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Mitral prosthetic valve regurgitation due to stent fracture of a porcine bioprosthesis

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ABSTRACT Mitral prosthetic valve regurgitation due to stent fracture with consequent cusp prolapse occurred in two patients with a Wessex porcine bioprosthesis.

The Wessex porcine bioprosthesis is Britain's first commercially available tissue valve and has been available for clinical implantation since December 1982. Early in vitro and in vivo evaluation[1] showed satisfactory biodurability and haemodynamic performance, and a recent three centre study concluded that clinical results compared favourably with those of other bioprostheses.[2] So far some 5000 valves have been implanted. There have been no published reports of valve failure. We report two cases in which valve failure occurred as a direct result of stent fracture, leading to leaflet prolapse. This complication has not been reported for porcine bioprostheses.

Case reports

CASE 1

A 58 year old woman was admitted to the coronary care unit with a three week history of worsening dyspnoea of effort and a four day history of paroxysmal nocturnal dyspnoea and orthopnoea. At the age of 31 she had undergone mitral valvotomy for rheumatic mitral stenosis, followed by mitral valve replacement with a 31 mm Carpentier-Edwards porcine bioprosthesis 21 years later. A bioprosthesis was chosen because she had a history of peptic ulceration and haematemesis. Three years later, and three years before the present admission, the Carpentier valve failed because of a torn leaflet and was replaced with a 29 mm Wessex porcine bioprosthesis. On the present occasion she was admitted with florid pulmonary oedema, and echocardiography showed regurgitation through the mitral prosthetic valve with cusp prolapse. At emergency operation the Wessex valve was found to have well preserved leaflets but cusp prolapse was caused by fracture of the septal strut of the stent (figs 1 and 2). The valve was replaced with a 29 mm St Jude prosthesis and the patient subsequently made a good recovery.

CASE 2

A 45 year old woman with an eight month history of increasing dyspnoea on exertion, paroxysmal nocturnal dyspnoea, and orthopnoea was admitted for replacement of her mitral prosthetic valve. At the age of 33 she had undergone mitral valve replacement with a Hancock porcine bioprosthesis. Nine years later the Hancock valve failed acutely owing to torn leaflets and emergency mitral prosthetic replacement with a 31 mm Wessex porcine bioprosthesis was performed. The patient remained well until a few months before the current admission, some 33 months after her last operation. Investigations showed regurgitation

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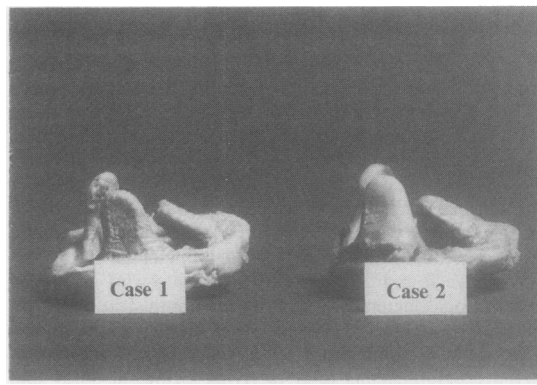

Fig 1 *Profile view of two explanted Wessex procine bioprostheses showing bent commissural posts.*

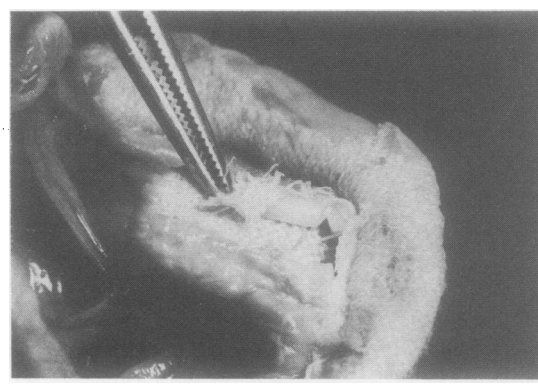

Fig 2 *Exploded view of commissural-annular junction showing fracture of commissural stent.*

through the mitral prosthetic valve. At operation the Wessex valve was again found to have well preserved leaflets but the septal stent was fractured (fig 1). The valve was replaced with a 31 mm St Jude prosthesis and the patient made a good recovery.

Discussion

The Wessex porcine bioprosthesis is essentially similar to established porcine bioprostheses available on the market, but with the added features of a computer designed stent, a special method of controlled pressure tissue fixation, the use of pericardial tape to reduce turbulence, and enhanced quality control.[1] The valve stent is made from Hostaform C27021, an acetal copolymer not previously used in prosthetic heart valve manufacture and chosen for its creep resistance. It is designed with precise commissural flexion characteristics to minimise stress on the leaflets, and each commissural support is tested for flexibility over a 2 mm flexion during manufacture (valve literature, Wessex Medical).

In our patients both valves failed because of fractures of the stent at the junction between valve annulus and commissural support (fig 2). The resulting distortion in stent geometry led directly to cusp prolapse despite macroscopically preserved cusps. We suggest that repeated systolic flexion of the commissural support led to fatigue of the creep resistant stent material and subsequent fracture. The fact that both our patients were young and had good left ventricular function may have been important in this respect.

The stents are most unlikely to have been fractured at the time of implantation or explantation. Against the former is the fact that both patients had clinically normal prosthetic function until their recurrence of symptoms, and against the latter the fact that both patients developed symptoms as a direct result of mitral prosthetic regurgitation, and that these symptoms predated the explantations by three weeks in one case and eight months in the other.

We conclude that prosthetic failure occurred in our patients as a result of stent fracture with cusp prolapse, a hitherto unreported mode of bioprosthetic failure.

References

1 Reece IJ, Anderson JD, Wain WH, *et al.* A new porcine bioprosthesis: in vitro and in vivo evaluation. *Life Support Systems* 1985;3:207–27.

2 Reece IJ, Wheatley DJ, Munro JL, *et al.* Early results with the Wessex porcine bioprosthesis in 245 patients. In: Bodnar E, Yacoub M, eds. *Biological and bioprosthetic valves*. USA: Yorke Medical Books, 1986:760–7.