Extraluminal compression of an aortic graft simulating recoarctation

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SUMMARY Restenosis rarely develops after surgical correction of coarctation of the aorta in adults. Late morbidity is usually related to residual hypertension or progressive aortic valve disease. A patient in whom symptoms and signs of recurrent coarctation developed 19 years after initial graft repair is described. Dehiscence of the original silk suture line was found at operation. Extensive thrombus had produced graft compression. Milder hypertension persisted in the postoperative period despite relief of the aortic obstruction.

Late morbidity after repair of coarctation of the aorta in adulthood is usually caused by residual hypertension, development of coronary atherosclerosis, aneurysm formation, stroke, aortic valve disease, or congestive heart failure.¹⁻³ Recoarctation rarely develops when the initial operation is performed in adulthood, but stenosis may develop when the enlarging aorta outgrows a synthetic graft inserted earlier in life. We encountered a case in which apparent recoarctation developed as a result of dehiscence of the original suture line 19 years after initial repair. Extensive thrombus formation resulted in graft compression.

Case report

A 38 year old white woman presented with hypertension and claudication in the legs 19 years after surgery for repair of coarctation of the aorta. She had presented initially with similar findings, and angiography had identified a juxtaductal aortic coarctation beyond the left subclavian artery with preserved distal flow and associated ductus arteriosus. During the original operation the ductus arteriosus was ligated, a long coarctation was resected, and a 14 mm Dacron

graft was inserted with a silk suture. The patient did well in the postoperative period; blood pressure became normal, claudication remitted, and she enjoyed an active lifestyle and completed two full term pregnancies without difficulty. At 38 she experienced fatigue and dyspnoea and claudication of the calves, thighs, hips, and buttocks on both sides. She was admitted to hospital with congestive heart failure and the blood pressure was then 230/112 mm Hg. Congestion and hypertension responded to administration of diuretic and vasodilator medications. At readmission her medications included metolazone (10 mg daily) and clonidine (0.1 mg three times a day); she had stopped using aspirin nine months before.

Physical examination found the patient in no distress at rest and well developed with a full, but not webbed, neck. The supine blood pressure was 168/90 mm Hg in the left arm and 180/95 in the right. The heart rate was 60 beats/min with occasional extrasystoles. The lung fields were clear. A systolic murmur was heard over the left chest posteriorly and radiated toward the midline and left renal fossa. Anteriorly, the murmur was heard over the left pectoral and manubrial areas. There was no jugular venous distension and the carotid arterial pulse was normal. An apical mid-systolic murmur and click were heard but no diastolic murmurs were heard. Examination of the abdomen showed no organ enlargement. The femoral pulse and more distal pulses of the legs were delayed and markedly diminished in volume. Systolic pressure at the ankles, registered with the Doppler ultrasound device, was

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75 mm Hg at rest while supine and 70 mm Hg after exercise.

The electrocardiogram displayed normal sinus rhythm and large voltages characteristic of left ventricular hypertrophy. Chest x ray showed normal cardiac dimensions and pulmonary vasculature but there was evidence of mild rib notching. Cross sectional echocardiography showed normal chamber

dimensions. The aortic valve was not well visualised; however, intracardiac Doppler analysis demonstrated aortic insufficiency.

CARDIAC CATHETERISATION AND ANGIOGRAPHY

Right and left heart catheterisation was performed from the right antecubital approach. The pulmonary artery pressure was 30/14 mm Hg and the left ventricular pressure was 240/16 mm Hg at rest. The cardiac index measured by the thermodilution technique was 3 l/min/m². Angiography showed severe eccentric narrowing of the descending aorta just distal to the origin of the left subclavian artery and a large filling defect at the site of narrowing (fig 1). Three aortic sinuses were identified and aortic insufficiency (grade 2+) was demonstrated.

SURGICAL FINDINGS AND POSTOPERATIVE COURSE

Left thoracotomy was performed through the original incision site at the level of the previously resected fifth rib. The area of original coarctation repair was identified and appeared well healed without evidence of infection. An aneurysmal dilatation approximately 3.5 cm in diameter was found in the region of the proximal anastomosis. The proximal suture line appeared to have dehiscence along half its circumference. Dissection around the graft and extensive thrombus formation had compressed the graft to less than 10% of its potential diameter (fig 2). The distal suture line was intact and there was slight neointimal hyperplasia. The graft was excised and replaced with a new 18 mm preclotted woven Dacron prosthesis and a Prolene suture was used to fashion the end to end anastomosis. Postoperatively, hypertension developed and treatment with intravenous nitroprusside, propranolol, and methyldopa was needed. Eventually an oral regimen of labetalol and diuretic was sufficient to maintain pressure in the normal range and the patient was discharged from the hospital on the eighth postoperative day after an otherwise uneventful recovery. A month after operation she was symptom free, with no dyspnoea or claudication. Blood pressure was well controlled, no murmurs were heard over the thorax, and pulses throughout the legs were strong.

Discussion

Repair of aortic coarctation by synthetic graft replacement has become increasingly common since its introduction in 1960, and the technique was used in approximately 65% of patients in a series reported by DeBakey's group.¹ Features of coarctation calling for graft interposition include (a) involvement of a

Fig 1 Aortic angiogram in the left anterior oblique projection showing severe eccentric narrowing beyond the origin of the left subclavian artery as well as a large filling defect at the site of narrowing (arrow).

Fig 2 Excised segment of aorta (A) and graft (G) showing extensive thrombus (T) compressing the lumen of the graft.

long aortic segment in the coarctation, (b) a small calibre proximal aortic segment, (c) the need for resection of an associated aneurysm, (d) inelasticity of the aorta in older patients, and (e) technical intra-operative complications. In all forms of surgery for relief of coarctation, late morbidity and mortality are usually associated with residual hypertension and take the form of myocardial infarction, congestive heart failure, stroke, and aneurysm formation. Important aortic valve disease may also develop but complications directly related to the graft anastomotic suture line are unusual.

Pennington *et al* reported an experience of the Massachusetts General Hospital with surgical treatment of aortic coarctation (1947–76).⁴ Fifty nine (36%) of 164 patients underwent resection and interposition grafting. Fifty three per cent of patients aged > 10 years and 66% of patients aged > 40 years had interposition grafts. Only one of the late deaths was related to rupture of a pseudoaneurysm at a silk suture line. This was found 17 years after operation. There were no cases of restenosis in patients with synthetic grafts or even of relative stenosis caused by outgrowing the graft.

Of the 190 patients who underwent surgical correction of coarctation of the descending aorta at Baylor, late postoperative mortality occurred in 18 cases.¹ Two thirds of these deaths were attributed to myocardial infarction or heart failure. Fatal rupture of aortic aneurysm occurred in two cases. Late morbidity developed in 14 patients: recurrent coarctation in three, true aneurysm in four, and pseudoaneurysm in one; six patients required aortic valve replacement. Aneurysms generally developed in patients with longstanding hypertension. The data provided mean that we cannot tell which patients were treated with synthetic grafts and which

by primary anastomosis, but the rather low rate of recurrent stenosis was ascribed to the high proportion of cases managed with grafts.

Although resection and primary end to end anastomosis is the procedure of choice for repair of coarctation of the aorta, many patients, especially those undergoing late repair, require interposition of a synthetic graft. After reports of suture line failures with pseudoaneurysm formation⁵ silk has been abandoned as a suture material for graft anastomosis. In patients presenting with signs and symptoms of recoarctation long after initial repair by interposition of a synthetic graft, the differential diagnosis includes coarctation at a second site, stenosis within the graft, or functional stenosis related to further growth of the aorta. Though less common, the possibility of a significant anastomotic complication, such as occurred in our patient, warrants prompt evaluation if catastrophic rupture is to be avoided.

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