A Case of Cardiomyopathy Induced by Premature Ventricular Complexes

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Tachycardia-induced cardiomyopathy is a well-known and reversible condition, but the left ventricular dysfunction caused by frequent isolated premature ventricular complexes (PVCs) has been rarely reported. Apparent dilated cardiomyopathy was resolved in a patient after the focal source of PVCs was eliminated by radiofrequency catheter ablation. Echocardiography showed progressive improvement of the abnormal wall motion. Frequent PVCs could be the cause of left ventricular dysfunction in a subset of patients with dilated cardiomyopathy and radiofrequency ablation should be the choice of therapy in those patients. (Circ J 2002; 66: 1065–1067)

Key Words: Cardiomyopathy; Catheter ablation; Premature ventricular complex

Case Report

A 53-year-old man presented with palpitations and exertional fatigue, but without clinical or laboratory evidence of ischemic heart disease. He was diagnosed by echocardiography as having DCM with fractional shortening (FS) of the left ventricle of 25%. All 4 chambers were dilated: LV diastolic diameter 65 mm, LV systolic diameter 49 mm, and left atrial diameter 46 mm. The chest X-ray showed mild cardiomegaly (cardio thoracic ratio) (CTR) 54%, and the serum brain natriuretic peptide (BNP) level was mildly elevated to 54 pg/ml. The electrocardiogram (ECG) did not record any abnormal findings other than frequent PVCs. Repeated Holter recordings revealed about 50,000 isolated or couplet PVCs in a 24-h period, the majority with a uniform left bundle branch block (LBBB) pattern and inferior-axis morphology. PVCs bigeminy appeared constantly throughout the day. During an exercise test, the number of PVCs decreased with the loading of exercise and increased again after the test. Tl-myocardial scintigraphy showed a mildly decreased uptake in the apicoanteroseptal wall. Cardiac magnetic resonance imaging did not show evidence of infiltration of fatty tissue.

It was not possible to use antiarrhythmic drugs for this patient with a low ejection fraction (EF) and the cause of his symptoms was considered to be the frequent PVCs. He was referred to the electrophysiologic laboratory for RF-CA of the PVC focus without medication, including β-antagonists. During the electrophysiologic study, the number of PVCs did not change after infusion of isopro-
terenol, and neither nonsustained nor sustained ventricular tachycardia could be induced with ventricular single and double extrastimuli or burst stimuli, even in the presence of isoproterenol. However, there were frequent PVCs with a morphology similar to those on the Holter recordings (ie, LBBB pattern and inferior axis; Fig 1). Endocardial mapping was performed with a 64-pole basket catheter introduced from the right femoral vein, which was positioned at the right ventricular outflow tract (RVOT). A standard curve ablation catheter (7Fr, 4-mm tip EPT Boston Scientific Co.) was introduced from the left femoral vein for further localization of the earliest activation site.

The earliest activation site of the PVCs was identified on the anteroseptal aspect of the RVOT (Fig 2) and pace mapping at this location reproduced the morphology of the PVCs in 10 of the 12 ECG leads (Fig 3). Therefore, RF-CA was applied to this site and after the first delivery of energy up to 50 W at 50°C for 60 s, the frequent PVCs disappeared. At the end of the procedure, no PVCs of the culprit morphology were elicited even with the infusion of isoproterenol.

After RF-CA, coronary angiography, left ventriculography, and biopsy of myocardium of the right ventricular septum were performed and they showed diffuse severe hypokinesis of the left ventricle (EF: 34%) (Fig 4), no significant stenosis, and no pathological findings, respectively.

Echocardiography showed that the LV wall motion progressively improved after RF-CA (Fig 5) and the asynergic wall motion of the LV septum, which is often observed in patients with LBBB, gradually disappeared after 1 month. Left ventricular function was normalized with FS of 33% on the 210th day after RF-CA. The chamber dimensions had also decreased: LV diastolic diameter 58 mm, LV systolic diameter 39 mm, and left atrial diameter 43 mm.

During the 6-month follow-up, the patient reported the absence of palpitations and fatigue. The CTR decreased to 50% on chest X-ray and the serum BNP level decreased to 15 pg/ml. The ECG showed several PVCs originating from the other sites, but no other abnormal findings. Follow-up 24-h Holter recordings showed the number of PVCs had decreased to a total of 1,100–1,800 per day without the culprit LBBB pattern and inferior axis morphology.

**Discussion**

There have been cases of improvement of cardiomyopathy after treatment of the rapid ventricular response caused by atrial fibrillation, atrial tachycardia, atrioventricular reciprocating tachycardia, atrioventricular nodal reentrant tachycardia, and incessant ventricular tachycardia, and these cases are considered to be tachycardia-induced cardiomyopathy. However, this concept does not apply to the present case, because the Holter recordings did not show sustained arrhythmias, only frequent PVCs.

Complete LBBB (CLBBB) is an independent factor in determining the prognosis of patients with DCM because the duration of mitral regurgitation flow is prolonged and diastolic function is impaired by the shortening of LV filling time, which decreases stroke volume. In another report, cardiac function improved after catheter ablation in a patient with type B Wolff-Parkinson-White syndrome with an old inferior myocardial infarction. Thus, the asynergic wall motion caused by LBBB might cause the decreasing cardiac function observed in some cases.
Chugh et al published the first report of a case of a cardiomyopathy reversed by focal RF-CA of the source of isolated PVCs, the morphology of which was LBBB and inferior axis, as in the present case. Usually, LV contraction moves from the apical to the basal portion to give the 'squeezing' effect in sinus rhythm, but the direction is reversed in the case of PVCs that originate from the outflow tract. Therefore, frequent PVCs, especially those originating from the RVOT, can decrease cardiac function.

In the present case, the delay in the contraction of the LV septum, as is often seen in patients with LBBB, gradually normalized on echocardiography over 13 days after the ablation procedure and the time course of recovery was possibly influenced by the effect of frequent PVCs on LV wall motion. Frequent PVCs do not change the depolarization and repolarization phase in ECG, but may effect the pattern of LV wall motion.

There are many patients with frequent PVCs, but few are accompanied by LV dysfunction, so it is not clear if frequent PVCs are an independent cause of LV dysfunction. It may be at least one important factor in the worsening of LV function, if there is some myocardial damage, such as the possible myocardial damage in the anteroseptal wall observed on the TI scintigram of the present case.

**Conclusion**

In a subset of patients with apparent idiopathic cardiomyopathy and frequent isolated monomorphic PVCs, RF-CA is the choice of therapy to eliminate the arrhythmic focus. We advise ablation therapy for cases of apparent LV dysfunction when there are few abnormal findings on the ECG at rest because that reflects the presence of minimal organic myocardial damage.

**References**