Titanium and Nitride Oxide-Coated Stents and Paclitaxel-Eluting Stents for Coronary Revascularization in an Unselected Population

Pasi P. Karjalainen, MD, Antti Ylitalo, MD, PhD, Juhani K.E. Airaksinen, MD, FESC

ABSTRACT: The aim of this study was to compare clinical outcome of a stainless-steel stent coated with titanium nitride oxide (TITANOX) and a paclitaxel-eluting stent (PES) in routine clinical practice represented by two prospective registries including all patients with *de novo* coronary artery disease treated exclusively with a TITANOX stent (n = 201) or with a PES (n = 204) between May 2003 and November 2004 (63% of all PCI patients). The primary endpoint of the study was major adverse cardiac events (MACE) at 30 days and 12 months. The TITANOX stent patients were more frequently (p = 0.011) treated for acute myocardial infarction and had more complex B- and C-type lesions (p = 0.004). The PES patients had longer (p < 0.001) total stent length. At 30 days, the rate of MACE was 0% and 4.9% for the TITANOX stent and PES groups, respectively (p = 0.001). A significant difference in target vessel revascularization (TVR) was seen in favor of the TITANOX stent (0% vs. 2.9% for PES; p = 0.014). This was mainly driven by stent thrombosis (n = 7). At 12 months, the difference in MACE was no longer significant (10.9% vs. 13.7%; p = 0.40), but the rate of myocardial infarction was lower in the TITANOX stent group (4.5% vs. 10.3%; p = 0.025). The rate of TVR (8% vs. 6.9%; p = 0.67) was similar between the two groups. In conclusion, both the TITANOX-coated stent and PES resulted in good clinical outcome with infrequent need for repeat interventions in the real-world setting of high-risk patients and complex coronary lesions.

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Coronary stents have reduced the risk of periprocedural complications and restenosis compared to balloon angioplasty alone.[1,2] In spite of this progress, restenosis is still a clinical problem of bare metal stents, particularly in certain high-risk patient subsets.[3,4] Widespread use of drug-eluting stents (DES) is the most effective way to reduce restenosis according to randomized, controlled trials in selected patient groups[5–9] and in everyday clinical practice.[10–12] Paclitaxel, a lipophilic molecule derived from the Pacific yew tree Taxus brevifolia, is capable of inhibiting cellular division, motility, activation, secretory processes and signal transduction.[13,14] A polymer-based, paclitaxel-eluting stent (PES) consistently reduced the rate of restenosis and the need for repeated revascularization procedures, as compared with bare metal stents.[6,7,15] Modifications in stent geometry,[16] strut thickness[17] and surface material[18] have been shown to influence the restenosis rate after bare metal stent implantation. Recently, patients with nickel allergy have been reported to be at an increased risk for restenosis after bare metal stent implantation.[19] Attempts to reduce restenosis after angioplasty with the use of various stent coatings have been largely unsuccessful.[20,21] Some studies have suggested that titanium features superior biocompatibility compared with stainless steel, gold or other surface coatings.[22,23] *In vitro* titanium nitride oxide shows diminished platelet adhesion and fibrinogen binding compared with stainless steel.[24] The Titan® stent *(Hexacath, France)* is a thin-strut (0.07–0.09 mm), balloon-expandable stent made of stainless steel and coated with titanium and nitride oxide (TITANOX) that completely prevents discharge of nickel, chromium and molybdenum. Stents coated with titanium nitride oxide reduced angiographic and ultrasonic measures of restenosis compared with stainless steel control stents in a prospective, randomized, multicenter trial (The TiNOX Trial).[25]

The aim of this study was to report one-year clinical outcome of unrestricted use of TITANOX stents and PES.

Methods

Patients and study design. The Titan PORI Registry is a prospective, single-center registry with the main purpose of evaluating the safety and efficacy of TITANOX stent implantation for consecutive unselected patients treated in daily practice. Since May 2003, PES *(Taxus®; Boston Scientific, Calway, Ireland)* have been used in our hospital as the default stent for all patients selected for DES implantation. The Taxus PORI Registry is a prospective, single-center registry designed with the purpose of evaluating the safety and efficacy of PES implantation for patients treated in daily practice. Between May 2003 and November 2004, all consecutive patients with symptoms or signs of myocardial ischemia and *de novo* coronary lesion(s) scheduled for stent implantation were considered for these registries. A total of 405 patients fulfilled the criteria and entered this study. A total of 201 patients received only 1 or more TITANOX stents, and 204 received only 1 or more PES. The study material comprised 63% of all patients who underwent percutaneous coronary intervention (PCI) during the study period. The choice of a particular stent was at the discretion of the operator, with no exclusion criteria.

The study was conducted according to the declaration of Helsinki, and written informed consent was obtained from all patients. This protocol was approved by the Ethics Committee of Satakunta Central Hospital.

From Satakunta Central Hospital, Pori, Finland.
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Address for correspondence: Pasi P. Karjalainen, MD, Satakunta Central Hospital, Sairaalatie 3, Pori, 28100, Finland. E-mail: pasi.karjalainen@satshp.fi

Table 1. Baseline clinical characteristics.

	TITANOX Stent *(201 patients)*	PES *(204 patients)*	p-Value
Age *(years)*	67 ± 10	64 ± 10	0.022
Men, *n (%)*	143 (71)	147 (72)	0.85
Diabetes, *n (%)*	34 (17)	37 (18)	0.78
Current smoking, *n (%)*	58 (29)	53 (26)	0.31
Hypercholesterolemia, *n (%)*	181 (90)	167 (82)	0.017
Hypertension, *n (%)*	133 (66)	110 (54)	0.016
Medical treatment, *n (%)*			
Acetylsalicylic acid	189 (94)	190 (93)	0.72
ß-blockers	152 (76)	160 (78)	0.50
ACE inhibitor	33 (16)	36 (18)	0.74
Lipid-lowering agents	177 (88)	164 (80)	0.034
Previous MI, *n (%)*	89 (44)	65 (32)	0.010
Previous PCI, *n (%)*	29 (14)	49 (24)	0.015
Previous CABG, *n (%)*	25 (12)	20 (10)	0.40
Multivessel disease, *n (%)*	133 (66)	137 (67)	0.94
Acute STEMI, *n (%)*	62 (31)	41 (20)	0.013
Primary angioplasty, *n (%)*	22 (11)	10 (5)	0.024
Rescue angioplasty, *n (%)*	40 (20)	31 (15)	0.21
Acute NSTEMI, *n (%)*	52 (26)	49 (24)	0.67
Unstable angina, *n (%)*	20 (10)	20 (10)	0.96

Data are mean (SD) or percentage. PES = paclitaxel-eluting stent; MI = myocardial infarction; PCI = percutaneous coronary intervention; CABG = coronary artery bypass graft surgery; STEMI = ST-elevation myocardial infarction; NSTEMI = non-ST-elevation myocardial infarction.

Table 2. Procedural and lesion characteristics.

	TITANOX Stent *(218 lesions/ 221 stents)*	PES *(244 lesions/ 247 stents)*	p-Value
Target vessel, *n (%)*			
LAD	100 (46)	124 (51)	0.29
LCX	48 (22)	37 (15)	0.060
RCA	54 (25)	68 (28)	0.45
Left main	7 (3)	7 (3)	0.83
Bypass graft *(venous)*	9 (4)	8 (3)	0.63
Lesion type, *n (%)*			
A	26 (12)	54 (22)	0.004
B1/ B2	137 (63)	171 (70)	0.10
C	55 (25)	19 (8)	< 0.001
Thrombus present, *n (%)*	35 (16)	34 (14)	0.63
Diameter of reference vessel *(mm)*	2.95 ± 0.34	2.97 ± 0.35	0.27
Lesion length *(mm)*	13.1 ± 3.4	13.5 ± 4.2	0.21
Stent diameter *(range, mm)*	2.98 (2–3.5) ± 0.34	2.97 (2.25–3.5) ± 0.34	0.83
Stent length used *(range,mm)*	15.6 (7-28) ± 3.5	21.2 (8-32) ± 6.7	< 0.001
Total stent length per lesion *(mm)*	16.0 ± 4.0	21.4 ± 6.8	< 0.001
Direct stenting, *n (%)*	48 (22)	46 (19)	0.40
GP IIb/IIIa inhibitor, *n (%)*	54 (27)	49 (24)	0.38
Clopidogrel prescription *(months)*	7.7 ± 3.3	8.2 ± 3.0	0.20

Data are mean (SD) or percentage. GP = glycoprotein; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery.

Coronary stent procedure. All patients were pretreated with aspirin (100 mg daily) and received intravenous enoxaparin (1 mg/kg) during the procedure. Oral clopidogrel was administered as a loading dose of 300 mg before or immediately after the procedure. Patients treated with PES were prescribed clopidogrel (75 mg/day) for a minimum of 6 months, based on data from randomized, controlled trials.[6] For patients treated with a TITANOX stent, clopidogrel was prescribed for a minimum of 3 months. Lesions were treated according to current standard interventional techniques, with the final strategy (direct stenting, post-dilatation, periprocedural glycoprotein IIb/IIIa inhibitor, intravascular ultrasound) left entirely to the operator's discretion. Angiographic success was defined as a residual stenosis < 30 % by visual analysis in the presence of Thrombolysis In Myocardial Infarction (TIMI) flow grade 3.

TITANOX stents were available in lengths of 7, 10, 13, 16, 19, 22 and 28 mm, and in diameters of 2, 2.25, 2.50, 2.75, 3.0 and 3.5 mm. PES were available in lengths of 8, 12, 16, 20, 24, 28 and 32 mm, and in diameters of 2.25, 2.50, 2.75, 3.0 and 3.5 mm.

Endpoint definitions and clinical follow up. The primary endpoint was major adverse cardiac events (MACE), defined as the occurrence of any of the following within 12 months after the index procedure: death from cardiac causes, Q-wave or non-Q-wave myocardial infarction, or revascularization of the target vessel (emergency or elective coronary artery bypass grafting or repeated coronary angioplasty).

Q-wave myocardial infarction was defined as either (1) the presence of chest pain or other acute symptoms consistent with myocardial ischemia and new pathologic Q-waves in ≥ 2 continuous electrocardiographic leads, or (2) elevated cardiac enzyme levels > 2 times the upper limit of normal associated with any elevation above the upper limit of normal in creatine kinase-MB levels in the presence of new pathologic Q-waves. Non-Q-wave myocardial infarction was defined as an elevated creatine kinase > 2 times the upper limit of normal associated with any elevation above the upper limit of normal in creatine kinase-MB levels.

Target lesion revascularization (TLR) was defined as a repeat intervention to

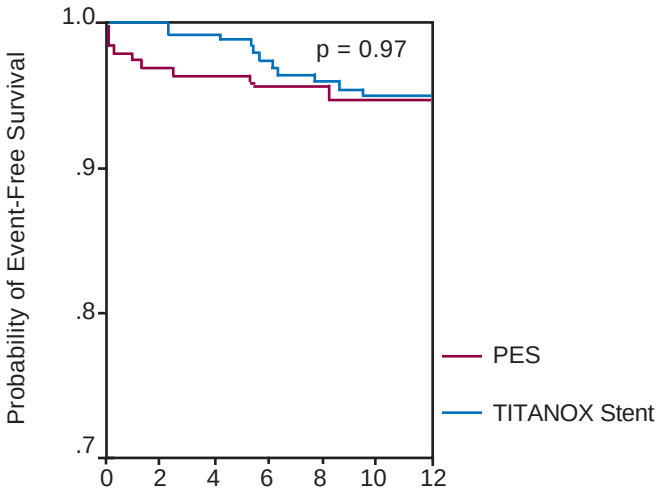

Figure 1. *Kaplan-Meier Survival Curves for target lesion revascularization (TLR).*

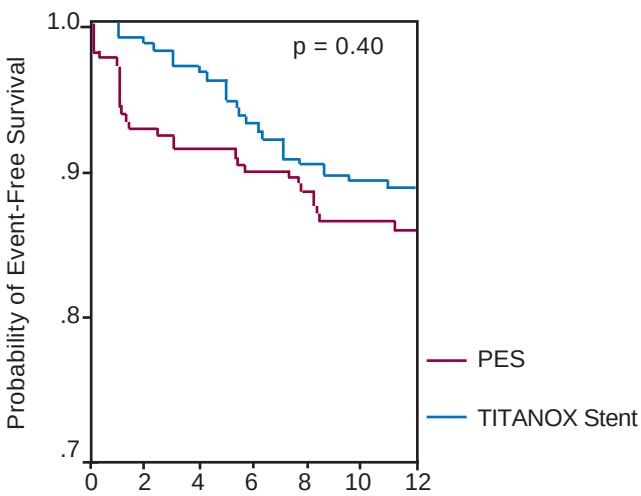

Figure 2. *Kaplan-Meier Survival Curves for major adverse cardiac events (MACE).*

treat a stenosis within the stent or in the segments 5 mm distal or proximal to the stent. Target vessel revascularization (TVR) was defined as a reintervention driven by any lesion located in the stented vessel. Stent thrombosis was diagnosed in the presence of an acute coronary syndrome with angiographic evidence of either vessel occlusion or thrombus within the study stent, or in autopsy.

All MACE were reviewed by two cardiologists (P.K, A.Y). All patients underwent clinical follow up. Adverse events were monitored at hospital discharge and by office visits or telephone interviews by the cardiologist at 1, 6 and 12 months. In addition, all data available from hospital records, the institutional electronic clinical database and the referring physicians were checked at the end of the follow-up period (February 2006) and entered into the computer database.

In both groups, follow-up coronary angiography was clinically driven by symptoms or signs suggestive of myocardial ischemia. Indication for repeat revascularization was a significant luminal stenosis (> 50% diameter stenosis) in the presence of anginal symptoms and/or proven myocardial ischemia in the target vessel territory.

Table 3. Clinical events during follow up.

	TITANOX Stent *(201 patients)*	PES *(204 patients)*	p-Value
0–1 Month			
Death, *n (%)*	0 (0)	3 (1.5)	0.09
Death from cardiac causes, *n (%)*	0 (0)	3 (1.5)	0.09
Myocardial infarction, *n (%)*	0 (0)	8 (3.9)	0.004
TVR, *n (%)*	0 (0)	6 (2.9)	0.014
TLR, *n (%)*	0 (0)	5 (2.5)	0.025
TVR (non-TLR), *n (%)*	0 (0)	1 (0.5)	0.32
MACE, *n (%)*	0 (0)	10 (4.9)	0.001
Stent thrombosis, *n (%)*	0 (0)	7 (3.4)	0.008
0–12 Months			
Death, *n (%)*	5 (2.5)	8 (3.9)	0.41
Death from cardiac causes, *n (%)*	1 (0.5)	5 (2.5)	0.104
Myocardial infarction, *n (%)*	9 (4.5)[a]	21 (10.3)[b]	0.025
TVR, *n (%)*	16 (8.0)	14 (6.9)	0.67
TLR, *n (%)*	10 (5.0)	10 (4.9)	0.97
TVR (non-TLR), *n (%)*	6 (3.0)	4 (2.0)	0.51
MACE, *n (%)*	22 (10.9)	28 (13.7)	0.40
Stent thrombosis, *n (%)*	0 (0)	7 (3.4)	0.008

TLR = target lesion revascularization; TVR = target vessel revascularization; MACE = major adverse cardiac events. [a] = 9 of 9 underwent control angiography; [b] = 16 of 21 underwent control angiography.

Statistical analysis. Continuous variables are presented as mean (SD) and were compared by Student's unpaired t-test. Categorical variables are presented as counts and percentages and were compared by the chi square or Fisher's exact test. The associations between variables were evaluated by univariate technique (Spearman's correlation coefficient). After the univariate analyses ($p < 0.1$), a logistic multivariable regression analysis was performed to identify independent predictors for MACE and stent thrombosis. The regression analysis was made for the whole population and separately in both registries. A two-sided p-value < 0.05 was required for statistical significance. Target lesion revascularization (TLR) and MACE were analyzed by means of Kaplan-Meier survival curves. All data were analyzed with the use of SPSS software, version 11.[26]

Results

Baseline and procedural characteristics. Between May 2003 and November 2004, 405 patients (462 lesions/468 stents) were enrolled; 201 patients (218 lesions/21 stents) were

Table 4. Characteristics of individual cases of PES stent thrombosis.

Patient Number	Age, Sex	Indication for PCI	Lesion Type	Target Vessel	Nominal Stent Diameter *(mm)*	Total Stent Length *(mm)*	GP IIb/IIIa Inhibitor
< 30 Days							
1	60, Male	STEMI	A	LCX	2.75	32	+
2	83, Male	UAP	B1	SVG	3.00	16	0
3	73, Male	STEMI	B2	RCA	2.75	32	+
4	76, Male	STEMI	B2	LAD	2.50	16	+
5	44, Male	STEMI	B1	RCA	2.75	24	+
6	65, Female	STEMI	A	LAD	2.50	20	0
7	67, Male	STEMI	B2	LAD	3.50	28	+
LAST							
1	54, Male	STEMI	C	LAD	2.50	28	0
2	68, Male	UAP	B1	RCA	3.00	42	0
3	76, Male	NSTEMI	B1	LAD	2.75	28	0
4	60, Male	STEMI	B1	LAD	3.00	24	+
5	60, Male	UAP	B1	LAD	3.00	20	0
6	65, Male	NSTEMI	A	RCA	3.50	16	+

LAST = late angiographic stent thrombosis; PCI = percutaneous coronary intervention; NSTEMI = non-ST-elevation myocardial infarction; STEMI = ST-elevation myocardial infarction; UAP = unstable angina pectoris; LCX = left circumflex coronary artery; SVG = saphenous vein graft; RCA = right coronary artery; LAD = left anterior descending coronary artery.

treated with TITANOX coated stent(s), and 204 patients (244 lesions/247 stents) with PES. Table 1 shows the baseline clinical characteristics of the study population. The procedural characteristics are shown in Table 2. The TITANOX stent patients ($p = 0.011$) had acute myocardial infarction more often as their presenting symptom and more ($p = 0.004$) complex B- and C- type lesions treated. The total stent length was longer ($p < 0.001$) in PES patients. The other deployment and implantation variables were similar in the two groups.

One- and twelve-month follow up. MACE during follow up are listed in Table 3. Complete follow up at 12 months was achieved in all 405 patients. At 30 days, the rate of MACE was 0% and 4.9% for the TITANOX stent and PES groups, respectively ($p = 0.001$). A significant difference in TVR was also seen in favor of the TITANOX stent patients (0% vs. 2.9%; $p = 0.014$). This was mainly driven by stent thrombosis ($n = 7$). At 12 months, the rate of myocardial infarction was higher in the PES group ($p = 0.025$), but the TLR rate was similar in both groups (Figure 1). The rate of clinical restenosis was 5% for TITANOX and 2.5% for PES ($p = 0.18$). At 1 year, 2.5% of patients in the TITANOX stent group and 3.9% in the PES group had died ($p = 0.41$). Event-free survival was 89.1% in the TITANOX stent group, as compared with 86.3% in the PES group (Figure 2). Clinically-driven control angiography was performed in 20% of the patients in the TITANOX stent group and 19% of the patients in the PES group during the 12-month follow-up period.

Late follow up. At the end of follow-up period (February 2006), the mean follow up based on hospital records was 17 ± 4 months (median 20) for the TITANOX stent patients, and 20 ± 6 months (median 25) for the PES patients. The main finding in the late follow-up data was that there were 6 cases of late (after 1 year) stent thrombosis documented angiographically, and all of them were in the PES group. Characteristics of individual cases of PES stent thrombosis are shown in Table 4. In addition, there were 4 myocardial infarctions, 2 noncardiac deaths and 1 cardiovascular death in the PES group, and 1 myocardial infarction and 1 noncardiac death in the TITANOX stent group.

Predictors of stent thrombosis and MACE. In the PES group, the patients with stent thrombosis had myocardial infarction more often as their presenting symptom before the index procedure (11% vs. 3%; $p = 0.02$), but there were no other significant predictors of stent thrombosis. MACE at 12 months was predicted by older age (70 vs. 63 years; $p = 0.001$), previous PCI ($p = 0.002$), previous CABG ($p = 0.006$) and multivessel disease ($p = 0.020$). Multivariate analysis showed that patient age ($p = 0.014$,) and previous PCI ($p = 0.012$) were the only independent predictors of MACE. In the TITANOX group, we found no significant predictors of MACE. When both registries were taken together, multivariate analysis revealed that total stent length ($p = 0.042$) was the only independent predictor of subacute, late and cumulative stent thrombosis.

Discussion

To our knowledge this is the first prospective comparison of titanium nitride oxide-coated stent with paclitaxel-eluting stent in routine clinical practice. The major finding of this study was that the unrestricted use of TITANOX stents and PES in *de novo* lesions leads to favorable and comparable outcomes after clinical decision making, even in high-risk patients with complex coronary lesions. Secondly, although the overall risk of

Table 4 (cont.). Characteristics of individual cases of PES stent thrombosis.

Patient Number	Number of Stents	Time to Thrombosis	Notes	Clinical Presentation	Angiographic Finding and Therapy	Clinical Outcome at Discharge
< 30 Days						
1	1	8 days	0	NSTEMI	New AMI after discharge, Thrombus within stent, PCI, Cardiac death	Dead
2	1	26 days	0	STEMI	New AMI, Thrombus within stent, PCI	Alive
3	1	15 days	0	NSTEMI	AMI, Cardiac death, Autopsy: Totally occluded vessel	Dead
4	1	3 days	0	STEMI	New AMI after discharge, Thrombus within stent, PCI	Alive
5	1	5 days	0	STEMI	New AMI after discharge, Thrombus within stent, PCI	Alive
6	1	1 day	0	STEMI	ST-elevation in ECG, Totally occluded vessel, PCI	Alive
7	1	29 days	0	STEMI	AMI, Totally occluded vessel, Thrombus within stent, PCI	Alive
LAST						
1	2	13 months	Clopidogrel stopped 1 month prior[a]	STEMI	ST- elevation in ECG, Thrombolysis, Thrombus within stent, PCI	Alive
2	2	15 months	Clopidogrel stopped 3 months prior[a]	STEMI	ST- elevation in ECG, Totally occluded vessel, PCI	Alive
3	1	16 months	Clopidogrel stopped 4 months prior[a]	STEMI	ST- elevation in ECG, Thrombus within stent, PCI	Alive
4	1	17 months	Clopidogrel stopped 5 months prior[a]	STEMI	ST- elevation in ECG, Thrombus within stent, PCI	Alive
5	1	23 months	Clopidogrel stopped 11 months prior[a]	STEMI	ST- elevation in ECG, Thrombus within stent, PCI	Alive
6	1	25 months	Clopidogrel stopped 13 months prior[a]	STEMI	ST-elevation in ECG, Thrombus within stent, PCI	Alive

LAST = late angiographic stent thrombosis; NSTEMI = non ST-elevation myocardial infarction; STEMI = ST-elevation myocardial infarction; AMI = acute myocardial infarction; PCI = percutaneous coronary intervention; ECG = electrocardiogram; [a] = acetylsalicylic acid monotherapy.

stent thrombosis was low, it was concentrated in the use of PES in the setting of acute myocardial infarction.

Currently, the use of DES is considered to be the most effective tool to prevent restenosis.[5–9] There is no evidence that DES could influence mortality or prevent myocardial infarction after stent implantation. PES have been shown to reduce the risk of restenosis in a broad range of lesions and patients undergoing PCI.[6,7] In the present study, the rates of clinical restenosis were low for both stent groups, substantiating the results of previous DES studies.[5–7] The three principal determinants of restenosis after coronary stent implantation are diabetes, vessel size and lesion length.[3–7] In the present

study, there was no difference in the vessel size or prevalence of diabetes between the TITANOX stent- and PES-treated patients (Tables 1 and 2). The total stent length was, however, significantly longer in the PES group, although the actual stenosis length was comparable between the two groups.

Clinically, the most alarming and unexpected finding was the high rate of stent thrombosis in the PES group. The overall rate of stent thrombosis in the PES group was higher in the present study than in previous DES studies.[27–30] The higher rate may have been due to the inclusion of patients with more complex conditions and lesions and a higher prevalence of acute coronary syndromes, since acute myocardial infarction and stent length were the significant predictors of stent thrombosis. In everyday clinical practice, late stent thrombosis may be an underestimated problem for DES, and the operators may not become aware of all late complications. Secondly, our findings underscore the importance of long-term follow up, particularly in the DES studies.

The mechanisms of late stent thrombosis are unknown, but may be related to malapposition, inadequate endothelial coverage of DES, thrombogenic surface,[31] polymer coatings[32] and drugs in high doses.[33] In the present study all cases of late stent thrombosis occurred quite shortly (range 1–13 months) after clopidogrel withdrawal, stressing the importance of adequate long-term antiplatelet therapy after PES implantation.[28] Earlier studies[28,31,34] have shown that longer stented segments may predispose patients to stent thrombosis after DES implantation. In our patient cohort, stent length was a significant predictor only when both registries were taken together.

The rate of restenosis was acceptable and there were no cases of stent thrombosis in the TITANOX group. Our current practice is to try to cover the entire plaque area with the PES, compared with a spotlike approach with bare metal stents in similar lesions. This difference in approach may reduce the risk of restenosis and thrombotic events in the TITANOX group.[28,31,35] Stent coating may also contribute to the findings, since an *in vitro* study has suggested that titanium nitride oxide reduces platelet adhesion and fibrinogen binding compared with stainless steel.[24] Similarly, a recent study compared the behavior of endothelial cells cultured on different stent materials. Metallic sheaths coated with titanium nitride (TiN) or titanium oxide (TiO2) exhibited higher cell density values on their surface compared to those without coating, supporting the view that deployment of stents coated with TiN or TiO2 may achieve earlier complete endothelial coverage.[36]

Study strengths and limitations. The strength of our single-center registry is the fact that Satakunta Central Hospital is the only center with coronary angiography capacity in the referral area. In this rural area, the population is stationary, enabling complete and sufficiently long follow up of an all-inclusive, unrestricted PCI population reflecting daily clinical practice. One of the limitations of our study is the limited size of the patient groups for subgroup analysis. This study also carries the general problems of registry-based observational studies with nonblinded outcome assessment. The fact that this is a single-center, low-patient-number registry may also give rise to unrecognized selection and performance bias. Angiographic control was performed in a minority of patients, and we may have underestimated the incidence of angiographic restenosis and silent stent thrombosis. However, there is no evidence that they were more frequent in either of the groups because the clinical outcomes were similar. On the other hand, by relying on clinical follow up only, we avoided the chance of unnecessary target lesion revascularization procedures due to the oculostenotic reflex or the patient's unjustified anxiety. In addition to the characteristics listed in the Tables, the patients in the registries may have other unrecognized differences, *e.g.*, we observed a less frequent use of PES in the setting of acute myocardial infarction during the latter part of the study period, and it is conceivable that patients at the highest risk of restenosis were more likely to be treated with PES.

Conclusions

In conclusion, both TITANOX coated stent and PES resulted in good clinical outcomes with infrequent need for repeat interventions in the real-world setting of high-risk patients and complex coronary lesions. Secondly, although the overall risk of stent thrombosis was low, it was concentrated in the use of PES in the setting of acute myocardial infarction. Further studies are warranted to randomly compare the TITANOX stent (to other passive and active coated stents) as an alternative to current DES, particularly in patients with acute myocardial infarction.

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