Progressive and Late Obstruction of an Aorto-Coronary Venous Bypass Graft

By Claude M. Grondin, M.D., Claude Meere, M.D., Yves Castonguay, M.D., Gilles Lepage, M.D., and Pierre Grondin, M.D.

SUMMARY
Progressive late obstruction of an aorto-coronary venous bypass graft is reported in a 44-year-old man who died of bi-ventricular failure 114 days after surgery. The first portion of the vein graft had a residual lumen of one mm. Histology showed marked intimal fibrosis. Mechanism for this occlusion and fibrosis is unclear. This represents the first documented case of late obstruction of an aorto-coronary bypass graft with angiographic evidence of early postoperative patency.

Additional Indexing Words:
Acquired coronary artery disease
Surgery for coronary artery disease
Postoperative angiographic evaluation

Aorto-coronary bypass grafts have gained an accepted place in the surgical treatment of atherosclerotic coronary artery disease.1-4 Similar use of the long saphenous vein for bypass in peripheral arterial surgery has proved satisfactory in long-term follow-up.5, 6 Experimental and clinical evidence of preservation of the histological structure of the long saphenous vein used in femoro-popliteal bypass graft has been established.7, 8 However, long-term follow-up of vein grafts used for aorto-coronary bypass is not available at present.

The purpose of this communication is to report the case of a patient who died 114 days after aorto-coronary bypass graft to the right and anterior descending coronary arteries. His vein graft—initially shown to be widely patent on selective postoperative angiography—became progressively occluded by endoluminal fibrous thickening.

Case History
A 44-year-old white male was admitted to the Montreal Heart Institute in May 1970 for acute pulmonary edema. The patient had a long history of angina pectoris and had complained of shortness of breath on exertion for more than a year and a half. He had been hospitalized 2 months prior to this admission for his second episode of acute myocardial infarction. During that hospitalization he presented congestive heart failure and a clinical episode compatible with pulmonary embolism. He was given anticoagulants, diuretics, and digitalis.

The electrocardiogram taken on admission in May showed occasional premature ventricular contractions, evidence of old inferior and anterior wall myocardial infarctions and left atrial enlargement. Following appropriate treatment of the acute left ventricular failure, the patient underwent further studies.

Cardiac fluoroscopy showed cardiomegaly and evidence of pulmonary venous hypertension. Left heart catheterization revealed a pressure of 95/60 mm Hg in the ascending aorta and 95/8 mm Hg in the left ventricle. The end-diastolic pressure in the left ventricle rose from 18 to 30 mm Hg following left ventriculography. Selective coronary arteriography, by a method previously described,9 showed two areas of narrowing of more than 80% of the lumen in the right coronary artery immediately proximal and distal to the marginal branch. Stenosis of 80% of the lumen of the left anterior descending artery just distal to
the first septal branch was also noted. The circumflex and diagonal arteries were free of significant stenosis. Left ventriculogram showed marked akinesia of the distal two-thirds of the anterior wall and of the inferior wall. The patient's serum cholesterol, total lipids, and triglycerides were within normal limits.

The patient underwent a double aorto-coronary saphenous vein graft to the right and to the left anterior descending coronary arteries. Blood flow was 55 ml/min "at rest" in the graft to the right coronary artery and rose to 90 ml/min following injection of 20 mg of papaverine into the graft. The vein graft to the left anterior descending artery was anastomosed proximally to the aorta, and the graft to the right coronary artery was anastomosed end-to-side to the vein graft to the left anterior descending artery. Blood flow in the graft to the left anterior descending artery was 20 ml/min and rose to 70 ml/min following papaverine injection. The left anterior descending artery measured 1.5 mm just distal to the site of anastomosis and the right coronary artery, 2 mm. No difficulties were encountered during surgery or in the immediate postoperative period.

Diuretics and digitalis were started again on the seventh postoperative day when the patient gained weight and presented ankle edema and moderate dyspnea. Postoperative electrocardiogram showed no change when compared to the preoperative tracing. On the fourteenth postoperative day cardiac catheterization studies were performed to evaluate patency of the vein grafts. Pressure in the aorta was 95/50 mm Hg and pressure in the left ventricle was 95/0 mm Hg. There was no improvement in the left ventricular akinesia noted prior to surgery. After ventriculography, the left ventricular end-diastolic pressure remained elevated at 35 mm Hg. The graft to the right coronary artery was widely patent with excellent filling of the distal coronary tree (fig. 1). The graft to the left anterior descending artery was occluded. It was elected not to re-explore and attempt to reestablish blood flow through the occluded graft because of the low flow recorded in the graft at the time of surgery.

The patient was discharged on the twentieth postoperative day still taking digitalis, diuretics, and anticoagulants. He was readmitted 12 days later with fever, cough, and right chest pain. Chest X-rays showed plate-like atelectasis of the right base. Symptoms abated and the patient was discharged 4 days later. He had had one episode of retrosternal pain which was relieved by nitroglycerin the day before admission. The electrocardiogram did not show any change. Sputum and blood cultures were negative.

He was readmitted 1 month later in acute left ventricular failure. Despite intensive medical treatment his condition worsened, and he died 2 weeks later of bi-ventricular failure.

Figure 1

Postoperative cineangiogram showing direct injection into the graft (upper left) to the right coronary artery and excellent filling of the distal coronary tree (lower right).
Figure 2

Segments of the vein graft showing marked narrowing of the lumen in the first portion of the graft (right) in comparison to the "normal" lumen-wall ratio of the distal segment of the vein graft (left).

Figure 3

Histological section of the vein graft in the area of stenosis showing marked fibrosis of all layers, but predominantly of the intimal layer (Int.). M = media; A = adventitia. Hematoxylin and eosin; × 65.

Postmortem examination revealed, in addition to marked mediastinal and pericardial adhesions, dilatation of all four cardiac chambers and complete occlusion of both aorto-coronary grafts. The occlusion of the graft to the left anterior descending artery was old but the occlusion of the graft to the right coronary artery was recent. There was marked narrowing, beginning immediately at the take-off of the vertical limb of the Y-graft, on the aorta down to the bifurcation of the
AORTO-CORONARY VENOUS BYPASS GRAFT

graft, with a residual lumen of 1 mm (fig. 2). The narrowing tapered off in the first portion of the graft to the right coronary artery. The lumen of the graft from that point to the right coronary artery was normal in size although occluded by a fresh thrombus.

On histological sections, the intima was markedly thickened by dense fibrous tissue. There was also thickening, but to a lesser extent, of the media and adventitia (fig. 3). The muscle and elastic layers of the media were preserved, although in some areas the fibers were fragmented and replaced by fibrous tissue. Section of the left ventricular myocardium showed extensive fibrosis. There was no evidence of recent myocardial infarction or of pulmonary or systemic embolization. The pulmonary arteries showed evidence of hypertension and arteriosclerosis. The cause of death was believed to be chronic left and right ventricular failure as well as occlusion of both aorto-coronary bypass grafts.

Discussion

Vein grafts subjected to arterial pressure and flow get thicker with time and ultimately take the gross appearance of arteries. Unlike other veins, the saphenous vein does not dilate or develop aneurysm. It may, however, dilate when utilized as a patch graft, in larger arteries such as the thoracic aorta or the femoral artery. In this situation, according to Laplace’s Law, the distending pressure, which is directly related to the diameter of the artery, may overcome the elasticity of the vein and cause rupture or aneurysm formation. The small diameter of the saphenous vein probably does not allow the development of distending pressures of such magnitude. In addition, as pointed out by Linton, the long saphenous vein has already been subjected to high internal pressures prior to its utilization as a bypass conduit.

Long-term follow-up of saphenous veins utilized in femoro-popliteal bypass grafts has shown that, while all layers of the vein thicken, the internal diameter remains relatively constant, and late occlusion is more a consequence of progression of the disease in the distal arterial tree than of local changes in the vein itself.

On the other hand early occlusion of femoro-popliteal or aorto-coronary vein grafts may be secondary to local factors such as the size of anastomosis, length of vein, and twist or trauma to the vein. Blood flow through these vein grafts may be critical in the initial phase, although correlation of intraoperative blood flow measurements and postoperative patency has been the subject of controversy. In our own series of aorto-coronary bypass grafts, grafts with flow greater than 45 ml/min remained open, and grafts with flow of 20 ml/min or less usually became occluded. In the present case, one graft carried a flow of 20 ml/min and was found to be occluded in the early postoperative angiographic evaluation. The graft to the right coronary artery carried a flow of 55 ml/min and remained patent.

Long-term follow-up has shown that saphenous veins utilized in femoro-popliteal bypass retain their histological structure. In the experimental animal, Jones demonstrated fibrous thickening of all three layers of patent vein grafts and found little difference between early and late changes occurring from 39 to 790 days after femoro-popliteal bypass. In a clinical study of late results of femoro-popliteal bypass grafts, DeWeese found that occluded grafts differed histologically from patent grafts by marked fibrosis and thickening of the intima and adventitia.

In the present case the mechanism for progressive occlusion and intimal fibrosis of the vein graft is not clear. Although marked scarring was noted in the mediastinum at autopsy, fibrosis of the vein was mainly limited to the intimal layer. Obstruction did not appear to be secondary to perivenous fibrosis. The relative lack of intimal fibrosis in the distal half of the graft cannot be easily explained either.

It is possible that the intimal fibrosis in this case represents a local reaction of the vein to pressure and turbulence—a variant form of jet lesions such as is seen in aortic valvular stenosis.

The aortic valve was normal in this patient but the anastomosis on the right lateral aspect of the ascending aorta may have been located precisely at the point of maximum impact of the systolic ejection of blood through the
aortic valve. The lack of intimal lesion in the normal aorta at that point does not rule out the hypothesis of a modified jet lesion since the intima of a transplanted vein and that of the ascending aorta may respond differently to stress. This phenomenon should occur more frequently, however, since anastomosis of grafts to the right coronary artery are usually made at that point on the ascending aorta. Intimal fibrosis in this instance would probably be limited to the ostium and not extend several centimeters into the vein as was seen in this case.

References


Progressive and Late Obstruction of an Aorto-Coronary Venous Bypass Graft
CLAUDE M. GRONDIN, CLAUDE MEERE, YVES CASTONGUAY, GILLES LEPAGE and PIERRE GRONDIN

Circulation. 1971;43:698-702
doi: 10.1161/01.CIR.43.5.698
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/43/5/698

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/