Replacement of mitral valve in active infective endocarditis

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Infective endocarditis which is resistant to medical treatment is fatal. In a woman of 32 years the mitral valve was replaced, in the presence of active infection, by a fascia lata valve. Her postoperative course was entirely satisfactory. Twelve months after operation, after a normal full-term pregnancy, she gave birth to healthy twins. The place of valvar replacement in the treatment of active infective endocarditis of the mitral valve is discussed.

The majority of patients with infective endocarditis respond satisfactorily to antibiotic treatment. Some develop severe valvar regurgitation, and others resistant infection or repeated embolism. In such patients excision of the infected valve and correction of the haemodynamic lesion during the active stage may be indicated (Yeh, Hall, and Ellison, 1964; Branniff, Shumway, and Harrison, 1967; Hurley, Eldridge, and Hultgren, 1967; Kretschmer and Lawrence, 1969; Cleland *et al.*, 1969). In most of the reported patients, in whom operation was performed, the aortic valve was infected (Wallace, Young, and Osterhout, 1965; Kaiser *et al.*, 1967; McDonald, 1967; Scott, Fish, and Crutcher, 1967). In only 4 reported patients was the mitral valve the site of infection (Robicsek *et al.*, 1967; Wilcox *et al.*, 1967; Windsor and Shanahan, 1967). This report describes a patient with resistant active bacterial endocarditis of the mitral valve, who was treated by valvar replacement, using a fascia lata valve. The value and risks of valvar replacement in acute infective endocarditis are discussed.

Case report

The patient was a woman of 32 years who had had rheumatic fever at the age of 13, and was seen in August 1969. After a miscarriage, in October 1968, she became febrile and there was clinical and laboratory evidence of infective endocarditis. A *Streptococcus pyogenes* was grown from her blood. Antibiotic treatment was begun early in the illness but the patient discharged herself from hospital and, on and off, since that time had had

recurrent clinical evidence of infective endocarditis. Furthermore, there had been several episodes of systemic embolism, including one to the right femoral artery on 29 May. On 6 August she presented with abdominal pain and jaundice.

On examination she was jaundiced and overweight. The arterial pulses were irregular, and of full volume, and blood pressure was 120/80 mmHg. The jugular venous pressure and pulse were normal, and the cardiac impulse impalpable. On auscultation, in the mitral area, the first heart sound was slightly accentuated and followed by a moderate mitral pansystolic murmur. In diastole there was a faint and fairly late opening snap of the mitral valve and a left ventricular third heart sound followed by a diastolic murmur of moderate intensity and length. In the aortic area there was a faint ejection systolic murmur with normal first and second heart sounds. There were basal crepitations which cleared with coughing and the liver was one finger's breadth enlarged. Electrocardiogram in August 1969 showed atrial fibrillation; previously there had been right axis deviation and evidence of moderate left atrial hypertrophy. X-rays showed moderate enlargement of the ventricular mass and slight to moderate left and right atrial enlargement, with slight prominence of the pulmonary artery and no pulmonary venous congestion. The urine contained neither albumin nor sugar. Blood counts showed a polymorphonuclear leucocytosis and raised sedimentation rate, without significant anaemia.

Thus, there was evidence of rheumatic heart disease with mitral valvar disease, in which the lesion was mixed with regurgitation and stenosis, and of infective endocarditis, which was and remained resistant to antibiotic therapy. The infective endocarditis was not controlled by a further course of intensive antibiotic treatment, and further systemic emboli occurred, and the heart

FIG. 1 Chest x-ray on 14 October 1969, showing moderate enlargement of the heart and pulmonary artery, also pulmonary venous congestion. See text.

FIG. 2 Chest x-ray on 9 March 1970. The heart and pulmonary artery are smaller than in Fig. 1 and the pulmonary veins are not congested. See text.

remained enlarged (Fig. 1). Replacement of the mitral valve was therefore undertaken in October 1969. This was performed through a median sternotomy. The external appearances of the heart were consistent with moderately severe mitral regurgitation. The left atrial mean pressure measured at operation, before starting cardiopulmonary bypass, was 15 mmHg with a V wave of 38 mmHg. There was no gradient across the aortic valve. Normothermic bypass was established with arterial return to the ascending aorta. The left atrium was opened behind Sonesgaard's sulcus. The mitral valve was thickened and looked stenotic and regurgitant with an opening measuring 1.8 cm in length. There was a soft vegetation on the atrial surface of the aortic cusp. The chordae tendineae were fused into two columns of fibrous tissue. The valve was excised and replaced by an autogenous fascia lata valve fixed to a frame measuring 26 mm in diameter (Ionescu and Ross, 1969). After getting rid of air, bypass was discontinued. The postoperative recovery was rapid and uncomplicated. A chest x-ray on 9 March 1970 (Fig. 2) showed a reduction in the size of the heart and pulmonary artery, and no pulmonary venous congestion. Twelve months after operation the patient was safely delivered of twins, after a normal term pregnancy.

Discussion

The use of antibiotics has favourably influenced the prognosis of patients suffering from bacterial endocarditis with a reduction in the immediate mortality rate (Hickie, 1961; Robinson and Ruedy, 1962). Destruction of the valve resulting in severe heart

failure, the presence of resistant infection, and repeated systemic embolism may be regarded as indications for valvar replacement. In 279 patients with infective endocarditis who were seen between 1950 and 1960, Hickie (1961) found that the commonest cause of death was cardiac failure. It occurred in 60 patients, and in a further 54, vegetations leading to systemic emboli (43 patients), coronary emboli (10 patients), and valvar stenosis (1 patient) were the cause of death.

The value of valvar replacement during the active stage of infective endocarditis has recently been stressed (Kretschmer and Lawrence, 1969). In most of the reported patients, the aortic valve was the site of the infection (Yeh *et al.*, 1964; Wallace *et al.*, 1965; Branniff *et al.*, 1967; Hurley *et al.*, 1967; Kaiser *et al.*, 1967; McDonald, 1967; Scott *et al.*, 1967). In contrast, the mitral valve was involved in only 4 reported cases (Robicsek *et al.*, 1967; Wilcox *et al.*, 1967; Windsor and Shanahan, 1967). The risks of dissemination or recurrence of the infection, or rupture of a mycotic aneurysm during or after the operation, have been slight. In the patient described here, a fascia lata graft (Ionescu and Ross, 1969) was used because autogenous tissues are believed to be more resistant to infection, and also to avoid the risk of long-term anticoagulant treatment in a woman in the childbearing age. Her successful pregnancy and the birth of twins indicate a satisfactory operative result.

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Symposium on Platelets, Drugs, and Thrombosis

The above symposium is to be held 16-18 October, 1972, at McMaster University Medical Centre, Hamilton, Ontario, Canada. The symposium is sponsored by the Faculty of Medicine, McMaster University, the Ontario Heart Foundation, and the Canadian Heart Foundation. Thirty-six internationally respected investigators have agreed to present papers. There will be a fee of \$30 for registration and the total attendance will be limited to 300 so that discussion will be possible and profitable.

Further information can be obtained from Ontario Heart Foundation, 247 Davenport Road, Toronto 180, Canada.