Successful Catheter Ablation for Incessant Ventricular Tachycardia in a Patient With Hypertrophic Cardiomyopathy

Yasuo Okumura, MD; Ichiro Watanabe, MD; Kimie Ohkubo, MD; Satoshi Saito, MD

A 35-year-old man was referred to Nihon University Hospital because of repetitive ventricular tachycardia (VT) at 180–200 beats/min. QRS morphology of the VT was right bundle branch block with a northwest axis. Transthoracic echocardiography showed hypertrophic cardiomyopathy. Coronary angiography was normal and left ventriculography showed neither obstruction in the left ventricle (LV) nor any pressure gradients within the LV or between the LV and aorta. Hemodynamic deterioration occurred during VT. Intracardiac mapping showed that the VT originated from the posteroseptal portion of the LV near the apex and Purkinje potentials that preceded the onset of the QRS complex by 58–70 ms were documented. Radiofrequency ablation at these sites terminated the VT, which has not recurred for 25 months. (Circ J 2007; 71: 1164–1168)

Key Words: Ablation; Hypertrophic cardiomyopathy; Purkinje potential; Ventricular tachycardia

Monomorphic ventricular tachycardia (VT) in the presence of structural heart disease is largely attributable to anatomically bound macro-re-entry involving regions of myocardial scarring or the bundle branches. In contrast, repetitive, idiopathic right and left ventricular (LV) outflow tract VTs are attributable to triggered activity or abnormal automaticity of the ventricular myocardium. There is evidence that triggers originating from distal Purkinje arborization play an important role in the initiation of malignant arrhythmias such as polymorphic VT and ventricular fibrillation in a variety of clinical scenarios. We report a patient with hypertrophic cardiomyopathy (HCM) in whom life-threatening repetitive monomorphic VTs developed. Radiofrequency (RF) ablation targeting the Purkinje-like potentials preceding the VT prevented recurrence.

Case Report

A 35-year-old man with HCM presented with recurrent episodes of palpitation and chest discomfort. Electrocardiographic monitoring during palpitation showed an incessant nonsustained, wide QRS tachycardia with a QRS duration of 130 ms at 180–200 beats/min. On admission to hospital, 12-lead ECG showed incessant nonsustained VT with right bundle branch block (RBBB) morphology and a northwest axis deviation (Fig 1). Intravenous lidocaine and verapamil were effective in terminating the VT but ineffective in preventing recurrence. A 12-lead ECG obtained during sinus rhythm after intravenous administration of verapamil showed poor R wave progression in the precordial leads (Fig 2). The signal-averaged ECG was recorded during sinus rhythm after intravenous administration of 5 mg of verapamil. Oral administration of ß-blocker, class I antiarrhythmic drugs (pilsicainide and cibenzoline), and amiodarone was ineffective in preventing the VT. The late potential was positive (filtered QRS duration: 139 ms, root mean square voltage of the terminal 40 ms of the filtered QRS complex: 6.6 µV; the duration of low-amplitude signals <40µV of the terminal filtered QRS complex: 56 ms). Transthoracic echocardiography showed nonobstructive HCM (LV ejection fraction: 88.8%, left atrial dimension: 36.9 mm, LV end-diastolic dimension: 31.8 mm, LV end-diastolic dimension: 13.4 mm, interventricular septum thickness: 19.4 mm, LV posterior wall thickness: 10.9 mm) and 2-dimensional echocardiography showed mid-septal hypertrophy. After written informed consent was given by the patient, coronary angiography, left ventriculography and electrophysiologic study were performed in the postabsorptive state after all antiarrhythmic drugs, except amiodarone, had been discontinued for at least 5 lives. Coronary angiography was normal and left ventriculography showed there was neither obstruction in the LV nor pressure gradients within the LV or between the LV and aorta. Three diagnostic catheters were inserted via the femoral veins and placed at the high right atrium, His bundle position and right ventricular apex. A decapolar catheter was inserted into the coronary sinus via the right internal jugular vein. Endocardial bipolar electrograms were recorded with a filter bandwidth of 30–500 Hz. Electrograms were recorded simultaneously with the 12-lead surface ECG and stored in a computer system (CardioLab, Prucka Engineering, Houston, TX, USA). Incessant nonsustained VT with a QRS duration of 130 ms, RBBB pattern, northwest axis and cycle length of 320 ms was recorded (VT1). The QRS morphology was identical to that during a clinical attack. VT1 was not induced by programmed atrial or ventricular stimulation. Rapid atrial or ventricular pacing was not performed be-
cause of hemodynamic deterioration, RF catheter ablation and LV mapping were performed with a 7-F deflectable electrophysiology catheter (4-mm tip electrode, 2-mm interelectrode spacing, and embedded thermistor [EP Technologies, Inc, Sunnyvale, CA, USA]). Unipolar RF current was applied at a target temperature of 60°C and maximum power output of 50 W (EPT-1000, EP Technologies) at the left posteroseptum near the apex (Fig 3, Right panel), the site where a sharp presystolic Purkinje potential had been recorded preceding the earliest ventricular activation on the surface ECG by 58 ms during VT (Fig 3, Left panel) and where a small Purkinje potential had been recorded just before local ventricular activation during sinus rhythm (Fig 3, Left panel, LV 3–4, short arrow). RF energy application 3 times at this site successfully terminated VT1, and VT1 became noninducible with isoproterenol infusion of 2 μg/min and programmed ventricular stimulation.

During application of RF energy, transient acceleration of the VT rate was noted before disappearance of the VT. However, incessant slow VT with a similar morphology to that of VT1 with a QRS duration of 112 ms, RBBB pattern, and northwest axis, and cycle length of 560 ms (VT2) recurred 1 week later. Thus, RF catheter ablation was repeated adjacent to the VT1 ablation site (Fig 4, Right panel), the site where a sharp presystolic Purkinje potential had been recorded preceding the earliest ventricular activation on the surface ECG by 38 ms during VT (Fig 4, Left panel). RF energy application (3 times) at this site successfully eliminated VT2, and an isolated sharp Purkinje potential appeared in the mid-diastolic phase during sinus rhythm (Fig 5). Surface 12-lead ECG after the second session showed a shift of the electrical axis in the inferior direction (from –23° before ablation to +65° after ablation) and the appearance of S I Q III T III pattern, suggesting left posterior hemiblock and an intraventricular conduction disturbance (Fig 6). Antiarrhythmic drugs were not administered and no clinical recurrence of VT1 or VT2 was documented during the 24-month follow-up period.

**Discussion**

We report a case of hemodynamically intolerable incessant VT in a patient with nonobstructive HCM. The incessant VT was triggered, and possibly maintained, by activity originating from distal Purkinje arborization localized to the left posterior fascicle of the left bundle branch and it was successfully abolished by RF ablation. Horowitz et al.9 and Arnar et al.10 showed that the subendocardial Purkinje network was the origin of ventricular arrhythmias occurring 24 h after myocardial infarction in dog models, and there is emerging evidence that Purkinje arborization is a dominant source of triggers for polymorphic VT or ven-
Fig 3. Intracardiac electrograms during incessant ventricular tachycardia (VT1, Left) and catheter position (Right) during the first radiofrequency ablation session. A sharp presystolic Purkinje potential (long arrow) precedes the onset of earliest ventricular activation on the surface ECG by 58 ms. A Purkinje potential is also present during sinus rhythm and fusion beat (short arrow). The ablation catheter (ABL) is located at the left posteroseptum near the apex (arrow). HRA, high right atrium; HBE, His bundle electrogram; LV, left ventricle; CS, coronary sinus; RV, right ventricle; TCL, tachycardia cycle length; AP, anteroposterior view; LAO, left anterior oblique view.

Fig 4. Intracardiac electrograms obtained during incessant ventricular tachycardia (VT2, Left) and catheter position (Right) during the second radiofrequency ablation session. A sharp presystolic Purkinje potential (arrow) precedes the onset of earliest ventricular activation on the surface ECG by 38 ms. The ablation catheter (ABL) is located at the left posteroseptum near the apex (arrow). HRA, high right atrium; HBE, His bundle electrogram; LV, left ventricle; CS, coronary sinus; RV, right ventricle; TCL, tachycardia cycle length; RAO, right anterior oblique view; LAO, left anterior oblique view; MCV, mid-cardiac vein.
tricular fibrillation in patients with various clinical conditions. Ablation of these triggers eliminates further arrhythmias. The electrocardiographic and electrophysiological characteristics of incessant VT in the present patient were similar to those in previous reports (i.e., all premature ventricular beats originated from the distal Purkinje network and the sources were eliminated by focal energy delivery). The mechanism of VT in this case could not be determined, but we speculate that it was triggered activity or abnormal automaticity, although the possibility of unstable reentry within the Purkinje network still remains. Kim et al reported that reentry within the Purkinje network adjacent to the papillary muscle played an important role in the initiation and maintenance of ventricular fibrillation in a model using isolated swine right ventricle. In the present case, the VT rate after the first session was slower than that before the first session and we speculate that the VT originated from the distal Purkinje network area, with the first ablation targeting the faster VT foci only. Thus, the slower VT that appeared after the first session may have originated from residual distal Purkinje network area where the slower VT was suppressed by the faster VT. In addition, we observed retrograde Purkinje activation during sinus rhythm following successful catheter ablation of the incessant VT during the second session. The mechanism of retrograde Purkinje activation is conduction block of the left posterior fascicle by ablation and retrograde activation of the distal posterior fascicle through right bundle branch or left anterior fascicle activation. Kuboki et al reported hypertrophied Purkinje fibers in the left bundle branch in a patient with HCM who developed torsades de pointes, and they suggested a role of Purkinje fibers in the development of this condition. Thus, similar to idiopathic VT and ventricular fibrillation, and some ischemia-related VTs, incessant non-sustained VT originating from the Purkinje fiber also occurs in HCM, and RF ablation targeting the presystolic Purkinje potentials is effective for curing the VT.

References


Fig 5. Isolated sharp Purkinje potential in the mid-diastolic phase during sinus rhythm after successful ablation of VT2 (arrow). HRA, high right atrium; HBE, His bundle electrogram; LV, left ventricle.

Fig 6. Twelve-lead ECG after the second ablation session. Note the electrical axis has shifted in the inferior direction (from ~23° before ablation to +65° after ablation) and the intraventricular conduction disturbance as shown by the wide QRS interval.


