A Case of Heart Failure Caused by Frequent Premature Ventricular Contractions

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A 43-year-old woman was found to have premature ventricular contractions (PVCs) during a health check conducted 6 months ago. She was admitted to our hospital 1 month ago because of heart failure. Frequent PVCs occurring in a bigeminal or trigeminal rhythm originated from the right ventricular outflow tract (RVOT), and the total heart rate was 62,830 pulses (48%). The patient was diagnosed with heart failure caused by frequent PVCs. We performed a catheter ablation under the guidance of an electroanatomic mapping system. PVCs of 2 types were detected, and PVC1 with a notch in the inferior axis was frequently observed. Activation mapping revealed that the free wall of the RVOT was the site of earliest activation. After ablation, PVC1 and PVC2 disappeared but PVC3 and later PVC4 appeared. The notch shape of the PVCs changed. Repeat activation mapping of PVC4 showed that the earliest excited site was now the posterior wall of the right ventricle (the initial deflection preceded the QRS complex during the PVCs by 40 ms). Ablation of the posterior wall completely inhibited the PVCs. On day 3 after the ablation, the brain natriuretic peptide levels decreased from 1,294 to 86 pg/dL, indicating significant improvement in heart failure. An echocardiogram taken 3 months after discharge revealed that the ejection fraction had improved by 50–71%.

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Key words: Premature ventricular contraction, Heart failure, Tachycardia-induced cardiomyopathy, Preferential conduction

Introduction

A patient without structural heart disease may at times experience heart failure resulting from cardiomyopathy induced by supraventricular tachycardias such as atrial fibrillation. However, few studies have reported frequent premature ventricular contractions (PVCs) resulting in tachycardia-induced cardiomyopathy and heart failure. We report the case of a patient with frequent PVCs that caused tachycardia which in turn induced cardiomyopathy and eventually heart failure. In this patient, cardiac function was improved after catheter ablation.

Case report

A 43-year-old woman was found to have PVCs during a health check conducted 6 months ago. She experienced subjective symptoms such as shortness of breath, palpitation, and general malaise in mid-
June 2008, and examination in a nearby clinic revealed frequent PVCs. She was then referred to our hospital. An echocardiogram showed a general decline in wall movement and an ejection fraction (EF) of 50%. A chest radiograph showed bilateral pleural effusion, and she was immediately admitted to the cardiology department for treatment.

Frequent PVCs occurring in a bigeminal or trigeminal rhythm originated from the right ventricular outflow tract (RVOT), and the total heart rate was 62,830 pulses (48%). Treatments such as human atrial natriuretic polypeptide (hANP) and diuretic administration did not improve the heart failure sufficiently. On day 4 after admission, coronary angiography was performed; no significant coronary stenosis was detected. Right ventricular enlargement was not observed on the angiogram ruling out arrhythmogenic right ventricular dysplasia. Thereafter, catheter ablation was performed under the guidance of an electroanatomic mapping system. A vigesimal catheter and ablation catheter with a 4-mm tip (Navistar, Biosense Webster) were inserted into the RVOT from the right femoral vein. PVCs of 2 types were detected, of which PVC1 with a notch in the inferior axis was frequent (Figure 1A). Activation mapping revealed that the free wall of the RVOT was the earliest site of activation (Figure 1B). However, initial deflection preceded the beginning of the QRS complex during PVC1 by 25 ms, and an almost identical origin in a relatively wide range and a good pace map were obtained. After ablation, PVC1 and PVC2 disappeared but PVC3 and later PVC4 appeared, with a change in the notch shape of the PVCs (Figure 2). Repeat activation mapping of PVC4 (Figure 3) showed that the earliest excited site was now the posterior ventricular wall, located 2 cm away from the previous site (the initial deflection preceded the beginning of the QRS complex during PVC4 by 40 ms). After ablation of the posterior wall, the PVC disappeared (Figure 4). Therefore, the electroanatomic mapping system was found to be effective for assessing changes in the breakout site of PVC. Brain natriuretic peptide (BNP) levels decreased from 1,294 to 86 pg/dL 3 days after the ablation. Chest radiography revealed a 64–50% decrease in CTR and improvement of pleural effusion and lung congestion. Heart failure was significantly improved, and the patient was discharged from the hospital on the next day. Echocardiography performed 3 months after discharge revealed a 50–71% improvement in EF, unchanged left ventricular (LV) end-diastolic dimensions.

![Figure 1A](image_url)  
**Figure 1A**  
Electrocardiogram taken at the time of admission.  
Premature ventricular contractions (PVCs) of 2 types are seen, of which PVC1 with a notch in the inferior axis is frequent.
(LVDd; 55 mm), and decreased LV end-systolic dimension (LVDs; 41 mm–33 mm).

**Discussion**

We report the case of a patient with heart failure possibly due to frequent PVCs.

Supraventricular tachycardia can induce cardiomyopathy, and PVC is a type of arrhythmia often encountered in clinical practice.

Some authors have reported that highly frequent PVCs were the root cause of tachycardia-induced cardiomyopathy.1–7) Chugh et al.1) first reported in 2000 that PVCs impair LV function and are inhibited by catheter ablation. Kanei et al.2) found a significant correlation between the frequency of PVCs and decrease in cardiac function. Bogun et al.3) reported a possible link between frequent PVCs and LV dysfunction. Catheter ablation was found to normalize LV function in 18 (82%) of 22 patients with an abnormal EF (baseline EF, 34%; post-ablation EF, 59% ± 7%) within 6 months. The mechanism underlying the induction of LV dysfunction by frequent PVCs is unclear. In patients with PVCs of the RVOT, myocardial activation was asynchronous, resulting in a decline in the left ventricular function.
mitral regurgitation, and finally a decrease in the stroke volume. Recent clinical trials show that right ventricular apex pacing causes left bundle-branch block and cardiac dysfunction.

In our patient also, PVCs accounted for 48% of the total heart rate, and cardiac function was impaired for the above-mentioned reasons. Catheter ablation completely inhibited PVCs and recovered cardiac function. To the best of our knowledge, no other case has yet been reported in which heart failure, lung congestion, and pleural effusion, and significantly high BNP levels were remarkably

Figure 3
Electroanatomic map and radiogram of the site of catheter ablation for PVC4 showing the posterior wall of the right ventricle as the new site of earliest excitation (the initial deflection preceded the QRS complex during the PVCs by 40 ms).

Figure 4
Electrocardiogram after catheter ablation of the posterior wall of the right ventricle showing no PVCs.
improved within a few days of catheter ablation. Therefore, we consider our case to be clinically useful.

In our patient, the morphology of PVC changed after ablation. Tada et al.\(^5\) stated that the morphology of PVCs is altered after catheter ablation therapy for PVC, and additional ablation at another site may be required. Yamada et al.\(^9\) used an electroanatomic mapping system in patients with PVCs of the RVOT and demonstrated preferential conduction. In the case of our patient, catheter ablation resulted in a gradual displacement of the breakout site and also blocked PVCs of the posterior wall of the right ventricle. The use of an electroanatomic mapping system can prove very helpful, particularly in cases in which the morphology of PVCs changes after catheter ablation.

Recently, ablation therapy for PVCs has been reported to be indicated in patients without structural heart disease. Takemoto et al.\(^4\) have reported that catheter ablation is beneficial in patients in whom PVCs exceed 20% of the total heart rate, impair cardiac function, and induce heart failure. Baman et al.\(^10\) concluded that PVCs exceeding 24% of the total heart rate are independently associated with PVC-induced cardiomyopathy, and induce heart failure. The findings of the above studies show that PVCs exceeding 20% of the heart rate are an indication for catheter ablation in patients without structural heart disease. In our patient, the PVCs accounted for 48% of the total heart rate, and catheter ablation was clinically beneficial.

PVC is a common type of arrhythmia, and if PVCs occur frequently, they may impair cardiac function. We found that catheter ablation can cure even patients with PVC-induced cardiac failure.

References