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Left Ventricular Rupture Following Mitral Valve Replacement with a Hancock Bioprosthesis*

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A rare complication of mitral valve replacement with the Hancock bioprosthesis was observed in three patients presenting with prevalent mitral stenosis and normally-sized left ventricular cavity. In the patients, a deep erosion of the left ventricular free wall was present which evolved into cardiac rupture in one of them. The laceration is ascribed to the friction between a prosthetic strut and the myocardium; a disproportion between the "high-profile" device and the left ventricular chamber is suggested to explain this complication, which may be avoided by employing a "low-profile" prosthesis.

Left ventricular rupture is an uncommon, often lethal complication of prosthetic mitral valve replacement. The rupture may be located either at the posterior atrioventricular groove or at the midportion of the posterior left ventricular wall; it may occur during surgery or within a few hours following operation, regardless of the type of prosthesis employed.5,8

We have recently encountered this complication in a patient who had undergone mitral valve replacement with a Hancock bioprosthesis. Postmortem examination of the heart disclosed a tear in the posterolateral wall of the left ventricle due to erosion produced on the myocardium by one of the prosthetic struts. Only a few cases of this type of rupture have been reported so far.4,5

A review of our pathologic collection of heart specimens with porcine mitral prostheses allowed us to recognize two other cases with a laceration of the left ventricular wall at the same level. In these cases, however, rupture did not occur.

CASE REPORTS

Case 1

A 59-year-old woman with prevalent mitral stenosis and tricuspid insufficiency was referred to our hospital for surgery. On March 13, 1974, she underwent mitral valve replacement with a flexible stented Hancock-SCP xenograft (33 mm) and a De Vega tricuspid annuloplasty. On the second postoperative day, signs of low cardiac output appeared and reoperation was performed due to a suspected cardiac tamponade. No intrapericardial clot or bleeding were found. Since the right atrium was extremely dilated and the central venous pressure high, the tricuspid valve, whose annuloplasty had disrupted, was replaced with a Björk-Shiley prosthesis (31 mm). The patient died the following day despite intensive pharmacologic treatment. At postmortem examination, an erosion of the endomyocardium was visible (arrows), with hemorrhagic infiltration of adjacent tissues.

FIGURE 1. Case 1, left ventricular view. A 33-mm Hancock bioprosthesis is inserted in mitral position. In lateral wall of left ventricle, 2 cm below mitral annulus, a large, deep erosion of the endomyocardium is visible (arrows), with hemorrhagic infiltration of adjacent tissues.

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the heart weighed 520 gm, including ascending aorta, pulmonary trunk, and subepicardial fat; the wall of the left ventricle was 11 mm thick. The porcine mitral bioprosthesis, implanted in the subannular position, partially obstructed the left ventricular outflow tract with one of its struts. On the lateral wall of the left ventricle, 2 cm below the mitral annulus, a deep erosion corresponding to a prosthetic prong was observed (Fig 1).

CASE 2

A 51-year-old woman had undergone a closed mitral commissurotomy at the age of 38 years, with immediate clinical improvement. Twelve years later, the signs of cardiac failure reappeared, and a cardiac catheterization demonstrated a severe stenosis with mild incompetence of the mitral valve and tricuspid insufficiency. On Oct 8, 1978, the calcified mitral valve was replaced with a Hancock-SGP flexible stented xenograft (31 mm), and a tricuspid annuloplasty was performed. Following surgery, the cardiac output appeared inadequate, and despite intense pharmacologic support, death occurred on the seventh postoperative day. At autopsy, the heart weighed 400 gm, including the ascending aorta, the pulmonary trunk, and the subepicardial fat; the left ventricular wall was 9 mm thick. A porcine mitral valve was inserted in the supra-annular position, and thrombotic deposits were noted on the sewing ring. A deep erosion of the posterolateral free wall of the left ventricle, 2 cm below the mitral annulus, was observed.

CASE 3

A 54-year-old woman with a severely calcified mitral stenosis had previously experienced an episode of embolization to the right leg. On Feb 2, 1978, the mitral valve was replaced with a Hancock-SGP xenograft (31 mm). One hour after surgery, while in the intensive care unit, a massive hemorrhagic drain was observed through the pericardial tubes. The chest was immediately opened and a tear in the posterolateral wall of the left ventricle was noted. All attempts to repair the laceration were unsuccessful, and the patient died. At postmortem examination, the heart weighed 450 gm including the great arteries and the subepicardial fat; the left ventricular wall was 1 cm thick. A normally oriented mitral porcine valve was in the supra-annular position. A deep erosion, 2 cm below the mitral annulus with rupture of the posterolateral wall of the left ventricle, was observed. A hemorrhagic infiltration in the subepicardial space, around the site of the laceration which was far from the atrioventricular groove, was also present (Fig 2).

DISCUSSION

The possible disproportion between "high-profile" mechanical valvular prostheses and cardiac cavities is a well-recognized problem. This may occur also with the use of porcine heterografts, and lead mainly to left ventricular outflow tract obstruction following mitral valve replacement. The cases reported here show deep erosion or rupture of the posterior or lateral wall of the left ventricle, which occurred early after mitral valve replacement with a Hancock valve. All patients had severe mitral disease with prevalent stenosis and had a relatively normal left ventricular cavity. The location of the tears, 2 cm below the atrioventricular groove, suggests that they were most likely caused by one of the struts of the porcine bioprosthesis which rubbed the myocardium continuously during the cardiac contractions, and progressively eroded it. In patient 3, the myocardial lesion was deep enough to

![Figure 2. Case 3. A (left), Left ventricular view. Mitral valve has been replaced with a 31-mm Hancock xenograft. Deep laceration of endomyocardium (arrow) is present in posterolateral wall of left ventricle, corresponding to posterior strut of the device. B (right), lateral external view of left ventricle showing a hematoma of subepicardium and a patch (arrow) inserted in attempt to repair rupture, which is quite far from atrioventricular groove.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21105/)
caused a fatal hemorrhage. This type of left ventricular rupture is quite different from those usually observed following prosthetic mitral valve replacement and which are ascribed either to a weakening of the anulus or to an excessive resection of the papillary muscles.1-3,8-14

It is well known that the mitral anulus is not a rigid structure. In fact, its size changes in an eccentric way along with the cardiac cycle, and decreases mainly during atrial systole, the left atrioventricular orifice being quite reduced before the onset of the ventricular systole.15 Prosthetic valve replacement prevents the movements of the mitral anulus, while the underlying musculature still contracts. When the left ventricular cavity is almost normal in size, as in mitral stenosis, insertion of a large Hancock bioprosthesis may determine a disproportion between the volume of the cavity itself and the size of the valve and cause a continuous friction by one strut on the left ventricular free wall. This occurred in our patients even though a flexible stented prosthesis had been used in each case. We have also considered that resuscitative efforts might have played a role in the etiology of the laceration; nonetheless, the spontaneous occurrence of cardiac rupture in the third case, at the very same level, led us to regard as less likely such possibility.

Left midventricular rupture with a Hancock mitral bioprosthesis has been previously reported,4,5 and different mechanisms have been advocated. Cobbs and colleagues4 suggested that the sudden elongation of the left ventricular inflow tract untethered from its normal attachment to the mitral anulus, possibly associated with a surgical trauma, is probably the major etiologic factor. On the other hand, Katske and colleagues5 excluded an iatrogenic factor and considered the transient postoperative hypertension, which was observed in each of their cases, as the determinant of a hyperdynamic left ventricular contraction against the strut of the bioprosthesis. The latter hypothesis could not be confirmed by the postoperative clinical history of our patients.

In our opinion, however, the main factor in determining this complication is the above-mentioned disproportion which the surgeon should consider when choosing a device to replace the mitral valve. A "low-profile" prosthesis is preferred in patients with mitral stenosis and relatively normal left ventricular cavity.

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Bronchial Coarctation*

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A 38-year-old white woman with chronic bronchitis was found to have proximal coarctation of several third and fourth order bronchi. These lesions persisted despite treatment with prednisone, 40 mg a day for eight weeks. We think these lesions are congenital in origin and were exacerbated by chronic bronchitis.

Severe narrowing of large bronchi may occur in chronic bronchitis, asthma, tuberculosis, sarcoidosis, and amyloidosis. In chronic bronchitis, these changes are said to occur "distal to the third or fourth generation of..."