device is effective for prevention of distal embolization. (J Am Coll Cardiol 2005;45:212–5) © 2005 by the American College of Cardiology Foundation

One of the major complications of percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI) is distal embolization due to thrombi and plaque components liberated by balloon inflation. Recently, distal protection (DP) devices have been used during PCI in patients with AMI for the prevention of distal embolization. However, the randomized, multicenter Enhanced Myocardial Efficacy and Removal by Aspiration of Liberalized Debris (EMERALD) trial (1) showed that DP devices cannot improve ST-segment resolution, infarct size, myocardial brush, or 30-day major adverse cardiac events in patients with AMI. This result was disappointing for cardiologists and raised questions regarding: 1) whether distal embolization actually occurs and what amounts of emboli are produced by PCI in AMI; and 2) whether the DP device can reduce embolic events. To answer these questions, it is necessary to establish a method that can detect and quantify the embolic particles liberated by PCI.

Emboli in the cerebral artery can be detected as high-intensity transient signals (HITS) by transcranial Doppler ultrasound (2). These HITS have been detected in patients with mechanical prosthetic valves (3), carotid stenosis (4), and ischemic stroke (3), as well as during carotid endarterectomy (5). In a previous study, we reported that an intracoronary Doppler guide wire is a simple and reliable device for the detection of epicardial coronary flow velocity during PCI (6). Recently, it was reported (7) that the Doppler guide wire can also detect HITS immediately after balloon deflation during elective PCI. However, it remains unclear whether these HITS are caused by emboli themselves, because they cannot exclude the possibility that the observed signals might have been caused by restoration of anterograde flow after balloon deflation. Therefore, it is necessary to perform a negative control study to monitor the coronary blood flow velocity spectrum after restoration of anterograde flow after balloon deflation in the absence of liberated emboli.

In the present study, we investigated whether the Doppler guide wire could detect HITS during PCI in patients with AMI and whether these HITS disappeared with the use of the DP device; this represents a negative control study of HITS, and the results confirm the effect of the DP device.

METHODS

Study population. We performed a prospective study in 18 consecutive patients with AMI referred to our hospital between January 2004 and June 2004 who fulfilled the following criteria: 1) chest pain >30 min in duration and presentation ≤24 h after symptom onset; 2) ST-segment
Abbreviations and Acronyms

AMI = acute myocardial infarction
DP = distal protection
EMERALD = Enhanced Myocardial Efficacy and Removal by Aspiration of Liberalized Debris
HITS = high-intensity transient signals
PCI = percutaneous coronary intervention
TIMI = Thrombolysis In Myocardial Infarction

Coronary flow velocity was recorded continuously during this procedure. Finally, coronary stenting was performed for all patients to achieve a residual diameter stenosis of <25%. The results of the present study demonstrated for the first time that: 1) an intracoronary Doppler guide wire can detect embolic debris in patients with AMI (8,9). However, it remains unknown whether this device can adequately reduce the embolic burden in a clinical setting. The results of the present study directly showed that PCI indeed produces embolic particles in patients with AMI, and that the DP device can aspirate liberated thrombotic debris in patients with AMI (8,9).

**DISCUSSION**

The results of the present study demonstrated for the first time that: 1) an intracoronary Doppler guide wire can detect embolic debris associated with PCI, allowing us to count their number; and 2) use of the DP device can effectively reduce the number of embolic particles. The DP device can aspirate liberated plaque and thrombotic debris in patients with AMI (8,9). However, it remains unknown whether this device can adequately reduce the embolic burden in a clinical setting. The results of the present study directly showed that PCI indeed produces embolic particles in patients with AMI, and that the DP device can reduce their number. The randomized, multicenter EMERALD trial (2) failed to show the usefulness of the DP device in patients with AMI and suggested that distal embolization caused by the PCI procedure does not play a major role in the myocardial injury associated with AMI. The results of the present study supported this observation, as embolic events may only occur just after the
PCI procedure, the number of embolic particle is small (mean 12 ± 9 counts), and the DP device works well. In our clinical experience, however, the DP device is sometimes useful, especially in patients whose culprit lesions have some volume of thrombi and/or plaque. We showed that the Doppler guide wire can easily detect emboli as HITS and allowed us to determine their number. Further Doppler guide wire studies are required to determine the characteristics of the culprit lesions that are responsible for the production of large numbers of embolic particles during PCI.

**Study limitations.** In the present study, it is not clear what size of distal emboli can be detected by the Doppler guide wire. We tried to calculate the size by the duration and velocity of HITS. The size was calculated according to the equation: size of emboli = velocity of HITS (mm/s) × duration of HITS (s), and the calculated size was 1.73 ± 0.81 mm. We are not sure that this equation really reflects the size of emboli, and further basic studies are required using techniques such as intracoronary injection of microspheres to determine the size limitation of detection by the Doppler guide wire.

**Table 1. Baseline Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Non-DP Group (n = 8)</th>
<th>DP Group (n = 8)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>59 ± 8</td>
<td>59 ± 13</td>
<td>0.94</td>
</tr>
<tr>
<td>Male gender</td>
<td>7 (88)</td>
<td>6 (75)</td>
<td>0.52</td>
</tr>
<tr>
<td>Coronary risk factor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>5 (63)</td>
<td>6 (75)</td>
<td>0.59</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>3 (38)</td>
<td>3 (38)</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (25)</td>
<td>2 (25)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>5 (63)</td>
<td>3 (38)</td>
<td>0.32</td>
</tr>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>116 ± 23</td>
<td>99 ± 19</td>
<td>0.15</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>81 ± 21</td>
<td>75 ± 26</td>
<td>0.62</td>
</tr>
<tr>
<td>Location of culprit lesions (LAD/RCA/LCx)</td>
<td>6/2/0</td>
<td>2/4/2</td>
<td>0.1</td>
</tr>
<tr>
<td>Symptom onset to reperfusion time (h)</td>
<td>8.5 ± 8.2</td>
<td>7.5 ± 5.8</td>
<td>0.78</td>
</tr>
<tr>
<td>TIMI flow grade before PCI (0/1/2)</td>
<td>3/0/5</td>
<td>5/1/2</td>
<td>0.25</td>
</tr>
<tr>
<td>TIMI flow grade after PCI (1/2/3)</td>
<td>0/0/8</td>
<td>0/0/8</td>
<td></td>
</tr>
<tr>
<td>Stent implantation</td>
<td>8 (100)</td>
<td>8 (100)</td>
<td></td>
</tr>
<tr>
<td>Peak creatine kinase (IU/l)</td>
<td>2,588 ± 1,969</td>
<td>3,213 ± 1,786</td>
<td>0.52</td>
</tr>
<tr>
<td>LVEF at discharge (%)</td>
<td>48 ± 8</td>
<td>47 ± 9</td>
<td>0.89</td>
</tr>
</tbody>
</table>

Data are expressed as the mean value ± SD or number (percentage) of subjects.

**Figure 1.** Representative coronary blood flow velocity spectra of the two groups. High-intensity transient signals (HITS) were detected in the non-distal protection (DP) group, but not in the DP group. D = diastole; S = systole.

**Figure 2.** Comparison of the number of high-intensity transient signals (HITS) among the two groups. Data are expressed as the mean value ± SD. DP = distal protection.
REFERENCES


