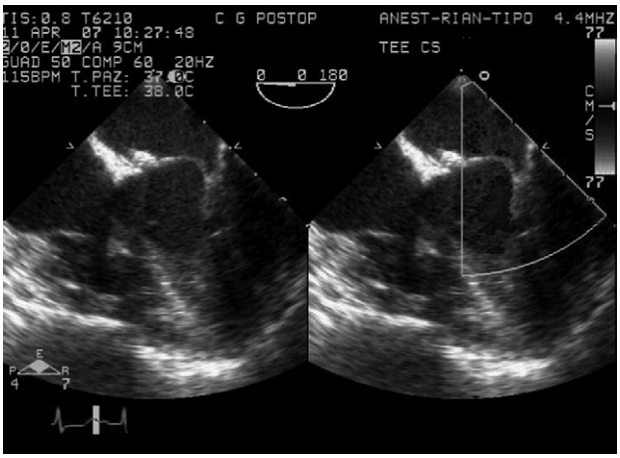

Fig 2. Postoperative transesophageal echocardiography, four-chamber view: absence residual mitral regurgitation.

mated was >2 cm^2 and coaptation length was 1.2 cm. He was asymptomatic, and a transthoracic echocardiogram at 11 months showed the absence of mitral regurgitation.

Comment

Primary congenital mitral valve abnormalities may be complex, affecting one or multiple anatomic components of the mitral valve, thereby leading to mitral insufficiency or stenosis [2]. Our case is an unusual cause of mitral valve insufficiency caused by a marked hypoplasia of the posterior leaflet and its subvalvular apparatus.

As far as we know, few cases of severe congenital hypoplasia and agenesis of the posterior mitral leaflet appear in the literature [2, 5]. In the case presented, the posterior mitral leaflet was almost completely absent, represented only by tags of fibrous tissue that strictly adhered to the posterior annulus with a total absence of chordae inserting into the hypoplastic leaflet. The anomaly was well tolerated and the mitral insufficiency was aggravated later in life by a progressive annular dilatation. Thus, interestingly, the presence of the posterior leaflet resulted not to be so critical for gaining valve continence. An adequately long and mobile anterior mitral leaflet guaranteed a proper valve closure until a significant annular dilatation occurred, translating into severe mitral regurgitation, which brought the patient to our attention.

The surgical correction consisted of mitral valve repair by restrictive annuloplasty, which, by reducing the septal-lateral dimensions, transformed the mitral valve in a "true" unicuspid valve where the mechanism closure was performed only by the anterior leaflet. Mitral valve repair gained a satisfactory postoperative coaptation length and no mitral insufficiency was present at subsequent echocardiographic controls.

We believe that the crucial point for successful repair under these circumstances is the mobility and length of the anterior leaflet (ie, a mobile anterior leaflet longer than anteroposterior annular diameter is required, in our opinion, to ensure successful repair).

Finally, in this case we preferred to implant a "saddle-shaped" rigid ring that mimics the physiologic shape of the mitral annulus and results in a lower tension on the whole valve apparatus.

In conclusion, severe hypoplasia of the posterior mitral leaflet is a rare cause of mitral regurgitation. It can be successfully corrected by a reductive ring annuloplasty in presence of a sufficiently long and mobile anterior leaflet. Nonetheless, the long-term durability of this repair remains uncertain.

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FEATURE ARTICLES

Aortic Valve Replacement for Aortic Stenosis During Orthotopic Cardiac Transplant

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Although concomitant coronary bypass, and mitral and tricuspid valve surgery have been used to expand the donor pool for cardiac transplantation, aortic valve disease is considered an absolute contraindication for use of an offered organ. A case is presented with the successful use of an organ requiring concomitant aortic valve replacement for calcific aortic stenosis on a congenitally bicuspid valve. Eighteen-month follow-up documented excellent allograft function with a normally functioning mechanical aortic prosthesis. Aortic valve disease in offered organs can be successfully treated with aortic valve replacement at the time of transplantation and should not preclude the use of the organ in the setting of a recipient who is a candidate for a marginal allograft.

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Assessment of appropriate cardiac allografts is the cornerstone to successful cardiac transplantation. Organs with structural valvular lesions are excluded at the time of assessment of the potential donor, with reliance placed on age, medical history, and appropriate adjuncts, such as echocardiography. Further assessment is performed during the organ procurement process with visual inspection and palpation of the allograft.

We report the successful intraoperative mechanical aortic valve replacement in a donor allograft during the orthotopic cardiac transplant procedure for undiagnosed moderate-to-severe aortic stenosis, secondary to a calcified congenitally bicuspid valve. Although inadvertent, the success of this patient highlights that correctable aortic valvular lesions do not preclude the use of offered organs.

Orthotopic cardiac transplantation was undertaken in a 55-year-old man for end-stage cardiac failure due to idiopathic dilated cardiomyopathy. The recipient had a known persistent left superior vena cava diagnosed during attempted percutaneous insertion of an LV lead for cardiac resynchronization therapy. Epicardial lead placement was performed using robotic surgical techniques. No other medical history was noted.

A 34-year-old man was referred as a donor from a very distant regional hospital after the diagnosis of brain death secondary to a head injury that occurred while intoxicated. No medical history was noted. The patient was reported as a fit and active young man, with no clinical evidence of cardiovascular disease. The donor's father had undergone coronary surgery at the age of 55. No vasoactive hemodynamic supports were required in the period prior to organ procurement. Due to limited services in the regional hospital, echocardiography was not performed. The retrieving time from another institution reported no abnormalities in the cardiac allograft. A bilateral sequential lung transplant was performed by this team with the procured lungs. The heart was then couriered 3,000 km unaccompanied to our institution.

Due to anticipated difficulties in preserving the recipient's persistent left superior vena cava and the tenacity of adhesions from the previous robotic surgery, cardiopulmonary bypass was established during transfer of the donor allograft. Recipient cardiectomy was performed with preservation of the left superior vena cava and a cuff of the right atrium containing the coronary sinus ostium, as described by Rabago and colleagues [1] in a bid to reduce donor ischemic time.

After arrival, the donor allograft was inspected prior to implantation at which time marked LV hypertrophy was noted. Inspection of the aortic valve revealed heavy calcification on a congenitally bicuspid valve. The leaflets were thickened and immobile, and it was immediately evident that valve replacement was required. A 25-mm St. Jude medical mechanical prosthesis (St. Jude Medical, St. Paul, MN) was implanted with everted pledgeted 2-0 Ticron interrupted sutures (Tyco Healthcare, Mansfield, MA). The valve was seated satisfactorily with normal prosthetic leaflet function. The remainder of the allograft implant was performed using a standard bi-caval technique and intermittent antegrade cold blood cardioplegia through the aortic root.

Cardiopulmonary bypass was weaned on low-dose adrenaline and noradrenaline infusions, intra-aortic balloon counterpulsation at 1:1 and nitric oxide at 40 ppm. A combination of the 3,000 km procurement distance and technical challenges led to a total ischemic time of 492 minutes. Post-bypass transesophageal echocardiography revealed normal prosthetic valvular function with a peak gradient of 29 mm Hg and a mean gradient of 12 mm Hg. Marked LV hypertrophy was noted with associated global left and right ventricular dysfunction.

The patient was returned to the intensive care unit in stable condition. Ventilatory support was weaned during the ensuing 24 hours. Adrenaline was exchanged for dobutamine, which was continued at a low dose for 5 days postoperatively. The recipient made an uncomplicated recovery and was discharged on day 14 on warfarin, prednisolone, mycophenolate mofetil, and cyclosporin. His management also included irbesartan and metoprolol. Endomyocardial biopsy at day 7 showed no evidence of rejection. Transthoracic echocardiography revealed a well-seated and normally functioning mechanical prosthesis with low normal left ventricular (LV) systolic function and mild LV hypertrophy.

Subsequent transthoracic echocardiograms were performed at 1, 3, 6, and 18 months, documenting significant reduction in LV mass. Immediately postoperative LV mass index was 124.5 g/m^2, which had reduced at last review to 95 g/m^2 (Table 1).

The function of the aortic valve prosthesis has been within normal limits throughout the serial transthoracic echocardiography studies.

Comment

This case report describes the unintentionally successful use of a marginal cardiac allograft with aortic stenosis not recognized at organ procurement in a patient with dilated cardiomyopathy, for whom recognition of the valvular disease in the offered allograft would have led to its rejection for transplantation. However, in the face of declining transplant numbers with growing waiting lists, use of marginal organs has become more common. The definition of the "marginal" allograft is dependent on the donor selection criteria of individual centers; however, in subgroups of patients, the use of a suboptimal organ may represent the only hope of undergoing cardiac transplantation.

Heterotopic cardiac transplants [2], valvular and coronary intervention on donor allografts have been well described to enable the use of smaller organs with longer ischemic times, and organs with valvular and coronary

Table 1. Echocardiographic Measurements of Left Ventricular Mass and Diastolic Function and Aortic Prosthesis Function[a]

	Postoperative	1 Month	3 Months	6 Months	18 Months
LV mass index g/m^2	124.46	120.66	113.24	105.43	94.90
LV systolic function					
Fractional shortening (%)	45.1	44.8	42.2	48.5	39.7
Aortic valve function					
V2 V_{max} m/sec	2	1.9	1.7	2.1	2.1
Maximum gradient (mm Hg)	16	14.3	11.4	18.3	18

[a] Normal left ventricular mass (76 g/m^2 [12]).

LV = left ventricular; V2 = maximal velocity at aortic valve orifice.

disease that may have been functioning suboptimally prior to procurement. Mitral and tricuspid valve repairs [3] and coronary artery bypass grafting [4] have been used to reduce post-transplant tricuspid regurgitation, reduce known pre-procurement mitral regurgitation, and bypass known coronary artery disease.

Although mitral valve disease is a relative contraindication for cardiac allograft donation, aortic stenosis with significant LV hypertrophy has been considered an absolute contraindication. Valve replacement surgery is well described in patients during the early years post-transplant, presumably for progression of valvular disease present, but not hemodynamically significant at the time of transplantation [5]. However, the reported replacement of the aortic valve during the orthotopic cardiac transplant procedure is very rare. Aortic valve repair for moderate central aortic regurgitation has been reported during orthotopic transplant from a donor with normal LV function.

Aortic valve replacement during orthotopic heart transplant has been reported for new onset aortic regurgitation diagnosed at the conclusion of the implant [6]. The bicuspid valve was functioning normally at the time of procurement, and torsion of the aortic root was the postulated mechanism of the aortic regurgitation. In contrast to our patient, the donor heart in each of these 2 patients had not been exposed to the pathophysiology of chronic stenotic aortic valvular disease with its consequent LV remodeling.

The long-term survival of marginal organs is compromised. Allografts with evidence of obstructive coronary disease have a reduced long-term survival compared with nondiseased allografts and more commonly required revascularization [7].

The presence of LV hypertrophy has been shown to adversely affect both the short-term and longer-term survival of cardiac transplant recipients. Echocardiographic findings of LV hypertrophy have been correlated with high rates of primary allograft failure resulting in death, a requirement for mechanical circulatory support and re-transplantation [8].

Hypertrophic changes in the left ventricle are associated with suboptimal myocardial preservation and subsequent subendocardial ischemia [8]. Furthermore, cardiac ischemia and ischemia–reperfusion injury exacerbates the restrictive physiology seen in hypertrophic hearts. Ischemia–reperfusion injury leads to myocardial interstitial edema, cellular dysfunction, areas of necrosis that physiologically manifest as the restrictive pre-load dependent heart commonly seen post-cardiopulmonary bypass, and exacerbated by the longer periods of ischemia experienced during transplantation.

In the nontransplant cardiotomy, these inflammatory changes regress in time. In aortic stenotic hearts, adequate relief of any aortic outflow tract obstruction and control of systemic hypertension leads to near complete regression of LV mass. However, in the transplanted heart, LV hypertrophy often develops denovo postoperatively [9], related in part to cyclosporine, hypertension, and the altered neurohormonal state seen in transplant recipients. There is some evidence this can be ameliorated with medical therapy [10].

Despite appropriate medical management, donor LV hypertrophy, particularly with a history of hypertension has been show to decrease 1-year survival by 15% to 35% [11]. However, the prognostic influence of LV hypertrophy related to corrected aortic valve disease in the post- transplant setting, and whether it portends a similar prognosis to other causes of LV hypertrophy is unknown. The reduction in LV mass seen in the reported case suggests that some improvement will be seen in time, likely related to the correction of the LV outflow tract obstruction.

This case presentation indicates the feasibility of valve replacement during organ implant, with subsequent regression of LV hypertrophy during the early follow-up period. The patient's early post-transplant course was not marred by significant hemodynamic problems, and cardiac allograft function has remained preserved, with normal left and right ventricular systolic function and normal mechanical aortic valve function seen at 18-months post-transplant.

In conclusion, traditional selection criteria for cardiac allografts have strictly excluded those organs with known aortic valvular disease or significant LV hypertrophy. This report documents the transplantation of an organ requiring simultaneous mechanical aortic valve replacement for aortic stenosis. A successful short-term outcome with preserved allograft function, a well-seated mechanical prosthesis, and regression in LV mass without any major morbidity suggests that strict exclusion of organs because of known aortic valvular disease may not be justified in all cases. In patients in whom a marginal allograft is being considered, an offered heart with aortic valve disease that would require concomitant valve replacement does not preclude successful transplantation.

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FEATURE ARTICLES

Thoratec Left Ventricular Assist Device Removal After Toxic Myocarditis

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The clinical manifestation and natural history of myocarditis range is variable from asymptomatic stages to intractable circulatory compromise and death. Supportive therapy is paramount in the treatment of this condition. The use of mechanical circulatory support as bridge-to-recovery or bridge-to-transplantation in cases of cardiovascular collapse is often the only therapeutic option for these patients. We report the case of an adolescent boy with toxic myocarditis, due to cannabis abuse, who was supported with a Thoratec left ventricular assist device (Thoratec Laboratories Corp, Pleasanton, CA) for 96 days before device removal.

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The broad spectrum cause, clinical expression, and natural history of myocarditis, together with the difficulty in the accurate diagnostic evaluation of the disease, render this condition very challenging for the clinician. Supportive treatment with standard heart failure medicine including inotropic support and intra-aortic balloon counterpulsation, combined with hemodynamic monitoring is paramount in treating this condition. The use of mechanical circulatory support as bridge-to-recovery or bridge-to-transplantation in cases of cardiovascular collapse has emerged as a very effective treatment for these patients.

We report the case of a young boy with severe heart failure secondary to a drug-induced (cannabis) myocarditis who required support by a Thoratec left ventricular assist device (LVAD) (Thoratec Laboratories Corp, Pleasanton, CA). His cardiac function fully recovered 96 days later, and the LVAD could be successfully removed.

A previously healthy 16-year-old teenager (63 kg, 180 cm, body surface area 1.80 m^2) was admitted to our hospital after being diagnosed with acute left heart failure due to acute myocarditis. Transesophageal echocardiography showed a severely dilated left ventricle with an ejection fraction of 15% and global hypokinesis, moderate to severe mitral regurgitation, moderate pulmonary hypertension, small pericardial effusion, and a mobile left ventricular apical thrombus. The electrocardiogram recorded showed sinus tachycardia, right axis deviation, slow R-wave progression in leads V_{1-4} and negative T waves in leads II, III, aVF, and V_{4-6}. His initial blood investigation results, echocardiography measurements, and hemodynamic status are shown in Table 1. The usual screening for infectious myocarditis was negative, but the patient's urine testing for cannabinoids was positive. He was initially treated with intravenous diuretics, ACE inhibitors, and dobutamine. Despite maximal medical treatment, including levosimendan therapy, his clinical and hemodynamic status deteriorated 36 hours later with hypotension, cardiac index of 1.60 L/min/m^2 and cardiogenic shock, with threatening multiple organ failure. The patient was evaluated and accepted for ventricular assist device therapy.

In cardiopulmonary bypass, the inflow cannula was inserted in the apex of the left ventricle after removal of the apical thrombi; the outflow cannula was inserted into the ascending aorta. The Thoratec LVAD (Thoratec Lab-

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