CASE REPORTS

Regression of left ventricular hypertrophy in a transplanted heart

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Abstract

A woman with advanced coronary artery disease underwent heart transplantation. The donor heart had left ventricular hypertrophy. The electrocardiographic and echocardiographic evidence of left ventricular hypertrophy regressed during follow up; estimated

left ventricular mass decreased from 393 g to 171 g. The adaptation of myocardial mass and performance after transplantation is not fully understood. This case illustrates the potential for regression of left ventricular hypertrophy in response to altered loading conditions.

Figure 1 12 lead electrocardiograms: (A) Immediately after transplantation, (B) 25 months after transplantation. Comparison shows a reduction in left ventricular hypertrophy.

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Figure 2 *M Mode echocardiograms at the level of the left ventricle: (A) Immediately after transplantation, (B) 25 months after transplantation. Comparison shows a considerable reduction in left ventricular posterior wall and septal thickness.*

The human myocardium responds to changes in demand upon it by changes in myocardial mass. The capacity of the transplanted heart to respond in this way is not well documented. A case is described in which the features of left ventricular hypertrophy in a donor heart regressed within a few months of transplantation.

Case report

A 53 year old woman with advanced heart failure secondary to coronary artery disease underwent orthotopic cardiac transplantation in November 1988. She received the heart of a 29 year old man of 71 kg who had died of a subarachnoid haemorrhage. He had had no medical history. His chest x ray was normal. When the organ was harvested it was noted to be bulky with left ventricular hypertrophy but no evidence of aortic stenosis. It was assumed by the donor team that the changes must have been due to previous hypertension, although his blood pressure recorded on the intensive care unit had been normal.

Standard orthotopic transplantation was carried out without complications and subsequent progress of the recipient has been uneventful; she remains well two and a half years after transplantation. She has had a single episode of moderate rejection on biopsy (grade 3a), which quickly resolved with a short period of augmented oral steroids. Her immunosuppressive regime is; 125 mg of cyclosporin twice daily and 5 mg of prednisolone and 150 mg of azathioprine once daily. She takes 10 mg of nifedipine and 40 mg of sotalol twice daily for cyclosporin induced hypertension and recurrent atrial tachyarrhythmias.

Electrocardiogram and echocardiogram immediately after the transplantation (figs 1 and 2) showed left ventricular hypertrophy

with a left ventricular mass of 393 g (estimated from the cube rule¹). Over several months these changes resolved with the electrocardiogram being normal at nine months. Her most recent electrocardiogram and echocardiogram are also shown in figs 1 and 2. The current estimate of left ventricular mass is 171 g.

Discussion

Normal myocardium responds to changes in demands upon it by changes in mass. Hypertrophy can occur quickly in experimental animals one to six weeks^{2,3} and this has also been shown in studies on the response of human myocardium to training⁴ and pregnancy,⁵ when again echocardiographic measurements of left ventricular mass show an increase within a period of weeks.

In transplantation it is unlikely for recipients to receive a donor heart that is exactly matched to their needs and therefore some degree of hypertrophy or involution might be expected. Little work has been done to show that this does in fact occur. Angermann *et al* have shown that the transplanted myocardium can both hypertrophy and regress in response to recipient hypertension and its treatment.⁶ Hosenpud *et al* compared donor:recipient weight ratio at three months after transplantation and found that although normal cardiac output is maintained in proportion to recipient weight, in patients who have received a heart from a lighter donor this is achieved at the expense of a higher heart rate and resting pulmonary capillary wedge pressure.⁷ This suggests that the myocardium has either failed to adapt, or has done so only partially by three months after transplantation. It has been suggested that this relation does not persist at 12 months,⁸ which would be in keeping with the idea of gradual adapta-

tion of the donor heart to its new loading conditions over a period of three to 12 months. A recent report retrospectively assessed the effect of donor:recipient weight mismatch on outcome and suggested that, contrary to popular belief, oversizing (that is a donor weight in excess of recipient ideal weight) results in a poorer outcome.⁸ There was a continuous (negative) relation between degree of oversizing and survival. It was also concluded that undersizing was not detrimental to allograft function and survival.

Further work on the correlation between size match and ultimate outcome is required to define the optimal limits of size matching.

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