Saphenous Vein Graft Shrinkage as a Mechanism of Stenosis Soon After Bypass Surgery

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A 63-year-old man with left main coronary artery disease underwent aortocoronary bypass surgery using saphenous vein grafts. Less than 1 month later, severe narrowing occurred in the mid-portion of the vein graft to the left anterior descending coronary artery. Preintervention intravascular ultrasonography revealed prominent vein graft shrinkage. Percutaneous transluminal angioplasty failed because the stenotic lesion could not be dilated, even by high-pressure balloon inflation. Saphenous vein graft shrinkage appears to be one of the mechanisms of early saphenous vein graft stenosis, and balloon angioplasty to the vein graft stenosis with prominent shrinkage may be of limited value. (Jpn Circ J 1998; 62: 382–384)

Key Words: Saphenous vein graft; Paradoxical shrinkage; Intravascular ultrasound

Histologic studies of saphenous vein grafts obtained at reoperation or during postmortem examination have shown that diffuse intimal fibrous proliferation occurs 1 month after surgery, and atherosclerotic changes occur in vein grafts after 1 year. Recent intravascular ultrasound studies have demonstrated that adaptive remodeling occurs to compensate for the accumulation of atherosclerotic plaque. Thus, paradoxical arterial wall shrinkage is an important cause of severe coronary artery narrowing. Thrombotic stenosis or occlusion is a common cause of early saphenous vein graft occlusion within 1 month. Severe intimal proliferation is another mechanism of early saphenous vein narrowing. To our knowledge, it remains unknown whether paradoxical vein graft shrinkage contributes to the development of early severe narrowing of saphenous vein graft. Here we report a case of early saphenous vein graft stenosis, and the mechanism of severe narrowing is suggested to be vein graft shrinkage.

Case Report

A 63-year-old man was admitted for evaluation of recent onset of effort angina. Diagnoses of essential hypertension, non-insulin-dependent diabetes mellitus, hypercholesterolemia, and atrial fibrillation had been made 10 years earlier, and he had been treated with nicardipine, lisoprol, prednisolone, glibenclamide, and warfarin. He had a family history of coronary artery disease, and his lipoprotein(a) level was 5 mg/dl. Cardiac catheterization was performed on February 26, 1997. Coronary angiography revealed 90% stenosis of the left main coronary artery and 99% stenosis of the proximal left anterior descending coronary artery. Left ventriculography revealed akinesis in the apical region and the left ventricular ejection fraction was 68%. He underwent coronary bypass surgery because of left main coronary artery disease on March 3, 1997. Saphenous veins weregrafted to the left anterior descending coronary artery and the high lateral branch. Special attention was paid to the manipulation of the saphenous vein graft and there was no apparent trauma to the vein grafts during preparation and harvesting of the grafts. To assess the patency of the vein grafts, coronary angiography was performed on April 7, 1997. The saphenous vein graft to the left anterior descending coronary artery was severely narrowed in the middle portion (Fig 1A and B). Percutaneous transluminal angioplasty was attempted on April 14, 1997. Preintervention intravascular ultrasonography (Cardiovascular Imaging Systems Inc) revealed prominent shrinkage in the lesion site (Fig 2). Vessel cross-sectional area (CSA) at the lesion site was 6.8 mm² (Fig 2B), which was smaller than the diameter of the proximal (Fig 2A) and distal (Fig 2C) regions (13.9 mm² and 11.9 mm², respectively). However, there was no apparent difference in vessel wall CSA (vessel CSA — luminal CSA) between the lesion site (4.8 mm²) and the proximal (6.4 mm²) or distal (4.4 mm²) sites. A non-compliant balloon with a diameter of 3.0 mm was used, and an indentation of the balloon did not disappear, even after high-pressure inflation up to 20 atmospheres (Fig 1C). After angioplasty, residual vein graft stenosis remained severe (Fig 1D).

Discussion

Morphologic and histologic studies of saphenous vein grafts at autopsy or after reoperation have shown that vein graft disease can be divided into 3 phases as follows. Acute thrombosis causes the majority of graft occlusions within 1 month after surgery. Intimal proliferation is the next stage of morphologic change beyond 1 month after operation. Atherosclerotic change occurs in vein grafts after 1 year.

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Although the early occlusive mechanism of saphenous vein grafts is mainly thrombosis, intimal proliferation is reported to be a cause of early saphenous vein graft narrowing. Batayas et al. described a patient with intimal thickening of a vein graft resulting in narrowing of the lumen a few months after graft insertion. And Waller et al. observed morphologically severe restenotic narrowing of a saphenous vein graft 3 months after surgery. Morphologically the common finding is severe diffuse luminal narrowing caused by intimal thickening. These reports, however, did not report vein graft shrinkage at the site of the stenotic lesion. Recent morphologic and intravascular ultrasound studies have demonstrated that compensatory enlargement is a common phenomenon in atherosclerotic arteries. Inadequate compensatory enlargement or paradoxical arterial wall shrinkage is an important mechanisms of severe narrowing of the coronary artery. To our knowledge, it remains unknown whether vessel wall shrinkage plays a role in severe stenosis of saphenous vein grafts early after graft insertion. The present intravascular ultrasound study showed that vein graft shrinkage is significant as a mechanism of early vein graft stenosis. Initial endothelial damage to the saphenous vein graft resulting from mechanical trauma and/or acute exposure to arterial pressure may lead to subsequent vessel wall changes. Andersen et al. have shown that neoadventitial formation occurs 3 weeks after balloon injury in porcine coronary artery, and late luminal narrowing is caused predominantly by circumferential neoadventitial shrinkage. They hypothesized that the
adventitial damage triggers neoadventitial formation that may be followed by neoadventitial shrinkage. It is, however, unknown whether the same mechanisms responsible for the porcine coronary artery shrinkage operate on the saphenous vein graft.

Percutaneous transluminal angioplasty is an effective therapeutic modality for bypass graft stenosis or occlusion. There are, however, no available data regarding the effectiveness of percutaneous transluminal angioplasty on early vein graft narrowing caused by vessel shrinkage as shown in the present study. Even after balloon angioplasty with high-pressure inflation, the stenotic lesion could not be dilated. Waller et al. suggested that the dilating mechanism of graft stenosis with intimal thickening is not intimal compression but rather graft stretching. Percutaneous transluminal angioplasty in vein graft stenosis with prominent vessel shrinkage and intimal thickening may have only a limited effect on stretching of the vein graft. Although several new techniques, such as rotational atherectomy and laser angioplasty, have been developed, catheter interventions are suggested to have a limited role in achieving a larger luminal area because the vessel cross-sectional area of the lesion itself is about half of the proximal or distal vessel cross-sectional area. Assessment of lesion morphology by intravascular ultrasoundography may have significant therapeutic implications, especially in the case of early saphenous vein graft narrowing, as shown in the present study.

References