Clinical Case Reports

CASE REPORT

Successful recovery of tachycardia-induced cardiomyopathy with severely depressed left ventricular systolic function by catheter ablation with mechanical hemodynamic support: a case report

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Key Clinical Message

We describe the case that persistent atrial fibrillation refractory to rhythm control by pharmacotherapy and electrical cardioversions caused tachycardia-induced cardiomyopathy with low ejection fraction and hemodynamic instability. Mechanical hemodynamic support using an intra-aortic balloon pump is one of the choices of hemodynamic support during catheter ablation by pulmonary vein isolation.

Keywords

Cardiogenic shock, clinical: catheter ablation – atrial fibrillation, intra-aortic balloon pump, mechanical hemodynamic support, tachycardia-induced cardiomyopathy.

Introduction

Tachycardia-induced cardiomyopathy (TIC) due to atrial fibrillation (AF) occurs particularly often [1–3]. Although catheter ablation is effective therapy for congestive heart failure (CHF) with AF [4, 5], it might be avoided in patients with a severely depressed left ventricular (LV) systolic function because of the possibility of coexisting primary idiopathic dilated cardiomyopathy with AF [1, 6]. We herein report a case of TIC with low-output CHF caused by persistent AF with a rapid ventricular response refractory to antiarrhythmic drugs and electrical cardioversions. Although the patient showed vital signs consistent with shock during the procedure, he was successfully rhythm-controlled with catheter ablation by pulmonary vein isolation (PVI) under mechanical hemodynamic support using an intra-aortic balloon pump (IABP).

Case Report

A 42-year-old man was referred to our institution because of a 1-month history of progressive general malaise and dyspnea. He had no history of heart disease. On admission, his pulse rate was 188 beats per minute and irregular and blood pressure was 109/85 mmHg. He was assessed as New York Heart Association (NYHA) functional class III. His brain natriuretic peptide concentration was elevated at 929.2 pg/mL. Blood tests for antibodies to viruses were negative. A 12-lead electrocardiogram showed AF with a rapid ventricular response. On transthoracic echocardiography, the left atrium (LA) diameter was 48 mm and the LV end-diastolic diameter and end-systolic diameter was 63 and 56 mm, respectively. LV wall motion exhibited globally severe hypokinesis with an LV ejection fraction (LVEF) of 15%, calculated using Simpson's method (Fig. 1A). Neither significant valvular dis-
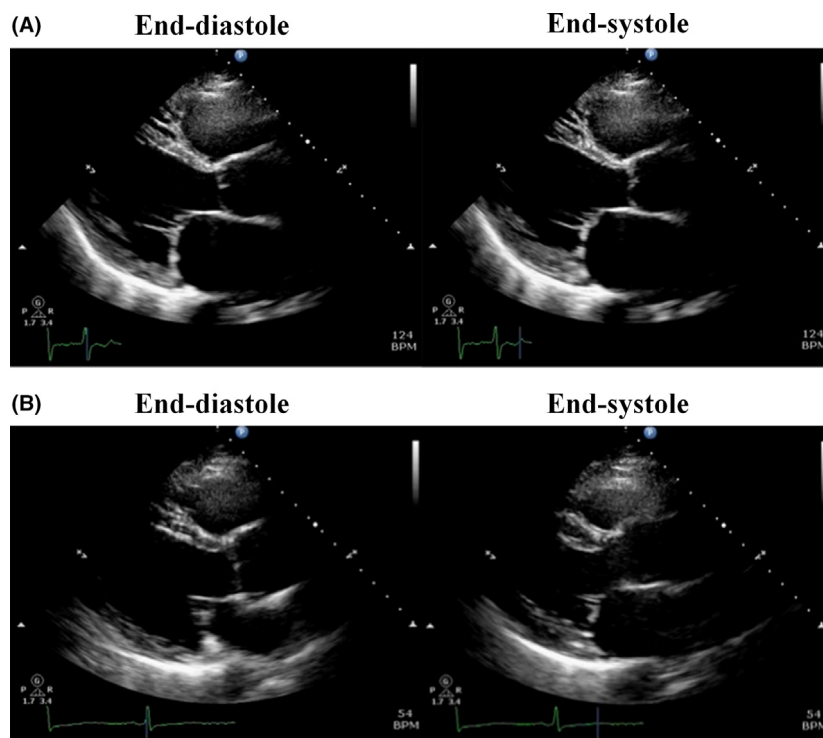
Figure 1. (A) Transthoracic echocardiography on admission showed a dilated left atrium and left ventricle. The left ventricular wall motion was severely and diffusely hypokinetic. The left ventricular ejection fraction calculated by the biplane Simpson's method was 15%. (B) Transthoracic echocardiography after 6 months depicted systolic function improvement in both the left atrium and left ventricle. The left ventricular ejection fraction was 50%.

ease including mitral regurgitation nor pericardial effusion was present. We started intravenous heparin followed by oral warfarin. Despite pharmacotherapy with carperitide, furosemide, spironolactone, and landiolol, and several biphasic electrical cardioversions, the AF with a rapid ventricular response sustained, and his symptoms did not improve. On mild effort, the patient showed vital signs consistent with shock that did not respond to additional medications including catecholamines (dopamine and dobutamine) and amiodarone. Because the patient's condition had become drug-refractory, we decided to try catheter ablation by PVI. However, we needed to wait 3 weeks while the patient underwent warfarin anticoagulant therapy to keep his prothrombin time–international normalized ratio at >2.0 because a left atrial appendage (LAA) thrombus was found during preoperative cardiac computed tomography and transesophageal echocardiography (TEE) under catecholamine support. On day 32, we confirmed the absence of an LAA thrombus with TEE. On day 37, right heart catheterization, coronary angiography, and catheter ablation were performed. The right heart catheterization revealed that his hemodynamics corresponded to Forrester IV (cardiac index (CI), 1.6 L/min per m^2; pulmonary artery capillary wedge pressure

(PCWP), 19 mmHg), and coronary angiography showed no significant stenosis. After introduction of noninvasive positive pressure ventilation (AutoSet CS; Teijin, Tokyo, Japan), the patient was sedated with a 0.6 μg/kg per h dexmedetomidine hydrochloride infusion and 0.1 g thiamylal sodium bolus injections when necessary. A 20-pole electrode catheter with an atrial cardioversion system (BeeAT; Japan Lifeline, Tokyo, Japan) was inserted from the right internal jugular vein and positioned along the lateral right atrium and in the coronary sinus for pacing and cardioversion. Using a 3D mapping system (CARTO 3; Biosense Webster, Diamond Bar, CA) and a 20-pole electrode circular mapping catheter (LASSO; Biosense Webster), the left and right ipsilateral pulmonary veins were circumferentially isolated at the antrum during AF with the 3.5-mm tip irrigation catheter (Thermocool Smarttouch; Biosense Webster) at a power of 25 to 30 W. The AF terminated and sinus rhythm (SR) recovered by BeeAT intracardiac cardioversion at 20 J. During the procedure, the patient's systolic blood pressure (SBP) decreased from 68 to 50 mmHg. He needed 10 μg/kg per min dopamine infusion, 10 μg/kg per min dobutamine infusion, and several 0.05 mg of bolus injections of noradrenaline to maintain his blood pressure, but these

effects were temporal. So we decided to start mechanical hemodynamic support using an IABP (CARDIOSAVE; Maquet Japan, Tokyo, Japan). After starting an IABP, SBP gradually increased to 100 mmHg and we did not need noradrenaline injections any more. He was transferred to the coronary care unit and managed with catecholamines and IABP support, which were weaned in 3 days because SBP maintained to 100 mmHg, urinary output was sufficient, and right heart catheter revealed that his hemodynamics improved to Forrester I (CI, 2.9 L/min per m²; PCWP, 11 mmHg). His general status gradually improved, and SR was subsequently maintained, and his cardiac function gradually improved. After 6 months, SR was maintained with no antiarrhythmic drugs except carvedilol. The LVEF improved to 50% (Fig. 1B) and brain natriuretic peptide decreased to 6.6 pg/mL. He was assessed as NYHA functional class I. His LA size dramatically decreased on cardiac computed tomography after 12 months (Fig. 2, from 206 mL to 97 mL). We diagnosed the patient with TIC.

Discussion

We have herein presented a case involving a patient with TIC with severely reduced LV systolic function. LVEF

Before catheter ablation

12 months after catheter ablation

Figure 2. Cardiac computed tomography before (top) and 12 months after (bottom) catheter ablation. The left atrial size dramatically decreased after treatment.

normalization was achieved after catheter ablation in 6 months, although temporal mechanical hemodynamic support using an IABP was required. To the best of our knowledge, this is the first report of PVI with IABP support for hemodynamically unstable TIC.

Because catecholamines increase myocardial oxygen consumption and vasoconstrictors may impair microcirculation as well as tissue perfusion, and may cause arrhythmia, their use should be restricted to the shortest possible duration and the lowest possible dose [7]. Percutaneous left ventricular assist device such as Impella (Aibomed, MA) has not been available in Japan. IABP, which increases coronary perfusion and decreases myocardial oxygen consumption, has been the most widely used mechanical hemodynamic support device for nearly 50 years. Although recent European Society of Cardiology revascularization guidelines downgraded of the routine use of IABP in patients with cardiogenic shock with acute myocardial infarction as a new class IIIA recommendation, early IABP use at the onset of cardiogenic shock still shows a potential benefit in the prevention of multiorgan system dysfunction [7]. Although not all patients with AF with a rapid ventricular response with low LVEF need mechanical hemodynamic support, we believe IABP is one of the choices to stabilize hemodynamics during PVI especially in patients with hemodynamically unstable AF. In the present case, right heart catheterization showed his hemodynamics corresponded to Forrester IV and catecholamines such as dopamine, dobutamine and noradrenaline were ineffective to maintain blood pressure, but IABP support successfully stabilized his hemodynamics corresponded to Forrester I and it could be withdrawn in 3 days.

AF with a rapid ventricular response causes inadequate diastolic filling due to atrioventricular dyssynchrony, depletion of myocardial energy resources, and systemic vascular resistance elevation, all of which finally lead to CHF [1, 2]. However, in patients with TIC, the LVEF and symptoms improve or normalize when the tachycardia is controlled appropriately. LV function improvement is reportedly seen within days to 3–6 months, although some patients exhibit late improvement (after 6 months) [8]. In particular, patients with severe LV dysfunction (LVEF of <20%) may have a slower rate of improvement [9], which is consistent with the present case.

We usually try to treat AF with a rapid ventricular response using either rate control or rhythm control. In the AFFIRM study, treatment of patients with AF using rhythm control offered no survival advantage over rate control [10]. However, the substudy of the AFFIRM study showed that restoration and maintenance of SR were associated with a lower risk of death and that the use of antiarrhythmic drugs was associated with increased mortality [11]. Additionally, rhythm control by PVI is

superior to rate control in terms of sinus node function, LA pressure, and LVEF recovery [5]. Therefore, if rhythm control by PVI can be accomplished curatively, it will lead to a better prognosis.

In many patients, CHF coexists with AF. Whether AF is the cause or result of CHF is sometimes unclear in these patients. Theoretically, TIC can be diagnosed for the first time after observing improvement in ventricular function following rate or rhythm control. Therefore, in drug-refractory cases, some clinicians may select atrioventricular junction ablation (AVJA) combined with biventricular (BiV) pacing (AVJA/BiV), not PVI. Although AVJA/BiV is an effective treatment for patients with non-ischemic dilated cardiomyopathy with AF [12], we must choose this therapy carefully because BiV implantation is invasive and may cause considerable physical and mental stress. In a multicenter randomized controlled trial comparing PVI with AVJA/BiV in patients with concurrent CHF and symptomatic AF, PVI was found to be superior to AVJA/BiV in improvement of the LVEF and 6-minute walk distance after 3 and 6 months. In this study, PVI showed better quality-of-life variables after 6 months [13]. Especially, patients with CHF caused by AF with a rapid ventricular response refractory to antiarrhythmic drugs or electrical cardioversions receive benefits by PVI like the present case. Importantly, LV dysfunction may reverse if AF is treated adequately, although temporal mechanical hemodynamic support may be needed, as in the present case.

In conclusion, we have herein presented a case of severe LV dysfunction due to TIC by AF, which was successfully treated with rhythm control by catheter ablation under IABP support. This case emphasizes the importance of rhythm control by PVI prior to consideration of AVJA/BiV, even when need of temporal mechanical hemodynamic support is required.

Conflict of Interest

None.

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