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Long-term outcomes after a strategy of percutaneous coronary intervention of the infarct-related artery with drug-eluting stents or bare metal stents vs medical therapy alone in the Occluded Artery Trial (OAT)

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Abstract

Background—The OAT, a randomized study of routine percutaneous coronary intervention or optimal medical therapy (MED) alone for the treatment of a totally occluded infarct-related artery in the subacute phase after myocardial infarction, showed similar rates of death, reinfarction and congestive heart failure (CHF) between study groups. Although most percutaneous coronary intervention patients were treated with bare metal stents (BMS), drug-eluting stents (DES) were also implanted in the latter part of the study. The aim of the study was to conduct an exploratory analysis of long-term outcomes for DES vs. BMS deployment vs. MED in the OAT.

Methods—Patients enrolled after February 2003 (when first DES was implanted) were followed (DES n = 79, BMS n = 393, MED n = 552) up to a maximum of 6 years (mean survivor follow-up 5.1 years).

Results—The 6-year occurrence of the composite end point of death, reinfarction and class IV CHF was similar [20.4% of DES, 18.9% of BMS and 18.4% of MED ($P = .66$)] as were the rates of the components of the primary end point. During the follow-up period, 33.4% of DES, 44.4% of BMS and 48.1% of MED patients, developed angina ($P = .037$). The rate of revascularization during follow up was 11.3%, 20.5% and 22.5% among these groups, respectively ($P = .045$).

Conclusions—There is no suggestion of reduced long-term risk of death, reinfarction or class IV CHF with DES usage compared to BMS or medical treatment alone. An association between DES use and freedom from angina and revascularization relative to medical therapy is suggested.

The OAT was a randomized study of routine percutaneous coronary intervention (PCI) or optimal medical therapy (MED) alone for the treatment of a totally occluded infarct-related artery (IRA) in the subacute phase after myocardial infarction in stable patients who met a high-risk criterion: ejection fraction of <50% or proximal occlusion.¹ After 2.9-year mean as

well as an extended 6-year mean follow-up, the rates for the combined end point of death, reinfarction and congestive heart failure (CHF) were similar between study groups.^{1,2}

OAT was conducted predominantly in the bare metal stent (BMS) era; the use of drug-eluting stents (DES) has been linked to better angiographic outcomes.^{3,4} Our group previously reported a comparison of 1-year angiographic and 3-year clinical outcomes between BMS and DES in PCI assigned patients in the TOSCA-2, the ancillary angiographic study of OAT.⁵ Although the reduction of late loss and trend to reduction in reocclusion with the use of DES did not translate into a signal for reduction in death, reinfarction or class IV heart failure, DES use was associated with less angina over time.

Our aim was to determine the durability of these findings over the period of National Heart, Lung, and Blood Institute–funded extended long-term follow-up, and to also compare for the first time the clinical outcomes of DES and BMS treated patients to those in the MED group over the same period of observation.

Methods

Patient selection

The design of OAT and TOSCA-2 has been previously described.^{6,7} Briefly, between 2000 and 2006, 2201 patients (2166 between February 2000 and December 2005 in the main OAT trial and 35 in the extension phase of the OAT-NUC substudy in 2006) who were stable in the subacute phase post myocardial infarction with persistent IRA occlusion were randomly assigned to PCI with stenting plus optimal medical therapy versus optimal medical therapy alone.

Patients were eligible if they had a 100% stenosis of the IRA with TIMI grade 0 or 1 antegrade flow suitable for PCI and stenting on coronary angiography performed [>24 hours (on calendar days 3–28, with day 1 defined as the date of symptom onset)] after a documented myocardial infarction. OAT patients assigned to PCI underwent stenting with locally approved stents that were in general chosen at the discretion of the operator. An observational drug-eluting-stent sub-study within the NIH-funded angiographic ancillary study TOSCA-2 enrolled 30 patients who underwent implantation of a DES, including 20 who were assigned to receive a Cypher stent per protocol (Cordis; Johnson & Johnson, Miami Lakes, FL) and 10 who received a DES at the operator's discretion (all Taxus stents; Boston Scientific Corporation, Boston, MA). In addition, we included all OAT participants enrolled after Feb 2003 (when DES became available) who consented to long-term follow-up. Patients were categorized as DES if they received at least one DES in the target lesion, whether this occurred by protocol within the TOSCA-2 DES substudy or by operator discretion alone. PCI-assigned patients receiving only BMS and no DES were categorized as BMS. Only patients in whom a stent was successfully deployed were included in the analysis of the PCI group.

Treatment intervention

All patients were to receive optimal medical therapy, including aspirin, anticoagulants if indicated, angiotensin-converting-enzyme inhibitors, β -blockers, and lipid-lowering agents, unless contraindicated. Patients assigned to PCI were to undergo the procedure within 24 hours of randomization. Use of glycoprotein IIb/IIIa inhibitors was strongly recommended. Stenting was recommended for the IRA as well as for high-grade stenoses in major proximal or distal segments. PCI for stenoses in non-infarct-related arteries was permitted in the PCI and MED groups. In accordance with practice at trial start-up, thienopyridine therapy was initiated before PCI and continued for 2 or 4 weeks in patients treated with a BMS and 3 to 6 months in those who underwent deployment of a DES. After publication of data supporting

longer-term thienopyridine therapy in the setting of an acute coronary syndrome, clopidogrel was recommended for 1 year in both groups.⁸

Clinical end points and follow-up

The predefined primary clinical outcome was the composite of death, reinfarction or development of New York Heart Association Class IV heart failure with hospitalization or admission for a stay in a short-stay unit.

The prespecified definition of reinfarction required two of three criteria: the persistence of symptoms for 30 or more minutes, electrocardiographic changes, and elevated cardiac biomarkers defined as 2-fold or greater elevation of both creatinine kinase and troponin I or T, adjudicated by an independent morbidity and mortality committee. All suspect reinfarction events were re-reviewed centrally by a group of 5 investigators to permit classification according to the universal definition of myocardial infarction.⁹ Post-PCI and post-CABG myocardial infarction were defined as elevation of cardiac biomarkers 3- and 5-fold or greater respectively.⁹ Stent thrombosis was defined as myocardial infarction with documented stent thrombosis by angiography or autopsy.^{2,9} Two reviewers, blinded to treatment assignment, reviewed each event. Disagreements were adjudicated by the investigator group. Other clinical outcomes analyzed included the individual components of the primary end point, repeat revascularization and angina.

Adverse and other clinical events were tracked from randomization. Long-term patient follow-up was conducted by telephone. A national or local death index was used to determine vital status at study completion for patients who did not participate in continued follow-up or who could not be contacted.

Statistical analysis

Baseline characteristics were summarized as frequencies and percentages for categorical variables and as means and standard deviations for continuous variables. Comparisons of groups were performed using chi square test for categorical variables and 1-way analysis of variance for continuous variables. Estimates of the cumulative event rates were calculated by the Kaplan-Meier product-limit method and groups were compared by the log-rank test. Cox proportional hazards regression models were utilized to calculate hazard ratios and 99% confidence intervals. The 6-year event rates are presented because DES was first utilized in 2003 and follow-up ended in 2009. Data for patients lost to follow-up were censored as of the time of the last contact. Analyses were performed according to the intention-to-treat principle. To control for the type I error rate, the study protocol pre-specified a *P* value of .01 as evidence of differences in the secondary analyses, and a *P* value .01 to .05 as evidence of a trend toward a difference. SAS version 9.2 (SAS Institute, Cary, NC) was used for statistical analyses.

Results

Characteristics of the patients and treatment assignments

From Feb 2003 (when the first DES was implanted) to June 2006, 1024 patients were included in the OAT, of which 552 patients were assigned to MED and 472 to PCI. Among them, 393 patients underwent implantation of a BMS and 79 underwent DES implantation (Cypher *n* = 47 and Taxus *n* = 32). Assignment to DES was by protocol in the TOSCA-2 DES substudy (*n* = 20) while the remaining patients received BMS or DES according to operator preference. Patients were clinically evaluated up to a maximum follow-up of 6 years (mean survivor follow-up 5.1 years).

As shown in Table I, no differences were observed in any baseline clinical characteristics except for a higher estimated glomerular filtration rate and a lower blood pressure range in the DES group. Compliance with medical therapy at 1 year was high and balanced among study groups with two exceptions. As expected, the PCI assigned patients and especially those with DES, were treated for longer periods with dual anti-platelet therapy. In addition, fewer DES-treated patients were receiving diuretics (Table II).

Main outcomes

The 6-year cumulative event rate for the primary OAT end point of death, reinfarction and class IV heart failure was similar between groups: DES 20.4%, BMS 18.9% and MED 18.4%; $P = .66$ (Figure 1), as were the rates of the components of the primary end point. In addition, the incidence of reinfarction by the universal definition was statistically similar among groups (DES 11.8%, BMS 7.6% and MED 6.4%; $P = .18$; Figure 2) as was the incidence of reinfarction associated with stent thrombosis between DES and BMS (DES 5.4%, BMS 2.9%; $P = .17$; Figure 3). Table III shows the unadjusted comparison of hazard ratios and 99% confidence limits for the DES group to the other groups for the main study outcomes. Reinfarction according to the OAT definition tended to occur more often in the DES group than the MED group and, to a lesser extent, also more often than in the BMS group. The results were consistent with those observed using the universal definition (above and Figure 2), and were similar after adjustment for the estimated glomerular filtration rate and diastolic blood pressure (Table IV). Additional data showing the 4- and 6-year event rates are also provided in Table V.

A sensitivity analysis comparing the primary end point between the DES group and all patients who underwent BMS or MED for the entire OAT enrolment period from Feb 2000 through Jun 2006 ($n = 79$ DES, $n = 865$ BMS, $n = 1100$ MED) revealed a similar result (DES 20.4% versus BMS 20.4% vs MED 20.1%).

Angina and repeat revascularization

As shown in the Kaplan Meier curve in Figure 4, a trend was observed toward a higher event rate for developing angina in MED compared to DES (DES 33.4%, BMS 44.4%, MED 48.1%; $P = .037$). There was also a trend toward more repeat revascularization by either PCI or coronary artery bypass surgery (CABG) in MED patients (DES 11.3%. BMS 20.5% and MED 22.5%; $P = .045$; Figure 5).

Discussion

The main finding of our study is the consistency of the overall OAT long term findings of similar rates of death, reinfarction or class IV heart failure in the subset of patients undergoing PCI with DES deployment of an occluded infarct-related artery up to 4 weeks after myocardial infarction, compared to patients treated with a BMS and to patients managed with an initial strategy of medical therapy only. The lack of benefit of PCI of the occluded IRA on death, reinfarction and heart failure observed in the OAT could possibly have been due to the predominance of BMS utilized in the trial, and the resulting relatively high rate of restenosis and reocclusion at one year in the PCI group. Although the number of OAT PCI patients receiving DES was relatively small, we might have nonetheless anticipated a signal of a lower event rate based upon the higher patency rate observed at one year in the DES-treated TOSCA-2 subset. In fact, we observed a nonsignificantly higher rate of reinfarction with the PCI strategy, and more specifically with DES use, although this analysis was underpowered and the observation was not accompanied by differences in death or heart failure.

The low rate of dual anti-platelet therapy at 1 year did not explain the observed finding since the differences were primarily seen after the second year (Figure 2). Of note, greater use of clopidogrel in the PCI group might have been expected to lead to a lower rate of reinfarction in that group, based on the known benefit of clopidogrel post acute coronary syndrome. A higher risk of very late stent thrombosis in the DES group might be a plausible explanation for our results (Figure 3). Our rates of long-term stent thrombosis were consistent with those reported in studies of stenting in patients with acute myocardial infarction. A recent meta-analysis of randomized trials comparing BMS versus DES in acute myocardial infarction¹⁰ reported similar rates of stent thrombosis (4.6%) and a higher incidence of very late stent thrombosis with DES after a mean follow-up of 3.6 years. At 3 years, comparable rates of stent thrombosis with DES (5%) have also been reported in patients with PCI of total coronary occlusions.¹¹

We observed an association between DES use and freedom from both angina and non-protocol revascularization relative to medical therapy. Over 6 years of follow-up, patients treated with DES were less likely to have any angina, and less likely to require non-protocol revascularization procedures than those treated with BMS or MED. These findings extend our previously reported observations at 3-year follow-up⁵ and confirm data from previous studies comparing outcomes after BMS vs. DES deployment in total coronary occlusions.¹² The significant improvements in 1-year IRA patency and restenosis also reported in DES-treated TOSCA-2 patients likely accounts for this clinical benefit.⁵ Angina severity, angina frequency, and revascularization end points have previously been reported to influence quality of life^{13,14} and healthcare costs.¹⁵ The presence of collateral flow to the IRA has also been considered a potential determinant of future outcomes and symptoms.^{16,17} Due to the small number of patients, especially in the DES group, we did not assess the impact of collateral flow between study groups. It is unlikely that collateral status would appreciably modulate the results since in a prior OAT analysis, Steg et al found presence of collaterals to the occluded infarct artery not to be an independent predictor of major clinical outcomes.¹⁸

All DES utilized in the OAT (Taxus and Cypher) belonged to the first generation of DES. Given the better long-term outcomes and reduced late stent thrombosis resulting from the use of newer DES platforms,^{19,20} it is possible that with current DES technology, the observed trends to reduced angina and revascularization may be more accentuated, and the trend towards higher reinfarction rates reduced.²⁰

Whether or not the observed trends towards less angina occurrence and revascularization without favorable changes in death, reinfarction or heart failure rates is sufficient to recommend a DES strategy in this particular scenario will be a matter of controversy. Considering the small number of patients treated with DES, the exclusive use of first-generation DES and the non-randomized nature of this analysis, new clinical trials will be necessary to clarify if DES usage might provide any significant advantage over the medical treatment.

Limitations

The main limitation of the current analysis is the absence of randomization in patients who received DES. Although we only detected differences in estimated glomerular filtration and baseline blood pressure between study groups, patients may have differed in other unrecorded clinical or angiographic characteristics that may have influenced outcome. Another limitation is the relatively underpowered nature of the analysis due to the small number of patients allocated to DES implantation. In addition, the absence of randomization might lead operators to use DES in selected complex cases that could predispose to future reinfarction episodes. The exclusive use of first generation DES (Taxus and Cypher) and the fact that 1-year dual antiplatelet therapy was not followed in all DES patients can also be

seen as a limiting factors. Other limitations of the study include the absence of angiographic follow-up after the first year and the relatively small percentage of patients treated with dual anti-platelet therapy during the first year (especially in the PCI-assigned group).

Conclusion

There is no suggestion of reduced long-term risk of death, reinfarction or class IV CHF with DES usage compared to BMS or medical treatment alone in patients with totally occluded IRA's in the subacute phase after myocardial infarction. A potentially important association between DES use and freedom from angina and revascularization relative to BMS and medical therapy is suggested. These data must be interpreted with caution due to the small number of patients treated with DES and the non-randomized nature of this analysis.

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References

1. Hochman JS, Lamas GA, Buller CE, et al. Coronary intervention for persistent occlusion after myocardial infarction. *N Engl J Med*. 2006; 355:2395–2407. [PubMed: 17105759]
2. Hochman JS, Reynolds HR, Dzavik V, et al. Long-term effects of percutaneous coronary intervention of the totally occluded infarct-related artery in the subacute phase after myocardial infarction. *Circulation*. 2011; 124:2320–2328. [PubMed: 22025606]
3. Morice MC, Serruys PW, Sousa JE, et al. A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. *N Engl J Med*. 2002; 346:1773–1780. [PubMed: 12050336]
4. Stone GW, Ellis SG, Cox DA, et al. A polymer-based, paclitaxel-eluting stent in patients with coronary artery disease. *N Engl J Med*. 2004; 350:221–231. [PubMed: 14724301]
5. Dzavik V, Buller CE, Devlin G, et al. Angiographic and clinical outcomes of drug-eluting versus bare metal stent deployment in the Occluded Artery Trial. *Catheter Cardiovasc Interv*. 2009; 73:771–779. [PubMed: 19309733]
6. Hochman JS, Lamas GA, Knatterud GL, et al. Design and methodology of the Occluded Artery Trial (OAT). *Am Heart J*. 2005; 150:627–642. [PubMed: 16209957]
7. Dzavik V, Buller CE, Lamas GA, et al. Randomized trial of percutaneous coronary intervention for subacute infarct-related coronary artery occlusion to achieve long-term patency and improve ventricular function: the Total Occlusion Study of Canada (TOSCA)-2 trial. *Circulation*. 2006; 114:2449–2457. [PubMed: 17105848]
8. Mehta SR, Yusuf S, Peters RJ, et al. Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: the PCI-CURE study. *Lancet*. 2001; 358:527–533. [PubMed: 11520521]
9. Thygesen K, Alpert JS, White HD, et al. Universal definition of myocardial infarction. *Circulation*. 2007; 116:2634–2653. [PubMed: 17951284]
10. Piccolo R, Cassese S, Galasso G, et al. Long-term safety and efficacy of drug-eluting stents in patients with acute myocardial infarction: A meta-analysis of randomized trials. *Atherosclerosis*.

11. Rahel BM, Laarman GJ, Kelder JC, et al. Three-year clinical outcome after primary stenting of totally occluded native coronary arteries: a randomized comparison of bare-metal stent implantation with sirolimus-eluting stent implantation for the treatment of total coronary occlusions (Primary Stenting of Totally Occluded Native Coronary Arteries [PRISON] II study). *Am Heart J*. 2009; 157:149–155. [PubMed: 19081412]
12. Ma J, Yang W, Singh M, et al. Meta-analysis of long-term outcomes of drug-eluting stent implantations for chronic total coronary occlusions. *Heart Lung*. 2011; 40:e32–e40. [PubMed: 21419488]
13. Gravely-Witte S, De Gucht V, Heiser W, et al. The impact of angina and cardiac history on health-related quality of life and depression in coronary heart disease patients. *Chronic Illn*. 2007; 3:66–76. [PubMed: 18072698]
14. Andreß P, Ekre O, Grip L, et al. Fatality, morbidity and quality of life in patients with refractory angina pectoris. *Int J Cardiol*. 2009
15. Arnold SV, Morrow DA, Lei Y, et al. Economic impact of angina after an acute coronary syndrome: insights from the MERLIN-TIMI 36 trial. *Circ Cardiovasc Qual Outcomes*. 2009; 2:344–353. [PubMed: 20031860]
16. Moretti C, Biondi-Zoccai GG, Sciuto F, et al. Appraising the pathophysiologic impact of coronary collaterals as measured by fractional flow reserve on symptoms and signs of myocardial ischemia. *J Cardiovasc Med (Hagerstown)*. 2008; 9:1120–1126. [PubMed: 18852584]
17. Berry C, Balachandran KP, L'Allier PL, et al. Importance of collateral circulation in coronary heart disease. *Eur Heart J*. 2007; 28:278–291. [PubMed: 17218452]
18. Steg PG, Kerner A, Mancini GB, et al. Impact of collateral flow to the occluded infarct-related artery on clinical outcomes in patients with recent myocardial infarction: a report from the randomized occluded artery trial. *Circulation*. 121:2724–2730. [PubMed: 20547926]
19. Garg S, Serruys P, Onuma Y, et al. 3-year clinical follow-up of the XIENCE V everolimus-eluting coronary stent system in the treatment of patients with de novo coronary artery lesions: the SPIRIT II trial (Clinical Evaluation of the Xience V Everolimus Eluting Coronary Stent System in the Treatment of Patients with de novo Native Coronary Artery Lesions). *JACC Cardiovasc Interv*. 2009; 2:1190–1198. [PubMed: 20129545]
20. Garg S, Serruys PW, Miquel-Hebert K. Four year clinical follow up of the XIENCE V everolimus eluting coronary stent system in the treatment of patients with de novo coronary artery lesions: The SPIRIT II trial. *Catheter Cardiovasc Interv*.

Kaplan-Meier curves for the OAT combined primary endpoint comparing DES, BMS, and MED patients. Log-rank test = 0.66.

Figure 1.

Kaplan-Meier curves for the OAT combined primary endpoint comparing DES, BMS, and MED patients. Log-rank test = 0.66.

Figure 2.
Kaplan-Meier curves for myocardial infarction according to the universal definition of MI⁹ comparing DES, BMS and MED patients. Log-rank test = 0.18.

Figure 3.
Kaplan-Meier curves for stent thrombosis (Type 4b) MI according to the Universal Definition of MI⁹ comparing DES and BMS patients. Log-rank test = 0.17.

Figure 4.
Kaplan-Meier curve for angina comparing DES, BMS, and MED patients. Log-rank test $P = .04$.

Figure 5.
Kaplan-Meier curves for revascularization comparing DES, BMS, and MED patients. Log-rank test = .45.

Table 1

Baseline clinical characteristics of PCI patients with deployment of a DES, those receiving BMS, and those in MED group

Variable	PCI DES (N = 79)		PCI BMS (N = 393)		MED (N = 552)		P
	N	% (mean ± SD)	n	% (mean ± SD)	n	% (mean ± SD)	
Age (years)	79	(56.9 ± 11.3)	393	(57.4 ± 10.6)	552	(58.7 ± 10.9)	.13
Female gender	20	25.3	89	22.6	116	21	.63
Race							.06
White	61	77.2	312	79.4	431	78.1	
Black	4	5.1	9	2.3	16	2.9	
Hispanic	7	8.9	63	16	80	14.5	
Other	7	8.9	9	2.3	25	4.5	
Diabetes	14	17.7	71	18.1	127	23	.14
Insulin treatment	7	8.9	21	5.3	32	5.8	.48
Hypertension	39	49.4	189	48.1	289	52.4	.42
Hypercholesterolemia	43	54.4	212	53.9	280	50.8	.59
Current Cigarette Smoker	38	48.1	148	37.7	210	38	.20
History – Angina	9	11.4	81	20.6	120	21.7	.10
History - Myocardial Infarction	4	5.1	37	9.4	60	10.9	.25
History - Cerebrovascular disease	5	6.3	21	5.3	17	3.1	.14
History - Peripheral vascular disease	2	2.5	11	2.8	22	4	.55
History - Congestive heart failure	23	29.1	106	27	149	27	.92
Prior PCI	3	3.8	10	2.5	25	4.5	.28
Prior CABG	0	0	2	0.5	4	0.7	.71
NYHA class at randomization							
II	11	13.9.	61	15.6.	92	16.7	.78
ST-elevation Q-Wave or loss of R-Wave	67	84.8	350	89.1	484	87.7	.54
Estimated GFR (ml/min/1.73 m ²)	79	(89.2 ± 23.8)	391	(80.8 ± 19.4)	548	(81.3 ± 21.4)	.004
HR (bpm)	79	(72.9 ± 11.8)	393	(71.3 ± 11.4)	550	(71.2 ± 12)	.50
SBP (mmHg)	79	(118.4 ± 17.6)	393	(119.1 ± 15.1)	551	(122 ± 18.3)	.02
DBP (mmHg)	79	(71.2 ± 12.1)	393	(71.9 ± 10.3)	551	(73.6 ± 11.4)	.03
Multivessel disease	18	22.8.	64	16.3.	110	20.1	.22

Variable	PCI DES (N = 79)		PCI BMS (N = 393)		MED (N = 552)		P
	N	% (mean ± SD)	n	% (mean ± SD)	n	% (mean ± SD)	
Infarct-related artery	79		393		552		.80
LAD	26	32.9	137	34.9	210	38	
Lcx	12	15.2	60	15.3	75	13.6	
RCA	41	51.9	196	49.9	267	48.4	
TIMI flow grade	79		392		547		.75
0	61	77.2	296	75.5	428	78.2	
I	18	22.8	93	23.7	117	21.4	
II	0	0	3	0.8	2	0.4	
Collateral Vessels Present	71	89.9	349	89	489	89.6	.96
LVEF	79	(48.4 ± 11.1)	393	(47.7 ± 10)	545	(46.9 ± 10.9)	.34
LVEF <40%	19	24.1	68	17.3	120	22	.15
Angiographic data							
Stenosis, (%)	79	100	393	99.8	547	99.9	.36
Minimum lumen diameter (mm)	79	0	393	0.005	547	0.003	.69
Reference diameter (mm)	72	3.10	332	3.16	405	3.33	.01 *

NYHA, New York Heart Association; GFR, glomerular filtration rate; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; LAD, left anterior descending; Lcx, left circumflex; RCA, right coronary artery; LVEF, left ventricular ejection fraction.

* Pbetween DES and MED <0.01. Pbetween DES and BMS non significant.

Table II

Medical therapy at 1 year in the PCI DES and BMS patients and those in MED group

Medication	PCI DES (N = 74)		PCI BMS (N = 363)		MED (N = 510)	
	n	%	N	%	N	P
Aspirin	68	91.9	342	94.2	460	90.2 .10
Thienopyridine	38	51.4	83	22.9	84	16.5 <.0001
Aspirin AND thienopyridine	34	45.9	73	20.1	74	14.5 <.0001
Aspirin OR thienopyridine	72	97.3	352	97	470	92.2 .005
β -Blocker	63	85.1	305	84	441	86.5 .59
ACE inhibitor/ARB	60	81.1	302	83.2	430	84.3 .75
Lipid-lowering Drug	66	89.2	326	89.8	452	88.6 .86
Spironolactone	4	5.4	24	6.6	49	9.6 .19
Calcium Blocker	3	4.1	30	8.3	41	8 .45
Diuretics	12	16.2	71	19.6	131	25.7 .04

ACE, Angiotensin converting enzyme; ARB, angiotensin receptor blockers.

Table III

Hazard Ratios and 99% CLs for Main Outcomes (unadjusted)

Outcome	DES[79] vs BMS[393]			DES[79] vs MED[552]		
	P	Hazard ratio	HR 99%CL	P	Hazard ratio	HR 99%CL
Death, MI or CHF Class IV *	.53	1.20	0.57–2.50	.36	1.29	0.63–2.65
Death	.40	0.70	0.23–2.13	.42	0.71	0.24–2.13
Fatal and Nonfatal Recurrent MI *	.09	2.01	0.69–5.89	.04	2.32	0.82–6.60
Nonfatal Recurrent MI *	.07	2.12	0.72–6.26	.03	2.43	0.85–6.92
MI associated with Stent thrombosis †	.17	2.27	0.48–10.8			
CHF Class IV	.30	1.71	0.45–6.47	.36	1.57	0.44–5.59
CHF Class III–IV	.74	1.16	0.36–3.74	.71	1.18	0.38–3.67
Death or MI *	.74	1.11	0.50–2.43	.48	1.24	0.57–2.68
Death, MI, CHF Class III–IV *	.71	1.11	0.54–2.26	.49	1.20	0.60–2.41
Revascularization (PCI or CABG)	.18	0.61	0.23–1.60	.04	0.47	0.18–1.21
Angina	.09	0.69	0.39–1.21	.02	0.60	0.35–1.05

MI, Myocardial infarction.

* MI according to the OAT-Protocol definition.

† MI according to the Universal definition.

Table IV

Hazard Ratios and 99%CLs for main outcomes with adjustment for eGFR and DBP

Outcome	DES (79) vs BMS (393)			DES (79) vs MED (552)		
	P	Hazard ratio	HR 99%CL	P	Hazard ratio	HR 99%CL
Death, MI or CHF Class IV *	.34	1.32	0.63–2.78	.27	1.37	0.66–2.83
Death	.58	0.79	0.26–2.43	.51	0.76	0.25–2.29
Fatal and Nonfatal Recurrent MI *	.10	2.00	0.67–5.96	.03	2.47	0.86–7.11
Nonfatal Recurrent MI *	.07	2.15	0.72–6.45	.02	2.58	0.89–7.46
MI associated with Stent thrombosis †	.22	2.14	0.44–10.5			
CHF Class IV	.17	2.06	0.53–7.93	.19	1.92	0.53–6.95
CHF Class III–IV	.43	1.43	0.44–4.65	.56	1.30	0.41–4.09
Death or MI *	.53	1.21	0.55–2.68	.40	1.29	0.59–2.81
Death, MI, CHF, Class III–IV *	.44	1.24	0.61–2.54	.41	1.25	0.62–2.52
Revascularization (PCI or CABG)	.14	0.57	0.21–1.51	.03	0.46	0.18–1.18
Angina	.11	0.70	0.40–1.24	.02	0.61	0.35–1.06

* MI according to the OAT-Protocol definition.

† According to the Universal Definition.

Table V

Four- and 6-year event rates* for DES vs BMS vs MED

Outcome	DES (n = 79)		BMS (n = 393)		MED (n = 552)		P
	n	rate	n	rate	n	rate	
Death, MI, CHF Class IV							
4 y	14	18.8	56	14.6	71	13.1	
6 y	15	20.4	66	18.9	89	18.4	.66
Death							
4 y	5	6.5	36	9.4	48	8.9	
6 y	6	8.1	44	12.2	62	12.9	.70
Fatal and nonfatal recurrent MI							
4 y	7	9.9	17	4.5	21	4.1	
6 y	8	11.8	21	6.6	26	5.8	.10
Nonfatal recurrent MI							
4 y	7	9.9	16	4.2	20	3.9	
6 y	8	11.8	20	6.4	25	5.6	.077
CHF Class IV							
4 y	5	6.8	14	3.7	20	3.7	
6 y	5	6.8	15	4.3	23	4.5	.57
CHF Class III-IV							
4 y	6	8.1	25	6.7	31	5.8	
6 y	6	8.1	26	7.2	37	7.5	.93
Death or MI							
4 y	11	14.9	51	13.4	62	11.5	
6 y	13	18.3	61	17.4	80	16.8	.74
Death, MI, CHF Class III-IV							
4 y	15	20.0	65	16.9	81	15.0	
6 y	16	21.7	75	21.1	101	20.7	.77
Revascularization (PCI or CABG)							
4 y	7	9.3	55	14.7	106	19.8	
6 y	8	11.3	66	20.5	112	22.5	.064

Outcome	DES (n = 79)		BMS (n = 393)		MED (n = 552)		P
	n	rate	n	rate	n	rate	
Angina [†]							
4 y	23	30.2	148	40.4	232	44.3	
6 y	24	33.4	158	44.4	243	48.1	.037
Universal definition of MI							
4 y	7	9.9	21	5.5	23	4.4	
6 y	8	11.8	25	7.6	29	6.4	.18
Stent thrombosis							
4 y	4	5.4	8	2.1			
6 y	4	5.4	9	2.9			.17

* Life table event rates are presented.

[†] Proportion with angina (%) : Year 4: 9.8 DES vs 16.7 BMS vs 16.8 MED, *P*=.43; Year 6: 9.1 DES vs 13.7 BMS vs 17.9 MED, *P*=.51.