

Long-term clinical outcome of titanium-nitride-oxide-coated stents versus everolimus-eluting stents in acute coronary syndrome: Final report of the BASE ACS trial



Pasi P. Karjalainen ^{a,*}, Wail Nammas ^{a,1}, Antti Ylitalo ^{a,1}, Bernard de Bruyne ^{b,1}, Jacques Lalmand ^{c,1}, Adam de Belder ^{d,1}, Fernando Rivero-Crespo ^{e,1}, Kari Kervinen ^{f,1}, Juhani K.E. Airaksinen ^{g,1}

^a Heart Center, Satakunta Central Hospital, Pori, Finland

^b Cardiovascular Center Aalst, OLV-Clinic, Aalst, Belgium

^c CHU de Charleroi, Charleroi, Belgium

^d Department of Cardiology, Brighton and Sussex University Hospital NHS Trust, Brighton, UK

^e Interventional Cardiology Section, La Princesa University Hospital, Madrid, Spain

^f Clinical Research Center, Oulu University Hospital, University of Oulu, Oulu, Finland

^g Heart Center, Turku University Hospital, Turku, Finland

ARTICLE INFO

Article history:

Received 3 June 2016

Received in revised form 28 July 2016

Accepted 30 July 2016

Available online 01 August 2016

Keywords:

Titanium-nitride-oxide-coated stents

Everolimus-eluting stents

Acute coronary syndrome

Outcome

ABSTRACT

Background: The BASE ACS randomized trial demonstrated non-inferiority of titanium-nitride-oxide-coated bioactive stents (BAS), compared with everolimus-eluting stents (EES), for the primary endpoint of major adverse cardiac events (MACE) in patients presenting with acute coronary syndrome (ACS) at 12-month follow-up. We report the final long-term clinical outcome of the trial.

Methods: We randomly assigned 827 patients with ACS to receive either BAS (417) or EES (410). The primary endpoint was MACE: a composite of cardiac death, non-fatal myocardial infarction (MI) or ischemia-driven target lesion revascularization (TLR) at 12-month follow-up. Analysis was performed by intention to treat. Follow-up was planned at 12 months, and yearly thereafter through 7 years.

Results: Mean follow-up duration was 4.2 ± 1.9 years (median 5.0 years). At 5-year follow-up, BAS was non-inferior to EES for the primary endpoint of MACE (14.4% versus 17.8%, respectively; hazard ratio for BAS versus EES, 0.82; 95% confidence interval, 0.58–1.16; $p = 0.26$ for superiority; $p < 0.001$ for non-inferiority). The rate of non-fatal MI was lower in the BAS group (5.9% versus 9.7%, respectively, $p = 0.028$). The rates of cardiac death and ischemia-driven TLR were comparable (2.8% versus 3.8%, and 8.3% versus 9.9%; $p = 0.76$ and $p = 0.58$, respectively).

Conclusions: In the current final report of the randomized BASE ACS trial in patients with ACS, BAS implantation was associated with a rate of cumulative MACE at long-term follow-up that was statistically non-inferior to EES.

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1. Introduction

Percutaneous coronary intervention (PCI) with drug-eluting stents (DES) implantation is the current standard of care for treatment of obstructive coronary artery disease. Evidence suggests a higher risk of late – and very late – stent thrombosis (ST) associated with first-generation DES [1,2]. The second-generation everolimus-eluting stents (EESs) improved safety outcome compared with first-generation DES [3,4]. Yet, most randomized controlled trials comparing alternative DES have been underpowered to detect a difference in ST; the rare nature of the event needs a large sample size and long-term follow-up in order for a difference in event rates to become apparent. Definite/probable ST continued to occur at an appreciable rate following EES implantation in an adequately powered real-world registry, at 4-year follow-up [5]. Additionally, nearly 10% of patients who undergo coronary

angioplasty with stenting, have a permanent indication for oral anticoagulation; extended dual antiplatelet therapy after DES implantation increases the risk of bleeding in such patients [6].

Titanium-nitride-oxide-coated bioactive stents (BAS) are based on a 316L stainless-steel platform coated with a thin atomic layer of titanium-nitride-oxide, with a strut thickness of 91 μm . Several reports from unselected populations and from randomized trials in patients presenting with acute coronary syndrome (ACS) demonstrated the safety of BAS [7–10]. The BASE ACS trial showed non-inferiority of BAS, versus EES, for the primary endpoint of major adverse cardiac events (MACE) in patients with ACS, at 1-, 2- and 4-year follow-up [10–12]. We present the final report of the trial outcome at long-term follow-up, with focus on definite ST.

2. Methods

2.1. Trial design and patient selection

The trial design was described elsewhere [10]. Briefly, the BASE ACS trial was a prospective assessor-blinded randomized controlled trial conducted in 14 centers. From January 2009 to September 2010, we randomized 827 patients (1:1) presenting with ACS who underwent early PCI to receive either BAS (Titan-2[®], Hexacath, Paris, France) or EES (Xiience V[®], Abbott Vascular, Santa Clara, CA, USA). Follow-up was planned at 12 months, and yearly thereafter through 7 years.

2.2. Ethical issues

The study was initiated by the investigators and conducted according to the ethical guidelines of the 1975 Declaration of Helsinki, as revised in 2013. Informed written consent was obtained from every patient after full explanation of the study protocol. The study protocol was approved by the ethics committees of the co-ordinating center (Satakunta Central Hospital, Pori, Finland), and the other participating centers. The study was registered in www.clinicaltrials.gov, number NCT00819923.

2.3. Procedures and pharmacological intervention

Predilatation of the culprit lesion, PCI technique, selection of access site, antithrombotic agent, and use of glycoprotein IIb/IIIa inhibitors were all left to the operator's discretion. In patients not maintained on aspirin, the study protocol recommended premedication with aspirin at a loading dose of 100–500 mg orally, or 250–500 mg intravenously. Clopidogrel was administered at a loading dose of 300–600 mg orally before or immediately after the index procedure. At discharge, aspirin was prescribed at a dose of 100 mg daily orally, indefinitely, and clopidogrel at a dose of 75 mg daily orally, for at least 6 months. Operators were by necessity not blinded to stent group allocation; however, the study investigators who performed data management and analysis, and the patients were blinded.

2.4. Study endpoints and definitions

The diagnostic criteria for ST-elevation myocardial infarction (MI), non-ST-elevation MI, and unstable angina were previously described [10]. The primary endpoint was the first occurrence of MACE, defined as a composite of cardiac death, non-fatal MI (safety endpoints), and ischemia-driven target lesion revascularization (TLR) (efficacy endpoint). The definitions of these endpoints were previously described [10]. Secondary endpoints included all-cause death, non-cardiac death, a composite of cardiac death or non-fatal MI, and ST. We adopted the 'definite' category of ST as defined by the Academic Research consortium [13]. An independent clinical events committee whose members were blinded to stent group allocation adjudicated the individual endpoints according to the prespecified definitions. A Data and Safety Monitoring Committee reviewed safety data periodically, and recommended each time that the study continues without modification.

2.5. Statistical analysis

Continuous data are presented as mean \pm SD, whereas categorical data are described as counts and proportions (percentage). Data analysis was based on the intention-to-treat principle. Unless otherwise stated, comparisons between the two groups were performed using the unpaired two-tailed *t*-test for continuous variables, and the Pearson chi-square test or Fisher Exact test for categorical variables, as appropriate. Time-to-event curves were constructed using Kaplan–Meier estimates, and the incidences compared using log-rank testing. Landmark analyses were performed for the curves of MACE and definite ST using a landmark point of 1 year and between 1 and 7 years. The 5-year incidences (percentages) of the clinical outcome endpoints were presented as Kaplan–Meier estimates, and compared with log-rank testing. The hazard ratios (HR) and 95% confidence intervals (CI) of the clinical outcome endpoints were derived from Cox proportional hazard models. Patients who received ESS served as the reference group for all analyses. In order to identify the independent predictors of adverse outcome (MACE and definite ST), initially, univariate logistic regression analysis was performed for each of the baseline, angiographic, and procedural variables. Thereafter, the variables significantly associated with the adverse outcome (2-sided $p < 0.1$) in univariate analysis were included as covariates in a multivariable Cox proportional hazard model in which the dependent variable was the index adverse outcome (MACE or definite ST). The results of the multivariable regression were presented as HR with 95% CI. All tests were two-sided and statistical significance was set at 5%. All data were analyzed with SPSS version 16.

3. Results

3.1. Baseline clinical, angiographic and procedural data

We randomized 827 patients to receive either BAS (417 patients, 480 lesions) or EES (410 patients, 484 lesions). The mean age was 63 ± 12 years; 76.1% were males; 16.9% diabetic. The baseline clinical, angiographic, and procedural data were matched between the 2 groups

(Table 1). In particular, presentation by ST-elevation MI and the frequency of complex lesions (AHA/ACC type B and C) were comparable ($p = 0.98$, and $p = 0.33$, respectively). More vessels (and lesions) were treated in the EES group, compared with the BAS group ($p < 0.05$ both); however, the number of stents per culprit lesion, and the total stent length per lesion were comparable ($p > 0.05$ both).

3.2. Clinical outcome

Median follow-up duration was 5.0 years; mean (SD) 4.2 (1.9) years. At 5-year follow-up, BAS was non-inferior to EES with respect to the primary composite endpoint of MACE (14.4% versus 17.8%, respectively; HR, 0.82; 95% CI, 0.58–1.16; $p = 0.26$ for superiority; $p < 0.001$ for non-inferiority) (Table 2, Fig. 1A). The Kaplan–Meier curves started to diverge after 1 year, and continued to diverge thereafter till the end of follow-up (Fig. 2A). During the first year, the incidence of MACE was comparable between the 2 stent arms (9.6% versus 9.0%, respectively; HR, 1.04; 95% CI, 0.81–1.32; $p = 0.81$); during the period from 1 through 7 years, the incidence of MACE was also comparable (although numerically lower) in patients who received BAS versus those who received EES (4.8% versus 8.8%, respectively; HR, 0.57; 95% CI, 0.33–1.01; $p = 0.052$). The incidence of non-fatal MI at 5-year follow-up was lower in the BAS group (5.9% versus 9.7%, respectively; HR, 0.56; 95% CI, 0.33–0.95; $p = 0.028$). This was driven by fewer events during the first year

Table 1

Baseline clinical, angiographic and procedural data of the 2 study groups.

Variable	BAS group N = 417	EES group N = 410	<i>p</i> value
Age (years)	62.9 \pm 12.0	63.0 \pm 11.8	0.92
Female gender	100 (24.0)	98 (23.9)	0.94
Diabetes mellitus	65 (15.6)	75 (18.3)	0.30
Insulin-treated	19 (4.6)	17 (4.1)	0.77
Hypertension	201 (48.2)	212 (51.7)	0.31
Hyperlipidemia	191 (45.8)	197 (48)	0.51
Current smoking	144 (34.5)	134 (32.7)	0.57
Family history of IHD	192 (46.0)	185 (45.1)	0.79
Prior MI	56 (13.4)	40 (9.8)	0.10
Prior PCI	40 (9.6)	43 (10.5)	0.66
Prior CABG	20 (4.8)	17 (4.1)	0.65
Prior heart failure	17 (4.1)	20 (4.9)	0.57
Peripheral vascular disease	12 (2.9)	11 (2.7)	0.86
Prior cerebrovascular stroke	18 (4.3)	14 (3.4)	0.50
Presentation by ST-elevation MI	162 (38.8)	159 (38.8)	0.98
Unfractionated heparin	105 (25.2)	110 (26.8)	0.58
Low-molecular weight heparin	248 (59.5)	235 (57.3)	0.53
Glycoprotein IIb/IIIa inhibitor	123 (29.5)	119 (29.0)	0.88
Bivalirudin	59 (14.1)	62 (15.1)	0.69
Radial access	246 (59.0)	237 (57.8)	0.72
ACC/AHA lesion type B or C	373 (89.4)	358 (87.3)	0.33
Thrombus	193 (46.3)	171 (41.7)	0.18
Calcified lesions	183 (43.9)	169 (41.2)	0.43
Bifurcation lesions	81 (19.4)	96 (23.4)	0.16
Reference vessel diameter (mm)	3.13 \pm 0.43	3.14 \pm 0.43	0.65
Lesion length (mm)	14.4 \pm 5.4	14.3 \pm 6.5	0.73
Stent diameter (mm)	3.15 \pm 0.44	3.15 \pm 0.45	0.96
Stent length (mm)	17.9 \pm 5.2	18.5 \pm 5.6	0.18
Number of stents per culprit lesion	1.15 \pm 0.38	1.14 \pm 0.36	0.72
Total stent length per lesion (mm)	20.8 \pm 9.4	20.6 \pm 8.2	0.69
Number of lesions treated per patient	1.15 \pm 0.42	1.23 \pm 0.57	0.015
Number of vessels treated per patient	1.12 \pm 0.34	1.17 \pm 0.44	0.042
Direct stenting	134 (32.1)	126 (30.7)	0.66
Thrombus aspiration	82 (19.7)	72 (17.6)	0.43
Post-dilatation	177 (42.2)	180 (43.9)	0.67
Stent failure	0 (0.0)	5 (1.2)	0.03
Procedural success	416 (99.8)	409 (99.8)	1.00

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

ACC indicates American College of Cardiology; AHA, American Heart Association; BAS; bio-active stent; CABG, coronary artery bypass grafting; EES, everolimus-eluting stent; IHD, ischemic heart disease; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table 2
Clinical outcome in the 2 study groups at 5-year follow-up.

	BAS group N = 417	EES group N = 410	HR for EES versus BAS (95% CI)	p value
MACE	57 (14.4)	69 (17.8)	0.82 (0.58–1.16)	0.26
Cardiac death	11 (2.8)	13 (3.8)	0.89 (0.40–1.95)	0.76
Non-cardiac death	20 (5.7)	17 (5.6)	1.14 (0.59–2.17)	0.69
Total death	31 (8.4)	30 (9.2)	0.91 (0.63–1.69)	0.91
Non-fatal MI	21 (5.9)	37 (9.7)	0.56 (0.33–0.95)	0.028
Cardiac death or MI	30 (8.1)	48 (12.7)	0.63 (0.39–0.99)	0.041
Ischemia-driven TLR	33 (8.3)	37 (9.9)	0.88 (0.55–1.40)	0.58
Definite ST	4 (1.1)	14 (3.8)	0.28 (0.09–0.85)	0.015

Data are presented as number (percentage as Kaplan–Meier estimate). Hazard ratios (95% CI) are derived from Cox proportional hazard models; p values from log-rank testing. BAS indicates bioactive stent; CI, confidence interval; EES, everolimus-eluting stent; HR, hazard ratio; MACE, major adverse cardiac events; MI, myocardial infarction; ST, stent thrombosis; TLR, target lesion revascularization.

(2.2% versus 5.9%, respectively; HR, 0.67; 95% CI, 0.54–0.83; $p = 0.007$); yet, the rates were comparable from 1 through 7 years (3.7% versus 3.8%, respectively; HR, 0.97; 95% CI, 0.44–2.16; $p = 0.94$). The rates of ischemia-driven TLR were comparable between the 2 stent arms at 5 years (8.3% versus 9.9%, respectively; HR, 0.88; 95% CI, 0.55–1.40; $p = 0.58$), and during the first year (6.5% versus 4.9%, respectively; HR, 1.17; 95% CI, 0.83–1.64; $p = 0.37$); however, from 1 through 7 years, fewer ischemia-driven TLR events occurred in the BAS group (1.8% versus 5.0%, respectively; HR, 0.37; 95% CI, 0.14–0.94; $p = 0.028$).

Additionally, the incidence of definite ST at 5-year follow-up was lower in patients who received BAS (1.1% versus 3.8%, respectively; HR, 0.28; 95% CI, 0.09–0.85; $p = 0.015$) (Table 2, Fig. 1E). The Kaplan–Meier curves started to diverge as early as the first month, and continued to diverge thereafter till the end of follow-up (Fig. 2B). The definite ST rates were numerically lower in the BAS arm during the first year (0.7% versus 2.2%, respectively; HR, 0.66; 95% CI, 0.47–0.92; $p = 0.07$); and from 1 through 7 years (0.4% versus 1.6%, respectively; 0.16; 95% CI, 0.02–1.35; $p = 0.054$). During the first year, all definite ST events (12 events) occurred while the patients were on dual antiplatelet therapy with aspirin and clopidogrel; whereas from 1 through 7 years, ‘very late’ definite ST events (6 events) occurred while the patients were on aspirin monotherapy. At 1-year follow-up, 51.3% of patients in the BAS group were still maintained on clopidogrel compared with 68.3% of patients in the EES group ($p < 0.001$). The mean duration of clopidogrel use was 8.7 ± 3.6 versus 10.2 ± 3.0 months, respectively, ($p < 0.001$).

3.3. Predictors of adverse clinical outcome

In univariate analysis, the predictors of MACE at long-term follow-up were age ($p = 0.001$), diabetes ($p = 0.003$), hypertension ($p < 0.001$), prior MI ($p = 0.005$), prior PCI ($p = 0.008$), target lesion calcification ($p = 0.004$), reference vessel diameter ($p = 0.022$), and the number of vessels treated ($p = 0.011$). In multivariable analysis, the independent predictors of MACE were hypertension (HR, 1.59; 95% CI, 1.09–2.33, $p = 0.047$), target lesion calcification (HR, 1.63; 95% CI, 1.13–2.34, $p = 0.008$), reference vessel diameter (HR, 0.58; 95% CI, 0.38–0.88, $p = 0.011$), and the number of vessels treated (HR, 1.59; 95% CI, 1.10–2.31, $p = 0.013$).

Similarly, in univariate analysis, the predictors of definite ST at long-term follow-up were stent type ($p = 0.016$), target lesion calcification ($p = 0.037$), prior PCI ($p = 0.08$), prior CABG ($p = 0.011$), and hyperlipidemia ($p = 0.03$). In multivariable analysis, the independent predictors of definite ST were stent type (HR, 3.61; 95% CI, 1.19–10.96, $p = 0.023$), target lesion calcification (HR, 2.69; 95% CI, 1.01–7.19, $p = 0.048$).

4. Discussion

4.1. Major findings

The current final report of the BASE ACS trial demonstrated that in patients undergoing early PCI for ACS, BAS implantation was associated with a rate of cumulative MACE that was statistically non-inferior to EES at long-term follow-up. Moreover, BAS was associated with a better safety and similar efficacy profile versus EES at long-term follow-up; the rates of non-fatal MI, and definite ST were lower in the BAS group; the rates of ischemia-driven TLR were comparable. To the best of the authors' knowledge, this is the first trial to report the clinical outcome of a head-to-head randomized comparison of BAS versus EES in the setting of ACS, at long-term follow-up.

4.2. Safety endpoints

The rates of MACE amounted to an absolute risk reduction of 3.4% with BAS versus EES, which would translate into 34 major events avoided at long-term follow-up per 1000 ACS patients treated with BAS rather than EES. Consequently, the number of ACS patients needed to treat with BAS rather than EES, to prevent one major event would be 30 patients, a rather small figure in real-world practice. Moreover, the slightly higher rate of cumulative MACE associated with EES versus BAS at long-term follow-up was mainly driven by a higher rate of non-fatal MI; the latter probably driven by a higher rate of ST. At 12-month follow-up, the HR for definite ST (safety endpoint) associated with EES versus BAS was 3.1 ($p = 0.07$), this ratio slightly increased to 3.59 ($p = 0.015$) at long-term follow-up. However, it should be noted that the current study is underpowered for comparison of ST events even at long-term follow-up; hence, we cannot rule out the possibility of type I statistical error underlying this finding. This finding should, therefore, be taken as hypothesis-generating, rather than conclusive. Long-term follow-up data from randomized trials comparing alternative stent designs are of paramount importance in order to gain insight into the clinical safety and efficacy profile of such devices; nevertheless, the large randomized trials that reported extended follow-up of EES in real-life clinical practice are limited. The rate of definite ST associated with EES at 5-year follow-up in the current report (3.8%) was higher than that observed in the 5-year reports of the RESOLUTE All-Comers, SPIRIT III, ISAR TEST IV, and SORT OUT IV trials (0.8%, 1.1%, 0.6%, and 0.4%, respectively) [4,14–16]. The lower rates of definite ST associated with EES in such trials can be seen in light of the risk profile of the patients enrolled. These trials enrolled all-comer populations with much lower risk profile than that enrolled in the BASE ACS trial. The enrolment of patients presenting with ACS was most probably the main drive for the higher incidence of ST events in the EES arm of the BASE ACS trial, at comparable time points. Autopsy studies demonstrated that culprit sites in patients presenting with acute MI (underlying plaque rupture) had more delayed arterial healing (incomplete strut coverage and fibrin deposition), compared with stable patients who have underlying fibroatheroma with a thick fibrous cap; the prevalence of late ST was also higher in such patients [17]. Indeed, ‘very late’ definite ST continued to occur in the EES arm at an annual rate of nearly 0.3% in the current report. Similarly, real-world data on ‘unrestricted’ use of EES showed that very late ST occurred at a steady rate of 0.2% per year at long-term follow-up [5]. Indeed, the 5-year incidence of definite ST following EES implantation in patients presenting with ST-elevation MI was 2% in the EXAMINATION trial [18]. Yet, early ST rate associated with EES was also high in the current study (1.7%: 3 cases acute ST, 4 cases subacute ST) [10]. Possible underlying mechanisms of early ST after EES implantation include initial TIMI flow grade 0/1, high thrombus burden, and procedural factors such as stent undersizing, edge dissection, and periprocedural use of bivalirudin [10]. Initial TIMI flow grade 0/1 (observed in 6 out of 7 cases of early ST after EES) was an independent predictor of early ST in a post-hoc analysis of the

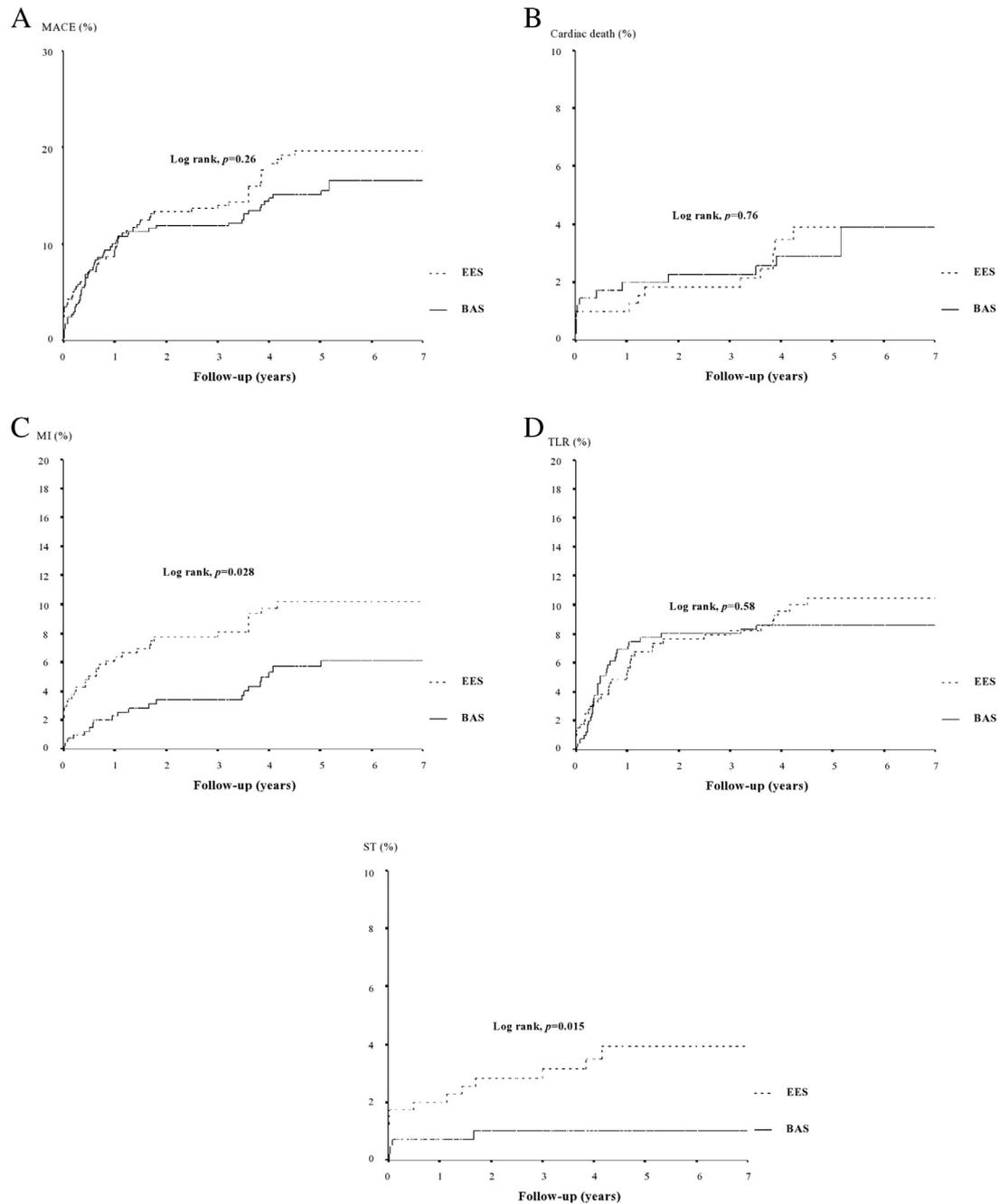


Fig. 1. Kaplan–Meier estimates for MACE and the individual endpoints through 7 years of follow-up (median 5.0 years, mean (SD) 4.2 (1.9) years): Kaplan–Meier curves show the cumulative incidence of MACE (the primary endpoint), a composite of cardiac death, non-fatal MI, or ischemia-driven TLR (Panel A); cardiac death (Panel B); non-fatal MI (Panel C); ischemia-driven TLR (Panel D); and definite ST (Panel E). BAS indicates bioactive stents; EES, everolimus-eluting stents; MACE, major adverse cardiac events; MI, myocardial infarction; ST, stent thrombosis; TLR, target lesion revascularization.

HORIZONS AMI trial [19]. Additionally, periprocedural bivalirudin (used in 2 out of 3 cases of acute ST after EES) was associated with high rates of acute ST in patients with ST-elevation MI undergoing primary PCI in the HORIZONS AMI trial [20]. Comparably, the 5-year rate of definite ST associated with BAS in the current report was similar to that observed with the same stent in the 5-year report of TITAX AMI trial (1.1% versus 0.9% respectively) [9].

4.3. Efficacy endpoint

The hazard ratio for ischemia-driven TLR (efficacy endpoint) associated with EES versus BAS at 1-year follow-up was 0.74 ($p = 0.37$). This

hazard ratio was 1.14 ($p = 0.58$) at 5-year follow-up in the current report; again, it should be noted that the current study was underpowered for comparison of the individual endpoints. There was a slightly higher incidence of ischemia-driven TLR events in the EES arm of the trial during the period from 1-year to long-term follow-up. This can be explained by the ‘late catch-up’ phenomenon: late revascularization more than 1 year following the index procedure [21]. In the j-Cypher Registry, late TLR continued to occur at an annual rate of 2.2% following sirolimus-eluting stent implantation through 5-year follow-up [22]. Predictors of late TLR after DES implantation include insulin-treated diabetes, stent diameter, and first-generation DES [23]. The exact mechanism of late restenosis following second-generation DES is far from

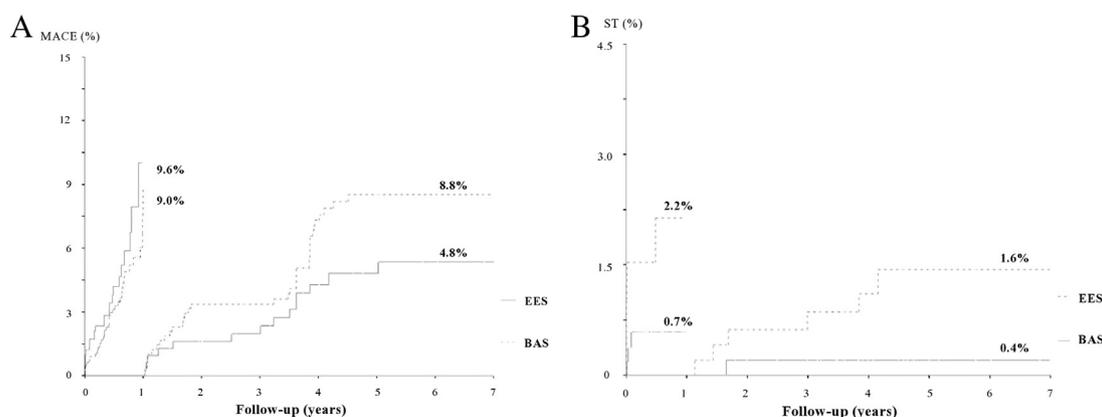


Fig. 2. Kaplan–Meier estimates for MACE (Panel A) and definite ST (Panel B) through 7 years of follow-up with landmark analysis at 1 year. BAS indicates bioactive stent; EES, everolimus-eluting stent; MACE, major adverse cardiac events; ST, stent thrombosis.

clear. The fact that we did not perform routine angiographic follow-up might have influenced the relative rates of TLR between the two stent arms. Admittedly, angiographic follow-up increases the absolute difference of TLR rates between stents beyond that which would otherwise be found with clinical follow-up. Yet, clinical follow-up reflects more faithfully real-world practice: it avoids repeat intervention for asymptomatic angiographically significant stenoses. Interestingly, the incidence of ischemia-driven TLR associated with EES at 5-year follow-up in the current report (9.9%) was comparable to those reported at 5 years in the RESOLUTE All-Comers and SPIRIT III trials, and slightly lower than that in the ISAR TEST IV trial (8.9%, 8.6%, and 12.6% respectively); however, it was higher than that in the SORT OUT IV trial (4.8%) [4,14–16]. Comparably, the incidence of ischemia-driven TLR associated with BAS in the current report was less than that reported in the TITAX AMI trial at 5 years (8.3% versus 11.2%, respectively) [9].

4.4. Study limitations

Although the current trial was well-powered to detect non-inferiority of BAS versus EES for the primary composite endpoint, it was underpowered to address the individual components of safety and efficacy, such as cardiac death, non-fatal MI, or ischemia-driven TLR, nor the secondary endpoints, such as definite ST. Second, the single-blinded nature of the trial is a weakness of the study design; however, all the study endpoints were objective, and were adjudicated by an independent events committee whose members were blinded to stent group allocation. Moreover, the current trial is not an all-comer trial; instead, some exclusion criteria existed in a background cohort of patients presenting with ACS. Exclusion criteria such as aorto-ostial lesions and lesions longer than 28 mm, might have, to some extent, favored the outcome of BAS, introducing selection bias. We also acknowledge the limitation that dual antiplatelet therapy was not extended for 12 months in all patients, as recommended by the last update of the guidelines for management of ACS. Finally, whether the results of the current trial can be extrapolated to other second-generation DES remains unclear.

5. Conclusion

In the current final report of the prospective randomized BASE ACS trial in patients presenting with ACS who underwent early PCI, BAS implantation was associated with a rate of cumulative MACE at long-term follow-up that was statistically non-inferior to EES.

Conflict of interest

All authors state that no conflict of interest exists.

Funding

The study was supported by grants from the Finnish Foundation for Cardiovascular Research, Helsinki, Finland. This work was also supported by unrestricted institutional grant from Hexacath, Paris, France; however, the company had no role in study design, data collection, data analysis, and interpretation, or manuscript writing and submission for publication.

Acknowledgements

The authors thank Tuija Vasankari, RN, Eija Niemelä, RN, and Minna Ampio, RN, for their support in the conduct of this study. We gratefully acknowledge the help of the research nurses and medical staff in participating hospitals whose cooperation made this study possible. We would like to dedicate this work to the memory of Prof. Otto M Hess (Swiss Cardiovascular Center, Bern University Hospital, Bern, Switzerland) who sadly passed away in 2011.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.ijcard.2016.07.267>.

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