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Impact of lesion location on intravascular ultrasound findings and short-term and five-year long-term clinical outcome after percutaneous coronary intervention for saphenous vein graft lesions

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ABSTRACT

Background: Little is known about intravascular ultrasound (IVUS) findings and acute post-percutaneous coronary intervention (PCI) outcome and long-term clinical outcome between aorto-ostial lesion and shaft lesion after PCI for saphenous vein graft (SVG) lesions.
Methods: Aorto-ostial lesion was defined as those arising within 3 mm of the origin of SVG and shaft lesion was defined as those arising in remaining portion of SVG. We evaluated pre- and post-PCI IVUS images of 311 SVG lesions and compared IVUS findings and acute post-PCI outcome and 5-year clinical outcome between aorto-ostial lesion ($n=64$) and shaft lesion ($n=247$).
Results: The presence of positive remodeling (40% vs. 22%, $p=0.026$), hypoechoic plaque (47% vs. 36%, $p=0.035$), plaque rupture (23% vs. 8%, $p=0.008$), multiple plaque rupture (9% vs. 2%, $p=0.038$), and an intraluminal mass (54% vs. 25%, $p<0.001$) were significantly more common in shaft lesion than in aorto-ostial lesion. Post-PCI no-reflow (15% vs. 5%, $p=0.033$), post-PCI tissue prolapse (TP) (40% vs. 23%, $p=0.014$), and post-PCI creatine kinase-MB elevation more than 3 times normal (14% vs. 8%, $p=0.047$) were observed more frequently after PCI for shaft lesion than for aorto-ostial lesion. At 5-year clinical follow-up, the incidences of death (25% vs. 13%, $p=0.036$) and myocardial infarction (24% vs. 11%, $p=0.028$), but not the rate of target vessel revascularization (36% vs. 25%, $p=0.096$), were significantly higher in patients with shaft lesion compared with those with aorto-ostial lesion.
Conclusions: SVG shaft lesion has more unstable plaque morphology and this may contribute to the worse acute PCI outcomes and long-term outcomes.

1. Introduction

Percutaneous coronary intervention (PCI) for saphenous vein graft (SVG) disease is limited by distal embolization [1,2] that can cause creatine kinase-MB elevations [3–6] or no-reflow [7–10], the occurrence of which is associated with about 15% rate of mortality and 30% rate of acute myocardial infarction [4]. Although several studies have reported that PCI of SVG aorto-ostial lesions is less likely to be associated with post-procedural complications [4,11], little is known about intravascular ultrasound (IVUS) findings and acute post-PCI outcome and long-term clinical outcome between aorto-ostial lesions and shaft lesions after PCI for SVG lesions. Our hypothesis was that SVG shaft lesions may be associated with more unstable plaque characteristics and more PCI-related complications such as no-reflow or creatine kinase-MB elevation and worse long-term clinical outcomes compared with aorto-ostial lesions. Therefore, the purpose of the present study was to evaluate pre- and post-PCI IVUS characteristics and post-PCI complications and long-term outcome according to the SVG lesion location after PCI for SVG disease.

2. Methods

2.1. Patient population

We reviewed 311 consecutive patients retrospectively who underwent pre-PCI IVUS, IVUS-guided PCI, and post-PCI IVUS for 311 SVG lesions between June 2000

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and December 2005, and we investigated pre- and post-PCI IVUS characteristics and post-PCI complications and 5-year clinical outcome according to the SVG lesion location after PCI for SVG disease. We excluded 25 patients with restenotic SVG lesions, 6 patients treated for stent thrombosis, 10 patients with cardiogenic shock, and 10 patients in whom adequate IVUS images could not be obtained. The protocol was approved by the institutional review board. The patients were followed by telephone communication, office visit, or by contacts with primary physicians or referring cardiologists. At each evaluation, a determination was made of the occurrence of interim adverse events.

2.2. Angiographic variables

Aorto-ostial lesion was defined as those arising within 3 mm of the origin of SVG and shaft lesion was defined as those arising in remaining portion of SVG. All patients were treated with stent implantation: 154 patients with sirolimus-eluting stents, 56 patients with paclitaxel-eluting stents, and 101 patients with bare-metal stents. No-reflow was defined as post-PCI Thrombolysis In Myocardial Infarction (TIMI) grade 0, 1, or 2 flow in the absence of mechanical obstruction. If TIMI flow post-PCI was 0, 1, or 2 in the absence of angiographic stenosis, repeated IVUS was performed to exclude the possibility of mechanical vessel obstruction. Angiographic thrombus was defined as discrete, intraluminal filling defect with defined borders, largely separated from the adjacent wall [11].

2.3. Quantitative coronary angiography (QCA) analysis

Quantitative analysis (CAAS II, Pie Medical, The Netherlands) was performed using standard protocols [12]. With the outer diameter of the contrast-filled catheter as the calibration standard, the reference diameter and minimal lumen diameter (MLD) were measured in diastolic frames from orthogonal projections. Perfusion was evaluated according to TIMI criteria [13].

2.4. IVUS imaging protocol

All IVUS examinations were performed after intra-SVG administration of 200 μg nitroglycerin using a commercially available IVUS system (Boston Scientific Corporation/SCIMed, Minneapolis, MN). The IVUS catheter was advanced distal to the target lesion, and imaging was performed retrograde to the aorto-ostial junction at an automatic pullback speed of 0.5 mm/s.

2.5. IVUS analysis

Quantitative and qualitative analyses were performed according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [14]. Before PCI, we measured SVG and lumen cross-sectional area (CSA) using planimetry software (TapeMeasure, INDEC Systems Inc., Mountain View, CA). The SVG CSA was measured by tracing the outer border of the entire SVG [15]. Plaque CSA was calculated as SVG CSA minus lumen CSA, and plaque burden was calculated as plaque CSA divided by SVG CSA. The lesion site was the image slice with the smallest lumen; if there were multiple image slices with the same minimum lumen CSA, then the image slice with the largest SVG and plaque CSAs was measured. Remodeling index was the ratio of lesion site SVG CSA divided by the average of the proximal and distal reference SVG CSA. Positive remodeling was defined as a remodeling index > 1.05, intermediate remodeling as a remodeling index between 0.95 and 1.05, and negative remodeling as a remodeling index < 0.95 [16].

An intraluminal mass had a layered lobulated appearance, evidence of blood flow (microchannels) within the mass, and speckling or scintillation (similar to thrombus in native coronary arteries). No attempt was made to identify thrombus because it could not be distinguished from degenerated, fragmented graft atheroma. A ruptured plaque contained a cavity that communicated with the lumen with an overlying residual fibrous cap fragment. A fragmented and loosely adherent plaque without a distinct cavity and without a fibrous cap fragment was not considered a plaque rupture. Rupture sites separated by a length of SVG containing smooth lumen contours without cavities were considered to represent different plaque ruptures [17,18]. Hypoechoic plaque was less bright than the adventitia, hyperechoic, noncalcified plaque was as bright as or brighter than the adventitia without acoustic shadowing, and hyperechoic, calcified plaque was brighter than the adventitia with acoustic shadowing. When there was no dominant plaque composition, the plaque was classified as mixed. At post-PCI, we measured the minimum stent CSA. Stent expansion was calculated as minimum stent CSA divided by mean reference lumen CSA. Tissue prolapse (TP) was defined as tissue protrusion through the stent strut post-procedure, and the volume of TP was calculated by subtracting lumen volume from stent volume.

2.6. Long-term clinical outcomes

We examined major adverse cardiac events at 5-year after PCI for SVG lesions. Death includes mortality from all causes. Myocardial infarction was defined as: (1) evolutionary ST-segment elevation, development of new Q-waves in 2 or more contiguous electrocardiographic leads, or new left bundle branch block patterns on the electrocardiogram, or, (2) biochemical evidence of myocardial necrosis, manifested as (a) creatine kinase-MB ≥ 3 times the upper limit of normal, or, if creatine kinase-MB was not available, (b) total creatine kinase ≥ 3 times the upper limit of normal, or (c) troponin value above the upper limit of normal. Target vessel revascularization (TVR) was defined as a repeat revascularization involving the initially treated SVG. Native vessel TVR was not included in the TVR endpoint.

2.7. Statistical analysis

The statistical Package for Social Sciences (SPSS) for Windows, version 15.0 (Chicago, Illinois) was used for all analyses. Continuous variables were presented as the mean value ± 1SD; comparisons were conducted by student's *t*-test or nonparametric Wilcoxon test if normality assumption was violated. Discrete variables were presented as percentages and relative frequencies; comparisons were conducted by chi-square statistics or Fisher's exact test as appropriate. A p value < 0.05 was considered statistically significant.

3. Results

3.1. Patient characteristics

The baseline characteristics are summarized in Table 1. There were no significant differences in graft age; risk factors for coronary artery disease; or the use of bivalirudin, glycoprotein IIb/IIIa inhibitors, and pre-PCI clopidogrel loading comparing the two groups. However, distal protection devices were used more frequently in patients with shaft lesion compared with those with aorto-ostial lesion. The PercuSurge Guardwire Plus System (Medtronic AVE, Sunnyvale, CA) was used in 80 patients, the Angioguard Filter System (Cordis, Miami, FL) was used in 25 patients, and Filterwire EX (Boston Scientific EPI, Santa Clara, CA) was used in 20 patients.

3.2. Angiographic and procedural results

Angiographic findings and procedural results are summarized in Table 2. More patients with aorto-ostial lesion had SVG to LAD lesion, and more patients with shaft lesion had SVG to RCA lesion. Angiographic thrombus was observed more frequently in patients with shaft lesion compared with those with aorto-ostial lesion. Reference diameter was significantly greater in patients with shaft lesion compared with those with aorto-ostial lesion. Stent length was

Table 1
Baseline clinical characteristics.

	Aorto-ostial lesion (n = 64)	Shaft lesion (n = 247)	p
Age (years)	71.4 ± 10.9	69.1 ± 10.3	0.19
Male gender (n)	50 (78%)	184 (75%)	0.5
Clinical presentation (n)			0.5
Stable angina	22 (34%)	68 (28%)	
Unstable angina	36 (56%)	140 (57%)	
NSTEMI	6 (9%)	38 (15%)	
STEMI	0 (0%)	1 (0%)	
Diabetes (n)	24 (38%)	93 (38%)	1.0
Hypertension (n)	50 (78%)	17 (72%)	0.3
Smoking (n)	28 (44%)	98 (40%)	0.6
Hypercholesterolemia (> 220 mg/dl) (n)	47 (73%)	188 (76%)	0.7
Family history of coronary disease (n)	25 (39%)	70 (28%)	0.097
Graft age (years)	10.5 ± 7.4	11.3 ± 6.4	0.4
Ejection fraction (%)	53 ± 10	48 ± 8	0.7
Distal protection device use (n)	15 (23%)	110 (45%)	0.002
Bivalirudin use (n)	53 (83%)	216 (87%)	0.3
Glycoprotein IIb/IIIa inhibitor use (n)	13 (20%)	57 (23%)	0.6
Pre-PCI clopidogrel loading (n)			0.6
None	37 (58%)	125 (51%)	
300 mg	13 (20%)	59 (24%)	
600 mg	14 (22%)	63 (26%)	

NSTEMI: non-ST segment elevation myocardial infarction, STEMI: ST segment elevation myocardial infarction, PCI: percutaneous coronary intervention.