When stenting an ostial or proximal coronary lesion, 1 fundamental decision is whether to extend the proximal end of the stent into the aorta (in the case of the left main [LM] or right coronary ostium) or into the polygon of confluence of the LM (in the case of the left anterior descending [LAD] ostium). Complete angiographic and intravascular ultrasound data and 9-month follow-up angiographic and clinical data were available from 459 patients with 138 ostial lesions (angiographic diameter stenosis within the ostium of ≥50%) or 321 nonostial lesions in which the proximal end of the stent ended at or near the coronary ostium. Strut protrusion was more frequent in the LM than in the right or LAD ostium (68% vs 59% vs 53%, p = 0.010). The length of strut protrusion was 3.4 ± 1.7 mm in the LM ostium, 1.7 ± 1.0 mm in the LAD ostium, and 2.4 ± 1.4 mm in the right ostium (p = 0.001). In contrast, incomplete stent coverage of the ostium was similar among the LM, LAD, and right coronary artery (23% vs 33% vs 28%, p = 0.084) with a residual uncovered segment plaque burden of 42 ± 11%. Ostial restenosis was similar between the lesions with versus without strut protrusion (3.2% vs 2.3%, p = 0.775) and between the lesions with incomplete versus complete stent coverage of the ostium (2.4% vs 3.0%, p = 0.100). Ostial restenosis was seen in only 2 of 61 lesions (3.3%) with acute malapposition. In conclusion, when treating an ostial or proximal coronary artery lesion with a drug-eluting stent, the decision of whether to protrude the proximal end of the stent or leave the ostium uncovered does not appear to be critical. © 2013 Elsevier Inc. All rights reserved. (Am J Cardiol 2013;111:1401–1407)
documented ischemia. Myocardial infarction was diagnosed by the presence of ischemic symptoms or signs plus cardiac enzyme elevation (creatine kinase-MB elevation >3 times or creatine kinase elevation >2 times the upper limit of normal or troponin I >1.5 ng/ml). The diagnosis of stent thrombosis was according to the Academic Research Consortium criteria. All patients provided written informed consent.

Quantitative angiographic analysis was done using automated edge-detection algorithms (CAAS-5, Pie Medical Imaging, Maastricht, The Netherlands) in the angiographic analysis center of the CardioVascular Research Foundation (Seoul, Korea). All images were independently analyzed by investigators who were unaware of the clinical data. The minimum lumen diameter and diameter stenosis were measured in stent and in segment to include 5-mm-long segments adjacent to the distal stent edge. Aorto-ostial lesions of the LM or right coronary artery were located within 3 mm of the aorta on the least foreshortened angiographic projection. The ostial LAD lesions were within 3 mm distal to the carina. Angiographic restenosis was defined as diameter stenosis of ≥50% at the follow-up examination, and ostial restenosis was defined as <3 mm of the coronary ostium. Patterns of restenosis were assessed using the Mehran classification.

IVUS imaging was performed after intracoronary administration of 0.2 mg nitroglycerin using motorized transducer pullback (0.5 mm/s) and a commercial scanner (Boston Scientific Scimed, Minneapolis, Minnesota) consisting of a rotating 40-MHz transducer within a 3.2F imaging sheath. Using computerized planimetry (EchoPlaque, version 3.0, Indec Systems, Mountain View, California), off-line IVUS analysis was performed. In-stent segment analysis included the minimum lumen area, minimum stent area, and external elastic membrane area. The plaque burden was calculated as follows: [(external elastic membrane – lumen)/external elastic membrane] × 100 (%). Stent underexpansion was defined as <8.0 mm² for the LM and <6.0 mm² for the LAD and right coronary arteries. The IVUS definition of each ostium paralleled the angiographic definition. In the LM and right coronary ostia, the length of the stent struts protruding into the aorta was measured (Figure 1). In contrast, if full lesion coverage was not present, the length of the ostial segment without stent coverage was also measured (Figure 1). Similarly, in ostial LAD lesions, the length of stent protrusion into the polygon of confluence of the distal LM ostium (distance from the carina to most proximal stent strut) and the length of the uncovered ostium were measured (Figure 2). Malaposition was defined as separation of ≥1 stent strut not in contact with the intimal surface of the vessel wall that was not overlapping a side branch and had evidence of blood speckling behind the strut.

All statistical analyses were performed using SPSS, version 10.0 (SPSS, Chicago, Illinois). All values are expressed as the mean ± SD for continuous variables or as counts and percentages for categorical variables. Continuous variables were compared using the unpaired t test, and categorical variables using chi-square statistics or Fisher’s exact test. p Values <0.05 were considered statistically significant. In the post hoc analysis, parameters were...
compared among the 3 groups—LM, LAD, and right ostia. Bonferroni corrections were made for multiple comparisons of the continuous variables. All p values were 2-sided, and p > 0.05 after Bonferroni correction was considered statistically significant.

Results

The clinical characteristics are listed in Table 1: Quantitative coronary angiographic data are listed in Table 2. Overall, 138 lesions were located at the coronary ostium (minimum lumen diameter located at the true ostium with angiographic diameter stenosis ≥50%), and 321 lesions were nonostial (ostial diameter stenosis <50%) but with the proximal end of the stent ending at or near the ostium. The pre- and post-stenting IVUS data are summarized in Tables 3 and 4.

With a follow-up duration of 8.7 ± 2.8 months, 24 lesions (5.2%) had angiographic in-stent restenosis, with restenosis located at the ostium in 13 (2.8%). Individual patient data are listed in Table 5. Ostial restenosis was more frequent in the right ostia (10.3%) than in the LM (1.7%) and LAD (1.2%) ostia (p < 0.001). The frequency of ostial restenosis was similar between the 138 ostial lesions and 321 nonostial lesions in which the proximal end of the stent ended at or near the ostium (2% vs 5%, p = 0.058).

Table 1
Clinical and procedural characteristics (n = 459)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>61.8 ± 9.5</td>
</tr>
<tr>
<td>Men</td>
<td>357 (78%)</td>
</tr>
<tr>
<td>Smoker</td>
<td>249 (54%)</td>
</tr>
<tr>
<td>Hypertension*</td>
<td>268 (58%)</td>
</tr>
<tr>
<td>Hypertension†</td>
<td>317 (69%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>173 (38%)</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>58.8 ± 6.4</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td>126 (27%)</td>
</tr>
<tr>
<td>Previous coronary bypass</td>
<td>12 (3%)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>16 (4%)</td>
</tr>
<tr>
<td>Renal failure†</td>
<td>36 (8%)</td>
</tr>
<tr>
<td>Drug-eluting stent type</td>
<td></td>
</tr>
<tr>
<td>Endeavor</td>
<td>52 (11%)</td>
</tr>
<tr>
<td>Endeavor Resolute</td>
<td>91 (20%)</td>
</tr>
<tr>
<td>Promus</td>
<td>76 (17%)</td>
</tr>
<tr>
<td>Xience</td>
<td>160 (35%)</td>
</tr>
<tr>
<td>Cypher</td>
<td>77 (17%)</td>
</tr>
<tr>
<td>Maximal balloon pressure (atm)</td>
<td>18.8 ± 4.5</td>
</tr>
<tr>
<td>Total stent length (mm)</td>
<td>39.5 ± 18.5</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD or n (%).
* Systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg or receiving antihypertensive treatment.
† Total cholesterol >200 mg/dl or receiving antilipidemic treatment.
‡ Serum creatinine >1.4 mg/dl.

Figure 2. (A) LAD ostium with strut protrusion into polygon of confluence zone of distal LM coronary artery (arrows), with length of strut protrusion above carina (arrow) of 2.4 mm (green bar). (B) LAD ostium with uncovered ostium (arrows), with length of uncovered ostial segment of 3.0 mm (green bar).
The frequency of ostial restenosis was not significantly different between the lesions with and without strut protrusion (9 of 282 [3.2%] vs 4 of 177 [2.3%), \( p = 0.775 \)). Among 169 lesions with >2 mm of strut protrusion into the aorta, only 3 (2.8%) showed ostial restenosis.

No significant difference was found in ostial restenosis between the patients with an uncovered ostial segment and those with complete stent coverage of the coronary ostium (3 of 126 [2.4%] vs 10 of 333 [3.0%], \( p = 0.10 \)). Only 2 of

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**Table 3**

Pre- and post-stenting intravascular ultrasound (IVUS) data

<table>
<thead>
<tr>
<th>Coronary Artery</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preprocedural intravascular ultrasound of ostial segment</td>
<td></td>
</tr>
<tr>
<td>Lumen area (mm²)</td>
<td>199</td>
</tr>
<tr>
<td>External elastic membrane area (mm²)</td>
<td>21.3 ± 5.9* ( \times )</td>
</tr>
<tr>
<td>Plaque burden (%)</td>
<td>60.1 ± 15.5</td>
</tr>
<tr>
<td>Post-stenting intravascular ultrasound</td>
<td></td>
</tr>
<tr>
<td>In-segment</td>
<td></td>
</tr>
<tr>
<td>Minimum stent area (mm²)</td>
<td>7.8 ± 2.7* ( \times )</td>
</tr>
<tr>
<td>External elastic membrane area at minimum stent area site (mm²)</td>
<td>16.8 ± 7.6* ( \times )</td>
</tr>
<tr>
<td>Ostial segment</td>
<td></td>
</tr>
<tr>
<td>Stent area (mm²)</td>
<td>11.0 ± 2.6* ( \times )</td>
</tr>
<tr>
<td>External elastic membrane area (mm²)</td>
<td>23.6 ± 5.0* ( \times )</td>
</tr>
<tr>
<td>Stent/vessel area ratio</td>
<td>0.47 ± 0.09†</td>
</tr>
<tr>
<td>Strut protrusion</td>
<td>156 (68%)* ( \times )</td>
</tr>
<tr>
<td>Length of strut protrusion (mm)</td>
<td>3.4 ± 1.7* ( \times )</td>
</tr>
<tr>
<td>Strut protrusion &gt;2 mm</td>
<td>123 (54%)* ( \times )</td>
</tr>
<tr>
<td>Strut protrusion &gt;3 mm</td>
<td>89 (39%)* ( \times )</td>
</tr>
<tr>
<td>Incomplete ostial stent coverage</td>
<td>53 (23%)</td>
</tr>
<tr>
<td>Uncovered segment length (mm)</td>
<td>-2.3 ± 1.3</td>
</tr>
<tr>
<td>Uncovered segment &gt;2 mm</td>
<td>28 (12%)</td>
</tr>
<tr>
<td>Plaque burden within uncovered ostial segment (%)</td>
<td>38.1 ± 11.9* ( \times )</td>
</tr>
<tr>
<td>Malapposition ostium</td>
<td>43 (19%)* ( \times )</td>
</tr>
</tbody>
</table>

* \( p < 0.05 \), LM versus LAD.
† \( p < 0.05 \), LM versus right coronary artery.
‡ \( p < 0.05 \), LAD versus right coronary artery.
Post-stenting intravascular ultrasound
65 43 30 164 119 38
ostium; none had restenosis.

Table 4
Intravascular ultrasound (IVUS) findings of ostial segments in lesions with and without significant ostial disease

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Coronary Ostial Lesion</th>
<th>Nonostial Coronary Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LM</td>
<td>LAD</td>
</tr>
<tr>
<td>Preprocedural intravascular ultrasound</td>
<td>57</td>
<td>23</td>
</tr>
<tr>
<td>Lumen area within ostial segment (mm²)</td>
<td>5.0 ± 2.0</td>
<td>3.1 ± 1.6</td>
</tr>
<tr>
<td>External elastic membrane area within ostial segment (mm²)</td>
<td>18.6 ± 5.3</td>
<td>14.9 ± 4.5</td>
</tr>
<tr>
<td>Plaque burden within ostial segment (%)</td>
<td>71.5 ± 10.9</td>
<td>78.9 ± 9.4</td>
</tr>
<tr>
<td>Post-stenting intravascular ultrasound</td>
<td>65</td>
<td>43</td>
</tr>
<tr>
<td>Stent area within ostial segment (mm²)</td>
<td>10.9 ± 2.3</td>
<td>8.3 ± 1.6</td>
</tr>
<tr>
<td>External elastic membrane area within ostial segment (mm²)</td>
<td>22.5 ± 5.2</td>
<td>17.5 ± 3.5</td>
</tr>
<tr>
<td>Lesions with strut protrusion</td>
<td>61 (94%)</td>
<td>31 (72%)</td>
</tr>
<tr>
<td>Strut protrusion length (mm)</td>
<td>3.7 ± 1.7</td>
<td>1.7 ± 1.0</td>
</tr>
<tr>
<td>Strut protrusion length &gt;2 mm</td>
<td>54 (83%)</td>
<td>9 (21%)</td>
</tr>
<tr>
<td>Strut protrusion length &gt;3 mm</td>
<td>38 (59%)</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>Incomplete ostial stent coverage</td>
<td>1 (2%)</td>
<td>6 (14%)</td>
</tr>
<tr>
<td>Uncovered segment length (mm)</td>
<td>−0.4</td>
<td>−1.6 ± 1.0</td>
</tr>
<tr>
<td>Uncovered segment &gt;2 mm</td>
<td>0 (0%)</td>
<td>2 (5%)</td>
</tr>
<tr>
<td>Plaque burden within uncovered ostial segment (%)</td>
<td>45.8 ± 14.0</td>
<td>44.6 ± 14.0</td>
</tr>
<tr>
<td>Malapposition</td>
<td>9 (14%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

* p < 0.05 versus ostial lesions (ostial diameter stenosis ≥50%).

Table 5
Patients with ostial restenosis

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Lesion Location</th>
<th>Ostial Lesion</th>
<th>Protrusion</th>
<th>Protrusion Length (mm)</th>
<th>Incomplete Coverage</th>
<th>Length of Incomplete Coverage (mm)</th>
<th>Plaque Burden of Uncovered Ostium (%)</th>
<th>Acute Malapposition</th>
<th>Stent Area at Ostium (mm²)</th>
<th>EEM Area Ostium (mm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LM</td>
<td>Yes</td>
<td>Yes</td>
<td>2.4</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>10.3</td>
<td>13.92</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>LM</td>
<td>No</td>
<td>No</td>
<td></td>
<td>Yes</td>
<td>2.2</td>
<td>40.3</td>
<td>Yes</td>
<td>17.28</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>LM</td>
<td>No</td>
<td>No</td>
<td></td>
<td>Yes</td>
<td>4.3</td>
<td>48.7</td>
<td>No</td>
<td>20.5</td>
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<tr>
<td>4</td>
<td>LM</td>
<td>Yes</td>
<td>Yes</td>
<td>3.6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>7.0</td>
<td>14.83</td>
<td></td>
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<tr>
<td>5</td>
<td>LAD</td>
<td>No</td>
<td>No</td>
<td></td>
<td>Yes</td>
<td>1.6</td>
<td>54.9</td>
<td>No</td>
<td>6.3</td>
<td>14.02</td>
</tr>
<tr>
<td>6</td>
<td>LAD</td>
<td>No</td>
<td>Yes</td>
<td>1.8</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>7.9</td>
<td>15.55</td>
<td></td>
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<tr>
<td>7</td>
<td>Right</td>
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<td>Yes</td>
<td>3.4</td>
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<td>No</td>
<td>No</td>
<td>9.5</td>
<td>14.67</td>
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<tr>
<td>8</td>
<td>Right</td>
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<td>Yes</td>
<td>5.6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>9.9</td>
<td>22.15</td>
<td></td>
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<tr>
<td>9</td>
<td>Right</td>
<td>Yes</td>
<td>Yes</td>
<td>1.2</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>10.1</td>
<td>22.01</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Right</td>
<td>No</td>
<td>Yes</td>
<td>2.2</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>11.9</td>
<td>22.4</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Right</td>
<td>Yes</td>
<td>Yes</td>
<td>1.2</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>12.1</td>
<td>22.94</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Right</td>
<td>Yes</td>
<td>Yes</td>
<td>1.9</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>9.0</td>
<td>16.57</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Right</td>
<td>No</td>
<td>No</td>
<td></td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>10.6</td>
<td>20.8</td>
<td></td>
</tr>
</tbody>
</table>

EEM = external elastic membrane; Pt. No. = patient number.

57 patients (3.5%) with ≥2 mm of an uncovered segment showed ostial restenosis. The residual plaque burden within the uncovered ostial segment was 50.0 ± 9.4% in patients with ostial restenosis and was not different from those without restenosis (41.3 ± 11.3%, p = 0.17).

Ostial restenosis was identified in 2 of 61 lesions (3.3%) with acute stent vessel wall malapposition at the ostium versus 11 of 398 lesions (2.8%) with complete stent vessel wall apposition (p = 0.7). Although acute malapposition was most common in the LM ostium (43 of 229 [18.8%]), ostial restenosis was found in only 1 LM (2.3%), similar to the LM ostia without malapposition (1.6%, p = 0.6). A total of 3 lesions had a nonflow-limiting edge dissection at the ostium; none had restenosis.

Ostial restenosis was associated with a much smaller external elastic membrane before the procedure (15.4 ± 4.5 vs 19.1 ± 6.0 mm², p = 0.050) or after stenting (17.9 ± 3.5 vs 21.1 ± 5.1 mm², p = 0.025) compared to those without ostial restenosis. However, no significant differences were found in the preprocedural ostial lumen area (6.5 ± 2.1 vs 7.3 ± 4.2 mm², p = 0.7), preprocedural ostial plaque burden (56.7 ± 12.4% vs 61.9 ± 15.2%, p = 0.3), or final stent area at the ostium (9.5 ± 1.6 mm² vs 10.0 ± 2.5 mm², p = 0.7).

The clinical follow-up duration was 31.1 ± 11.4 months. Major adverse coronary events occurred in 18 patients (6.4%). Of the 18 patients, 4 (0.9%) died from cardiac causes; acute myocardial infarction occurred in 6 (1.3%), including 3 (0.7%) with definite stent thrombosis; and target lesion revascularization was performed in 26 (5.7%), of whom 9 underwent repeat percutaneous coronary intervention because of ostial restenosis. No significant differences were found in the incidence of major adverse coronary events between patients with proximal strut protrusion and...
those without proximal strut protrusion (6.4% vs 5.6%, p = 0.8), ostia with incomplete versus complete stent coverage (4.0% vs 6.9%, p = 0.3), and patients with acute ostial stent vessel wall malapposition and those with complete ostial stent vessel wall apposition (6.6% vs 6.0%, p = 0.7). The clinical variables did not predict major adverse coronary events.

Discussion

The major findings of the present study were as follows. First, during IVUS-guided DES implantation into lesions at or near the ostium of the LM, LAD, or right coronary artery, >1/2 showed strut protrusion, and 28% had incomplete stent coverage of the ostium. Strut protrusion and acute malapposition were more frequent in the LM than in the LAD and right ostia. However, no difference was found in full lesion coverage among the 3 locations. Second, although a smaller pre- or post-stenting external elastic membrane area at the ostium was a risk factor for ostial restenosis, strut protrusion, incomplete ostial coverage, and malapposition did not predict ostial restenosis or major adverse coronary events.

In the bare metal stent era, ostial lesions had greater restenosis rates than nonostial lesions. Histologic data showed that ostial lesions were heavily calcified and sclerotic, which led to more elastic recoil, even after stenting. Moreover, a greater frequency of ostial restenosis was reported in right than in LM ostia (50% vs 19%), explained in part by chronic stent recoil at the right ostium.

DES treatment of aorto-ostial lesions appeared safe and effective, with a significant improvement in restenosis and late clinical events. The use of DESs for aorto-ostial lesions resulted in a lower rate of in-segment restenosis and repeat revascularization than with bare metal stents; a small reference vessel diameter was the only independent predictors of angiographic restenosis.

In our study, ostial restenosis was more frequent in the right coronary ostium (10.3%) than in the LM (1.7%) and LAD (1.2%) ostia. The smaller vessel size of the right ostium relative to the LM and the greater elastic recoil and rigidity in the adjacent aortic wall might contribute to the greater restenosis rate.

For nonostial coronary lesions, stent underexpansion was an important predictor of restenosis. However, after IVUS-guided DES implantation and optimization, most of our patients showed a uniformly large final stent area at the ostium to diminish the effect of stent expansion on ostial restenosis.

In the present study, strut protrusion was seen in 68% of LM, 53% of LAD, and 59% of right ostia. This was not associated with ostial restenosis or major adverse coronary events. Even in lesions with a strut protrusion length >3 mm, only 3.0% lesions showed ostial restenosis. Thus, stent protrusion beyond the ostium should not be a procedural concern.

No significant difference was found in ostial restenosis between patients with an uncovered segment versus those with complete ostial coverage. However, with IVUS guidance, uncovered ostia with a significant residual plaque burden were mostly treated with additional stent placement, such that the final plaque burden of the uncovered ostial segment was only a small 40%. Thus, incomplete ostial stent coverage appeared to have little effect on restenosis or clinical events, as long as the residual plaque burden was modest, similar to that found in other DES edge restenosis studies. Consistent with data from nonostial lesions, acute malapposition at the ostium did not affect the incidence of restenosis or major adverse coronary events.

The present study was a retrospective, single-center study. The relatively low event rate might have affected the results. Because only a small number of patients underwent isolated left circumflex ostial stenting, these patients were not included in the present analysis. Also, because we did not have follow-up IVUS scanning, the restenosis mechanisms were not studied.

Disclosures

The authors have no conflicts of interest to disclose.


