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DOUBLE VALVE REPAIR IN A TRANSPLANTED HEART

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The first case of mitral valve replacement after heart transplantation was published in 1991. Since then, three additional cases of mitral valve replacement (one in combination with a tricuspid valve repair), two aortic valve replacements, and 12 cases of tricuspid valve replacement have been reported.[1-4] In all these cases the damaged valves were deemed beyond surgical repair and were replaced by either a porcine bioprosthesis or a mechanical valve prosthesis. In two instances bench mitral valve repair before heart transplantation was described.[5] In seven patients tricuspid valve repair was performed (combined with mitral valve replacement in one, as just mentioned).[2, 4] This report presents the first case of mitral and tricuspid valve repair in a transplanted heart.

The patient was a 60-year-old man with a history of heart failure resulting from dilated cardiomyopathy for which he underwent orthotopic heart transplantation in April 1990. The donor was a 12-year-old boy with no history of heart disease, normal auscultatory findings, and normal electrocardiographic findings. The procedure was performed by means of the biatrial method with Prolene running sutures (Ethicon, Inc., Somerville, N.J.) for the atria and the great vessels. The operation was uncomplicated.

Postoperatively the patient had bradycardia, for which he received isoproterenol (INN: isoprenaline) treatment for 11 days. Recovery was otherwise uneventful. Immunosuppressive therapy consisted of OKT3, followed by cyclosporine (INN: ciclosporin) and low-dose steroids as maintenance therapy.

Echocardiography in the first postoperative weeks showed some pericardial effusion. From the first week on, some billowing of the anterior mitral valve leaflet was noted. There were no rejection episodes. A total of seventeen endomyocardial biopsies were performed in the first year after transplantation and seven in the following years. Follow-up by transthoracic echocardiography showed gradual progression of mitral regurgitation. In 1993 moderate tricuspid regurgitation was also found. Despite this the man enjoyed a productive working life without symptoms during these 5 years. At the yearly catheterization, mild regurgitation of the mitral valve was noted on cineangiography.

In 1996, 6 years after cardiac transplantation, the patient began having easy fatiguability and dyspnea on exertion. On those occasions he also reported abdominal distention. Physical examination revealed a holosystolic apical murmur, no pulmonary abnormalities, and no hepatomegaly. Transesophageal echocardiography showed a nondilated hypertrophic left ventricle with good systolic function. Billowing of both mitral valve leaflets was noted, with a prolapse of the anterior leaflet and severe regurgitation. At that time, an insignificant tricuspid regurgitation was noted. Heart catheterization showed elevated filling pressures (right atrial pressure 15 mm Hg, pulmonary capillary wedge pressure 20 mm Hg, and left ventricular end-diastolic pressure 23 mm Hg), no significant stenosis of the coronary arteries, good left ventricular function, but severe mitral valve regurgitation (Sellers grade 3/4). Valve repair was proposed for this patient, and on February 4, 1997, he was operated on.

A redo sternotomy was performed. Cardiopulmonary bypass with selective venous cannulation of both the superior and inferior venae cavae and moderate hypothermia with cardioplegic arrest were used. The mitral valve was well exposed via a right atrial, transseptal approach. Inspection revealed a prolapse of both valve leaflets adjacent to the posteromedial commissure resulting from papillary muscle elongation. The papillary muscle appeared to be unnaturally pale, as did other parts of the endocardial tissues. Marked myxomatous degeneration was noted on the right side of both valve leaflets. The left side was unaffected. The anulus was severely dilated. Papillary muscle shortening was performed by folding back the tip of the muscle onto its base and suturing it into place. The annular dilation was corrected by inserting a size 34 Cosgrove-Edwards ring (Baxter Healthcare Corp., Irvine, Calif.). Transesophageal echocardiography after closure of the atrial septum and the atrial wall showed a good result. However, at this time tricuspid valve regurgitation was considered more severe than previously appreciated because of two flail valve leaflets. Cardiopulmonary bypass was restarted and the tricuspid valve was exposed with a beating heart. The chordae of the posterior part of anterior valve leaflet, as well as chordae of the posterior leaflet, were ruptured, with resulting partially flail leaflets. The septal and posterior valve leaflets were sutured together to make a functionally bicuspid valve. A small prolapsing segment of the anterior leaflet was resected, and a partial De Vega annuloplasty at the base of the posterior leaflet and adjoining commissures completed the reconstruction. Transesophageal echocardiography showed a good result with minor regurgitation of both the mitral and tricuspid valves.

Postoperative recovery was uneventful. Transthoracic echocardiography on the seventh day after operation

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showed a trace of mitral valve regurgitation and grade 1+ tricuspid valve regurgitation. The patient was discharged on the tenth postoperative day. One month after the operation he was seen in the outpatient clinic and had recovered well. The dyspnea had greatly diminished. At physical examination no heart murmurs were heard. Unfortunately, the patient died on April 30 as a result of intracranial hemorrhage, during the last week of a 3-month postoperative course of coumarin therapy.

Multiple causes seem to exist with regard to valve dysfunction after heart transplantation. Early atrioventricular valve regurgitation is a common finding in transplanted hearts. Edema of the transplanted heart and poor lymphatic drainage are possible explanations for this phenomenon, which subsides after about 3 months. Others stipulate that the enlargement of the atrial cavity in atrium-to-atrium suturing gives an atrium-to-ventricle size mismatch that lies at the basis of atrioventricular valve dysfunction. Chord rupture resulting from endomyocardial biopsies is thought to be a leading cause of late tricuspid valve regurgitation. However, annular dilatation as a consequence of long-standing tricuspid valve dysfunction or graft dilatation is nearly always reported in patients undergoing these operations. In three unusual cases, the septal leaflets were virtually absent.[2] The true incidence of chord damage in heart transplant recipients may be underestimated because loose tricuspid chordae will not always be apparent on echocardiography.[3]

A total of 10 cases of prosthetic tricuspid valve replacement after heart transplantation have been reported, the majority for chord damage after myocardial biopsies.[4] Severe tricuspid regurgitation leading to tricuspid valve repair has been reported in seven patients. From the information of the intermediate results after valve reconstruction on residual tricuspid valve regurgitation, it is unsettled whether the tricuspid valve should be replaced rather than repaired.[2, 3] One author suggests that, to prevent the recurrence of chord rupture after reconstruction, biopsy specimens should be taken with a sheath-covered bioptome.[3] After valve replacement the right side of the heart is inaccessible for endomyocardial biopsies.

Prosthetic mitral valve replacement was reported in two cases[1, 6] in which endomyocardial biopsy specimens were taken from the left ventricle and in one other patient with mitral regurgitation caused by bacterial endocarditis after transplantation.[4] Recently, a case was published describing valve replacement for a myxomatous degenerated mitral valve combined with tricuspid valve annuloplasty for iatrogenic damage. Those authors' observations are very similar to the abnormalities found in our patient.[4] Two interesting cases of mild donor mitral valve disease treated with bench repair at the time of transplantation were reported.[5] These are the only cases of mitral valve repair known in transplant recipients.

Our report shows the feasibility of double valve reconstruction in a heart transplant recipient. Damage assessment is a crucial step, as in other valve operations. Exposure of the mitral valve was easily obtained by a right atrial, transseptal approach. The mitral valve regurgitation is most likely caused by progression of valve disease in the donor at the time of transplantation. The tricuspid valve regurgitation was iatrogenic. Both valves could be repaired successfully. After reconstruction the tricuspid valve chordae remain exposed to the risk of iatrogenic damage, but future biopsies are not hampered by a valve prosthesis and long-term coumarin therapy is avoided.

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