Atrioventricular Nodal Ablation and Biventricular Pacing Therapy with Coronary Venoplasty for Severe Heart Failure with Drug Refractory Atrial Tachycardia

Seiji Hokimoto¹, Hiroki Usuku², Takeshi Tsuchiya³, Shuichi Oshima⁴, Hiroshi Kugimiya⁵ and Hisao Ogawa²

Abstract

A 53-year-old man was admitted to our hospital for dyspnea, associated with atrial tachycardia. He underwent mitral valve replacement and Maze operation for mitral regurgitation and atrial fibrillation and since then he suffered from drug refractory atrial tachycardia followed by cardiogenic shock with systolic heart failure. Atrial tachycardia with rapid ventricular response was medically refractory, and radical catheter ablation was thought to be very difficult due to post Maze operation, mitral mechanical valve replacement and unstable hemodynamics. Thus, atrioventricular nodal ablation, which was safe procedure compared to the radical ablation for this patient, and biventricular pacemaker implantation, which required coronary venoplasty, were performed. Combination therapy may be one of the treatments for heart failure patients with drug refractory tachyarrhythmias.

Key words: tachyarrhythmias, heart failure, ablation-catheter, pacing

Introduction

It has been demonstrated that combination therapy of atrioventricular (AV) nodal ablation and right ventricular (RV) pacing is effective for drug refractory tachyarrhythmias to allow control of the ventricular rate. However, RV apical pacing may create abnormal left ventricular (LV) contraction, or deteriorate the failing heart.

We report a case of treatment for heart failure with drug refractory atrial tachycardia.

Case Report

A 53-year-old man was transferred to our hospital due to aggravated dyspnea and faintness. Six months prior to this admission, the patient underwent mitral valve replacement (mechanical heart valve, SJM29) and Maze operation due to severe mitral regurgitation and atrial fibrillation. At that time, he was found to have atrial fibrillation and left ventricular (LV) dysfunction with an LV end-diastolic dimension of 80 mm. The LV ejection fraction was 35% and the left atrium (LA) dimension was 65 mm. Post operation, he suffered from atrial tachycardia followed by cardiogenic shock. The patient was put on amiodarone, carvedilol, digitoxin, or verapamil orally, however, atrial tachycardia with rapid ventricular response and exacerbation of heart failure (HF) with cardiogenic shock and faintness occurred repeatedly.

On admission, his blood pressure was 84/56 mmHg, and heart rate 130 beats/min. Physical examination showed moist rale in bilateral lung fields and a grade II/IV systolic murmur at the apex. An electrocardiogram revealed atrial tachycardia (130 beats/min) and the QRS interval of 120 msec (Fig. 1). A chest X-ray film demonstrated cardiomegaly (cardio-thoracic ratio, CTR 59%) and pulmonary

¹Department of Interventional Cardiology, Division of Coronary Intensive Care Unit, Kumamoto University Hospital, Kumamoto, ²Department of Cardiovascular Medicine, Graduate School of Medical Sciences, Kumamoto University, Kumamoto, ³EP Expert Doctors-Team Tsuchiya, Kumamoto, ⁴Division of Cardiology, Kumamoto Central Hospital, Kumamoto and ⁵Bethesda Clinic, Miyakonojo

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Correspondence to Dr. Seiji Hokimoto, seisan@momo.so-net.ne.jp
congestion. Echocardiography revealed diffuse severe hypokinesis of LV with ejection fraction of 14%. The LV end-diastolic dimension was 79 mm, and LA dimension of 60 mm.

Atrial tachycardia with 1:1 AV conduction (130 beat/min) was drug refractory, and intravenous administration of quinidine sulfate, procainamide hydrochloride, disopyramide, or pilsicainide hydrochloride was not effective for termination of atrial tachycardia. Radical catheter ablation for atrial tachycardia was thought to be very difficult due to post Maze and mitral valve replacement operation (mechanical valve) and unstable hemodynamics with temporal syncope. Thus, AV nodal ablation, which was a safe procedure compared to the radical ablation for this patient with hemodynamic compromise and mechanical mitral valve, and pacemaker implantation were selected. However, RV apical pacing may create abnormal LV contraction, or deteriorate the setting of heart failure. Thus, biventricular pacing was chosen.

During the biventricular pacemaker (Medtronic InSync Model 8040; Medtronic Inc., Minneapolis, MN, USA) implantation procedure, the over-the-wire (OTW)-type LV lead (Medtronic Attain 4193) did not pass through the lateral vein due to stenosis (Fig. 2A, B). Thus, venoplasty was carried out by dilatation using a 2.0 mm coronary balloon catheter, however the LV lead was not crossed. The trial of placement in the lateral site of LV through the midcardiac vein was also a failure because the guide sheath was not inserted into the orifice of midcardiac vein. Thus, the anterolateral vein was selected as the target vein, and dilated for severe stenosis with a 1.5-mm coronary balloon (Fig. 2A, C). Finally, the LV lead was placed in the anterolateral vein (Fig. 2D). Later, AV nodal ablation was performed and biventricular pacing was initiated. Comparing
Cardiac venograms during the biventricular pacemaker implantation procedure. A: Overall picture of cardiac vein, showing anterolateral vein stenosis (white arrowhead), and lateral vein (black arrowhead). B: Stenosis of proximal site of lateral vein (black arrowhead). C: Balloon dilatation for anterolateral vein. D: Deployment of left ventricular pacing lead.

the cardiac indices after ablation and pacing to those before treatment, CTR in chest X-ray was from 59% to 56%, ejection fraction from 15% to 20%, LV end-diastolic dimension from 79 to 73, LA dimension from 60 mm to 58 mm, B type natriuretic peptide from 1350 pg/mL to 590 pg/mL, and NYHA class from IV to II. There was no hospitalization due to exacerbation of heart failure for a follow-up period of 2 years.

Discussion

Cardiac resynchronization therapy (CRT) has been shown to reduce mortality and hospitalizations as well as to improve LV ejection fraction, exercise tolerance and quality of life in patients with heart failure (1, 2). On the other hand, combination therapy of AV nodal ablation and pacing is reported to be effective for patients with drug refractory tachyarrhythmia (3).

In the present case, atrial tachycardia with rapid ventricular response was refractory to conventional therapy including drugs and defibrillation. Radical ablation for this patient was very difficult due to post Maze operation, mechanical valve replacement, and unstable hemodynamics. The control of the ventricular rate may be important and result in stable hemodynamics, however, it was not effective to control the heart rate with antiarrhythmic agents such as amiodarone, carvedilol, digoxin, and verapamil, thus AV nodal ablation and pacing therapy was selected.

Dual-chamber pacing preserves AV synchrony and may reduce heart failure compared with RV pacing in patients with sick sinus syndrome. However, RV apical pacing sometimes results in prolonged QRS duration and ventricular desynchronization. Despite maintenance of AV synchrony, ventricular pacing conferred an increased risk of heart failure hospitalization compared with less pacing (4). Moreover, it was demonstrated that RV pacing may be deleterious in patients with advanced LV dysfunction in DAVID study (5). Consequently, the choice of biventricular pacing was rational in this patient, who had severe LV dysfunction, although dyssynchrony was not detected by tissue doppler echocardiography. After ablation and pacing, QRS duration was 200 msec, and 160 msec in RV and biventricular pacing, respectively (Fig. 3A, B), and septal-lateral delayed time in LV was 84 msec, and 17 msec, respectively. In addition, cardiac indices, such as CTR, ejection fraction, LV end-diastolic dimension, LA dimension, and B type natriuretic peptide, improved after treatment. These results support that the selection of biventricular pacing was rational.

The present case needed coronary vein balloon angioplasty, although it is unknown whether Maze and mitral
valve replacement during thoracotomy was associated with coronary vein stenosis or not. It has been demonstrated that coronary venoplasty is a safe and effective technique to permit LV lead insertion in cases of target vein stenosis (6, 7). In the present case, cardiac vein angioplasty for the lateral vein as the first target site was performed, but the LV pacing lead did not pass probably because of incomplete dilatation, dissection, or poor back up support of the guide sheath. The LV pacing lead was finally placed in the anterolateral vein after venoplasty.

The underlying heart disease of this case, before Maze operation and mitral valve replacement, was severe mitral regurgitation (non-ischemic) with progressive LV remodeling, but post thoracotomy, medically refractory tachyarrhythm-
mia sustained incessantly, and rate control was insufficient. Consequently, we suppose that tachycardia-induced cardiomyopathy (8) was partially associated with the development of ventricular dysfunction.

In conclusion, combination therapy of AV nodal ablation and biventricular pacing may be one of the treatment choices for heart failure patients with drug refractory tachyarrhythmias.

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