One-Year Results of Paclitaxel-Eluting Stent Implantation in Ostial Lesions of the Left Anterior Descending Artery

Li-Chin Sung, Ji-Hung Wang and Yu-Chih Chen

Background: Lesions in the ostial left anterior descending (LAD) coronary artery are usually considered an indication for bypass surgery because of possible distal left main (LM) involvement. Percutaneous coronary intervention for such lesions is considered a high-risk procedure because of high rates of acute complications and later restenosis. The implantation of drug-eluting stents has been reported to reduce angiographic restenosis and major adverse cardiac events (MACE) compared to bare-metal stent. To date, there is little literature regarding the safety and efficacy of drug-eluting stent implantation in ostial LAD lesions. We sought to investigate the immediate and late clinical outcomes of paclitaxel-eluting stents (PES) implantation for such lesions.

Methods: From July 2004 to October 2007, PES were implanted in 44 consecutive patients with ostial LAD stenoses in our hospital. We applied two different stenting strategies: (1) precise stent positioning, and (2) stent covering the distal LM coronary artery in the presence of intermediate distal LM lesions, thus ensuring full lesion coverage. We retrospectively reviewed the charts to assess MACE, which included cardiac death, non-fatal myocardial infarction (MI), target lesion revascularization (TLR) or target vessel revascularization. Results: Forty-four patients (30 males, 14 females) with a mean age of 64.4 ± 10.2 years (range, 46-83 years) were evaluated. The angiographic as well as procedural success rate was 100%, and none of the patients had in-hospital MACE. Clinical one-year follow-up was available for all patients. Angiographic follow-up was available for seventeen (38.6%) patients. Neither cardiac death nor stent thrombosis occurred in our patients, but one (2.3%) patient had non-fatal MI during the follow-up period. Four patients had clinically-driven angiographic restenosis and underwent TLR subsequently. The cumulative MACE-free survival rate was 88.6% at one year.

Conclusions: Paclitaxel-eluting stent implantation in ostial LAD lesions with complete lesion coverage achieves high procedural success rate and acceptable clinical outcomes during the one-year follow-up period.

Key Words: Ostial stenting • Paclitaxel-eluting stent • Percutaneous coronary intervention

INTRODUCTION

The treatment of ostial left anterior descending (LAD) coronary artery lesions is a challenge for interventional cardiologists because these lesions usually involve the distal left main (LM) coronary artery.1 Focal ostial stenting may result in incomplete lesion coverage or plaque shift into the ostium of the left circumflex (LCX) artery or LM coronary artery.2,3 Furthermore, the reported restenosis rate of 26.1% after implantation for such lesions with bare-metal stents (BMS) is high.4 Debubling atherectomy before stent implantation for ostial LAD stenosis has been advocated to reduce restenosis but is controversial in recent studies.5,6 For
these reasons, such patients are often referred for coronary artery bypass grafting (CABG), even patients who have single-vessel disease. Recently, the use of drug-eluting stents (DES) has been considered more important than debulking in the prevention of restenosis by suppression of neointimal growth. The implantation of paclitaxel-eluting stents (PES) in non-ostial LAD lesions has a lower restenosis rate compared to BMS for one year. So, DES implantation in ostial LAD lesions might be a reasonable option to reduce restenosis. However, there have been little publish data regarding the immediate and late results of PES implantation for ostial LAD stenosis. The purpose of the present study was to evaluate the clinical outcomes of patients following implantation of PES in ostial LAD lesions in our institution during a one-year period.

**METHODS**

**Study patients and design**

A study group comprised of 44 consecutive patients with 44 ostial LAD lesions who decided to receive coronary intervention between July 2004 and October 2007 at Hualien Tzu Chi medical center was analyzed. All the patients had been treated with PES (Taxus™, Boston Scientific Corp., Natick, Massachusetts). We defined the ostial LAD stenosis as a lesion with a diameter stenosis (DS) > 50% within 3 mm of the LAD orifice and a vessel diameter > 2.5 mm. Different from the previous published studies, patients with the following lesions involving the LAD ostium were not excluded from analysis: (1) chronic total occlusion; (2) long and diffuse lesions; (3) restenotic lesions; and (4) acute myocardial infarction (MI) with thrombotic lesions. Patients with coexisting significant disease (> 50% DS) in the distal LM coronary artery or the ostium of the LCX, or severe left ventricular dysfunction (ejection fraction < 30%) were excluded. Patients were followed up for at least 12 months. Repeat angiographic evaluation was left to the discretion of the physician. Clinical follow-up was performed by telephone contact or outpatient visit. The study endpoints included angiographic success, procedural success, target lesion revascularization (TLR) rate and any major adverse cardiac events (MACE) during the follow-up period. This was a retrospective, observational study. Records from the patient’s chart were obtained for review.

**Procedural protocol**

Each patient was adequately informed of the alternative surgical treatment and signed an informed consent. At the beginning of the procedure, the patient received heparin (100 u/kg) bolus with a repeat bolus to achieve an activated clotting time of > 250 sec. Glycoprotein IIb/IIIa inhibitors were administered at the discretion of the operator. All patients received combine antiplatelet therapy with aspirin 100 mg and clopidogrel 75 mg daily ≥ 3 days before the procedure. A loading dose of 300 mg of clopidogrel was administered to patients who were not pretreated. Aspirin (100 mg/day) was continued indefinitely after stenting; however, we shifted aspirin to clopidogrel if patients had peptic ulcer (PU) disease with bleeding after antiplatelet medications. All of our procedures were performed or under supervised by an experienced, high-volume interventional cardiologist.

**Angiographic analysis**

All coronary angiograms (CAG) were analyzed by two experienced angiographers. Using contrast-filled guiding catheter for calibration and an on-line quantitative coronary angiographic system (GE Healthcare Centricity Cardiology CA1000, Milwaukee, USA), reference vessel diameter, minimal lumen diameter (MLD) and percent DS were measured before and after the intervention and at follow-up from the single worst and least foreshortened view.

**Definitions**

Angiographic success was defined as < 20% angiographic residual DS in the presence of Thrombolysis In Myocardial Infarction flow grade 3.

Procedural success was defined as angiographic success without major procedural or in-hospital complications.

Restenosis was defined as > 50% DS within a stented segment at follow-up.

TLR was defined as CABG or repeat revascularization that was driven by restenosis within the stent or within the 5-mm borders proximal and distal to the stent.

Target vessel revascularization (TVR) was defined as any reintervention performed on the treated vessel.
MACE was defined as cardiac death, non-fatal MI, or the need of TLR or TVR.

Intermediate distal LM coronary artery stenosis was defined as a visually estimated DS < 50% by CAG.

Acute lumen gain was defined as the difference between MLD at the end of the stenting and the baseline MLD.

In-segment late lumen loss was defined as the difference between the final MLD and the MLD at the follow-up of the ostial LAD segment.

Loss index was defined as the ratio between late lumen loss and the acute gain.

**Statistical analysis**

Data are expressed as means ± one SD for continuous variables and as frequencies (%) for categorical variables. Continuous variables were compared using the unpaired Student’s 𝑡 test, and categorical variables were compared using the chi-square test or Fisher’s exact test. Cumulative analysis of event-free survival was expressed by Kaplan-Meier survival curve. Statistical significance was accepted for a two-sided, probability value of 𝑃 < 0.05.

**RESULTS**

**Baseline characteristics**

The baseline clinical characteristics of the patients are shown in Table 1. Forty-four patients (30 males, 14 females) with a mean age of 64.6 ± 10.2 years (range, 46-83 years) were included. All patients had either symptoms or documented ischemia by non-invasive test. The majority of these patients (24 patients, 54.5%) had unstable angina. The prevalence of hypertension was 70.5% and of diabetes 40.9%.

**Angiographic and procedural characteristics and in-hospital outcomes**

Angiographic and procedural characteristics are summarized in Table 2. The radial artery approach was utilized in 86.4% of procedures. A total of 14 (31.8%) patients had single-vessel disease, 14 (31.8%) patients had double-vessel disease, and 16 (36.4%) patients had three-vessel disease. Predilation using balloon angioplasty (59%) or debulking devices (4.55%) was discretionary, although a strategy of direct stenting (36.4%) in order to minimize balloon injury to the arterial wall was preferred. The mean stent deployment pressure was 15.23 ± 3.56 atmospheres (atm). Intermediate distal LM lesions were presented in 52.3% of patients, who were almost all treated by stent positioning from the distal LM coronary artery across the LCX ostium into LAD, ensuring full lesion coverage. Final kissing balloon dilatation was performed in 13.6% of patients due to significant stent compromise of the LCX ostium after the procedure. The angiographic and procedural success rates were 100% in our patients (Table 2). There were no cases of acute stent thrombosis or coronary perforation.

**Table 1. Baseline characteristics of patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>44</td>
</tr>
<tr>
<td>Number of lesions</td>
<td>44</td>
</tr>
<tr>
<td>Age (years)</td>
<td>64.6 ± 10.2</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>30 (68.2)</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>9 (20.5)</td>
</tr>
<tr>
<td>Former smoker, n (%)</td>
<td>10 (22.7)</td>
</tr>
<tr>
<td>DM, n (%)</td>
<td>18 (40.9)</td>
</tr>
<tr>
<td>Insulin</td>
<td>3 (6.8)</td>
</tr>
<tr>
<td>Oral Hypoglycemic agent</td>
<td>15 (34.1)</td>
</tr>
<tr>
<td>L1*, n (%)</td>
<td>26 (59.1)</td>
</tr>
<tr>
<td>HT, n (%)</td>
<td>31 (70.5)</td>
</tr>
<tr>
<td>Previous stroke, n (%)</td>
<td>1 (2.3)</td>
</tr>
<tr>
<td>Previous PCI, n (%)</td>
<td>19 (43.2)</td>
</tr>
<tr>
<td>Previous congestive heart failure, n (%)</td>
<td>4 (9.1)</td>
</tr>
<tr>
<td>CRF (Non-HD), n (%)</td>
<td>6 (13.6)</td>
</tr>
<tr>
<td>HD, n (%)</td>
<td>3 (6.8)</td>
</tr>
<tr>
<td>Left ventricular EF (%)</td>
<td>51.8 ± 19.7</td>
</tr>
<tr>
<td>Clinical presentation before PCI, n (%)</td>
<td></td>
</tr>
<tr>
<td>Stable angina pectoris</td>
<td>13 (29.5)</td>
</tr>
<tr>
<td>Unstable angina pectoris</td>
<td>24 (54.5)</td>
</tr>
<tr>
<td>Non ST elevation MI</td>
<td>6 (13.6)</td>
</tr>
<tr>
<td>ST elevation MI</td>
<td>1 (2.3)</td>
</tr>
</tbody>
</table>

*Total cholesterol > 200 mg/dL or receiving lipid-lowering treatment.

DM = diabetes mellitus; LI = dyslipidemia; HT = hypertension; PCI = percutaneous coronary intervention; CRF = chronic renal failure; HD = hemodialysis; EF = ejection fraction; MI = myocardial infarction.
### Table 2. Angiographic and procedural characteristics and in-hospital outcomes

<table>
<thead>
<tr>
<th>Characteristics and outcomes</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Approach vessel, n (%)</td>
<td>44 (100)</td>
</tr>
<tr>
<td>Radial artery</td>
<td>38 (86.4)</td>
</tr>
<tr>
<td>Brachial artery</td>
<td>2 (4.5)</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>4 (9.1)</td>
</tr>
<tr>
<td>Angiographic characteristics, n (%)</td>
<td></td>
</tr>
<tr>
<td>Three-vessel CAD</td>
<td>16 (36.4)</td>
</tr>
<tr>
<td>Double-vessel CAD</td>
<td>14 (31.8)</td>
</tr>
<tr>
<td>Single-vessel CAD</td>
<td>14 (31.8)</td>
</tr>
<tr>
<td>Intermediate distal LM lesions</td>
<td>23 (52.3)</td>
</tr>
<tr>
<td>No distal LM lesions</td>
<td>21 (47.7)</td>
</tr>
<tr>
<td>Severe calcified lesions</td>
<td>2 (4.6)</td>
</tr>
<tr>
<td>Restenotic lesions</td>
<td>6 (13.6)</td>
</tr>
<tr>
<td>Chronic total occlusion</td>
<td>2 (4.6)</td>
</tr>
<tr>
<td>Very long, diffuse lesions (&gt; 32 mm)</td>
<td>8 (18.2)</td>
</tr>
<tr>
<td>Procedural characteristics</td>
<td></td>
</tr>
<tr>
<td>Stent per lesion</td>
<td>1.18 ± 0.39</td>
</tr>
<tr>
<td>Max pressure inflation (atm)</td>
<td>15.23 ± 3.56</td>
</tr>
<tr>
<td>Rotablator debulking, n (%)</td>
<td>2 (4.55)</td>
</tr>
<tr>
<td>Cutting balloon, n (%)</td>
<td>2 (4.55)</td>
</tr>
<tr>
<td>Direct stent, n (%)</td>
<td>16 (36.4)</td>
</tr>
<tr>
<td>Final kissing balloon, n (%)</td>
<td>6 (13.6)</td>
</tr>
<tr>
<td>Multi-vessel PCI, n (%)</td>
<td>6 (13.6)</td>
</tr>
<tr>
<td>IVUS guidance, n (%)</td>
<td>5 (11.4)</td>
</tr>
<tr>
<td>GP IIb/IIIa inhibitor, n (%)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Precise location, n (%)</td>
<td>20 (45.5)</td>
</tr>
<tr>
<td>No distal LM lesion</td>
<td>18 (40.9)</td>
</tr>
<tr>
<td>Distal LM lesion</td>
<td>2 (10)</td>
</tr>
<tr>
<td>Stent covering distal LM, n (%)</td>
<td>24 (54.5)</td>
</tr>
<tr>
<td>No distal LM lesion</td>
<td>3 (12.5)</td>
</tr>
<tr>
<td>Distal LM lesion</td>
<td>21 (87.5)</td>
</tr>
<tr>
<td>In-hospital outcomes, n (%)</td>
<td>44 (100)</td>
</tr>
<tr>
<td>Angiographic success</td>
<td>44 (100)</td>
</tr>
<tr>
<td>Death</td>
<td>0 (0)</td>
</tr>
<tr>
<td>MI</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Emergency bypass surgery</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Repeat intervention</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

| Approach vessel, n (%)                                | 44 (100)                        |
| Radial artery                                         | 38 (86.4)                       |
| Brachial artery                                       | 2 (4.5)                         |
| Femoral artery                                        | 4 (9.1)                         |
| Angiographic characteristics, n (%)                   |                                 |
| Three-vessel CAD                                      | 16 (36.4)                       |
| Double-vessel CAD                                     | 14 (31.8)                       |
| Single-vessel CAD                                     | 14 (31.8)                       |
| Intermediate distal LM lesions                       | 23 (52.3)                       |
| No distal LM lesions                                  | 21 (47.7)                       |
| Severe calcified lesions                              | 2 (4.6)                         |
| Restenotic lesions                                    | 6 (13.6)                        |
| Chronic total occlusion                               | 2 (4.6)                         |
| Very long, diffuse lesions (> 32 mm)                  | 8 (18.2)                        |

### Table 3. Angiographic results

<table>
<thead>
<tr>
<th>Results</th>
<th>n = 44</th>
</tr>
</thead>
<tbody>
<tr>
<td>QCA before and after intervention</td>
<td></td>
</tr>
<tr>
<td>Lesion length (mm)</td>
<td>27.29 ± 12.25</td>
</tr>
<tr>
<td>Pre-RVD (mm)</td>
<td>3.16 ± 0.28</td>
</tr>
<tr>
<td>Pre-MLD (mm)</td>
<td>0.77 ± 0.41</td>
</tr>
<tr>
<td>Diameter stenosis (%)</td>
<td>75.45 ± 13.21</td>
</tr>
<tr>
<td>Post-RVD (mm)</td>
<td>3.39 ± 0.32</td>
</tr>
<tr>
<td>Post-MLD (mm)</td>
<td>3.12 ± 0.34</td>
</tr>
<tr>
<td>Residual stenosis (%)</td>
<td>7.95 ± 4.47</td>
</tr>
<tr>
<td>Acute Gain</td>
<td>2.35 ± 0.59</td>
</tr>
<tr>
<td>QCA at follow-up</td>
<td>17 (38.6)</td>
</tr>
<tr>
<td>Duration of follow-up (days)</td>
<td>239 ± 102</td>
</tr>
<tr>
<td>RVD (mm)</td>
<td>3.20 ± 0.25</td>
</tr>
<tr>
<td>MLD (mm)</td>
<td>2.67 ± 0.39</td>
</tr>
<tr>
<td>Diameter stenosis (%)</td>
<td>16.4 ± 11.0</td>
</tr>
<tr>
<td>Late loss (in-segment) (mm)</td>
<td>0.42 ± 0.45</td>
</tr>
<tr>
<td>Late loss index</td>
<td>0.20 ± 0.19</td>
</tr>
<tr>
<td>Angiographic F/U by, n (%)</td>
<td>9 (20.5)</td>
</tr>
<tr>
<td>Symptom of angina pectoris</td>
<td>7 (15.9)</td>
</tr>
<tr>
<td>Positive non-invasive test</td>
<td>1 (2.3)</td>
</tr>
<tr>
<td>Negative test but with symptom</td>
<td>18 (40.9)</td>
</tr>
<tr>
<td>Angina pectoris before PCI</td>
<td>16 (36.4)</td>
</tr>
<tr>
<td>Acute MI before PCI</td>
<td>2 (4.5)</td>
</tr>
<tr>
<td>Patients with only clinical F/U, n (%)</td>
<td>10 (22.7)</td>
</tr>
<tr>
<td>Negative non-invasive test, n (%)</td>
<td>7 (15.9)</td>
</tr>
<tr>
<td>Follow-up acute MI patients with, n (%)</td>
<td>5 (11.4)</td>
</tr>
<tr>
<td>Angiography or stress test</td>
<td>2 (4.5)</td>
</tr>
<tr>
<td>Clinical F/U (no symptom)</td>
<td></td>
</tr>
</tbody>
</table>

QCA = quantitative coronary angiography; RVD = reference vessel diameter; MLD = minimal lumen diameter; F/U: follow-up.

Patients had negative results on non-invasive test and the 18 (40.9%) asymptomatic patients did not have any further examination. Some asymptomatic patients refused to accept angiographic follow-up. We believe that the decision to perform follow-up CAG according to the presence of recurrent symptoms is reliable in patients with previous typical angina and without the history of MI. The follow-up CAG at a mean interval of 239 ± 102 days revealed late lumen loss of 0.42 ± 0.45 mm and late loss index of 0.20 ± 0.19. Two different strategies of the stenting procedure, covering the distal LM coronary artery (Group I) or precise location (Group II), were applied. The angiographic analysis of both groups is shown in Table 4. Baseline characteristics were well matched between the two groups. The rates of angiographic follow-up were 45.8% in group I and 30% in group II (P = 0.35). The 2 patients with distal LM lesions treated with precise stent positioning (group II) did not receive follow-up CAG because they were symptom-free. Patients in group I had more high-pressure balloon inflation (16.3 ± 3.2 atm vs. 13.9 ± 3.6 atm, P = 0.02) and a higher rate of final kissing balloon inflation (25% vs. 0%, P = 0.02) than in group II. Clinically-driven TLR at one year was more frequent in group I (16.7%) than group II (0%), but did not differ significantly (P = 0.11). Four patients with restenotic lesions belonged to group I, and their pattern of restenosis was focal (≤ 10 mm). Table 5 presents the details for all pa-
tients who had restenosis. In 3 of the 4 patients, restenosis occurred within the LCX ostium; of these, two patients had undergone PES implantation and the other patient had undergone cutting balloon angioplasty. In 1 of the 4 patients, restenosis occurred at the proximal edge of PES within the LM artery, which lesion was treated with another PES implantation into the LM ostium overlapped with the previous PES.

**Follow-up clinical outcomes**

Clinical follow-up was obtained in all patients for one year. During this period, one patient receiving clopidogrel monotherapy had non-fatal MI (2.3%) due to restenosis at the proximal edge of PES (Table 6). Clinically-driven TLR was performed in 4 (9.1%) of the 44 total patients. One (2.3%) of our patients underwent TVR. There were no incidences of stent thrombosis, cardiac death or revascularization with CABG during the follow-up period. The Kaplan-Meier estimate of overall MACE-free survival rate at one year was 88.6% (Figure 1).

**DISCUSSION**

The major findings of this study demonstrate that PES implantation for ostial LAD lesions: (1) has a high rate of immediate procedural success; (2) is safe without stent thrombosis; and (3) achieves acceptable one-year MACE-free survival.

In the BMS era, De Cesare et al. reported the experience of Palmaz-Schatz stent implantation in 23 patients with ostial LAD stenosis. Some patients had slight progression of LM stenosis during the follow-up. The authors advocated that stenting of ostial LAD lesions

<table>
<thead>
<tr>
<th>No.</th>
<th>Age/sex</th>
<th>Underlying disease/indications/stress test*</th>
<th>Direct stent</th>
<th>Distal LM lesion</th>
<th>Stenting group</th>
<th>Final kissing balloon</th>
<th>LCX lesion</th>
<th>EF**</th>
<th>Site of ISR</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60/M</td>
<td>DM/LI/AP/-</td>
<td>-</td>
<td>+</td>
<td>I</td>
<td>+</td>
<td>-</td>
<td>48</td>
<td>LCX(O)</td>
<td>stent</td>
</tr>
<tr>
<td>2</td>
<td>68/M</td>
<td>DM/HT/AP/-</td>
<td>+</td>
<td>+</td>
<td>I</td>
<td>-</td>
<td>-</td>
<td>67</td>
<td>Stent(P)</td>
<td>stent</td>
</tr>
<tr>
<td>3</td>
<td>81/F</td>
<td>HT/AP/-</td>
<td>+</td>
<td>+</td>
<td>I</td>
<td>+</td>
<td>+</td>
<td>61</td>
<td>LCX(O)</td>
<td>CB</td>
</tr>
<tr>
<td>4</td>
<td>56/M</td>
<td>LI/AP/-</td>
<td>-</td>
<td>+</td>
<td>I</td>
<td>-</td>
<td>-</td>
<td>58</td>
<td>LCX(O)</td>
<td>stent</td>
</tr>
</tbody>
</table>

* Stress test before follow-up angiography.  
** By echocardiography.  
M = male; F = female; AP = angina pectoris; ISR = in-stent restenosis; O = ostium; P = proximal edge; CB = cutting balloon.

Table 4. The angiographic results of 2 different strategies of stenting procedures

<table>
<thead>
<tr>
<th></th>
<th>Covering LM (I) (n = 24)</th>
<th>Precise location (II) (n = 20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivessel CAD, n (%)</td>
<td>16 (66.7)</td>
<td>14 (70)</td>
<td>1.0</td>
</tr>
<tr>
<td>Lesion length (mm)</td>
<td>26.69 ± 12.51</td>
<td>28.01 ± 12.22</td>
<td></td>
</tr>
<tr>
<td>Before intervention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVD (mm)</td>
<td>3.24 ± 0.27</td>
<td>3.05 ± 0.26</td>
<td></td>
</tr>
<tr>
<td>MLD (mm)</td>
<td>0.76 ± 0.40</td>
<td>0.78 ± 0.42</td>
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</tr>
<tr>
<td>Diameter stenosis (%)</td>
<td>76.09 ± 12.91</td>
<td>74.68 ± 13.87</td>
<td></td>
</tr>
<tr>
<td>Max pressure (atm)</td>
<td>16.3 ± 3.2</td>
<td>13.9 ± 3.6</td>
<td>0.02</td>
</tr>
<tr>
<td>After intervention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVD (mm)</td>
<td>3.41 ± 0.27</td>
<td>3.36 ± 0.37</td>
<td></td>
</tr>
<tr>
<td>MLD (mm)</td>
<td>3.13 ± 0.27</td>
<td>3.10 ± 0.41</td>
<td></td>
</tr>
<tr>
<td>Residual stenosis (%)</td>
<td>7.98 ± 4.27</td>
<td>7.92 ± 4.81</td>
<td></td>
</tr>
<tr>
<td>Final kissing balloon, n (%)</td>
<td>6 (25)</td>
<td>0 (0)</td>
<td>0.02</td>
</tr>
<tr>
<td>One-year angiographic follow-up, n (%)</td>
<td>11 (45.8)</td>
<td>6 (30)</td>
<td>0.35</td>
</tr>
<tr>
<td>Angiographic restenosis (%)</td>
<td>4/11 (36.4)</td>
<td>0/6 (0)</td>
<td></td>
</tr>
<tr>
<td>Clinically-driven TLR, n (%)</td>
<td>4 (16.7)</td>
<td>0 (0)</td>
<td>0.11</td>
</tr>
<tr>
<td>Stent thrombosis, n (%)</td>
<td>0 (0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TLR = target lesion revascularization.
should be avoided in the presence of distal LM disease, even if mild.\textsuperscript{9} The use of DES in ostial lesions resulted in more unfavorable outcomes than in non-ostial lesions, and previous reports suggested that the relatively high restenosis rate (14.7\%) might be associated with incomplete lesion coverage.\textsuperscript{10,11} Sirolimus-eluting stents for ostial LAD lesions with full lesion coverage have been shown to reduce restenosis and improve clinical outcomes compared with BMS.\textsuperscript{5} Furthermore, several recent studies have documented the safety of LM stenting with DES, and early follow-up results did not show excessive morbidity and mortality.\textsuperscript{12-14} With all the above issues are taken into consideration, we believe that PES positioning from the LM into the ostial LAD in the presence of intermediate distal LM narrowing results in complete lesion coverage and leads to a more favorable clinical outcome. We used the two stenting strategies similar to the technique reported by Seung et al.\textsuperscript{5} In our study, a higher final balloon pressure to deploy or postdilate the stent was applied in group I. The distal LM segments of the stents were usually under higher pressure balloon postdilatation (mean 16.3 $\pm$ 3.2 atm). Using a higher balloon pressure is essential to produce adequate stent expansion and complete vessel wall apposition, thus reducing later restenosis.\textsuperscript{14} However, it was difficult for us to deploy the stent at an optimal balloon inflation pressure in order to have stent better apposition without plaque shift. If these complications occurred, it turned out to be an LM-equivalent disease. The stent deployment under intravascular ultrasonography (IVUS) guidance and the use of kissing balloon technique to prevent or manage plaque shift are usually recommended in this condition. IVUS was used more selectively in our study population (11.4\%) compared to the report of Seung et al (89.7\%). According to our immediate clinical outcomes, it is at least feasible to perform crossover LM stenting with detailed angiographic assessment alone. Further investigation must be carried out to compare the results of routine IVUS with selective IVUS for such procedure.

The restenosis in this study deserves discussion. One of our patients developed restenosis at the proximal stent margin. The proximal part of the lesion uncovered by PES, residual dissection after the procedure, and balloon trauma outside the stent were several possible reasons for it. Three patients developed restenosis at the ostium of the LCX. Two of the three patients received a final kissing balloon angioplasty. The final kissing balloon inflation was assumed a possible etiology of restenosis at the ostium of the side branch,\textsuperscript{8} and it may not prevent side branch restenosis after main branch stenting.\textsuperscript{15} Also, plaque shift during high-pressure stent implantation (the snow-plough effect) or the presence of a narrow angle between the LAD and LCX are possible causes for late restenosis following ostial LAD stenting.\textsuperscript{2,4,16} All of our restenotic lesions were treatable with repeat percutaneous revascularization.

We treated 18 patients with isolated ostial LAD lesions utilizing precise positioning of PES, and no clinically-driven TLR was noted within one year. Cubeddu et al. reported that using LM stenting technique for isolated ostial LAD or LCX lesions is a reasonable choice. They

**Table 6. Major clinical events at follow-up**

<table>
<thead>
<tr>
<th>Clinical events</th>
<th>Follow up (12 months) (n = 44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MACE, n (%)</td>
<td>5 (11.4)</td>
</tr>
<tr>
<td>Death, n (%)</td>
<td></td>
</tr>
<tr>
<td>Cardiac death</td>
<td>0 (0)</td>
</tr>
<tr>
<td>No cardiac death</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Q-MI, n (%)</td>
<td>1 (2.3)</td>
</tr>
<tr>
<td>Non-Q-MI, n (%)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>TLR, n (%)</td>
<td>4 (9.1)</td>
</tr>
<tr>
<td>Group I</td>
<td>4 (9.1)</td>
</tr>
<tr>
<td>Group II</td>
<td>0 (0)</td>
</tr>
<tr>
<td>TVR (not TLR), n (%)</td>
<td>5 (11.4)</td>
</tr>
<tr>
<td>Group I</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Group II</td>
<td>1 (2.3)</td>
</tr>
<tr>
<td>CABG, n (%)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Stent thrombosis, n (%)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

MACE = major adverse cardiac events; TVR = target vessel revascularization; CABG = coronary artery bypass grafting.

**Figure 1.** Kaplan-Meier estimates of (death or nonfatal-MI)-free and MACE-free survival following PCI during the one year follow-up period.
enrolled 33 patients with 33 lesions. Three patients had restenosis at the ostium of the side branch, and 2 patients had developed restenosis at the proximal stent margin within the LM artery at 2 years. The incidence rates for cardiac death, non-fatal MI, and TLR were 3%, 3%, and 15%, respectively. The overall event-free survival rate was 79%. However, little information is found in the literature for direct comparison between the above two different stenting strategies for isolated ostial LAD lesions.

We used an LM stenting strategy in patients with distal LM lesions less restrictively than Seung et al. because they used this procedure only with a visually estimated distal LM DS ≥ 30% by angiography. Seung et al. reported no restenosis in this group. Although there was still insufficient data to support our hypothesis, the infrequent use of IVUS guidance and a higher incidence of diabetes in our patients (40.9% vs. 23.5%) may be the possible reasons for higher incidence of restenosis in our group I patients.

We thought that LM stenting with IVUS guidance in some conditions and precise stent positioning only in visually normal distal LM arteries were both applicable. We encourage IVUS examination in patients with concomitant ostial LCX lesions before intervention, compromise of the LCX after stenting, and narrow angle (<90°) between the LAD and LCX measured in the left anterior oblique caudal view. In cases of LCX ostium compromised after ostial LAD stenting, we should choose proper balloon size with the IVUS guidance to minimize the risk of LCX ostial dissection. A final kissing balloon inflation and provisional T stent of the LCX is then performed. In patients with a very low bifurcation angle (<50°) between the axis of the main vessel and the axis of the side-branch at its origin, bifurcation stenting such as crush/culotte stenting was suggested. Additionally, the ostial plaque morphology deserves investigation. Atherosclerotic lesions in the proximal LAD were usually eccentric in one previous study. These lesions appeared to be usually located on the lateral wall, opposite the LCX takeoff. For such eccentric lesions, we can deploy a stent without plaque shift to the LCX ostium irrespective of the angle between LAD and LCX.

No patients developed stent thrombosis within one year in our study. Only 65.9% of our patients were maintained on dual antiplatelet therapy for at least 6 months, which was lower compared to previous reports. About 52.3% of our patients were maintained on clopidogrel at one year. The premature discontinuation of dual antiplatelet therapy may increase the risk of MACE. In 3 of our 4 restenotic cases, dual antiplatelet therapy was prematurely discontinued; of these, 2 patients were on clopidogrel monotherapy due to PU with bleeding and 1 patient had dual therapy for only 2 months. Cilostazol is an antiplatelet drug which has been used as part of an antiplatelet regimen after coronary stent placement. Its antiplatelet action is mainly due to phosphodiesterase III inhibition. Triple therapy (aspirin plus clopidogrel plus cilostazol) resulted in more potent inhibition of platelet aggregation and prevention of stent thrombosis compared with dual antiplatelet therapy. More recently, a randomized, multicenter, prospective study - the DECLARE-DIABETES trial - concluded that the use of cilostazol in diabetes patients treated with sirolimus-eluting stents could reduce restenosis. We can consider adding cilostazol in patients with aspirin intolerance or severe PU disease. However, the effectiveness of using cilostazol plus clopidogrel compared with conventional dual antiplatelet therapy following stenting is not tested in clinical trial.

In the TAXUS IV trial, the implantation of PES in 126 single, proximal LAD lesions was safe and effective. The one-year incidence of TLR (6.3%) and MACE (13.3%) were reported. Our study showed that two stenting strategies may achieve acceptable clinical outcomes in more complex ostial LAD lesions, similar to the outcomes seen in simple, non-ostial LAD lesions as described above. Also, patients with specific lesions such as chronic total occlusion (4.6%), severe calcified lesions (4.6%), restenotic lesions (13.6%) and diffuse long lesions (18.2%) were also included in our study. Although DES in the treatment of the above lesions plus ostial lesions is off-label indication, no major in-hospital complications or MACE during the follow-up period were observed in this group. The clinical results of our study population regarding MACE-free (88.6%) and TLR-free (90.9%) survival are consistent with those obtained in previous studies that have been conducted with DES in de novo ostial LAD lesions.

**Limitations of study**

This investigation was a retrospective, observational study without control group from a single center and
thus has several limitations. Firstly, the study population was relatively small and had selection bias. Secondly, the rate of angiographic follow-up was low (38.6%). Some asymptomatic patients with restenosis were possibly ignored, which would lead to an underestimation of the restenosis rate. Thirdly, routine IVUS before and after stenting, which may provide useful information, was not performed in our study. Fourthly, the inconsistent duration of dual antiplatelet therapy in our patients may have altered the clinical outcomes. Finally, the lower rate of MACE can possibly be explained by small sample size, highly selective patients and low routine CAG follow-up rate. Although generalizability of this strategy to the entire population is uncertain, our data could reflect the real-world registry in daily practice. Further larger, multicenter, long-term, randomized study comparing among different types of DES, stenting strategies and CABG is necessary to find out the best method for treating ostial LAD lesions.

CONCLUSION

Paclitaxel-eluting stent implantation in ostial LAD lesions with complete lesion coverage achieves high procedural success rate and acceptable clinical outcomes during hospitalization and at one-year follow-up period. Our experience is in agreement with recent reports that DES implantation in ostial LAD lesions is feasible.

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REFERENCES

