Short- and Long-Term Evolution of Unstented Nonocclusive Coronary Dissection After Coronary Angioplasty

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OBJECTIVES We assessed the short- and long-term clinical and angiographic outcome of nonocclusive unstented dissection after percutaneous transluminal coronary angioplasty (PTCA) and its correlation with restenosis.

BACKGROUND The use of stents has dramatically increased both the number and the cost of coronary revascularization procedures. However, this technique is not completely risk free, and its benefits have not been fully demonstrated in uncomplicated dissections.

METHODS We studied 129 consecutive patients with 49 nonocclusive dissections after PTCA (grades A to D of National Heart, Lung, and Blood Institute classification) and good distal flow (TIMI [Thrombolysis in Myocardial Infarction] flow grade 3). All patients underwent coronary angiography at 24 h and at six months post-PTCA. Clinical status was assessed every three months in the outpatient clinic. Study subjects were matched with 60 other patients in whom stenting was performed for the presence of dissection.

RESULTS In the former group, all but two patients (with type E dissection, which evolved to coronary occlusion and myocardial infarction) improved their dissection score during follow-up: at six months only 18 dissections were still angiographically visible, and no clinical adverse events were recorded. In the dissected vessels, the restenosis rate was significantly lower than in those without dissection (12% vs. 44%, \(p < 0.001\)); in the stented vessels, the restenosis rate was 25% (15/60).

CONCLUSIONS In the presence of TIMI flow grade 3, coronary dissection is associated with a favorable outcome and predicts a low restenosis rate. These results caution against the indiscriminate use of intravascular prostheses in the event of nonocclusive coronary dissection. (J Am Coll Cardiol 1999;34:1484–8) © 1999 by the American College of Cardiology

The use of endovascular prostheses (stents) has dramatically influenced the growth of nonsurgical myocardial revascularization procedures. Indeed, the extensive application of stents yielded the extension of these procedures to a broader spectrum of clinical and anatomical situations, and allowed the accomplishment of better short- (1) and long-term (2,3) results. However, stenting has also produced an increase in procedural costs, and it is associated with a rate of restenosis that is not always as favorable as reported in major randomized trials (2,3). This is especially true when stenting is employed to repair a dissection, as the technique is not completely devoid of risks and complications (4–6). Among the various procedural events prompting stent application, coronary dissection remains the most frequent one. Indeed, regardless of angiographic definitions (7) or morphological classification (8), the incidence of coronary dissection after percutaneous transluminal coronary angioplasty (PTCA) ranges from 9% to 45% (9,10). Furthermore, autopic (11–15) and intravascular imaging studies (16,17) show the presence of dissection after percutaneous coronary interventions in 50% to 80% of patients.

Dissection grade is known to contribute to the immediate result of PTCA (6,8,18,19); therefore, stent insertion, besides allowing better management of this complication, has also determined a reduction in the incidence of abrupt occlusions. The more friendly use of stents without the need of systemic anticoagulation has increased the disposition of most operators to implant a stent following almost any dissection occurring after PTCA (20). Although this ap-
Abbreviations and Acronyms  

- CK-MB = creatine kinase–myocardial band  
- ECG = electrocardiogram, electrocardiographic  
- NHLBI = National Heart, Lung, and Blood Institute  
- PTCA = percutaneous transluminal coronary angioplasty  
- QCA = quantitative coronary angiography  
- TIMI = Thrombolysis in Myocardial Infarction

proach may contribute to the safety of the procedure by decreasing the occurrence of abrupt vessel closure, the frequent need of long or multiple stents to cover a dissection fully may not necessarily lead to a low restenosis rate. Therefore, it appears appropriate to evaluate the natural history of coronary dissection occurring after angioplasty, for a better definition of its natural evolution may lead to a more selective use of stents.

We report here our experience, gathered in a relatively large patient population, treated conservatively despite the angiographic evidence of a dissection-complicating PTCA. We review the short- and long-term effects of this complication, and we try to interpret our observations in the context of the available literature.

METHODS

Patients. We investigated the results obtained in 129 consecutive patients (103 men; mean age 53 ± 11 years) with stable angina, who were treated with only balloon angioplasty (PTCA). This cohort was recruited in the initial phase of our experience in interventional cardiology, when the use of coronary stenting was not readily available in our institution.

We also assessed a second group of 60 consecutive patients, clinically and angiographically matched with the previous group, predominantly (90%) with one-vessel disease, who came to our observation at a later stage of our interventional experience and underwent single-vessel PTCA and stent implantation for the presence of a dissection after PTCA (TIMI [Thrombolysis in Myocardial Infarction] flow grade 3 in all patients).

Angioplasty. All patients undergoing coronary angioplasty were pretreated with aspirin, and a heparin intravenous (IV) bolus of 10,000 U was administered at the beginning of the procedure. In all patients the percent stenosis (and restenosis defined as a stenosis ≥50% at follow-up angiography) was determined by quantitative coronary angiography (QCA); both electrocardiogram (ECG) and blood pressure were monitored continuously throughout the procedure and for 30 min thereafter. During this time, additional coronary injections were performed every 5 to 10 min after PTCA completion to verify vessel patency and contrast runoff. Total creatine kinase (CK) (normal <200 IU/liter) and creatine kinase–myocardial band (CK-MB) isoenzyme (normal <10%) were measured immediately after procedure and every 4 h for 12 h, and finally at 24 h post-PTCA.

Nonocclusive dissection. Nonocclusive dissection was noted by each operator and then reviewed by a second investigator. It was defined as an angiographically visible intraluminal filling defect, extraluminal opacity, or flap complicating the dilation site, unassociated with symptoms and/or ischemic ECG alterations, or with impaired distal filling, at the end of the procedure. The severity of the dissection was scored on the basis of the National Heart, Lung, and Blood Institute (NHLBI) classification (8), and defined as follows: type A, radiolucent area within the lumen with minimal or no persistence of contrast; type B, parallel double lumen separated by a radiolucent area with minimal or no persistence of contrast; type C, persistent presence of contrast outside the coronary lumen; type D, spiral luminal filling defect; type E, dissection with persistent filling defect; type F, dissection with total coronary occlusion.

In the “stented” group, the prostheses were implanted to cover completely the dissection utilizing high pressures for deployment. The decision to implant the stent in these patients was based on the operator’s choice (elective, following Institute guidelines). In all, a ticlopidine (250 mg b.i.d.) and aspirin (100 mg once daily [od]) combination was used after stenting for one month. Patients were then treated indefinitely with aspirin alone and other cardiac medications as required.

All patients of both groups underwent repeat coronary angiography 24 h and six months after PTCA; furthermore, they all attended regular visits to our outpatient clinic for a six-month follow-up.

Statistical analysis. All data were expressed as the mean value ± SD. The chi-square test was performed to analyze the association of restenosis with categoric variables (SPSS statistical software package, Cary, North Carolina). A p value < 0.05 was accepted as statistically significant.

RESULTS

The clinical and angiographic features of study patients are shown in Table 1. In the group of patients only undergoing balloon angioplasty (129 patients, 111 with single-vessel disease and 18 with two-vessel disease), 147 coronary stenoses were treated. At the end of the procedure an angiographically visible dissection was present in 51 sites (35%). Of these, two type E dissections, in patients with two-vessel disease, evolved toward complete artery occlusion, and every 4 h for 12 h, and finally at 24 h post-PTCA.

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TIMI grade 3 throughout the procedure. Prolonged inflations with a perfusion balloon were not performed in any case. As expected, the highest rate of dissections occurred in B and C lesions. No clinical or angiographic differences were present between patients with and without dissection.

Figure 1 shows the distribution of dissections in the different severity classes and their evolution at 24 h and at six months. At 24 h, coronary dissection was still visible in 41 (all in patients with single-vessel disease) of the 49 initial sites; worsening was noted in 2 lesions (4%, from grades B to C), whereas improvement was observed in 12 (24%). At six months, 25 other lesions (61%) had improved: 23 dissections had disappeared and only 18 (12%) were still angiographically visible.

Figure 2 illustrates an example of the angiographic evolution of type B dissection at 24 h and at six months.

Of the remaining 145 lesions treated, 51 (35%) showed restenosis at six months. Among these, 5 (5/41, 12%) occurred in dissected lesions (all in patients with single-vessel disease), whereas 46 (46/106, 44%) occurred in vessels with no dissection (in particular, 37 patients with single-vessel disease and 9 single lesions in patients with bivessel disease) ($\chi^2 = 13.2, p = 0.0002$). When the analysis was performed using the patients instead of the lesions, 5/41

### Table 1. Baseline Clinical and Angiographic Characteristics of Patients

<table>
<thead>
<tr>
<th></th>
<th>PTCA Group</th>
<th>STENT Group</th>
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</thead>
<tbody>
<tr>
<td>Mean age ± SD (yrs)</td>
<td>55 ± 11 50 ± 8</td>
<td>57 ± 10</td>
</tr>
<tr>
<td>No. of male patients (%)</td>
<td>103 (80) 40 (89)</td>
<td>51 (85)</td>
</tr>
<tr>
<td>Recent MI (&lt;3 months), no. (%)</td>
<td>14 (11) 4 (9)</td>
<td>7 (11)</td>
</tr>
<tr>
<td>Single-vessel disease, no. (%)</td>
<td>111 (86) 41 (91)</td>
<td>54 (90)</td>
</tr>
<tr>
<td>Vessel dilated no.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>66 (45) 20 (41)</td>
<td>30 (50)</td>
</tr>
<tr>
<td>LCX</td>
<td>49 (33) 6 (12)</td>
<td>11 (18)</td>
</tr>
<tr>
<td>RCA</td>
<td>32 (22) 23 (47)</td>
<td>19 (32)</td>
</tr>
<tr>
<td>No. type A, B, C lesions</td>
<td>29-81-37 8-12-29</td>
<td>10-35-15</td>
</tr>
<tr>
<td>Type A, B, C lesions (%)</td>
<td>20-55-25 16-24-60</td>
<td>17-58-25</td>
</tr>
<tr>
<td>No. type A-B-C-D dissections</td>
<td>33-10-4-2</td>
<td>0-34-19-7</td>
</tr>
<tr>
<td>Mean diameter stenosis pre-PTCA (%)</td>
<td>85 ± 11</td>
<td>87 ± 8</td>
</tr>
<tr>
<td>Mean reference artery diameter pre-PTCA (mm)</td>
<td>3.20 ± 0.54</td>
<td>3.18 ± 0.7</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA (%)</td>
<td>20 ± 7</td>
<td>8 ± 8</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA 24 h (%)</td>
<td>24 ± 5</td>
<td>10 ± 9</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA 6 months (%)</td>
<td>75 ± 9</td>
<td>71 ± 14</td>
</tr>
<tr>
<td>(nR)</td>
<td>20 ± 10</td>
<td>10 ± 8</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA (mm)</td>
<td>3.23 ± 0.65</td>
<td>3.41 ± 0.55</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA 24 h (mm)</td>
<td>3.09 ± 0.54</td>
<td>3.37 ± 0.48</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA 6 months (mm R)</td>
<td>0.78 ± 0.51</td>
<td>0.85 ± 0.48</td>
</tr>
<tr>
<td>(nR)</td>
<td>3.01 ± 0.47</td>
<td>3.24 ± 0.61</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA dissected vessels (%)</td>
<td>22 ± 18</td>
<td>23 ± 16</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA 24 h dissected vessels (%)</td>
<td>23 ± 16</td>
<td>24 ± 9</td>
</tr>
<tr>
<td>Mean diameter stenosis post-PTCA 6-month dissected vessels (%)</td>
<td>80 ± 8</td>
<td>24 ± 9</td>
</tr>
<tr>
<td>(nR)</td>
<td>3.11 ± 0.89</td>
<td>3.09 ± 0.16</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA dissected vessels (mm)</td>
<td>3.09 ± 0.16</td>
<td>3.02 ± 0.64</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA 24 h dissected vessels (mm)</td>
<td>3.09 ± 0.16</td>
<td>3.02 ± 0.64</td>
</tr>
<tr>
<td>Mean lumen diameter post-PTCA 6-month dissected vessels (mm R)</td>
<td>0.88 ± 0.42</td>
<td>0.88 ± 0.42</td>
</tr>
<tr>
<td>(nR)</td>
<td>3.09 ± 0.42</td>
<td>3.09 ± 0.42</td>
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MI = myocardial infarction; LAD = left anterior descending; LCX = left circumflex; RCA = right coronary artery; R = restenosis; nR = nonrestenosis. Data referring to patients with dissection after PTCA are in bold.

**Figure 1.** Evolution of the dissections during the first 24 h and at six-month follow-up. A, B, C, D = degree of dissection (see text for explanation).
In patients treated with stent (a single 15-mm stent in 55/60 lesions), the dissection grade was, respectively, 56%, 32% and 12% for grades B, C and D. Unstented patients had a higher prevalence of A and B dissection grades (85% vs. 56%) as compared to stented patients. However, no difference among stented and unstented patients was found when they were categorized according to dissection type.

The rates of restenosis and recurrent angina were 25% (15/60) and 20%, respectively. Albeit lower, these figures were not significantly different from those observed in “unstented” patients. No other relevant clinical events were observed in this cohort.

**DISCUSSION**

The results of our study indicate that discrete dissections are associated with a very low incidence of acute and long-term complications and do not increase the incidence of restenosis. Indeed, in the dissected vessels, the restenosis rate was 12%, a significantly lower figure than that observed in those without dissection (44%) and in the “stented group” (25%). Although the exiguity of the figures examined does not allow us to draw conclusions on the “protective” role of dissection against restenosis, our observation agrees with previous studies (21–23).

Our results suggest that the use of stents is not mandatory to manage dissection, although a different approach has to be used when this complication is associated with an unsatisfactory angiographic result, either because of significant residual stenosis or a filling defect, which suggests the presence of overlying thrombus or an intimal flap. The fundamental condition that warrants a conservative approach when facing this type of complication is represented by the clinical stability of the patient, namely the absence of symptoms, ECG and hemodynamic modifications along with the persistence of a TIMI grade 3 flow. Whenever all these conditions are met, the short- and long-term prognosis seems to be more than satisfactory.

**Comparison with previous studies.** The results of our study are in agreement with previous data by Huber et al. (8) and Sharma et al. (18), who showed that “minor” dissections are associated with a lower risk of ischemic complications than are “major” ones. Hermans et al. (21) as well as Cripps et al. (24) demonstrated that even more extensive dissections had a good long-term follow-up when patients have no ischemic manifestations at the end of the procedure.

**Clinical implications.** How can we manage the “good, bad and ugly coronary balloon angioplasty dissection”? (7). And when does the use of a stent become warranted? Probably, in all those cases, when the relation among dissection severity, vessel size, procedural difficulties and concomitant anatomic characteristics is largely favorable. In other words, a type A–B dissection, involving the middle or distal portion of a relatively tortuous vessel with ≦2.5 mm diameter, is probably to be treated conservatively. Conversely, a type D

![Figure 2. (A) Eccentric stenosis of the proximal segment of the left anterior descending coronary artery; (B) PTCA final result: a type B dissection is observed at the site of dilation. After 24 h this result was unchanged; (C) angiography six-months after the procedure.](image)
dissection involving the proximal portion of a ≥3 mm diameter artery will require stent application, especially when the result is suboptimal and/or an intimal flap is evident.

Conclusions. Our results show that nonocclusive coronary dissection is a frequent and relatively benign complication of PTCA that is not associated with increased incidence of restenosis. Moreover, both its short- and long-term clinical evolution and angiographic evolution are generally predictable, and the persistence of TIMI grade 3 flow, along with the absence of ischemia, generally permits us to exclude the occurrence of sudden coronary occlusion. We recognize that our study was conducted in a relatively limited patient cohort. However, all patients were carefully followed and all underwent angiography at 24 h and at six months post-PTCA. We believe that the lack of significant clinical and angiographic differences associated with stented and unstented dissections suggests that the use of endovascular prostheses should be carefully evaluated in patients without flow-limiting coronary artery dissection.

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REFERENCES