Late atrioventricular block and permanent pacemaker implantation after heart transplantation

Kalp nakli sonrası geç dönemde atriyoventriküler blok ve kalıcı kalp pili uygulaması

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The need for permanent pacemaker implantation due to late atrioventricular (AV) block after heart transplantation is rare. A 59-year-old male patient underwent heart transplantation. He presented with syncope eight months after transplantation. Ambulatory 24-hour Holter monitoring showed predominant sinus rhythm with a mean heart rate of 74 bpm, intermittent second-degree AV block, and high-degree AV block with pauses of up to 10.6 seconds. Percutaneous transvenous endomyocardial biopsy yielded a histologic diagnosis of grade IA rejection according to the ISHLT (International Society of Heart and Lung Transplantation) scoring system. A permanent pacemaker with DDD-R mode was implanted via the left subclavian vein, and he was discharged on the following day without any complication.

Key words: Bradycardia; heart block/therapy; heart transplantation; pacemaker, artificial; postoperative complications.

Approximately 2100 heart transplants are carried out in the United States each year and up to 21% of heart transplant recipients further receive permanent pacemakers.[1,2] Suspected causes of bradyarrhythmias requiring pacing include prolonged ischemic time, allograft rejection, sinus node dysfunction, damage to the sinus node of the donor heart at the time of transplant, and amiodarone use before transplantation.[3,4]

Although pacing offers heart transplant recipients the probability of a shorter postsurgical recovery time and an earlier initiation of cardiac rehabilitation, permanent pacing has not been shown to improve long-term survival.[5] It has even been argued that pacing in heart transplant recipients is used excessively.[5] Many patients recover from sinus node dysfunction without pacing within several weeks or months of receiving a heart transplant.[6,7]

We report here a heart transplant recipient who developed high-degree atrioventricular (AV) block causing a pause of up to 10.6 seconds.

CASE REPORT

A 59-year-old male with a history of dilated cardiomyopathy received a biventricular implantable cardioverter-defibrillator (CRT-ICD) in 2004 because of decompensation of heart failure and nonsustained ventricular tachycardia. In 2007, he presented with worsening functional capacity, for which echocardiography, selective coronary angiography, and right heart catheterization were performed. Transthoracic
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Echocardiography showed global hypokinesia, ejection fraction of 15%, and moderate mitral and mild aortic regurgitations. On angiography, coronary arteries were normal. During catheterization, pulmonary capillary wedge pressure was 34 mmHg, systolic pulmonary artery pressure was 77 mmHg, cardiac output was 2.34 l/min (Fick's method), and pulmonary vascular resistance was 4 Wood units. A decision for heart transplantation was made. In 2008, successful heart transplantation was performed and postoperative follow-up was uneventful. However, he presented with syncope eight months after transplantation. Physical examination findings, electrocardiogram, and chest radiography were unremarkable. Ambulatory 24-hour Holter monitoring showed predominant sinus rhythm with a mean heart rate of 74 bpm, intermittent second-degree AV block, and high-degree AV block with pauses of up to 10.6 seconds (Fig 1). Percutaneous transvenous endomyocardial biopsy was performed, which yielded a histologic diagnosis of grade IA rejection according to the ISHLT (International Society of Heart and Lung Transplantation) scoring system. A permanent pacemaker with DDD-R mode was implanted via the left subclavian vein, and he was discharged on the following day without any complication.

DISCUSSION

Bradyarrhythmia occurs in 64% of recipients in the first few weeks after cardiac transplantation, but often resolves spontaneously.[5,8] Persistent bradyarrhythmia may require permanent pacemaker implantation. Reported rates of permanent pacemaker implantation range from 8% to 24% at different transplant centers.[5,8,10]

Late requirement of permanent pacing after cardiac transplantation is rare,[7] with AV block being the most common reason.[9] Woo et al.[9] found a higher incidence of heart block in patients who required pacing beyond six months after transplantation. Approximately, 24% of the patients required pacing within the first month after transplantation. Of 11 patients who required pacing beyond six months, four had evidence for transplant vasculopathy.

The incidence of post-transplant AV block has rarely been reported. Cui et al.[12] reported that, of 1,047 patients, 113 patients developed AV block following heart transplantation. The most common isolated AV block on the post-transplant ECGs was first-degree AV block in 87 patients, accounting for 8.4%.

Miyamoto et al.[10] found that 72 (18%) of 401 adult orthotopic heart transplant recipients developed prolonged bradyarrhythmias within five days after transplantation. Permanent pacemaker implantation was performed in 17 patients within 40 days of transplantation. Only six patients received a permanent pacemaker between 5 and 31 months after transplantation. These patients had sinus rhythm at the time of discharge, but later developed bradyarrhythmia. Of these, three cases were associated with rejection, but three were not. All recovered to sinus rhythm after permanent pacemaker implantation. What causes late brady-
cardia in the absence of rejection is unknown. Partial rejection that cannot be detected by routine right ventricular biopsy, fibrosis, or temporary decreases in blood supply around the sinus node and conduction system may result in bradycardia.

Avitall et al.\cite{13} showed in the transplanted canine heart that allograft rejection first appeared in the right atrium and was much more severe in the atrium than in the ventricle. They proposed that the conduction tissue, including the sinus node and AV node, was a special target for allograft rejection, and that right atrial lymphocyte infiltration, myocyte necrosis, and fibrosis associated with acute or chronic rejection might contribute to intra- and inter-atrial conduction disturbances. Cooper et al.\cite{14} reported that eight of 20 pacemaker receivers were associated with episodes of rejection. Our case also had ISHLT grade 1A rejection and required late pacemaker implantation.

In conclusion, sinus node dysfunction mainly occurs during the early period of orthotopic heart transplantation and may be associated with surgical trauma, ischemic sinus node dysfunction, rejection, drug therapy, and increasing donor age; however, AV conduction abnormalities, which are far less common, generally occur late after transplantation and require lifelong permanent dual-chamber pacing.

**REFERENCES**