

Radiofrequency Catheter Ablation in a Patient with Tachycardiomyopathy due to Incessant Fascicular Tachycardia

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ABSTRACT

Fascicular tachycardia (FT) arising from the left posterior fascicle, one of the most common forms of idiopathic ventricular tachycardia (VT), is considered benign. Herein is presented our experience of a 25-year-old man presenting with palpitation, dyspnea and dilated cardiomyopathy due to drug-refractory FT who was successfully treated with radiofrequency catheter ablation. Echocardiography showed a dilated left ventricle (LV, 71 mm at end-diastole, 67 mm at end-systole) and decreased ejection fraction (18%). The 12-lead ECG showed a wide-QRS tachycardia (cycle length: 440 msec), with a monophasic R wave in the lead V1, left axis deviation and atrioventricular dissociation. The His-bundle electrogram revealed that the His potential preceded the ventricular activity with an abnormally short HV interval (15 msec) and dissociation of the atrial and ventricular activities. Endocardial mapping during the VT demonstrated that the Purkinje potential preceded the ventricular activity and was earliest at the upper posterior septum of LV, where the tachycardia was ablated with radiofrequency (RF) energy (30 W for 30 sec). The patient remained asymptomatic over a 6 months follow-up and the LV has returned to normal (51 mm at end-diastole, 34 mm at end-systole, ejection fraction: 62%). (*Korean Circulation J* 2004;34(11):1107-1112)

KEY WORDS : Tachycardia ; Cardiomyopathy ; Catheter ablation.

Introduction

Fascicular tachycardia (FT) arising from the anterior or posterior fascicle is a common form of idiopathic ventricular tachycardia (VT) coupled with right ventricular outflow tract tachycardia. FT is usually caused by reentry within the fascicles, but mechanical stimulation of the

fascicles or drug intoxication can also be the cause. FT due to reentry within the fascicles is called intrafascicular reentry tachycardia, or verapamil-sensitive left ventricular tachycardia, because of the excellent response of this tachycardia to intravenous verapamil or diltiazem.^{1,2)} FT may respond to adenosine or beta blocker when the mechanism is triggered activity.³⁾ Radiofrequency (RF) catheter ablation is highly successful in FT. Tachycardia-induced cardiomyopathy is caused persistent supraventricular or ventricular tachycardia and characterized by ventricular dilatation and systolic dysfunction, which is reversible with normalization of the heart rate.

Herein, is reported a case of FT complicated with

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tachycardia-induced cardiomyopathy.

CASE

A 25-year-old male patient presented with intermittent palpitations and dyspnea of 7 years duration. Over the last 2 years, the symptoms appeared more frequently and pit-

ting edema had developed. He was admitted to a local hospital due to exacerbation of his dyspnea. He was diagnosed as dilated cardiomyopathy and sustained VT. The VT was not terminated with intravenous administration of lidocaine, verapamil, esmolol, and amiodarone, so he

was transferred to our hospital.

The physical examination revealed mild jugular vein

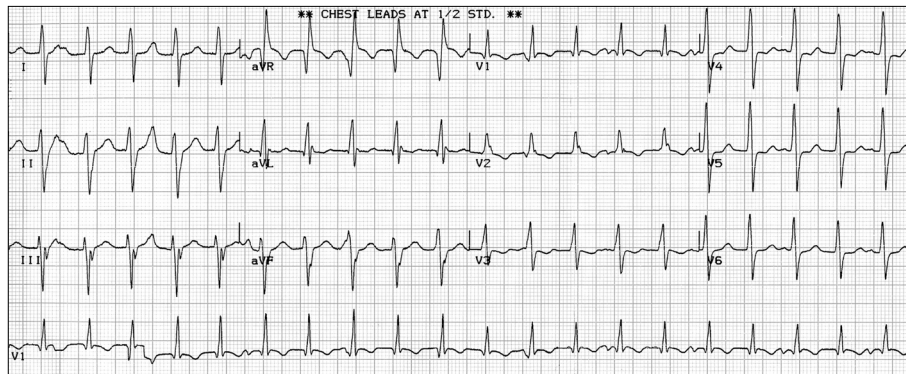


Figure 1. Surface electrocardiogram (ECG) during the fascicular tachycardia. The standard 12-lead ECG shows a wide QRS tachycardia with a QRS width of 130 msec, atrioventricular dissociation, monophasic R wave in the lead V1 and left axis deviation.

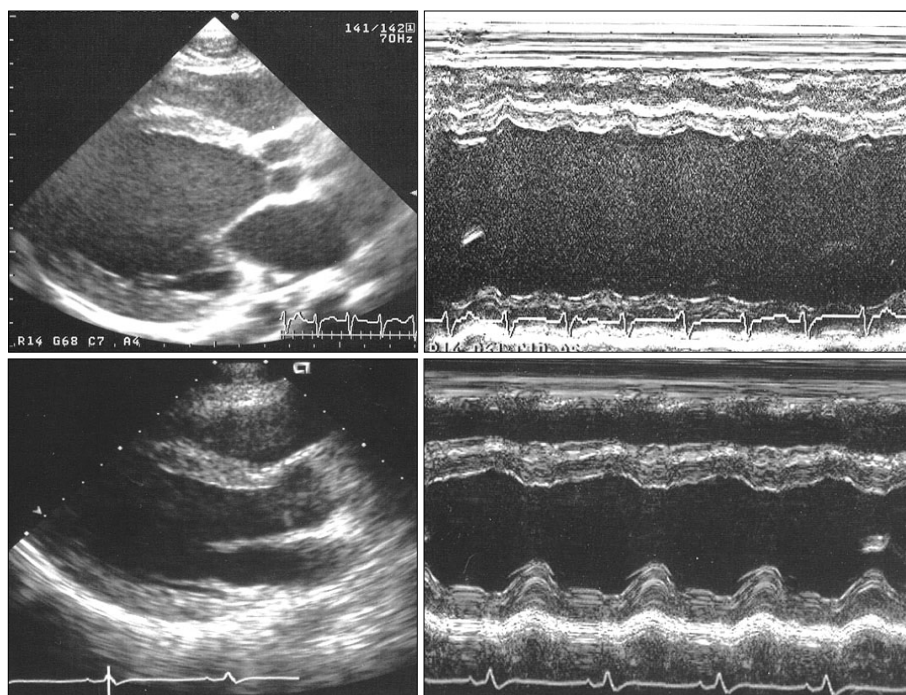
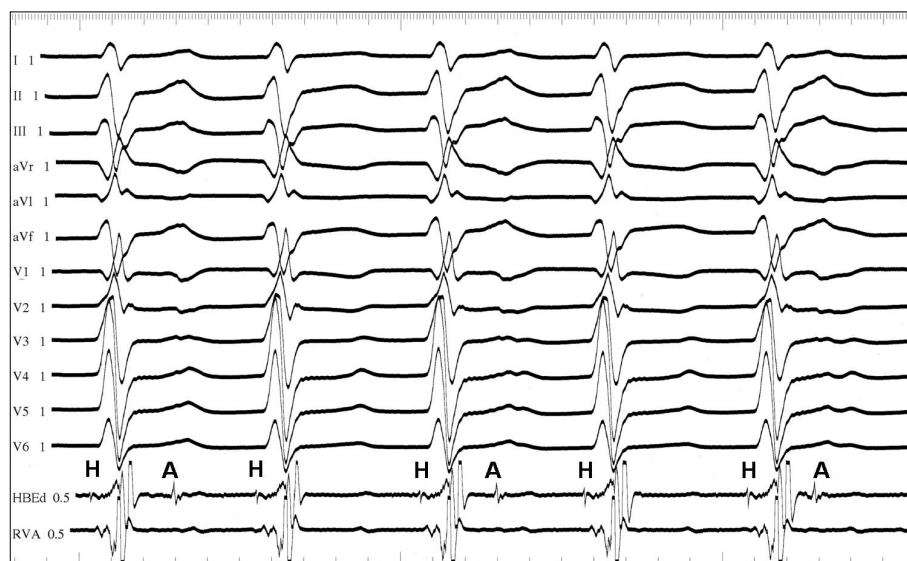


Figure 2. Two-dimensional echocardiogram on admission (upper panel) and 6 months later (lower panel). The echocardiogram demonstrates a marked dilatation of the left ventricle (LV) and a decrease in the LV contractile function. LV end-diastole dimension (LVEDD), 71 mm; LV end-systole dimension (LVESD), 67 mm; and ejection fraction (EF), 18% (upper panel). The follow-up echocardiography (lower panel) reveals a normal LV dimension and function (LVEDD: 51 mm, LVESD: 34 mm, EF: 62%).



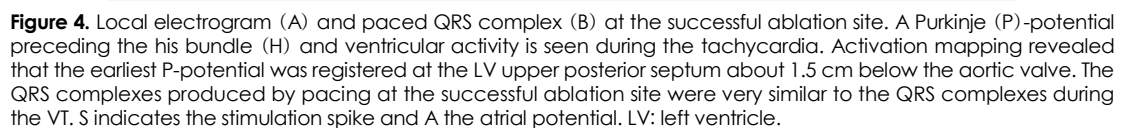
engorgement, clear breathing sounds and 2-finger breadth of hepatomegaly. The vital signs were as follows: pulse rate, 136 beats/min; blood pressure, 120/80 mmHg; and respiration rate, 20/min. His consciousness was clear. Cardiac auscultation revealed a regular but rapid heart beat, with no murmur and variable intensity of the first heart sound.

The standard 12-lead electrocardiography (ECG) showed a wide QRS tachycardia with a QRS width of 130 msec, AV dissociation, monophasic R wave in the lead V1 and left axis deviation (Figure 1). Cardiomegaly and increased broncho-vascular markings at the perihilar region were observed on the chest X-ray. Diuretics, digitalis and angiotensin converting enzyme inhibitor were given. The echocardiography demonstrated a marked dilatation of the left ventricle (LV) and a decreased LV contractile function (LV end-diastole dimension, 71 mm; LV end-systole dimension, 67 mm; ejection fraction, 18%) (Figure 2).

Electrophysiological study and catheter ablation

An electrophysiological study and radiofrequency catheter ablation were performed after informed written consent had been obtained and all antiarrhythmic drugs had been discontinued for at least 5 half-lives. Three 6-F

quadrupolar electrode catheters were positioned via a femoral vein at the high right atrium, His bundle and the right ventricular apex. A 7-F vascular introducer was inserted into the right femoral artery to monitor the arterial blood pressure. The surface ECG, intracardiac electrogram and blood pressure were stored in the cardiac electrophysiology data storing and analyzing computer (EP Lab system, Quinton Electrophysiology, Seattle, WA, USA). The tachycardia was sustained at a cycle length of 440 msec. The atrial activity was completely dissociated from the His bundle and ventricular activity. The His bundle activity always preceded the ventricular activity, but HV interval was abnormally short (15 msec) (Figure 3). These findings were compatible with VT. Single ventricular premature depolarization reset the VT and double ventricular premature depolarization terminated the VT. The VT spontaneously developed one or two sinus beats after termination of the VT. The VT did not respond to an intravenous bolus injection of adenosine (6 and 12 mg). This tachyarrhythmia was thought to be VT due to reentry involving the left posterior fascicle. A 7-F deflectable quadrupolar ablation catheter, with a 4 mm tip electrode (Livewire™, Daig Co., Minnetonka, MN, USA), was placed through the right femoral artery into



It was considered that this site was a good target for ablation. However, RF energy was applied at a site 5 mm below the presumed good target site, with a power of 30 W for 30 sec, as the site was thought too close to the main trunk of left bundle branch. The VT was terminated after 15 sec of RF delivery and could not be induced at the baseline study following termination, but non-sustained VT was induced following an infusion of iso-

proterenol at a rate of 2 μ g/min. Therefore, the target was moved to the initial mapping site, where the earliest P-potential was recorded, and then delivered RF energy for 30 sec while observing if left bundle branch block (LBBB) would develop. LBBB did not develop after the RF delivery and VT could no longer be induced. The follow-up echocardiography performed 7 days after catheter ablation showed a slight decrease in the LV dimension coupled with an increase in the EF to 35%. The patient was discharged without any antiarrhythmic drugs.

Follow-up

For the 6 months following his discharge, no symptoms developed and the ECG was normal. The LV dimension and function had returned to normal on the follow-up echocardiography (Figure 2).

DISCUSSION

The clinical presentation of VT varies from palpitation to sudden death according to the underlying disease, LV function and VT rate. It has been estimated that idiopathic VT generally accounts for 10% of all VT, but various incidences have been reported.⁴⁻⁷⁾ The incidence of idiopathic VT in Korea has been considered to be much higher than that of western countries. Lee, et al. reported that idiopathic VT accounts for 30.4% of all causes of VTs.⁷⁾

FT, one of the most common idiopathic VT, develops mostly in relatively young, healthy people without any structural heart disease. This case was accompanied by severe LV dilatation and systolic dysfunction, which was caused by the drug refractory, incessant FT. Tachycardiomyopathy was confirmed by the observation that the LV size and function returned to normal after the successful ablation of the FT. The QRS complex during FT usually shows a right bundle branch block (RBBB) and left or right axis deviation morphology.^{8,9)} FT is found to originate from the left posterior fascicle if the QRS complex has a RBBB with a left axis deviation pattern, or the left anterior fascicle if the QRS complex has a RBBB with a right axis deviation pattern. In this case, the QRS complex showed a RBBB with a left axis deviation pattern. FT often exhibits a relatively narrow QRS complex because

it originates from the fascicle, with impulses rapidly propagating through the fascicles to the ventricular myocardium.⁸⁾ The QRS width in our case was 130 msec, which was compatible with the FT finding. The electrical impulse in FT can propagate more rapidly to the His bundle than to the ventricle, and thus, the His bundle potential may appear earlier than the ventricular activity. Therefore, the His bundle potential was recorded as early as 15 msec prior to the ventricular activity in this case.

Although electrophysiologically distinct, but anatomically related to intrafascicular tachycardia, interfascicular or bundle branch reentry (BBR) tachycardia should be considered in the differential diagnosis of idiopathic left VT. In this case, an RBBB morphology, left axis deviation and an abnormally shorter HV interval during VT than sinus rhythm was thought to be differential points from the BBR or interfascicular tachycardia.^{10,11)}

FT usually responds very well to verapamil, but not to lidocaine or propranolol, which is in contrast to VT in ischemic heart disease.^{1,12-14)} It can also respond to ajmaline, sotalol or amiodarone. However, this case did not respond to any antiarrhythmic drugs, including lidocaine, verapamil, esmolol, amiodarone and adenosine. It was presumed that multiple drug resistance of the FT in this case was caused by electrophysiological remodeling of the LV following long standing tachycardia.

Tachycardia-induced cardiomyopathy, due to ventricular tachyarrhythmias, has been reported in association with right ventricular outflow tract tachycardia and idiopathic left ventricular tachycardia.^{15,16)} It can occur at any age, even in a fetus. The incidence of tachycardia-induced cardiomyopathy is unknown; most reports have been small retrospective studies from patients with atrial fibrillation.^{17,18)} The presumed risk factors include the type, rate and duration of tachyarrhythmia, and the underlying heart disease.

The RF catheter ablation technique is found to be a safe curative technique in FT. The P potential can be an aid to map the successful ablation site.¹⁹⁾ The LV septum is the successful target site for ablation when the QRS complex of the tachycardia shows an RBBB and left axis deviation pattern. Although there was a risk for developing LBBB, since the target site was located near the main trunk of the left bundle branch, it did not occur in

our case. However, since there have been reports of LBBB developing in such cases, extreme caution should be taken when ablation is attempted in the upper part of the LV septum.

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