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A Randomized Comparison of Sirolimus- Versus Paclitaxel-Eluting Stent Implantation in Patients With Diabetes Mellitus

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Objectives The aim of this study was to compare the effectiveness of sirolimus-eluting stents (SES) and paclitaxel-eluting stents (PES) in patients with diabetes mellitus (DM).

Background Drug-eluting stent implantation significantly improved the angiographic and clinical outcomes compared with bare-metal stent implantation in diabetic patients. However, comparison of SES with PES in diabetic patients has not been sufficiently evaluated.

Methods This prospective, multicenter, randomized study compared SES (n = 200) and PES implantation (n = 200) for diabetic patients (n = 400). The primary end point was in-segment restenosis at 6 months according to intention-to-treat principle.

Results The 2 groups had similar baseline clinical and angiographic characteristics. Six-month in-stent (3.4% vs. 18.2%, $p < 0.001$) and in-segment restenosis (4.0% vs. 20.8%, $p < 0.001$) and 9-month target lesion revascularization (2.0% vs. 7.5%, $p = 0.017$) were significantly lower in the SES versus the PES group. The incidence of death (0% in SES vs. 0.5% in PES, $p = 0.999$) or myocardial infarction (0.5% in SES vs. 0.5% in PES, $p = 0.999$) at 9-month follow-up was not statistically different between the 2 groups. Major adverse cardiac events including death, myocardial infarction, and target lesion revascularization at 9 months (2.0% vs. 8.0%, $p = 0.010$) were lower in the SES versus the PES group.

Conclusions Sirolimus-eluting stent implantation is superior in reducing angiographic restenosis and improving 9-month clinical outcomes in patients with DM and coronary artery disease compared with PES implantation. (J Am Coll Cardiol 2008;52:727–33) © 2008 by the American College of Cardiology Foundation

Diabetic patients often present unfavorable coronary anatomy with small and diffusely diseased vessels (1) and exhibit exaggerated neointimal hyperplasia after bare-metal stent (BMS) implantation compared with nondiabetic subjects (2). Although drug-eluting stent (DES) implantation significantly reduced the neointimal hyperplasia and angiographic restenosis compared with BMS in diabetic patients (3), presence of diabetes mellitus (DM) has been still associated with an increased risk of restenosis and unfavorable clinical outcomes in the era of DES (4,5). Recently, the relative efficacies of sirolimus-eluting stents (SES) and paclitaxel-eluting stents (PES) in patients with DM have been evaluated in randomized and registry studies (6–10). Although some studies found SES to have greater efficacy than PES in diabetic patients (9,10), controversy remains (6–8). Therefore, to compare the effectiveness of 2 DES

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Abbreviations and Acronyms

AMI = acute myocardial infarction
BMS = bare-metal stent(s)
CI = confidence interval
DES = drug-eluting stent(s)
DM = diabetes mellitus
IQR = interquartile range
MACE = major adverse cardiac events
MI = myocardial infarction
MLD = minimal lumen diameter
PES = paclitaxel-eluting stent(s)
QCA = quantitative coronary angiography
RR = relative risk
SES = sirolimus-eluting stent(s)
TLR = target lesion revascularization
TVR = target vessel revascularization

(SES and PES) in patients with DM, we performed a randomized, multicenter, prospective study comparing SES and PES in diabetic patients (DES-DIABETES [Drug-Eluting Stent in patients with DIABETES mellitus] trial).

Methods

Patient selection. This prospective randomized study included 400 patients ≥18 years of age with angina pectoris and/or a positive stress test and a native coronary lesion. The study involved 5 cardiac centers in Korea between May 2005 and March 2006. Patients were considered eligible if they had DM, presented with angina pectoris, or had a positive stress test and had clinically significant angiographic stenosis in a native coronary vessel with a diameter stenosis ≥50% and visual reference diameter ≥2.5 mm. Patients were excluded if they had a contraindication to aspirin, clopidogrel, or cilostazol; left main disease (diameter stenosis ≥50% by visual estimate); graft vessel disease; left ventricular ejection fraction <30%; recent history of hematologic disease or leukocyte count <3,000/mm^3 and/or platelet count <100,000/mm^3; hepatic dysfunction with asparatate aminotransferase (AST) or alanine aminotransferase (ALT) ≥3 times the upper normal reference limit; history of renal dysfunction or serum creatinine level ≥2.0 mg/dl; serious noncardiac comorbid disease with a life expectancy <1 year; planned bifurcation stenting in the side branch; primary angioplasty for acute myocardial infarction (AMI) within 24 h; or inability to follow the protocol. In patients with multiple lesions fulfilling the inclusion and exclusion criteria, the first stented lesion was considered as target lesion. The institutional review board at each participating center approved the protocol. All patients provided written informed consent.

Randomization and procedures. Once the guidewire had crossed the target lesion, patients were randomly assigned in a 1:1 ratio to SES or PES implantation. After DES randomization, patients were randomly allocated in a 1:1 ratio to the triple antiplatelet group (aspirin, clopidogrel, and cilostazol; triple group; n = 200) or the dual antiplatelet therapy group (aspirin and clopidogrel; standard group; n = 200) (antiplatelet arm) on the basis of a 2 × 2 factorial design with a computer-generated randomization sequence. Random assignments were stratified according to participation sites and blocked with block size of 4 or 6 and were distributed in sealed envelopes to each participating center. The block size was concealed. From at least 24 h before the procedure and thereafter, all patients received aspirin (200 mg daily) and clopidogrel (loading dose of 300 mg, followed by 75 mg daily for at least 6 months). Patients in the triple group received a loading dose of 200 mg cilostazol immediately after the procedure and 100 mg twice/day for 6 months.

Coronary stenting was performed with the standard technique. The decision of pre-dilation or direct stenting was made by the operator. The use of intravenous glycoprotein IIb/IIIa inhibitors was at the operator's discretion. A 12-lead electrocardiogram was obtained after the procedure and before discharge. Serum levels of creatine kinase-myocardial band isoenzyme were assessed 8, 12, and 24 h after the procedure and thereafter if considered necessary.

Study end point and definitions. The primary end point of this trial was in-segment restenosis on 6-month follow-up study (defined as in-segment stenosis of at least 50%). The secondary end points included 6-month angiographic outcomes such as in-segment late loss and the rate of in-stent restenosis at 6 months (defined as in-stent stenosis of at least 50%), stent thrombosis, target vessel revascularization (TVR), and major adverse cardiac events (MACE) including death, myocardial infarction (MI), and target lesion revascularization (TLR).

The diagnosis of DM was considered confirmed in all patients receiving active treatment with an oral hypoglycemic agent or insulin; for patients with a diagnosis of diabetes who were on a dietary therapy alone, enrollment in the trial required the documentation of an abnormal blood glucose level after an overnight fast. Angiographic success was defined as in-segment final diameter stenosis <30% by quantitative coronary angiography (QCA). A Q-wave MI was defined by the post-procedural presence of new Q waves of >0.04 s in 2 contiguous leads. Non–Q-wave MI was defined as a creatine kinase-myocardial band fraction >3 times the upper limit of normal. Target lesion revascularization was considered clinically driven if prompted by symptoms consistent with myocardial ischemia, if preceded by an abnormal stress test result consistent with myocardial ischemia, if there were other electrocardiographic changes consistent with myocardial ischemia, or if the lesion diameter stenosis was more than 70% at follow-up (11). Stent thrombosis was defined as any of the following after the procedure: angiographic documentation of stent occlusion with or without the presence of thrombus associated with an acute ischemic event, unexplained sudden death, or MI not clearly attributable to another coronary lesion (12,13).

Follow-up. Repeat coronary angiography was mandatory at 6 months after stenting or earlier if indicated by clinical symptoms or evidence of myocardial ischemia. Clinical follow-up visits were scheduled at 30, 90, 180, and 270 days. At every visit, physical examination, electrocardiogram, cardiac events, and angina recurrence were monitored. At each participating center, patient data were recorded prospectively on standard case report forms and gathered in the central data

management center (Asan Medical Center, Seoul, Korea). All adverse clinical events were adjudicated by an independent events committee blinded to the treatment groups.

QCA analysis. Coronary angiograms were obtained after intracoronary nitroglycerin administration. Procedure (baseline), post-procedure, and follow-up angiograms were submitted to the angiographic core analysis center (Asan Medical Center, Seoul, Korea), in which intraobserver and interobserver correlation coefficients were 0.92 and 0.93, respectively. Digital angiograms were analyzed with an automated edge detection system (CASS II, Pie Medical, Maastricht, the Netherlands). The core laboratory was blinded to the treatment assignment. Angiographic variables included absolute lesion length, stent length, reference vessel diameter, minimal lumen diameter (MLD), percent diameter stenosis, binary restenosis rate, acute gain, late loss, and the patterns of recurrent restenosis. The QCA measurements of target lesions were obtained for both the stented segment only (in-stent) and the region including the stented segment as well as the margins 5 mm proximal and distal to the stent (in-segment). In-segment late loss was calculated with maximal regional late loss method (14). Patterns of angiographic restenosis were quantitatively assessed with the Mehran classification (15).

Statistical analysis. On the basis of results from a previous study (10), we assumed an in-segment angiographic restenosis rate of 7% in the SES group and 19% in the PES group. With a 2-sided 5% significance level, we estimated that 163 patients/group were needed to detect this difference with a statistical power of 90%. Expecting that approximately 20% of the patients would not return for angiographic follow-up, total sample size was estimated to 400 patients (200 patients/group). Analyses of 2 groups were performed according to the intention-to-treat principle. Continuous variables are presented as mean ± SD or median (interquartile range [IQR]) and compared with Student unpaired *t* or Mann-Whitney *U* tests. Categorical variables are presented as numbers or percentages and were compared with chi-square or Fisher exact tests. The relative risk (RR) and its 95% confidence interval (CI) were computed for outcome measures. The Breslow-Day test was performed to assess the homogeneity of the RR across participating centers and use of cilostazol (16). The adjusted RR and CI after controlling the center and use of cilostazol were computed by the Mantel-Haenszel method. For the primary outcome, we also calculated the absolute difference between SES and PES patients with and without cilostazol. A p value <0.05 was considered to indicate a significant difference. All statistical analyses were performed with SAS version 9.1 (SAS Institute, Cary, North Carolina).

Results

Baseline characteristics of the patients. Table 1 shows the baseline clinical characteristics of the study groups. There were no significant differences between the 2 groups in baseline clinical characteristics and risk factors.

Table 1 Baseline Clinical Characteristics

Variable	SES (n = 200)	PES (n = 200)	p Value
Age, yrs	61.1 ± 8.9	60.7 ± 8.8	0.622
Men	122 (61.0%)	110 (55.0%)	0.224
Hypertension	114 (57.0%)	124 (62.0%)	0.308
Treatment of diabetes mellitus			0.972
Dietary therapy alone	18 (9.0%)	19 (9.5%)	
Oral hypoglycemic agent	150 (75.0%)	148 (74.0%)	
Insulin	32 (16.0%)	33 (16.5%)	
Glycosylated hemoglobin, %	7.7 ± 1.8	7.8 ± 1.6	0.682
Total cholesterol ≥200 mg/dl	55 (27.5%)	63 (31.5%)	0.380
Current smoker	54 (27.0%)	57 (28.5%)	0.738
Previous PCI	25 (12.5%)	25 (12.5%)	0.999
Previous CABG	4 (2.0%)	3 (1.5%)	0.999
Clinical diagnosis			0.098
Stable angina	86 (43.0%)	82 (41.0%)	
Unstable angina	80 (40.0%)	67 (33.5%)	
Acute myocardial infarction	34 (17.0%)	51 (25.5%)	
Left ventricular ejection fraction, %	59 ± 10	58 ± 10	0.370
Multivessel disease	119 (59.5%)	137 (68.5%)	0.170

CABG = coronary artery bypass surgery; PCI = percutaneous coronary intervention; PES = paclitaxel-eluting stent; SES = sirolimus-eluting stent.

Procedural results and in-hospital outcomes. Table 2 shows angiographic characteristics and procedural results. The 2 groups have similar anatomical and procedural characteristics. All stents were successfully implanted, and the angiographic success rate was 99.5% in all groups. The 2 groups were treated with similar stented lengths and number of implanted stents/target lesion. Procedure-related non–Q-wave MI occurred similarly in both arms. Acute stent thrombosis developed in 1 patient (0.5%) treated with SES during hospital stay. In-hospital events, including Q-wave MI, emergency bypass surgery, or death, did not occur in either group.

Angiographic outcomes. Baseline and post-procedural QCA outcomes for study groups are shown in Table 3. The 2 groups had similar baseline and post-procedural QCA characteristics, except for higher maximal inflation pressure in the SES group versus the PES group.

Follow-up angiography was performed in 330 patients (82.5%)—176 (88%) SES and 154 (77%) PES patients. The median duration of angiographic follow-up was similar in the 2 groups (187 [IQR: 178 to 201] and 188 [IQR: 178 to 203] days for the SES and PES groups, respectively, p = 0.762). Results of QCA measurements at follow-up are shown in Table 3. The in-stent and -segment late loss were significantly lower in the SES versus in the PES group. Follow-up in-stent and -segment MLD also were significantly larger in SES versus in PES. In-segment restenosis, the pre-specified primary end point, was identified in 7 (4.0%) SES and 32 (20.8%) PES patients (RR: 0.19; 95%

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Table 2 Angiographic Characteristics and Procedural Results

Variable	SES (n = 200)	PES (n = 200)	p Value
Target vessel			0.707
Left anterior descending artery	122 (61.0%)	118 (59.0)	
Left circumflex artery	28 (14.0%)	25 (12.5%)	
Right coronary artery	50 (25.0%)	57 (28.5%)	
Procedure-related non–Q-wave MI	16 (8.0%)	18 (9.0%)	0.720
Maximal inflation pressure, atm	15.4 ± 3.6	14.6 ± 3.6	0.028
Use of intravascular ultrasound	67 (33.5%)	64 (32.0%)	0.749
Use of glycoprotein IIb/IIIa inhibitor	11 (5.5%)	7 (3.5%)	0.470
Pre-dilation before stenting	194 (97.0%)	190 (95.0%)	0.445
Post-stenting adjunctive balloon dilation	97 (48.5%)	87 (43.5%)	0.316
Largest balloon size for adjunctive dilation, mm	3.18 ± 0.43	3.25 ± 0.42	0.104
Multivessel stenting	64 (32.0%)	69 (34.5%)	0.596
Number of used stents at the target lesion	1.28 ± 0.49	1.28 ± 0.56	0.936

MI = myocardial infarction; other abbreviations as in Table 1.

CI: 0.09 to 0.42; $p < 0.001$). In-stent restenosis rate was also lower in the SES than in the PES group (3.4% vs. 18.2%; RR: 0.19; 95% CI: 0.08 to 0.44; $p < 0.001$). The test for assessing the homogeneity of the RR across the center and use of cilostazol were not significant for in-segment (Breslow-Day test, $p = 0.317$) and in-stent restenosis (Breslow-Day test, $p = 0.246$). Although there was interaction between types of DES and use of cilostazol in relative measure ($p < 0.001$ for in-segment restenosis), absolute measure was not statistically significant ($p = 0.861$). This phenomenon was due to 0% in-segment restenosis of the SES + cilostazol group. With regard to in-segment restenosis, the pre-specified primary end point, absolute difference was 8% in SES patients and 6.8% in PES patients, according to use of the cilostazol ($p = 0.861$) (Fig. 1).

Table 3 Quantitative Angiographic Measurements

Variable	SES (n = 200)	PES (n = 200)	p Value
Reference diameter, mm	2.80 ± 0.43	2.80 ± 0.43	0.962
Lesion length, mm	25.8 ± 12.9	27.2 ± 14.2	0.338
Stented length, mm	32.5 ± 13.9	33.2 ± 15.2	0.665
Minimal lumen diameter, mm			
In-segment			
Before procedure	0.79 ± 0.50	0.73 ± 0.46	0.236
After procedure	2.23 ± 0.46	2.27 ± 0.47	0.392
At follow-up	2.24 ± 0.50	1.93 ± 0.60	<0.001
In-stent			
After procedure	2.55 ± 0.46	2.57 ± 0.41	0.559
At follow-up	2.44 ± 0.51	2.01 ± 0.67	<0.001
Diameter stenosis, %			
In-segment			
Before procedure	68.1 ± 14.7	69.4 ± 12.7	0.423
After procedure	20.2 ± 12.1	19.2 ± 10.3	0.379
At follow-up	21.3 ± 12.5	31.6 ± 18.2	<0.001
In-stent			
After procedure	10.5 ± 11.5	8.9 ± 11.6	0.230
At follow-up	14.1 ± 15.2	26.3 ± 22.0	<0.001
Acute gain, mm			
In-segment	1.43 ± 0.60	1.53 ± 0.59	0.119
In-stent	1.76 ± 0.60	1.84 ± 0.57	0.171
Late loss, mm			
In-segment	0.31 ± 0.40	0.67 ± 0.53	<0.001
In-stent	0.13 ± 0.43	0.53 ± 0.57	<0.001
Binary angiographic restenosis			
In-segment	7 (4.0%)	32 (20.8%)	<0.001
In-stent	6 (3.4%)	28 (18.2%)	<0.001

Abbreviations as in Table 1.

In patients with restenoses, a pattern of focal restenosis (type I) was more common in the SES than in the PES group (Table 4).

Clinical outcomes. A minimum 9-month clinical follow-up was performed in all patients. One cardiac death occurred in PES patients due to nontarget vessel MI.

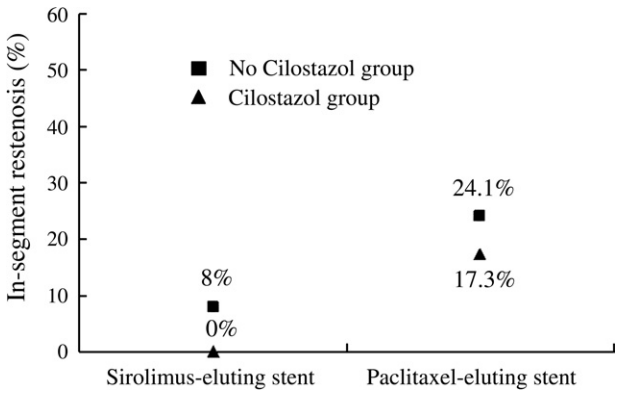

Figure 1 The In-Segment Restenosis Rate According to Drug-Eluting Stent and Use of Cilostazol

There is interaction between the type of drug-eluting stent and use of cilostazol for in-segment restenosis ($p < 0.001$). Although there was significant difference in relative measure of in-segment restenosis rate due to 0% in-segment restenosis rate of the sirolimus-eluting stent + cilostazol group, the absolute measure was not statistically different ($p = 0.861$).

Table 4 Angiographic Patterns of Restenosis

Variable	SES (n = 7)	PES (n = 32)	p Value
Focal	6 (85.7%)	18 (56.3%)	0.216
IA (articulation or gap)	0	0	
IB (margin)	1	5	
IC (focal body)	4	8	
ID (multifocal)	1	5	
Diffuse	1 (14.3%)	14 (43.8%)	0.216
II (intrastent)	1	10	
III (proliferative)	0	3	
IV (total occlusion)	0	1	
Length of restenosis	7.56 ± 5.21	15.5 ± 10.9	0.091

Classified with the Mehran criteria (15).
Abbreviations as in Table 1.

Myocardial infarction occurred in 1 patient/group. During 9 months, only 1 stent thrombosis occurred in the SES patients, which was angiographically documented at 6 h after index procedure. The patient was successfully treated with repeat intervention (Table 5).

The rates of TLR (2.0% vs. 7.5%; RR: 0.27; 95% CI: 0.09 to 0.79; p = 0.017) and TVR (3.5% vs. 8.0%, p = 0.053) were lower in the SES than in the PES group. Clinically driven TLR (1.5% vs. 6.0%, p = 0.032) and TVR (2.0% vs. 7.5%, p = 0.017) rates were also lower in the SES than in the PES group. At 9 months, MACE was lower in the SES than in the PES group (2.0% vs. 8.0%; RR: 0.25; 95% CI: 0.09 to 0.73; p = 0.010). The composite of death, MI, or TVR was also significantly lower in the SES than in the PES patients (3.5% vs. 8.5%, p = 0.035).

Discussion

The major finding of this study is that SES implantation is associated with reduction of late loss and 6-month angiographic restenosis, relative to PES implantation, which is translated to reduction of subsequent TLR and MACE with no difference of death or MI in patients with DM.

Drug-eluting stents significantly reduced angiographic restenosis and cardiac events compared with BMS in patients with DM (3). The presence of DM is associated with higher neointimal hyperplasia, restenosis, and unfavorable clinical outcomes in the era of DES (17,18). Recently, several randomized trials and registries showed inconsistent results regarding the superiority of SES over PES in diabetic patients (6–10). The current study shows that SES are more effective than PES in reducing angiographic restenosis and subsequent clinical outcomes.

In the current study, the in-stent (0.13 ± 0.43 mm vs. 0.53 ± 0.57 mm, p < 0.001) and in-segment late loss (0.31 ± 0.40 mm vs. 0.67 ± 0.53 mm, p < 0.001) were significantly lower in the SES than in the PES group. Although in previous studies, the late loss of the SES group (in-stent: 0.09 ± 0.4 mm to 0.26 ± 0.40 mm; in-segment: 0.41 ± 0.6 mm to 0.43 ± 0.45 mm) and the PES group (in-stent: 0.46 ± 0.64 mm to 0.50 ± 0.6 mm; in-segment: 0.67 ± 0.62 mm to 0.68 ± 0.6 mm) had somewhat different results than our study due to study design, enrolled patient/lesion characteristics, and stenting procedures (3,9,19,20), the 2 randomized studies comparing SES and PES in diabetic patients showed that the SES group had significantly reduced late loss compared with the PES group (9,20), which was a finding consistent with that of our study.

We also found that SES reduced in-segment restenosis (4.0% vs. 20.8%, p < 0.001), the pre-specified primary end point, by 81% (relative reduction) compared with PES. Previous registry data comparing SES and PES also showed a restenosis rate similar to those of our study (5.3% in SES and 23.1% in PES, p < 0.01) (10). However, this relative reduction is greater than those reported in previous randomized studies of various lesion subsets that showed a 7% to 77.2% relative reduction (9,21–23). The current study involved diabetic patients with a relatively long diseased segment, which might have contributed to the more pronounced differences between the 2 stents. According to previous randomized trials comparing SES versus PES, SES showed a more profound benefit over PES in more complex patients/lesions (9,22,23)—such as diabetic patients, in-stent restenosis, and long lesions—than in trials involving relatively simple lesions (11,24). The restenosis rate of ISAR-DIABETES (The Intracoronary Stenting and Angiographic Results: Do Diabetic Patients Derive Similar Benefit from Paclitaxel-Eluting and Sirolimus-Eluting Stents) (9), a randomized study comparing SES and PES in diabetic patients, was 6.9% in SES and 16.5% in PES patients. Our study had longer lesion length (25.8 mm of SES and 27.2 mm of PES) than those of ISAR-DIABETES (12.4 mm of SES and 13.4 mm of PES),

Table 5 Clinical Outcomes at 9 Months

Variable	SES (n = 200)	PES (n = 200)	p Value
Death	0	1 (0.5%)	0.999
Cardiac	0	1 (0.5%)	
Noncardiac	0	0	
MI	1 (0.5%)	1 (0.5%)	0.999
Q-wave	0	1 (0.5%)	
Non–Q-wave	1 (0.5%)	0	
TLR	4 (2.0%)	15 (7.5%)	0.017
Drug-eluting stent	1 (0.5%)	10 (5%)	
Cutting	1 (1%)	3 (1.5%)	
Bypass surgery	2 (1%)	2 (1%)	
Stent thrombosis	1 (0.5%)	0	0.999
Acute	1 (0.5%)	0	
Subacute	0	0	
Late	0	0	
TVR	7 (3.5%)	16 (8.0%)	0.053
Death/MI/TVR	7 (3.5%)	17 (8.5%)	0.035
MACE (death/MI/TLR)	4 (2.0%)	16 (8.0%)	0.010

MACE = major adverse cardiac events; MI = myocardial infarction; TLR = target lesion revascularization; TVR = target vessel revascularization; other abbreviations as in Table 1.

which would partly explain the profound superiority of SES over PES in our study.

Due to the reduced risk of angiographic restenosis, the subsequent TLR (2.0% vs. 7.5%; RR: 0.27; 95% CI: 0.09 to 0.79; p = 0.017) was significantly reduced in SES patients compared with PES patients. These results were also found in a recent meta-analysis comparing SES and PES in a variety of clinical settings and lesion complexity, in which SES significantly reduced the risk of reintervention (25). In our study, death or MI was similar in the 2 groups. Consequently, MACE including death, MI, and TLR was significantly lower in SES than in PES patients, mainly driven by TLR (2.0% vs. 8.0%, p = 0.010).

Diabetes mellitus has been reported to be associated with antiplatelet resistances (26). This is explained by aggressive atherosclerosis, abnormal endothelial function, impaired fibrinolysis, and platelet hyperactivity after arterial injury. Importantly, increased platelet activity is considered critically involved in the increased thrombogenic potential among diabetic patients. These findings might be associated with an increased risk of stent thrombosis after coronary stenting, which has been reported in previous registry data of DES (27). Furthermore, a recent meta-analysis showed that the PES had an increased risk of stent thrombosis compared with SES during a mean follow-up period ranging from 9 to 37 months (25). However, in our study, stent thrombosis occurred in only 1 patient receiving SES during 9 months. Thus, to evaluate the impact of DM on stent thrombosis after DES implantation, a larger population study would be required.

Study limitations. The present study has several limitations. First, our use of routine 6-month angiography might have resulted in an underestimation of the rates of restenosis and TLR compared with a study with a longer angiographic follow-up period. Second, there might be possible bias associated with clinical decisions related to TLR by operators, but this limitation has mostly been compensated for by ischemic-driven TLR. Third, the rate of angiographic follow-up was nonuniform in both groups.

Conclusions

The present study showed that SES implantation resulted in a significantly reduced risk of 6-month angiographic restenosis and 9-month TLR or MACE without a significant difference in MI or death compared with PES implantation.

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