Introduction and objectives. Diabetics are at an increased risk of restenosis and adverse events after coronary stenting. Drug-eluting stents may, therefore, be useful in these patients. Our objective was to evaluate the use of sirolimus-eluting stents in diabetics with complex coronary lesions.

Patients and method. Between May 2002 and August 2003, we treated 231 patients with 260 complex coronary lesions using sirolimus-eluting stents. Of these patients, 56% did not have diabetes (ND), 22% had non-insulin-dependent diabetes (NIRD), and 20% had insulin-dependent diabetes (IRD). The primary clinical endpoint was target vessel failure at 1 year. The primary angiographic endpoints in the stent were late loss and binary restenosis at 6 months.

Results. At 6 months, late loss was greater in the IRD group (0.35 [0.71] mm) than in the ND group (0.096 [0.54] mm; P = 0.016) or the NIRD group (0.058 [0.52] mm; P = 0.017), and restenosis was more frequent (IRD, 16.3%; ND, 6.3%; and NIRD 7.8%; P = 0.05 for linear trend). At 1 year, target vessel failure occurred more frequently in the IRD group (IRD, 17.4%; ND, 6.3%; and NIRD 7.7%; P = 0.07 for linear trend) and the rate of survival free of target vessel failure was lower in the IRD group (82.1%) compared with the ND group (92.3%, P = 0.06) or the NIRD group (92.3%, P = NS). The only independent predictor of restenosis and target vessel failure was female sex.

Conclusions. Despite IRD patients having greater late lumen loss and more frequent restenosis at six months and a trend towards a poorer clinical outcome at 1 year, no independent relationship was found between type of diabetes and clinical outcome.

Key words: Diabetes. Drug-eluting stent. Angioplasty.
Drug-eluting stents (DES) have been shown to have a considerably lower risk of restenosis\(^6\) and as a result there is growing interest in using such stents to treat coronary lesions in complex scenarios. Substudies carried out in diabetic patients from large clinical trials conducted with DES have found considerable decreases in the risk of restenosis and new revascularizations.\(^5,7\) Nevertheless, despite the availability use of DES, diabetic patients show a higher risk than nondiabetics.\(^8\,9\) Hypertension and insulin resistance are implicated in a variety of molecular mechanisms that could predispose diabetics to a higher incidence of restenosis.\(^10,11\) In addition, the angiographic prognosis after the placement of sirolimus-eluting stents (SES) could be influenced by the type of diabetes therapy.\(^1\)

Certain anatomic variables in native vessels are associated with an increased risk of restenosis a high risk of restenosis and raise issues on the best treatment strategy, particularly when the patient presents several risk factors for restenosis risk factors for restenosis at the same time, has multivessel disease, or is diabetic. The purpose of the present study is to analyze the impact of diabetes and the type of treatment on angiographic restenosis and the clinical prognosis in patients with complex coronary anatomies treated with SES.

**PATIENTS AND METHODS**

**Study Population**

Between May 2002 and August 2003, 1379 percutaneous revascularization procedures were performed at our hospital. Among these patients, 231 with a total of 260 complex coronary lesions and with proven ischemia were treated with the SES implant (Cypher\(^7\), Cordis, Johnson & Johnson Company) and were included in a prospective registry.

Coronary lesions with at least one of the following characteristics were included:

1. Significant stenosis of the left main.
2. Bifurcations.
3. Long lesions (>18 mm).
5. Stenosis of the proximal segment of the left anterior descending artery (LAD).
6. Restenotic lesions, particularly in-stent restenosis.
7. Total occlusions.
8. Ostial lesions.
9. Stenosis in small vessels (<2.75 mm) together with any of the above conditions.

**Procedure**

All patients received aspirin and clopidogrel. A 300-mg loading dose of clopidogrel was always administered before angioplasty to patients who were not receiving it previously. Heparin was administered at doses of 100 UI/kg, or 70 UI/kg if the patient was receiving glycoprotein IIb-IIIa inhibitors. Use during the procedure was left to the discretion of the catheterization specialist. The implant procedure was done according to the usual practice of our interventional cardiology unit.\(^12\) Periprocedure infarction was defined according to 3 criteria: 1) appearance of new Q waves after the procedure; 2) creatine kinase elevation; or 3) at least 2-fold troponin-T elevation in 2 samples drawn immediately before angioplasty and 12-18 h afterwards.

**Follow-Up**

At the time of discharge, patients received aspirin and clopidogrel, 75 mg/day, for 6 months. Angiographic follow-up at month 6 was proposed to all patients, who were informed of the procedure and its objectives, and informed consent was obtained from all patients who were reevaluated. Patients were contacted at month 1, 6, and 12 following the procedure.

**Angiographic Analysis**

The quantitative analysis was done using the MEDIS\(^9\) system, version 5.2 (Leiden, Netherlands), which includes the Drug Eluting Stent Analysis-QCA package and allows analysis by stent segment and by proximal and distal margins. The analysis was done by an observer from our group who was blinded to the patients’ clinical data.

**Definitions and Objectives of Analysis**

The angiographic endpoints were in-stent late loss and angiographic in-segment binary restenosis at 6 months. Restenosis was considered to exist when the angiographic stenosis in the stent or at its proximal and/or distal margins was >50%.

All deaths were recorded and an attempt was made to identify the causes by contacting the family or the attending physician. Cardiac deaths were considered to be sudden deaths, deaths due to acute myocardial infarction (AMI), or deaths due to heart failure.

Acute myocardial infarction in the follow-up was defined as simultaneous presence of prolonged precordial pain and the onset of new Q waves or significant elevations.
of creatine kinase or troponin. The clinicians in charge were asked to refer patients from the registry to undergo new catheterization if they presented new events during the follow-up period. The purpose was to determine whether the event was related to the target vessel and, if so, to identify the mechanism (restenosis, stent thrombosis, disease progression). Stent thrombosis was considered to be certain when visualized on angiography and probable when an event could be produced by a thrombus, but there was no angiographic confirmation.

The primary endpoint for the clinical analysis was target vessel failure (TVF), defined as the occurrence of any of the following events on follow-up: cardiac death, myocardial infarction related to the target vessel, and revascularization of the target vessel. With this endpoint we intended to analyze the incidence of adverse events related to the clinical decision to treat the culprit lesion with SES. Other secondary endpoints for clinical analysis were target lesion revascularization and the incidence of AMI in other sites. In some cases with angiographic restenosis at 6 months, new revascularization could be performed; this could result in an overestimation of the true incidence of revascularization of the target lesion, and therefore we recommended new revascularization only in cases of symptomatic restenosis or extremely critical restenosis in which the onset of symptoms may be expected shortly after angiography.

Study Design and Statistical Analysis

The present study represents an a post-hoc analysis of the clinical and angiographic prognosis of a cohort of patients with complex coronary lesions treated with SES according to the treatment modality for diabetes. The study group was divided in 3 subgroups: nondiabetic patients (ND, 133 patients), non-insulin-dependent diabetes mellitus patients (NIDDM, 52 patients), and insulin-dependent diabetes mellitus patients (IDDM, 46 patients). The NIDDM group included patients with type 2 diabetes who were receiving dietary management (15 patients) or oral antidiabetic therapy (37 patients) at the time of inclusion. The IDDM group included patients with diabetes who required insulin prior to inclusion; most were long-term type 2 diabetics who were receiving insulin because oral antidiabetic therapy had failed. The continuous variables are expressed as mean ± standard deviation and the qualitative variables as percentages. The continuous variables were compared by ANOVA; in the case of multiple comparisons, the Bonferroni test was used. Clinical and angiographic qualitative variables were compared by the chi-square test. The incidence of restenosis and adverse events in the 3 subgroups was compared using the linear-by-linear association test; TVF-free survival was analyzed by Kaplan-Meier. In patients who had 2 or more events of the combined endpoint, the time to first event was considered. Survival curves were compared using the log-rank test.

Since the study was not randomized, we expected considerable differences in the baseline characteristics of the 3 groups, which would reflect the differences in clinical and angiographic profile of these patients. To analyze the relationship between diabetes and in-stent late loss, we performed stepwise multiple regression analysis between diabetes and restenosis with logistic regression analysis and between diabetes and TVF with Cox regression analysis. These models were planned to include diabetes type, as well as any confounding variables that showed differences in distribution according to group. The SPSS 11.0 software was used (Chicago, Illinois) for the statistical analysis and significance was set at a P value of less than .05, or less than .016 in the case of multiple comparisons.

RESULTS

Baseline Clinical, Angiographic, and Procedure Characteristics

The patients’ clinical characteristics are shown in Table 1 and the quantitative coronary analysis in Table 2. The qualitative angiographic variables (Table 3) showed higher prevalences of proximal LAD involvement in ND, long lesions in IDDM, and type B2 or C lesions in diabetics. The procedure characteristics were homogenous in the 3 groups (Table 4), observing a nonsignificant trend for IDDM patients to require longer stent lengths and greater need for stent overlap. IIb-IIIa inhibitors as antiplatelet agents were used more often in IDDM. There were no differences in the incidence of periprocedure infarction between the 3 groups. Two samples were obtained for the enzymatic determinations in 107 (80.4%) ND patients, 38 (73.1%) NIDDM, and 37 (80.4%) IDDM (P=NS).

Angiographic Follow-Up at 6 Months

After 180±12 days of follow-up, 202 patients (87%) with 230 lesions (88%) were assessed by angiography.

Late Loss

In-stent late loss showed no significant difference between diabetics (0.20±0.63) and nondiabetics (0.09±0.54; P=NS), although it was significantly higher in IDDM (0.35±0.71 mm) compared to ND (0.09±0.54; P=0.016) and NIDDM (0.05±0.52; P=0.017). In the univariate analysis, late loss was also related to female sex (male, 0.09±0.51 mm, vs female, 0.30±0.74 mm; P=0.016), active smoker (no, 0.18±0.60 mm, vs yes, -0.01±0.44 mm; P=0.048), and renal failure.
The only independent determining factor of late loss was IDDM ($R^2=0.04$, $B=0.28$, 95% confidence interval [CI], 0.10-0.46, $P<0.003$).

**Binary Restenosis**

No cases of edge restenosis were found and there were no significant differences in the incidence of restenosis between diabetics (10.8%) and nondiabetics (6.5%; $P=NS$). Figure 1 presents the incidence of angiographic restenosis according to type of diabetes, showing a significant trend toward greater restenosis with increasing complexity of diabetes ($P=0.05$). In the univariate analysis, diabetes in general was not significantly associated with restenosis (hazard ratio [HR]=1.73; 95% CI, 0.67-4.49; $P=NS$), but IDDM did show a significant association (HR=2.89; 95% CI,
1.09-7.65; \( P = .02 \)), as did the variables female sex (HR=3.65; 95% CI, 1.40-9.49; \( P = .005 \)), depressed ejection fraction (HR=3.00; 95% CI, 1.13-7.98; \( P = .022 \)) and hypertension (HR=2.71; 95% CI, 0.87-8.47; \( P = .07 \)). In the multivariate analysis, only female sex was an independent predictor of restenosis (odds ratio \([OR]\)=3.42; 95% CI, 1.31-8.94; \( P = .012 \)).

**Clinical Progress at 1 Year**

Information on clinical progress was obtained at 1 year for 99.1% of the patients. Table 5 shows adverse clinical events after a mean follow-up of 357±83 days. The primary endpoint of TVF was not significantly higher in diabetics (12.2%) than nondiabetics (7.7%; \( P = .09 \)). Nevertheless, when considering the type of treatment, the incidence of TVF among IDDM patients (17.4%) was higher than in ND (7.7%) or NIDDM (7.7%) patients, although at the limit of statistical significance (\( P = .07 \)).

Six patients (2.6%) died, 3 of them due to sudden death at home and 2 due to heart failure, 1 after an infarction with ST segment elevation due to probable Berenguer A, et al. *Efficacy of Sirolimus-Eluting Stents in Diabetics With Complex Coronary Lesions*  

**TABLE 4. Procedure Characteristics in the 3 Study Groups**

<table>
<thead>
<tr>
<th></th>
<th>ND (n=133)</th>
<th>NIDDM (n=52)</th>
<th>IDDM (n=46)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total stent length, mean( \pm )SD, mm</td>
<td>25±12</td>
<td>25±11</td>
<td>28±13</td>
<td>NS</td>
</tr>
<tr>
<td>Number of lesions treated, mean( \pm )SD</td>
<td>1.11±0.32</td>
<td>1.07±0.27</td>
<td>1.24±0.60</td>
<td>NS</td>
</tr>
<tr>
<td>Number of stents per patient, mean( \pm )SD</td>
<td>1.23±0.44</td>
<td>1.34±0.54</td>
<td>1.31±0.47</td>
<td>NS</td>
</tr>
<tr>
<td>Length, mean( \pm )SD, mm</td>
<td>2.91±0.33</td>
<td>2.88±0.25</td>
<td>2.84±0.23</td>
<td>NS</td>
</tr>
<tr>
<td>Overlap</td>
<td>32 (21.8%)</td>
<td>11 (19.6%)</td>
<td>15 (25.3%)</td>
<td>NS</td>
</tr>
<tr>
<td>Direct stent</td>
<td>7 (4.9%)</td>
<td>4 (7.1%)</td>
<td>3 (5.4%)</td>
<td>NS</td>
</tr>
<tr>
<td>Rotablator</td>
<td>4 (2.8%)</td>
<td>1 (1.8%)</td>
<td>4 (7.1%)</td>
<td>NS</td>
</tr>
<tr>
<td>Post-dilation</td>
<td>40 (30.8%)</td>
<td>10 (21.7%)</td>
<td>16 (30.8%)</td>
<td>NS</td>
</tr>
<tr>
<td>Implant pressure, mean( \pm )SD, atm</td>
<td>16±13</td>
<td>17±2</td>
<td>16±2</td>
<td>NS</td>
</tr>
<tr>
<td>Ilb-lla inhibitors</td>
<td>39/113 (30.2%)</td>
<td>19/52 (37.3%)</td>
<td>23/46 (50%)</td>
<td>.02</td>
</tr>
</tbody>
</table>

**Clinical Progress at 1 Year**

Information on clinical progress was obtained at 1 year for 99.1% of the patients. Table 5 shows adverse clinical events after a mean follow-up of 357±83 days. The primary endpoint of TVF was not significantly higher in diabetics (12.2%) than nondiabetics (7.7%; \( P = .09 \)). Nevertheless, when considering the type of treatment, the incidence of TVF among IDDM patients (17.4%) was higher than in ND (7.7%) or NIDDM (7.7%) patients, although at the limit of statistical significance (\( P = .07 \)).

Six patients (2.6%) died, 3 of them due to sudden death at home and 2 due to heart failure, 1 after an infarction with ST segment elevation due to probable

![Figure 1. Incidence of binary restenosis in nondiabetic (ND), non-insulin-dependent diabetes mellitus (NIDDM), and insulin-dependent diabetes mellitus (IDDM) patients. An increased incidence of restenosis was observed with increasing complexity of diabetes.](image)

**TABLE 5. Clinical Follow-Up at 1 year. Incidence of Adverse Clinical Events**

<table>
<thead>
<tr>
<th>Event</th>
<th>Total (n=230)</th>
<th>ND (n=133)</th>
<th>NIDDM (n=52)</th>
<th>IDDM (n=46)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total deaths</td>
<td>6 (2.6%)</td>
<td>2 (1.6%)</td>
<td>1 (1.9%)</td>
<td>3 (6.5%)</td>
<td>.09</td>
</tr>
<tr>
<td>Cardiac death</td>
<td>5 (2.1%)</td>
<td>2 (1.6%)</td>
<td>1 (1.9%)</td>
<td>2 (4.4%)</td>
<td>NS</td>
</tr>
<tr>
<td>TLR</td>
<td>11 (4.7%)</td>
<td>5 (3.8%)</td>
<td>3 (5.8%)</td>
<td>3 (6.5%)</td>
<td>NS</td>
</tr>
<tr>
<td>AMI, target vessel</td>
<td>8 (3.4%)</td>
<td>4 (3.1%)</td>
<td>1 (1.9%)</td>
<td>3 (6.5%)</td>
<td>NS</td>
</tr>
<tr>
<td>AMI, another vessel</td>
<td>1 (0.4%)</td>
<td>1 (0.4%)</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
<td>NS</td>
</tr>
<tr>
<td>TVF</td>
<td>22 (9.4%)</td>
<td>10 (7.7%)</td>
<td>4 (7.7%)</td>
<td>8 (17.4%)</td>
<td>.07</td>
</tr>
</tbody>
</table>

**IDDM** indicates insulin-dependent diabetes mellitus; **NIDDM** non-insulin-dependent diabetes; **AMI** acute myocardial infarction; **ND** non-diabetic; **NS** nonsignificant.

*Relative variable for the 147 lesions in ND, 46 in NIDDM, and 57 in IDDM patients.

1.09-7.65; \( P = .02 \)), as did the variables female sex (HR=3.65; 95% CI, 1.40-9.49; \( P = .005 \)), depressed ejection fraction (HR=3.00; 95% CI, 1.13-7.98; \( P = .022 \)) and hypertension (HR=2.71; 95% CI, 0.87-8.47; \( P = .07 \)). In the multivariate analysis, only female sex was an independent predictor of restenosis (odds ratio \([OR]\)=3.42; 95% CI, 1.31-8.94; \( P = .012 \)).

*SD* indicates standard deviation; **IDDM**, insulin-dependent diabetes mellitus; **NIDDM**, non-insulin-dependent diabetes; **AMI**, acute myocardial infarction; **ND**, non-diabetic; **NS**, nonsignificant.

*Relative variable for the 147 lesions in ND, 46 in NIDDM, and 57 in IDDM patients.*
In the univariate analysis, diabetes in general was not independently related to restenosis or prognosis. In the multivariate analysis, only female sex was independently related with a poorer prognosis (HR=2.78; 95% CI, 1.21-6.43; P=0.016).

**DISCUSSION**

The main findings of our study were: 1) patients with IDDM had greater in-stent late loss and a higher incidence of restenosis than ND or NIDDM patients; 2) at 1 year of follow-up, patients with IDDM showed trends approaching statistical significance for a higher incidence of death, a higher incidence of TVF, and lower TVF-free survival than ND or NIDDM patients; 3) nevertheless, in scenarios with a high risk of restenosis, SES showed favorable results in IDDM patients, with a 6-month restenosis rate of 16.3% and 1-year event-free survival of 82.1%; and 4) IDDM was not independently related to restenosis or prognosis.

**Diabetics and Coronary Stents: From Conventional Stents to Drug-Eluting Stents**

Studies comparing balloon angioplasty to stent placement in diabetic patients have shown better results in the latter group, although, despite the use of stents, diabetic patients continue to have a higher risk than nondiabetic patients.

Drug-eluting stent experience in humans has shown them to be useful in reducing restenosis and clinical events. At the present time, attention has focused on determining if these results could be maintained in more complicated scenarios. In diabetic patients, a subanalysis of the main clinical trials with DES also showed decreases in the risk of restenosis and clinical events. Thus these devices appear to be a good alternative for diabetic patients, although more data are needed in the more complex lesions frequently observed in diabetic patients, which have not been assessed in previous studies.

**Comparison With Previous Studies**

In this study, ND patients had an incidence of restenosis and TVF of 6.3% and 7.7%, respectively, similar to the 7.8% and 7.7% observed among NIDDM patients. In contrast, IDDM patients had a 16.3% incidence of restenosis and 17.7% of TVF. These results for IDDM patients are better than those reported for conventional stents, which have shown a 6-month incidence of angiographic restenosis of 39.6% and a 1-year incidence of major events of 39.8%.

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partly related to the use of IL-1alpha inhibitors, which was 50.0% in our IDDM patients, since these drugs have been shown to be effective in diabetics.24 Nevertheless, it is important to mention that the results among ND and NIDDM patients were similar, whereas IDDM patients showed a poorer prognosis. Insulin therapy is an important predictor of new revascularization and worse prognosis with conventional stents25 as well as DES.5 Hyperinsulinemia and insulin resistance have been implicated in a variety of molecular mechanisms that could predispose diabetics to a higher incidence of restenosis. The actions of insulin on cellular growth and proliferation are mediated by 2 enzymatic cascades: the phosphatidylinositol 3-kinase (PI3K) pathway and mitogen-activated protein-kinase (MAPK) pathway.13 The PI3K pathway is usually hyperactivated in diabetic patients; however, in situations of severe insulin resistance the MAPK pathway becomes predominant and PI3K is downregulated.14 The PI3K pathway is modulated by signals that implicate the mTOR (mammalian target of rapamycin) protein and therefore, sirolimus (rapamycin) can inhibit it.14 In diabetic patients, sirolimus could inhibit proliferation processes dependent on the PI3K pathway; however, in severe diabetics with considerable insulin resistance, the biological effects of sirolimus could be attenuated when the MAPK pathway is upregulated.

Sirolimus-Eluting Stents in Diabetics

The DIABETES clinical study25 was the first randomized trial to assess the efficacy of SES in diabetic patients. IDDM patients treated with SES had the same degree of reduction in restenosis parameters as diabetic patients under oral therapy. Whereas our study had a late loss of 0.35 mm at 6 months among IDDM patients, the DIABETES study found 0.001 mm at 9 months. Since DES can delay the process of neointimal proliferation, the differences might be even greater at longer term. This difference in late loss among IDDM patients between the 2 studies can be explained by the potential of developing restenosis of the lesions in this registry: 1) in our study, the inclusion criterion was the presence of complex lesions with a high risk for restenosis; 2) the DIABETES study excluded certain scenarios with a high risk of restenosis, such as chronic occlusions, bifurcations, in-stent restenosis, and renal failure, which were not excluded in our study and were rather prevalent, in particular in the IDDM group; and 3) the lesions in our diabetic patients were typically longer. Another question to consider is why there are differences between IDDM and NIDDM in our study, while there were none in the DIABETES study. Our study is a prospective nonrandomized registry and therefore, presents differences in the baseline clinical and angiographic characteristics between the 3 groups studied, which reflects the differential characteristics that these patients present in real life. As a result, we cannot rule out the possibility that, as a whole, the poorer clinical and angiographic profile of IDDM patients may have contributed to these differences; in addition, the fact that up to 28.8% of diabetics treated with diet are included among the non-insulin-dependent diabetes mellitus (NIDDM) may have contributed to making their clinical progress similar to that of ND patients and also to lowering the severity of the latter with respect to insulin-dependent diabetes mellitus.

Other studies have also found differences. In a substudy of the SIRIUS study6 that compared treatment with diet and/or oral antidiabetic agents, insulin therapy was related to a higher incidence of restenosis (35% vs 12.3%; P<0.01) and adverse events (15.8 vs 6.5%; P<0.05).

These results do not allow definitive conclusions to be drawn about whether there are differences in the efficacy of SES according to the type of diabetes treatment. Nor can these differences be attributed to a difference in the biological efficacy of sirolimus or to the confluence of multiple factors favoring restenosis. These hypotheses should be confirmed in new studies, since it would be extremely valuable to know which drugs work best in the different situations, in this era of DES.26 The only independent predictor of restenosis and worse clinical progress was female sex.11

Limitations

Although the registry was carried out prospectively to assess SES in complex lesions, our study was done a posteriori. The study did not use a randomized design and therefore, there were important differences in the baseline characteristics of the 3 groups which makes them somewhat incomparable. Therefore, the poorer results observed among insulin-dependent diabetics may be due to the influence of variables with a confounding effect. Nevertheless, we address an aspect of current interest that has not been extensively evaluated in randomized studies with SES. Finally, more details on the duration and severity of the disease, parameters related to blood glucose control, and insulin resistance would have been extremely valuable in establishing the actual usefulness of SES in diabetic patients.

CONCLUSIONS

In a cohort of patients with SES-treated lesions having a high risk of restenosis, IDDM patients showed a tendency to present greater in-stent late loss, higher incidence of restenosis, and poorer clinical progress at 1 year, when compared to ND or NIDDM patients. Nevertheless, an independent association between the type of diabetes and the prognosis was not established.
REFERENCES


