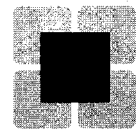

# Multiple myocardial infarctions associated with atheromatous emboli after PTCA of saphenous vein grafts

## A clinicopathologic correlation

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■ Percutaneous transluminal coronary angioplasty (PTCA) of stenotic saphenous vein grafts is less invasive than repeat coronary bypass grafting; however, this procedure is complicated by atheromatous embolization in 2.5%–3.0% of cases, an incidence significantly higher than in angioplasty of native coronary vessels. We reviewed the cases of 98 patients who underwent elective saphenous vein graft angioplasty at our institution. Five patients (5.1%) had non-Q-wave myocardial infarctions after the procedure, documented by elevated serum creatine kinase. In four, atheroembolism was suspected during the procedure. Two patients died, and autopsy showed that both had multiple atherosclerotic emboli to the distal epicardial coronary artery supplied by the vein graft. In addition, multiple infarcts of varying age, some clearly antedating the angioplasty procedure, were identified. These findings suggest that both ongoing, recurrent atheroembolism as well as angioplasty-related embolism may occur in older vein grafts.

□ INDEX TERMS: ANGIOPLASTY, TRANSLUMINAL; AORTOCORONARY BYPASS; EMBOLISM; MYOCARDIAL INFARCTION □ CLEVE CLIN J MED 1988; 56:581–584

PERCUTANEOUS transluminal coronary angioplasty (PTCA) is an acceptable and less invasive treatment than repeat coronary bypass grafting for some patients with stenotic saphenous vein bypass grafts and symptomatic disease.[1–3] The initial success rate is high, particularly at the distal anastomosis, and the complication rate is low during the procedure.[3] However, restenosis remains a major problem, reportedly occurring in 18%–50% of cases, depending upon the site of the angioplasty in the graft.[3] Very early graft occlusion (within one month) is generally thrombotic; later graft occlusion (one month to one year) is caused by progressive fibromuscular intimal hyperplasia of the vein graft.[4] Very late graft occlusion (generally after three years) is characterized by atheromatous plaque formation causing stenosis of the graft.[5] Embolization of atheromatous debris to the distal coronary arteries, with possible myocardial infarction, is a complication of PTCA of saphenous vein bypass grafts. An incidence of 2.5%–3.0% has been reported,[1] which is significantly higher than that reported for angioplasty of native coronary arteries.[6]

Of our patients who had PTCA for saphenous vein

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TABLE 1
MYOCARDIAL INFARCTION FOLLOWING SAPHENOUS VEIN GRAFT ANGIOPLASTY

| Patient | Age/sex | Type of graft | Age of graft | Stenosis | CPK peak IU/L | ECG | Comment |
|---|---|---|---|---|---|---|---|
| 1 | 72/M | SVG LAD | 16 yr | 80% distal | 440 (20% MB) | No change | Died 7 days after procedure |
| 2 | 61/M | SVG Cx | 6 yr | 80% proximal | 1420 (12% MB) | ST depression V4-V5 | Embolus to distal Cx at angioplasty, persistent enzyme elevation. Died 4 days after procedure. |
| 3 | 75/M | SVG RCA | 8 yr | 70–80% mid | 535 (11% MB) | No change | Distal embolus to PDA seen at angioplasty |
| 4 | 54/M | SVG "Y" Dg/Cx | 6 yr | 75% limb to Dg | 492 (12% MB) | No change | Enzyme elevation after 24 hr. Repeat cath: patent SVG |
| 5 | 61/M | SVG RCA | 4 yr | 90% proximal | 516 (26% MB) | No change | Embolus to PV branch of RCA at angioplasty |

*Cx* = circumflex coronary artery; *Dg* = diagonal branch; *LAD* = left anterior descending artery; *PDA* = posterior descending branch; *PV* = posterior ventricular branch; *RCA* = right coronary artery; *SVG* = saphenous vein graft.

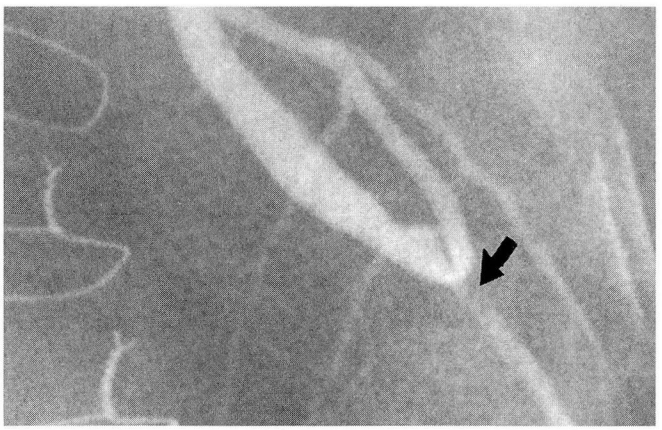

**FIGURE 1. Patient 1. Severe stenosis of saphenous vein graft to the left anterior descending coronary artery, distal anastomotic site (arrow).**

graft stenosis, we identified five who had associated myocardial infarction. In two patients this complication was fatal. In four of the five patients, atheroembolism was suspected at angioplasty or documented at autopsy. The pathologic findings are described and correlated with the clinical courses in the two fatal cases.

## METHODS

The clinical records of 98 patients who consecutively underwent PTCA for vein graft stenosis were reviewed for evidence of myocardial infarction, documented by elevated serum creatine kinase or ECG changes. In the two fatal cases, postmortem examination was done. At autopsy, the vein grafts were carefully dissected, fixed in buffered formalin, and decalcified. The grafts were then serially cross-sectioned and submitted for routine processing, maintaining orientation of the grafts throughout. In addition, multiple sections of myocardium, including areas supplied by the stenotic vein grafts, were examined for evidence of myocardial infarction and coronary atheroemboli.

## RESULTS

Of the 98 cases reviewed, five (5.1%) had myocardial infarction complicating saphenous vein graft angioplasty. The clinical data are summarized in *Table 1*. All five patients had remote saphenous vein grafts (older than four years). Non-Q-wave infarction was documented in each case by elevated serum creatine kinase. Embolization was suspected based on angiographic evidence at the time of angioplasty in four patients (patients 1, 2, 3, 5). Two patients died (patients 1 and 2); their clinical courses and autopsy findings are detailed below. The three remaining patients had uncomplicated courses after infarction. No patient in our series had evidence of atheroemboli without myocardial infarction.

## CASE REPORTS

*Patient 1* A 72-year-old white man who had undergone left internal mammary artery implant and saphenous vein graft to the left anterior descending coronary artery (LAD) 16 years before was admitted for further evaluation after an episode of congestive heart failure without myocardial infarction, new onset atrial

A, B

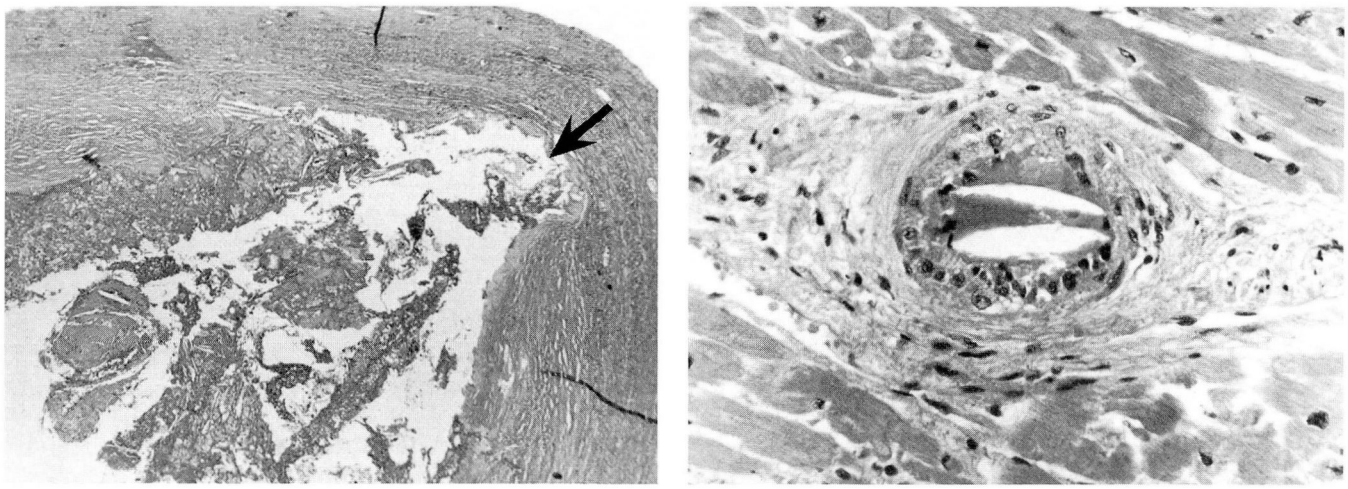

FIGURE 2A. Patient 1. Split in fibrotic intima of saphenous vein graft induced at angioplasty (arrow). The lumen of the graft is filled with atheromatous material from the ruptured plaque. FIGURE 2B. An atheromatous embolus containing cholesterol clefts occludes a small coronary artery.

fibrillation, and pneumonia. Recent cardiac catheterization showed stenosis of the vein graft (*Figure 1*) and severely impaired left ventricular function. Despite maximal medical therapy with nitrates and calcium channel blockers, the patient continued to have angina at rest, necessitating repeated admission to his local hospital.

The patient was refused for reoperation; subsequently, he underwent angioplasty of the distal vein graft–LAD anastomotic site. During the procedure, the proximal portion of the vein graft closed, possibly from guiding catheter dissection. This was re-opened with PTCA. Because of chest pain and hypotension, intra-aortic balloon pumping was begun during the procedure. Cardiac enzymes became elevated, with peak creatine kinase of 440 IU/L with 20% MB fraction; congestive heart failure developed and the patient died one week after angioplasty.

At autopsy there was diffuse atherosclerosis with stenosis of the saphenous vein graft–LAD anastomosis and stenosis of the distal LAD. Microscopically, atherosclerotic plaques in both the proximal and distal vein graft were ruptured with hemorrhage and dissection in the plaques and overlying thrombus. The fibrotic intima showed a characteristic angioplasty split (*Figure 2A*). The LAD distal to the graft and small coronary arteries at the left ventricular apex contained occlusive atheromatous emboli (*Figure 2B*). Multifocal subendocardial infarctions were present. Both the emboli and infarcts were of varying ages, recent and organizing.

*Patient 2* A 61-year-old white man with hypertension and hypercholesterolemia had undergone saphenous vein grafting to the LAD and left internal mammary artery implant in 1971, repeat vein graft to the LAD and vein graft to the circumflex artery in 1981, and unsuccessful angioplasty of the native circumflex artery in 1986. He returned one year later because of progressive rest angina and dyspnea on exertion. Cardiac catheterization showed stenosis of 50% in the LAD graft and 80% in the portion of the graft proximal to the circumflex artery (*Figure 3*). Left ventricular function was markedly impaired.

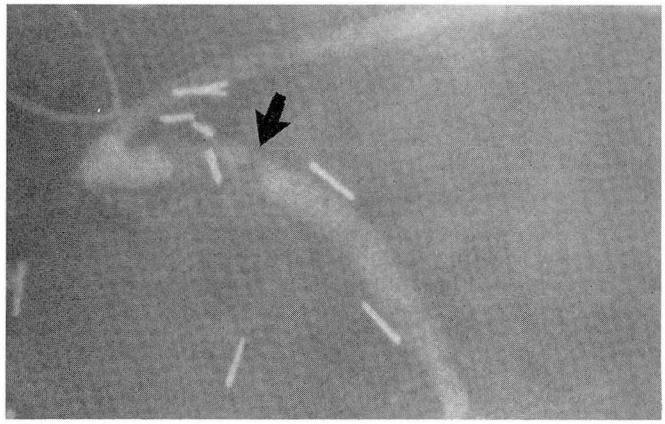

FIGURE 3. Patient 2. Severe proximal stenosis of the saphenous vein graft to the circumflex coronary artery (arrow).

Two cardiac surgeons refused to perform a third bypass procedure. Because of recurrent angina, angioplasty of the vein graft to the circumflex was performed.

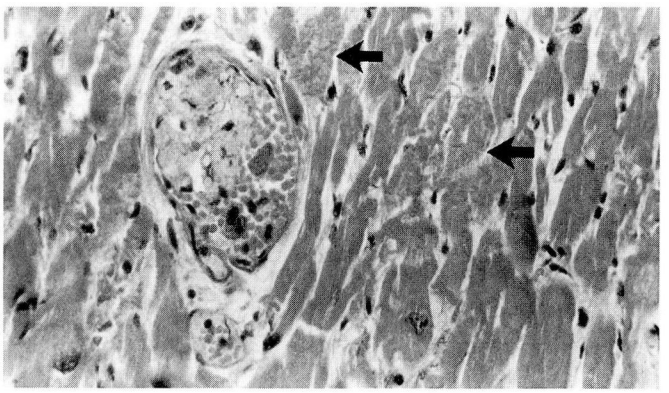

**FIGURE 4. Patient 2. An organizing atheromatous embolus partially occludes an artery. Focal contraction band necrosis is present in adjacent myocardium (arrows).**

Though dilatation was successful, embolization of atheroma to the distal native vessel was noted during the procedure. Creatine kinase peaked at 1420 IU/L with 12% MB fraction. Despite inotropic and intra-aortic balloon pump support, the patient died four days after angioplasty because of progressive left ventricular failure. At autopsy, the midportion of the graft to the circumflex was totally occluded by ruptured atheromatous plaque and secondary thrombus. In the distribution of the circumflex there were atheroemboli and multiple myocardial infarcts varying in age from 24 hours to 10 days (*Figure 4*). Thus, there were areas of infarction that clearly antedated the PTCA.

## DISCUSSION

Balloon angioplasty is less invasive than reoperation, but often is the only alternative for patients with stenotic saphenous vein grafts who are poor candidates for reoperation. Embolization of atheroma at the time of PTCA is a recognized and potentially fatal complication of older, stenotic saphenous vein graft angioplasties.[7] In our series of 98 patients, five (5.1%) had myocardial infarctions directly related to the angioplasty. In two patients, this complication was fatal. In four of the five patients, atheroembolization was suspected at angioplasty or documented at autopsy. The cause of infarction in the other patient (patient 4) may have been spasm or emboli that were too small to be detected angiographically.

In the two fatal cases, atherosclerotic embolism to the distal epicardial artery was clearly documented at autopsy. In addition, multiple atherosclerotic emboli and myocardial infarctions of varying ages were present. These findings suggest that in older saphenous vein grafts, there may be progressive fragmentation of atheroma, leading to recurrent microscopic embolism and progressive myocardial cell loss over several days, unrelated to any angioplasty procedure. The atherosclerotic lesion may also be a nidus for thrombus formation and recurrent embolization of thrombus fragments.[8,9] These embolic events may cause recurrent angina and sustained elevation of serum creatine kinase and may produce multiple myocardial infarctions of varying ages, as seen in our two cases. In one of our patients (patient 2), persistent enzyme elevation over three days suggested continuing infarction because of recurrent embolization; however, severe left ventricular dysfunction could have contributed to the continuing ischemia.

In conclusion, in patients with poor left ventricular function and late vein graft atherosclerosis, balloon angioplasty may result in potentially fatal myocardial infarction from thrombotic and atherosclerotic emboli. In addition, autopsy findings in our two fatal cases confirm that progressive fragmentation of atheroma of these older vein grafts may also occur unrelated to any invasive procedure.

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