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Usefulness of Post-coronary Dilation to Prevent Recurrent Myocardial Infarction in Patients Treated With Percutaneous Coronary Intervention for Acute Coronary Syndrome (from the BASE ACS Trial)

Pasi P. Karjalainen, PhD[a,*], Matti Niemelä, PhD[b], Mika Laine, PhD[c], Juhani K.E. Airaksinen, PhD[d], Antti Ylitalo, PhD[a], and Wail Nammas, PhD[a]

Stent underexpansion is associated with worse outcome after stent implantation. Whether post-dilation (PD) improves outcome in patients with acute coronary syndrome (ACS) remains unclear. We performed post hoc analysis of outcome in patients from the BASE ACS (A prospective randomized comparison of titanium-nitride-oxide-coated bioactive stents with everolimus-eluting stents in acute coronary syndrome) trial who underwent PD versus those who did not. The BASE ACS trial randomized 827 patients (1:1) with ACS to receive either titanium-nitride-oxide–coated bioactive stents or everolimus-eluting stents. The primary end point was major adverse cardiac events (MACE): a composite of cardiac death, nonfatal myocardial infarction (MI), or ischemia-driven target lesion revascularization. Follow-up was planned at 12 months and yearly thereafter for up to 7 years. Of 827 patients enrolled in the BASE ACS trial, 357 (43.2%) underwent PD. Median follow-up duration was 5 years. Patients who underwent PD had less frequent nonfatal MI events at long-term follow-up, compared with those who did not (4.5% vs 8.5%, respectively, $p = 0.02$). The rates of MACE (15.7% vs 15.1%, respectively, $p = 0.81$), and the other end-points, were not significantly different ($p > 0.5$ for all). The results were consistent in propensity score–matched analysis (270 pairs). In patients treated with bioactive stents, those who underwent PD had a trend for a fewer nonfatal MI events ($p = 0.076$). Comparably, in patients treated with everolimus-eluting stents, MACE and all the individual end points were comparable ($p > 0.5$ for all). In conclusion, patients treated with early percutaneous coronary intervention for ACS who underwent PD had less frequent nonfatal MI events at long-term follow-up, compared with those who did not; MACE rates were not significantly different. **© 2016 Elsevier Inc. All rights reserved.** **(Am J Cardiol 2017;119:345–350)**

In the era of bare-metal stents, adjunctive post-dilation (PD) with noncompliant balloons inflated at higher pressure increased the final minimal stent area and doubled the frequency of optimal stent deployment.[1] With modern stent delivery systems, optimal stent deployment improved from 35.6% to 56.5% after PD in unselected patients.[2] Stent underexpansion independently predicted stent thrombosis (ST) after sirolimus-eluting stent implantation.[3] Likewise, minimal stent area was smaller in patients with in-stent restenosis after sirolimus-eluting stent implantation for both de novo and restenotic lesions.[4,5] Yet, the role of PD after implantation of new-generation drug-eluting stents in patients presenting with acute coronary syndrome (ACS) remains unclear. Several reports demonstrated safety of titanium-nitride-oxide–coated bioactive stents (BAS) in unselected cohorts and in randomized trials of ACS.[6–9] The BASE ACS (A prospective randomized comparison of titanium-nitride-oxide-coated bioactive stents with everolimus-eluting stents in acute coronary syndrome) trial showed noninferiority of BAS versus everolimus-eluting stents (EES) for the primary end point of major adverse cardiac events (MACE) in patients with ACS, at long-term follow-up.[9–12] In post hoc analysis of the trial, we explored the long-term clinical outcome of patients who underwent PD versus those who did not.

Methods

The trial design was previously described.[9] In short, the BASE ACS trial was a prospective single-blinded randomized trial conducted in 14 centers. From January 2009 to September 2010, we randomized 827 patients (1:1) presenting with ACS who underwent early percutaneous coronary intervention to receive either BAS (Titan-2; Hexacath, Paris, France) or EES (Xience V; Abbott Vascular, Santa Clara, California). Follow-up was planned at 12 months and yearly thereafter through 7 years. The trial was initiated by the investigators and conducted according to the ethical

[a]Heart Center, Satakunta Central Hospital, Pori, Finland; [b]Department of Internal Medicine, Division of Cardiology, University of Oulu, Oulu, Finland; [c]Helsinki University Hospital, Helsinki, Finland; and [d]Heart Center, Turku University Hospital, Turku, Finland. Manuscript received August 17, 2016; revised manuscript received and accepted September 29, 2016.

See page 349 for disclosure information.

*Corresponding author: Tel: (+358) 2-6277755; fax: (+358) 2-6277757.
E-mail address: pasi.karjalainen@satshp.fi (P.P. Karjalainen).

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http://dx.doi.org/10.1016/j.amjcard.2016.09.057

Table 1
Baseline clinical, angiographic and procedural characteristics of the 2 study groups

Variable	Post-Dilatation		p Value
	Yes (N = 357)	No (N = 470)	
Age (years)	63.6 ± 11.3	62.5 ± 12.3	0.19
Women	86 (24.1%)	112 (23.8%)	0.93
Diabetes mellitus	64 (17.9%)	76 (16.2%)	0.50
Current smoker	116 (32.5%)	162 (34.5%)	0.55
Hyperlipidemia	195 (54.6%)	193 (41.1%)	<0.001
Hypertension	176 (49.3%)	237 (50.4%)	0.74
Presentation by ST elevation myocardial infarction	135 (37.8%)	186 (39.6%)	0.60
Stent used (BAS/EES)	49.6%/50.4%	51.1%/48.9%	0.67
Prior myocardial infarction	39 (10.9%)	57 (12.1%)	0.59
Prior percutaneous coronary intervention	35 (9.8%)	48 (10.2%)	0.84
Prior coronary bypass	16 (4.5%)	21 (4.5%)	0.99
ACC/AHA Lesion type B/C	320 (89.6%)	411 (87.4%)	0.33
Thrombus	154 (43.1%)	210 (44.7%)	0.65
Calcified lesions	178 (49.9%)	174 (37.0%)	<0.001
Bifurcation lesions	84 (23.5%)	93 (19.8%)	0.19
Reference vessel diameter (mm)	3.16 ± 0.43	3.12 ± 0.43	0.15
Lesion length (mm)	14.9 ± 6.7	13.9 ± 5.3	0.015
Stent diameter (mm)	3.18 ± 0.44	3.12 ± 0.44	0.08
Stent length (mm)	18.8 ± 5.3	17.8 ± 5.5	0.009
Total stent length per lesion (mm)	21.9 ± 8.9	19.8 ± 8.7	0.001
Number of vessels treated per patient	1.15 ± 0.39	1.14 ± 0.39	0.84
Number of lesions treated per patient	1.20 ± 0.52	1.18 ± 0.49	0.59
Stents per culprit lesion	1.19 ± 0.41	1.11 ± 0.33	0.004
Direct stenting	85 (23.8%)	175 (37.2%)	<0.001
Stent failure	2 (0.6%)	3 (0.6%)	1.00
Procedural success	355 (99.4%)	470 (100%)	0.18
Unfractionated heparin	78 (21.8%)	137 (29.1%)	0.018
Low-molecular weight heparin	226 (63.3%)	257 (54.7%)	0.013
GP IIb IIIa inhibitor	99 (27.7%)	143 (30.4%)	0.39
Bivalirudin	45 (12.6%)	76 (16.2%)	0.15

Continuous variables are presented as mean ± SD, whereas categorical variables are presented as frequency (percentage).

ACC = American College of Cardiology; AHA = American Heart Association; BAS = bioactive stent; GP = glycoprotein.

guidelines of the 1964 Declaration of Helsinki, as revised in 2013. Informed written consent was obtained from every patient after explanation of the trial protocol; the protocol was approved by the ethics committees of the coordinating center (Satakunta Central Hospital) and the other participating centers. The trial is registered under *ClinicalTrials.gov*, with number NCT00819923.

Patients not previously maintained on aspirin were pretreated with aspirin at a loading dose of 250 mg orally or 250 to 500 mg intravenously and continued at a dose of 75 to 150 mg daily indefinitely. Oral clopidogrel was initiated at a loading dose of 300 to 600 mg before or immediately after the procedure and continued at a dose of 75 mg daily. Patients in either group were prescribed clopidogrel for a minimum of 6 months and, thereafter, for extended periods (maximum 12 months) at operator's discretion. During the procedure, low−molecular-weight or unfractionated heparin was administered intravenously in the standard dosage. Use of glycoprotein IIb and IIIa inhibitors or bivalirudin was left to operator's discretion.

PD was performed using a noncompliant balloon slightly larger (0.25 to 0.5 mm) than the stent deployment balloon, inflated at higher pressures (≥16 bars). The diagnostic criteria for non−ST-segment elevation ACS and ST-segment elevation myocardial infarction (MI) were previously described.[9] The primary end point was the first occurrence of MACE: a composite of cardiac death, nonfatal MI, or ischemia-driven target lesion revascularization (TLR). Secondary end points included noncardiac death and definite ST. Cardiac death was defined as death from cardiovascular causes or any death without known cause. ST was adjudicated according to the criteria of definite ST described by the Academic Research Consortium.[13] An independent clinical events committee whose members were blinded to stent group allocation adjudicated all the individual end points according to the prespecified definitions.

Continuous variables were presented as mean ± SD, whereas categorical variables were described with absolute and relative (percentage) frequencies. Comparisons between the 2 subgroups (patients who underwent PD vs those who did not) were performed using the unpaired *t* test for continuous variables and the Pearson chi-square test or Fisher's exact test for categorical variables, as appropriate. Data analysis was based on the intention-to-treat principle. We observed significant differences between the 2 subgroups in several baseline characteristics. Therefore, we performed a propensity score−matched analysis of the 2 subgroups to estimate the impact of PD on the clinical outcome. We calculated the propensity score using a logistic regression model in which we included—as covariates—all the baseline clinical, angiographic, and procedural variables with a difference between the 2 subgroups as indicated by a $p < 0.1$ in univariate analysis. The unmatched subgroup variable (PD vs non-PD) was entered in the model as the dependent variable. Probabilities predicted by the model were saved as a new variable: propensity score, which was then used to identify and select the matched pairs. Hosmer-Lemeshow test was used to assess the fit of the logistic regression model (chi-square: 12.18, $p = 0.143$). Finally, we used the "Caliper and Radius" matching method for selection of the matched pairs. Matching was performed based on an estimated caliper width of 0.2 the SD of the propensity score logit. Time-to-event curves were constructed using Kaplan-Meier estimates, based on all the available follow-up data for MACE, and were compared with the log-rank test. Comparison of the 2 subgroups (based on PD) for the clinical outcome was also performed stratified by stent group. All tests were 2 sided and statistical significance was set at 5%. Data were analyzed with SPSS, version 16.

Results

Of the 827 patients enrolled in the BASE ACS trial, 357 (43.2%) underwent PD. Median follow-up duration was

Table 2
Clinical outcome in the 2 study groups at long-term follow-up

Outcome Event	Post-Dilatation		Hazard Ratio (95% CI)	p Value
	Yes (N = 357)	No (N = 470)		
MACE	56 (15.7%)	71 (15.1%)	1.05 (0.71 − 1.53)	0.81
Cardiac Death	15 (4.2%)	11 (2.3%)	1.83 (0.83 − 4.04)	0.12
Non-fatal MI	16 (4.5%)	40 (8.5%)	0.50 (0.28 − 0.92)	0.02
Ischemia-driven TLR	33 (9.2%)	36 (7.7%)	1.23 (0.75 − 2.01)	0.41
Non-cardiac Death	20 (5.6%)	17 (3.6%)	1.58 (0.82 − 3.07)	0.17
Definite ST	8 (2.2%)	10 (2.1%)	1.05 (0.41 − 2.69)	0.91

Variables are presented as frequency (percentage).
CI = confidence interval; MACE = major adverse cardiac events; MI = myocardial infarction; ST = stent thrombosis.

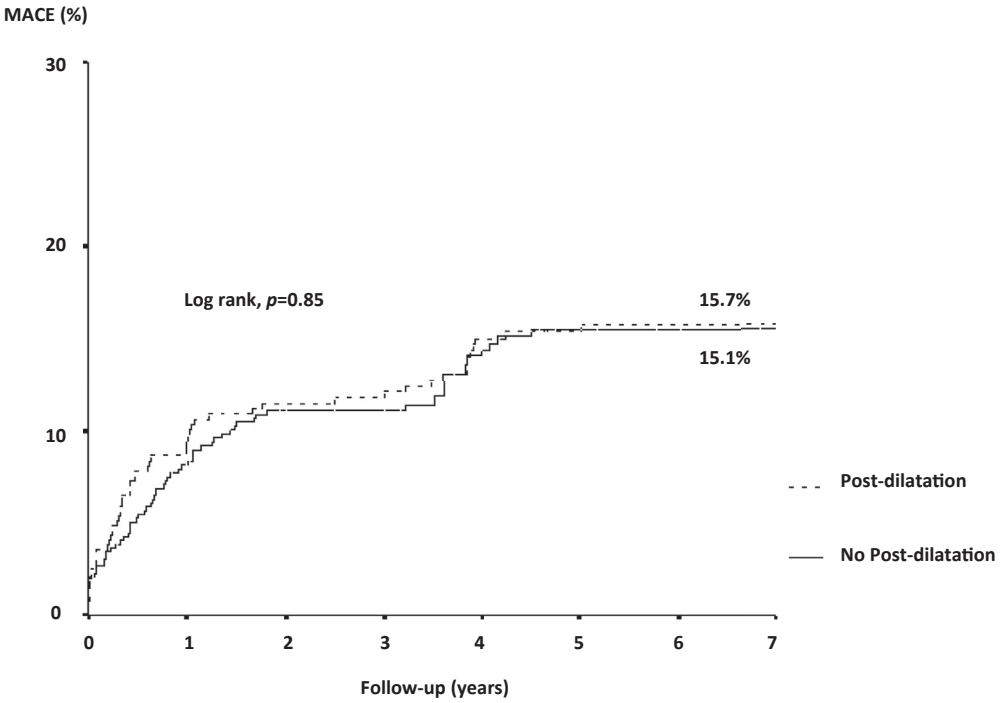

Figure 1. Kaplan-Meier estimates of the primary end point (a composite of cardiac death, nonfatal myocardial infarction, or ischemia-driven TLR) in the 2 subgroups at long-term follow-up.

5.0 years; mean (SD) 4.2 years (1.9). Compared with those who did not, patients who underwent PD were more often dyslipidemic and had longer and more frequently calcified target lesions ($p < 0.05$ for all). They underwent more often pre-dilation and received longer stents, with more stents per culprit lesion ($p < 0.05$ for all). The other baseline clinical, angiographic, and procedural data were matched (Table 1).

Patients who underwent PD had less frequent nonfatal MI events at long-term follow-up, compared with those who did not (4.5% vs 8.5%, respectively, $p = 0.02$). In patients who underwent PD, 16 patients developed nonfatal MI events: in 9 patients (56.3%), MI occurred while the patients were still on dual antiplatelet therapy; in these, 5 events (55.6%) occurred during the first 30 days. In patients who did not undergo PD, 40 patients developed nonfatal MI events: in 18 patients (45%), MI occurred while the patients were still on dual antiplatelet therapy; in these, 12 events (66.7%) occurred during the first 30 days. The cumulative incidence of MACE was not significantly different between the 2 subgroups (15.7% vs 15.1%, respectively, $p = 0.81$) (Table 2, Figure 1). The rates of cardiac death and ischemia-driven TLR were not significantly different ($p > 0.05$ both). Definite ST and noncardiac death were not significantly different ($p > 0.05$ both) (Table 2). Propensity score matching yielded 540 patients (270 pairs) with balanced baseline characteristics (Table 3). Consistently, in the propensity score−matched pairs, patients who underwent PD had less frequent nonfatal MI events, compared with those who did not (3.7% vs 10.0%, respectively, $p = 0.004$). MACE and all the other individual end points were not significantly different between the 2 matched subgroups ($p > 0.05$ for all) (Table 4). In patients treated with BAS, those who underwent PD had a trend for a fewer nonfatal MI

Table 3
Baseline clinical, angiographic and procedural characteristics of the 2 matched groups

Variable	Post-Dilatation		p Value
	Yes (N = 270)	No (N = 270)	
Age (years)	63.9 ± 10.7	62.3 ± 12.3	0.096
Women	68 (25.2%)	67 (24.8%)	0.93
Diabetes mellitus	53 (19.6%)	43 (15.9%)	0.26
Current smoker	88 (32.6%)	85 (31.5%)	0.78
Hyperlipidemia	139 (51.5%)	129 (47.8%)	0.38
Hypertension	142 (52.6%)	132 (48.9%)	0.38
Presentation by ST elevation myocardial infarction	106 (39.3%)	96 (35.6%)	0.37
Stent used (BAS/EES)	52.2%/47.8%	53.7%/46.3%	0.73
Prior myocardial infarction	33 (12.2%)	32 (11.9%)	0.89
Prior percutaneous coronary intervention	29 (10.7%)	32 (11.9%)	0.68
Prior coronary bypass	14 (5.2%)	12 (4.4%)	0.68
ACC/AHA Lesion type B/C	239 (88.5%)	241 (89.3%)	0.78
Thrombus	118 (43.7%)	115 (42.6%)	0.79
Calcified lesions	124 (45.9%)	118 (43.7%)	0.60
Bifurcation lesions	61 (22.6%)	65 (24.1%)	0.68
Reference vessel diameter (mm)	3.13 ± 0.42	3.15 ± 0.43	0.52
Lesion length (mm)	14.3 ± 5.6	14.3 ± 5.5	0.94
Stent diameter (mm)	3.15 ± 0.44	3.16 ± 0.45	0.90
Stent length (mm)	18.5 ± 5.2	18.3 ± 5.6	0.70
Total stent length per lesion (mm)	21.1 ± 8.9	21.1 ± 9.4	0.91
Number of vessels treated per patient	1.13 ± 0.36	1.14 ± 0.39	0.73
Number of lesions treated per patient	1.17 ± 0.44	1.18 ± 0.50	0.85
Stents per culprit lesion	1.16 ± 0.39	1.16 ± 0.37	0.82
Direct stenting	79 (29.3%)	67 (24.8%)	0.24
Stent failure	1 (0.4%)	3 (1.1%)	0.62
Procedural success	269 (99.6%)	270 (100%)	1.00
Unfractionated heparin	68 (25.2%)	63 (23.3%)	0.61
Low-molecular weight heparin	166 (61.5%)	175 (64.8%)	0.42
GP IIb IIIa inhibitor	72 (26.7%)	84 (31.1%)	0.25
Bivalirudin	34 (12.6%)	31 (11.5%)	0.69

Continuous variables are presented as mean ± SD, whereas categorical variables are presented as frequency (percentage).

ACC = American College of Cardiology; AHA = American Heart Association; BAS = bioactive stent; EES = everolimus-eluting stent; GP = glycoprotein.

events, compared with those who did not (2.8% vs 6.7%, respectively, p = 0.076). MACE and the other end points were not significantly different (p >0.05 for all). Comparably, in patients treated with EES, MACE and all the individual end points were not significantly different (p >0.5 for all).

Discussion

The current post hoc analysis of the BASE ACS trial demonstrated that patients treated with early percutaneous coronary intervention for ACS who underwent PD following the index procedure had less frequent nonfatal MI events at long-term follow-up, compared with those who did not; such better outcome persisted after propensity score–matched analysis. Moreover, the incidence of MI was slightly lower in patients who underwent PD after BAS implantation; yet, such incidence was comparable (between those who underwent PD and those who did not) after EES implantation. The current report is the first to address the impact of PD on the long-term clinical outcome after stent implantation in patients with ACS.

Drug-eluting stents effectively reduced restenosis rates and obviated the need for TLR in most patients who underwent percutaneous coronary intervention in contemporary clinical practice. Because PD was not routinely performed in trials that confirmed the efficacy of DES, the role of PD after drug-eluting stent implantation has ultimately come into question. In clinical practice, PD is usually operator decided and is rarely performed as a standard procedure. A few studies reported the angiographic and clinical outcome of high-pressure balloon PD after drug-eluting stent implantation. In an early study of unselected patients (n = 6,479) who underwent drug-eluting stent implantation, operator-decided PD was associated with reduction of in-stent and in-segment late lumen loss and binary restenosis rates at 7-month follow-up; yet, the rates of overall and individual MACE (death, MI, and TLR) were similar; ST rates were similar.[14] In a more recent study, unselected patients who underwent routine PD after drug-eluting stent implantation (n = 279, nearly 55% first-generation drug-eluting stents) were compared with historical controls who underwent ad hoc PD for suboptimal results (n = 262, 32% PD). The former group demonstrated better immediate angiographic outcome versus the latter; at 12-month follow-up, routine PD was associated with lower rates of MACE (death, MI, target vessel revascularization, definite/probable ST), TLR, and target vessel revascularization; however, MI included cases of periprocedural MI that were frequent in both groups (8.2% vs 8.4%, respectively).[15] Another study explored the outcome of operator-decided PD in patients with ST-elevation MI (n = 191) who underwent primary percutaneous coronary intervention with drug-eluting stents: compared with those who did not, patients who underwent PD had less often target vessel revascularization and definite/probable ST at 6-month follow-up; yet, the 2 groups had similar immediate angiographic outcome (Thrombolysis In Myocardial Infarction flow, myocardial blush).[16] Nevertheless, no propensity score matching was performed in the aforementioned studies (2 of which are small sized); the comparison groups remained unmatched for several key baseline characteristics. Moreover, outcome was reported at mid-term follow-up. Two other retrospective studies suggested worse outcome in patients who underwent PD, versus those who did not. In post hoc analysis of the National Heart, Lung, and Blood Institute Dynamic Registry, patients who presented with acute MI and underwent PD had a higher risk of death/MI at 1 year compared with non-PD, and repeat revascularization was similar; in those who presented without acute MI, outcome was similar.[17] In large registry data, PD was associated with a higher restenosis risk, similar ST risk, but a lower death risk.[18] In the current post hoc analysis of a

Table 4
Clinical outcome in the 2 matched groups at long-term follow-up

Outcome Event	Post-Dilatation		Hazard Ratio (95% CI)	p Value
	Yes (N = 270)	No (N = 270)		
MACE	40 (14.8%)	44 (16.3%)	0.89 (0.56 − 1.42)	0.63
Cardiac Death	10 (3.7%)	7 (2.6%)	1.45 (0.54 − 3.85)	0.46
Non-fatal MI	10 (3.7%)	27 (10.0%)	0.35 (0.16 − 0.73)	0.004
Ischemia-driven TLR	24 (8.9%)	18 (6.7%)	1.37 (0.72 − 2.58)	0.33
Non-cardiac Death	16 (5.9%)	9 (3.3%)	1.83 (0.79 − 4.21)	0.15
Definite ST	6 (2.2%)	4 (1.5%)	1.51 (0.42 − 5.42)	0.52

Variables are presented as frequency (percentage).
CI = confidence interval; MACE = major adverse cardiac events; MI = myocardial infarction; ST = stent thrombosis; TLR = target lesion revascularization.

randomized trial, patients who underwent PD had a lower incidence of nonfatal MI at long-term follow-up versus those who did not, both in crude and propensity score−matched analysis. The incidence of definite ST was comparable between the 2 groups, both in crude and matched analysis. Cases of probable ST (acute MI in the index vessel territory or cardiac death within 30 days of the index procedure) could have contributed to the discrepancy between the relative rates of nonfatal MI and definite ST between the 2 comparison groups. Interestingly, the 6-month incidence of definite/probable ST was lower with PD in patients with ST-elevation MI who underwent primary percutaneous coronary intervention with drug-eluting stents.[16]

Stent underexpansion is common and portends a high risk of ST and restenosis after implantation of first-generation drug-eluting stents.[3−5,19] Stent underexpansion is related to acute strut malapposition immediately after stent implantation.[20] Stent segments with acute malapposition portend a higher risk of delayed neointimal strut coverage and late malapposition, compared with well-apposed segments at implantation.[21] Moreover, thrombus resolution underneath the implanted stent might occur in patients treated for ACS, further contributing to late strut malapposition. In a study by intravascular ultrasound at long-term follow-up, late acquired malapposition could be attributed to positive vessel remodeling and/or plaque/thrombus resolution.[22] In a meta-analysis of 17 studies with intravascular ultrasound performed at 6 to 9 months, the risk of (very) late ST was sixfold higher in patients with, versus those without, late malapposition (late acquired or persistent).[23] Moreover, in 2 recent studies by optical coherence tomography, strut malapposition was the most common identifiable mechanism in patients presenting by late and very late ST after drug-eluting stent implantation.[24,25]

The BASE ACS trial was not designed a priori to explore specific differences in outcome based on PD following the index procedure. Because of the retrospective nature of this post hoc analysis, some data relevant to the outcome might have been missed. In addition, the trial cohort is underpowered for specific subgroup analysis. Moreover, analysis of patient data in 1 subgroup that includes different stent designs should be interpreted with caution. Furthermore, the current post hoc analysis was a nonrandomized subgroup analysis: the trial cohort was not randomized based on the index subgroup analysis (PD versus non-PD), but instead, PD was performed ad hoc, based on operator decision; this might limit the conclusiveness of the results. Finally, medication use was reported only at baseline, but not at different time points of follow-up; this might have potential effect on the results.

Acknowledgment: The authors thank Tuija Vasankari, Eija Niemelä, and Minna Ampio for their support in the conduct of this study.

Disclosures

The authors declare that there is no conflict of interest. The current post hoc analysis received no grants from any funding agency in the public, commercial, or not-for-profit sectors.

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