Intravascular Ultrasound Predictors for Edge Restenosis After Newer Generation Drug-Eluting Stent Implantation

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The aim of the present study was to assess the intravascular ultrasound predictors for angiographic edge restenosis after newer generation drug-eluting stent implantation. A total of 820 patients (987 lesions) who underwent newer generation drug-eluting stent placement (236 Endeavor zotarolimus-eluting stents, 246 Resolute zotarolimus-eluting stents, and 505 everolimus-eluting stents) with 9 months of angiographic surveillance were enrolled. The post-stenting angiographic and intravascular ultrasound images of 1,668 reference segments (681 proximal and 987 distal) were analyzed. Overall, 37% of angiographically normal proximal reference segments and 21% of angiographically normal distal reference segments had plaque burden >50%. In the overall cohort of 1,668 reference segments, 47 (2.8%) had 9-month angiographic edge restenosis (diameter stenosis >50%). Edge restenosis was predicted by a post-stenting reference segment plaque burden >54.5% (sensitivity 81%, specificity 80%) and a reference segment minimum lumen area of 5.7 mm^2 (sensitivity 72%, specificity 59%). The edge restenosis rate was 2.1% in the Endeavor zotarolimus-eluting stents, 2.4% in the Resolute zotarolimus-eluting stents, and 3.4% in the everolimus-eluting stents lesions ($p = 0.311$). The predictive cutoff of the reference plaque burden was 56.3% for Endeavor zotarolimus-eluting stents, 57.3% for Resolute zotarolimus-eluting stents, and 54.2% for everolimus-eluting stents. The criteria for residual plaque burden were similar between proximal and distal reference segments (56.4% vs 51.9%, respectively), but the minimum lumen area criteria were quite different (<7.1 mm^2 for proximal vs <4.8 mm^2 for distal reference segments). In conclusion, after newer drug-eluting stent implantation, edge restenosis was predicted by post-stenting reference segment plaque burden >55%. **(Am J Cardiol 2013;111:1408−1414)**

Stent underexpansion and incomplete lesion coverage are consistent and important procedural factors responsible for stent failure.[1−8] Intravascular ultrasound (IVUS) studies have reported that the reference segment plaque burden predicted both edge restenosis and stent thrombosis in lesions treated with bare metal stents and first-generation drug-eluting stents (DESs).[4−8] The newer generation DESs differ from the first-generation devices regarding antiproliferative agents, polymer coatings, and metallic platforms. Although the newer DESs are generally used in daily practice, because they provide better safety and efficacy, no IVUS optimization criteria are available to determine the appropriate landing zones suitable for stent deployment to prevent edge restenosis and improve the long-term clinical outcomes.[9,10] Thus, the aim of the present study was to assess the IVUS cutoffs for the reference segment plaque burden and lumen area to predict angiographic edge restenosis in patients undergoing newer generation DES implantation.

Methods

From January 2008 to August 2010, 820 patients (with 987 lesions) underwent newer generation DES implantation in ≥1 of 3 major coronary arteries with 9 months of follow-up angiographic surveillance at the Asan Medical Center, Seoul, Korea. The exclusion criteria were chronic total occlusion, in-stent restenosis, left main or saphenous vein graft lesions, and vessels with a reference lumen diameter <2.5 mm. The newer generation DESs used were the Endeavor zotarolimus-eluting stent (Medtronic, Santa Rosa, California) in 236 lesions, the Resolute zotarolimus-eluting stent (Medtronic) in 246 lesions, and everolimus-eluting stent (EES, Xience V, Abbott Vascular, Santa Clara, California, and Promus, Boston Scientific, Natick, Massachusetts) in 505 lesions. In 306 lesions, no proximal reference segment was present, because the lesions were ostial in location or were just distal to a major branch; therefore, a total of 1,668 reference segments (681 proximal and 987 distal reference segments) were included in the IVUS analysis.

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Table 1
Clinical and procedural characteristics (n = 820 patients)

Variable	Total (n = 820)	Edge Restenosis		p Value
		With (n = 42)	Without (n = 778)	
Age (yrs)	60 ± 9	58 ± 9	60 ± 9	0.163
Men	199 (24%)	8 (19%)	191 (25%)	0.272
Smoker	447 (55%)	20 (48%)	427 (55%)	0.223
Hypertension*	474 (58%)	26 (62%)	448 (58%)	0.350
Hyperlipidemia†	546 (67%)	28 (67%)	518 (67%)	0.569
Diabetes mellitus	286 (35%)	17 (41%)	269 (35%)	0.266
Ejection fraction (%)	59 ± 6	59 ± 9	59 ± 6	0.931
Previous bypass surgery	13 (2%)	0 (0%)	13 (2%)	0.502
Previous myocardial infarction	29 (4%)	1 (2%)	28 (4%)	0.556
Previous stroke	46 (6%)	9 (10%)	42 (5%)	0.204
Renal failure	7 (1%)	0 (0%)	7 (1%)	0.691
Multivessel disease	479 (58%)	30 (71%)	449 (58%)	0.053
Clinical presentation				0.963
Stable angina pectoris	550 (67%)	30 (71%)	520 (67%)	
Unstable angina pectoris	183 (22%)	7 (17%)	176 (22%)	
Acute myocardial infarction	87 (11%)	5 (12%)	82 (11%)	

Data are presented 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.

Revascularization was defined as "ischemia driven" if angiographic diameter stenosis (DS) of ≥50% was present, with a documented positive functional study, ischemic changes on an electrocardiogram, or ischemic symptoms. In addition, lesions with angiographic DS of ≥70%, as assessed by quantitative coronary analysis, were considered to be "ischemia-driven," even in the absence of 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 determined using the Academic Research Consortium criteria.[11] All patients provided written informed consent, and the ethics committee approved our study.

Qualitative and quantitative angiographic analysis was done using standard techniques with automated edge-detection algorithms (CAAS-5, Pie Medical Imaging, Maastricht, The Netherlands) in the angiographic analysis center of the CardioVascular Research Foundation (Seoul, Korea).[12] On the final post-stenting angiogram, the minimal lumen diameter and DS were measured within the stent (in-stent) and within 5 mm of the proximal and distal edges of the stent. For bifurcation lesions, angiographic measurement was performed only in the main vessel. Angiographic restenosis was defined as DS >50% at the follow-up examination. The patterns of angiographic restenosis were assessed using the Mehran classification.[13]

Final post-stenting 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 stent area and external elastic membrane area as measured using 2-dimensional planimetry. At both the proximal and the distal reference segments (5-mm-long segment adjacent to the stent edge), the minimum lumen area (MLA) and external elastic membrane area were measured. The maximum plaque burden within the reference segment was calculated as plaque/external elastic membrane × 100 (%).

All statistical analyses were performed using SPSS, version 10.0 (SPSS, Chicago, Illinois). All values are expressed as the mean ± SD (continuous variables) or as counts and percentages (categorical variables). Continuous variables were compared using the unpaired *t* test or nonparametric Mann-Whitney *U* test. Categorical variables were compared using chi-square statistics or Fisher's exact test. In post hoc analysis, all IVUS parameters were compared among the 3 DES types. Bonferroni corrections were made for multiple comparisons of continuous variables. All p values were 2-sided, and p values after Bonferroni correction of <0.05 were considered statistically significant.

To predict edge restenosis within the corresponding reference segment, a receiver operating characteristic curve was used to identify the optimal cutoff value of the reference segment plaque burden that minimized the distance between the curve and upper corner, using MedCalc (MedCalc Software, Mariakerke, Belgium). The sensitivity and specificity were obtained. A p value <0.05 was considered statistically significant.

Results

The clinical characteristics of the 820 patients are summarized in Table 1. The quantitative coronary angiographic data from the 987 lesions with 1,668 reference segments (681 proximal and 987 distal) are summarized in Table 2. With a follow-up duration of 8.7 ± 2.6 months,

Table 2
Quantitative coronary angiographic data from 987 lesions

Variable	Total	E-ZES	R-ZES	EES
Lesion number	987	236	246	505
Proximal reference segments	681	197	176	308
Distal reference segments	987	236	246	505
Left anterior descending artery	622 (63%)	139 (59%)	154 (63%)	329 (65%)
Left circumflex artery	119 (12%)	29 (12%)	35 (14%)	55 (11%)
Right coronary artery	246 (25%)	68 (29%)	57 (23%)	121 (24%)
Preprocedure angiographic data				
Proximal reference lumen diameter (mm)	3.7 ± 0.5	3.6 ± 0.4	3.7 ± 0.4	3.6 ± 0.5
Distal reference lumen diameter (mm)	2.7 ± 0.5	2.7 ± 0.5	2.7 ± 0.5	2.7 ± 0.5
Minimum lumen diameter (mm)	1.1 ± 0.6	1.0 ± 0.5	1.0 ± 0.5	1.1 ± 0.7
Diameter stenosis (%)	67.7 ± 14.5	67.9 ± 15.4	69.8 ± 14.3	66.5 ± 13.9
Lesion length (mm)	28.6 ± 16.0	27.5 ± 15.4	27.9 ± 15.2	29.5 ± 16.7
Post-stenting angiographic data				
Proximal reference lumen diameter (mm)	3.2 ± 0.5	3.2 ± 0.5	3.3 ± 0.6	3.2 ± 0.5
Proximal reference diameter stenosis (%)	10.0 ± 9.1	8.5 ± 8.1	9.5 ± 9.5	11.2 ± 9.3
Distal reference lumen diameter (mm)	2.5 ± 0.6	2.6 ± 0.6	2.5 ± 0.5	2.4 ± 0.5
Distal reference diameter stenosis (%)	14.7 ± 10.2	13.8 ± 9.3	14.5 ± 10.4	15.2 ± 10.4
Total stent length (mm)	36.1 ± 16.2	34.5 ± 14.3	36.4 ± 15.8	36.7 ± 17.1
In-stent minimum lumen diameter (mm)	2.8 ± 0.5	2.9 ± 0.5	2.9 ± 0.5	2.8 ± 0.5
In-stent diameter stenosis (%)	6.9 ± 7.8	7.9 ± 6.3	6.4 ± 7.9	6.7 ± 8.4
Thrombolysis In Myocardial Infarction flow 3	987 (100%)	236 (100%)	246 (100%)	505 (100%)
Follow-up angiographic data				
Proximal reference lumen diameter (mm)	3.0 ± 0.6	3.0 ± 0.6	3.1 ± 0.6	3.0 ± 0.6
Proximal reference diameter stenosis (%)	15.8 ± 13.2	15.1 ± 13.3	14.9 ± 13.8	16.8 ± 12.9
Distal reference lumen diameter (mm)	2.4 ± 0.6	2.3 ± 0.6	2.6 ± 0.5	2.6 ± 0.6
Distal reference diameter stenosis (%)	17.2 ± 12.1	28.1 ± 16.7	14.9 ± 11.7	15.7 ± 15.1
In-stent minimal lumen diameter (mm)	2.5 ± 0.6	2.5 ± 0.6	2.4 ± 0.5	2.4 ± 0.6
In-stent diameter stenosis (%)	18.5 ± 15.7	16.7 ± 11.3	17.2 ± 10.8	17.5 ± 12.9
9-Mo angiographic edge restenosis				
Proximal edge restenosis	24 (2.4%)	5 (2.1%)	7 (2.8%)	12 (2.4%)
Distal edge restenosis	19 (1.9%)	4 (1.7%)	3 (1.2%)	12 (2.4%)
Both	2 (0.2%)	0 (0%)	0 (0%)	2 (0.4%)
9-Mo angiographic in-stent restenosis	60 (6.1%)	26 (11.0%)	11 (4.5%)	23 (4.5%)
Marginal	26 (43.3%)	4 (15.4%)	9 (81.8%)	13 (56.5%)
Focal body	17 (28.3%)	9 (34.6%)	2 (18.2%)	6 (26.1%)
Diffuse in-stent	8 (13.3%)	8 (30.8%)	0 (0%)	0 (0%)
Proliferative	7 (11.7%)	5 (19.2%)	0 (0%)	2 (8.7%)
Total occlusion	2 (3.3%)	0 (0%)	0 (0%)	2 (8.7%)

Data are presented as n, n (%), or mean ± SD.
E-ZES = Endeavor zotarolimus-eluting stent; R-ZES = Resolute zotarolimus-eluting stent.

angiographic edge restenosis and in-stent restenosis was observed in 45 (4.6%) and 60 (6.0%) lesions, respectively.

The post-stenting IVUS data are listed in Table 3. Post-stenting angiographic DS showed only a weak correlation with maximum plaque burden at the proximal ($r = 0.293$, $p < 0.001$) or distal ($r = 0.155$, $p < 0.001$) reference segments (Figure 1). Of 785 normal-looking proximal reference segments (post-stenting DS <20%), 290 (37%) had a maximum plaque burden >50%. In 724 normal-looking distal reference segments (post-stenting DS <20%), 153 (21%) also had a maximum plaque burden >50%.

The angiographic and IVUS findings between the reference segments with angiographic edge restenosis and those without edge restenosis are listed in Table 4. In both proximal and distal reference segments, edge restenosis was associated with a smaller post-stenting IVUS reference segment MLA, a larger post-stenting IVUS reference segment plaque burden, and a greater reference segment angiographic DS. In addition, only a trend was seen for longer stent length (40.4 ± 19.0 mm vs 35.9 ± 16.0 mm, $p = 0.070$) in lesions with in-stent restenosis. However, edge restenosis was not related to the reference segment external elastic membrane area, edge dissection, or plaque rupture.

In the overall cohort of 1,668 reference segments, 47 (2.8%) were angiographic edge restenosis: 24 with proximal edge restenosis, 19 with distal edge restenosis, and 2 with both proximal and distal edge restenosis. A post-stenting reference segment plaque burden >54.5% predicted edge restenosis, with a sensitivity of 81% and specificity of 80% (Figure 2). No edge restenosis was present in 99% of the reference segments with a post-stenting edge plaque burden of ≤54.5%. A post-stenting reference segment MLA <5.7 mm^2 also predicted edge restenosis, with a sensitivity of 72% and specificity of 59%.

Edge restenosis was found in 9 of 433 Endeavor zotarolimus-eluting stent lesions (2.1%), 10 of 422 Resolute

Table 3
Post-stenting intravascular ultrasound (IVUS) findings

Variable	Total	E-ZES	R-ZES	EES
Proximal reference segments	681	197	176	308
Minimum lumen area (mm^2)	9.0 ± 3.4	8.9 ± 3.6	9.2 ± 3.2	8.9 ± 3.5
External elastic membrane area at minimum lumen area site (mm^2)	17.4 ± 5.6	17.2 ± 5.5	17.5 ± 5.0	17.5 ± 5.9
Maximum plaque burden (%)	48.1 ± 11.5	47.9 ± 11.2	47.4 ± 11.5	48.7 ± 11.7
Proximal edge dissection	5 (0.7%)	1 (0.5%)	1 (0.6%)	2 (1.0%)
Distal reference segments	987	236	246	505
Minimum lumen area (mm^2)	5.7 ± 2.8	5.8 ± 2.6	5.6 ± 2.3	5.7 ± 3.0
External elastic membrane area at minimum lumen area site (mm^2)	9.8 ± 5.2	10.0 ± 5.3	9.4 ± 4.3	9.9 ± 5.5
Maximum plaque burden (%)	38.6 ± 14.6	38.7 ± 14.2	38.1 ± 14.5	38.6 ± 14.8
Distal edge dissection	14 (1.4%)	5 (2.1%)	0 (0%)	9 (1.8%)
In-stent segments	987	236	246	505
Minimum stent area (mm^2)	6.3 ± 2.2	6.3 ± 2.3	6.1 ± 1.9	6.3 ± 2.2
External elastic membrane area at minimum stent area site (mm^2)	12.3 ± 5.0	12.7 ± 5.2	12.0 ± 4.5	12.2 ± 5.1

Data are presented as n, n (%), or mean ± SD.
Abbreviations as in Table 2.

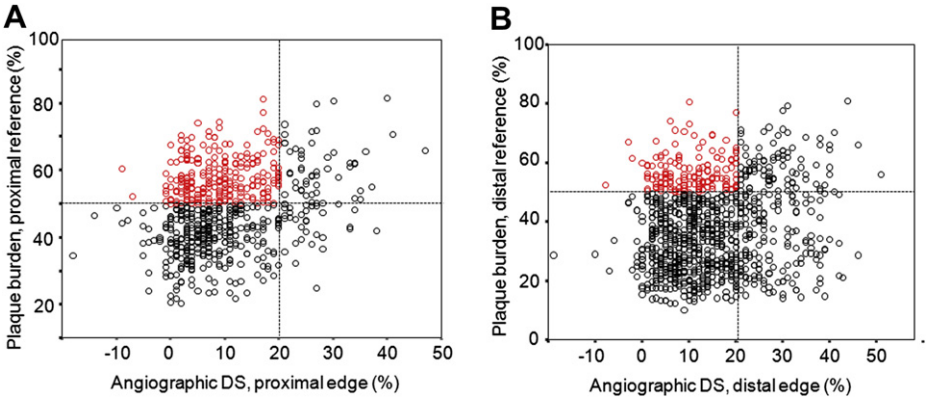

Figure 1. *(A)* Of 785 normal-looking proximal reference segments with post-stenting angiographic DS <20%, 290 (37%) had reference segment maximal plaque burden >50%. *(B)* Of 724 distal reference segments with DS <20%, 153 (21%) had plaque burden >50%.

zotarolimus-eluting stent lesions (2.4%), and 28 of 813 EES lesions (3.4%; p = 0.311). To predict edge restenosis, the best cutoff for the post-stenting reference plaque burden was 56.3% for Endeavor zotarolimus-eluting stents (sensitivity 67%, specificity 86%), 57.3% for Resolute zotarolimus-eluting stents (sensitivity 80%, specificity 87%), and 54.2% for EES (sensitivity 86%, specificity 80%; Figure 3).

Of 681 proximal reference segments, 26 (3.8%) showed proximal edge restenosis. The follow-up angiographic DS at the proximal edge correlated with the post-stenting reference plaque burden (r = 0.273, p <0.001) and MLA (r = −0.293, p <0.001). For the prediction of proximal edge restenosis, the cutoff for the reference plaque burden was 56.4%, and the cutoff for the reference segment MLA was 7.1 mm^2.

Of 987 distal reference segments, 21 (2.1%) showed distal edge restenosis. Follow-up angiographic DS at the distal edge had a positive correlation with the post-stenting reference segment plaque burden (r = 0.149, p <0.001) and a negative correlation with post-stenting MLA of the distal reference segment (r = −0.294, p <0.001). For the prediction of distal edge restenosis, the cutoff for the reference plaque burden was 51.9%, and the cutoff for the reference segment MLA was 4.8 mm^2.

Edge dissection without flow limitation was seen in 5 proximal reference segments (0.7%) and 14 distal reference segments (1.4%). Stent thrombosis occurred in 1 lesion with distal edge dissection 2 weeks after stenting, but no edge restenosis was seen at 9 months in lesions with post-stent edge dissection.

In the overall cohort of 820 patients, the mean clinical follow-up duration was 29.9 ± 12.7 months (median 29.7, interquartile range 20.0 to 39.4). Major adverse cardiac events occurred in 53 patients (6.5%) at 2 years. Of these, 6 patients (0.7%) died (3 cardiac deaths and 3 deaths from unknown causes). Acute myocardial infarction occurred in 6 patients (0.7%), including 3 (0.4%) with definite stent thrombosis (2 subacute and 1 very late stent thrombosis). Target lesion revascularization was performed in 46 patients (5.6%). In 13 patients, edge restenosis was responsible for repeat revascularization. Only 1 myocardial infarction with stent thrombosis was related to edge restenosis.

Discussion

The major findings of the present study evaluating the newer generation DESs were as follows. First, angiographic

Table 4
Angiographic and intravascular ultrasound (IVUS) data in 1,668 edges

	Proximal Edges (n = 681)			Distal Edges (n = 987)		
	With Edge Restenosis	Without Edge Restenosis	p Value	With Edge Restenosis	Without Edge Restenosis	p Value
Lesions (n)	26	655		21	966	
Post-stenting angiographic data						
Minimum lumen diameter (mm)	2.9 ± 0.5	3.3 ± 0.5	<0.001	2.0 ± 0.5	2.5 ± 0.5	<0.001
Diameter stenosis (%)	18.5 ± 11.0	9.7 ± 8.9	<0.001	27.5 ± 12.7	14.4 ± 9.9	<0.001
9-mo follow-up angiographic data						
Minimal lumen diameter (mm)	1.5 ± 0.4	3.1 ± 0.5	<0.001	1.1 ± 0.6	2.4 ± 0.5	<0.001
Diameter stenosis (%)	57.2 ± 9.8	14.2 ± 10.4	<0.001	61.7 ± 18.9	16.3 ± 9.9	<0.001
Post-stenting intravascular ultrasound data						
Minimum lumen area (mm^2)	6.3 ± 2.4	9.0 ± 3.4	<0.001	3.8 ± 2.2	5.7 ± 2.8	0.001
External elastic membrane area at minimum lumen area site (mm^2)	16.6 ± 4.5	17.4 ± 5.6	0.456	9.2 ± 4.6	9.8 ± 5.2	0.595
Maximum plaque burden (%)	61.7 ± 12.9	47.6 ± 11.1	<0.001	58.7 ± 12.7	38.1 ± 14.3	<0.001
Edge dissection	0 (0%)	5 (0.8%)	0.753	0 (0%)	14 (1.4%)	0.792
Plaque rupture	0 (0%)	7 (1.1%)	0.573	1 (4.8%)	14 (1.4%)	0.063

Data are presented as mean ± SD or n (%).

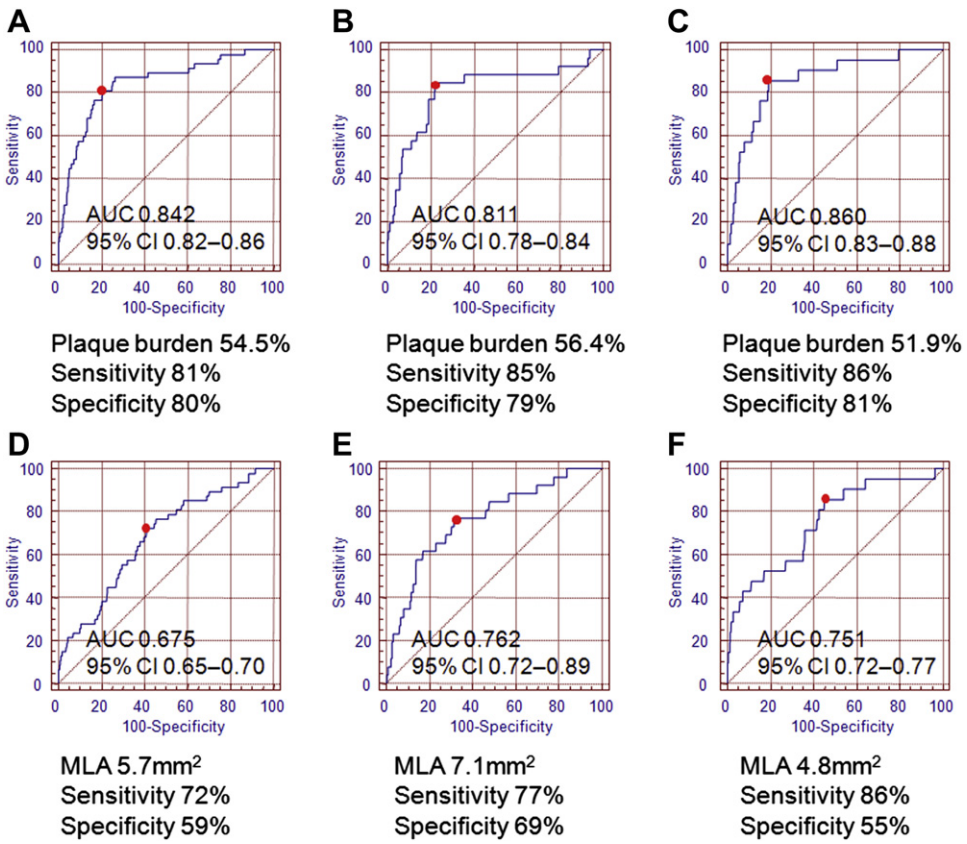

Figure 2. Receiver operating characteristic curves for IVUS criteria of 9-month angiographic edge restenosis. Cutoff values for reference segment maximum plaque burden for predicting edge restenosis in overall 1,668 lesions *(A)*, 681 proximal reference segments *(B)*, and 987 distal reference segments *(C)*. Cutoff values for MLA of reference segments in overall 1,668 lesions *(D)*, 681 proximal reference segments *(E)*, and 987 distal reference segments *(F)*. AUC = area under the curve; CI = confidence interval.

DS of the reference segments after stent implantation correlated poorly with the reference segment plaque burden as assessed by IVUS. Second, a maximum post-stenting reference segment plaque burden >55% predicted 9-month angiographic edge restenosis and was similar among the Endeavor zotarolimus-eluting stents, Resolute zotarolimus-eluting stents, and EESs.

Mintz et al[14] previously reported that the plaque burden in 884 angiographically normal reference segments was 51 ± 13%. Our present study has consistently demonstrated

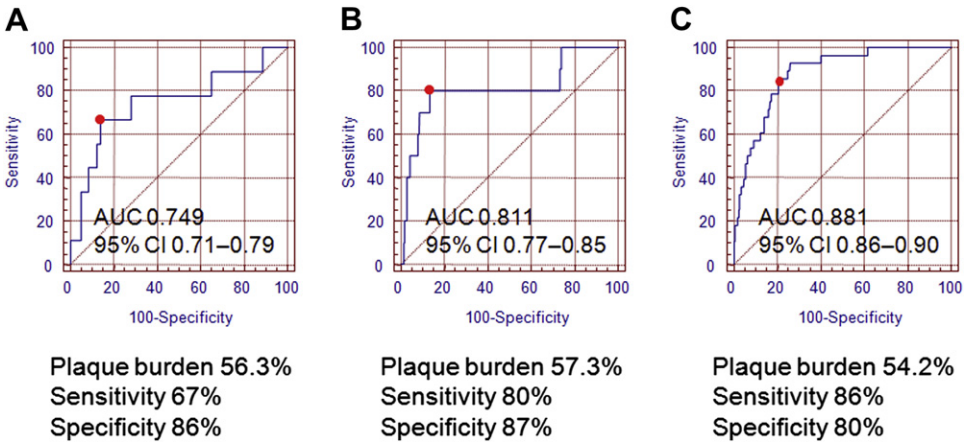

Figure 3. Receiver operating characteristic curves for IVUS criteria for 9-month angiographic edge restenosis. Cutoff values for reference segment maximum plaque burden for predicting edge restenosis in 433 reference segments of Endeavor zotarolimus-eluting stents *(A)*, 422 reference segments of Resolute zotarolimus-eluting stents *(B)*, and 813 reference segments of EESs *(C)*. Abbreviations as in Figure 2.

the poor correlation between post-stenting angiographic DS and IVUS-measured plaque burden at the reference segments. Overall, 37% of post-intervention angiographically normal proximal reference segments and 21% of post-intervention angiographically normal distal reference segments had a plaque burden >50%. Because IVUS revealed considerable disease even in the angiographically normal reference segments, guidance for the appropriate stent landing site could be 1 of the unique roles of IVUS.

It has been proposed that a greater reference segment plaque burden was the strongest predictor for edge stenosis after bare metal, sirolimus-eluting, and paclitaxel-eluting stent implantation.[4–8] Furthermore, Okabe et al[5] reported that lesions with stent thrombosis after sirolimus- and paclitaxel-eluting stent placement showed a smaller stent area and more residual disease at the stent edges. Our study has extended these observations to the newer generation DES. In our present study, the reference segment plaque burden was the consistent predictor of edge restenosis. Although full lesion coverage has been important for better clinical outcomes, the use of a greater length of stent has also been cited as a risk factor for stent thrombosis and restenosis.[2,15] Thus, acceptable residual plaque at the stent deployment site must be clarified to avoid unnecessarily long stents, particularly in diffuse coronary lesions. In the present analysis, stent length only showed a trend toward longer stents in lesions with in-stent restenosis.

With regard to specific IVUS criteria for the prediction of edge restenosis, the various published studies have been remarkably consistent. An integrated analysis of the IVUS substudies of the many TAXUS stent trials suggested a cutoff for a plaque burden of >47% for both bare metal and paclitaxel-eluting stents that predicted edge restenosis.[7] Similarly, an edge plaque burden of 52% was reported in 1 study of sirolimus-eluting stents.[6] In the present study of the newer generation DESs, a reference segment maximum plaque burden >55% predicted edge restenosis, with a sensitivity of 81%, specificity of 80%, and high negative predictive value, such that 99% of the reference segments with a plaque burden of ≤55% were free of edge restenosis at the follow-up examination. For the Endeavor zotarolimus-eluting stents, Resolute zotarolimus-eluting stents, and EESs, a residual plaque burden of approximately 55% can be used to determine the optimal stent landing zone.

The cutoff criteria for residual plaque burden were similar between the proximal and distal reference segments (56.4% and 51.9%, respectively). However, the cutoff criteria of the reference segment MLA were quite different (<7.1 mm^2 for proximal vs <4.8 mm^2 for distal). However, the predictabilities of the MLA criteria were lower than for the residual plaque burden criteria. Thus, at both proximal and distal edges, an identical threshold of edge plaque burden—approximately 55%—could be a feasible and practical IVUS criterion for the various types of newer generation DESs. In the present study, no significant relation was found between edge dissection and subsequent edge restenosis or clinical events; however, only 19 lesions had edge dissection, and none had flow limitation. These findings were consistent with those from previous studies.[16,17]

For the prediction of in-stent restenosis after newer generation DES implantation, a recent study reported that the cutoff for the post-stenting minimal stent area was 5.5 mm^2 for sirolimus-eluting stents, 5.3 mm^2 for zotarolimus-eluting stent, and 5.4 mm^2 for EESs.[18] Thus, combining these minimum stent area cutoffs with a plaque burden of ≤55% in both proximal and distal reference segments could optimize the efficacy of newer generation DES.

The present study was a retrospective, single-center study that included patients who had post-stenting IVUS scans and 9-month follow-up angiograms available. Therefore, the possibility of selection bias was not excluded. Furthermore, the relatively low rates of edge restenosis and cardiac events might have affected the results. Because preintervention IVUS analysis was not performed in all patients, the effect of preprocedural IVUS findings on edge restenosis could not be assessed. Finally, with the lack of follow-up IVUS scans, the precise mechanisms of restenosis were not assessed.

Disclosures

The authors have no conflicts of interest to disclose.

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