Intravascular Ultrasound Assessment of Drug-Eluting Stent Coverage of the Coronary Ostium and Effect on Outcomes

Soo-Jin Kang, MD, PhD[a], Jung-Min Ahn, MD[a], Won-Jang Kim, MD[a], Jong-Young Lee, MD[a], Duk-Woo Park, MD, PhD[a], Seung-Whan Lee, MD, PhD[a], Young-Hak Kim, MD, PhD[a], Cheol Whan Lee, MD, PhD[a], Gary S. Mintz, MD[b], Seong-Wook Park, MD, PhD[a], and Seung-Jung Park, MD, PhD[a,*]

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. **(Am J Cardiol 2013;111:1401−1407)**

When stenting an ostial or proximal coronary artery 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] coronary artery or right coronary artery) or into the polygon of confluence of the LM (in the case of the left anterior descending [LAD]). In part because of the radiolucency of the stent, this is difficult to assess angiographically. In contrast, intravascular ultrasound (IVUS) can accurately assess the relation between the proximal end of the stent and the true coronary ostium and provide information regarding expansion and stent−vessel wall apposition that is often incomplete in ostial lesions because of the size of the proximal vessel lumen.[1−5] Thus, the aim of the present study was to use IVUS to assess the relation between the proximal end of a drug-eluting stent (DES) and the coronary ostium to determine whether protrusion, incomplete proximal vessel coverage, or acute malapposition affects subsequent restenosis or major adverse coronary events.

Methods

From March 2008 to September 2010, 493 patients (493 lesions) underwent IVUS-guided DES placement in which the proximal end of the stent was positioned at or near the coronary ostium and in whom 9-month angiography was performed at the Asan Medical Center (Seoul, Korea). The patients were excluded if stent implantation was performed during cardiogenic shock or as a bridge to emergency bypass surgery, if antiplatelet agents were contraindicated, or if the left ventricular ejection fraction was <35%. The lesion-related exclusion criteria were chronic total occlusion, in-stent restenosis, and saphenous vein graft. Because of incomplete IVUS visualization of the ostial segment either before or after stenting, an additional 34 patients were excluded. Thus, a total of 459 lesions (229 LM, 162 LAD, and 68 right coronary ostia) were finally included. All had post-stenting IVUS scans available. After excluding the lesions with predilation before IVUS, preprocedural IVUS scans were available for 354 lesions.

Major adverse coronary events was defined as death from cardiac causes, target lesion revascularization, or myocardial infarction. Revascularization was defined as "ischemia driven" if the angiographic diameter stenosis was ≥50%, with a documented positive functional study such as a thallium scan or treadmill test, ischemic changes on the electrocardiogram, or ischemic symptoms. In addition, lesions with an angiographic diameter stenosis of ≥70% were considered to be "ischemia driven," even in the absence of

[a]Department of Cardiology, University of Ulsan College of Medicine, Asan Medical Center, Seoul, Korea; and [b]Cardiovascular Research Foundation, New York, New York. Manuscript received December 6, 2012; revised manuscript received and accepted January 20, 2013.

This study was supported by a grant of the Korea Healthcare Technology Research and Development Project, Ministry of Health and Welfare, A120711, and Cardiovascular Research Foundation (Seoul, Republic of Korea).

See page 1406 for disclosure information.

*Corresponding author: Tel: (82) 2-3010-4812; fax: (82) 2-475-6898.
E-mail address: sjpark@amc.seoul.kr (S.-J. Park).

0002-9149/13/$ - see front matter
http://dx.doi.org/10.1016/j.amjcard.2013.01.291

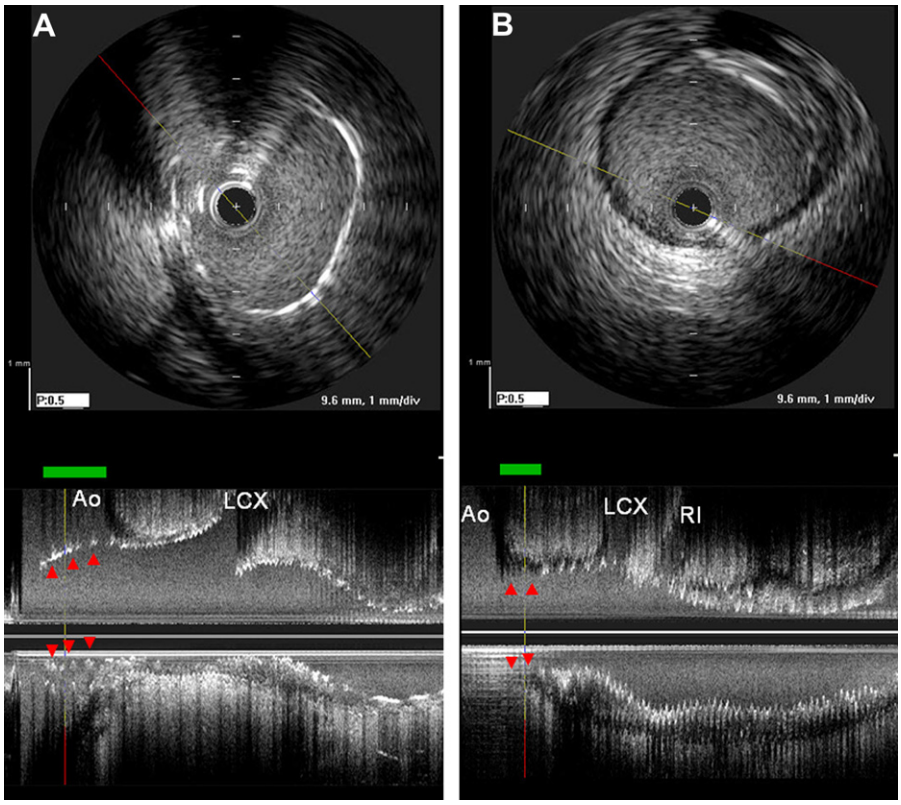

Figure 1. *(A)* LM coronary artery ostium with strut protrusion into aorta *(arrows)*, with length of protruded struts of 4.8 mm *(green bar)*. *(B)* LM coronary artery with uncovered ostium *(arrows)*, with length of ostial segment without stent coverage of 3.0 mm *(green bar)*.

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.[6] 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).[7] 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.[8]

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 mm^2 for the LM and <6.0 mm^2 for the LAD and right coronary arteries.[9] The IVUS definition of each ostium paralleled the angiographic definition.[10] 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). Malapposition 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.[11]

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

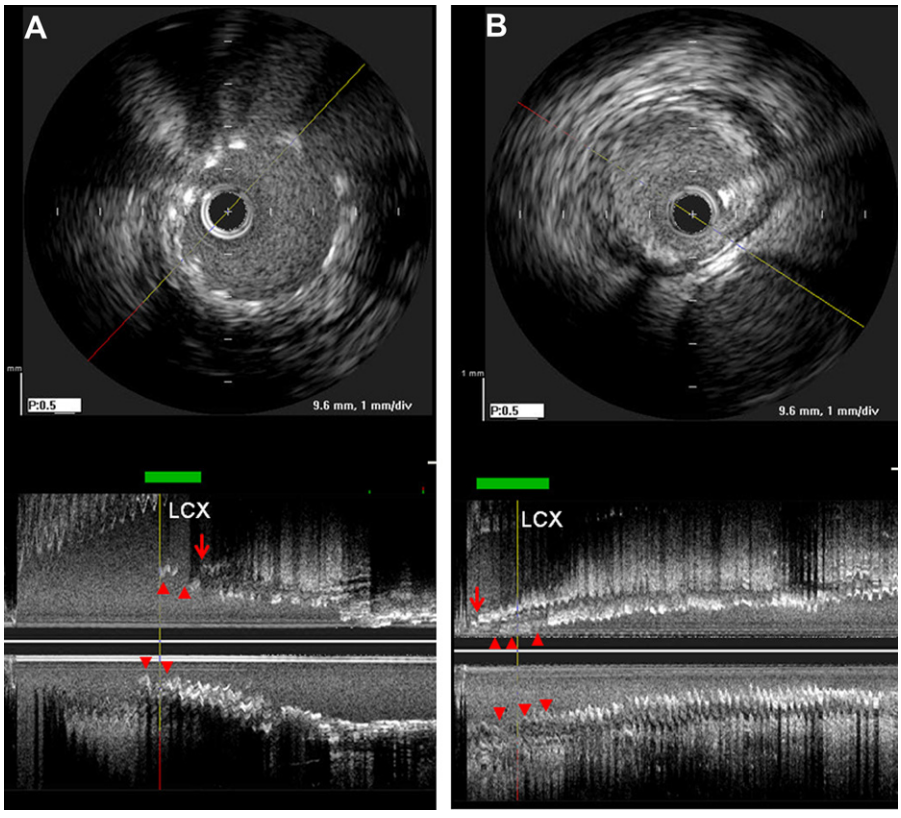

Figure 2. *(A)* LAD ostium with strut protrusion into polygon of confluence zone of distal LM coronary artery *(arrows)*, with length of strut protrusion above carina *(arrow)* of 2.4 mm *(green bar)*. *(B)* LAD ostium with uncovered ostium *(arrows)*, with length of uncovered ostial segment of 3.0 mm *(green bar)*.

Table 1
Clinical and procedural characteristics (n = 459)

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

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.

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 2
Quantitative coronary angiographic data

	LMCA	LAD	RCA	p Value
Lesions (n)	229	162	68	—
Preprocedure angiographic data				
Minimum lumen diameter (mm)	1.5 ± 0.7*,†	1.1 ± 0.9	1.1 ± 0.6	<0.001
Diameter stenosis (%)	57.9 ± 16.4*,†	68.7 ± 14.1	70.4 ± 16.3	<0.001
Lesion length (mm)	30.5 ± 16.4	34.1 ± 14.5	30.9 ± 18.4	0.092
Thrombolysis In Myocardial Infarction flow 3	197 (87%)	138 (86%)	55 (81%)	
Post-stenting angiographic data				
Minimum lumen diameter (mm)	3.2 ± 0.6*	2.7 ± 0.4	3.1 ± 0.4	<0.001
In-stent diameter stenosis (%)	6.0 ± 8.4†	7.2 ± 7.3	8.7 ± 6.7	0.031
Thrombolysis In Myocardial Infarction flow 3	229 (100%)	162 (100%)	68 (100%)	1.000
Follow-up angiographic data				
Minimum lumen diameter (mm)	2.9 ± 0.7*,†	2.5 ± 0.5	2.6 ± 0.7	<0.001
In-stent diameter stenosis (%)	14.3 ± 15.2†	17.0 ± 13.4‡	26.1 ± 19.1	<0.001
Angiographic in-stent restenosis	10 (4.4%)	6 (3.7%)	8 (11.8%)	<0.001
Marginal	2 (20%)	3 (50%)	4 (50%)	0.532
Focal body	4 (40%)	2 (33%)	4 (50%)	
Diffuse in-stent	2 (20%)	1 (17%)	0 (0%)	
Total occlusion	2 (20%)	0 (0%)	0 (0%)	

* p <0.05, LM coronary artery versus LAD.
† p <0.05, LM coronary artery versus right coronary artery.
‡ p <0.05, LAD versus right coronary artery.

Table 3
Pre- and post-stenting intravascular ultrasound (IVUS) data

	Coronary Artery			p Value
	LM	LAD	Right	
Preprocedural intravascular ultrasound of ostial segment	199	117	38	
Lumen area (mm^2)	8.6 ± 4.5*,†	5.8 ± 2.9	5.3 ± 2.5	<0.001
External elastic membrane area (mm^2)	21.3 ± 5.9*,†	15.8 ± 4.1	15.5 ± 4.7	<0.001
Plaque burden (%)	60.1 ± 15.5	63.8 ± 14.6	65.1 ± 14.7	0.052
Post-stenting intravascular ultrasound	229	162	68	
In-segment				
Minimum stent area (mm^2)	7.8 ± 2.7*	5.7 ± 1.7‡	7.9 ± 2.0	<0.001
External elastic membrane area at minimum stent area site (mm^2)	16.8 ± 7.6*	10.9 ± 4.1‡	15.9 ± 4.7	<0.001
Ostial segment				
Stent area (mm^2)	11.0 ± 2.6*,†	8.6 ± 1.6‡	10.2 ± 2.1	<0.001
External elastic membrane area (mm^2)	23.6 ± 5.0*,†	17.8 ± 3.4‡	19.7 ± 4.0	<0.001
Stent/vessel area ratio	0.47 ± 0.09†	0.49 ± 0.07	0.52 ± 0.08	<0.001
Strut protrusion	156 (68%)*,†	86 (53%)	40 (59%)	0.010
Length of strut protrusion (mm)	3.4 ± 1.7*,†	1.7 ± 1.0	2.4 ± 1.4	<0.001
Strut protrusion >2 mm	123 (54%)*,†	25 (15%)‡	21 (31%)	<0.001
Strut protrusion >3 mm	89 (39%)*,†	6 (4%)‡	14 (21%)	<0.001
Incomplete ostial stent coverage	53 (23%)	54 (33%)	19 (28%)	0.084
Uncovered segment length (mm)	−2.3 ± 1.3	−1.8 ± 1.1	−1.7 ± 1.0	0.050
Uncovered segment >2 mm	28 (12%)	22 (14%)	7 (10%)	0.782
Plaque burden within uncovered ostial segment (%)	38.1 ± 11.9*	45.1 ± 10.8	40.6 ± 8.5	0.006
Malapposition ostium	43 (19%)*,†	10 (6%)	9 (12%)	0.001

* p <0.05, LM versus LAD.
† p <0.05, LM versus right coronary artery.
‡ p <0.05, LAD versus right coronary artery.

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 5 (3.0%) showed ostial restenosis. Among 109 lesions with >3 mm of stent 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

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

	Coronary Ostial Lesion			Nonostial Coronary Lesion		
	LM	LAD	Right	LM	LAD	Right
Preprocedural intravascular ultrasound	57	23	15	142	94	23
Lumen area within ostial segment (mm^2)	5.0 ± 2.0	3.1 ± 1.6	4.6 ± 2.3	10.0 ± 4.5*	6.4 ± 2.7*	5.7 ± 2.5
External elastic membrane area within ostial segment (mm^2)	18.6 ± 5.3	14.9 ± 4.5	14.3 ± 5.1	22.4 ± 5.9*	16.0 ± 4.0	16.2 ± 4.4
Plaque burden within ostial segment (%)	71.5 ± 10.9	78.9 ± 9.4	66.1 ± 15.7	55.4 ± 14.6*	60.1 ± 13.2*	64.4 ± 14.3
Post-stenting intravascular ultrasound	65	43	30	164	119	38
Stent area within ostial segment (mm^2)	10.9 ± 2.3	8.3 ± 1.6	9.7 ± 1.9	11.0 ± 2.7	8.7 ± 1.6	10.6 ± 2.1
External elastic membrane area within ostial segment (mm^2)	22.5 ± 5.2	17.5 ± 3.5	19.4 ± 4.4	24.1 ± 4.8*	17.9 ± 3.5	20.1 ± 3.7
Lesions with strut protrusion	61 (94%)	31 (72%)	25 (83%)	95 (58%)*	55 (46%)*	15 (40%)*
Strut protrusion length (mm)	3.7 ± 1.7	1.7 ± 1.0	2.4 ± 1.5	3.1 ± 1.7*	1.7 ± 1.0	2.3 ± 1.3
Strut protrusion length >2 mm	54 (83%)	9 (21%)	12 (40%)	69 (42%)*	16 (13%)	9 (24%)
Strut protrusion length >3 mm	38 (59%)	3 (7%)	9 (30%)	51 (31%)*	3 (3%)	5 (13%)
Incomplete ostial stent coverage	1 (2%)	6 (14%)	2 (7%)	52 (32%)*	48 (40%)*	17 (45%)*
Uncovered segment length (mm)	−0.4	−1.6 ± 1.0	−1.6 ± 1.4	−2.4 ± 1.3	−1.8 ± 1.1	−1.7 ± 1.0
Uncovered segment >2 mm	0 (0%)	2 (5%)	1 (3%)	28 (17%)*	20 (17%)	6 (16%)
Plaque burden within uncovered ostial segment (%)	45.8 ± 14.0	44.6 ± 14.0	41.7 ± 9.5	37.8 ± 12.0	45.1 ± 10.5	40.4 ± 8.5
Malapposition	9 (14%)	0 (0%)	3 (10%)	34 (21%)	10 (8%)*	5 (13%)

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

Table 5
Patients with ostial restenosis

Pt. No.	Lesion Location	Ostial Lesion	Protrusion	Protrusion Length (mm)	Incomplete Coverage	Length of Incomplete Coverage (mm)	Plaque Burden of Uncovered Ostium (%)	Acute Malapposition	Stent Area at Ostium (mm^2)	EEM Area Ostium (mm^2)
1	LM	Yes	Yes	2.4	No			No	10.3	13.92
2	LM	No	No		Yes	2.2	40.3	Yes	10.3	17.28
3	LM	No	No		Yes	4.3	48.7	No	10.5	20.5
4	LM	Yes	Yes	3.6	No			No	7.0	14.83
5	LAD	No	No		Yes	1.6	54.9	No	6.3	14.02
6	LAD	No	Yes	1.8	No			No	7.9	15.55
7	Right	Yes	Yes	3.4	No			No	9.5	14.67
8	Right	Yes	Yes	5.6	No			No	9.9	22.15
9	Right	Yes	Yes	1.2	No			Yes	10.1	22.01
10	Right	No	Yes	2.2	No			No	11.9	22.4
11	Right	Yes	Yes	1.2	No			No	12.1	22.94
12	Right	Yes	Yes	1.9	No			No	9.0	16.57
13	Right	No	No		No			No	10.6	20.8

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 mm^2, $p = 0.050$) or after stenting (17.9 ± 3.5 vs 21.1 ± 5.1 mm^2, $p = 0.025$) 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 mm^2, $p = 0.7$), 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 mm^2 vs 10.0 ± 2.5 mm^2, $p = 0.7$).

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.[12−14] Histologic data[15,16] 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.[17]

DES treatment of aorto-ostial lesions appeared safe and effective, with a significant improvement in restenosis and late clinical events.[18,19] 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.[10]

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.[15,16]

For nonostial coronary lesions, stent underexpansion was an important predictor of restenosis.[1,2] 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.[3,5] Consistent with data from nonostial lesions, acute malapposition at the ostium did not affect the incidence of restenosis or major adverse coronary events.[11,20,21]

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.

1. Sonoda S, Morino Y, Ako J, Terashima M, Hassan AH, Bonneau HN, Leon MB, Moses JW, Yock PG, Honda Y, Kuntz RE, Fitzgerald PJ, SIRIUS Investigators. Impact of final stent dimensions on long-term results following sirolimus-eluting stent implantation: serial intravascular ultrasound analysis from the SIRIUS trial. *J Am Coll Cardiol* 2004;43:1959−1963.
2. Hong MK, Mintz GS, Lee CW, Park DW, Choi BR, Park KH, Kim YH, Cheong SS, Song JK, Kim JJ, Park SW, Park SJ. Intravascular ultrasound predictors of angiographic restenosis after sirolimus-eluting stent implantation. *Eur Heart J* 2006;27:1305−1310.
3. Sakurai R, Ako J, Morino Y, Sonoda S, Kaneda H, Terashima M, Hassan AH, Leon MB, Moses JW, Popma JJ, Bonneau HN, Yock PG, Fitzgerald PJ, Honda Y; SIRIUS Trial Investigators. Predictors of edge stenosis following sirolimus-eluting stent deployment (a quantitative intravascular ultrasound analysis from the SIRIUS trial). *Am J Cardiol* 2005;96:1251−1253.
4. Morino Y, Tamiya S, Masuda N, Kawamura Y, Nagaoka M, Matsukage T, Ogata N, Nakazawa G, Tanabe T, Ikari Y. Intravascular ultrasound criteria for determination of optimal longitudinal positioning of sirolimus-eluting stents. *Circ J* 2010;74:1609−1616.
5. Liu J, Maehara A, Mintz GS, Weissman NJ, Yu A, Wang H, Mandinov L, Popma JJ, Ellis SG, Grube E, Dawkins KD, Stone GW. An integrated TAXUS IV, V, and VI intravascular ultrasound analysis of the predictors of edge restenosis after bare metal or paclitaxel-eluting stents. *Am J Cardiol* 2009;103:501−506.
6. Cutlip DE, Windecker S, Mehran R, Boam A, Cohen DJ, van Es GA, Steg PG, Morel MA, Mauri L, Vranckx P, McFadden E, Lansky A, Hamon M, Krucoff MW, Serruys PW; Academic Research Consortium. Clinical end points in coronary stent trials: a case for standardized definitions. *Circulation* 2007;115:2344−2351.
7. Ryan TJ, Faxon DP, Gunnar RM, Kennedy JW, King SB III, Loop FD, Peterson KL, Reeves TJ, Williams DO, Winters WL Jr. Guidelines for percutaneous transluminal coronary angioplasty: a report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Percutaneous Transluminal Coronary Angioplasty). *Circulation* 1988;78:486−502.
8. Mehran R, Dangas G, Abizaid AS, Mintz GS, Lansky AJ, Satler LF, Pichard AD, Kent KM, Stone GW, Leon MB. Angiographic patterns of in-stent restenosis: classification and implications for long-term outcome. *Circulation* 1999;100:1872−1878.
9. Kang SJ, Ahn JM, Song H, Kim WJ, Lee JY, Park DW, Yun SC, Lee SW, Kim YH, Lee CW, Mintz GS, Park SW, Park SJ. Comprehensive intravascular ultrasound assessment of stent area and its impact on restenosis and adverse cardiac events in 403 patients with unprotected left main disease. *Circ Cardiovasc Interv* 2011;4:562−569.
10. Park DW, Hong MK, Suh IW, Hwang ES, Lee SW, Jeong YH, Kim YH, Lee CW, Kim JJ, Park SW, Park SJ. Results and predictors of angiographic restenosis and long-term adverse cardiac events after drug-eluting stent implantation for aorto-ostial coronary artery disease. *Am J Cardiol* 2007;99:760−765.

11. Hong MK, Mintz GS, Lee CW, Park DW, Park KM, Lee BK, Kim YH, Song JM, Han KH, Kang DH, Cheong SS, Song JK, Kim JJ, Park SW, Park SJ. Late stent malapposition after drug-eluting stent implantation: an intravascular ultrasound analysis with long-term follow-up. *Circulation* 2006;113:414−419.
12. Chin K. An approach to ostial lesion management. *Curr Interv Cardiol Rep* 2001;3:87−89.
13. Toutouzas K, Stankovic G, Takagi T, Spanos V, Di Mario C, Albiero R, Corvaja N, Gaglione A, Colombo A. Outcome of treatment of aorto-ostial lesions involving the right coronary artery or a saphenous vein graft with a polytetrafluoroethylene-covered stent. *Am J Cardiol* 2002;90:63−66.
14. Hoffmann R, Mintz GS, Mehran R, Pichard AD, Kent KM, Satler LF, Popma JJ, Wu H, Leon MB. Intravascular ultrasound predictors of angiographic restenosis in lesions treated with Palmaz-Schatz stents. *J Am Coll Cardiol* 1998;31:43−49.
15. Stewart JT, Ward DE, Davies MJ, Pepper JR. Isolated coronary ostial stenosis: observations on the pathology. *Eur Heart J* 1987;8:917−920.
16. Popma JJ, Dick RJ, Haudenschild CC, Topol EJ, Ellis SG. Atherectomy of right coronary ostial stenoses: initial and long-term results, technical features and histologic findings. *Am J Cardiol* 1991;67:431−433.
17. Tsunoda T, Nakamura M, Wada M, Ito N, Kitagawa Y, Shiba M, Yajima S, Iijima R, Nakajima R, Yamamoto M, Takagi T, Yoshitama T, Anzai H, Nishida T, Yamaguchi T. Chronic stent recoil plays an important role in restenosis of the right coronary ostium. *Coron Artery Dis* 2004;15:39−44.
18. Iakovou I, Ge L, Michev I, Sangiorgi GM, Montorfano M, Airoldi F, Chieffo A, Stankovic G, Vitrella G, Carlino M, Corvaja N, Briguori C, Colombo A. Clinical and angiographic outcome after sirolimus-eluting stent implantation in aorto-ostial lesions. *J Am Coll Cardiol* 2004;44: 967−971.
19. Seung KB, Kim YH, Park DW, Lee BK, Lee CW, Hong MK, Kim PJ, Chung WS, Tahk SJ, Park SW, Park SJ. Effectiveness of sirolimus-eluting stent implantation for the treatment of ostial left anterior descending artery stenosis with intravascular ultrasound guidance. *J Am Coll Cardiol* 2005;46:787−792.
20. Kimura M, Mintz GS, Carlier S, Takebayashi H, Fujii K, Sano K, Yasuda T, Costa RA, Costa JR Jr, Quen J, Tanaka K, Lui J, Weisz G, Moussa I, Dangas G, Mehran R, Lansky AJ, Kreps EM, Collins M, Stone GW, Moses JW, Leon MB. Outcome after acute incomplete sirolimus-eluting stent apposition as assessed by serial intravascular ultrasound. *Am J Cardiol* 2006;98:436−442.
21. Tanabe K, Serruys PW, Degertekin M, Grube E, Guagliumi G, Urbaszek W, Bonnier J, Lablanche JM, Siminiak T, Nordrehaug J, Figulla H, Drzewiecki J, Banning A, Hauptmann K, Dudek D, Bruining N, Hamers R, Hoye A, Ligthart JM, Disco C, Koglin J, Russell ME, Colombo A; TAXUS II Study Group. Incomplete stent apposition after implantation of paclitaxel-eluting stents or bare metal stents: insights from the randomized TAXUS-II trial. *Circulation* 2005;111:900−905.