Severe late failure of a porcine xenograft mitral valve: clinical, echocardiographic, and pathological findings

GIANCARLO CRUPI, DEREK GIBSON, BRIAN HEARD, AND CHRISTOPHER LINCOLN

From the Department of Surgery, Cardiothoracic Institute, Brompton Hospital, London

ABSTRACT A case of dysfunction of a Carpentier-Edwards porcine xenograft mitral prosthesis is presented. Valve failure was diagnosed by echocardiography and confirmed at operation. Histology showed valve tissue degeneration without evidence of prosthetic endocarditis.

Several clinical studies have demonstrated that the porcine aortic xenograft prosthesis fulfils many of the criteria for a satisfactory valve substitute for atrioventricular valve replacement.¹⁻³ The durability of this prosthesis has been increased by the use of a flexible stent and glutaraldehyde preservation, and shown to exceed that of previously available tissue valves.

However, several instances of valve failure have been reported even with porcine xenograft prostheses.²⁻⁶

In this report we describe the clinical, echocardiographic, and pathological findings in an 11-year-old patient in whom dysfunction of a Carpentier-Edwards porcine xenograft mitral valve occurred two and a half years after its implantation. Valve failure was diagnosed by echocardiography and confirmed at operation. This appears to be the first reported case of valve failure of the Carpentier-Edwards xenograft since its clinical use began in 1975.

Case report

A 9-year-old boy was admitted to Brompton Hospital for investigation of mitral valve disease. Cardiac catheterisation confirmed the presence of severe mitral regurgitation. At operation he was found to have a parachute mitral valve with very abnormal leaflets. Satisfactory repair was not thought to be feasible and so the valve was excised and replaced with a 29 mm Carpentier-Edwards porcine xenograft prosthesis. After operation, the patient's clinical state was greatly improved, and it was possible to discontinue

treatment two months later. He remained well for two and a half years and then suddenly became severely breathless when walking. He was admitted to hospital and found to be in severe left ventricular failure which responded initially to digoxin and diuretics. He was transferred to Brompton Hospital where he was found to have evidence of pulmonary oedema, and to have a soft systolic murmur at the apex. The pulmonary component of the second heart sound was accentuated. A chest radiograph showed a normal sized heart with dilatation of the upper lobe veins. Echocardiography showed a transverse left ventricular dimension of 5.0 cm at end-diastole. Peak diastolic rate of dimension increase was 25 cm/s. The pattern of movement of the cusps of the prosthesis was abnormal. There was an amorphous group of echoes within the valve ring during diastole which disappeared during systole. The diagnosis was severe mitral regurgitation through rather than around the prosthesis associated with disorganisation of the cusps. At reoperation one of the cusps of the xenograft showed heavy calcification and another was torn off at its commissural attachment. The prosthesis was excised and replaced with a 2M 6120 Starr-Edwards valve. The postoperative course was uneventful.

PATHOLOGY

The valve cusps were rigid, the orifice admitting only the tip of the finger with difficulty. Their general shape was preserved, but they were diffusely thickened and opaque as a result of patches of calcification. There was a hole 2 mm in diameter near the centre of one of the cusps and part of this cusp was detached from the

Address for reprint requests: Christopher Lincoln, Brompton Hospital, Fulham Road, London SW3 6HP.

(a)

(b)

Fig Atrial (a) and ventricular (b) views of a porcine xenograft mitral prosthesis removed two and a half years after its insertion. Heavy calcification is present in two leaflets. The superior leaflet is detached from the prosthetic annulus at one commissure (arrow).

annulus (fig). The attachments of the other cusps were normal. Section of the cusps showed degenerative lesions of the elastin and collagen framework with focal calcification.

Discussion

The major disadvantage of tissue valves as human valve substitutes has been the unaccept-

ably high rates of failure. The use of a flexible stent and glutaraldehyde preservation has reduced this significantly, and more encouraging results have been achieved with the porcine xenograft prosthesis.¹⁻³ Indeed, the durability of the Hancock porcine xenograft appears to be comparable with that of currently available mechanical prostheses. However, several cases of valve failure from tissue degeneration have been reported with the Hancock prosthesis.²⁻⁶ Cusp perforation, leaflet calcification, and tears at the point of attachment of the commissure to the prosthetic struts have been the usual cause of valve failure. Histology invariably showed degenerative lesions of elastin and collagen framework with focal calcification. Findings were similar in our case whose glutaraldehyde fixed prosthesis had been inserted two and a half years earlier. Several hypotheses have been advanced to explain occasional failure of porcine aortic xenografts, but none seems entirely satisfactory.⁵⁻⁷ The degenerative lesions of the collagen and elastin framework have been interpreted as fatigue lesions and considered to be both progressive and inevitable in view of the nature of the bioprosthesis itself.⁸

Our findings seem to confirm this hypothesis, although a longer follow-up is required to assess whether the newly designed strut can improve the durability of the glutaraldehyde fixed aortic xenograft.

This case also illustrates the use of echocardiography in the management of these cases. Recording from the prosthesis indicated major abnormalities of the cusps. Digitisation of the left ventricular echogram demonstrated the presence of significant mitral regurgitation, at the same time excluding significant obstruction to inflow by the prosthesis or left ventricular disease, both of which might have explained the clinical picture on presentation.

References

- 1 Carpentier A, Deloche A, Relland J *et al*. Six year follow-up of glutaraldehyde-preserved heterografts. *J Thorac Cardiovasc Surg* 1974; **68**:771-82.
- 2 McIntosh CL, Michaelis LL, Morrow AG, Itscoitz SC, Redwood DR, Epstein SE. Atrio-ventricular valve replacement with the Hancock porcine xenograft: a five years' clinical experience. *Surgery* 1975; **78**:768-75.
- 3 Stinson EB, Griep RB, Oyer PE, Shumway NE. Long-term experience with porcine aortic valve xenografts. *J Thorac Cardiovasc Surg* 1977; **73**:54-63.

- 4 Zuhdi H, Hawley W, Voehl V, Hancock W, Carey J, Greer A. Porcine aortic valves as replacement for human heart valves. *Ann Thorac Surg* 1974; **17**:479–91.
- 5 Levitsky S. Discussion of Stinson *et al.* *J Thorac Cardiovasc Surg* 1977; **73**:62.
- 6 Brown JW, Dunn JM, Spooner E, Kirsh MM. Late spontaneous disruption of a porcine xenograft mitral valve. Clinical, haemodynamic, echocardiographic and pathologic findings. *J Thorac Cardiovasc Surg* 1978; **75**:606–11.
- 7 Carpentier A. Discussion of Stinson *et al.* *J Thorac Cardiovasc Surg* 1977; **73**:61.
- 8 Broom ND. Fatigue-induced damage in glutaraldehyde-preserved heart valve tissue. *J Thorac Cardiovasc Surg* 1978; **76**:202–11.