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【 CASE REPORT 】

Successful Catheter Ablation as a Substitute for Cardiac Resynchronization Therapy in Patient with an Accessory Pathway-induced Cardiomyopathy

Keisuke Nakabayashi, Ryo Sugiura, Yusuke Mizuno, Hiroko Kato, Naomi Nakazawa, Toshiaki Suzuki, Hideki Saito, Naomi Kawakatsu, Masayuki Goto, Daichi Isomura, Hisayuki Okada and Toshiaki Oka

Abstract:

A 50-year-old man presented with exertional dyspnea and orthopnea. An electrocardiogram showed a delta wave and a wide QRS complex, similar to left bundle branch block. Cardiac echocardiography revealed diffuse severe hypokinesis and dyssynchrony. The patient was diagnosed with congestive heart failure. We considered that the patient's condition was caused by an accessory pathway-induced cardiomyopathy after heart failure compensation with guideline-oriented medical therapy. We therefore performed catheter ablation for right-sided pre-excitation syndrome as cardiac resynchronization therapy. The left ventricular dyssynchrony was resolved immediately after the procedure, and the patient's ventricular contraction improved, with a reduced cardiac volume at 6 months after the procedure-thus suggesting that the accessory pathway had affected the patient's cardiac function.

Key words: right-sided pre-excitation syndrome, accessory pathway-induced cardiomyopathy, radiofrequency catheter ablation

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Introduction

Cardiac dysfunctions and heart failures have definite etiologies. Although identifying the definite etiology is essential to providing appropriate treatment, it is often difficult. The prevalence of accessory pathway is 0.1-0.3%. Most cases are related to Wolff-Parkinson-White (WPW) syndrome (1). Right-sided pre-excitation syndrome (type B WPW syndrome), even without supraventricular arrhythmias, can cause a left ventricular dysfunction due to left ventricular dyssynchrony (2). Currently, radiofrequency catheter ablation (RFCA) is considered a curative treatment for this condition. Physicians should consider this etiology and treatment particularly for patients who have left ventricular dysfunction without a typical etiology but who have a right-sided accessory pathway. We herein present a case in which RFCA was successfully used to improve the cardiac function in a patient with type B WPW syndrome.

Case Report

A 50-year-old man who had been diagnosed with type B WPW syndrome 30 years previously, without signs of tachycardia, presented at our department with exertional dyspnea and orthopnea. He was a smoker and had untreated hypertension and dyslipidemia, but no other specific family medical history was noted. A physical examination revealed the following findings: blood pressure, 140/98 mmHg; heart rate, 111/min; respiratory rate, 20/min; oxygen saturation, 92% on 4 L of O_2; and body temperature, 36.8℃. He had jugular vein distension, a coarse crackle in the bilateral lung fields, third heart sound, and bilateral pedal edema. An electrocardiogram revealed sinus tachycardia, a delta wave, a

Department of Cardiology, Seirei Hamamatsu General Hospital, Japan
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Correspondence to Dr. Keisuke Nakabayashi, keisuke2018@gmail.com

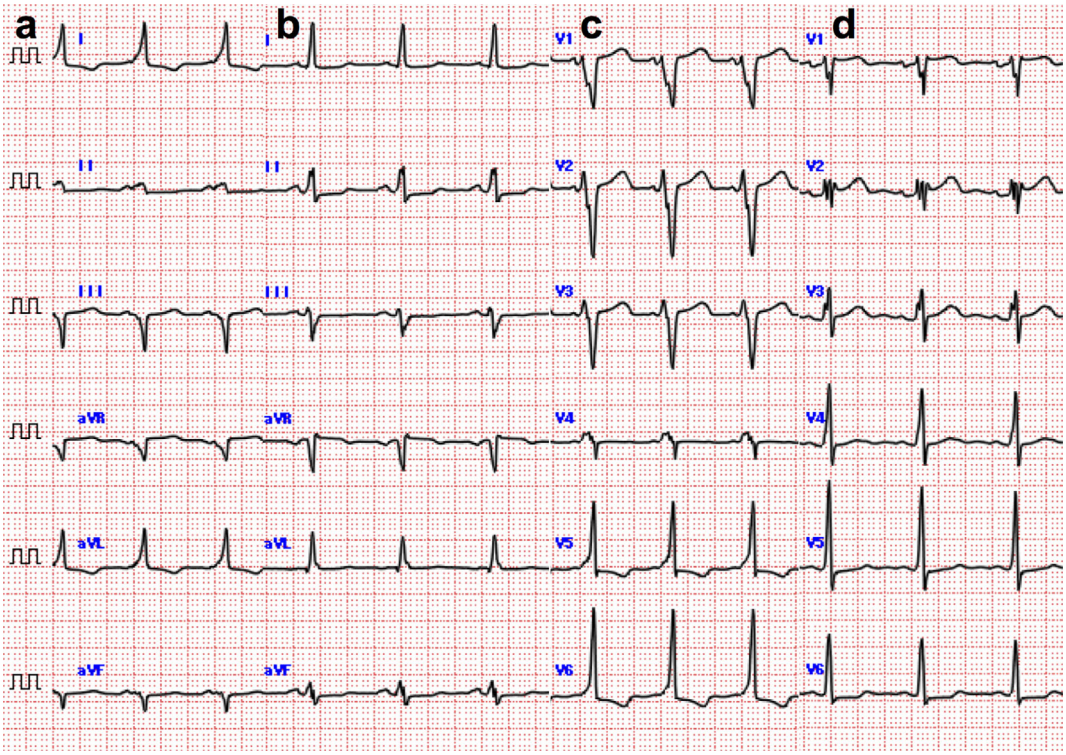

Figure 1. **Electrocardiograms before and after catheter ablation. (a, c) An electrocardiogram before catheter ablation showed sinus rhythm, a heart rate of 100 bpm, delta wave, a short PR duration (90 ms), and a wide QRS complex (QRS, 144ms), similar to that observed in left bundle branch block. (b, d) An electrocardiogram after catheter ablation showed sinus rhythm, a heart rate of 86 bpm, a normal PR duration (148 ms), and notch in the precordial leads (QRS, 110ms).**

biphasic P wave, a wide QRS complex (144 ms) similar to left bundle branch block, and ST depression in the lateral leads (Fig. 1a and c). A chest X-ray showed pulmonary edema and cardiac silhouette enlargement. Transthoracic echocardiography revealed severe left ventricular dysfunction (ejection fraction: 17.6%) and left ventricle enlargement with left ventricular end-diastolic and end-systolic diameters of 73 mm and 67 mm, respectively (Fig. 2). The laboratory results, including the patient's electrolyte levels and renal and hepatic functions, were close to the normal ranges; however, his brain natriuretic peptide level was 354.5 pg/mL.

The patient was initially diagnosed with congestive heart failure due to a non-specific etiology. The initial treatment with diuretics, nitrates, and human atrial natriuretic polypeptide was successful. An angiotensin II receptor blocker [valsartan (40 mg)] and a beta-blocker [carvedilol (1.25 mg)] were administered; however, full titration could not be achieved due to relative hypotension. Coronary angiography after heart failure compensation ruled out ischemic heart disease, and the myocardial biopsy finding was non-specific. The laboratory data did not indicate sarcoidosis or amyloidosis. Echocardiography clearly showed left ventricular dyssynchrony due to right-sided pre-excitation. We considered that the accessory pathway induced a contraction disturbance of the left ventricle, leading to left ventricular dysfunction. After obtaining informed consent, we performed RFCA of the accessory pathway on the 10th day after admission, expecting that it could be used as a substitute for cardiac resynchronization therapy (CRT). An electrophysiological study revealed a normal sinus function, normal atrioventricular conduction, no ventriculoatrial conduction, a Kent blocking rate of 150/min on coronary sinus pacing, and a Kent effective refractory period of 480 ms. Considering that no atrial fibrillation was detected during hospitalization, these findings suggested that a rapid ventricular response was less likely to have caused the ventricular dysfunction and that the risk of supraventricular arrhythmia was low. Mapping confirmed the presence of the accessory pathway at the posterolateral tricuspid valve (Fig. 3 upper panel). RFCA at this point removed the accessory pathway (Fig. 3 lower panel). Thereafter, the Wenckebach rate at the atrioventricular node was 120/min. He was discharged a day after the procedure, without any complications. An electrocardiogram on the same day showed the disappearance of the delta wave and a reduction of the QRS duration (106 ms; Fig. 1b, d); echocardiography revealed the resolution of left ventricular dyssynchrony.

His cardiac function dramatically improved (Fig. 2), and his brain natriuretic peptide level decreased to 7.8 pg/mL at 6 months after RFCA, despite the administration of the minimum guideline-oriented medical therapy at discharge [losartan (25 mg) and carvedilol (1.25 mg)]. We concluded that the patient's right-sided accessory pathway could have

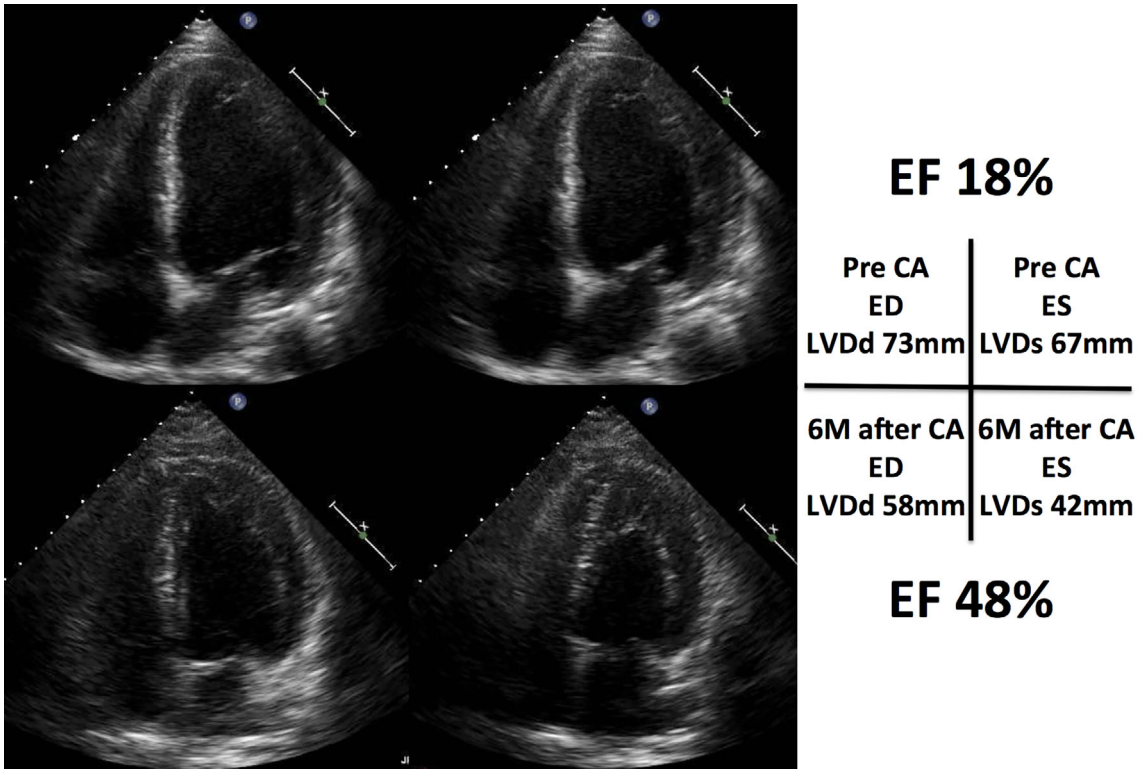

Figure 2. **Transthoracic echocardiography before and 6 months after catheter ablation. The patient's left ventricular dyssynchrony resolved immediately after catheter ablation and his left ventricular function showed dramatic improvement at 6 months after catheter ablation. EF: ejection fraction, CA: catheter ablation, ED: end-diastolic phase, ES: end-systolic phase, LVDd: left ventricular diameter on diastole, LVDs: left ventricular diameter on systole, 6M: 6 months**

affected his cardiac dysfunction. The patient agreed to the publication of this case report.

Discussion

In the present case, RFCA was used as a substitute for CRT in the treatment of a patient with type B WPW syndrome who had intolerance for the titration of guideline-oriented medical therapy. Patients with left branch bundle block often have other cardiac disorders, such as mechanical dyssynchrony, mitral valve apparatus deformation, and left ventricular remodeling, which lead to a poor prognosis (3). CRT has been established as an effective heart failure therapy, especially for patients who are resistant to medical therapy (4).

Patients with type B WPW syndrome have an accessory pathway on the right side of the heart and therefore have a wide QRS complex similar to that in the left branch bundle block pattern and left ventricular dyssynchrony. RFCA was reported to improve the cardiac function of patients with a right-sided accessory pathway. Tomaske et al. retrospectively confirmed the improving cardiac function of 34 symptomatic patients with type B WPW syndrome after RFCA (5). Iwasaku et al. performed RFCA in type B WPW syndrome patients with dilated cardiomyopathy, regardless of whether full doses of angiotensin-converting-enzyme inhibitors and beta-blockers were administered, and confirmed resynchronization, as well as improved cardiac function, mitral regurgitation, and brain natriuretic peptide levels (6).

These patients were evaluated to determine whether they had secondary cardiomyopathy due to type B WPW syndrome or cardiomyopathy of another etiology with type B WPW syndrome as a comorbidity. Udink et al. retrospectively investigated 10 consecutive children (median age, 8 years) with dilated cardiomyopathy and ventricular preexcitation syndrome (7). All cases had a right-sidedd accessory pathway: 2 disappeared and 8 were found to be residual by an electrophysiological study. All 8 cases underwent RFCA and showed an improved cardiac function. The study suggested that there was a relationship between accessory pathways and dilated cardiomyopathy and that early intervention was highly effective.

The patient in the current case had incipient heart failure; thus, idiopathic dilated cardiomyopathy could not be ruled out. Besides the evaluation of typical etiologies on admission, further or repeated evaluations should be performed in the chronic phase. The electrocardiogram obtained after catheter ablation showed residual intraventricular conduction disturbance, and the cardiac function remained at the lower limit of the normal ejection fraction at the 1.5 year follow-up examination. These findings suggested a coexisting cardiac disorder, such as dilated cardiomyopathy. However, we

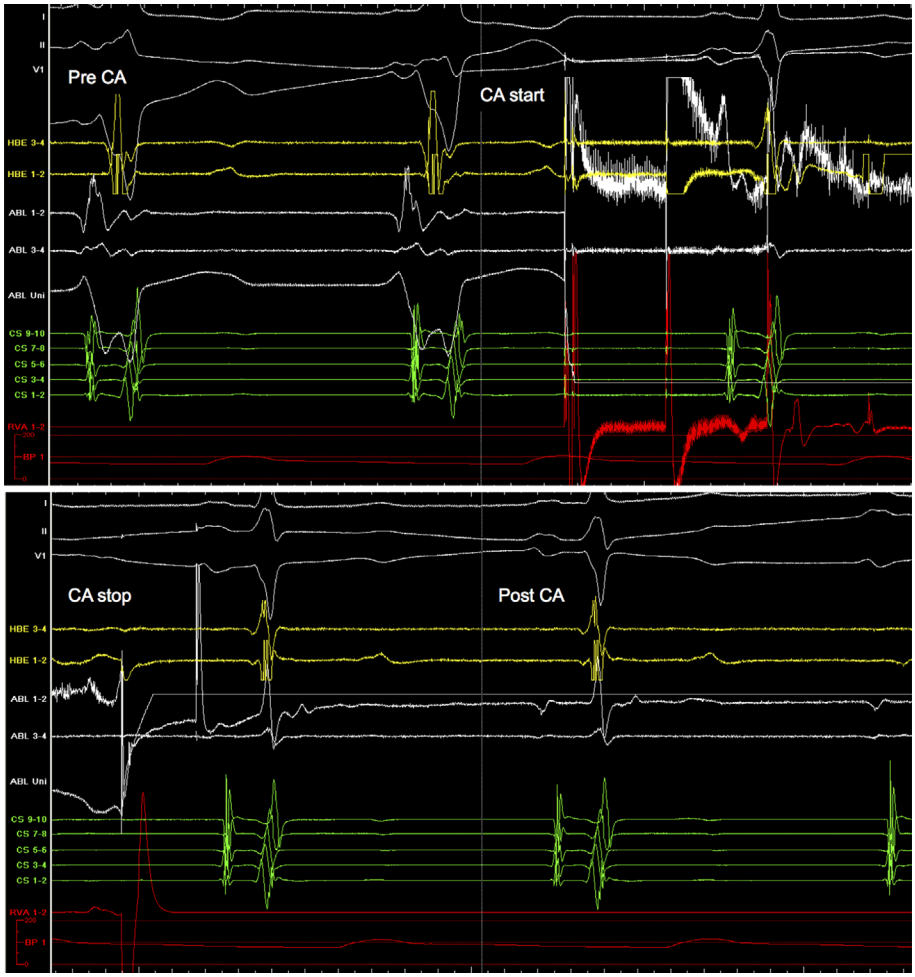

Figure 3. The electrophysiological study and catheter ablation. The upper panel shows the potential on the right-sided accessory pathway before catheter ablation. The lower panel shows an extracardiac and intra-cardiac electrocardiogram after catheter ablation. CA: catheter ablation

believe that the accessory pathway might have affected the cardiac dysfunction in the present case, as we could not use a beta-blocker or an angiotensin II receptor blocker because of hypotension, and the improvement of the cardiac function was observed after a relatively short period. It is important to obtain information on an accessory pathway-induced cardiomyopathy as an etiology for cardiac dysfunction and heart failure, without missing secondary cardiomyopathies that require specific treatments.

Our case was missing specific left ventricular dyssynchrony parameters, such as septal-to-posterior wall motion delay (8), interventricular mechanical delay (9), septal-to-lateral delay (10), and intraventricular conduction delay, which are evaluated by tissue Doppler echocardiography (11) before and after RFCA, because of testing time limitation. Our medical staff aggressively evaluated these left ventricular dyssynchrony parameters after this case.

In general, the indication for RFCA in patients with type B WPW syndrome is tachyarrhythmia. However, we advocate the use of RFCA as an additional treatment after (or concomitantly with) the guideline-oriented medical therapy for accessory pathway-induced cardiomyopathy due to type B WPW syndrome because of its potential to reduce the risk of sudden cardiac death and recurrence of decompensation.

The authors state that they have no Conflict of Interest (COI).

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