Journal of the American College of Cardiology Vol. 45, No. 7, 2005

Published by Elsevier Inc.
ISSN 0735-1097/05/$30.00
doi:10.1016/j.jacc.2004.11.060

Treatment of Saphenous Vein Graft Lesions With Drug-Eluting Stents

Immediate and Midterm Outcome

Lei Ge, MD,*† Ioannis Iakovou, MD,* Giuseppe M. Sangiorgi, MD,* Alaide Chieffo, MD,* Gloria Melzi, MD,* John Cosgrave, MD,* Matteo Montorfano, MD,* Iassen Michev, MD,* Flavio Airoldi, MD,* Mauro Carlino, MD,* Nicola Corvaja, MD,* Antonio Colombo, MD, FACC*

Milan, Italy; and Shanghai, China

OBJECTIVES The purpose of the present report was to evaluate clinical and angiographic outcomes of drug-eluting stent (DES) implantation in saphenous vein graft (SVG) lesions.

BACKGROUND The safety and efficacy of DES implantation for the treatment SVG lesions remains uncertain.

METHODS We evaluated in-hospital and six-month outcomes in 61 consecutive patients treated with DES in SVG lesions from March 2002 to March 2004 (DES group), as compared to 89 consecutive patients treated with bare-metal stents (BMS) in the 24 months immediately before the introduction of DES (BMS group). Major adverse cardiac events (MACE) including death, myocardial infarction, target lesion revascularization (TLR), and target vessel revascularization (TVR) were recorded in-hospital and at six-month follow-up.

RESULTS The rate of in-hospital MACE was similar between the two groups (6.6% vs. 5.6%, p = 1.0). Cumulative MACE at six months was 11.5% in the DES group and 28.1% in the BMS group (p = 0.02). The DES group had a significantly lower incidence of in-segment restenosis (10.0% vs. 26.7%, p = 0.03), TLR (3.3% vs. 19.8%, p = 0.003), and TVR (4.9% vs. 23.1%, p = 0.003). By Cox regression analysis, diabetes (hazard ratio [HR]: 3.03; 95% confidence interval [CI]: 1.33 to 6.90; p = 0.008), usage of BMS (HR: 2.53; 95% CI: 1.07 to 5.97; p = 0.03), and age of SVG (HR: 1.10; 95% CI: 1.02 to 1.19; p = 0.02) were identified as predictors of MACE at six-month follow-up.

CONCLUSIONS Compared to BMS implantation, DES implantation in SVG lesions appears safe with favorable and improved mid-term outcomes. (J Am Coll Cardiol 2005;45:989–94) © 2005 by the American College of Cardiology Foundation

Percutaneous revascularization of saphenous vein graft (SVG) lesions remains a challenge for interventional cardiologists. Balloon angioplasty in SVG lesions is associated with a high complication rate and a high incidence of restenosis (1,2). Compared to balloon angioplasty, bare-metal stent (BMS) implantation in SVG lesions has been shown to improve procedural outcomes and reduce major cardiac events (3,4). However, the incidence of in-stent restenosis remains 20% to 37% (3–5). Recently, the introduction of drug-eluting stents (DES), either sirolimus-eluting stents (Cypher, Cordis, Johnson & Johnson Company, Warren, New Jersey) or paclitaxel-eluting stents (Taxus, Boston Scientific, Natick, Massachusetts), has shown promising results in selected de novo native coronary artery lesions (6–10). The safety and efficacy of DES implantation for the treatment of SVG lesions remains uncertain (11). The aim of the present report was, therefore, to evaluate clinical and angiographic outcomes of DES implantation in SVG lesions.

From the *EMO Centro Cuore Columbus and San Raffaele Hospital, Milan, Italy; and the †Department of Cardiology, Zhongshan Hospital, Fudan University, Shanghai, China.

Manuscript received September 14, 2004; revised manuscript received October 29, 2004, accepted November 2, 2004.

METHODS

We identified 61 consecutive patients (with 69 lesions) who underwent percutaneous revascularization in SVG lesions using DES from March 2002 to March 2004 (DES group). During this time period, an additional 103 patients were treated for lesions located in SVGs; 25 of them were included in a registry utilizing a covered stent, and others were treated with BMS. The main reasons for utilizing BMS were: 1) a lesion <10 mm in length evaluated to be at low risk of restenosis; 2) a lesion located on an SVG with a reference vessel diameter (RVD) >3.5 mm for which there were no appropriately sized DES available. A control group (BMS group) was composed of 89 consecutive patients (with 120 lesions) who underwent percutaneous treatment in SVG lesions with BMS in the 24 months immediately before the introduction of DES. Patients were excluded if any of the following was present: an acute myocardial infarction (MI) <1 week before the index procedure, implantation of a covered stent, or brachytherapy.

All patients were pretreated with aspirin and either ticlopidine or clopidogrel. A 300-mg loading dose of clopidogrel before the index procedure was administered if patients were not pretreated. During the procedure, patients received intra-

Abbreviations and Acronyms

BMS = bare-metal stent
CI = confidence interval
DES = drug-eluting stent
HR = hazard ratio
MACE = major adverse cardiac events
MI = myocardial infarction
MLD = minimal lumen diameter
RVD = reference vessel diameter
SVG = saphenous vein graft
TLR = target lesion revascularization
TVR = target vessel revascularization

venous unfractionated heparin (100 IU/kg) to maintain activated clotting time between 250 to 300 s. Platelet glycoprotein IIb/IIIa receptor inhibitors and distal protection devices were used at the discretion of the operator. Stent implantation methods have been described previously (12). All stents were implanted with high deployment pressure (>12 atm). Patients received lifelong aspirin and clopidogrel or ticlopidine for at least three to six months after DES implantation and for at least one month after BMS implantation.

Clinical definitions and follow-up. Clinical follow-up was performed by either telephone contact or office visit at 6 months after the index procedure (follow-up window to 210 days). Angiographic follow-up was scheduled for between six and eight months after the procedure unless clinically indicated at an earlier time. The events analyzed in this report included death (cardiac and noncardiac), MI (Q-wave and non–Q-wave), restenosis, stent thrombosis, target lesion revascularization (TLR), and target vessel revascularization (TVR), either percutaneous or surgical.

All deaths were considered cardiac unless otherwise documented. A non–Q-wave MI was defined as creatine kinase-MB enzyme elevation ≥3 × the upper limit of the normal value; when in addition to enzyme elevation there were new pathological Q waves on the electrocardiogram, the event was defined as a Q-wave MI. Target lesion revascularization was defined as repeat revascularization secondary to a stenosis ≥50% within the stent or within the 5-mm borders proximal or distal to the stent at the follow-up angiogram. Target vessel revascularization was defined as repeat revascularization within the treated vessel. Stent thrombosis was defined as any of the following: angiographic documentation of intrastent filling defect or stent occlusion associated with a clinical event, unexplained sudden death, or MI after stent implantation and without concomitant demonstration of a patent stent (13,14). Major adverse cardiac events (MACE) were defined as cardiac death, MI, TLR, and TVR. Cumulative MACE were defined as the in-hospital and six-month follow-up MACE.

Quantitative coronary angiographic analysis. Coronary angiograms were analyzed using a validated edge detection system (CMS, version 5.2, MEDIS, the Netherlands). Minimal lumen diameter (MLD), RVD, and percent diameter stenosis at baseline, post-procedure, and at follow-up were measured, respectively. Acute gain was defined as the difference between the MLD immediately after the procedure and the baseline. Late lumen loss was defined as the difference between the MLD immediately after the procedure and at follow-up (15). Angiographic restenosis was defined as diameter stenosis ≥50% by quantitative coronary angiographic analysis within a previously stented segment (stent and 5 mm proximal and distal) at the follow-up angiogram. No reflow was defined as Thrombolysis In Myocardial Infarction (TIMI) (16) flow grade ≤1 that was not due to dissection or high-grade residual stenosis adjacent to the target lesion (17). Angiographic success was defined as a final residual stenosis <30% with TIMI flow grade 3. Procedural success was defined as the achievement of angiographic success without in-hospital MACE.

Statistical analysis. Continuous variables are presented as mean values ± SD and categorical variables as frequency (%). Continuous variables were compared using independent sample *t* test. Categorical variables were compared with chi-square statistics. Survival free of MACE was estimated using the Kaplan-Meier method, and the differences between the two survival curves were compared with the log-rank test. The Cox proportional hazards regression model was used to identify the independent predictors of MACE at six-month follow-up. The results are presented as hazard ratios (HR) with 95% confidence interval (CI). A p value of <0.05 was considered statistically significant, and all reported p values are two-sided. Statistical analysis was performed using SPSS Version 11.5 (SPSS Inc., Chicago, Illinois).

RESULTS

Baseline clinical characteristics. The baseline clinical characteristics were similar between the two groups, except for a trend toward a higher incidence of hypercholesterolemia in the DES group (65.6% vs. 49.4%, p = 0.07) (Table 1).

Angiographic and procedural characteristics. Angiographic and procedural characteristics are shown in Table 2. The percentage of restenotic lesions were significantly higher in the DES group than in the BMS group (34.8% vs. 6.7%, p < 0.001). In the DES group, sirolimus-eluting stents were implanted in 35 patients (57.4%) and paclitaxel-eluting stents in 26 patients (42.6%). Compared to the BMS group, the DES group had a smaller mean maximum balloon diameter (3.35 mm vs. 3.83 mm, p < 0.001) and a longer stent length per lesion (29.4 mm vs. 20.4 mm, p < 0.001).

Serial quantitative coronary angiographic analysis. Serial quantitative coronary angiographic analyses are shown in Table 3. The mean MLD was 1.01 mm for the DES group and 1.24 mm for the BMS group (p = 0.009). The DES group had a trend toward smaller RVD compared to the BMS group (p = 0.08). Post-procedure RVD and MLD were significantly larger in the BMS group.

Angiographic follow-up was available in 43 patients (71%) (with 50 lesions) in the DES group and 61 patients (69%) (with 86 lesions) in the BMS group. The mean time

Table 1. Baseline Clinical Characteristics

	DES Group (n = 61 Patients)	BMS Group (n = 89 Patients)	p Value
Age, yrs	67 ± 8	67 ± 8	0.85
Male, n (%)	51 (83.6)	79 (88.8)	0.46
Family history of CAD, n (%)	23 (37.7)	24 (27.0)	0.21
Hypercholesterolemia, n (%)	40 (65.6)	44 (49.4)	0.07
Hypertension, n (%)	37 (60.7)	48 (53.9)	0.50
Diabetes mellitus, n (%)	12 (19.7)	14 (15.7)	0.66
Prior MI, n (%)	36 (59.0)	56 (62.9)	0.73
Age of SVG, yrs	9.7 ± 5.6	9.2 ± 4.8	0.58
Unstable angina, n (%)	18 (29.5)	36 (40.4)	0.23
Multivessel coronary disease, n (%)	59 (96.7)	89 (100)	0.32
LVEF, %	50.6 ± 8.1	48.7 ± 10.4	0.24

Values are presented as numbers (%) or mean ± SD.
BMS = bare-metal stent; CAD = coronary artery disease; DES = drug-eluting stent; LVEF = left ventricular ejection fraction; MI = myocardial infarction; SVG = saphenous vein graft.

to angiographic follow-up was not statistically different between the two groups (6.9 ± 3.2 months vs. 6.2 ± 2.1 months, respectively, p = 0.3). Compared to the BMS group, late lumen loss was significantly smaller in the DES group (0.37 ± 0.97 mm vs. 1.09 ± 1.10 mm, p = 0.003). In-segment restenosis occurred less frequently in the DES group (10.0% vs. 26.7%, p = 0.03). There was no statistical difference regarding the incidence of late occlusion during six-month follow-up (6.0% vs. 8.1%, p = 0.90).

In-hospital results and clinical follow-up outcomes. In-hospital results and clinical follow-up outcomes are shown in Table 4. The incidence of in-hospital MACE was similar between the two groups (6.6% vs. 5.6%, p = 1.0). Non–Q-wave MI occurred in four patients (6.6%) of the DES group and in five (5.6%) of the BMS group (p = 1.0). Among those who suffered a non–Q-wave MI in the BMS group, one patient (1.1%) died two days after the procedure.

Six-month clinical follow-up was available in all patients. The cumulative MACE at six months was 11.5% in the DES group and 28.1% in the BMS group (p = 0.02). There were no statistically significant differences in the cumulative incidence of death and MI between the two groups. Compared to the BMS group, the rates of TLR and TVR were significantly lower in the DES group (TLR: 3.3% vs. 19.8%, p = 0.003; TVR: 4.9% vs. 23.1%, p = 0.003, respectively). The rate of MACE-free survival was 88.5% in the DES group and 71.9% in the BMS group (p = 0.03) (Fig. 1).

Cox regression analysis was used to identify independent predictors of MACE at six months follow-up. Variables entered into analysis included: age of patient, age of SVG, diabetes, hypercholesterolemia, unstable angina, left ventricular ejection fraction, usage of distal protection devices, administration of glycoprotein IIb/IIIa inhibitors, duration of dual antiplatelet therapy, restenotic lesions, occluded lesions, stent type, baseline RVD, lesion length, post-procedural MLD, maximal balloon inflation pressure, and stent length. By Cox regression analysis, diabetes (HR: 3.03; 95% CI: 1.33 to 6.90; p = 0.008), usage of BMS (HR: 2.53; 95% CI: 1.07 to 5.97; p = 0.03), and age of SVG

Table 2. Baseline Lesion and Procedural Characteristics

	DES Group	BMS Group	p Value
Lesions characteristics	n = 69	n = 120	
Location of lesion, n (%)			0.46
Ostial	13 (18.8)	18 (15.0)	
Proximal	22 (31.9)	34 (28.3)	
Mid	18 (26.1)	27 (22.5)	
Distal and anastomotic	16 (23.2)	41 (34.2)	
Restenotic lesions	24 (34.8)	8 (6.7)	<0.001
Total occlusion	3 (4.3)	4 (3.3)	0.71
Calcium	6 (8.7)	6 (5.0)	0.36
Thrombus	9 (13.0)	26 (21.7)	0.18
Procedural characteristics	n = 61	n = 89	
Number of stents per lesion, n	1.20 ± 0.61	1.08 ± 0.30	0.050
Mean length of stent per lesion, mm	29.4 ± 19.8	20.4 ± 8.8	<0.001
Maximum balloon diameter, mm	3.35 ± 0.39	3.83 ± 0.58	<0.001
Maximum balloon inflation pressure, atm	17.7 ± 3.9	15.1 ± 3.5	<0.001
No reflow, n (%)	0	1 (1.1)	1.0
Distal protection devices, n (%)	19 (31.1)	20 (22.5)	0.26
Glycoprotein IIb/IIIa inhibitors, n (%)	9 (14.8)	19 (21.3)	0.40

Values are presented as numbers (%) or mean ± SD.
Abbreviations as in Table 1.

Table 3. Serial Quantitative Coronary Angiography Analysis

	DES Group	BMS Group	p Value
Baseline	n = 69 lesions	n = 120 lesions	
RVD, mm	3.06 ± 0.65	3.30 ± 0.96	0.08
MLD, mm	1.01 ± 0.44	1.24 ± 0.59	0.009
Diameter stenosis, %	67.6 ± 14.8	63.9 ± 15.2	0.11
Mean lesion length, mm	14.8 ± 12.7	12.8 ± 8.3	0.20
Post-procedure	n = 69 lesions	n = 120 lesions	
RVD, mm	3.38 ± 0.58	3.67 ± 0.69	0.005
MLD, mm	2.97 ± 0.54	3.22 ± 0.73	0.02
Diameter stenosis, %	11.8 ± 8.5	12.8 ± 9.4	0.45
Acute gain, mm	1.97 ± 0.61	1.97 ± 0.76	0.99
Six-month follow-up, n (%)	n = 50 lesions	n = 86 lesions	
RVD, mm	3.33 ± 0.61	3.32 ± 0.64	1.0
MLD, mm	2.56 ± 0.95	2.03 ± 1.05	0.02
Diameter stenosis, %	17.3 ± 21.4	33.1 ± 29.7	0.02
Mean lesion length, mm	5.55 ± 5.41	5.47 ± 3.30	0.73
Late lumen loss, mm	0.37 ± 0.97	1.09 ± 1.10	0.003
In-segment restenosis rate, n (%)	5 (10.0)	23 (26.7)	0.03
Occlusion at follow-up, n (%)	3 (6.0)	7 (8.1)	0.90

Values are presented as numbers (%) or mean ± SD.
MLD = minimal lumen diameter; RVD = reference vessel diameter. Other abbreviations as in Table 1.

(HR: 1.10; 95% CI: 1.02 to 1.19; p = 0.02) were identified as predictors of MACE during six-month follow-up.

DISCUSSION

The main findings of this report are that utilization of DES for treatment of SVG lesions appears safe and feasible and DES implantation in SVG lesions seems effective in reducing the incidence of restenosis and improves MACE-free survival at six months.

It is estimated that at least 50% of SVG lesions will develop stenosis or occlusion within 10 years of implantation (18). Due to the higher mortality and morbidity associated with repeat bypass surgery, percutaneous revascularization is the preferred approach for treatment of SVG lesions (19). Early results using balloon angioplasty alone to treat SVG lesions were disappointing (20). Although the introduction of stents achieved more predictable results and higher success rates, the incidence of restenosis remained still as high as 37% (3,4).

In comparison with previous studies (3,4), the present report enrolled patients with more challenging lesion characteristics, including 18.8% ostial lesions, 4.3% total occlusion, and 34.8% restenotic lesions. These subgroups are known to be associated with less favorable outcomes

Table 4. In-hospital Results and Clinical Outcomes at Six-Month Follow-up

	DES Group (n = 61 Patients)	BMS Group (n = 89 Patients)	p Value
In-hospital			
Angiographic success, n (%)	60 (98.4)	83 (93.3)	0.24
Procedural success, n (%)	56 (91.8)	80 (89.9)	0.78
In-hospital MACE, n (%)	4 (6.6)	5 (5.6)	1.0
Cardiac death	0	1 (1.1)	1.0
Noncardiac death	0	0	—
Q-wave MI	0	0	—
Non–Q-wave MI	4 (6.6)	5 (5.6)	1.0
TLR	0	0	—
TVR	0	0	—
Stent thrombosis, n (%)	0	0	—
Six-month follow-up			
Cumulative six-month MACE, n (%)	7 (11.5)	25 (28.1)	0.016
Cardiac death	0	2 (2.2)	0.65
Noncardiac death	1 (1.6)	0	0.85
Q-wave MI	0	1 (1.1)	0.85
Non–Q-wave MI	5 (8.2)	7 (7.9)	0.82
TLR	2 (3.3)	18 (19.8)	0.003
TVR	3 (4.9)	21 (23.1)	0.003
Stent thrombosis, n (%)	0	0	—

Values are presented as numbers (%).
MACE = major adverse cardiac events; TLR = target lesion revascularization; TVR = target vessel revascularization. Other abbreviations as in Table 1.

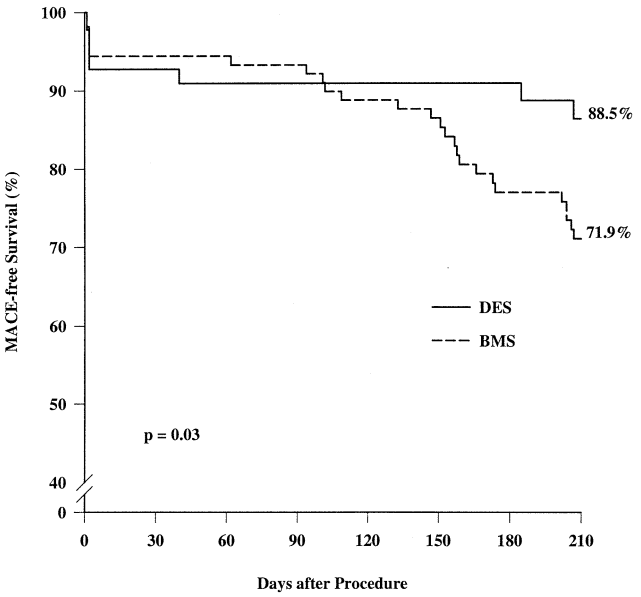

Figure 1. Kaplan-Meier survival curves for freedom from major adverse cardiac events (MACE) at six-month follow-up. BMS = bare-metal stents; DES = drug-eluting stents.

(1,21,22). However, these lesions treated with DES had a significant improvement in late lumen loss (0.37 ± 0.97 mm vs. 1.09 ± 1.10 mm, p = 0.003), resulting in lower in-segment restenosis rate (10.0% vs. 26.7%, p = 0.03). Compared to the studies using DES implanted in de novo native coronary artery lesions, late lumen loss obtained in the present report appears larger (6,7). It is worth noting that three of the five restenotic lesions in the DES group were found to be total occlusions, and this fact may have influenced the quantification of the late loss. Late occlusion is a known problem associated with stenting of SVG lesions. It is reported that late occlusion occurs in 4% to 7% of the cases that received BMS (23,24). However, to date, no data are available about the rate of late occlusion in SVG lesions after DES implantation. It is also unclear whether the pattern of restenosis after DES implantation in SVG lesions is different from the one found in native coronary arteries (25).

Compared to the BMS group, MACE-free survival rates at six months in the DES group were higher (88.5% vs. 71.9%, p = 0.03). By Cox regression analysis, diabetes, usage of BMS, and age of SVG were identified to be the predictors of MACE during six-month follow-up. It has been shown that diabetes is an independent risk factor for worse clinical and angiographic outcomes in native coronary arteries and SVG lesions (26–28). Compared to the stenting in SVG lesions of nondiabetic patients, diabetics have higher TLR and late mortality (27). Marked intimal abnormalities and more rapid progression of the atherosclerotic disease in diabetic subjects may contribute to unfavorable outcomes (29). Graft age was described as one of the risk factors for less favorable results in previous studies (30,31). In the present report, the mean graft age was 9.4 years.

Despite the encouraging findings of this report, we cannot ignore that 30% to 50% of late cardiac events (after one year) in patients with SVG lesions are due to disease progressions at different sites rather than the target one (3,32). This knowledge is important when evaluating the clinical impact of reducing restenosis in SVG lesions after DES implantation. For these reasons the long-term clinical benefit of DES in SVG lesions remains to be determined. Adjunctive therapies (e.g., treatment of the concomitant diseases associated with coronary heart disease, extended antithrombotic therapy) may be important to impact on late events, which may occur independently of restenosis.

Study limitations. The present report has some limitations: 1) it is a retrospective study; 2) the DES group included two different types of DES; 3) not all patients performed angiographic follow-up; and 4) clinical follow-up is limited to seven months. Despite these limitations, this report represents a large cohort of patients treated on SVG by DES implantation with complete clinical follow-up.

Some patients with SVG lesions were treated by BMS between March 2002 and March 2004. This fact may be perceived as a selection bias. If anything, these patients had very focal lesions or lesions located in large SVGs for which no appropriately sized DES were available; this group represents a lower-risk cohort for TLR. Despite the limitations in this study design, a conclusive randomized trial comparing DES to BMS appears progressively less feasible due to ethical difficulties with treating high-risk cohorts with BMS.

Conclusions. Percutaneous revascularization in SVG lesions with DES appears feasible with a high procedural success rate. Compared to BMS implantation, DES implantation in SVG lesions is associated with a reduction in the restenosis rate and a beneficial effect on MACE-free survival at six-month follow-up.

Reprint requests and correspondence: Dr. Antonio Colombo, EMO Centro Cuore Columbus, 48 Via M. Buonarroti, 20145 Milan, Italy. E-mail: info@emocolumbus.it.

REFERENCES

1. de Feyter PJ, van Suylen RJ, de Jaegere PP, Topol EJ, Serruys PW. Balloon angioplasty for the treatment of lesions in saphenous vein bypass grafts. J Am Coll Cardiol 1993;21:1539–49.
2. Morrison DA, Crowley ST, Veerakul G, Barbiere CC, Grover F, Sacks J. Percutaneous transluminal angioplasty of saphenous vein grafts for medically refractory unstable angina. J Am Coll Cardiol 1994;23:1066–70.
3. Savage MP, Douglas JS Jr., Fischman DL, et al. Stent placement compared with balloon angioplasty for obstructed coronary bypass grafts. Saphenous Vein De Novo Trial Investigators. N Engl J Med 1997;337:740–7.
4. Hanekamp CE, Koolen JJ, Den Heijer P, et al. Randomized study to compare balloon angioplasty and elective stent implantation in venous bypass grafts: the Venestent study. Catheter Cardiovasc Interv 2003; 60:452–7.
5. Frimerman A, Rechavia E, Eigler N, Payton MR, Makkar R, Litvack F. Long-term follow-up of a high risk cohort after stent implantation in saphenous vein grafts. J Am Coll Cardiol 1997;30:1277–83.

6. Moses JW, Leon MB, Popma JJ, et al. Sirolimus-eluting stents versus standard stents in patients with stenosis in a native coronary artery. N Engl J Med 2003;349:1315–23.
7. Colombo A, Drzewiecki J, Banning A, et al. Randomized study to assess the effectiveness of slow- and moderate-release polymer-based paclitaxel-eluting stents for coronary artery lesions. Circulation 2003; 108:788–94.
8. 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;346:1773–80.
9. 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;350:221–31.
10. Stone GW, Ellis SG, Cox DA, et al. One-year clinical results with the slow-release, polymer-based, paclitaxel-eluting TAXUS stent: the TAXUS-IV trial. Circulation 2004;109:1942–7.
11. Hoye A, Lemos PA, Arampatzis CA, et al. Effectiveness of the sirolimus-eluting stent in the treatment of saphenous vein graft disease. J Invasive Cardiol 2004;16:230–3.
12. Colombo A, Tobis J. Techniques in Coronary Artery Stenting. London: Martin Dunitz Publishers, 2000.
13. Cutlip DE, Baim DS, Ho KK, et al. Stent thrombosis in the modern era: a pooled analysis of multicenter coronary stent clinical trials. Circulation 2001;103:1967–71.
14. Jeremias A, Sylvia B, Bridges J, et al. Stent thrombosis after successful sirolimus-eluting stent implantation. Circulation 2004;109:1930–2.
15. Lansky AJ, Dangas G, Mehran R, et al. Quantitative angiographic methods for appropriate end-point analysis, edge-effect evaluation, and prediction of recurrent restenosis after coronary brachytherapy with gamma irradiation. J Am Coll Cardiol 2002;39:274–80.
16. Sheehan FH, Braunwald E, Canner P, et al. The effect of intravenous thrombolytic therapy on left ventricular function: a report on tissue-type plasminogen activator and streptokinase from the Thrombolysis In Myocardial Infarction (TIMI phase I) trial. Circulation 1987;75: 817–29.
17. Stankovic G, Colombo A, Presbitero P, et al. Randomized evaluation of polytetrafluoroethylene-covered stent in saphenous vein grafts: the Randomized Evaluation of polytetrafluoroethylene COVERed stent in Saphenous vein grafts (RECOVERS) trial. Circulation 2003;108:37–42.
18. Fitzgibbon GM, Kafka HP, Leach AJ, Keon WJ, Hooper GD, Burton JR. Coronary bypass graft fate and patient outcome: angiographic follow-up of 5,065 grafts related to survival and reoperation in 1,388 patients during 25 years. J Am Coll Cardiol 1996;28:616–26.
19. Morrison DA, Sethi G, Sacks J, et al. Percutaneous coronary intervention versus repeat bypass surgery for patients with medically refractory myocardial ischemia: AWESOME randomized trial and registry experience with post-CABG patients. J Am Coll Cardiol 2002;40:1951–4.
20. Platko WP, Hollman J, Whitlow PL, Franco I. Percutaneous transluminal angioplasty of saphenous vein graft stenosis: long-term follow-up. J Am Coll Cardiol 1989;14:1645–50.
21. Fenton SH, Fischman DL, Savage MP, et al. Long-term angiographic and clinical outcome after implantation of balloon-expandable stents in aortocoronary saphenous vein grafts. Am J Cardiol 1994;74:1187–91.
22. Wong SC, Baim DS, Schatz RA, et al. Immediate results and late outcomes after stent implantation in saphenous vein graft lesions: the multicenter U.S. Palmaz-Schatz stent experience. The Palmaz-Schatz Stent Study Group. J Am Coll Cardiol 1995;26:704–12.
23. Heidland UE, Heintzen MP, Michel CJ, Strauer BE. Risk factors for the development of restenosis following stent implantation of venous bypass grafts. Heart 2001;85:312–7.
24. Schachinger V, Hamm CW, Munzel T, et al. A randomized trial of polytetrafluoroethylene-membrane-covered stents compared with conventional stents in aortocoronary saphenous vein grafts. J Am Coll Cardiol 2003;42:1360–9.
25. Colombo A, Orlic D, Stankovic G, et al. Preliminary observations regarding angiographic pattern of restenosis after rapamycin-eluting stent implantation. Circulation 2003;107:2178–80.
26. Elezi S, Kastrati A, Pache J, et al. Diabetes mellitus and the clinical and angiographic outcome after coronary stent placement. J Am Coll Cardiol 1998;32:1866–73.
27. Ahmed JM, Hong MK, Mehran R, et al. Influence of diabetes mellitus on early and late clinical outcomes in saphenous vein graft stenting. J Am Coll Cardiol 2000;36:1186–93.
28. The Bypass Angioplasty Revascularization Investigation (BARI) Investigators. Comparison of coronary bypass surgery with angioplasty in patients with multivessel disease. N Engl J Med 1996;335:217–25.
29. Lorusso R, Pentiricci S, Raddino R, et al. Influence of type 2 diabetes on functional and structural properties of coronary artery bypass conduits. Diabetes 2003;52:2814–20.
30. Dorogy ME, Highfill WT, Davis RC. Use of angioplasty in the management of complicated perioperative infarction following bypass surgery. Cathet Cardiovasc Diagn 1993;29:279–82.
31. Kahn JK, Rutherford BD, McConahay DR, et al. Early postoperative balloon coronary angioplasty for failed coronary artery bypass grafting. Am J Cardiol 1990;66:943–6.
32. Keeley EC, Velez CA, O'Neill WW, Safian RD. Long-term clinical outcome and predictors of major adverse cardiac events after percutaneous interventions on saphenous vein grafts. J Am Coll Cardiol 2001;38:659–65.