Case reports

Rupture of a saphenous vein bypass graft during coronary angioplasty

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SUMMARY A self-limited rupture of a five month old saphenous vein bypass graft occurred during coronary angioplasty when the balloon used to stretch the vessel proved to be too large. A lack of fibrous tissue in the new graft may have predisposed it to rupture.

Percutaneous transluminal coronary angioplasty has been used to treat obstructions of the saphenous vein graft anastomosis or of the vein body itself.¹⁻⁵ Rupture and perforation of coronary arteries during angioplasty is rare.⁶⁻¹¹ Rupture or perforation of saphenous vein bypass grafts has not been described. We report a case of rupture of a saphenous vein bypass graft during percutaneous transluminal coronary angioplasty.

Case report

This 47 year old hypertensive man had first complained of angina pectoris five years before percutaneous transluminal coronary angioplasty. The angina was progressive and eventually chest pain at rest developed. This was relieved by nitrates. The patient then underwent coronary artery bypass surgery to the left anterior descending artery, right coronary artery, and circumflex coronary artery. He did well until angina recurred a month before the present admission. Initially, he only had angina with moderate exertion, but this progressed to episodes during minimal exertion and at rest. Repeat cardiac catheterisation demonstrated that the grafts to the circumflex and right coronary arteries were widely patent. The saphenous vein graft to the left anterior descending artery had a long segment of smooth narrowing of 95-99% with slow antegrade filling of the distal native left anterior descending artery (fig 1).

Angioplasty was attempted with a 9 French El Gamal guiding catheter and a 4 mm USCI low profile balloon (United States Catheter and Instrument Corporation, Billerica, MA). The balloon was

passed over a 0.018 inch high torque floppy ACS guide wire (Advanced Cardiovascular Systems, Mountain View, CA). There was no difficulty negotiating the obstruction and in passing the guide wire to the distal portion of the left anterior descending coronary artery. The balloon was then inflated four times to a maximum pressure of 8 atmospheres for a maximum of 60 seconds. At 8 atmospheres, the ratio of the inflated balloon to the distal artery was 1.22/1, and the balloon slightly overdilated the distal vein. During balloon inflations the patient had transient angina pectoris associated with 1 mm ST segment depression in lead V5. The initial transstenotic gradient across the lesion was 60 mm Hg and this was reduced to 4 mm Hg after coronary angioplasty. After the last balloon inflation the patient continued to have mild residual chest discomfort which was unlike his angina pectoris. This was not associated with any ST segment changes on his electrocardiogram. After removal of the balloon, angiograms revealed that the area of the saphenous vein graft obstruction was widely patent with slight residual narrowing. There was extravasation of contrast material outside the wall of the vein, however, which continued proximally in a linear fashion along the vein in a sinus tract (fig 2). Repeat fluoroscopic examination of this area showed that the dye remained in this extraluminal area for the 45 minute observation period in the laboratory. The patient's blood pressure and heart rate remained stable and a Swan-Ganz catheter was inserted to monitor haemodynamic variables: initial pulmonary artery pressure 16/6 mm Hg (mean 8 mm Hg); mean pulmonary capillary wedge pressure 2 mm Hg; and mean right atrial pressure 1 mm Hg. The patient was transferred to the cardiac care unit for continued monitoring. The patient did well and had no appreciable increase in his right heart pressures and no

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Fig 1 Angiogram in the left anterior oblique projection with craniocaudal angulation showing saphenous vein graft to the left anterior descending coronary artery. There was severe narrowing in the vein just before the distal anastomosis and slow anterograde filling of the distal left anterior descending coronary artery.

important change in vital signs. Repeat fluoroscopy the next day did not show residual contrast material at the area of the extravasation. At no time did any signs of cardiac tamponade develop. The patient's non-anginal chest pain resolved approximately two to three hours after the procedure and he was discharged from the hospital on the third hospital day. Four months after angioplasty he is active and free of angina.

Discussion

The most common coronary vascular complications of angioplasty are dissection, acute occlusion, and spasm. A recent series of patients indicates that this occurs in approximately 5% of cases.^{6,8} Perforation of the native coronary arteries is rare, but it has been described in several case reports.^{7,9-11} Perforation was usually caused by passage of a guide wire into a false channel and this can cause cardiac tamponade

or may not have any adverse effects. Rupture of the native coronary artery after safe passage of the balloon is rare. It has been reported twice.^{7,9} In both cases rupture of the coronary artery was thought to have been caused by the use of a balloon that was too large for the size of the artery. When a small diagonal vessel was affected the clinical course was not life threatening,⁹ but balloon rupture of a dominant right coronary artery resulted in pericardial tamponade, progressive hypotension, and death.

In our case a five month old saphenous vein graft was ruptured. Rupture was self-limited and had no adverse outcome. The ratio of the inflated balloon diameter to the vessel diameter was 1.22/1. The diameter of the inflated balloon was not too large for the size of the distal artery or for dilatation of native arteries.¹² Dorros has proposed the use of 4.2 and 5.0 mm balloons in saphenous vein angioplasty to reduce the recurrence rate.¹³ This vein graft had only been in place for five months, however. It is

Fig2 Angiogram in the left anterior oblique projection with craniocaudal angulation obtained after angioplasty. There was slight residual narrowing in the area of obstruction and extravasation of dye outside the wall of the vein, which appeared to travel along the vein in a sinus tract.

known that fibrosis of the walls of a vein graft takes time to develop.¹⁴ The longer the vein graft is in place, the greater the replacement of smooth muscle cells by fibrous elements in the media and adventitia. If fibrosis of the vein has not had time to develop, the vein may be more susceptible to rupture if a large balloon is used.

The presence of chest pain and the absence of extensive haemorrhage into the chest are two other interesting aspects of this case. The constancy of the pain in the absence of electrocardiographic changes suggests that it originated in the pericardium or arterial wall. Pericarditic chest pain sometimes follows angioplasty.¹⁵ Even in the absence of arterial perforation, patients may show arterial haemorrhage over the dilated native arteries at subsequent operation for elective or emergency bypass surgery. Presumably, this haemorrhage can cause pericardial irritation and pericarditic chest pain.

The patient showed no signs of pericardial tam-

ponade or extensive blood loss despite clear rupture of the vessel. The dye appeared to be contained in a tissue sac surrounding the vein. This presumably was connective tissue; such tissue is often seen at reoperation, densely adhering to the vein structure. In this patient it must have prevented tamponade or serious intrathoracic haemorrhage.

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