Incidence of stent thrombosis and adverse cardiac events 5 years after sirolimus stent implantation in clinical practice

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Background The long-term incidence of stent thrombosis (ST) and complications after sirolimus-eluting stents (SES) implantation is still a matter of debate.

Method We conducted a systematic follow-up on the day of their 5-year SES implantation anniversary, in a series of consecutive real-world patients treated with a SES. The use of SES implantation was not restricted to "on-label" indications, and target lesions included in-stent restenosis, vein graft, left main stem locations, bifurcations, and long lesions. The Academic Research Consortium criteria were used for ST classification.

Results Three hundred fifty consecutive patients were treated with SES between April and December 2002 in 3 Swiss hospitals. Mean age was 63 ± 6 years, 78% were men, 20% presented with acute coronary syndrome, and 19% were patients with diabetes. Five-year follow-up was obtained in 98% of eligible patients. Stent thrombosis had occurred in 12 patients (3.6%) [definite 6 (1.8%), probable 1 (0.3%) and possible 5 (1.5%)]. Eighty-one percent of the population was free of complications. Major adverse cardiac events occurred in 74 (21%) patients and were as follows: cardiac death 3%, noncardiac death 4%, myocardial infarction 2%, target lesion revascularization 8%, non–target lesion revascularization target vessel revascularization 3%, coronary artery bypass graft 2%. Non-TVR was performed in 8%.

Conclusion Our data confirm the good long-term outcome of patients treated with SES. The incidence of complications and sub acute thrombosis at 5 years in routine clinical practice reproduces the results of prospective randomized trials. (Am Heart J 2009;157:883-8.)

The use of stents has significantly improved the outcome of percutaneous coronary interventions (PCI).[1] However, despite major advances in angioplasty and stenting, in-stent restenosis and stent thrombosis (ST) have remained major limitations. In 2002, sirolimus-eluting stents (SES) have emerged as a very promising approach in preventing restenosis and have been shown to have a major beneficial impact on both the angiographic and the clinical outcome.[2,3] However, even more than with bare metal stents (BMSs), ST represents a clinical challenge after SES implantation. Because some have suspected that ST is more frequent after SES placement,[4] obtaining long-term follow-up data is of critical importance. Longer-term follow-up is still very scarce. We report here the complete 5-year follow-up of our first 350 consecutive patients treated with SES just after the introduction on the Swiss market to determine the incidence of major adverse cardiac events (MACE) and ST in routine clinical practice.

Methods

Between March 2002 and December 2002, all consecutive patients with SES implantation (Cypher, Cordis, Miami Lakes, FL) were included in a prospective registry. During the same period 625 additional patients underwent percutaneous revascularization. The institutional review boards of the 3 participating institutions approved the study. Eligible patients provided written informed consent. The study complied with the Declaration of Helsinki regarding investigations in humans. Some patients were included in the e-Cypher registry. It is an Internet-based, postmarketing surveillance registry with the primary objective to assess the safety of the Cypher coronary SES in daily clinical practice. The database was tested and validated as previously described.[5] The other patients were included in the present registry upon the decision of the operator to implant a DES instead of a BMS. Procedures were performed via the femoral or the radial artery. A 5F, 6F, or 7F guiding catheter was used. All patients were pretreated with aspirin 100 mg/day. Intravenous heparin (70 U/kg) was given

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Table I. Demographic data (n = 350)

Men	274 (78%)
Mean age	63 ± 6 y
Stable angina pectoris	280 (80%)
ACS	70 (20%)
Diabetes	67 (19%)
Family history	174 (50%)
Hypertension	217 (62%)
Hyperlipidemia	270 (77%)
Smokers	
Past	98 (28%)
Current	77 (22%)
Previous MI	89 (25%)
Previous CABG	52 (15%)
Previous PCI	98 (28%)
Multivessel disease	168 (48%)
Stable angina pectoris	280 (80%)

Table II. Angiographic data

Treated vessel:	
LAD	195 (55%)
RCA	78 (22%)
Circumflex	69 (20%)
Left main	9 (3%)
SVG	15 (4%)
Reference vessel diameter (mm)	2.8 ± 0.6
MLD (before stenting) (mm)	0.62 ± 0.12
MLD (post-stenting) (mm)	2.75 ± 0.8
No. of stents per patient	1.4 ± 0.2
Lesion length	18 ± 6
Stented length (mm)	21 ± 6
Multivessel stenting	37 (11%)
In-stent restenosis	38 (11%)
Bifurcation	49 (14%)

LAD, Left anterior descending coronary artery; *RCA*, right coronary artery; *SVG*, saphenous vein graft; *MLD*, minimal lumen diameter.

Table III. Incidence of MACE at 5 years (n = 344) (patients can appear more than once)

Death	26 (8%)
Cardiac	10 (3%)
Noncardiac	16 (5%)
MI	6 (2%)
TLR	26 (8%)
TVR	9 (3%)
CABG	6 (2%)
ST	12 (3.6%)
Definite	6 (1.8%)
Probable	1 (0.3%)
Possible	5 (1.5%)
Event-free	277 (81%)

at the beginning, and a 300 or 600 mg loading dose of clopidogrel was administered either before or immediately at the end of the procedure. Standard interventional techniques were used to treat the patients and performed at the discretion of the operator. Intravascular ultrasound (IVUS) was not used. When predilatation was used, balloon diameter and length were both generally smaller than the stent dimensions. A successful procedure was defined as a residual stenosis <20% and the absence of major adverse cardiac event during the inhospital stay. Bifurcation lesion were treated with provisional stenting only.

After the procedure, patients were usually monitored in an intermediate care unit, and CK, creatine kinase–MB (CK-MB), and troponin values were measured at least once 8 to 24 hours after the procedure. A 12-lead electrocardiogram was recorded at the end of the procedure for all patients, and further tracings were obtained if indicated by the clinical course. Patients were discharged the same day or on the day after the procedure. Aspirin 100 mg/d was given long term and clopidogrel 75 mg/d was prescribed for 3 to 12 months. Patients who received 12 months were those with "off-label" indication, acute coronary syndrome (ACS), and patients with diabetes.

Quantitative coronary angiography evaluation was obtained in multiple views. Analyses were performed after administration of intracoronary nitrates before, during, and after the procedure. For patients with angiographic follow-up, restenosis was defined as a ≥50% reduction of the luminal diameter occurring within the 5 proximal and distal segment millimeters of the stented segment. The use of SES implantation was not restricted to "on-label" indications, and target lesions included in-stent restenosis, lesions in saphenous vein grafts or the left main stem, chronic total occlusions, bifurcations, and long lesions. Twenty-five percent (88/350) of the patients were treated for ≥1 off-label indication defined as follows: acute myocardial infarction, left ventricular ejection fraction <35%, in-stent restenosis, left main stenosis, chronic total occlusion, saphenous vein graft, and in-stent restenosis. The cohort of patients included in the trial did not differ from the cohort of patients treated with BMSs. A bias of selection can be therefore excluded.

Clinical follow-up was obtained by a visit or by telephone contact with the patient or his/her referring physician after 6 months, 1 year, and on the day of the 5-year procedural anniversary or shortly after. Angiographic follow-up was performed only in patients with clinical indication (silent or symptomatic ischemia).

The primary end point was ST. Secondary end points included a composite of MACE, defined as death, myocardial infarction, or target vessel revascularization (TVR) (target lesion revascularization [TLR] and non-TLR).

Target vessel revascularization was defined as a repeat revascularization based on a stenosis within the stent or within the 5-mm borders proximal or distal to the stent. Target vessel revascularization was defined as any other revascularization in the stented vessel. Target vessel revascularization was considered if the stenosis of the target lesion or vessel was >50% based on angiography in the presence of ischemic signs or symptoms or if there was a stenosis of >70% in the absence of ischemic signs or symptoms.

The diagnosis of myocardial infarction (MI) after the intervention was established whenever new Q waves of at least 0.4 seconds in duration in at least 2 contiguous leads appeared on the electrocardiogram with an elevated CK-MB fraction level or, in the absence of pathologic Q waves, an elevation in total creatine kinase levels to more than twice the upper limit of normal with an elevated CK-MB or troponin I level. Stent thrombosis was classified according to the Academic Research Consortium criteria.[6]

Table IV. Characteristics of patients with ST

Patient	Age	Indication for stenting	Vessel treated	Stent size and length (mm)	Delay after stenting (d)	Symptoms	Treatment	Outcome	APT
1	36	De novo stenosis	LAD	3.0/18	1770	MI	PCI	Favorable	Aspirin
2	56	De novo stenosis	Marginal	2.5/18	1080	Sudden death	none	Death	Aspirin
3	83	In-stent restenosis	Circumflex	2.5/18	1202	Sudden death	none	Death	Aspirin
4	59	De novo stenosis	Marginal	2.75/18	751	Sudden death	none	Death	Aspirin
5	57	De novo stenosis	RCA	3.0/33	1349	MI	PCI	Favorable	Aspirin, stop clopidogrel 10 d before
6	57	De novo stenosis	LAD	3.0/18	1048	Sudden death	none	Death	Aspirin
7	68	In-stent restenosis	LAD	3.0/33	813	MI	PCI	Death	Aspirin + clopidogrel
8	75	De novo stenosis	Diagonal	2.5/13	725	MI	PCI	Death	Aspirin
9	72	In-stent restenosis	LAD	3.0/18	348	MI	none	Death	None
10	62	De novo stenosis	LAD	2.5/18	570	Angina	CABG	CABG	Aspirin
11	54	In-stent restenosis	SVG	3.0/18	693	MI	PCI	Favorable	Aspirin
12	63	De novo stenosis	RCA	3.0/18	1168	ACS	PCI	Favorable	Aspirin

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Statistical analysis

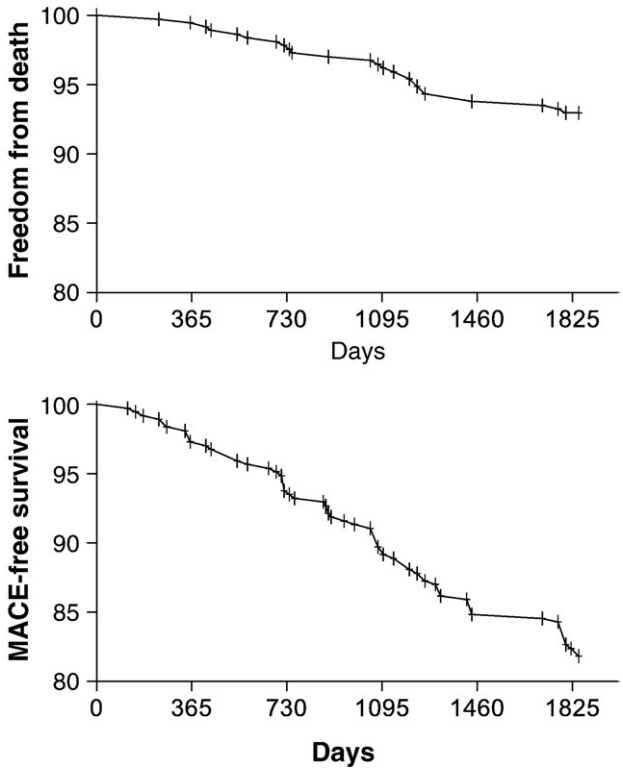
Values are expressed as mean ± SD. The Fischer exact test was used for statistical analysis. A 2-tailed *P* value <.05 was considered to be of statistical significance. The cumulative incidence of adverse events was estimated according to the Kaplan-Meier method, and curves were compared using the log-rank test. Patients lost to follow-up were considered at risk until the date of last contact, at which point they were censored.

Results

Three hundred fifty consecutive patients were included in the registry. Demographic data are shown in Table I. Mean age was 63 ± 6 years, 78% were men, 20% presented with ACS, and 19% were patients with diabetes. Angiographic data are shown in Table II. Multivessel procedures were performed in 11% of patients even if 48% had multivessel disease because only the culprit lesion was treated. The mean lesion length was 18 ± 6 mm, the mean total stent length was 21 ± 5 mm, and the vessel reference diameter 2.8 ± 0.6 mm (Table III). Mean number of SES per patient was 1.4 ± 0.2. On the day of their 5-year procedure anniversary or shortly thereafter, 318 (98%) of 324 surviving patients were contacted by telephone or during a clinical visit. Stent thrombosis had occurred in 12 patients (3.6%) (definite 6 [1.8%], probable 1 [0.3%] and possible 5 [1.5%]), and the annual rate of definite and probable ST was 0.4% a year. Characteristics of patients with ST are shown in Table IV. The status of antiplatelet therapy at the time of ST was available for 11 of these 12 patients. The time course of MACE and ST is shown in Figures 1 and 2. Surprisingly, most of ST were late ST, and only 1 (8% of all ST) occurred during the first year of follow-up. This patient had stopped clopidogrel 1 week before ST. Another patient with lifetime prescription of clopidogrel stopped this medication for elective abdominal surgery 4 years after stent implantation. He experienced a very late ST a few days after surgery and had nonfatal myocardial infarction. The 6 other patients were on aspirin 100 mg/d when they had ST. Overall mortality of ST was 58% (7/12). It was 28% (2/7) when only definite and probable ST were considered. One patient experienced a second ST after reperfusion of the occluded stent even treated with dual antiplatelet agent and died.

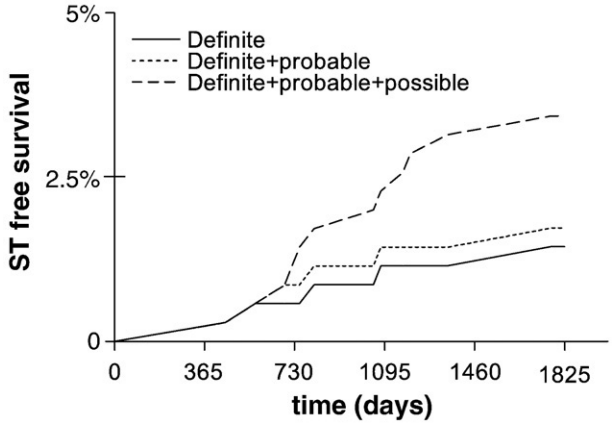
Eighty-one percent of the population was free of complications (Figure 2). Major adverse cardiac events were as follows: cardiac death 3%, noncardiac death 4%, MI 2%, TLR 8%, non-TLR TVR 3%, and coronary artery bypass graft (CABG) 2%. In 8%, non-TVR revascularization was performed. All additional revascularization

Figure 1



Survival curve and MACE free-survival curves at 5 years.

Figure 2



Stent thrombosis survival curve at 5 years.

procedures (TLR, TVR, and non-TVR) were clinically driven. Target vessel revascularization was more frequent during the first year of follow-up, but a significant number of them are late TLR as shown in Table V. Most of TLR were needed because of focal in-stent restenosis. No particular pattern was noted.

Table V. Time course of TLR

0-1 y	9 (35%)
1-2 y	5 (19%)
2-3 y	5 (19%)
3-4 y	3 (12%)
4-5 y	4 (15%)

Table VI. Incidence of ST in previously registries and trials compared with our data

	Definite and probable ST	Follow-up duration	Stent type
Urban et al[5] (e-Cypher)	0.87%	12 m	SES
Flores-Rios[15]	2.8%	34 m	PES
Daemen et al[16]	2.7%	3 y	SES
ESTROFA registry[17]	2%	3 y	SES/PES
Stone et al[18]	1.2%	4 y	SES
Mauri et al[19]	1.5%	4 y	
Daemen et al (RESEARCH) registry[20]	2.3%	4 y	SES
Stettler et al[21]	1.9%	4 y	SES
Galloe et al[32]	2.5%		SES
Our data	2.2%	5 y	SES

PES, Paclitaxel-eluting stent.

Discussion

In this report, we described long-term clinical outcome after implantation of SES in routine clinical practice. Our report mainly addresses the 5-year incidence of MACE and ST. Our data provide the longest follow-up reported for patients treated with SES for various and often complex lesions outside the contain of a randomized controlled trial. Our data are consistent with those of previous clinical trials even in most of the latter have reported shorter follow-up periods. The 4 main early randomized SES trials (RAVEL, SIRIUS, C-SIRIUS, and E-SIRIUS) found no significant differences in 4-year rates of mortality or ST between SES and BMS,[7-10] and read-judicated ST rates according to the Academic Research Consortium definitions were 0.7% and 0.4% when only definite and probable ST were considered. Freedom from MACE in the SES group was 88 and 89% at 4 years and therefore higher than in our cohort of patients but in a very selected group of randomized patients. It is therefore likely that different baseline clinical and angiographic characteristics explain the less favorable clinical outcome of our patients, even with a very similar rate of ST. Our MACE rates at 5 years (21%) are closer to those observed in the Swedish registry[11] and in the RESEARCH registry[12] which reported 23% of MACE at 4 years. Anstrom et al[13] recently reported the results from the Duke registry with an incidence of MACE of 16%, but follow-up did not

exceed 2 years. The same is true for the report of Kelbaeck et al[14] and Planer et al[15] who reported a 3-year incidence of MACE of, respectively, 12% and 16% in a randomized trial of patients with complex lesions and from the Israeli arm of the e-Cypher registry. These numbers are very similar to our incidence of MACE at 2 and 3 years of follow-up. As shown by our data, some TLRs occurred very late after stenting and explain a small but constant progression of adverse events.

Our 5-year TVR rate is also comparable with the one reported in the RESEARCH registry (8.2%). The 2-year adjusted TVR rates in the community practice reported by Anstrom et al,[13] at 6.6% was less than the one reported for the RAVEL trial (7.7%) and than ours but limited to 2 years of follow-up. These are small differences at least partially, explained by the fact that in most randomized clinical trials and even in some registries, systematic repeat angiography is performed. That has been shown to increase the frequency of repeated TVR procedures even in patients without clinical symptoms. In contrast, restenosis in clinical practice is typically detected when symptoms prompt repeat angiography. Another explanation is the late occurrence of TLR as shown by our data (up to 5 years after stenting). This may indicate that at least in some patients a catch-up phenomenon exist after SES implantation. The rate of definite and probable SES thrombosis that we observed (2.1%) as well the rate of ST reported in several registries or trials (Table V) for follow-up periods of up to 4 years is higher than in a meta-analyses of the randomized trials (1.2% at 2 years using the Academic Research Consortium definitions for definite and probable ST).[16-22] In the ESTROFA registry, no difference was found between stent types, and the incidence of ST at 3 years was 2%. In a large 2-institution registry,[21] the incidence of ST remain constant at 0.6% per year over a follow-up of 3 years, with a total incidence of 2.5%—again very comparable with our data with a yearly incidence of 0.4% per year. The e-Cypher registry[5] reported a cumulative ST incidence of 0.87%, but follow-up was limited to 12 months (Table VI). Higher incidence 1.1% per year have been reported with paclitaxel-eluting stents with a cumulative incidence of 2.8% at 34 months,[17] with most of ST occurring late after stent implantation. Our results also underscore the persistent occurrence of late ST during follow-up as shown by the Kaplan-Meier curve of cumulative hazard and are in keeping with the recently published study by Daemen et al[21] and Stettler et al[22] with a follow-up up to 4 years. This confirms that the risk persists over the time, at least for some patients. Clinical and angiographic features may explain the differences in the incidence of early ST and particularly late ST. Furthermore, our study emphasizes the clinical importance of late ST in routine cardiologic practice, given not only its frequency and constant presentation but also the high risk of overall and cardiac death[23,24] and nonfatal MI related to ST.

Several factors have previously been identified to predict ST.[23] Withdrawal of antiplatelet drug therapy was an independent predictor of ST in some studies.[25-27] In our series, most ST presented beyond the first year after stent placement (90%) and therefore occurred well after the cessation of dual antiplatelet treatment. Stent thrombosis was sometimes related to aspirin or clopidogrel withdrawal, underlining the importance of adherence to long-term antiplatelet monotherapy, perhaps especially when the patient undergoes a major surgical procedure.[28] However, whether prolonging dual antiplatelet therapy could have prevented the events that were not related to antiplatelet monotherapy, withdrawal is an unanswered question to date, and the optimal duration and intensity of antiplatelet therapy after coronary stent implantation remains an unresolved issue. Recently, a new antiplatelet drug, prasugrel, has been shown to reduce the rate of ST during the first year after PCI for ACSs in comparison with clopidogrel,[29-31] but its potential longer-term benefits have not been evaluated yet. Finally, the absence of early ST (during the first month after implantation) is surprising. As angiographic control was not systematic, some ST could have been underestimated as shown in the SORT OUT trial,[32] but our low rate of early ST might also be related to the size of our cohort of patients. Indeed, in 508 patients in the RESEARCH registry, they report only 2 cases of early ST.[12]

Limitations

The major weakness of this study is that it concerns a relatively small number of patients so that the CIs for the MACE and ST rates remain comparatively wide. In addition, no fixed duration of dual antiplatelet therapy was used, and patients received 3 to 12 months of aspirin + clopidogrel, at the discretion of the treating physician. This may have influenced our results, particularly during the first 12 months. It is unlikely to have impacted on most of the observed ST events; however, because they largely occurred beyond the first year.

In conclusion, confirming what was already known for larger series of patients followed up to 4 years, the present data suggest that the safety of SES is acceptable for up to 5 years in real-world patients, despite having included a large number of moderate- to high-risk patients. Even if a catch-up phenomenon cannot be definitively ruled out, efficacy of SES was excellent with a low rate of TLR (8%).

Disclosures

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