Wolff-Parkinson-White syndrome after transplantation of the heart

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SUMMARY The classic features of Wolff-Parkinson-White syndrome developed in a 49 year old man after he was given the heart of a donor who had had a normal electrocardiogram. The recipient showed type A pre-excitation on the surface electrocardiogram and clinically important paroxysmal supraventricular tachycardia. An electrophysiological study showed a left sided accessory pathway and dissociation between donor and recipient atrial activity during tachycardia. The arrhythmia was controlled by flecainide.

The suitability of heart donors is based on clinical examination, electrocardiogram, chest x ray, and echocardiogram.1 A careful history taken from a member of the donor's family will help to assess the likelihood of a cardiovascular abnormality that might not be detected by these tests. We report the case of a patient who was given the heart of a symptomless donor who had had a normal electrocardiogram. The recipient subsequently presented with Wolff-Parkinson-White syndrome.

Case report

In September 1986, a 49 year old white man was accepted for heart transplantation. He had severe dilated cardiomyopathy caused by ischaemic heart disease and was in severe heart failure (New York Heart Association class IV). Heart transplantation was performed at the University Hospital of Zurich on 2 February 1987. The donor, a white man aged 19, had irreversible severe cerebral damage after a car accident. The donor did not have a history of cardiovascular disease and according to relatives had never had palpitations. His electrocardiogram showed non-specific ST segment changes, without any signs of pre-excitation (fig 1a); the chest x ray and echocardiogram were within normal limits. Heart transplantation was performed and the patient was extubated on the second postoperative day. The immediate postoperative electrocardiogram showed normal sinus rhythm with sinus tachycardia and non-specific ST segment modification. On day 7 he had an infection caused by Pseudomonas aeruginosa; this was successfully treated with antibiotics. The patient was maintained on azathioprine 100 mg/daily and cyclosporin 220 mg/daily.

When he first attended as an outpatient for myocardial biopsy he complained of two or three episodes of dizziness that had coincided with a rapid heart rate. The clinical examination was normal apart from a high blood pressure of 170/110 mm Hg. The electrocardiogram surprisingly showed typical patterns of Wolff-Parkinson-White syndrome (PR interval 80 ms) (type A) with a delta wave axis that was compatible with a left sided accessory pathway (fig 1b).

An electrophysiological investigation was performed. The atrioventricular conduction intervals were normal, as were effective refractory periods of the atrioventricular node and atrium (AH = 90 ms, HV = 50 ms, H-delta = 10 ms). Orthodromic tachycardia (cycle length 300 ms) was easily induced by two atrial extrasystoles. The refractory period of the accessory pathway was 380 ms for anterograde conduction and 260 ms for retrograde conduction. During tachycardia the earliest atrial electrocardiogram was recorded in the distal coronary sinus confirming the left sided site of the accessory pathway. In sinus rhythm and during tachycardia (fig 2a) two atrial depolarisations could be seen (A1 and A2). A1 represents the depolarisation of the remaining part of the recipient atrium and A2 is the depolarisation of the donor atrium.

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Fig 1  (a) Twelve lead electrocardiogram recorded in the donor. (b) Twelve lead electrocardiogram showing the typical patterns of Wolff-Parkinson-White syndrome.
Fig 2  (a) Endocavitary electrocardiogram showing two different atrial depolarisations, A1 and A2. A1 is the signal of the recipient atrium and A2 is the depolarisation of the donor atrium. HRA, high right atrium electrocardiogram; HBE, His bundle electrocardiogram. (b) Endocavitary electrocardiogram during orthodromic reentrant tachycardia. A1 (recipient atrium) was not part of the circuit A2 (donor atrium) and was retrogradely activated. The earliest site of retrograde activation was in the coronary sinus. RA, high right atrium electrocardiogram; HBE, His bundle electrocardiogram; CS, coronary sinus.
heart was involved in the reentry circuit, while the recipient heart remained in normal sinus rhythm with sinus tachycardia (fig 2b). Episodes of tachycardia were poorly tolerated and required conversion to sinus rhythm after 30 to 60 seconds with programmed atrial stimulation. Atrial fibrillation was not induced. Amiodarone 600 mg/daily was prescribed, but was stopped a month later when tachycardia recurred and there were side effects. The patient was successfully treated with flecainide (100 mg twice a day), which controlled the symptoms and suppressed episodes of spontaneous paroxysmal tachycardia.

Discussion

As the number of heart transplants increases donor-transmitted cardiac disease, although rare, is likely to occur. Wolff-Parkinson-White syndrome is found in 0.1–0.2% of the general population,6 and this case emphasises the need for taking a thorough history of the donor and to have several recent and, if possible, earlier electrocardiograms. If doubt persists about the presence of an accessory pathway, limited atrial and ventricular stimulation should be performed before removal of the heart.

Patients with accessory pathways should not be considered as heart donors, even if they do not have a history of palpitation. Supraventricular tachycardia may be incapacitating or dangerous to the recipient. Moreover, as this case shows, tachycardia and evidence of accessory pathways may become apparent only after transplantation. The explanation of this phenomenon remains unclear. It is theoretically possible that conducting pathways might develop between the donor and recipient atria, and might form the basis of a reentry circuit.

However, in our patient the dissociated activity in the recipient atria rules out this possibility. A further possibility is that intermittent pre-excitation might be present in the donor heart—this phenomenon is well known in the Wolff-Parkinson-White syndrome especially with left sided accessory pathways.3 The location and the relatively long anterograde refractory period may explain the absence of pre-excitation in the donor heart. After transplantation vagal control was lost but the response to sympathetic nervous stimuli persisted, resulting in shortening of the refractory periods of the accessory pathway.4 Thus pre-excitation occurred and orthodromic reciprocating tachycardia could be initiated.

References