Late Atrioventricular Block After Heart Transplantation

Yen-Wen Wu,1 Wen-Jone Chen,1 Nai-Kuan Chou,2 Shoei-Shen Wang,2 and Yuan-Teh Lee1

Abstract: Bradyarrhythmia requiring permanent pacing after heart transplantation remains a common problem. Sinus node dysfunction is the most common indication, and late onset of atrioventricular (AV) block has rarely been reported. We report the case of a patient who developed advanced AV block at 41 months after transplantation. Right bundle branch block with progressive increase of QRS complex duration was noted in serial electrocardiograms. At the time of late AV block development, the patient did not have acute rejection and coronary angiogram was normal. The mechanism of late onset of AV block is unclear, but it may be caused by progressive conduction.

Key words: Heart transplantation; Bundle-branch block


Bradyarrhythmia requiring permanent pacemaker implantation after heart transplantation has been reported to occur in 4 to 29% of transplant recipients, and its most common indication is sinus node dysfunction.1-5 Atrioventricular (AV) block, especially with late onset after heart transplantation, has rarely been reported.6-9 There is limited information regarding this condition.

A total of 163 patients received cardiac transplant at our medical center between July 1987 and December 2001, and permanent pacemaker implantation was required in 5 patients, including 1 for sinus node dysfunction and 4 for AV block. Here we report 1 of these cases that developed symptomatic high-degree AV block 41 months after heart transplantation.

Case Report

A 63 year-old man underwent orthotopic heart transplantation at age 59 years because of ischemic cardiomyopathy with severe congestive heart failure. He had a 10-year history of diabetes mellitus, and was a heavy smoker (1 pack per day for 30 years). The post-transplant course was unremarkable for 41 months. One day before admission, sudden onset of dizziness and many episodes of near-syncope and syncope developed. He visited our outpatient clinic and marked bradycardia (rate less than 40/min) was noted. Telemetry monitoring showed high-degree AV block with slow escape rhythm, and he was hospitalized immediately for temporary pacemaker insertion. Patent coronary angiograms and no elevation of cardiac enzymes were documented. Echocardiography showed normal regional wall motion and cardiac size with good systolic contractility (left ventricular ejection fraction, 65%).

Endomyocardial biopsies found no evidence of acute rejection. Nevertheless, foci of old subendocardial scarring with fat infiltration were noted. A permanent dual-chamber rate adaptive (DDDR) pacemaker was implanted on the fourth day due to persistent bradycardia.

The donor heart was from a 38-year-old man without any history of systemic disease who was a hepatitis B virus carrier. The ischemic time was 119 minutes. The bypass time was 165 minutes and total operation time was 420 minutes. The patient was followed weekly in the first 4 to 6 weeks post-transplant, and then once per year according to the post-transplant follow-up schedule in our hospital. The most severe rejection episode was grade II according to the International Society for Heart and Lung Transplantation scale10 occurring at 6 months after transplantation. The pathology reports of serial biopsies are listed in Table 1.

At 2 weeks postoperatively, surface 12-lead electrocardiograms showed incomplete right bundle branch block (RBBB), and the width of QRS complex duration...
was 96 ms. Complete RBBB morphology with progressive prolongation of QRS complex duration was noted thereafter (Fig.). He did not take any medication postoperatively which might have resulted in bradycardia, such as amiodarone, or a β-blocker or calcium channel blocker. He was symptom-free at 10 months after pacemaker implantation.

### Discussion

Heart transplantation is considered standard therapy for end-stage heart disease and permanent pacing is an important issue in this patient population. The majority of donor hearts are functionally depressed and require rate support with positive chronotropic agents or temporary pacing within the first 24 hours following transplantation. Previous studies have reported prolonged bradyarrhythmias in 14 to 44% of transplant recipients, and permanent pacemaker implantation was indicated in 4 to 29%. The causes of bradyarrhythmias have been considered to be related to prolonged graft ischemic time, surgical trauma, abnormal torsion of donor sinoatrial nodal artery, older donor age, cardioplegia and organ storage temperature, total bypass time, preoperative use of amiodarone or the presence of rejection. Sinus node dysfunction is predominant in the early post-transplant period, and many of these patients recover spontaneously. The incidence of bradyarrhythmias occurring later than 1 month after heart transplantation is quite low, and its pathogenesis is not clear (Table 2). Most of the reported late bradyarrhythmias were AV block. In the absence of autonomic innervation in post-transplant hearts, long pause or extreme bradycardia might occur when sinus or AV nodal dysfunction develops, and the risk of syncope or sudden death is much higher.

Conduction abnormalities following heart transplantation are not rare, occurring in 12 to 79% of patients in previous reports, with RBBB the most common. Jessen et al reported that the outcome of heart transplantation was not influenced by conduction abnormality. However, in a retrospective case-control study of 87 post-transplant recipients, a higher mortality rate was found in patients with progressive conduction defects. Sudden death associated with complete heart block (2 patients) or ventricular arrhythmias (3 patients) was exclusively confined to those with progressive conduction defects.

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**Table 1. Serial follow-up of endomyocardial biopsy.**

<table>
<thead>
<tr>
<th>Postoperative follow-up</th>
<th>11 days</th>
<th>18 days</th>
<th>27 days</th>
<th>46 days</th>
<th>3 months</th>
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<tr>
<td>ISHLT scale</td>
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<td>0</td>
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</table>

<table>
<thead>
<tr>
<th>Postoperative follow-up</th>
<th>6 months</th>
<th>1 year</th>
<th>2 years</th>
<th>3 years</th>
<th>41 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISHLT scale</td>
<td>II</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</tbody>
</table>

ISHLT = International Society for Heart and Lung Transplantation.

**Fig. Serial electrocardiograms:**

A) before transplantation (PR = 164 ms, QRS = 92 ms); B) pre-discharge ECG (18th postoperative day; PR = 156 ms, QRS = 96 ms); C) 1 year after transplantation (PR = 136 ms, QRS = 112 ms); and D) 3 years after transplantation (PR = 149 ms, QRS = 124 ms).
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All of these patients had documented coronary artery disease or myocardial infarction. Progression of RBBB, particularly if it occurs during the first year post-transplant, is a sign of poor prognosis.15

At the time of late AV block development, this patient did not have acute rejection, and the coronary angiograms were normal. Focal fibrosis and fat infiltration was found in the endomyocardial biopsies. The presence of progressive RBBB might suggest the development of progressive conduction defects after heart transplantation. In this patient, the heart rate increased post-transplant (to around 90 to 110/min). However, there is limited information concerning the possibility of rate-dependent RBBB and the relationship between PR interval and QRS duration change in the denervated transplanted heart.

No uniform predictors of late onset AV block following transplantation have been identified, and it is unclear if progressive RBBB is an early indicator of late AV block due to the limited case numbers in previous studies. Further investigation of progressive conduction abnormalities in patients after heart transplantation is needed to explore the mechanism of late onset bradyarrhythmia.

References


Table 2. Characteristics of patients in previous reports of atrioventricular block occurring in the late post-transplantation period.

<table>
<thead>
<tr>
<th>Report</th>
<th>Number of patients</th>
<th>Onset</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Miyamoto et al (1990)1</td>
<td>6/401</td>
<td>5 to 31 months</td>
<td>Three cases with acute rejection and all 6 cases recovered normal sinus rhythm</td>
</tr>
<tr>
<td>Weinfeld et al (1996)7</td>
<td>11/213</td>
<td>1 to 10 years</td>
<td>Acute rejection (-)</td>
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<tr>
<td>Cataldo et al (1996)8</td>
<td>2/200</td>
<td>28, 30 months</td>
<td>Acute rejection (-)</td>
</tr>
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<td>This report</td>
<td>1/163</td>
<td>41 months</td>
<td>Acute rejection (-)</td>
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