Nonreentrant proximal fascicular ventricular tachycardia, with normal QRS duration and normal axis, originating from a region remote from the His bundle

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Introduction

Nonreentrant fascicular ventricular tachycardia (VT) is a rare type of idiopathic VT[1–3] that often presents with relatively narrow QRS and left (LBBB) or right (RBBB) bundle branch block patterns. Unlike reentrant fascicular VT, it is usually not responsive to verapamil; however, it is responsive to lidocaine and β-blocker treatment. Catheter ablation, targeting the earliest Purkinje potential, is often effective at eliminating the VT. In the present case, tachycardia with a normal QRS duration and a normal axis was proven to be nonreentrant fascicular VT. The VT was successfully suppressed using catheter ablation at a site remote from the His bundle.

Case report

A 48-year-old woman was referred to our hospital with uncontrolled tachycardia. An implantable cardioverter-defibrillator had been implanted 1 year earlier, at another institute, because of a complete atrioventricular (AV) block and a history of cardiac arrest due to ventricular fibrillation. Initial imaging (ultrasound cardiogram, coronary angiogram, cardiac magnetic resonance) and blood testing did not reveal any structural abnormalities, and positron-emission tomography findings were unremarkable. An endomyocardial biopsy was not performed. Tachycardia had been documented after implantable cardioverter-defibrillator implantation, and the tachycardia initiated and terminated incessantly and was accompanied with AV dissociation. The R-R interval was completely irregular, with cycle lengths of 384–560 ms (mean, 441 ms). The tachycardia QRS duration was 74 ms and the QRS axis was normal, without demonstrating bundle branch block patterns (VT1; Figure 1). The tachycardia could not be suppressed using bisoprolol, verapamil, or bepridil. Therefore, radiofrequency (RF) catheter ablation was performed under the guidance of an electroanatomical mapping system (CARTO3, Biosense-Webster, Diamond Bar, CA).

The multipolar electrode catheters were inserted through the femoral vein and were placed in the upper right atrium, His bundle region, and right ventricular apex. Initially, the baseline intracardiac electrocardiogram showed a complete A-H block during sinus rhythm; a His potential was not confirmed during the sinus rhythm. Ventriculoatrial conduction was never observed during ventricular pacing. During tachycardia, the catheter located in the His bundle region recorded atrial potentials (amplitude, 0.3 mV) from the right ventricle as well as His bundle potentials preceding the QRS onset by 34 ms (Figure 2A). The tachycardia initiated and terminated incessantly at baseline, with a cycle length of 480–592 ms (mean, 522 ms). The paced QRS morphology, from the right ventricle, was dissimilar to the patient's previously documented tachycardia. In addition, the tachycardia was not entrained.

Next, an ablation catheter (NAVISTAR, Biosense-Webster) was inserted into the left ventricle, via the femoral artery, and mapping was performed. At the left aspect of the basal ventricular septum (site A in Figure 3A), a presystolic Purkinje potential, preceding the onset of the surface QRS pattern by 40 ms, was recorded during tachycardia (Figure 2A); a fair pace map (score, 11/12) was obtained at this site. During pace mapping, the latency between the pacing and QRS complex was also 40 ms, corresponding to the interval between the Purkinje potential and QRS onset during the tachycardia. Given the presence of a complete AV block, we delivered RF energy to this site that was proximal to the His-Purkinje system. The application of RF energy to this site gradually changed the tachycardia QRS morphology from a narrow QRS pattern with a normal axis to an RBBB pattern with an inferior axis (VT2; Figures 1 and 2B). After the QRS

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KEY TEACHING POINTS

- Ventricular tachycardia can present with a normal QRS duration and a normal axis.
- Modification of related pathways can cause drastic QRS morphology changes.
- Detailed mapping is necessary to target the earliest Purkinje potentials, not only around the His bundle but also in other regions.
- Ablation of the proximal aspect of the His-Purkinje system is not always required.

morphology changed, the His bundle potential disappeared (Figure 2A); however, the tachycardia cycle length remained unchanged.

Repeated mapping revealed Purkinje potentials that preceded the QRS onset by 20 ms at the proximal area of the left anterior fascicle (site B in Figure 3A). RF energy application to this area changed the tachycardia axis from an inferior to superior axis (VT3; Figure 1). Repeated mappings also showed Purkinje potentials, preceding QRS onset by 18 ms, at the proximal area of the left posterior fascicle (site C in Figure 3A). RF energy application again altered the tachycardia QRS morphology, resulting in an RBBB pattern with a right axis deviation (VT4; Figure 1). However, the tachycardia cycle length remained unchanged, varying from 424 ms to 544 ms (mean, 495 ms).

Finally, extensive mapping showed a Purkinje potential at the basal anterior wall (Figure 3B) that preceded QRS onset by 68 ms (Figure 3C). This site (site D in Figure 3A) was located lateral to the site of the first RF ablation; RF energy application immediately terminated and suppressed the tachycardia. After this ablation, the VT became noninducible using either an isoproterenol infusion or ventricular burst pacing. During the 24-month follow-up period, VT has not recurred and the patient has not needed antiarrhythmic drugs.

Discussion

This case report describes a patient with nonreentrant, focal, Purkinje VT, with a very narrow QRS duration (74 ms) and a normal axis. The tachycardia was characterized as having the following features: (1) a narrow QRS and a normal QRS axis, (2) an irregular tachycardia cycle length, (3) AV dissociation, (4) spontaneous initiation and termination, and (5) changes in QRS morphology after RF application to the Purkinje fibers. Additionally, the tachycardia was eliminated following RF energy application at the earliest Purkinje potential recording site.

Differential diagnoses for a tachycardia with narrow QRS complex and ventriculoatrial dissociation include AV nodal reentrant tachycardia (AVNRT) with an exit block to the atrium, orthodromic reentrant tachycardia with a concealed nodoventricular or nodofascicular pathway as the retrograde limb, and junctional ectopic tachycardia (JET).[4] In this case, supraventricular tachycardia was less likely because of the complete AV block. However, supraventricular tachycardia is possible, though rare, in cases of AVNRT with bidirectional blocks of the upper common pathway; orthodromic reentrant tachycardia, with a concealed nodoventricular or nodofascicular pathway, which is accompanied with a bidirectional block at a site proximal to the level of emergence of these pathways; and JET with a bidirectional block at a site proximal to the origin. These diagnoses can be excluded by their

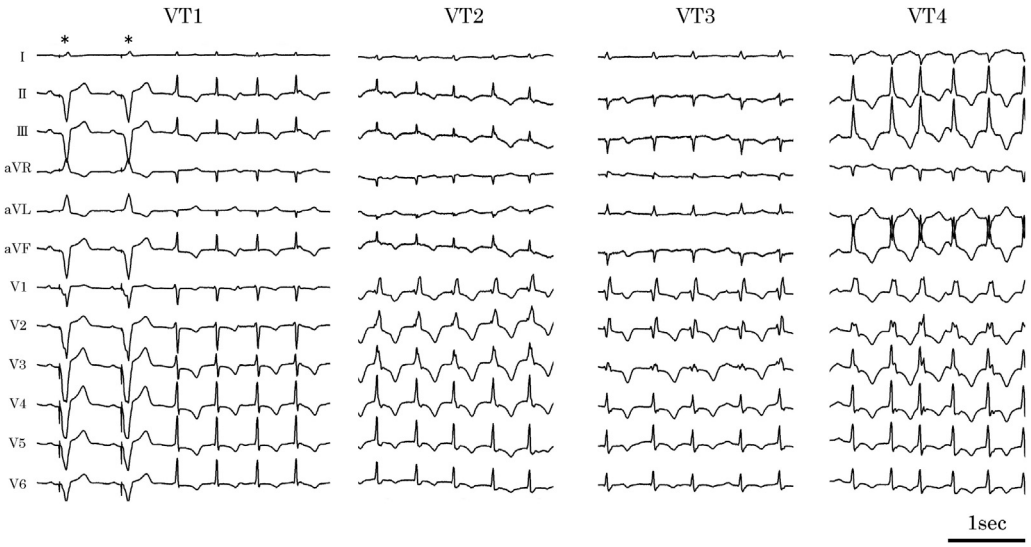

Figure 1 The morphologies of each ventricular tachycardia (VT; VT1–4). A clinical VT (VT1) with a normal QRS duration and a normal axis is observed at baseline. (* indicates right ventricular pacing.) VT2 with a right bundle branch block (RBBB) and an inferior axis is evident after radiofrequency (RF) energy application at site A (described in Figure 3). VT3 with an RBBB and a superior axis is observed after RF energy application at site B (described in Figure 3). VT4 with an RBBB and a right axis deviation is evident after RF energy application at site C (described in Figure 3).

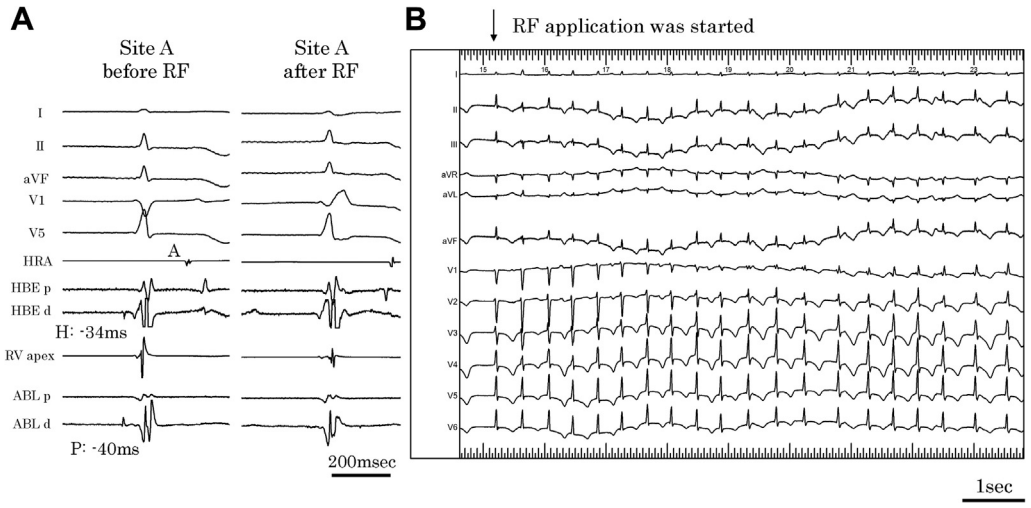

Figure 2 Radiofrequency (RF) energy application to the left aspect of the basal ventricular septum. **A:** The local potential in the left aspect of the basal intraventricular septum preceded the QRS onset by 40 ms. A His potential, preceding QRS onset by 34 ms, is evident at the His electrode. The His potential disappears just after the ablation at site A. **B:** The QRS morphology gradually changes into a right bundle branch block (RBBB) pattern during RF energy application at site A.

demonstrating longer HV intervals during sinus rhythm than during tachycardia. In the present case, the isolated junctional beat presented an HV interval of 34 ms, but the HV interval during tachycardia was 24 ms.

The administration of adenosine might help to confirm participation of the AV node in the tachycardia circuit. Further, the introduction of ventricular pacing during tachycardia when the His bundle was refractory might also contribute to distinguishing AVNRT from atrioventricular reciprocating tachycardia, nodoventricular reciprocating tachycardia, and nodofasicular reciprocating tachycardia; advancement of AV-dissociated tachycardia by His-synchronous ventricular extrastimuli indicates the presence of a nodoventricular or nodofascicular pathway. However, in this case, the completely irregular cycle length interfered with confirmation of the resetting phenomenon.

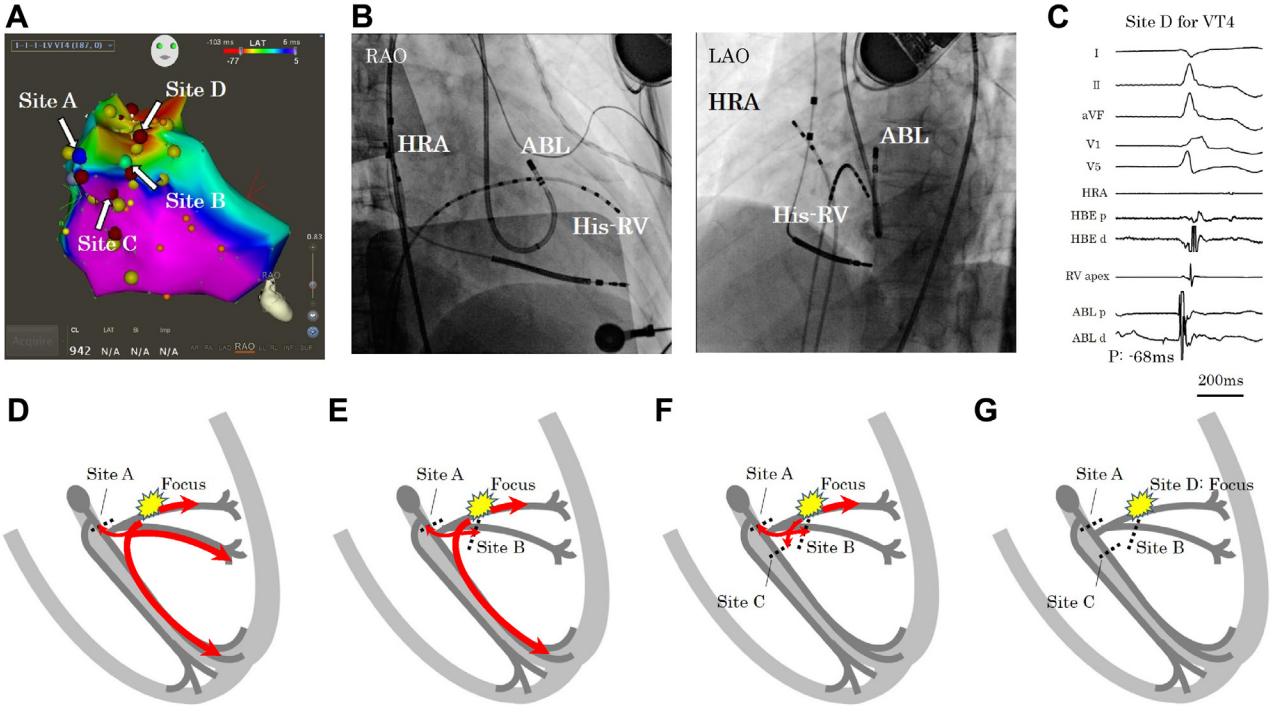

Figure 3 The site where the tachycardia was eliminated and a description of the assumed mechanism of the QRS morphology change. **A:** The CARTO image indicates the point of each ablation (sites A–D). **B:** (Site D) Fluoroscopy shows the ablation catheter at the basal anterior wall. **C:** A Purkinje potential at the basal anterior wall, preceding the QRS by 68 ms, is the earliest in the session. **D–G:** Assumed mechanism of QRS morphology changes, illustrating the assumed correlation between the ablation points and the tachycardia origin. ABL = ablation catheter; His-RV = catheter at the His bundle region and right ventricular apex; HRA = catheter at the high right atrium; LAO = left anterior oblique; RAO = right anterior oblique.

JET could be excluded for the following reasons. First, the Purkinje potential at the first ablation site was earlier than at the His bundle during tachycardia. Second, in this case, the QRS morphology gradually changed to an RBBB pattern, and the His bundle potential that preceded the QRS complex disappeared after RF energy application to the left aspect of the basal ventricular septum. In the case of JET, the QRS morphology might change into an LBBB pattern after diminishing the proximal part of the left bundle branch. If the ablation site was at the His bundle, proximal to the JET origin, the QRS complex would not change. In addition, if the ablation site was the His bundle, distal to the JET origin, the tachycardia might not be conducted to the ventricle because of a complete HV block. Third, RF energy application to a site remote from the His bundle successfully eliminated the tachycardia.

The mechanism of this VT was not precisely determined, but it was assumed to be nonreentrant, since the tachycardia initiated and terminated incessantly and spontaneously, had an irregular cycle length, was refractory to verapamil, and was not entrained. In this case, the QRS morphology of the VT gradually changed during RF energy application, while the cycle length remained unchanged. This phenomenon indicated that the VTs likely originated from a single site. In addition, the earliest Purkinje potential, preceding QRS onset by 68 ms, was recorded at the successful ablation site. Considering these observations, we believe that the tachycardia originated from the left anterior fascicle region. The mechanism of the marked change in QRS morphology was likely due to scission or modification of a related pathway, rather than originating from multiple sites (Figure 3D–G). However, the QRS morphology change from VT3 to VT4 might be due to an incomplete left anterior fascicular block or to partial conduction recovery after ablation of the left posterior fascicle. QRS morphology changes in reentrant fascicular VT, during or after ablation, have been previously reported.[5] Similarly, the QRS morphology of nonreentrant fascicular VT might also change during or after ablation of noncritical Purkinje fibers.[2]

We suppose that a conduction block, caused by ablation of related pathways, prolonged the conduction time from the origin to exit, ie, QRS onset. Because the earliest Purkinje potential was not initially recognized, the interval between the Purkinje potential origin and the QRS onset of the baseline VT remained indeterminable. Mapping was performed using a straight multipolar catheter, but a 20-pole, high-density mapping catheter would be helpful for determining the earliest Purkinje potential.

This case report underscores the significance of performing detailed mapping to target the earliest Purkinje potentials in cases of nonreentrant fascicular VTs with very narrow QRS durations, as ablation of the proximal aspect of the His-Purkinje system is not always required. As previously reported, the fascicular potential can be misidentified as an origin potential.[6] Determination of the earliest Purkinje potential is important, and it may be located in the left anterior or posterior fascicular areas, remote from the His bundle, even if the VT shows a normal QRS duration and a normal axis. Further, RF energy application, remote from the His bundle, may eliminate the VT.

The findings and outcomes of this case may help prevent AV blocks or complete LBBBs in cases requiring catheter ablation for nonreentrant fascicular VTs with normal QRS durations and normal axes.

Acknowledgments

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