Radiofrequency Catheter Ablation of Ventricular Tachycardia Originating in the Left Posterior and Left Anterior Fascicles in a Patient With Prior Myocardial Infarction

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SUMMARY

A 61-year-old man with prior anteroseptal myocardial infarction (ejection fraction: 40%) presented with recurrent episodes of palpitations. Twelve-lead ECG during palpitations showed an incessant ventricular tachycardia (VT1) with right bundle branch block (RBBB) morphology and inferior axis. Electrophysiologic study revealed that the clinical VT originated from the anterolateral left ventricle. A Purkinje potential preceded onset of the QRS complex by 34 ms. Radiofrequency ablation guided by the Purkinje potential terminated the VT1. Another ventricular tachycardia (VT2) showing RBBB morphology with superior axis and originating from the posteroseptal left ventricle, was induced by programmed ventricular stimulation. A Purkinje potential preceded onset of the local ventricular potential by 120-130 ms in this VT. Radiofrequency ablation guided by the Purkinje potential terminated the VT2. (Int Heart J 2008; 49: 119-127)

Key words: Ventricular tachycardia, Myocardial infarction, Purkinje potential, Catheter ablation

MONOMORPHIC ventricular tachycardia (VT) in postmyocardial infarction (MI) is largely attributable to anatomically bound macro-reentry involving regions of myocardial scarring. Increased understanding of such mechanisms has led to the ability to map and identify critical isthmuses that create the substrate necessary for these arrhythmias, allowing for their ablation.1-5) With regard to VT that includes the His-Purkinje system in post-MI patients, bundle branch reentry and interfascicular reentry have been reported, but monomorphic reentrant VT...
originating from the left fascicular Purkinje fibers and not involving another fascicle or the right bundle branch is rare. Herein, we describe a previously unreported combination of two different morphologies of fascicular VT in the same post-MI patient and successful ablation of these VTs.

**CASE REPORT**

A 61-year-old man who had suffered anteroseptal MI 13 years earlier experienced palpitations and visited his family doctor. Twelve-lead ECG during sinus rhythm showed a QS pattern in V1 to V5 and ventricular premature complexes (VPCs) with right bundle branch block (RBBB) morphology (Figure 1). VT with a cycle length of 431 ms occurred spontaneously during ECG recording (Figure 2). The patient was referred to our hospital. Serum enzyme tests revealed no myocardial enzyme elevation. Left ventriculography, coronary angiography, and electrophysiologic study were performed 2 days after admission. Left ventriculography showed dyskinesia at the apex and akinesia at the mid-apical septum and anterolateral wall of the left ventricle with an ejection fraction of 40%. Coronary angiography revealed an occluded left anterior descending coronary artery with

![Figure 1](image1.png)  
*Figure 1.* Twelve-lead electrocardiogram (ECG) recorded at the family doctor’s office. Twelve-lead ECG shows a QS pattern in V1-V5 and ventricular premature beats with right bundle branch block morphology.
grade II collateral blood flow from the right coronary artery. No significant stenosis was found in the left circumflex or right coronary arteries.

**Electrophysiologic study and catheter ablation:** Written informed consent for electrophysiologic study and ablation was obtained from the patient. The study was performed with the patient in the fasting state. Sedation was achieved with midazolam and fentanyl. Electrode catheters were placed via the femoral veins: a quadripolar electrode catheter in the right atrial appendage and another in the right ventricular apex and an octapolar electrode catheter in the His-bundle region. A decapolar catheter with an end-hole was positioned in the coronary sinus via the right internal jugular vein. Surface and bipolar endocardial electrograms were monitored continuously and recorded on a Cardio Lab system (Prucka Engineering, Houston TX, USA) at a filter setting of 30 to 500 Hz. Bipolar pacing was performed with a BC-02 cardiac stimulator (Fukuda Denshi, Tokyo). Programmed right ventricular stimulation was performed with a maximum of 3 extra stimuli at 2 basic cycle lengths of 600 and 400 ms from the right ventricular apex and the outflow tract, respectively. During sinus rhythm, the AH interval was 129 ms and the HV interval was 57 ms. Sustained VT with a cycle length of 370 ms and a QRS configuration matching that of the clinical VPC was
Figure 3. Twelve-lead ECG of the VT1 and VT2.
VT1 showed an RBBB morphology with an inferior axis that was similar to that of the clinical VT shown in Figure 2. VT2 showed an RBBB pattern with a left superior axis.

Figure 4. Surface and intracardiac recordings during VT1.
Vertical caliper lines on the second beat of the tracing indicate a high frequency potential and the onset of the surface QRS complex. High-frequency Purkinje-like potentials (arrows) precede the onset of the VT electrograms by 34 ms. HRA indicates high right atrium; HIS, His bundle electrogram recording site; RV, right ventricle; and ABL, ablation catheter.
reproducibly induced by performing programmed stimulation with double extra-
stimuli (400/220/230 ms) at the right ventricular apex (VT1). Another VT (VT2) with a cycle length of 360 ms, RBBB morphology, and left superior axis was induced by performing programmed stimulation with double extrastimuli (400/260/260 ms) at the right ventricular outflow tract (Figure 3). A 7F 4-mm-tip quadripolar catheter (Blazer II, Standard Curve, EP Technologies, San Jose, CA, USA) introduced via a retrograde transaortic approach was used to perform left ventricular mapping and catheter ablation. Ablation was performed using a radiofrequency generator (EPT-1000-XP, EP Technologies). During VT1, a pre-
systolic Purkinje potential was recorded just before onset of the local ventricular electrogram and 34 ms before onset of the QRS complex was recorded at the mid-
portion of the anterolateral left ventricle (Figures 4, 5). No delayed potentials or fragmented potentials were recorded during sinus rhythm at that site. Radiofre-
quency energy (55°C and 30 W for 60 seconds) delivered to this site terminated VT1 within 5 seconds. During radiofrequency energy delivery, left anterior hemiblock developed, and a Purkinje potential was located after the ventricular electrogram during sinus rhythm (Figure 6). VT1 could not be induced by pro-
Figure 6. Surface and intracardiac recordings after ablation of VT1. High-frequency Purkinje-like potentials are recorded after the local ventricular electrogram (arrows) after radiofrequency ablation of the VT1. Abbreviations as in Figure 4.

Figure 7. Entrainment of the VT2 by rapid right atrial pacing. Manifest entrainment of the VT2 (cycle length of 360 ms) was shown by right atrial pacing from the right atrium at a cycle length of 340 ms.
grammed ventricular stimulation. However, VT2 was still inducible. During VT2, a diastolic Purkinje potential recorded at the mid-portion of the posteroseptum preceded the ventricular electrogram by 120-130 ms. Rapid racing from the high right atrium at a cycle length of 340 ms during VT2 showed manifest entrainment with progressive fusion (Figure 7). VT2 was terminated with Purkinje-ventricular block (Figure 8). Radiofrequency energy (55°C and 30 W for 60 seconds) delivered to this site terminated VT2 within 4 seconds. The QRS morphology and HV interval of the sinus rhythm did not change by VT2 ablation. VT1 and VT2 could not be induced after ablation, but rapid VT with hemodynamic deterioration was induced by programmed ventricular stimulation at the right ventricular outflow tract. An implantable cardioverter-defibrillator (ICD) was implanted on the next day. No antiarrhythmic drugs were administered, and the patient did not experience ICD discharge for 3 years.

**DISCUSSION**

VT that involves the left posterior or anterior fascicle as a part of the reentrant circuit occurs in patients without obvious structural heart disease, and the

![Diagram of DP VT2](image-url)
mechanism of this VT has been shown to be macro-reentry.\textsuperscript{12-15)} Experimental studies have shown that VT with a focal mechanism originating from subendocardial Purkinje fibers is common in the early ischemic period.\textsuperscript{16,17)} VPCs triggering polymorphic VT or ventricular fibrillation after MI have also been shown to originate from the left ventricular Purkinje fibers in humans, and triggered activity from surviving Purkinje fibers has been hypothesized as the underlying mechanism of the VPCs.\textsuperscript{18-20)} In the present case, spontaneous clinical VT (VT1) was incessant with an irregular RR interval, thus, the mechanism of the VT in our case might indicate triggered activity as shown by previous studies.\textsuperscript{18-20)} However, a recent study showed that reentrant monomorphic VT originating from the left posterior Purkinje fibers can develop in the acute or chronic phase of MI.\textsuperscript{11)} The mechanism of the VT1 may be bundle-branch reentry or interfascicular reentry. However, a His bundle electrogram was not recorded during VT1, while it was recorded during sinus rhythm. Thus, the possibility of bundle-branch reentry or interfascicular reentry is less likely. A limitation of this case report is that we did not perform entrainment mapping during VT1. Thus, the exact mechanism of VT1 could not be determined. His bundle was also not recorded during VT2, but it was recorded during sinus rhythm. Thus, the possibility of bundle-branch reentry or interfascicular reentry is less likely. However, manifest entrainment with progressive fusion was obtained by rapid atrial pacing during VT2 (Figure 7). Thus, reentry around the posterior fascicle is the mechanism of the VT2. A previous case report described idiopathic VT originating from the left anterior and left posterior fascicles.\textsuperscript{21)} A previous report on polymorphic VT after MI indicated that the Purkinje potentials triggering polymorphic VT showed more than 2 morphologies in 4 of the 5 patients.\textsuperscript{20)} However, to the best of our knowledge, ours is the first report of 2 morphologies of monomorphic VT originating from the left anterior and left posterior fascicles in a patient with prior MI as well as successful ablation of these VTs.

REFERENCES