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CASE REPORT

**Tamotsu Sakamoto · Akira Fujiki · Yosuke Nakatani
Masao Sakabe · Koichi Mizumaki · Hiroshi Inoue**

Narrow QRS ventricular tachycardia from the posterior mitral annulus without involvement of the His-Purkinje system in a patient with prior inferior myocardial infarction

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Abstract A 54-year-old man with prior inferior myocardial infarction suffered from monomorphic ventricular tachycardia (VT) with narrow QRS complex of 120 ms. During VT, a fragmented prepotential preceding QRS onset by 30 ms at the right ventricular posterior septum and a late diastolic potential preceding QRS onset by 70 ms at the infarcted posterior mitral annulus were recorded. Radiofrequency energy delivered to the late diastolic potential at the posterior mitral annulus eliminated VT. During sinus rhythm, the late diastolic potential shifted to the end of QRS complex and no Purkinje potentials were observed. Synchronized excitation of both ventricles from the posterior infarcted mitral annulus in this patient may make the QRS width during VT narrow, without involvement of the His-Purkinje system.

Key words Ventricular tachycardia · Narrow QRS complex · Myocardial infarction · Radiofrequency ablation · Synchronized excitation

Introduction

Monomorphic ventricular tachycardia (VT) originating in the His-Purkinje fiber has narrow QRS morphology despite the presence of structural heart disease.[1–3] It is also known that VT from the septal region shows a narrow QRS complex compared with VT from the free wall region.[4] On the other hand, VT originating from the mitral annulus showed a wide QRS complex, and VT from the anterolateral and the posterior mitral annulus had an average QRS duration of 164 ms.[5] This report describes a VT with narrow QRS complex (120 ms) originating from the posterior mitral annulus in a patient with prior inferior myocardial infarction.

T. Sakamoto · A. Fujiki (✉) · Y. Nakatani · M. Sakabe · K. Mizumaki · H. Inoue
Second Department of Internal Medicine, Faculty of Medicine, University of Toyama, 2630 Sugitani, Toyama 930-0194, Japan
Tel. +81-76-434-7297; Fax +81-76-434-5026
e-mail: fujiki@med.u-toyama.ac.jp

Case report

A 54-year-old man was admitted with a complaint of anterior chest pain that began after jogging. On admission the patient's electrocardiogram (ECG) showed sinus rhythm and ST-segment elevation in leads II, III, and aVF. A diagnosis of acute coronary syndrome was made on the basis of the presence of typical chest pain, significant ECG changes, echocardiographic signs, and elevated levels of cardiac enzymes including the MB fraction of creatine phosphokinase and troponins. The patient underwent emergency coronary angiography, which demonstrated an occlusion of the mid right coronary artery (RCA). The RCA lesion was dilated with a balloon, stented with a bare-metal stent, and thrombolytic therapy with tissue plasminogen activator was administered; however, revascularization was not achieved because of severe coronary thrombosis. Left ventriculography showed an ejection fraction of 51% with hypokinetic inferior wall motion.

The patient experienced palpitation on effort 1 month after an acute episode of myocardial infarction, and 12-lead ECG during palpitation showed narrow QRS complex (120 ms) tachycardia at a rate of 140/min (Fig. 1, right). The existence of atrioventricular dissociation and fusion beats led to the diagnosis of VT. Catheter ablation was performed using standard methods after obtaining written informed consent with regard to the possibility of His-Purkinje related VT. Multielectrode catheters were inserted and positioned at the high right atrium (HRA), the His bundle electrogram (HBE) region, the right ventricle (RV), and the coronary sinus (CS). Bipolar intracardiac electrograms filtered between 30 and 500 Hz were simultaneously recorded and stored digitally on a CardioLab system (Prucka Engineering, Houston, TX, USA) with a 12-lead surface ECG. Stimuli were delivered from a programmable stimulator (Nihon-Kohden SEC3102, Tokyo, Japan) as rectangular

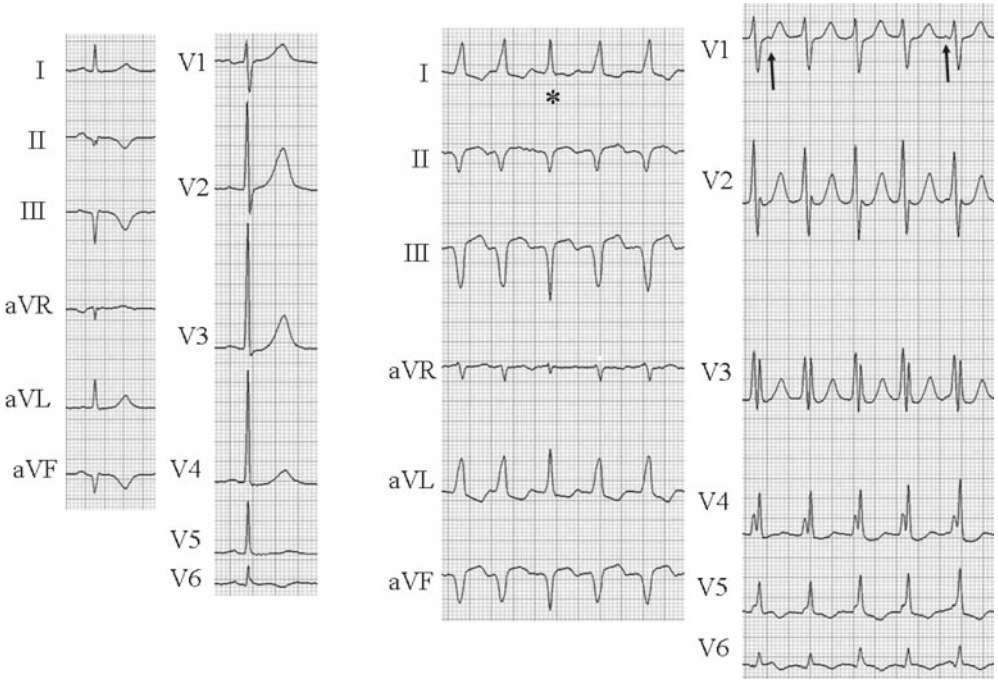

Fig. 1. Twelve-lead ECG during sinus rhythm and ventricular tachycardia (VT). **Left** During sinus rhythm, abnormal q and coronary T in leads II, III, and aVF, and tall R and tall T in lead V2 suggest previous inferoposterior myocardial infarction. **Right** During VT, QRS width was narrow at a rate of 140/min. This narrow QRS tachycardia is diagnosed as VT on the basis of the existence of atrioventricular dissociation (*arrows*) and fusion beat (*asterisk*)

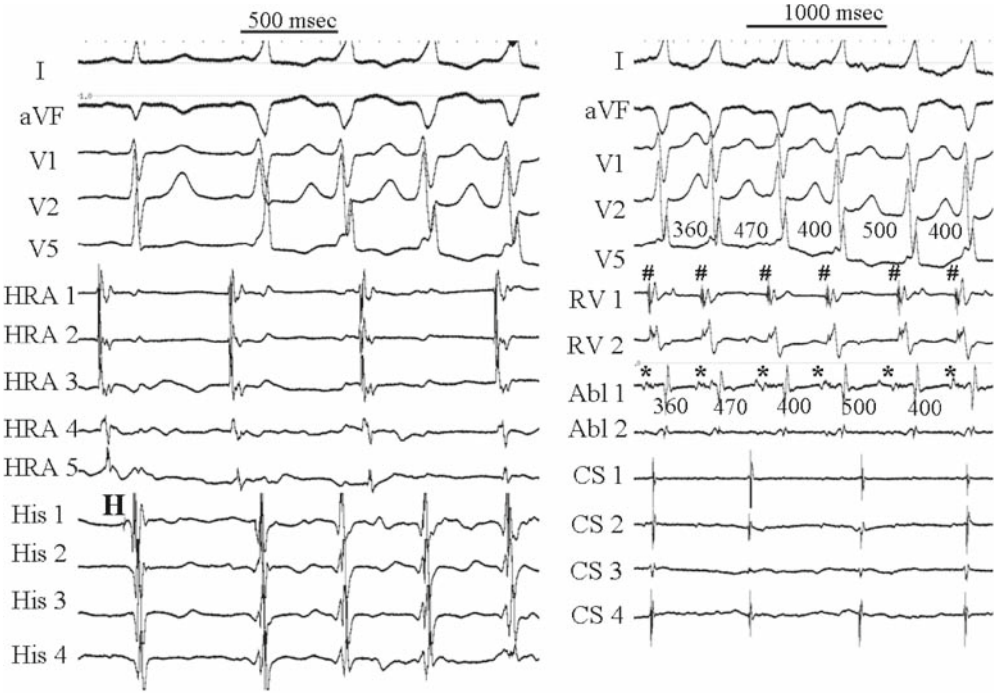

Fig. 2. Intracardiac recordings during VT. **Left** During sinus rhythm HV interval (40 ms) was normal and His bundle electrogram (*H*) disappeared during VT. On the figure, five surface ECG leads are represented (*I*, *aVF*, *V1*, *V2*, *V5*) as well as nine bipolar intracardiac recordings. *HRA*, high right atrium electrogram; *HBE*, His bundle electrogram. **Right** During VT, a fragmented prepotential (*hatches*) at the right ventricular posterior septum, and a late diastolic potential (*asterisks*) at the left ventricular posterior wall close to the mitral annulus were recognized. The RR interval during VT changed according to the preceding interval of the late diastolic potentials (*asterisks*). *Abl*, ablation catheter electrogram; *RV*, right ventricular electrogram; *CS*, coronary sinus electrogram

pulses of 2 ms and twice the diastolic threshold. During pace mapping, stimulation strength was increased up to 9 V until successful ventricular capture was achieved at a basic cycle length of 400 ms. Ablation was performed between the distal electrode of a 7-F deflectable catheter with a 4-mm ablation tip (EP Technologies, Sunnyvale, CA, USA) and a skin electrode with a 550-kHz unmodulated radiofrequency (RF) current from a generator with temperature monitoring (EP Technologies). The RF energy was applied in a temperature-controlled mode, with an upper temperature limit of 55°C, maximal power output of 30 W, and duration of 40 s for each application.

During sinus rhythm at a cycle length of 750 ms, the AH interval was 120 ms and the HV interval was 40 ms. Non-sustained VT at a cycle length ranging from 360 to 470 ms with the same QRS configuration as the clinical VT occurred spontaneously. Initially, VT was thought to originate from the His-Purkinje system because of narrow QRS complex VT, but the His bundle electrogram did not precede the ventricular electrogram during VT (Fig. 2, left). Ventricular

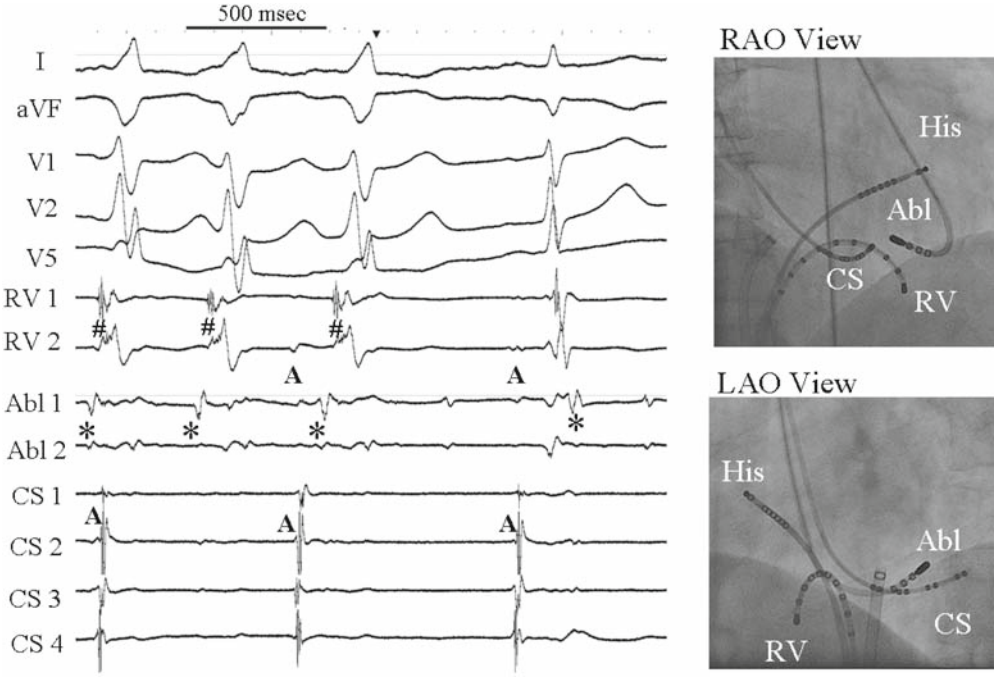

Fig. 3. Intracardiac recordings during VT (**left**) and catheter position (**right**). During VT, a fragmented prepotential was recorded in the RV 1 at the right ventricular posterior septum and it preceded QRS onset by 30 ms (*hatches*). In the Abl 1, located at the left ventricular posterior wall near the mitral annulus, a delayed potential during sinus rhythm became a late diastolic potential during VT, which preceded QRS onset by 70 ms (*asterisks*). Other diastolic potentials in the Abl 1 did not have significant relation to occurrence of VT. *Abl*, ablation catheter electrogram; *RV*, right ventricular electrogram; *CS*, coronary sinus electrogram; *LAO*, left anterior oblique view; *RAO*, right anterior oblique view

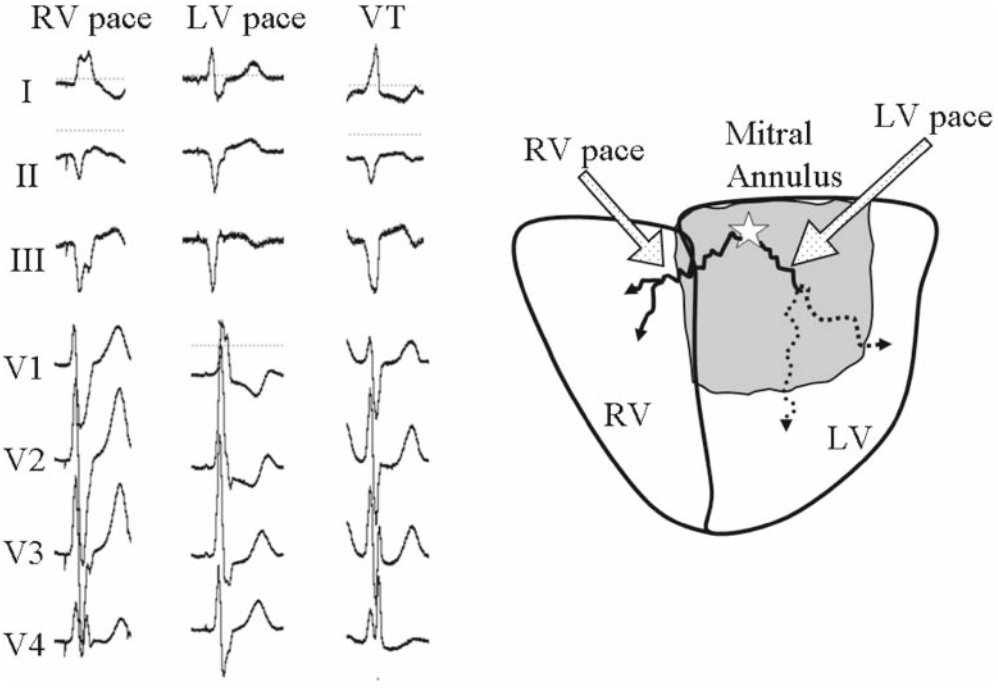

Fig. 4. QRS morphology during pace mapping and hypothetical mechanism of this narrow QRS complex VT. **Left** Pace mapping at the right ventricle posterior septum involved in the right-sided breakthrough site of excitation wavefront from the infarcted area resulted in left bundle branch block morphology. Pace-mapping at the left ventricular posterior wall close to the mitral annulus (the successful ablation site) resulted in right bundle branch block morphology. **Right** *Gray area* indicates the infarcted zone. *Star* indicates the ablation success site. *Dotted lines* in the LV indicate speculated excitation route. *RV*, right ventricle; *LV*, left ventricle

tachycardia could not be induced by programmed atrial or ventricular stimulation, and entrainment pacing was difficult because of short duration of VT. No direct evidence suggesting re-entry as the mechanism of this VT was obtained. During VT, a fragmented prepotential (hatches in figures) at the right ventricular posterior septum, which preceded QRS onset by 30 ms, and a late diastolic potential preceding QRS onset by 70 ms (asterisks in figures) at the left ventricular posterior wall close to the mitral annulus were recognized (Fig. 2, right and Fig. 3). The RR interval during VT changed according to the preceding interval of the late diastolic potentials. The stimulus to QRS interval at the right posterior septum was similar to the fragmented prepotential to QRS interval during VT; however, the QRS morphology was different from spontaneous VT (Fig. 4, RV pace) and RF application failed to suppress VT. During sinus rhythm, the late diastolic potential shifted to the end of the QRS complex and no Purkinje activities were recognized (Fig. 3). Stimulation with maximum strength could not capture the ventricle at this site because of the high threshold of the infarcted myocardium. Stimulation around the site of the late diastolic potential induced right bundle

branch block (RBBB) morphology with prolonged stimulation-QRS interval of 70 ms (Fig. 4, LV pace). Radiofrequency energy delivered to the late diastolic potential recoding site induced bursts of clinical VT transiently, which was terminated within 10 s. After ablation, the patient remained free of VT recurrences during a follow-up of 12 months.

Discussion

In the present case, despite the posterior mitral annular origin of VT and the prior inferior infarction, the QRS complex of VT was narrow. During VT, the fragmented prepotential at the right posterior septum preceded the QRS onset by 30 ms, but the left bundle branch block morphology during the stimulation at this right ventricular septal site was wide and different from the spontaneous VT morphology. The successful ablation site showed the late diastolic potential preceding QRS onset by 70 ms at the posterior mitral annuls in the infarcted area. This late diastolic potential shifted to the end of the QRS complex during sinus rhythm, and no Purkinje potentials were observed. Stimulation at the infarcted left ventricular area induced RBBB morphology with a relatively narrow QRS complex, suggesting the left ventricular predominance of the fusion beat (Fig. 4). The fusion beat of both morphologies from the right and the left ventricular stimulation seems to be similar to the spontaneous VT morphology.

These observations suggest that the origin of the VT was located at the posterior mitral annular region (the successful ablation point), and the excitation waves from this site conducted through the infarcted area to the narrow exit at the right posterior septal site and to the left ventricular apical area simultaneously. Although the exact exit sites from the infarcted area to the left ventricular apical area could not be determined, it is possible that synchronized excitation of both ventricles makes the width of QRS complex during VT narrow, without involvement of the conduction through the His-Purkinje system. The ablation at the posterior mitral annulus induced spontaneous VT transiently, but the electrical stimulation around this site could not induce the spontaneous VT morphology. The mechanism of this discrepancy is unclear, but complexity of the conduction properties of the surviving myocardium within the infarcted area may play a role. In conclusion, synchronized excitation of both ventricles similar to cardiac resynchronization therapy from the infarcted area may cause narrow QRS VT without involvement of the His-Purkinje system.[6] A closer evaluation that reveals the underlying mechanism of ECG changes is essential for the individualized treatment of arrhythmia.[7]

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