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Comparison of the Effectiveness of Sirolimus- and Paclitaxel-Eluting Stents for Small Coronary Artery Lesions

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Background: The sirolimus-eluting stent (SES) and the paclitaxel-eluting stent (PES) reduce restenosis in small coronary artery lesions. However, it is not clear which of these stents is superior in terms of clinical outcomes. Methods: The authors retrospectively examined 197 patients with 245 de novo small coronary artery lesions (≤2.75 mm) that were treated with either the SES (156 lesions) or the PES (89 lesions). Six-month angiographic restenosis rates and the 9-month target lesion revascularization (TLR) rates were compared between the two groups. Results: In terms of baseline clinical and angiographic parameters, the two groups well matched together. Six-month angiographic follow-up was performed on 170 patients (86.3%), comprising 135 SES lesions (86.5%) and 76 PES lesions (85.4%). At 6-month angiographic follow-up, the late lumen loss was less in the SES group than in the PES group (0.29 ± 0.42 vs. 0.69 ± 0.63 mm, $P < 0.01$). Therefore, the SES group showed a lower rate of angiographic restenosis than the PES group (6.7% vs. 27.7%, $P < 0.01$). At 9 months there were no deaths or myocardial infarctions in either group. The 9-month TLR rate was lower in the SES group than in the PES group (3.3% vs. 14.4%, $P < 0.01$). The Kaplan-Meier estimate of freedom from TLR at 9 months was 96.7% for the SES patients and 86.5% for the PES patients ($P < 0.01$). Conclusions: The SES treatment may be superior to the PES treatment in terms of long-term clinical and angiographic outcomes in patients with small coronary artery lesions.

Key words: stent; restenosis; coronary artery disease

INTRODUCTION

The use of bare metal stents (BMS) for small coronary artery lesions has not been promising because of the high restenosis rate [1,2]. Recent data show that drug-eluting stents (DES) are superior to the BMS in terms of restenosis rates in native coronary artery lesions [3,4]. Published clinical studies using the most promising DES, namely the sirolimus-eluting stents (SES) and the paclitaxel-eluting stents (PES), have shown that the benefits of DES may be extended to the small coronary artery lesions [5–7]. However, little is known regarding which DES is better at inhibiting restenosis in small coronary artery lesions.

The present study compared the efficacy of the SES (Cypher: Cordis, Johnson & Johnson, Miami Lakes, FL) with the PES (Taxus: Boston Scientific, Natick, MA) when used for de novo small coronary artery lesions.

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METHODS

Study Design and Population

A retrospective analysis was undertaken for all patients undergoing percutaneous coronary intervention (PCI) for small coronary artery lesions using the SES or PES between June 2003 and July 2004. Eligible patients had to be aged more than 18 years and should have been diagnosed of stable or unstable angina pectoris. Patients with multivessel disease were also eligible to be enrolled in the study. Angiographic inclusion criteria were specified de novo coronary lesions with a diameter stenosis $\geq$70% and a reference diameter $\leq$2.75 mm by visual assessment. Exclusion criteria were acute myocardial infarction within the previous 48 hr, left ventricular ejection fraction $\leq$40%, more than 50% stenosis of the left main coronary artery lesion, bifurcation lesion, chronic total occlusion, previous history of PCI or bypass surgery, in-stent restenotic lesion, or the patients who were not treated with aspirin or clopidogrel because of allergic reaction.

Stenting Procedure

Patients were treated according to current standard interventional techniques. All patients received aspirin (200 mg/day) and clopidogrel (a loading dose of 300 mg 24 hr before the procedure) both before and after the procedure. A base-line electrocardiogram was obtained, and creatine kinase and cardiac enzyme levels were measured. During the procedure, intravenous boluses of heparin were administered to maintain an activated clotting time longer than of 250 sec and were discontinued immediately after the procedure. The use of intravenous glycoprotein IIb/IIIa inhibitors was at the operator's discretion. The choice of either predilation or direct stenting was made by the operator. Angiographic success was defined as residual stenosis $\leq$30% by visual analysis in the presence of a thrombolysis in myocardial infarction flow grade 3 [8]. A postprocedural electrocardiogram was obtained and isoenzyme levels were measured every 8 hr for 24 hr. Patients were discharged with a regimen of aspirin (200 mg daily indefinitely) and clopidogrel (75 mg daily for more than six months).

Study Endpoints

The primary endpoint was in-segment angiographic restenosis at 6 months after the procedure. The secondary end points were major adverse cardiac events (MACE) including death from any cause, myocardial infarction (Q-wave or non-Q-wave), and target lesion revascularization (TLR) at 9 months after the procedure. The TLR was defined as repeat revascularization (either by PCI or CABG) of the initial target lesion including in-stent and in-segments 5 mm from both proximal and distal stent edges. When the patients had the medically uncontrolled angina and the positive stress test (thallium scan or treadmill test), the TLR was considered. The Q-wave myocardial infarction was defined as the postprocedural presence of new pathological Q-waves greater than 0.04 sec in at least two contiguous leads with an elevated creatine kinase MB fraction level [7]. Non-Q-wave myocardial infarction was defined as a creatine kinase MB fraction greater than three times the normal upper limit in the absence of pathological Q waves.

Quantitative Angiographic Analysis

Coronary angiograms were obtained prior to the procedure (baseline), following the procedure, and at the 6-month follow-up. All of the coronary angiograms were performed in greater than two orthogonal projections after an injection of intracoronary nitroglycerin (200 μg). The follow-up angiograms were also done in the identical projections to the initial procedure. Coronary angiographs were analyzed by two independent angiographers who were not involved in the stenting procedures. Quantitative coronary angiographic measurements of the target lesions were obtained using a guiding catheter for magnification-calibration and an on-line system (ANCOR V 2.0, Siemens, Germany), with the minimal lumen diameter (MLD) of a lumen segment and a reference segment diameter were measured before and after stenting and at the 6-month follow-up. The analysis of segments included vessel regions 5 mm adjacent to the proximal and distal stent edges. Late lumen loss was defined as the change in the MLD between postprocedure and follow-up measurements. Angiographic restenosis was defined as a stenosis diameter >50% at 6 months. The patterns of angiographic in-stent restenosis were classified by Mehran et al. [9].

Statistical Analysis

Statistical analysis was performed using commercially available software (SPSS ver.11 for Windows, SPSS Inc., Chicago, Illinois). Data are presented as mean $\pm$ SD for continuous variables and as frequencies for categorical variables. Comparisons were performed using Pearson's χ^2 and unpaired t tests. TLR-free survival distributions in the two groups were estimated using the Kaplan-Meier method and were compared between groups using the log-rank test. A P-value < 0.05 was considered to indicate a significant difference.

TABLE I. Baseline Clinical Characteristics of Patients

	SES	PES	*P*-value
Patients	121	76	
Age (years)	61.0 ± 8.6	59.2 ± 10.6	0.22
Male, *n* (%)	76 (62.8)	54 (71.0)	0.28
LVEF (%)	59.1 ± 8.5	58.8 ± 7.6	0.79
Hypertension, *n* (%)	62 (51.2)	34 (44.7)	0.38
Diabetes mellitus, *n* (%)	39 (32.2)	21 (24.5)	0.53
Current smoker, *n* (%)	35 (28.9)	28 (36.8)	0.21
Hypercholesterolemia, *n* (%) (total cholesterol ≥200 mg/dL)	39 (32.2)	30 (39.5)	0.36
Clinical diagnosis			0.20
Stable angina, *n* (%)	60 (49.6)	43 (56.6)	
Unstable angina, *n* (%)	48 (39.7)	27 (35.5)	
AMI, *n* (%)	13 (10.7)	6 (7.8)	
Diseased vessels			0.51
1 vessel, *n* (%)	29 (24.0)	13 (17.2)	
2 vessel, *n* (%)	50 (41.3)	35 (46.0)	
3 vessel, *n* (%)	42 (34.7)	28 (36.8)	

SES, sirolimus-eluting stent; PES, paclitaxel-eluting stent; LVEF, left ventricular ejection fraction; AMI, acute myocardial infarction. Values represent mean ± SD or number (%).

TABLE II. Angiographic and Procedural Characteristics of Lesions

	SES	PES	*P*-value
Lesions	156	89	
Lesion location			0.21
Left anterior descending artery, *n* (%)	82 (52.6)	53 (59.6)	
Left circumflex artery, *n* (%)	50 (32.0)	20 (22.4)	
Right coronary artery, *n* (%)	24 (15.3)	16 (18.0)	
Type B2 and C lesion, *n* (%)	110 (70.5)	66 (74.2)	0.56
Before procedure			
Lesion length (mm)	25.2 ± 14.7	27.1 ± 12.7	0.34
Mean reference diameter (mm)	2.47 ± 0.21	2.44 ± 0.25	0.19
Minimal lumen diameter (mm)	0.86 ± 0.33	0.81 ± 0.42	0.31
Diameter stenosis (%)	65.4 ± 13.0	67.5 ± 16.0	0.22
After procedure			
Mean reference diameter (mm)	2.47 ± 0.21	2.44 ± 0.25	0.27
Minimal lumen diameter (mm)	2.52 ± 0.33	2.42 ± 0.35	0.45
Diameter stenosis (%)	3.7 ± 7.1	5.8 ± 8.3	0.06
Acute gain (mm)	1.67 ± 0.44	1.62 ± 0.50	0.48
Procedural characteristics			
Total stent length (mm)	31.4 ± 17.5	32.8 ± 14.4	0.69
Maximal inflation pressure (atm)	15.9 ± 3.0	15.1 ± 2.9	0.09
Maximal stent diameter (mm)	2.91 ± 0.2	3.00 ± 0.22	0.23
Follow-up			
Lesions, *n* (%)	135 (86.5)	76 (85.4)	0.85
Mean reference diameter (mm)	2.46 ± 0.28	2.43 ± 0.39	0.54
Minimal lumen diameter (mm)	2.32 ± 0.56	1.77 ± 0.77	<0.01
Diameter stenosis (%)	5.38 ± 22.5	31.7 ± 34.9	<0.01
Late loss (mm)	0.29 ± 0.42	0.69 ± 0.62	<0.01
Restenosis, *n* (%)	9 (6.7)	21 (27.7)	<0.01

SES, sirolimus-eluting stent; PES, paclitaxel-eluting stent. Values represent mean ± SD or number (%).

RESULTS

During the study period, 1238 patients underwent stenting using DES. In these patients, 264 patients (21.3%) had small coronary artery lesions, and 67 of these patients did not satisfy the inclusion criteria. Thus, 197 patients were enrolled in the trial; 121 patients (61.4%) were treated with the SES and 76 patients (38.6%) were treated with the PES. There were no significant differences between the two groups in terms of baseline clinical characteristics (Table I). Similarly, there were no significant differences between the two groups in terms of angiographic and procedural characteristics (Table II). Adverse cardiac events during hospitalization are shown in Table III. There were no incidents of cardiac death or Q-wave myocardial infarction in either group. The two groups do not differ significantly in terms of the rate of post-procedural non-Q-wave myocardial infarctions (12.4% vs. 13.2%, $P = 0.54$). Angiographic follow-up at 6 months was performed in 170 patients, comprising 135 SES treated-lesions and 76 PES-treated lesions (86.5% vs. 85.4%, $P = 0.84$). The SES group was found to have a larger MLD at 6 months (2.32 ± 0.56 vs. 1.77 ± 0.77 mm, $P < 0.01$), corresponding with a smaller late loss in the SES compared to the PES group (0.29 ± 0.42 vs. 0.69 ± 0.62 mm, $P < 0.01$). The 6-month angiographic restenosis rate was lower in the SES group than the PES group (6.7% vs. 27.7%, $P < 0.01$). There was no difference between the groups in terms of the patterns of restenosis as Table IV. Multivariate analysis was performed to identify independent predictors of in-segment restenosis (Table V). This analysis identified the type of DES, postprocedure in-stent MLD, and a diffuse long lesion as independent predictors of in-segment restenosis in small coronary artery disease.

All of the patients had 9-month clinical follow-up by means of medical record and phone interview. At the time of this follow-up, there were no deaths or myocardial infarctions in either group (Table III). The SES group showed a lower incidence of the 9-month TLR (3.3% vs. 14.4%, $P < 0.01$) and MACE (15.7% vs. 27.6%, $P < 0.01$) compared to the PES group. Consequently, the Kaplan-Meier estimate of freedom from TLR at 9 months was 96.7% for the SES group and 86.5% for the PES group ($P < 0.01$; Figure 1).

DISCUSSION

The long-term results of percutaneous intervention for small coronary artery lesions have not been promising. Previous studies such as by Park et al., SISA, and COAST have shown that the BMS implantation

TABLE III. Clinical Outcomes During Hospitalization and at 9-Month Follow-Up

	SES	PES	*P*-value
Patients	121	76	
In-hospital outcomes			
Death	0	0	1.0
Myocardial infarction, *n* (%)	15 (12.4)	10 (13.2)	0.54
Q myocardial infarction	0	0	1.0
Non-Q myocardial infarction, *n* (%)	15 (12.4)	10 (13.2)	0.54
Target lesion revascularization	0	0	1.0
MACE, *n* (%)	15 (12.4)	10 (13.2)	0.54
Nine-month outcomes			
Death	0	0	1.0
Myocardial infarction, *n* (%)	15 (12.4)	10 (13.2)	0.54
Q myocardial infarction	0	0	1.0
Non-Q myocardial infarction, *n* (%)	15 (12.4)	10 (13.2)	0.54
Target lesion revascularization, *n* (%)	4 (3.3)	11 (14.4)	<0.01
MACE, *n* (%)	19 (15.7)	21 (27.6)	<0.01

MACE, major adverse cardiac event.

TABLE IV. Follow-Up Angiographic Characteristics of Lesions and Patterns of In-Stent Restenosis Using the Mehran Criteria

	SES	PES	*P*-value
Lesions, *n* (%)	135 (86.5)	76 (85.4)	0.85
Mean reference diameter (mm)	2.46 ± 0.28	2.43 ± 0.39	0.54
Minimal lumen diameter (mm)	2.32 ± 0.56	1.77 ± 0.77	<0.01
Diameter stenosis (%)	5.38 ± 22.5	31.7 ± 34.9	<0.01
Late loss (mm)	0.29 ± 0.42	0.69 ± 0.62	<0.01
Restenosis, *n* (%)	9 (6.7)	21 (27.7)	<0.01
Patterns of in-stent restenosis			0.49
Focal, *n* (%)	7 (77.8)	10 (47.6)	
Diffuse, *n* (%)	2 (22.2)	11 (52.4)	
Intra-stent, *n* (%)	2 (22.2)	7 (33.4)	
Proliferative, *n* (%)	0	2 (9.5)	
Total occlusion, *n* (%)	0	2 (9.5)	

SES, sirolimus-eluting stent; PES, paclitaxel-eluting stent. Values represent mean ± SD or number (%).

TABLE V. Independent Predictors of In-Segment Restenosis

	Hazard ratio	95% CI	*P*-value
SES implantation	0.26	0.08–0.85	<0.05
MLD after procedure	0.02	0.01–0.24	<0.01
Lesion length per 10 mm	1.49	1.09–2.02	<0.05

SES, sirolimus-eluting stent; MLD, minimal lumen diameter.

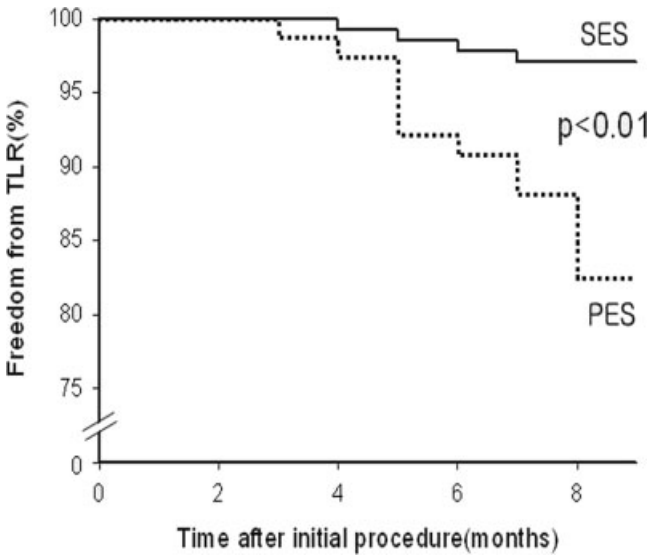

Fig. 1. Kaplan–Meier survival curves displaying cumulative rates of freedom from TLR after 9 months follow-up. SES, sirolimus-eluting stent; PES, paclitaxel-eluting stent; and TLR, target lesion revascularization.

was not clearly superior to the balloon PTCA for small coronary artery lesions [10–12], with the major problem being the relatively high restenosis rates associated with BMS implantation. Indeed the reported incidence of restenosis after BMS implantation for small coronary lesions is 35–45% [5,6]. The inverse relationship between vessel size and angiographic restenosis rate in BMS implantation [1] may also be applicable to the DES. In SIRIUS study, there was an inverse relationship between vessel size and angiographic restenosis rate in the SES-treated vessels, with large (> 3.3 mm) and small (< 2.3 mm) vessels showing restenosis rates of 1.9% and 18.6%, respectively [3,13]. In the TAXUS-IV study, the 9-month restenosis rates for the small (≤ 2.5 mm) and large (> 3.5 mm) vessels were 8.8% and 5.5%, respectively [7]. Despite the inverse association between vessel size and restenosis rate, the benefits of the SES and the PES may extend to small vessel disease due to their ability to inhibit neointimal hyperplasia. In the present study, both the SES and the PES were associated with low angiographic restenosis and TLR rates without serious adverse events. These results are consistent with findings of previous large randomized clinical studies. Substudies of the SIRIUS trial, the E-SIRIUS and C-SIRIUS studies, showed that the SES was superior to the BMS in terms of restenosis rate in native coronary arteries that had reference diameters between 2.5–3.0 mm [5,6]. In the E-SIRIUS trial, there was an 86% reduction in the in-segment restenosis rate when using the SES compared to the BMS (5.9% vs. 42.3%, $P < 0.001$). Similarly, the TAXUS IV trial showed that the PES was superior to the BMS in a subset analysis of restenosis rates in small coronary artery (< 2.5 mm) disease (8.8% vs. 40.8%, $P < 0.001$) [7].

Currently, little is known about which DES is better at inhibiting restenosis in small coronary artery disease. A recent study of native coronary lesions showed SES were superior to PES in terms of 9-month angiographic restenosis rates (6.6% vs. 11.7%, $P = 0.02$) [14]. Another

recent study revealed that the SES decreased the extent of late loss compared with the PES in diabetic patients (0.43 ± 0.45 vs. 0.67 ± 0.62 mm, $P = 0.002$) [15]. In the present study, the SES appeared to be more effective at reducing 6-month angiographic restenosis (57% reduction) and MACE (62% reduction) compared to the PES. In the present study, it is unclear why the SES appears to be more effective than the PES for use in small coronary lesions. In small coronary lesions, the mechanical configuration of the stent strut as well as the biological efficacy of the DES may affect angiographic restenosis. Briguori et al. compared outcomes using stents with strut thicknesses of <0.1 mm and ≥ 0.1 mm in vessels smaller than 3.0 mm in diameter. They reported that the restenosis rate for thinner-strutted stents was 56% lower than for thicker strutted stents (28.5% vs. 36.6%, $P = 0.009$) [16]. In the present study, the strut thicknesses of the PES and the SES were very similar (0.132 mm vs. 0.140 mm, respectively), suggesting that the differing results obtained using the two stents were not due to mechanical differences. The biological efficacy of the DES over the BMS is well established. Using intravascular ultrasound (IVUS), Hoffmann et al. showed that the DES had greater antiproliferative efficacy than the BMS [17]. In that study, the IVUS assessment at 6 months after stent deployment showed that the mean intimal hyperplasia thickness was reduced by 49% and 90% when using the PES and SES, respectively, compared to using the BMS ($P = 0.048$ and $P < 0.001$). While the exact mechanism underlying the present findings of the SES superiority remains to be determined, the data may reflect pharmacological or drug delivery differences between the two stent types.

Late loss is defined as the difference between the follow-up MLD and the postprocedural MLD. Late loss provides a measure of neointimal hyperplasia and has been used as a surrogate end point in many interventional trials. In the DES era, late loss may be a strong end point for discriminating between different DES, for which binary restenosis rates are anticipated to be low [18]. In addition, late loss may give great effect on small coronary artery lesions because same amount of late loss could be a greater restenosis issue than large coronary artery lesions [13]. Studies to date report that the SES are associated with a greater reduction of late loss compared to the PES according to angiographic follow-up data [14,15,19]. Consistent with those findings, the present study found that the PES were associated with greater late loss than the SES (0.69 ± 0.62 vs. 0.29 ± 0.42 mm, $P < 0.001$), and consequently showed a higher restenosis rate.

In the present study, the restenosis rate for the PES was higher than that reported in the TAXUS IV trial (27.7% vs. 7.9%). The major determinants of restenosis after intracoronary stent implantation are the reference vessel diameter and lesion length. Compared to the TAXUS IV trial, the PES population in the present study had longer lesion lengths (27.1 ± 12.7 vs. 13.4 ± 6.3 mm) and smaller reference diameters (2.44 ± 0.25 vs. 2.75 ± 0.47 mm). These differences may explain the differences in the PES-associated restenosis rates between the two studies.

In this study, in spite of the long lesions, there was no subacute thrombosis because 73.5% (69.7% in PES vs. 75.6% in SES, $P = 0.37$) of lesions were evaluated with intravascular ultrasound in order to find and fix the stent malapposition and 34.2% of the patients were prescribed dual antiplatelet treatment (aspirin + clopidogrel) for 6 months and the 65.8% (63.1% in PES vs. 67.6% in SES, $P = 0.62$) of patients prescribed for more than 6 months. According to Saucedo et al. after successful stenting, the incidence of periprocedural non-Q-wave myocardial infarction (CK-MB elevation > 5 times normal) was about 8.5% and it is associated with an increased risk of MACE [20]. In this study, the incidence of non-Q-wave myocardial infarction (CK-MB elevation > 3 times normal) was 12.7%. If we made an application of CK-MB elevation > 5 times normal, the incidence of non-Q-wave myocardial infarction was 4.1%.

There is a possibility that the greater effectiveness of the SES shown in the present report relates to the retrospective study design, indicating further research using a prospective randomized approach is warranted. However, considering the well-matched clinical and procedural characteristics between the two groups, selection bias was unlikely to be a factor in the present results.

CONCLUSIONS

The present retrospective comparison study indicates that the SES might be associated with the PES that is superior in terms of angiographic and clinical outcomes in treatment of small coronary artery lesions.

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