Rapidly Progressive Coronary Artery Bypass Graft Atherosclerosis*

Report of a Case Documented by Serial Coronary Arteriograms and Pathologic Examination

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Atherosclerosis occurred in a coronary artery saphenous vein bypass graft. Serial angiograms are presented documenting subtotal occlusion of this graft which occurred over a nine-month period. Pathologic examination of graft material obtained at surgery demonstrated ulcerating atherosclerotic plaque. The occurrence of early saphenous vein graft atherosclerosis is discussed with particular reference to the possible effects of hyperlipidemia, which was present in this patient.

Coronary artery bypass surgery with autologous saphenous vein grafts has been employed in the treatment of obstructive coronary atherosclerosis and its resultant symptoms since 1967. The efficacy of this procedure in relieving severe angina pectoris has been clearly demonstrated, but the uncertain ultimate fate of the implanted grafts is an important consideration in assessing other possible indications for this operation.

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Pathologic studies of aortocoronary saphenous vein grafts have revealed intimal proliferation, sometimes to the point of occlusion, in grafts implanted for less than one year, but early atherosclerotic occlusion is rare. We report herein a case of histologically-proven atherosclerotic subtotal occlusion of a saphenous vein coronary artery bypass graft with angiographic documentation that this progressed over a ninth-month interval. Though early occurrence of atherosclerotic lesions in these grafts has been previously noted in several patients, to our knowledge, this report provides the first example of sequential angiography with confirmatory pathologic findings.

CASE REPORT

The patient was a 61-year-old white male farmer with a 30-year history of adult-onset diabetes mellitus treated with an orally administered agent and insulin, moderate hypertension controlled by diuretics and alpha-methylidopa, and combined hyperlipidemia (cholesterol 474 mg/100 ml, triglycerides 582 mg/100 ml) treated with diet and clofibrate. He began to experience exertional angina pectoris in 1976. As a result of worsening of his symptoms, he underwent cardiac catheterization in January 1978. Significant lesions were found in the right and left anterior descending coronary arteries and the major marginal branch of the circumflex. Left ventricular function was normal (ejection fraction = 70 percent). The same month, he underwent coronary artery bypass grafting to the left anterior descending, first diagonal and obtuse marginal arteries, with an uncomplicated postoperative course.

After discharge, he was asymptomatic until April 1978, when he noted recurrence of angina with exertion. Medical treatment was reinstated, but his symptoms continued and he was recatheterized in August 1978. At that time, the left anterior descending (Fig 1) and diagonal grafts were free of lesions, but the graft to the obtuse marginal had a high grade stenosis due to kinking of the graft at the distal anastomotic site. He was discharged on medical therapy and continued to have moderately severe stable angina.

In May 1979, his symptoms worsened. Despite hospitalization and maximal medical therapy, he continued to have
frequent chest pain with ischemic electrocardiographic changes in the anterolateral leads. Catheterization in May, 1979, again showed the tight lesion at the distal anastomotic site of the obtuse marginal graft, a patent graft to the diagonal artery, and a new 90 percent stenotic lesion in the mid-left anterior descending graft (Fig 2), which had appeared since August 1978.

He was taken to the operating room the same day, where a 2-cm high grade, ulcerating lesion was visualized in the proximal portion of the left anterior descending graft. This was excised, and a segment of vein was interposed. In addition, a new saphenous vein graft was implanted distal to the anastomotic narrowing of the obtuse marginal vessel. Although the patient was weaned from cardiopulmonary bypass without inotropic support and was subsequently hemodynamically stable, he suddenly developed electromechanical dissociation two hours later. Despite extensive resuscitative efforts and open cardiac massage, the patient died.

At autopsy, the heart weighed 580 g and exhibited marked left ventricular hypertrophy. An organized, nontransmural myocardial infarction was found in the posterior-septal left ventricle. The native coronary arteries exhibited severe calcific atherosclerosis. The resected portion of the left anterior descending graft was almost totally occluded by atheromatous material. Microscopically, the vein grafts exhibited marked intimal thickening, composed of smooth muscle cells, abundant collagen, and elastic fibers. Atherosclerotic fibrous plaques were present throughout the grafts, with microscopic findings of foam cells, cholesterol clefts, and calcium deposits within the thickened intima. No pathologic cause for the terminal event was found.

**Discussion**

The most striking feature of this case is the documented, rapid progression of severe atherosclerotic disease in a saphenous vein graft. The left anterior descending graft appeared angiographically normal seven months postoperatively, although some degree of intimal proliferation was almost certainly present at that time. Just nine months later, this graft was nearly totally occluded. The timing of the rapid worsening of symptoms which led to his second operation and the location of the electrocardiographic changes during pain suggest that this lesion was the cause of his deterioration. Pathologic examination confirmed its atherosclerotic nature. Of particular note in this patient is his large number of risk factors for atherosclerotic disease, and especially, his uncontrolled hyperlipidemia.

The classic lipid-containing fibrous intimal plaque of atherosclerosis occurs rarely in veins transplanted to the arterial circulation. The long history of vein graft bypass surgery for peripheral vascular disease suggests that vein implants are relatively resistant to atherosclerosis despite exposure to arterial pressure. On the other hand, intimal proliferation occurs rapidly and appears to be a universal phenomenon.

In 1973, Vlodaver and Edwards reported on 53 patients with 88 grafts, describing the intimal proliferative lesion, which had resulted in thrombosis in some cases, but did not note any atherosclerotic fibrous plaque. This description was followed by many other reports (Table 1), which generally showed a high frequency of intimal proliferation with a low incidence of intimal fibrous plaque. In 1974, Barborigo et al described six cases of unequivocal atherosclerotic plaque in aortocoronary vein grafts, but only one of these occurred in less than one year, and it did not contribute directly to the death of the patient. In fact, it was their conclusion that vein graft atherosclerosis does not pose an immediate threat to long-term survival. Kennedy et al, in reviewing 108 patients, mention one graft which exhibited atherosclerotic occlusion eight months after surgery. In 1977, Lie et al failed to find the fibrous plaque of atherosclerosis in any graft prior to 12 months after implantation but did note that hyperlipidemia...
Table 1—Pathologic Studies of Saphenous Vein Coronary Artery Grafts

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No. of Patients</th>
<th>No. of Grafts</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vlodaver and Edwards</td>
<td>1979</td>
<td>53</td>
<td>86</td>
<td>No fibrous plaque*</td>
</tr>
<tr>
<td>Vlodaver and Edwards</td>
<td>1973</td>
<td>21</td>
<td>29</td>
<td>No fibrous plaque</td>
</tr>
<tr>
<td>Unni et al</td>
<td>1974</td>
<td>40</td>
<td>62</td>
<td>No fibrous plaque</td>
</tr>
<tr>
<td>Barboriak et al</td>
<td>1974</td>
<td>33</td>
<td>...</td>
<td>6 Cases of fibrous plaque, 1 early†</td>
</tr>
<tr>
<td>Kennedy et al</td>
<td>1974</td>
<td>108</td>
<td>...</td>
<td>1 Case fibrous plaque</td>
</tr>
<tr>
<td>Lawrie et al</td>
<td>1976</td>
<td>41</td>
<td>78</td>
<td>No fibrous plaque</td>
</tr>
<tr>
<td>Bulkley and Hutchins</td>
<td>1976</td>
<td>55</td>
<td>97</td>
<td>No fibrous atherosclerotic plaque.</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>High incidence of concentric intimal hyperplasia frequently with infiltrating lipids in older grafts.</td>
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<tr>
<td>Lie et al</td>
<td>1977</td>
<td>55</td>
<td>99</td>
<td>14 Grafts with fibrous plaque, none early</td>
</tr>
<tr>
<td>Klima et al</td>
<td>1979</td>
<td>109</td>
<td>168</td>
<td>29 Grafts with fibrous plaque, 1 early</td>
</tr>
</tbody>
</table>

*Intimal fibrous plaque of atherosclerosis.
†Early indicates occurring in the first 12 months after implantation.

seemed to significantly increase the degree of both intimal proliferation and the extent of atherosclerosis, a phenomenon also noted by Barboriak et al.1-17 and an observation for which there is ample experimental basis.18-20

Thus, the early occurrence of fibrous plaque with classic histologic features of atherosclerosis in aortocoronary vein grafts appears to be uncommon, while fibrous intimal proliferation is almost universal. The pathologic features of each lesion bear review, since at first glance, these two processes seem to be relatively distinct entities. Microscopically, the concentric intimal fibrosis universally seen in these grafted veins consists of smooth muscle cells, abundant collagen, and elastin, with lipid stains that are usually negative.5,4,10 Conversely, the intimal fibrous plaque of classic atherosclerosis is a focal, eccentrically-placed lesion composed of lipid-laden smooth muscle cells, abundant collagen and elastin, and collections of extracellular lipid and cellular debris.21

In arteries, fibrous intimal thickening appears to be a precursor of the fully-developed fibrous atherosclerotic plaque.22 However, little is known about the development of atherosclerosis in saphenous vein grafts. One possibility is that the intimal proliferation commonly seen in vein grafts is a precursor of more severe atherosclerotic lesions, as appears to be the case in the arterial circulation.4,10 In most patients, there may be a long latency period before more advanced atherosclerotic changes take place, just as there is in the native arteries. This would explain the infrequency with which severe atherosclerosis has been reported in arteriographic and pathologic studies thus far. It would also not be surprising if factors such as hyperlipidemia, which predisposes patients to severe atherosclerosis of their native circulation, accelerate its appearance in vein grafts.

Certainly, the present case demonstrates the rapidity with which atherosclerotic graft occlusion may occur and suggests the need for continued vigorous treatment or modification of these risk factors after coronary artery revascularization. This case also raises the question of the long-term fate of bypass grafts, particularly if the common phenomenon of intimal proliferation is a precursor of atherosclerosis. If subsequent studies prove this to be the case, the recent demonstration that administration of platelet-inhibiting drugs reduces the degree of early intimal thickening in canine bypass grafts22 may take on added clinical significance.

REFERENCES


Traumatic Rupture of the Thoracic Aorta during Pregnancy*

Surgical Considerations

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This is a report of successful management of a traumatic rupture of the thoracic aorta in pregnancy. Both mother and daughter are alive and well 24 months after the injury. The various aspects of the surgical and supportive treatment are discussed.

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Traumatic rupture of the thoracic aorta indicates a major physical insult, and survival depends on early diagnosis and successful repair. Once this is accomplished, the fate of the patient is determined in part by the severity of associated injuries. Accurate assessment and meticulous management are essential for a successful outcome. A case of traumatic rupture of the thoracic aorta in pregnancy associated with fracture of the femur and the zygoma is described to illustrate this point.

CASE REPORT

A 27-year-old woman in the sixth month of her first pregnancy was admitted to another hospital following an accident on the road. On admission, her blood pressure was 80/60 mm Hg, with a pulse rate of 130 beats per minute. Fractures of the right femur and the right zygoma were diagnosed. The chest x-ray film showed some widening of the mediastinum, contusion of the left lung, and fracture of the seventh left rib. The patient developed respiratory failure, and assisted ventilation with positive end-expiratory pressure of 6 mm Hg was instituted. External fixation of the right thigh was performed.

On the sixth day of hospitalization, the patient developed a sudden pain in the chest accompanied by diminution of the femoral pulses, and a bruit was heard over the right subclavian artery. A repeated chest x-ray film showed further widening of the mediastinum. An aortogram confirmed the diagnosis of traumatic rupture of the thoracic aorta. The patient was transferred to our hospital for surgical treatment. On admission the patient was hemodynamically stable, with moderate respiratory insufficiency. Prior to surgery, 100 mg of progesterone was given intravenously. In order to ensure blood supply to the fetus, we elected to use partial cardiopulmonary bypass at the time of aortic cross-clamping. The right femoral vessels were therefore exposed and cannulated. Partial bypass with a flow of 2 L/min was instituted and continued for 49 minutes, while the ruptured descending aorta was repaired by interposing a 20-mm Dacron tube graft. After surgery, fetal monitoring was performed continuously, and additional doses of progesterone (100 mg every eight hours) were given. No change in fetal pulse rate was noted throughout. The patient needed additional respiratory support initially. During recovery internal fixation of the fractured right femur was performed, and the zygoma was repositioned.

The patient was discharged three weeks following repair, to be readmitted electively for a cesarean section two months later. She gave birth to a normal, 3,200-gm full-term girl. Follow-up examination 24 months later showed both mother and daughter to be in excellent health. The baby is physically and psychomotorally in the 90th percentile for her age.

DISCUSSION

Eighty percent of the patients who suffer traumatic rupture of the aorta die before arrival at the hospital. Increased awareness of this condition, together with faster evacuation and wider surgical experience, should reduce the mortality in survivors of the initial impact. The intraoperative management of traumatic rupture of the aorta may include the use of an aorto-aortic shunt, left-atrial-to-femoral bypass, femoral vein-femoral artery bypass, or no provisions for perfusion of the lower half of the body at all. Zitnik* reported in 1968 that the