Echocardiographic features in spontaneous disruption of implanted tissue aortic valves

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SUMMARY The echocardiographic features were recorded in three patients with spontaneous disruption of implanted tissue aortic valves. Two of the patients had xenografts, and one had a homograft. Aortic regurgitation had developed in all three spontaneously in the absence of active infective endocarditis. In each case there was severe disruption of the valve. Similar echocardiographic features have been found, usually in association with infective endocarditis or with myxomatous degeneration of the aortic valve. They have not previously been reported in association with implanted tissue aortic valves.

Specific M mode echocardiographic features are thought to be typical of a ruptured or flail aortic valve.¹ These features have most often been reported in association with active infective endocarditis.²⁻¹⁰ They have also been found in patients with a floppy or myxomatous aortic valve.^{6,11-15}

We report three patients, two with aortic xenografts and one with a homograft, in whom the typical echocardiographic appearances of a ruptured aortic valve were recorded. In none was the aetiology active infective endocarditis. These features have not previously been associated with implanted tissue valves.

Patients and methods

M mode echocardiography was performed in the three patients using an Ekoline 20 instrument and cross sectional echocardiography using a Varian 3000 phased array ultrasound.

CASE REPORTS

Case 1

At the age of 15 in 1977 this man had his aortic valve replaced with a glutaraldehyde fixed Carpentier-Edwards xenograft, after which he was reviewed annually. There was no clinical evidence of aortic incompetence until February 1981, when at routine review an early diastolic murmur was heard for the first time. He was asymptomatic despite clinically severe aortic incompetence.

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M mode echocardiography showed dilatation of the left ventricle. There was fluttering of both leaflets of the mitral valve, which closed at the normal time. There were fine vibrating echoes in the aortic root, both in systole and diastole, with similar vibrating echoes in the left ventricular outflow tract in diastole (Fig. 1a). Cross sectional echocardiography showed prolapse of the cusp in the non-coronary position into the left ventricular outflow tract (Fig. 1b).

Aortic valve replacement was undertaken in March 1981, without previous angiography. At operation there was a notable loss of tissue from all three cusps of the porcine aortic valve, with calcification of two. There was a tear in one of the cusps, a small piece of which was hanging free.

Case 2

A 33 year old man had an aortic valve replacement with a fresh homograft in July 1971. In October 1971 an early diastolic murmur was first noted. Electrocardiographic evidence of left ventricular hypertrophy developed over the succeeding years. In 1981 he had clinical evidence of severe aortic incompetence. He was asymptomatic.

M mode echocardiography in February 1981 showed that the left ventricle was normal in size. There was fluttering of the anterior leaflet of the mitral valve and of the septum. There were fine fluttering echoes in diastole in the aortic root (Fig. 2).

At operation in April 1981 the homograft was found to have a hole in the cusp in the right coronary position causing severe regurgitation. There was some retraction of the other two cusps. There was no histological evidence of inflammation or of endocarditis.

Fig. 1 (a) *M mode* echocardiographic scan from the left ventricular outflow tract (LVOT) to the aorta (AO) showing fluttering diastolic echoes (case 1). AML, anterior mitral leaflet. (b) Cross sectional echocardiogram (long axis parasternal view) showing diastolic prolapse of the non-coronary cusp of the aortic valve (arrowed) (case 1). LV, left ventricle; IVS, interventricular septum.

Case 3

A 35 year old woman, with a history of rheumatic fever, had an aortic valve replacement with a glutaraldehyde fixed Hancock xenograft in 1975. After this she was asymptomatic until December 1978, when she complained of weight loss and muscle pains. This illness was diagnosed and treated as subacute bacterial endocarditis, although no organisms could be cultured from the blood. There was no clinical evidence

Fig. 2 *M mode* echocardiogram of the aortic valve showing fine fluttering echoes in diastole (case 2). AO, aorta; LA, left atrium.

of aortic incompetence before or during this illness, and she made a good recovery.

M mode echocardiography performed during this illness showed a normally functioning bioprosthetic valve with no evidence of either aortic regurgitation or of vegetations (Fig. 3a).

In February 1980 an early diastolic murmur was noted for the first time. In February 1981 she complained of increasing dyspnoea. There was clinical evidence of pronounced aortic incompetence. *M mode* echocardiography showed notable diastolic fluttering of both leaflets of the mitral valve and of the septum. Mitral valve closure was normal in timing. The left ventricle was dilated and contracted poorly. There were fluttering echoes in systole in the aortic root (Fig. 3b). Diastolic vibrating echoes were seen in the aortic root and left ventricular outflow tract (Fig. 3c). Cross sectional echocardiography showed that the non-coronary cusp prolapsed into the left ventricular outflow tract.

At operation in November 1981 the Hancock valve was seen to have deficiencies in all three cusps, leaving a central orifice at all times (Fig. 3d). The non-

Fig. 3 M mode echocardiogram showing the Hancock xenograft in the aortic position (case 3) (a) on 23 February 1979 and (b) on 19 February 1981 when fluttering systolic echoes were evident. (c) M mode echocardiographic scan from the aortic valve to the left ventricle showing fluttering diastolic echoes in the aortic root and in the left ventricular outflow tract (case 3). (d) Morphological appearance of Hancock xenograft showing deficiencies in all three cusps (case 3). AO, aorta; AML, anterior mitral leaflet; LV, left ventricle.

coronary cusp was not supported and had fallen into the left ventricular outflow tract. No vegetations were seen. Histologically there was no evidence of an inflammatory response nor of bacterial infection.

Discussion

In each of the three patients with implanted tissue aortic valves in whom echocardiography showed a ruptured valve the diagnosis was confirmed at operation. There was a hole in one cusp of the homograft in one patient (case 2), deficiencies in all cusps of the xenografts with prolapse of a cusp in one (case 3) and of part of a cusp in one (case 1). In none was there any evidence of active infective endocarditis, although case 3 had had a febrile illness three years before.

Degeneration of the valve was thought to be spontaneous in all three.

Specific M mode echocardiographic features were first described in association with rupture of the aortic valve by Lee *et al* who reported a band of high frequency echoes in early to mid diastole, with fine linear echoes during systole, in the aortic root in four patients in whom a ruptured aortic valve was subsequently confirmed.¹

Since the initial description, similar echocardiographic appearances have been reported most often in association with active infective endocarditis affecting the aortic valve. Similar M mode echocardiograms have been recorded in patients who had recovered from endocarditis one, two, four, and five years before¹⁶⁻¹⁹ and in patients with a myxomatous or

floppy valve.¹¹

Wray suggested that irregular diastolic echoes in the left ventricular outflow tract, continuous with the aortic valve, were diagnostic of a flail cusp.²³ Roy *et al* interpreted them as representing prolapse of a cusp or part of a cusp or a vegetation on the ventricular aspect of the valve.⁵ These echocardiographic appearances have not previously been described in association with implanted tissue valves.

Two of our patients had xenografts, one a Carpentier-Edwards and the other a Hancock valve. These glutaraldehyde fixed porcine valves occasionally lose their integrity.²⁰ Cohn *et al* reported dysfunction in seven of 528 such valves in the mitral or aortic area after six years.²¹ Calcification of the valves can occur, especially in children and young adults.²²⁻²⁴ Rupture of the cusps has been reported, Cohn *et al* having found two in 528.²¹ Ferrans *et al* reported six with perforations.²³

Aortic homografts probably lose their integrity more often than do xenografts. Barratt-Boyes *et al* reported aortic incompetence in eight patients with aortic homografts who had died.²⁵ There was rupture of a cusp in three. At reoperation for aortic incompetence rupture of a cusp was the cause in nine of 17.

M mode and cross sectional echocardiography may be used to detect disorganisation of the aortic valve. Abnormal fluttering echoes in systole and diastole in the aortic root suggest valve disruption. Vibrating diastolic echoes in the left ventricular outflow tract may be caused by prolapse of cusp tissue. When these features are recorded from an implanted tissue valve, either a homograft or a xenograft, they imply severe disruption, which may be due to spontaneous degeneration. This is likely to be progressive and may be an indication for aortic valve replacement.

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