Purkinje-related ventricular tachycardia with upper septal and left posterior fascicular type in a patient of post–myocardial infarction

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Introduction

Idiopathic left ventricular tachycardia (ILVT) is commonly recognized as a Purkinje-related reentrant ventricular tachycardia (VT) in patients without underlying heart disease,[1] but it has also been reported to manifest subsequent to myocardial infarction (MI).[2] In this report, we present 2 forms of narrow QRS VT that emerged during the chronic phase of MI. These tachycardias were diagnosed as reentrant tachycardia involving the injured Purkinje network and treated with ablation without damaging the fascicular conduction system. Additionally, VT with an extremely narrow QRS was speculated to have similarities to the upper septal type of ILVT.

Case report

A 74-year-old man, with a history of inferior MI 25 years ago, had palpitations associated with narrow QRS tachycardia and had been treated with verapamil by a previous physician for about a year as paroxysmal supraventricular tachycardia. However, the tachycardia recurred frequently after verapamil was discontinued because of sinus bradycardia. Coronary angiography showed no new coronary lesion. Therefore, the patient was referred to our hospital.

Twelve-lead electrocardiogram exhibited a normal axis and poor R progression in V_1 lead during sinus rhythm. During tachycardia, a narrow QRS complex with 114 ms beating at 130 beats per minute and a right bundle branch block (RBBB) pattern in V_1 lead were observed. Atrioventricular dissociation was observed, indicating VT (Figure 1A). The echocardiogram revealed thinning and reduced wall motion from the base to the middle of the inferior wall.

Catheter ablation for tachycardia was performed with the patient's consent. During sinus rhythm (53 beats/min), AH interval was 120 ms, HV interval was 44 ms, and QRS duration was 118 ms. Both atrial and ventricular programmed stimulations induced clinical tachycardia showing atrioventricular dissociation with a cycle length (CL) of 420 ms. HV interval was 33 ms, which was shorter than in sinus rhythm, suggesting VT (VT1).

Overdrive pacing from the right ventricle apex during VT1 resulted in constant and progressive fusion. VT1 presented complete right bundle branch block pattern and the pace map from the upper septum of the right ventricle showed different QRS morphology from VT1, indicating left ventricular origin. The left ventricle (LV) voltage map during sinus rhythm and activation map during VT1 were obtained by using the EnSite system (Abbott, St. Paul, MN) and a multipolar catheter (Advisor HD Grid; Abbott). The voltage map during sinus rhythm showed a low-voltage area (<1.5 mV) extending from the basal septum to the inferior wall (Figure 1B). The activation map of VT1 showed a preceding excitation entering from the basal septum, followed by a spreading excitation from the proximal left bundle branch to the peripheral Purkinje system and LV myocardium

KEY TEACHING POINTS

- Even in patients with post–myocardial infarction, verapamil-sensitive fascicular ventricular tachycardia may occur. This type of tachycardia is characterized by relatively narrow QRS waveform.
- The injured Purkinje network might create a reentrant circuit of ventricular tachycardia, and abnormal Purkinje potentials can be detected during tachycardia using a multipolar mapping catheter.
- Catheter ablation targeting the abnormal Purkinje potentials allowed termination of ventricular tachycardia without disrupting the normal conduction system.

KEYWORDS Purkinje-related ventricular tachycardia; Narrow QRS tachycardia; Reentrant tachycardia; Catheter ablation; Verapamil
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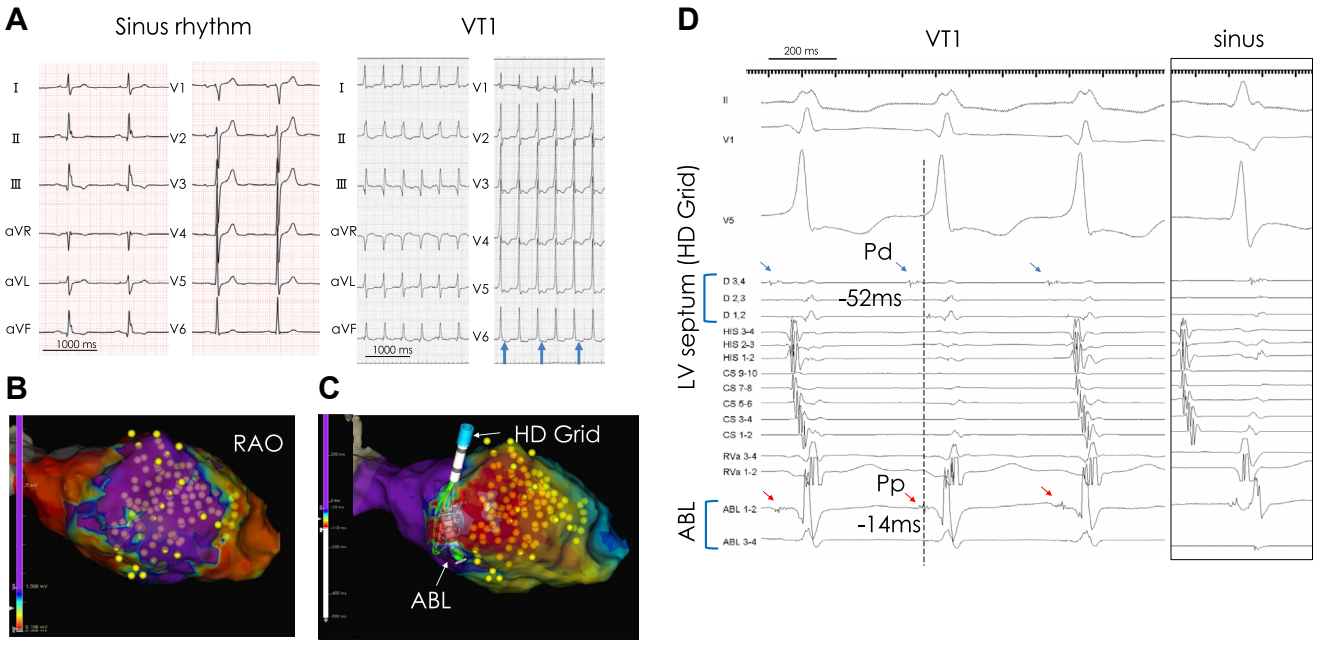

Figure 1 Electrocardiograms and 3-dimensional maps obtained in the case of VT1. **A:** Twelve-lead electrocardiography at baseline and tachycardia (VT1). VT1 is characterized by a relatively narrow QRS tachycardia, with dissociation of p wave from QRS complex, as denoted by the blue arrowhead. **B:** Voltage map (Ensite; Abbott, St. Paul, MN) showed the fascicular and Purkinje potential (yellow tags). Low-voltage area extends from the basal septum to the posterior wall. **C:** Activation map (Ensite) during VT1; multipolar catheter (Advisor HD Grid; Abbott) and ablation catheter were placed at the basal septum. **D:** Intracardiac electrograms of VT1 and sinus rhythm. VT1-Pd (indicated by the blue arrow) and VT-Pp (marked with the red arrow) were recorded by HD Grid and ablation catheter during VT1. VT1-Pd, observed on HD Grid D3,4 electrodes, precedes the onset of the QRS complex by 52 ms, while VT-Pp also precedes the QRS complex onset by 14 ms. The earliest excitation sites are highlighted in the white area. Dotted line indicates the onset of QRS complex. During sinus rhythm, VT1-Pd represented fractionated potential within the QRS complex. ABL = ablation catheter; LV = left ventricular; Pd = diastolic Purkinje potential; Pp = presystolic Purkinje potential; RAO = right anterior oblique.

(Figure 1C). To clarify the normal conduction system, the fascicular and Purkinje potentials picked up by the HD Grid during sinus rhythm were yellow tagged, as shown in Figure 1B. At the upper septal region, during VT1 a diastolic Purkinje potential (VT1-Pd) was detected in HD Grid D3,4 electrodes that preceded the QRS complex onset by 52 ms, and a presystolic Purkinje potential (Pp) was recorded by the distal tip of the ablation catheter placed behind the left posterior fascicle (LPF) (Figure 1D). Entrainment pacing at the VT1-Pd exhibited concealed fusion and postpacing interval (PPI) – tachycardia cycle length (TCL) was 40 ms. These findings suggested that this site was the adjacent bystander or on the circuit of the VT with decremental conduction. The VT1-Pd potential was observed late in the QRS complex during sinus rhythm, indicating an anterograde conduction delay in the Purkinje system.

During the VT1 mapping, a relatively wide QRS VT (VT2) showing a QRS duration of 136 ms and CL of 405 ms appeared transiently, which was similar to a common type of ILVT with RBBB pattern and left axis deviation (Figure 2A). In VT2, Pd potential (VT2-Pd) preceding the QRS onset by 58 ms was recorded at the ablation catheter at the same site shown in Figure 1C and 1D; on the contrary, this potential was detected as VT1-Pp during VT1. Instead, VT1-Pd on HD Grid preceded QRS the most in VT1 (Figure 2B). Moreover, the HD Grid D3,4 recording VT1-Pd detected systolic Purkinje potential (VT2-Ps) within the QRS complex during VT2. Interestingly, the CL of VT1 was dependent on the VT1-Pd interval, and the CL of VT2 was dependent on the VT2-Pd interval. During VT2, the HD Grid showed an intermittent conduction block to VT1-Pd potential, indicating that VT1-Pd was a bystander during VT2. At the transition from VT2 to VT1, the interval from VT2-Pd to VT1-Pd was 435 ms, which is approximately equivalent to the QRS interval of 430 ms, and the slight difference was inferred to be approximately from each Pd to the QRS onset. These findings suggest that VT1 and VT2 circuits are independent pathways.

Thirty-five watts of radiofrequency energy was applied to the LV septum targeting the VT1-Pd, leading to the successful termination of VT1 and elimination of the Pd potential (Figure 3A and 3B). Following this, VT2 was spontaneously induced, and the VT2-Pd that was located behind the distal part of the LPF was targeted. Radiofrequency applications to VT2-Pd terminated VT2, and no further VT was induced.

The 12-lead electrocardiogram after ablation was unchanged, and the HV interval was not prolonged. The patient has been free from recurrent tachycardia for 2 years.

Discussion

There were 2 types of narrow QRS VT in a patient with post-MI presenting a reentrant mechanism. The estimated VT schemas are shown in Figure 3C. During VT1, VT1-Pd

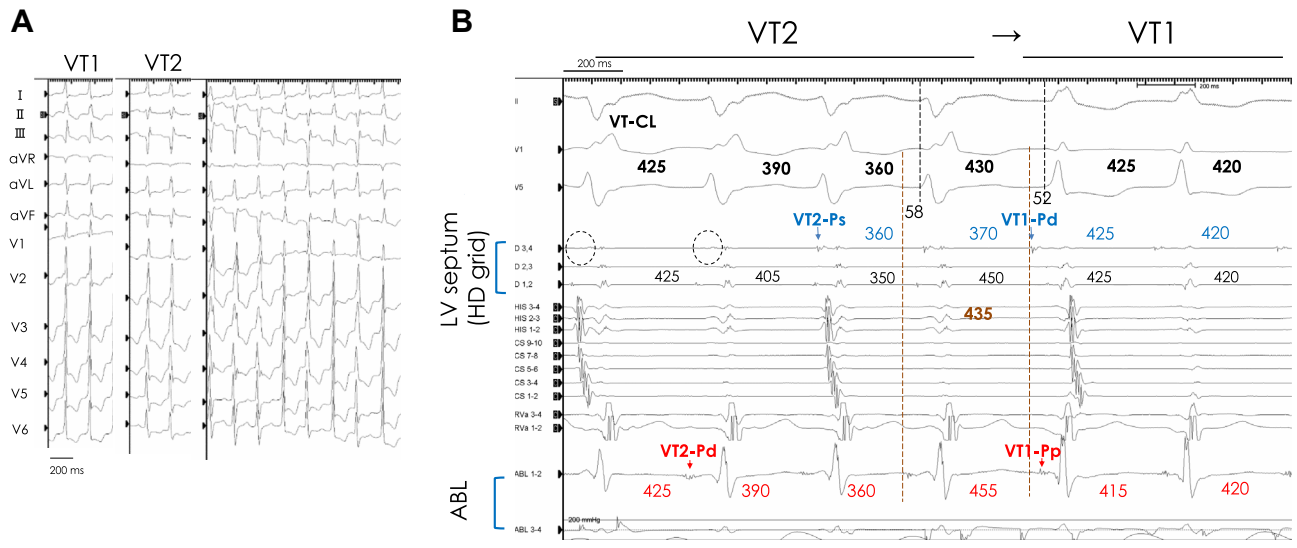

Figure 2 Electrocardiograms of VT1 and VT2. **A:** Twelve-lead electrocardiography of VT1 and VT2. These arrhythmias exhibit intermittent transitions between VT1 and VT2. **B:** Intracardiac electrograms of transition from VT2 to VT1. The HD Grid catheter (Abbott, St. Paul, MN) was placed at the basal septum of the left ventricle (LV) and the ablation catheter (Abl) was placed behind the left posterior fascicle. During VT2, the VT2-Pd on the ablation catheter precedes QRS onset, whereas during VT1 the VT1-Pd on the HD Grid precedes QRS onset. And during VT2, the Pd on the ablation catheter was the earliest activation and defined the QRS interval of VT. On the other hand, during VT1 the Pd potential on the HD Grid preceded and defined the QRS interval of ventricular tachycardia. Moreover, during VT2, the potential on HD Grid 3-4 showed an intermittent conduction block, indicating that it was not related to the VT2 circuit. When transitioning from VT2 to VT1, the interval from the Pd defining VT2 to the Pd defining VT1 was 435 ms, which was almost equivalent to the QRS interval of 430 ms. Dotted line indicates the onset of QRS complex. Dotted circle indicates intermittent block of VT1-Pd during VT2. Ps = systolic Purkinje potential.

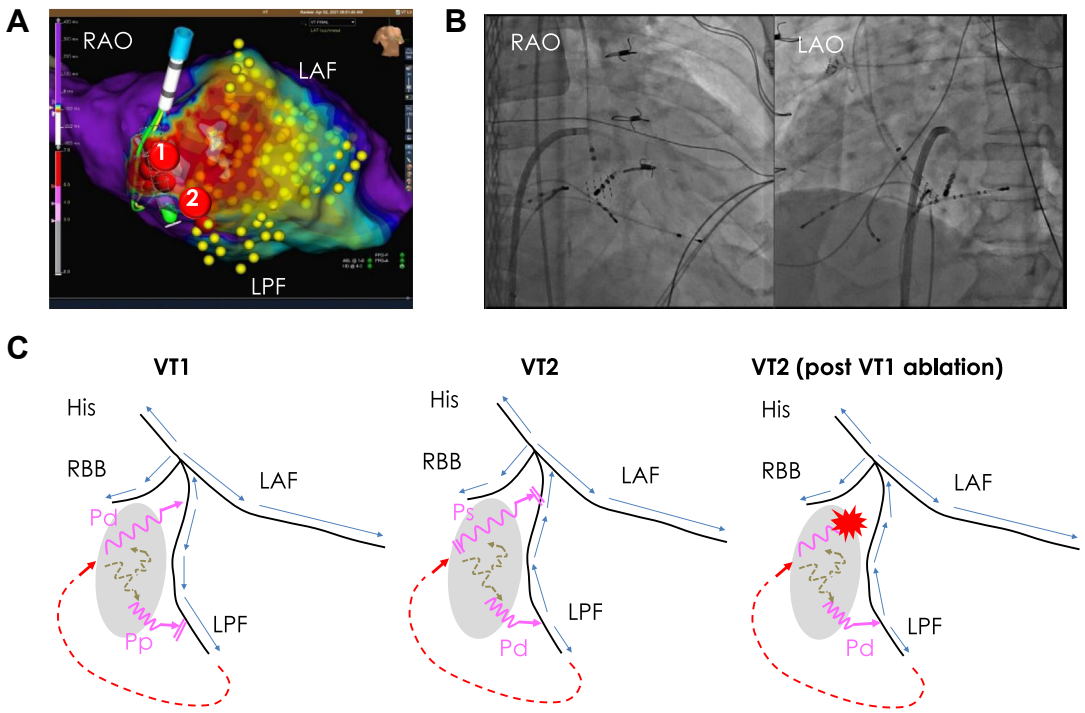

Figure 3 Ablation site and schematic picture of the estimated reentrant circuit of VT1 and VT2. **A:** Three-dimensional map shows ablation catheter at the success site of VT1 termination. Sites labeled 1 and 2 indicate the positions where VT1 and VT2 have terminated, respectively. **B:** Fluoroscopic image shows the location of the ablation catheter at the site of successful VT1 termination. **C:** During VT1, VT1-Pd connects to the proximal part of the left posterior fascicle (LPF), and excitation initiates upstream of the LPF and proceeds to retrograde His activation, left anterior fascicle (LAF), and right bundle branch (RBB). Therefore, QRS complex of VT1 was relatively narrow and the morphology was similar to that of idiopathic left ventricular tachycardia of the upper septal type. During VT2, VT1-Pd was passive activation from the Purkinje network, as shown by the gray wavy lines in the scar area. This finding suggests that conduction to VT1-Pd may be blocked owing to the refractory period. Conduction initially occurs in the direction of VT2-Pd, consequently exciting the LPF retrogradely, thereby preventing conduction from VT1-Pd. Following the ablation of VT1, conduction to VT1-Pd was blocked and only the circuit of VT2 remained. LAO = left anterior oblique; Pd = diastolic Purkinje potential; Ps = systolic Purkinje potential; RAO = right anterior oblique.

connects to the proximal part of the LPF, and excitation initiates upstream of the LPF and proceeds to retrograde His activation, left anterior fascicle, and right bundle branch (RBB), while the other excitation descends the LPF to reach the LV myocardium and reenter the VT1-Pd. The voltage map revealed a scar region extending from the basal septum to the inferior wall, where VT-Pd was located, suggesting the survival of injured Purkinje fibers, as indicated by continuous high-frequency potential findings and conduction delay behind the QRS onset during sinus rhythm. These pathways might serve as critical slow conduction pathways for reentrant VTs. However, we cannot rule out the possibility that the slow conduction site of this circuit also existed in the remaining myocardium within the infarct upstream of the Pd potential.

During VT2, VT1-Pd was detected within the QRS duration and not associated with the circuit, showing passive activation from the Purkinje network, as shown by the gray wavy lines in the scar area (Figure 3C). This finding suggests that conduction to VT1-Pd may be blocked owing to the refractory period. Conduction initially occurs in the direction of VT2-Pd, consequently exciting the LPF retrogradely, thereby preventing conduction from VT1-Pd. The conduction connected toward the LPF via VT2-Pd, which is positioned more distally than VT1-Pd. Consequently, the QRS duration was relatively wide in VT2. The transition between VT1 and VT2 circuits might be explained by a refractory period of VT1-Pd. Based on this speculation, it can be inferred that VT2 tends to have a shorter CL than VT1 because of the difference between the proximal and distal connection points of the LPF. Finally, after VT1 was terminated by ablation, only the circuit involving VT2-Pd remained, leading to the induction of VT2. Subsequent ablation targeting VT2-Pd also terminated VT2 (Figure 3C).

The finding that the PPI – tachycardia cycle length at the VT1-Pd recording site was >30 ms is presumed to be due to prolongation resulting from the decremental properties of injured Purkinje fibers or as an adjacent bystander within the VT circuit. Injured Purkinje tissue has been reported to exhibit decremental properties and sensitivity to verapamil.[3] Considering the decremental conduction of VT1-Pd, the corrected PPI was calculated by subtracting the difference in conduction time (stim-QRS and egm-QRS) from PPI.[4] In this case, stim-QRS was 90 ms and egm-QRS was 45 ms; thus the correct PPI was PPI − (90 − 45) = 415 ms, which approximated tachycardia cycle length (420 ms). Taken together with the effect of the ablation, this strongly suggests that VT1-Pd played a crucial role in the circuit.

ILVT without organic heart disease is the most common type of Purkinje-related VT. However, similar VT associated with post-MI has also been reported. Hayashi and colleagues[2] reported 4 cases of reentrant VT originating from the left posterior branch Purkinje after MI. The QRS morphology and successful ablation site closely resemble VT2 in this case. Furthermore, Bogun and colleagues[5] compared narrow QRS (<145 ms) and wide QRS (>145 ms) in 81 patients with VT that occurred after MI. They demonstrated that the narrow QRS VT was characterized by an RBBB pattern and often associated with inferior wall MI. Our case is also consistent with the RBBB type and the post-MI of the inferior wall. However, VT1 in this case had a narrower QRS than previously described and a normal axis, resembling the upper septal type ILVT (US-ILVT).

The injured Purkinje system is vulnerable to being a substrate for reentrant circuits owing to a disrupted gating mechanism.[6] According to Nogami and colleagues,[7] the septal branch of the left fascicle functions as a retrograde limb in the US-ILVT circuit. In the present case, the injured Purkinje fibers connecting to the proximal LPF might have functioned similarly to the septal branches in ILVT. However, it is difficult to distinguish whether these retrograde conducting Purkinje fibers connect to the LPF or the septal branch, because the dissection of the left septal fascicles is complicated and can be classified into 5 types based on their relationship to the left bundle branch, as reported by Pérez-Riera and colleagues.[8] In the US-ILVT, one of the main challenges is the risk of damage to the conduction system caused by ablation because the proximal part of the conduction system is in close proximity to the VT circuit. However, in this case the VT was successfully eliminated by ablating the Pd potential without impacting the normal conduction system.

Conclusion

We experienced 2 types of narrow QRS VTs in a patient with post-MI. Both types of VT were considered to be reentrant tachycardia involving the impaired Purkinje network in the circuit, and ablation targeting the diastolic Purkinje potentials successfully abolished the tachycardia.

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