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Risk Factors for Restenosis after Percutaneous Coronary Intervention with Sirolimus- and Paclitaxel-eluting Stents

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Abstract : To identify risk factors for restenosis after percutaneous coronary intervention with sirolimus (SES)- or paclitaxel (PES)-eluting stents. The clinical outcomes of 894 patients treated with either SES (n＝462) or PES (n＝432) between January 2005 and January 2010 were evaluated. Multivariate logistic regression analysis showed that long (＞20 mm) (odds ratio [OR], 1.87 ; 95% confidence interval [CI], 1.07～3.33 ; $P＝0.03$) or bent (angle＞45°) lesions (OR, 2.57 ; 95% CI, 1.47～4.49 ; $P＜0.01$) were independent risk factors for restenosis with SES, and that hemodialysis (OR, 7.61 ; 95% CI, 2.78～20.85 ; $P＜0.01$) and long (OR, 2.63 ; 95% CI, 1.18～5.84 ; $P＝0.02$) or bent lesions (OR, 3.47 ; 95% CI, 1.65～7.27 ; $P＜0.01$) were independent risk factors for target lesion revascularization (TLR) with SES. In contrast, no independent risk factors for restenosis and TLR were found for lesions treated with PES. The rate of TLR was significantly higher in patients on hemodialysis or in those with long lesions in the SES group (hemodialysis, 30.4% vs. 11.1%, $P＝0.02$; long lesions, 13.2% vs. 4.4%, $P＜0.01$; for SES vs. PES, respectively). Rates of restenosis and TLR were significantly higher in patients with bent lesions in the SES group (restenosis, 30.8% vs. 15.6%, $P＜0.01$; TLR, 20.0% vs. 5.8%, $P＜0.01$; for SES and PES, respectively). Most clinical studies have described better angiographic results for SES compared to PES. However, PES might result in better clinical outcomes than SES for patients on hemodialysis or for those with long or bent lesions.

Key words : restenosis, drug-eluting stent, sirolimus-eluting stent, paclitaxel-eluting stent

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Introduction

The introduction of drug eluting stents (DES) has remarkably improved the restenosis rate of percutaneous coronary intervention (PCI). However, although DES have revolutionized PCI by significantly reducing the occurrence of restenosis and revascularization compared with bare metal stents during short- and long-term follow-up, restenosis and revascularization continue to occur in some patients treated with DES.

Sirolimus-eluting stents (SES; CYPHER®; Cordis Corporation / Johnson and Johnson, Miami Lakes, FL) and paclitaxel-eluting stents (PES; TAXUS™; Boston Scientific Corp., Natick, MA) are the most studied DES to date. However, the bare metal stent platform, permanent polymer and the antiproliferative drugs significantly differ between SES and PES. Sirolimus is an immunosuppressive drug with antiinflammatory properties that arrests the cell-cycle at the G1 / S phase transition, whereas paclitaxel is a cytotoxic, antineoplastic drug that causes cell-cycle arrest at the G2 / M phase transition[1, 2]. Sirolimus-eluting stents are based on the rigid and closed-cell, BX velocity®, whereas PES are based on EXPRESS® or LIBERTE® that have a flexible, open-cell design. Because these devices differ in terms of stent design and polymer construction, the question arises as to whether they differ with respect to implementation in PCI.

Diabetes mellitus[3–6], dialysis[7, 8], long lesions and small vessels[9], as well as chronic and total occlusion (CTO)[10–15], have been described as risk factors for restenosis after PCI with bare metal stents. In contrast, risk factors for PCI using DES compared with bare metal stents have not been investigated in detail. A large multi-center study demonstrated similar DES efficacy profiles in patients with and without calcified coronary lesions. However, patients with severely calcified lesions were excluded from that trial[16, 17]. Therefore, we compared the clinical outcomes of PCI with SES and PES to determine independent risk factors for restenosis in such patients.

Methods

Study population

We analyzed data from 894 consecutive patients with coronary artery disease who were treated with SES (January 2005 to May 2007) or PES (May 2007 to January 2010). The type, length and number of coronary lesions did not influence the choice of stent. The data were extracted from a retrospective registry of patients at our center where over 400 procedures per year are performed by five expert operators. Baseline clinical, angiographic and procedural characteristics as well as in-hospital outcomes were entered into a database by physicians. Clinical outcomes, most importantly major adverse cardiac events (MACE), were recorded at the clinic, or by formal telephone interviews at 1, 8 and 12 months and annually thereafter, and later entered into our database. Patients treated both with SES and PES were included in the study.

Angioplasty procedures

All angioplasties were performed using a 7- or 8-Fr guiding catheter and the femoral approach. Heparin was administered in boluses to achieve and maintain an activated clotting time of more than 250 s. Aspirin (at least 100 mg for an indefinite period) was administered immediately after the procedure and continued for as long as possible. Clopidogrel (loading dose of 300 mg, followed by 75 mg / day for at least one year) or ticlopidine (loading dose of 200 mg, followed by 300 mg / day for at least one year) was also started immediately after the procedure. Additional DES were used as necessary when dissection arose or lesions were not completely covered.

Stents were deployed with or without pre-dilation according to standard techniques and positioned to completely cover lesions. Dilation pressure was applied to the stent until the lesion was sufficiently dilated under transillumination. Intravascular ultrasound was used during all procedures to determine stent diameter pre- and post-dilation and after stenting to reconfirm the position of the stent.

The lengths of SES and PES ranged from 13～33 mm and 12～33 mm, respectively, and diameters ranged from 2.5～4 mm and 2.25～3.5 mm, respectively. Aspirin and clopidogrel or ticlopidine were started immediately after stent implantation, as described above.

Definitions

Anginal symptoms were defined according to the classification of the Canadian Cardiovascular Society. Major adverse cardiac events were defined as death, myocardial infarction, or target lesion revascularization. Target restenosis was defined as stenosis of ≥50% on follow-up coronary angiography. Target vessel revascularization was defined as clinically-driven percutaneous revascularization or bypass of the target lesion or any segment of the epicardial coronary artery including the target lesion. Target lesion revascularization was defined as any repeat revascularization procedure (percutaneous or surgical) at the original target lesion site. The primary endpoint was the occurrence of MACE during follow-up and independent predictors, which were compared between the groups. Calcified lesions were defined as identifiable radiopaque images on still images obtained before injecting contrast agent or an identifiable dark area on moving images. Lesions were defined as being long if they were ＞20 mm in length, or being bent if they had a ≥45° bend at the center. Small vessel lesions were defined as having a diameter of ＜2.75 mm. Procedural success was taken as thrombolysis in myocardial infarction flow 3 on final images and a ≤25% residual rate of stenosis.

Quantitative coronary angiography

We used the QCA-CMS cardiovascular analysis system (Medis Medical Imaging Systems, Raleigh, NC) for coronary angiography. Lesion length, minimum vascular diameter and control vascular diameter were measured from dilation-phase frames taken from the same

angle of minimal lesion contraction during pre-treatment, post-treatment and at remote-phase follow-up coronary angiography. Rates of stenosis, acquired inner diameter during the acute phase and the loss of inner diameter in the remote phase were calculated.

Statistical analysis

Quantitative data are presented as means ± SD, and categorical data as ratios (%). Data were statistically analyzed using the chi-square or Fisher's exact test (two-tailed) for categorical variables. Continuous variables were compared using the Student *t* test and *P*-values ≤0.05 were considered significant. Univariate and multivariate analyses, including 95% confidence intervals (CI), were calculated using logistic regression analysis. Factors with *P*-values <0.05 in the univariate analysis were entered into the multivariate model. All data were statistically analyzed using commercially available software (Stat View for Windows version 5.0).

Results

Base characteristics

The baseline characteristics of 462 and 432 patients who were treated with PCI using SES and PES, respectively, over a period of seven years are shown in Table 1. Age, male sex, risk factors (hypertension, dyslipidemia, hyperuricemia, current or previous smoking habit, hemodialysis, family history of cardiovascular disease, left ventricular ejection fraction) and clinical presentation were similar in both groups.

Angiographic characteristics

The angiographic characteristics of the patients are shown in Table 2. The rate of left main trunk and left anterior descending artery lesions was significantly higher in the SES (14.5%), than in the PES group (10.0% ; respectively, $P=0.04$), while the rate of left anterior descending artery was significantly higher in the PES group (40.5%), than in the SES group (31.2% ; respectively, $P=0.04$). The rate of bent lesions was significantly larger in the PES group (35.6%), than in the SES group (26.0% ; $P=0.002$), while the rate of small vessel lesions was significantly larger in the SES group (39.8%), than in the PES group (32.2% ; $P=0.02$). Pre-PCI, the reference diameter and % diameter stenosis were significantly greater in the SES group, than in the PES group (reference diameter, 2.97 ± 1.24 mmvs. 2.67 ± 0.71 mm, $P=0.03$; % diameter stenosis, 85.1% ± 13.8% vs. 80.3% ± 17.6%, $P=0.01$, for SES vs. PES, respectively). The rate of CTO lesions was also significantly higher in the SES group compared to the PES group (20.1% vs. 12.5%, respectively ; $P=0.03$).

Clinical outcomes

Data from the one-year clinical follow-up are shown in Table 3. The rate of restenosis, TLR and MACE (death, myocardial infarction, target vessel revascularization) were similar

Table 1. Baseline characteristics of the patients.

	SES	PES	p
Patients, n	462	432	
Age, years	66.8±10.2	67.6±10.4	0.22
Male, n (%)	368 (79.5)	355 (82.2)	0.38
Risk factors			
Hypertension, n (%)	369 (80.0)	322 (74.5)	0.57
Diabetes mellitus, n (%)	257 (55.6)	231 (59.1)	0.31
Dyslipidemia, n (%)	319 (69.0)	253 (65.0)	0.21
Hyperuricemia, n (%)	135 (29.2)	55 (23.9)	0.14
Current or previous smoker, n (%)	303 (65.6)	195 (65.9)	0.93
Hemodialysis, n (%)	56 (12.1)	45 (10.4)	0.42
Family history, n (%)	171 (37.0)	85 (35.7)	0.66
Left ventricular ejection fraction, %	52.5±14.2	51.7±12.9	0.55
Clinical presentation			
Stable angina, n (%)	198 (42.9)	176 (40.7)	0.52
Unstable angina, n (%)	78 (16.9)	53 (12.3)	0.11
Acute myocardial infarction, n (%)	86 (18.6)	83 (19.2)	0.82
Number of diseased vessels			0.26
1, n (%)	280 (60.6)	243 (56.3)	
2, n (%)	134 (29.0)	147 (34.0)	
3, n (%)	48 (10.4)	42 (9.7)	

SES, sirolimus-eluting stent ; PES, paclitaxel-eluting stent.

between the two groups. The rate of TLR tended to be greater in the SES group, but the difference was not statistically significant.

Results of the univariate and multivariate logistic regression analyses of restenosis and TLR are shown in Tables 4 and 5. Univariate analysis showed that the clinical and angiographic risk factors for restenosis in lesions treated with SES were hemodialysis (odds ratio [OR], 2.96 ; 95% CI, 1.58～5.55 ; $P<0.01$) and calcified (OR, 3.11 ; 95% CI, 1.84～5.27 ; $P<0.01$), long (OR, 2.49 ; 95% CI, 1.48～4.19 ; $P<0.01$) and bent lesions (OR, 3.68, 95% CI, 2.19～6.16 ; $P<0.01$). Multivariate logistic regression analysis showed that long (OR, 1.87 ; 95% CI, 1.07～3.33 ; $P=0.03$) and bent lesions (OR, 2.57 ; 95% CI, 1.47～4.49 ; $P<0.01$) were independent risk factors for restenosis after treatment with SES. On the other hand, we found no risk factors for coronary restenosis among lesions treated with PES. Univariate analysis showed that the clinical and angiographic risk factors for TLR after treatment with SES were hemodialysis (OR, 7.26 ; 95% CI, 3.58～14.74 ; $P<0.01$) and calcified (OR, 3.59 ; 95% CI, 1.85～6.97 ; $P<0.01$), long (OR, 3.19 ; 95% CI, 1.55～6.55 ; $P<0.01$) and bent lesions (OR, 5.09 ; 95% CI, 2.60～9.99 ; $P<0.01$). Multivariate logistic regression analysis showed that hemodialysis (OR, 7.61 ; 95% CI, 2.78～20.85 ; $P<0.01$) and long (OR, 2.63 ; 95% CI, 1.18～5.84 ; $P=0.02$) and bent lesions (OR, 3.47 ; 95% CI, 1.65～7.27 ; $P<0.01$) were independent risk factors for TLR after treatment with SES. No risk

Table 2. Angiographic characteristics, procedural data and quantitative coronary findings.

	SES	PES	p
Angiographic characteristics			
Target vessels			
Left main trunk, n (%)	67 (14.5)	43 (10.0)	0.039
Left anterior descending, n (%)	144 (31.2)	175 (40.5)	0.036
Left circumflex, n (%)	111 (24.0)	94 (21.8)	0.42
Right coronary, n (%)	140 (30.3)	147 (34.0)	0.23
Lesion type			
Long, n (%)	220 (47.6)	217 (50.2)	0.44
Bent, n (%)	120 (26.0)	154 (35.6)	0.0015
Calcified, n (%)	104 (22.5)	100 (23.1)	0.11
Small vessel, n (%)	184 (39.8)	139 (32.2)	0.017
CTO, n (%)	93 (20.1)	46 (12.5)	0.033
Type B2 / C lesion, n (%)	382 (82.7)	343 (79.4)	0.19
Procedural data			
Stent diameter, mm	2.98 ± 0.38	2.90 ± 0.35	0.75
Stent length, mm	23.2 ± 5.2	23.1 ± 6.1	0.94
Balloon diameter, mm	3.14 ± 0.57	2.98 ± 0.47	0.57
Balloon pressure, atm	16.0 ± 4.2	14.2 ± 3.7	0.73
QCA			
Pre-PCI			
MLD, mm	0.44 ± 0.45	0.51 ± 0.43	0.13
RD, mm	2.97 ± 1.24	2.67 ± 0.71	0.032
%DS	85.1 ± 13.8	80.3 ± 17.6	0.013
Post-PCI			
MLD, mm	2.65 ± 0.68	3.71 ± 1.48	0.21
RD, mm	3.11 ± 0.42	3.05 ± 0.45	0.35
%DS	14.7 ± 9.9	20.7 ± 79.4	0.24
Follow-up			
MLD, mm	2.29 ± 0.76	2.12 ± 0.73	0.12
RD, mm	3.10 ± 0.63	4.31 ± 18.9	0.26
%DS	25.8 ± 21.0	25.6 ± 22.6	0.93
Acute gain, mm	2.22 ± 0.65	2.08 ± 0.59	0.27
Late loss, mm	0.37 ± 0.83	0.47 ± 0.68	0.15

CTO, chronic total occlusion ; MLD, minimal lumen diameter ; QCA, quantitative coronary analysis ; RD, reference diameter ; %DS, % diameter stenosis.

factors for TLR were found after treatment with PES.

Hemodialysis, long and bent lesions were risk factors for TLR in the SES group. The rates of restenosis and TLR in patients on hemodialysis are compared between the two groups, SES and PES, in Table 6. The rates of restenosis in these patients were 32.1% (n = 18) and 20.0% (n = 9) in the SES and PES groups, respectively ($P = 0.17$). The rates of TLR in these patients were 30.4% (n = 17) and 11.1% (n = 5), for SES and PES, respectively ($P = 0.02$). The rates of restenosis and TLR in patients with long lesions are

Table 3. Clinical outcomes of all patients.

	SES (n = 462)	PES (n = 432)	p
Variable			
Restenosis, n (%)	74 (16.0)	76 (17.6)	0.53
Target vessel revascularization, n (%)	49 (10.6)	38 (8.8)	0.36
Target lesion revascularization, n (%)	40 (8.7)	25 (5.8)	0.099
Death, n (%)	3 (0.65)	5 (0.93)	0.42
Myocardial infarction, (%)	3 (0.65)	3 (0.69)	0.93
CABG, n (%)	0 (0.0)	3 (0.69)	0.073
MACE, n (%)	43 (9.3)	34 (7.9)	0.44

MACE, major adverse cardiac events : all cause death, myocardial infarction, target lesion revascularization.

Table 4. Risk factors for restenosis in univariate and multivariate analyses.

		Univariate analysis			Multivariate analysis		
		Odds ratio	95% CI	p Value	Odds ratio	95% CI	p Value
SES							
	Diabetes mellitus	1.48	0.88 to 2.47	0.14			
	Hemodialysis	2.96	1.58 to 5.55	< 0.01	1.753	0.79 to 3.88	0.17
	Calcified lesion	3.11	1.84 to 5.27	< 0.01	1.881	1.00 to 3.66	0.063
	CTO	0.97	0.54 to 1.88	0.97			
	Long lesion	2.49	1.48 to 4.19	< 0.01	1.894	1.07 to 3.33	0.026
	Bent lesion	3.68	2.19 to 6.16	< 0.01	2.573	1.47 to 4.49	< 0.01
	Small vessel	1.64	0.98 to 2.70	0.53			
PES							
	Diabetes mellitus	0.87	0.53 to 1.42	0.57			
	Hemodialysis	1.01	0.45 to 2.28	0.97			
	Calcified lesion	1.81	0.99 to 3.31	0.052			
	CTO	0.54	0.21 to 1.42	0.21			
	Long lesion	0.93	0.57 to 1.52	0.77			
	Bent lesion	0.81	0.47 to 1.35	0.41			
	Small vessel	1.12	0.66 to 1.89	0.68			

CTO, chronic total occlusion.

compared between the SES and PES groups in Table 7. The rates of restenosis in these patients were 22.3% for SES (n = 49) and 17.3% for PES (n = 43 ; $P = 0.18$) and those of TLR were 13.2% (n = 29) and 4.4% (n = 11), for SES and PES, respectively ($P < 0.01$). The rates of restenosis and TLR in patients with bent lesions are compared between the SES and PES groups in Table 8. The rates of restenosis in these patients were 30.8% in the SES group (n = 37) and 15.6% in the PES group (n = 24 ; $P < 0.01$) and those of TLR were 30.8% (n = 37) and 5.8% (n = 9), in the SES and PES groups, respectively ($P < 0.01$).

Table 5. Risk factors for target vessel revascularization in univariate and multivariate analysis.

		Univariate analysis			Multivariate analysis		
		Odds ratio	95% CI	p Value	Odds ratio	95% CI	p Value
SES							
	Diabetes mellitus	0.17	0.87 - 3.45	0.12			
	Hemodialysis	7.26	3.58 - 14.74	< 0.01	7.61	2.78 - 20.86	< 0.01
	Calcified lesion	3.59	1.85 - 6.97	< 0.01	0.89	0.34 - 2.32	0.81
	CTO	0.99	0.44 - 2.23	0.98			
	Long lesion	3.19	1.55 - 6.55	< 0.01	2.63	1.18 - 5.84	0.018
	Bent lesion	5.09	2.60 - 9.98	< 0.01	3.47	1.65 - 7.27	< 0.01
	Small vessel	1.01	0.52 - 1.96	0.98			
PES							
	Diabetes mellitus	0.61	0.27 - 1.38	0.24			
	Hemodialysis	2.29	0.82 - 6.44	0.12			
	Calcified lesion	1.93	0.71 - 5.21	0.19			
	CTO	1.15	0.33 - 4.02	0.82			
	Long lesion	0.64	0.28 - 1.47	0.29			
	Bent lesion	1.01	0.44 - 2.34	0.98			
	Small vessel	0.51	0.19 - 1.36	0.19			

CTO, chronic total occlusion.

Table 6. Comparison of restenosis rates among patients on hemodialysis.

	SES (n = 56)	PES (n = 45)	p
Restenosis, n (%)	18 (32.1)	43 (20.0)	0.17
Target lesion revascularization, n (%)	17 (30.4)	5 (11.1)	0.02

Table 7. Comparison of restenosis rates among patients with long lesions.

	SES (n = 220)	PES (n = 248)	p
Restenosis, n (%)	49 (22.3)	43 (17.3)	0.18
Target lesion revascularization, n (%)	29 (13.2)	11 (4.4)	< 0.01

Table 8. Comparison of restenosis rates among patients with bent lesions.

	SES (n = 120)	PES (n = 154)	p
Restenosis, n (%)	37 (30.8)	24 (15.6)	< 0.01
Target lesion revascularization, n (%)	24 (20.0)	9 (5.8)	< 0.01

Discussion

Various investigators have cited diabetes, hemodialysis, calcified lesions, CTO, bent lesions, long lesions and vessel diameter as risk factors for restenosis after PCI with bare metal stents. However, independent clinical and angiographic risk factors for coronary restenosis after SES or PES implantation have never been reported as far as we can ascertain.

The rates of late loss are lower for SES than for PES. We also found no significant differences in late loss between SES and PES implantations, although SES was more frequently deployed than PES in small vessels and left main trunk lesions. In fact, the results of many randomized trials have indicated that SES can suppress neo-intimal hyperplasia more effectively then PES, and this results in a reduction of in-stent and in-segment late loss. However, this is not always associated with a reduction in binary restenosis, target vessel revascularization and MACE, as shown by the large randomized REALITY trial.18 On the other hand, some smaller randomized trials such as ISAR-SMART and SIRTAX have identified better angiographic or clinical parameters for SES than for PES[19, 20].

Although most clinical comparisons of SES and PES have found better angiographic results for SES than PES, the two largest randomized stent trials and several smaller randomized controlled trials and registries have found equivalent clinical outcomes for the two types of stents[21-24]. The present study found no differences between SES and PES in terms of clinical MACE, target lesion or vessel revascularization and restenosis, before and after adjustment for confounding factors in the setting of a routine practice. A meta-analysis of 16 randomized trials of SES versus PES in patients with coronary artery disease indicated that SES was more effective than PES in reducing the risk of re-intervention and stent thrombosis[25]. Another meta-analysis found a lower frequency of TLR within six months of SES deployment and of angiographic restenosis. However, these analyses included different study populations with variable follow-up durations and endpoint definitions, which might limit the ability to reach a firm conclusion. The TAXi-LATE trial compared long-term (3-year) clinical outcomes of stenting with SES versus PES. The findings of that study supported previously published data indicating that both are equivalent in terms of treating coronary artery lesions[26].

The present study uncovered significant differences in risk factors for restenosis and TLR between SES and PES. The risk factors for restenosis associated with SES were hemodialysis, as well as calcified, long and bent lesions, and the latter two were independent risk factors for coronary restenosis. On the other hand, no risk factors were found for coronary restenosis in the PES group. Risk factors in the SES group for TLR were hemodialysis, calcified, long and bent lesions. Independent risk factors for TLR in the SES group were hemodialysis, long and bent lesions, but no risk factors were associated with the PES group. Stent design might have played a role in these differences, but whether or not the stent platform is directly involved in restenosis has not been reported. However, some studies

have found that the stent platform is associated with stent fracture, which is involved in restenosis. The closed-cell design might cause SES to become more rigid and thus more prone to fracture than the open-cell design of PES. Liao *et al*[27] investigated the impact of the more rigid closed-cell design. Three-dimensional reconstructions of coronary arteries showed that more vessels became straightened with closed-, than with open-cell stents, thus changing the shape of the stent ends and generating hinge points that are prone to fracture. Bent lesions, overlapping stents and the use of SES have been cited as predictors of stent fracture. Considering that SES and PES almost always overlapped in long lesions in the present study, the risk factors for stent fracture and for in-stent restenosis seemed similar.

Diabetes is a known major risk factor for in-stent restenosis after implantation with bare metal stents. The angiographic rates of restenosis are decreased in patients with diabetes after the introduction of DES. However, Hong *et al*[28] reported restenosis rates of 20.9% and 14.6% in patients with and without diabetes, respectively, even after DES implantation. The present study found no significant differences in the rates of restenosis and TLR after PCI with SES and PES.

Mousssa *et al*[29] reported that the rate of TLR after the deployment of bare metal stents is higher in patients who are on hemodialysis than in those who are not. Stenosis in patients on hemodialysis can be caused by chronic vascular inflammation, poor dilation due to calcification and complications due to dysfunctional organs including the heart. We found no significant differences in the rates of restenosis and TLR in patients on hemodialysis implanted with PES, but the rate of TLR significantly differed between such patients implanted with SES and PES. We found no differences in post-procedural minimal lumen diameter analyzed by quantitative coronary angiography between patients implanted with SES or PES, indicating that technical factors such as poor vessel dilation did not cause the high frequency of TLR in patients on hemodialysis implanted with SES. However, the open-cell design of PES is more suitable than the closed-cell design of SES for treating complex lesions such as those that are calcified in patients on hemodialysis. This could explain the difference in the rate of TLR between patients on hemodialysis in the SES and PES groups.

Several limitations of the present study need to be addressed. This study is a registry of a single-center experience involving a small patient cohort. The present findings require confirmation in a larger study. The unblinded evaluation of coronary angiography might have affected operator decisions regarding revascularization. Although baseline clinical and angiographic characteristics did not significantly differ between the SES and PES groups, the selection of the stenting strategy was at the discretion of the operators. In addition, not all patients were followed up by coronary angiography and 12 months might not be a sufficient time to discern subsequent outcomes or the relationships identified in the present study.

Conclusion

Hemodialysis, and long and bent lesions were high risk factors for revascularization after PCI with SES. Rates of restenosis and TLR in the PES group did not significantly differ among these risk factors. The rate of TLR was significantly higher in patients with long or bent lesions or patients on hemodialysis in the SES group, than in the PES group. Most clinical studies comparing SES and PES have found better angiographic results for SES than PES. However, PES might result in better clinical outcomes than SES for patients on hemodialysis or for those with long or bent lesions.

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