

Permanent Pacemaker for Syncope after Heart Transplantation with Bicaval Technique

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Sinus node dysfunction occurs occasionally after heart transplantation and may be caused by surgical trauma, ischemia to the sinus node, rejection, drug therapy, and increasing donor age. However, the timing and indication of permanent pacemaker insertion due to sinus node dysfunction following heart transplantation is contentious. Here, we report a case of a permanent pacemaker insertion for syncope due to sinus arrest after heart transplantation, even with a bicaval technique, which has been known to associate with few incidences of sinus node dysfunction.

Key Words : Permanent pacemaker, sinus arrest, heart transplantation

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INTRODUCTION

In the past, permanent pacemaker implantation is required in a large number of transplantation patients principally because of sinus node dysfunction of the donor atrium. However, the need for permanent pacemaker implantation after transplantation was nearly eliminated as surgical technique changed from standard biatrial anastomosis to bicaval anastomosis.¹ This new bicaval anastomosis is technically simple and preserves the anatomic size, geometric configuration and physiologic function of the atria.² Since 1992, we have performed bicaval technique in our all heart transplantation patients at our cardiovascular center. In this report, we present our first case of a permanent pacemaker insertion for syncope due to sinus arrest after heart transplantation, even with bicaval technique.

CASE REPORT

A 44 year-old male visited our hospital because of shortness of breath and edema in both legs. He underwent prosthetic mitral valve replacement with Saint-Jude #27 due to severe mitral stenosis in 1992. He had no history of hypertension, diabetes mellitus and other diseases. On physical examination, enlarged liver was palpable with icteric sclera and neck veins were engorged. Metallic valve click sounds were audible without murmurs. Chest radiography showed marked cardiomegaly and no evidence of pulmonary edema. All laboratory findings were within normal limits except for mild elevation of total bilirubin level (2.7 mg/dL, normal range: 0.2 - 1.2 mg/dL). Abdominal sonography showed no remarkable

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findings. Echocardiography showed markedly enlarged cardiac chambers with severely reduced left ventricular (LV) systolic function [ejection fraction (EF) = 8%] and well functioning prosthetic mitral valve. Despite optimal medical treatment, including diuretics, digoxin, angiotensin converting enzyme inhibitors and inotropics, his symptoms were not relieved. He underwent heart transplantation with bicaval technique on 15th, July, 2007. The immunosuppressive regimen consisted of cyclosporine (target blood trough level: 250 - 350 ng/mL), mycophenolate mofetil and prednisolone. The blood trough level of cyclosporin was 323 ng/mL. Follow-up echocardiography after heart transplantation showed normal LV systolic function (EF = 60%). He felt mild dizziness with sinus bradycardia (< 50 beats/min) during hospital stay. On day 22, he had an event of syncope in the early morning that was due to prolonged sinus arrest (10 seconds), which was

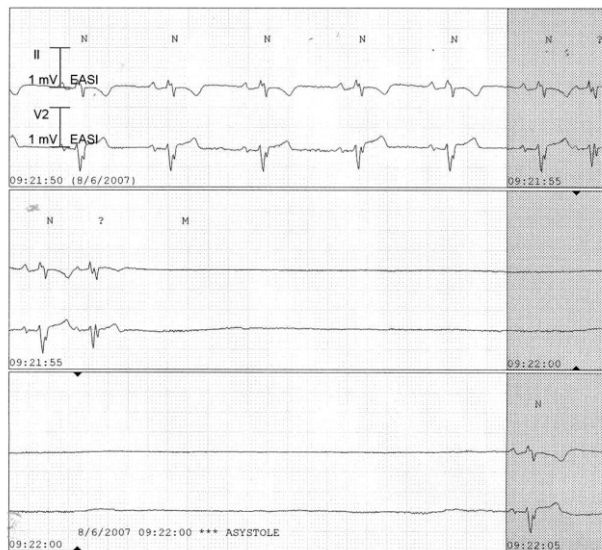


Fig. 1. Sinus arrest was captured for 10 seconds on telemetry on day 22 after heart transplantation.

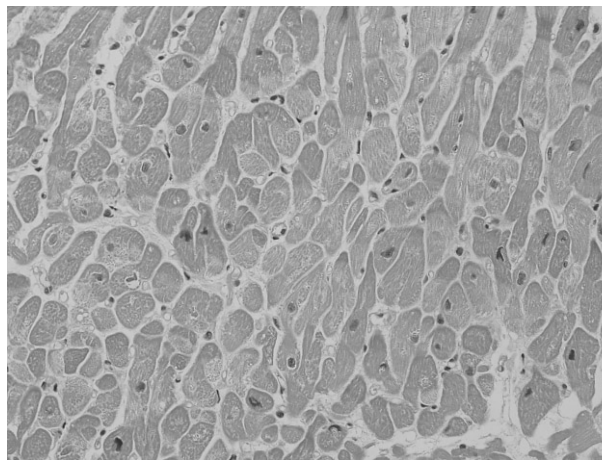


Fig 2. Endomyocardial biopsy showed no evidence of rejection. Normal myocardium without lymphocytic infiltration or myocyte damage.

confirmed on telemetry (Fig. 1). The endomyocardial biopsy specimen showed no rejection, according to International Society for Heart Transplantation Classification (Fig. 2). Follow-up echocardiography showed no significant interval changes. After temporary pacemaker insertion, he had still symptomatic bradycardia (< 50 beats/min) and sinus arrest (8 second). Finally, he underwent insertion of a permanent VVI pacemaker on day 32. He was discharged without symptoms and was followed at outpatient clinic.

DISCUSSION

The standard heart transplantation technique originally described by Lower and Shumway involves removal of both donor and recipient hearts at the midatrial level.³ This biatrial technique preserves the multiple pulmonary venous connections to the recipient's left atrium. The great vessels are resected just above the semilunar valves. This technique has been associated with postoperative problems in some patients, including atrial dysrhythmias, atrial dysfunction, thrombus formation, and tricuspid valve dysfunction. In the early 1990s, a variation of the standard procedure, called the bicaval technique was introduced.^{4,5} This procedure uses resection of the superior and inferior vena cavae, preserving the integrity of the right atrium. The theoretical advantages of this technique are the preservation of atrial contractility, sinus node dysfunction, and tricuspid valve competence. However, Meyer et al.⁶ reported disadvantages of bicaval technique such as prolonged cross-clamp and donor ischemic time.

The donor was a 37-year-old male who was sentenced to brain death due to acute cerebral hemorrhage. He had a history of hypertension on anti-hypertensive medication. The ischemic time of donor's heart was 180 minutes. Our patient successfully underwent heart transplantation by bicaval technique, and no evidence of rejection was then noted on endomyocardial biopsy and echocardiography. Perhaps, the most possible cause of sinus node dysfunction in this patient was surgical trauma at the time of transplantation. Although normalization of posttransplantation sinus node dysfunction and bradyarrhythmias occurs in up to 55 percent of patients during the first three postoperative months,⁷ patients with more severe symptomatic bradycardia that persists for more than two weeks or sinus arrest after transplantation usually require a permanent pacemaker.⁸ Rothman et al.⁹ noted sinus node dysfunction in 14 (42%) of 33 patients who underwent biatrial anastomosis and 2 (5%) of 37 patients who underwent bicaval anastomosis. Because sinus node dysfunction in the transplanted heart does not predict subsequent development of atrioven-

tricular node dysfunction, rate-responsive atrial pacing should be considered in majority of cases. However, considering the possibility of surgical trauma around superior vena cava at the time of transplantation in our case, we decided single lead pacemaker insertion (VVI) to prevent the opportunity of complication such as thrombosis.

In conclusion, the present case represents a permanent pacemaker insertion for syncope due to sinus arrest after heart transplantation, even with bicaval technique, which has been known to associate with few incidences of sinus node dysfunction.

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