A Case of Pure Thrombus in a Saphenous Vein Graft Six Years After Bypass Surgery

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Summary
A man showed a 90% narrowing at the origin of a saphenous vein graft (SVG) 6 years after bypass surgery. After 4 days of intravenous thrombolytic therapy the narrowing in the SVG disappeared completely. Thus, late stenosis of SVG can be caused by thrombosis not superimposed on organic narrowing.

Additional Indexing Words:
Unstable angina Urokinase Thrombosis Saphenous vein graft

The narrowing or occlusion of a saphenous vein graft (SVG) is said to be caused by thrombosis in the initial weeks after bypass surgery, by intimal proliferation or medial hyperplasia in the following several months and by atherosclerosis thereafter. Here we report a case with unstable angina showing a severe narrowing in the SVG, which completely vanished 4 days after treatment with intravenous urokinase.

Case Report
A 59-year-old man noticed effort angina in August 1979, which became more frequent in November. In another hospital he was given a beta-blocker following which his angina occurred less frequently and only with strenuous effort. He remained stable until September 1982, when he was admitted for diagnostic coronary arteriography. His risk factors included hyperlipemia (total cholesterol: 286 mg/dl, HDL-cholesterol 30 mg/dl, triglycerides 189 mg/dl), borderline glucose tolerance and smoking (20 cigarettes a day). He was not obese (162 cm and 62 kg) and had no family history of coronary heart disease. The coronary arteriogram indicated a 90% narrowing in

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the proximal segment of the left anterior descending artery (LAD) and occlusion in the distal segment of the left circumflex artery, which was opacified by the collaterals from the proximal segment of the circumflex artery itself. The right coronary artery (RCA) and the left ventricle were normal. Among the various choices of therapy, he chose aortocoronary bypass surgery of the LAD in October 1982. His postoperative course was good and the angiogram performed in December 1982 showed patency of the SVG to the LAD. He remained asymptomatic and a routine follow-up angiogram in February 1985 showed disease progression; the LAD was occluded at the very site of the previous stenosis, and there was a new 60% narrowing in the RCA just distal to the acute margin. The SVG and the circumflex artery showed no changes. He had been free of chest pain until the end of October 1985, when he began to have frequent anginal attacks and was readmitted. A repeat arteriogram showed subtotal occlusion at the very site of the 60% stenosis. The distal segment of the RCA was opacified by collaterals from the LAD and the circumflex artery. The SVG, the left ventricle and the circumflex artery were unchanged from the previous studies. Percutaneous transluminal coronary angioplasty (PTCA) was attempted in order to dilate the RCA. This was not successful because of the marked tortuosity of the RCA proximal to the acute margin. With maximal doses of diltiazem and long-acting isosorbide dinitrate his angina became stable. On March 26th, 1989, he was awakened by chest pain which subsided with sublingual isosor-
bide dinitrate. He fell asleep again only to be awakened by chest pain, which again subsided with sublingual isosorbide dinitrate. The next day, he again had chest pain associated with a cold sweat while walking in the morning and it continued for about 10 min even with 2 tablets of sublingual isosorbide dinitrate. He was admitted immediately. The electrocardiogram showed no significant changes and the serum creatine kinase level was 157 units with an MB fraction of 8% (in our laboratory, it is normally below 200 without an MB fraction). He was given 240,000 units of urokinase intravenously on admission with 60 mg of diltiazem and 20 mg of long-acting isosorbide dinitrate every 8 hours, had no chest pain at all until the diagnostic arteriogram on March 31st. The arteriography was performed after intravenous administration of 5 mg of isosorbide dinitrate and showed an
80–90% narrowing near the proximal segment of the SVG (Fig. 1A, B). The RCA was completely occluded at its proximal segment and its distal segments received collaterals from the LAD and the circumflex artery (Figs. 1, 2). We recommended that he have repeat bypass surgery of the LAD and the RCA. However, he chose to have a PTCA, which was then planned for April 5th. He received 120,000 units of intravenous urokinase daily until April 4th. He remained asymptomatic with large doses of diltiazem and long-acting isosorbide dinitrate. With all the equipment for emergency utilization of an intra-aortic balloon pump and subsequent bypass surgery on hand, an angiogram was performed, which, to our surprise, showed the SVG to have a normal appearance (Fig. 3A, B), and it appeared to be the same as in the angiogram performed 1 month after the bypass surgery. The PTCA was not performed and he was discharged with the same medication as before.

**DISCUSSION**

Stenoses of SVGs are, in general, said to be due to thrombosis when they occur within a month after bypass surgery, to intimal or medial hypertrophy during the following several months and to atherosclerosis thereafter. In the late follow-up periods after SVG surgery, thrombosis, if present, superimposes on atheromatous plaque. Accordingly, on the diagnostic arteriogram 5 days prior to the attempted PTCA, we believed the stenosis of the SVG in this patient to be due to atherosclerosis because more than 6 years had passed between the bypass surgery and the morphology of the narrowing was not suggestive of a thrombus. However, the stenosis in the SVG completely disappeared 5 days after the diagnostic arteriogram and we were forced to consider that thrombosis was the sole origin of the stenosis in the SVG which gave rise to the symptoms of unstable angina and that atherosclerosis of the SVG was, if present, minimal and not visible on the angiogram. Solymoss, on examining SVGs resected during repeat bypass surgery, reported that thrombosis plays some role in late occlusion of SVGs in many cases. However, in their report, all thromboses were associated with atherosclerosis, or in a small number of cases, with nonatherosclerotic intimal hyperplasia. Our case resembles 2 cases reported by Grill. In their report, a thrombus originating in the venous sinus having no significant atherosclerosis occluded the SVGs 8 and 10 years after bypass surgery. They suggested that localized stagnation of blood might have given rise to the thrombus which was resolved with intracoronary and intravenous streptokinase within 24 hours after administration without significant residual
stenosis. The narrowing in the SVG of our case was similar in its appearance to their second case. However, the site of the thrombus was not in the venous sinus on the angiogram in our case and the surgeons did not describe or remember that they had utilized the saphenous vein at a site having a venous sinus. Thus, our case might be the first one in which an occlusive thrombus arose in a SVG without significant atherosclerosis, and at a site without blood stagnation late in the follow-up period after an aortocoronary saphenous vein bypass operation.

As we have no way to positively differentiate the etiology of the narrowings seen on angiograms, we must always consider the possibility that the stenosis might not be fixed and might be due to a thrombosis or spasm in graft vessels as well as the native vessels.

REFERENCES