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Correlates of Restenosis after Deployment of the Palmaz-Schatz Stent: The Importance of Diabetes Mellitus in Patients with Coronary Artery Disease

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Summary. **This study was done to determine the correlates of restenosis after deployment of the Palmaz-Schatz stent. Clinical, procedural and quantitative angiographic data from 85 consecutive patients (92 lesions) with successful stent deployment (diameter stenosis $<50\%$) were analyzed. All patients had follow-up angiography 6.5 ± 2.5 months after stenting. Minimal luminal diameters and diameter stenosis before and immediately after the procedure were 0.82 ± 0.35 mm, $67.8\pm11.5\%$ and 2.96 ± 2.16 mm, $0.9\pm19.3\%$. The number of deployed stents was 1.1 ± 0.4. At follow-up, minimal luminal diameter was 1.93 ± 0.75 mm and diameter stenosis was $23.4\pm27.6\%$. Restenosis (diameter stenosis $\geqq50\%$) occurred in 16 (17.4%) lesions. The incidence of diabetes was significantly higher in the restenosis group than non-restenosis group (62.5% vs. 19.7%; $p=0.0005$), and lesion length was longer (16.2 ± 9.2 mm vs. 10.0 ± 6.2 mm; $p=0.0077$). Other variables did not show differences between the two groups. Using logistic regression analysis, a multivariate study showed that restenosis was correlated only with diabetes ($p<0.0001$, RR=18.18, 95%CI=3.38-97.68). In diabetic patients, HbAlc was higher in the restenosis group than the non-restenosis group at the time of stent deployment ($7.6\pm0.7\%$ vs. $6.3\pm1.4\%$; $p=0.0484$).**

Conclusion: Restenosis after the Palmaz-Schatz stent is strongly concerned with diabetes mellitus.

Key words—Palmaz-Schatz stent, restenosis, diabetes mellitus.

INTRODUCTION

Coronary stents have been proposed as a means of overcoming two major limitations of balloon angioplasty——sudden early closure and late restenosis. Early studies with the Palmaz-Schatz stent (PS stent), introduced 10 years ago, have documented a modest reduction in angiographic restenosis and clinical cardiac events.[1,2] However, the incidence of restenosis still remains at 20 to 30%, which has persisted as a clinical problem. The purpose of this study was to explore the clinical and procedural variables that modify the risk of restenosis after deployment of the PS stent.

METHODS

Patients

Consecutive patients who underwent successful deployment of a PS stent and who were eligible for 6-month angiographic follow-up are described in this report. A total of 85 patients with 92 lesions was enrolled. Stent deployment in primary angioplasty for acute myocardial infarction and chronically occluded vessels was excluded.

Clinical data

Patient age, sex, history of diabetes mellitus, hypertension, hyperlipidemia, cigarette smoking, diagnosis of ischemic heart disease, previous myocardial infarction, history of coronary angioplasty, and extent of coronary artery disease were analyzed.

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Angiographic data

Coronary angiography before stent deployment and at the follow-up period was performed after intracoronary administration of isosorbide dinitrate. An attempt was made to demonstrate the stenosis in orthogonal views. All cineangiograms were analyzed using a quantitative coronary angiographic automated edge detection algorithm[3] (Cardio 500, Kontron Electronik Co.). The outer diameter of the catheter was used for calibration. Minimal lumen diameter (MLD), reference diameter, percent diameter stenosis (DS), and lesion length before and after intervention and on follow-up were measured.

Definitions

Definitions are as follows: 1) Indication for suboptimal result: stenting for immediate post-angioplasty percent diameter stenosis $<50\%$ with substantial residual stenosis by visual estimation. 2) Indication for bailout: stenting for threatened closure due to coronary dissection after angioplasty. 3) restenosis: $\geq 50\%$ diameter stenosis at follow-up.

Statistical analysis

All data are expressed as mean $\pm$ SD unless otherwise indicated. Comparisons between groups with and without restenosis were performed using Student's t test and χ^2 analysis, respectively. Independent correlates of restenosis was determined by multivariate logistic regression analysis. The variables as possible correlates of restenosis were analyzed to see the correlates of poststenting restenosis. A p value less than 0.05 was considered significant.

RESULTS

Clinical and angiographic characteristics

The characteristics of patients are shown in Table 1. The mean age of patients was 67.3 years with a range of 43 to 82 years. Fifty-six of 85 (65.9%) patients were men. Thirty-three (38.8%) patients had stable angina, and 25 (29.4%) patients had unstable angina. Twenty-seven (31.8%) suffered no anginal attack. Fifty-one (60.0%) patients had a history of myocardial infarction and 45 (52.9%) had previously undergone coronary angioplasty. Twenty-five (29.4%) patients had a habit of smoking. Diagnoses of diabetes mellitus, hypertension, and hyperlipidemia were present in 24 (28.2%), 34 (40.0%) and 13 (15.3%) patients, respectively.

Table 1. Patient characteristics

Age (yrs)	67.3±8.7
Male (%)	56 (65.9)
History of:	
Smoking (%)	25 (29.4)
Diabetes mellitus (%)	24 (28.2)
Hypertension (%)	34 (40.0)
Hyperlipidemia (%)	13 (15.3)
Total cholesterol (mg/dl)	189.7±36.1
Triglyceride (mg/dl)	139.7±72.4
Uric acid (mg/dl)	4.6±2.0
Diagnosis	
Stable angina (%)	33 (38.8)
Unstable angina (%)	25 (29.4)
Silent ischema (%)	27 (31.8)
Prior MI (%)	51 (60.0)
PTCA history (%)	45 (52.9)
Extent of CAD	
1VD (%)	31 (36.5)
2VD (%)	34 (40.0)
3VD (%)	20 (23.5)

MI, myocardial infarction; CAD, coronary artery disease; VD, vessel disease.

Table 2. Lesion characteristics

Target	
LAD (%)	46 (50.0)
LCX (%)	11 (12.0)
RCA (%)	33 (35.9)
SVG (%)	2 (2.2)
ACC/AHA lesion type	
A (%)	10 (10.9)
B (%)	62 (67.4)
C (%)	20 (21.7)
Restenotic lesion (%)	25 (27.2)
Indication	
Elective (%)	36 (39.1)
Suboptimal (%)	25 (27.2)
Bailout (%)	31 (33.7)
No. of deployed stents	1.1±0.4

LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; SVG, saphenous vein graft.

The characteristics of the lesions are shown in Table 2. Forty-six (50.0%) lesions were located in the left anterior descending artery, 11 (12.0%) in the left circumflex artery, 33 (35.9%) in the right coronary

and 2 (2.2%) in saphenous vein grafts. According to ACC/AHA classification, 10 (10.9%) lesions were classified as type A, 62 (67.4%) as type B and 20 (21.7%) as type C. Twenty-five (27.2%) lesions were restenotic lesion. Thirty-one (33.7%) stentings were performed as a bailout procedure and 25 (27.2%) for suboptimal results of balloon angioplasty. The mean number of deployed stents was 1.1±0.4, with a range of 0.5 to 2.5. Five patients received stenting to 2 vessels, and one patient received 3 vessels. The mean length of stented lesions was 10.8±6.9 mm. The mean reference diameter was 2.59±0.75 mm, with a mean pretreatment MLD of 0.82±0.35 mm, corresponding to a mean DS of 67.8±11.5%. MLD and DS of immediately after stenting and at follow-up (6.5±2.5 months later) were 0.9±19.3% and 2.96±2.16 mm, 23.4±27.6% and 1.93±0.75 mm, respectively. Restenosis occurred in 16 (17.4%) lesions and 16 (18.8%) patients at follow-up.

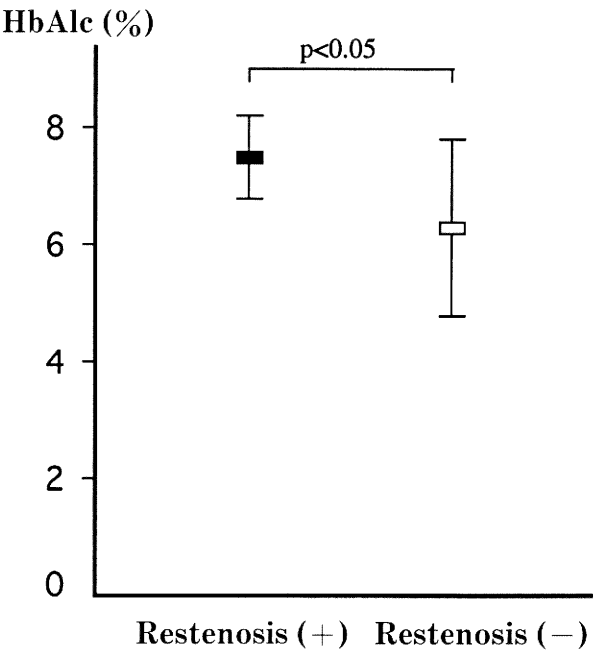
Correlates of restenosis

As shown in Tables 3 and 4, restenosis was associated with a much higher incidence of diabetes and longer lesions (lesion length>15 mm). At the baseline, many lesions in the restenosis group were classified as type C, but the incidence did not reach statistical significance. Analysis of angiography (Table 5) showed a longer length of stented lesions in the restenosis group. There was no difference in reference diameter, poststenting MLD or DS between groups with and without restenosis.

Variables selected for logistic regression analysis were sex (male gender), diabetes, hypertension, hyperlipidemia, cigarette smoking, prior myocardial infarction, unstable angina, target vessel, restenotic lesion, long lesion (lesion length>15 mm), type C lesion, deployment of multiple stents, unplanned deployment. Diabetes was the only significant independent correlate of restenosis ($p<0.001$, relative risk=18.18, 95% confidence interval=3.38-97.68).

Analysis of diabetic patients

Patients with diabetes were analyzed by dividing them into two groups with (10 patients) and without (14 patients) restenosis. Glycosylated hemoglobin (HbAlc) was higher in patients with restenosis than without restenosis (Fig. 1, 7.6±0.7% vs. 6.3±1.4%, $p=0.0484$). Treatment of diabetes (diet therapy alone in 10, oral hypoglycemic agents in 7 and insulin in 7) did not show differences between the two groups. Comparison of other variables revealed a high incidence of hyperlipidemia (30% vs. 0%, $p=0.0342$) and type C lesion (60% vs. 7%, $P=0.0128$) in the restenosis group.



Fig, 1. Plot of the mean±1SD of HbAlc in lesions of diabetic patients.
HbAlc, glycosylated hemoglobin.

DISCUSSION

Early randomized trials with high rates of angiographic follow-up reported a 22 to 32% incidence of restenosis after deployment of the Palmaz-Schatz stent,[1,2] and other reported studies identified relations between restenosis and several characteristics of patients and lesions.[4-22] These included diabetes,[4,6,7,19-21] lesion location,[6] residual stenosis, poststenting MLD,[6-10] deployment of multiple stents,[4,8,10] restenotic lesion,[5,7,8,19] and unplanned stenting.[12-14] Our results showed that the incidence of diabetes and longer lesions was higher in the restenosis group, and diabetes alone was the significant independent correlate of restenosis after logistic regression analysis.

Recent trials, after the proposal of high-pressure postdilatation using intravascular ultrasound (IVUS),[23] have reported a lower restenosis rate and fewer variables related to restenosis.[5,7,15,24] Reduction in the restenosis rate after introduction of these procedures might result from a complete expansion of the stent and good apposition to the vessel wall with reduced residual stenosis.[5,7,24] Despite the sub-

Table 3. Patient variables associated with restenosis

	Restenosis (−)	Restenosis (+)	p-value
Age (yrs)	67.3±9.1	67.7±6.7	0.8565
Male (%)	67.1	43.7	0.0786
History of:			
Smoking (%)	33.8	18.8	0.2397
Diabetes mellitus (%)	19.7	62.5	0.0005
Hypertension (%)	43.8	25.0	0.1644
Hyperlipidemia (%)	15.3	31.3	0.1340
Total cholesterol (mg/dl)	191.6±38.5	187.1±24.5	0.6731
Triglyceride (mg/dl)	144.2±75.3	117.2±44.8	0.2028
Uric acid (mg/dl)	4.6±2.1	4.1±1.6	0.3931
Diagnosis			0.7584
Stable angina (%)	39.5	31.3	
Unstable angina (%)	28.9	37.5	
Silent ischemia (%)	31.6	31.3	
Prior MI (%)	44.7	68.8	0.3211
PTCA history (%)	50.0	50.0	
Extent of CAD			0.9908
1VD (%)	34.2	33.3	
2VD (%)	40.8	40.0	
3VD (%)	25.0	26.7	

Abbreviations as in Table 1.

Table 4. Lesion variables associated with restenosis

	Restenosis (−)	Restenosis (+)	p-value
Target			0.7736
LAD (%)	48.7	56.3	
LCX (%)	13.2	6.3	
RCA (%)	35.5	37.5	
SVG (%)	2.6	0	
ACC/AHA lesion type			0.0664
A (%)	12.0	6.3	
B (%)	70.7	50.0	
C (%)	17.3	43.4	
Restenotic lesion (%)	26.3	31.3	0.6868
Indication			0.2721
Elective (%)	40.0	31.3	
Suboptimal (%)	24.0	43.8	
Bailout (%)	36.0	25.0	
No. of deployed stents	1.10±0.42	1.28±0.48	0.1302
Lesion length > 15 mm (%)	15.9	50.0	0.0123
Multiple stents (%)	22.1	37.5	0.1933
Unplanned stent deployment(%)	58.4	68.8	0.4433

Abbreviations as in Table 2.

stantial part of this study that consisted of restenotic lesion or unplanned stenting, restenosis rate was low compared with early studies that showed a reduction of restenosis after coronary angioplasty by use of the PS stent. Angiographic analysis in our patients showed adequate stent expansion (poststenting mean MLD was 2.96 mm and mean DS was 0.9%); this might result in the low incidence of restenosis and fewer correlates of restenosis.

Importance of diabetes

Findings in several studies have suggested that diabetic patients are at increased risk for restenosis after coronary balloon angioplasty.[25–27] The complex pathophysiological mechanisms involved in the restenosis process in diabetes have been reported to be exaggerated platelet aggregation and thrombus formation, impaired vasodilatory response, dysregulation of growth factor expression, increased

Table 5. Quantitative angiographic evaluation

	Restenosis (−)	Restenosis (+)	p-value
Lesion length (mm)	10.0±6.2	16.2±9.2	0.0077
Reference diameter (mm)	2.61±0.76	2.50±0.68	N. S.
pre. MLD (mm)	0.84±0.34	0.72±0.42	N. S.
pre. % stenosis	67.3±10.7	70.6±16.2	N. S.
post. MLD (mm)	3.05±2.36	2.63±0.59	N. S.
post. % stenosis	0.8±19.6	1.4±18.4	N. S.

MLD, minimal luminal diameter.

production of extracelluar matrix, etc.[20-22,28-32] Recent studies by IVUS analysis revealed that restenosis after deployment of a PS stent was the result of neointimal proliferation, and that chronic stent recoil did not contribute to the late lumen loss.[24,33-35] Therefore, diabetes appears to be an important factor for increased neointimal hyperplasia leading to restenosis after stenting.[28]

Van Belle et al.[36] reported that angiographic outcome was similar in different antidiabetic regimens as shown in this report. However, hypercoagulability, impairment of vasodilatory response, and increase in selected extracellular matrix gene transcription has been reported to be mediated by high glucose concentration.[30-32] In our patients with diabetes, glycosylated hemoglobin was higher in those with restenosis, and the results suggest that inadequate glycemic control is an important factor in restenosis after deployment of a PS stent in diabetic patients. To the best of our knowledge, no information is available on the importance of glycemic control as a risk factor for restenosis. A larger study is needed to address this issue.

Limitations

First, the sample size is small and the results need to be interpreted with caution. Second, the value of glycosylated hemoglobin was determined only at the time of stenting. Glycemic control of the entire period from stenting to follow-up was not confirmed. Third, IVUS studies were not performed. Confirmation of optimal stenting and the exact mechanisms of restenosis were not proved.

Conclusions

The correlates of restenosis after deployment of the Palmaz-Schatz stent were investigated. Diabetes mellitus appeared to be the most strong determinant of restenosis. In diabetics, glycosylated hemoglobin was higher in patients with restenosis than those without restenosis.

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