Evaluation of Culprit Saphenous Vein Graft Lesions With Optical Coherence Tomography in Patients With Acute Coronary Syndromes

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Objectives This study sought to assess, with optical coherence tomography (OCT), presumably culprit atherosclerotic lesions of saphenous vein grafts (SVGs) in patients with acute coronary syndromes (ACS).

Background Atherosclerotic lesions of SVGs have been studied in vivo with angioscopy and intravascular ultrasound. However, imaging with OCT, which has a higher resolution than intravascular ultrasound and better penetration than angioscopy, has not been conducted systematically.

Methods Using a nonocclusive OCT technique, we performed angiography and OCT of culprit SVG lesions in patients with unstable angina (UA), ST-segment elevation myocardial infarction (STEMI), and non-STEMI. Fibrous and fatty tissue, calcification, thrombus, and plaque rupture were defined according to OCT objective criteria.

Results Twenty-eight SVGs (average age 14.6 years) in 26 patients were imaged. Lesions on angiography were complex (96.4%), with ulceration in 32.1% and thrombus in 21.4%. OCT disclosed a fibrofatty composition in all lesions, calcification in 32.1%, plaque rupture in 60.7%, and thrombus in 46.4%. Thrombus was progressively more frequent across groups (UA to STEMI, \( p = 0.003 \); UA vs. myocardial infarction, \( p = 0.006 \)). A thin fibrous cap was marginally more frequent in myocardial infarction patients (UA vs. myocardial infarction, \( p = 0.06 \); STEMI 100% vs. non-STEMI 53.3% vs. UA 20%, \( p = 0.03 \)). OCT features of friability were present in 67.9% of SVGs not correlating with clinical presentation.

Conclusions OCT of culprit lesions of old SVGs in patients with ACS demonstrates fibrofatty composition, relatively thin fibrous cap, plaque rupture, and thrombus, which correlate with the clinical spectrum of ACS. This suggests that similar mechanisms with native vessels’ atherosclerosis may be involved in SVG-related ACS. (J Am Coll Cardiol Intv 2011;4:683–93) © 2011 by the American College of Cardiology Foundation
Saphenous vein graft (SVG) failure beyond the first month following coronary artery bypass graft is mainly due to atherosclerosis similar in nature to that of native coronaries (1). Acute coronary syndromes (ACS) occurring in patients with previous coronary artery bypass graft are attributed in 70% to 80% of cases to lesions located in SVG (1). Pathological (2), angiographic (3,4), and intravascular ultrasound (IVUS) studies (5) have shown that, similar to native lesions, SVG lesions have a fibrofatty composition with evidence of positive remodeling, and in patients with ACS plaques, SVG lesions may demonstrate a complex appearance with rupture. Therefore, apart from morphological similarities between SVG and native atherosclerotic disease, common mechanisms underlying ACS attributed to native and SVG lesions may also exist (4,5).

Optical coherence tomography (OCT) has a higher axial resolution than IVUS and its penetration allows visualization of the vessel wall in contrast to angiography (6,7). The efficiency and reproducibility of OCT for plaque characterization have been established and some OCT features such as thin-cap fibroatheroma (TCFA), lipid/necrotic core, and plaque rupture have been recognized as hallmarks of culprit atheromatous plaques in patients with native vessel ACS (6,7).

However, to the best of our knowledge, there are no in vivo studies using OCT to explore the structure and morphology of culprit atherosclerotic SVG lesions in patients with ACS. We conducted an OCT study of presumably culprit SVG atherosclerotic lesions in patients with ACS, aiming to assess OCT characteristics and to explore their correlation, if any, with clinical and angiographic findings.

**Methods**

**Patient population and angiographic data.** From November 2008 to May 2010, all consecutive patients with previous coronary artery bypass graft referred to our center for coronary angiography due to ACS were screened for inclusion in this study. Inclusion criteria were: presentation with an ACS, existence of at least 1 SVG with a significant angiographic stenosis (>50%), ST-T changes on the electrocardiogram obtained during symptoms in the territory of an SVG with a significant stenosis and/or absence of native vessels lesions distal to the SVG anastomosis that could be potentially responsible for the ACS, and patient’s consent for participation in the study.

Demographics, medical history, and clinical data were recorded. ACS included: unstable angina (UA) defined as new-onset, accelerated, or angina at rest; recent (<7 days) myocardial infarction defined as elevation of cardiac enzymes >3× the upper limit of normal and categorized as ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI). In every patient, coronary angiography was performed via a femoral approach before OCT and angioplasty. Previously used angiographic descriptions (8) were applied and included: “ulceration” (discrete luminal widening with luminal irregularity) (Figs. 1 and 2); “lumen irregularity” (irregular lumen border not classified as ulceration) (Fig. 3); and “aneurysm” (lumen dilation >25% larger than normal segment) (Figs. 4 and 5). Lesions were considered complex if they had 1 or more of the above specific morphologies; otherwise, they were classified as simple. Degenerated grafts were classified as those with lumen irregularities or ectasia comprising >50% of the length of the SVG shaft (9) (Figs. 4, 5, and 6). All patients’ lesions were treated with angioplasty and stenting using filter wire (Boston Scientific, Natick, Massachusetts) distal protection. Filter wires were placed in position after removal of the OCT catheter and guidewire. Thrombolyis In Myocardial Infarction (TIMI) flow grade <3 at any stage of the procedure was recorded.

**OCT protocol.** The M2 and C7-XR (for 40% of cases) Cardiology Imaging OCT Systems (LightLab Imaging, Inc., Westford, Massachusetts) were used. A 6- to 7-F guiding catheter was used, and a nonocclusive technique infusing contrast at 3 to 4 ml/s with motorized pullback at 3 mm/s (M2) or 20 mm/s (C7-XR) was employed. Thromboaspiration was conducted if thrombi prevented further insertion of the imaging probe (Fig. 7). The OCT pullbacks were performed before any intervention and continuous images were stored digitally for subsequent analysis.

**OCT analysis.** OCT analysis was performed independently by 2 physicians blinded to the patient’s history and angiographic findings. The OCT images were considered analyzable if the lumen contour was visualized in ≥3 quadrants. Homogeneous tissue with high reflectivity and low attenuation was identified as fibrous. Lipid pools appeared as diffusely bordered, signal-poor regions with overlying signal-rich bands, corresponding to fibrous caps. All cross sections were divided into 4 quadrants. Lipid was semiquantified as the number of involved quadrants on the cross-sectional OCT image. When lipid was present in ≥2 quadrants in any of the images within a plaque, the latter was considered lipid-rich. For each patient, the cross-sectional image with the highest number of lipid quadrants was used for analysis. The fibrous cap was measured...
**Figure 1. OCT of SVG Lesions in a Patient With Lateral Wall NSTEMI**

(A) Angiography of a saphenous vein graft (SVG) to the left anterior descending artery with severe narrowing and thrombus (white box). Plaque ulceration with a flap is shown in the magnification (asterisk). Optical coherence tomography (OCT) images at the site of the lesion: (B) red thrombus (arrow); (C) intimal rupture with a large cavity underneath (asterisk). (C, D) Arrows point to a signal-rich layer, underneath which a signal-free layer is evident, separating the former from the SVG wall. The signal-rich layer probably represents friable tissue “detached” from the SVG wall. In all OCT frames, fibrofatty composition of the intima is evident. NSTEMI = non-ST-segment elevation myocardial infarction.

**Figure 2. OCT of SVG Lesions in a Patient With Anterior Wall Ischemia**

(A) Angiography of an SVG to left anterior descending artery with evidence of plaque rupture (magnification top right, arrow). (B) Thin-cap fibroatheroma (40 μm) at the level of rupture (arrow) with a large cavity underneath (asterisk). (C) Successive OCT frame showing ruptured intima (arrow). In all OCT frames, fibrofatty composition of the intima is evident. Abbreviations as in Figure 1.
at its thinnest part across all analyzed images. A cutoff point of 65 μm was used to distinguish a TFCA (Figs. 2, 3, and 6). Calcifications within plaques were identified by the presence of well-delineated, low backscattering heterogeneous regions (Fig. 4). Plaque ruptures were identified as a cavity communicating with the lumen with an overlying residual fibrous cap fragment (Figs. 1 to 3 and 5 to 7). Red thrombi were defined as high-backscattering protrusions with signal-free shadowing (Figs. 1, 5, and 7), and white thrombi as signal-rich, low-backscattering billowing projections protruding into the lumen (Figs. 4 and 7). Both were categorized under the general title “thrombi.”

**Statistics.** Discrete data were summarized as frequencies and group percentages, and continuous data were summarized as mean ± SD. Student *t* test and 1-way analysis of variance, and Fisher exact and McNemar tests were used for comparison of continuous and categorical data, respectively. All tests were 2-tailed and statistical significance was considered for *p* values <0.05. All statistical analyses were performed using SPSS for Windows (version 16.0, SPSS, Inc., Chicago, Illinois). This study was performed with the approval of Hospital’s Ethics Committee.

**Results**

**Clinical findings.** A total of 26 patients were included in the study. In 1 patient with UA and 1 with NSTEMI, electro-
Cardiogram findings were inconclusive with 2 SVGs having potentially culprit lesions; therefore, both were imaged with OCT raising the number of SVGs studied to 28. Patients’ demographics, SVGs studied, and SVG age according to clinical presentation are presented in Table 1. There were 9 patients with UA, 14 with NSTEMI, and 3 with STEMI. Demographic data and coronary artery risk factors did not differ among ACS subgroups or between UA and myocardial infarction (MI) groups (the latter including both STEMI and NSTEMI). The average age of the patients was 68.4 years, and the average graft age was 14.6 years.

Angiographic and OCT findings. The angiographic and OCT findings according to clinical presentation are summarized in Table 2. The location of the SVG bearing the presumed culprit lesion did not differ among UA, NSTEMI, and STEMI. Most lesions were complex (96.4%), with visible angiographic ulceration in 32.1%, and thrombus in 21.4% of cases (Figs. 1 and 2). In total, 46.4% of grafts were characterized as degenerated (Figs. 4 to 6).

In all patients, a fibrofatty plaque was demonstrated (Figs. 1 to 7). Calcification (Fig. 4) was found in 32.1% of cases and did not vary significantly within the ACS subgroups. Thrombus was more frequently detected with OCT (Figs. 1, 4, 5, and 7) than with angiography (46.4% vs. 21.4%, McNemar test $p = 0.016$). The presence of thrombus on OCT was progressively more frequent moving from UA to STEMI ($p = 0.003$). Plaque rupture on OCT (Figs. 1 to 3 and 5 to 7) followed a similar distribution across the ACS subgroups, without reaching statistical significance (Table 2). A TFCA was demonstrated in all STEMI patients (100% for STEMI vs. 53.3% for NSTEMI and 20% for UA, $p = 0.03$) (Figs. 2, 3, and 6). The average thickness of the fibrous cap was 52.5 μm for STEMI, 81 μm for NSTEMI, and 80 μm for UA patients ($p = 0.3$). When we analyzed these parameters in MI versus UA patients, the former had significantly more frequent thrombus (66.7% vs. 10%, $p = 0.006$) and marginally more frequent TCFA (61.1% vs. 20%, $p = 0.06$), all other differences being nonsignificant.

Figure 4. OCT of SVG Lesions in a Patient With an Inferior Wall NSTEMI

(A) Angiography of an SVG to right coronary artery, showing severe degeneration and aneurysmal dilation with a 90% stenosis at its distal part. (B to F) Successive OCT images of the SVG. Severe aneurysmal dilation is evident in the long axis (bottom of all panels). (B) Arrow points to white thrombus at the site of angiographic stenosis. (C) Arrow points to calcific deposit. (D, E) Arrows point to white thrombus. (F) Arrow points to cholesterol clefts. In all OCT frames, fibrofatty composition of the intima is evident. Abbreviations as in Figure 1.
In most of the studied SVGs, we identified a distinct layer of tissue in discontinuity with the vein wall, loosely attached to the latter (Figs. 1, 3, 6, 7, and 8). Such fragmented and loosely adherent tissues without a distinct cavity and without a fibrous cap fragment were not considered plaque rupture. Instead, they correlated with areas of severe angiographic degeneration, and we speculate that they represent degenerated fragmented graft atheroma (friable tissue). Such OCT features of friability were present in 67.9% of SVGs without difference among the 3 ACS subgroups, or between UA and MI. A spectrum of OCT findings such as tissue friability or fragmentation, rarely found in native coronaries, is presented in Figure 8.

All patients’ lesions were treated successfully with angioplasty and stenting. The rate of TIMI flow grade \(<3\) (17.9%) progressively increased from UA to STEMI; however, it did not differ significantly across the clinical spectrum of ACS, or between UA and MI. All cases were associated with evidence of graft degeneration.

**Discussion**

Our OCT study shows that the presumed culprit angiographic lesions in old SVGs in patients with ACS have a fibrofatty composition and a relatively thin fibrous cap. A complex plaque morphology (rupture and thrombus) associated with a TCFA is common and may occur more frequently in patients with STEMI and NSTEMI compared with patients with UA. Finally, the OCT feature corresponding to the angiographic appearance of friable-degenerated SVGs was identified as an internal layer of tissue loosely attached and/or in discontinuity with the vein wall (Figs. 1, 3, 6, and 8).

OCT features of culprit SVG lesions similar to native lesions.
Our OCT findings regarding the fibrofatty composition and complexity of culprit SVG plaques are in accord with angioscopic studies showing a yellow (lipid-rich) plaque in most SVG lesions in patients with UA, along with ulceration and thrombus in a significant proportion, which was similar to that of patients with native vessels disease presenting with UA (3,4). A previous IVUS study has demonstrated that ruptured SVG plaques occur almost exclusively in old SVGs (>12 years), are found more often in patients with ACS, have a complex angiographic appearance, and demonstrate similar IVUS features as ruptured plaques in native coronary arteries (e.g., positive remodeling) (5). Similarly, in our study, the vast majority of lesions demonstrated severe angiographic complexity (96.4%) with evidence of rupture in 60.7% on OCT (Figs. 1 to 3 and 5 to 7). SVG atheromas may be more prone to rupture as they contain more foam and inflammatory cells and have poorly developed or absent fibrous caps (1,10,11). In native coronaries, culprit lesions with TCFA, which correlates with plaque rupture, are found progressively more frequently in patients with stable angina, UA, NSTEMI, and STEMI (7,12).

Similarly, we detected relatively thin fibrous caps (71.4 ± 27.3 μm) over most of the culprit SVG lesions, whereas TCFA frequency correlated with clinical presentation (Figs. 2, 3, and 6). Although the incidence of plaque rupture detected by OCT in our study was higher compared with that detected by angioscopy and IVUS (3–6), the mechanism of ACS in the remaining cases is not explained easily, and we cannot exclude the possibility that a region of plaque rupture or erosion was missed during OCT interpretation. These observations are in accord with the suggestion that the mechanisms underlying plaque rupture and ACS are similar between SVGs and native coronaries (4,5).

OCT features unique to SVG atherosclerotic disease. SVG atherosclerotic plaques are larger with frequent aneurysmal dilations and intraluminal friable tissue compared with those in native coronaries (2,10,11). It is very interesting that in most of our cases with evidence of plaque rupture, a large cavity was demonstrated, something that is infrequent in native vessel disease (Figs. 1 to 3 and 5 to 7). We speculate that the larger size of the SVGs as well as of their atherosclerotic lesions compared with those of native coro-
nary arteries, and/or the high lipid content of these plaques, may partly account for this finding. Degenerated fragmented graft atheroma (friable plaque) corresponds to loose fibroatheromatous debris lining on the internal surface of the vessel and is rarely found in atherosclerosis of native coronary arteries. Angiographically, it presents as an irregular or serrated luminal border. On angioscopy, friable plaques usually appear as fragmented, loosely adherent plaques lining the SVG wall, and they are found in approximately 45% of patients with UA (3,4). With OCT, we detected circumferential signal-free zones inside the walls of SVGs and, over those, a distinct high-signal layer of tissue loosely attached and/or in discontinuity with the vein wall, correlating with areas of degenerated SVGs on angiography, which we presume represent friable tissue (Figs. 1, 3, 6, 7, and 8). In accord with others (3), we did not find any correlation between plaque friability and graft age; however, the number of our patients with grafts <5 years old was small. There is evidence that the presence of friable material or age of the graft (age ≥3 years) correlates with distal

**Table 1. Patient Demographics**

<table>
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<tr>
<th></th>
<th>Total (n = 26)</th>
<th>UA (n = 9)</th>
<th>NSTEMI (n = 14)</th>
<th>STEMI (n = 3)</th>
<th>p Value</th>
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<tr>
<td>Age, yrs</td>
<td>68.4 ± 7.7</td>
<td>71.2 ± 6.5</td>
<td>65.6 ± 8.9</td>
<td>72.8 ± 7.0</td>
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<td>7 (77.8)</td>
<td>10 (71.4)</td>
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<td>Hypertension</td>
<td>14 (53.8)</td>
<td>6 (66.7)</td>
<td>7 (50)</td>
<td>1 (33.3)</td>
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<tr>
<td>Current smoking</td>
<td>10 (38.5)</td>
<td>3 (33.3)</td>
<td>5 (35.7)</td>
<td>2 (66.7)</td>
<td>0.7</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>8 (30.8)</td>
<td>1 (11.1)</td>
<td>7 (50)</td>
<td>0 (0)</td>
<td>0.07</td>
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<tr>
<td>Family history of CAD</td>
<td>8 (30.8)</td>
<td>2 (22.2)</td>
<td>4 (28.6)</td>
<td>2 (66.7)</td>
<td>0.4</td>
</tr>
<tr>
<td>SVGs studied/patient</td>
<td>1.08 ± 0.3</td>
<td>1.1 ± 0.3</td>
<td>1.1 ± 0.3</td>
<td>1 ± 0.0</td>
<td>0.8</td>
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<tr>
<td>Graft age, yrs</td>
<td>14.6 ± 4.3</td>
<td>16.5 ± 2.8</td>
<td>13.3 ± 5.0</td>
<td>15.0 ± 2.6</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Values are mean ± SD or n (%). The p values refer to comparisons among UA, NSTEMI, and STEMI patients.

CAD = coronary artery disease; NSTEMI = non–ST-segment elevation myocardial infarction; STEMI = ST-segment elevation myocardial infarction; SVG = saphenous vein graft; UA = unstable angina.

**Figure 7. OCT of SVG Lesions in a Patient With an Anterior STEMI**

(A) Angiography of a degenerated SVG to right coronary artery, showing narrowing with plaque rupture (arrow). (B to F) OCT images at the site of the lesion. (B, C) The fibrous cap (B) and rupture (C) of the thin-cap fibroatheroma (60 μm) with a large cavity underneath (arrows). D to F demonstrate signal-rich friable material loosely attached at the SVG wall (arrows). In all OCT frames, fibrofatty composition of the intima is evident. Abbreviations as in Figure 1.
emobilization and periprocedural mortality during percutaneous coronary intervention (13). We detected possible distal embolization (TIMI flow grade <3) during the procedure in 5 grafts (17.9%), which all demonstrated degeneration. Finally, culprit SVG lesions demonstrating aneurysmal dilation were also reliably detected by OCT especially in the long-axis images (Figs. 4 and 5).

Severe vessel degeneration, aneurysmal dilation, and plaque friability are very rarely seen in native coronary arteries and constitute unique findings on angiography and/or intravascular imaging of SVGs (4,14). All these, along with a spectrum of rare SVG plaque characteristics, such as “tissue fragmentation” (Fig. 8), were depicted in detail in this OCT study. In this rare SVG plaque characteristics, such as “tissue fragmentation” (Fig. 8), were depicted in detail in this OCT study. In this OCT study, we took all necessary precautions to avoid injury of SVGs by placing the filter wire after having performed the OCT, we cannot exclude the possibility that some OCT findings could be due to the passage of guidewires or OCT catheters. Although tissue penetration remains essentially the same (1.5 to 2 mm) between the 2 OCT systems used, M2 and C7-XR, the C7-XR has a larger field of view (10 mm vs. 7 mm), allowing SVGs with larger diameters to be visualized better. Hence, more than 1 pullback over the region of interest was needed with the M2 system to allow acquisition of satisfactory and analyzable images. Due to the slower pullback speed, lower frame rate, and smaller field of view of the M2 system, more contrast was needed. Our results are preliminary. We did not correlate OCT findings with clinical endpoints, and we did not compare OCT findings with clinical endpoints, and we did not compare OCT with other invasive imaging modalities such as IVUS. Therefore, we cannot conclude the possible clinical utility of OCT in SVG lesions. By contrast, we have shown that OCT allows detailed imaging and characterization (according to objective OCT criteria) of atherosclerotic lesions of SVGs. It would be very interesting for future research to examine any possible correlation of OCT findings, such as

<table>
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<tr>
<th>Graft location</th>
<th>Total (n = 28)</th>
<th>UA (n = 10)</th>
<th>NSTEMI (n = 15)</th>
<th>STEMI (n = 3)</th>
<th>p Value</th>
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<td>LAD</td>
<td>9 (32.1)</td>
<td>5 (50)</td>
<td>3 (20.1)</td>
<td>1 (33.3)</td>
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<tr>
<td>RCA</td>
<td>7 (25.0)</td>
<td>1 (10)</td>
<td>5 (33.3)</td>
<td>1 (33.3)</td>
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<td>LCx</td>
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<td>4 (40)</td>
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<td>1 (33.3)</td>
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<td>Luminal irregularity</td>
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<td>8 (80)</td>
<td>15 (100)</td>
<td>3 (100)</td>
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<td>Ulceration</td>
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<td>2 (20)</td>
<td>6 (40)</td>
<td>1 (33.3)</td>
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<td>Thrombus</td>
<td>6 (21.4)</td>
<td>0 (0)</td>
<td>5 (33.3)</td>
<td>1 (33.3)</td>
<td>0.08</td>
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<td>Complex lesion</td>
<td>27 (96.4)</td>
<td>9 (90)</td>
<td>15 (100)</td>
<td>3 (100)</td>
<td>0.5</td>
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<tr>
<td>Aneurysm</td>
<td>8 (28.6)</td>
<td>2 (20)</td>
<td>6 (40)</td>
<td>0 (0)</td>
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<tr>
<td>Degeneration</td>
<td>13 (46.4)</td>
<td>5 (50)</td>
<td>7 (46.7)</td>
<td>1 (33.3)</td>
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<td>5 (17.9)</td>
<td>1 (10)</td>
<td>3 (20)</td>
<td>1 (33.3)</td>
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</table>

**Table 2. Angiographic and OCT Findings in Studied SVGs**

Values are n (%) or mean ± SD. The p values refer to comparisons among UA, NSTEMI, and STEMI patients.

LAD = left anterior descending artery; LCx = left circumflex artery; OCT = optical coherence tomography; RCA = right coronary artery; TCFA = thin-cap fibroatheroma; TIMI = Thrombolysis In Myocardial Infarction; other abbreviations as in Table 1.
a large lipid pool, TCFA, friable material, and so forth, with the rate of distal embolization during percutaneous coronary intervention of SVGs.

Conclusions

OCT imaging of presumed culprit atherosclerotic lesions of old SVGs in patients with ACS demonstrates features similar to native atherosclerosis such as fibrofatty composition, a relatively thin fibrous cap, and a high percentage of plaque rupture with thrombus, which all correlate with the clinical spectrum of ACS. These findings suggest that similar mechanisms to native vessels’ atherosclerosis may be involved in SVG-related ACS. OCT demonstrated, in detail, features unique to SVG disease, such as large cavities at the site of plaque rupture, aneurysms, and tissue friability.

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Key Words: acute coronary syndrome(s) ■ optical coherence tomography ■ saphenous vein graft.