Successful catheter ablation of focal ventricular tachycardia originating from right bundle branch without making right bundle branch block, using pharmacologic induction by landiolol

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Introduction

Right ventricular outflow tract tachycardia is one of the most common ventricular tachycardia (VT) observed in young patients, accounting for 60%–80% of all idiopathic VTs.[1] The QRS morphology of this tachycardia is left bundle branch block (LBBB) type, inferior axis, and wide QRS complex. In contrast, focal VT originating from the right bundle branch (RBB) is a rare idiopathic VT. Here we report the successful catheter ablation of focal VT originating from the RBB (RBB-VT), the QRS morphology of which is LBBB type, inferior axis, and narrow QRS complex.

Case report

A 6-year-old female patient was referred to our hospital after a school-based electrocardiogram (ECG) screening revealed VT at 120 beats per minute (bpm). The surface 12-lead ECG revealed a narrow QRS VT competing with sinus rhythm. Her heart rate was 100 bpm. The QRS morphology of sinus rhythm was normal (QRS width, 72 ms; QRS axis, 92°), whereas that of VT was LBBB type and inferior axis (QRS width, 92 ms; QRS axis, 58°). She neither presented with any symptoms nor showed any evidence of structural heart disease or tachycardia-induced cardiomyopathy. Notably, VT was suppressed by the exercise stress test. The 24-hour Holter ECG revealed minimum, maximum, and average heart rates of 85 bpm, 169 bpm, and 106 bpm, respectively, and VT at 55%. Owing to the relatively high frequency of VT, oral administration of bisoprolol (0.1 mg/kg/day) was initiated. In addition, the repeated 24-hour Holter ECG revealed minimum, maximum, and average heart rates of 110 bpm, 159 bpm, and 132 bpm, respectively, and VT at 99%. Although sinus

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rhythm was suppressed by bisoprolol, VT was accelerated. Thus, oral administration of bisoprolol was stopped and she was followed up without any medication. At the age of 8 years, when she visited an outpatient clinic for a follow-up, she demonstrated sustained VT at 150 bpm (Figure 1A), the QRS morphology of which was the same as described previously. A week later, electrophysiology study and catheter ablation were performed under general anesthesia.

At the beginning of the electrophysiology study, heart rhythm was VT competing with sinus rhythm at 100 bpm. We used the CARTO 3 electroanatomic mapping system (Biosense Webster Inc, Diamond Bar, CA) and placed 3 catheters as follows: a 5F decapolar electrode catheter each in the right atrium and the His position and a 7F decapolar mapping catheter in the right ventricle. We performed atrial pacing at 120 pulses per minute to identify the His bundle potential. After the atrial pacing was stopped, VT was suppressed and it disappeared. Heart rhythm was converted to sinus rhythm at 100 bpm, which suggested the automaticity of VT. Although we performed detailed program pacing of rapid atrial pacing, extra-atrial pacing, and rapid ventricular pacing, VT was not completely induced. Hence, we administered landiolol (a short-acting β-blocker), and VT reappeared. We initiated landiolol infusion at 3 μg/kg/min and increased it up to 6 μg/kg/min. Heart rhythm was converted to VT at 100 bpm.

During sinus rhythm, the His potential preceded the QRS onset and its sequence was from proximal to distal portion on the His catheter, and the H–V duration was 22 ms. In contrast, during VT, the His potential preceded the QRS onset and its sequence was from distal to proximal portion, and the H–V duration was 12 ms, which was shorter than that of sinus rhythm (Figure 1B). These findings and the QRS morphology suggested RBB-VT.

VT mapping using the CARTO 3 system revealed that the earliest ventricular excitation was widely spread around the mid-anterior portion of the right ventricle and was only as early as the QRS onset. Continued meticulous mapping revealed that small and high-frequency Purkinje potentials preceded the ventricular local excitation. The earliest Purkinje

potential, which preceded the His potential, was located at the anterior portion of the right ventricular septum and at the same level as that of the His potential. The earliest Purkinje–His duration was 17 ms.

During mapping of the mid-anterior portion of the right ventricular free wall, right bundle branch block (RBBB) appeared and the QRS morphology of VT changed to RBBB

type without change in the cycle length. After 80 seconds, RBBB disappeared and the QRS morphology returned to its original configuration.

We replaced the 7F decapolar mapping catheter with a 7F nonirrigated ablation catheter. The second application of radiofrequency current (limit 55°C, 25 W), which targeted the earliest Purkinje potential, induced rapid firing of VT with or without RBBB; this current was stopped when rapid firing of VT with RBBB was sustained (Figure 2). The application duration was 17 seconds. Following ablation, heart rhythm was sinus rhythm without RBBB and VT was no longer inducible. No recurrences or late complications were observed over a 16-month follow-up period.

Parental consent was provided for this case report to be published.

Discussion

RBB is a part of the cardiac conduction system, which inferiorly continues as if it were a continuation of the His bundle, traveling along the right side of the muscular interventricular septum. It proximally runs just deep to the endocardium and slightly inferior to the medial papillary muscle of the tricuspid valve before dividing into fibers that spread
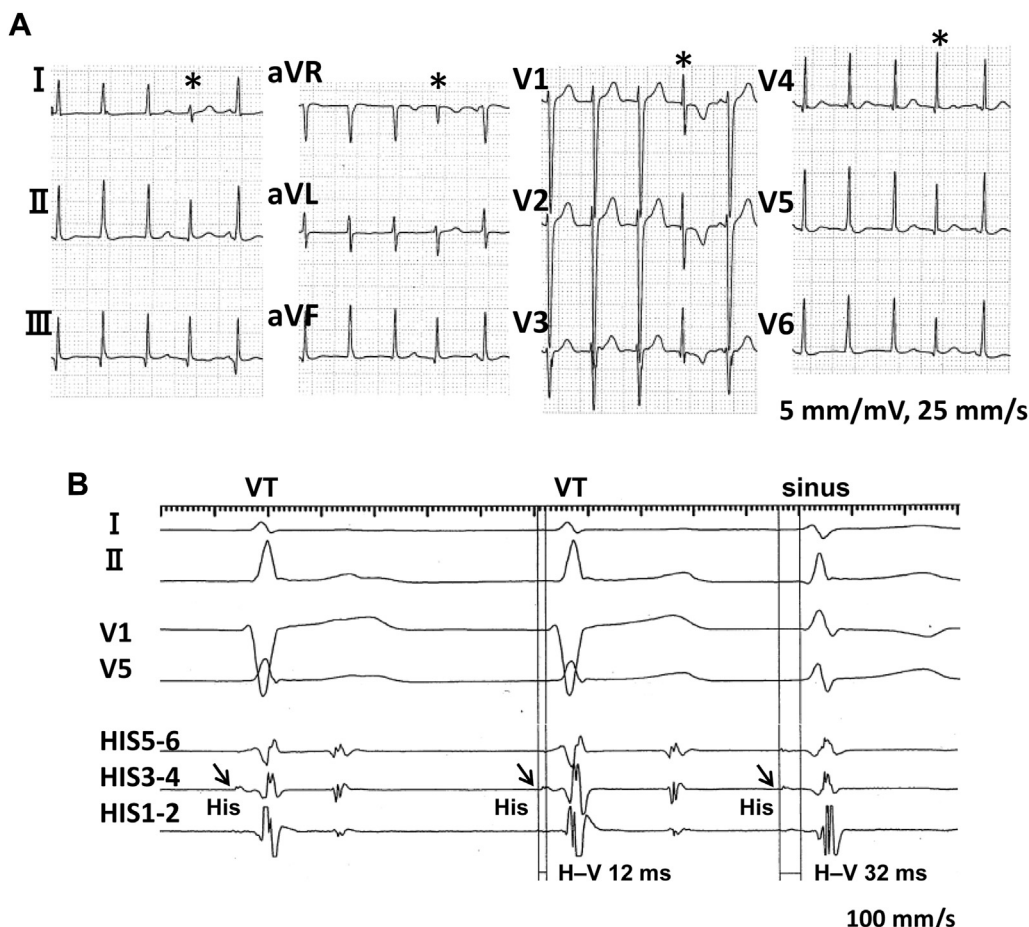

Figure 1 **A:** The surface 12-lead electrocardiogram revealed sustained ventricular tachycardia (VT) occasionally with sinus capture (*). VT rate was 150 beats/min. **B:** The first and second beat was VT beat and the third beat was sinus capture. During VT, the His potential preceded the QRS onset, and the sequence of the His potential was inverted from sinus capture. During VT, the H–V duration was shorter than that of sinus capture.
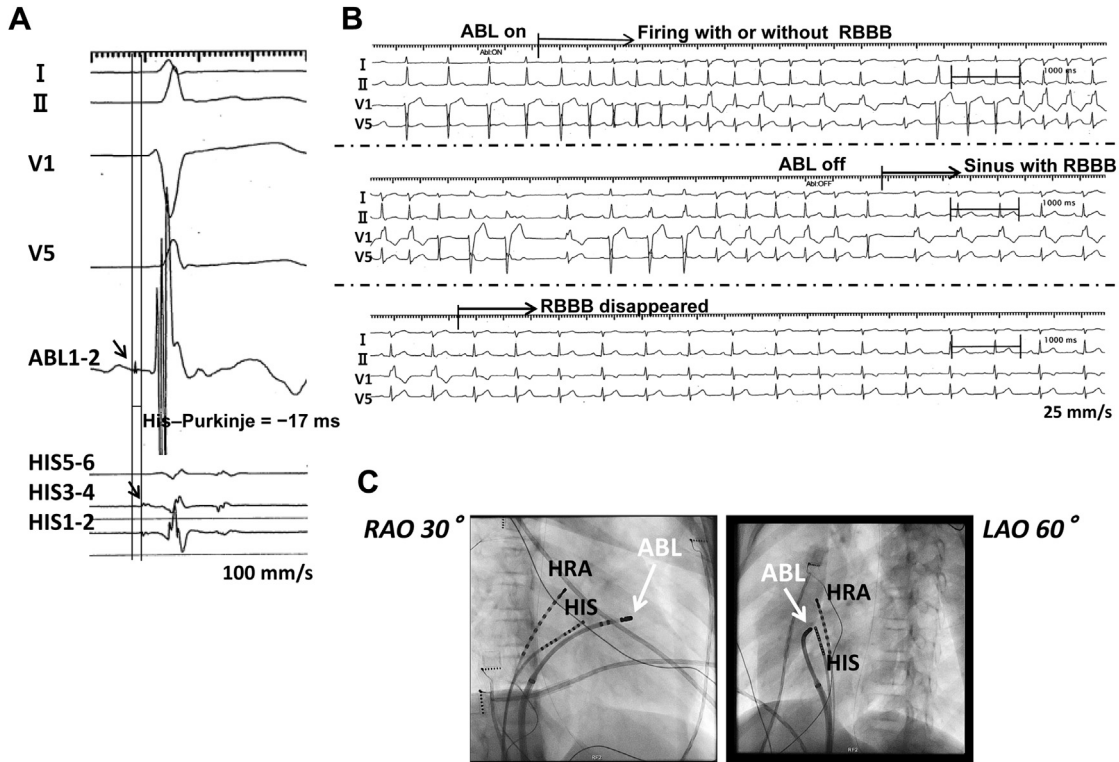
Figure 2 **A:** The sharp Purkinje potential of the successful ablation site (ABL1-2) preceded the His potential by 17 ms. **B:** The surface lead electrocardiogram during radiofrequency current revealed rapid firing of ventricular tachycardia with or without right bundle branch block (RBBB). Several seconds after radiofrequency current was stopped, heart rhythm was converted to sinus rhythm without RBBB. **C:** Fluoroscopy revealed successful ablation site targeting the earliest Purkinje potential located at the anterior portion of the right ventricular septum and at the same level as that of the His potential.

throughout the right ventricle.[2] Few studies have reported catheter ablation for RBB-VT.[3–7] In these cases, although the success rates were high, all cases were accompanied by the morbidity of RBBB. To the best of our knowledge, this is the first report of successful catheter ablation of RBB-VT without RBBB. We suggest that RBB-VT got cured without RBBB because low-energy radiofrequency current, the

power of which was limited to <25 W, fortunately eliminated only the RBB tissue with automaticity and spared the normal RBB tissue. The fact that the Purkinje potential still remained at the ablation site during sinus rhythm after ablation implied that the origin of the VT was not the side branch of the RBB but the RBB itself. Cryoablation may be useful for ablation of RBB-VT. We performed radiofrequency
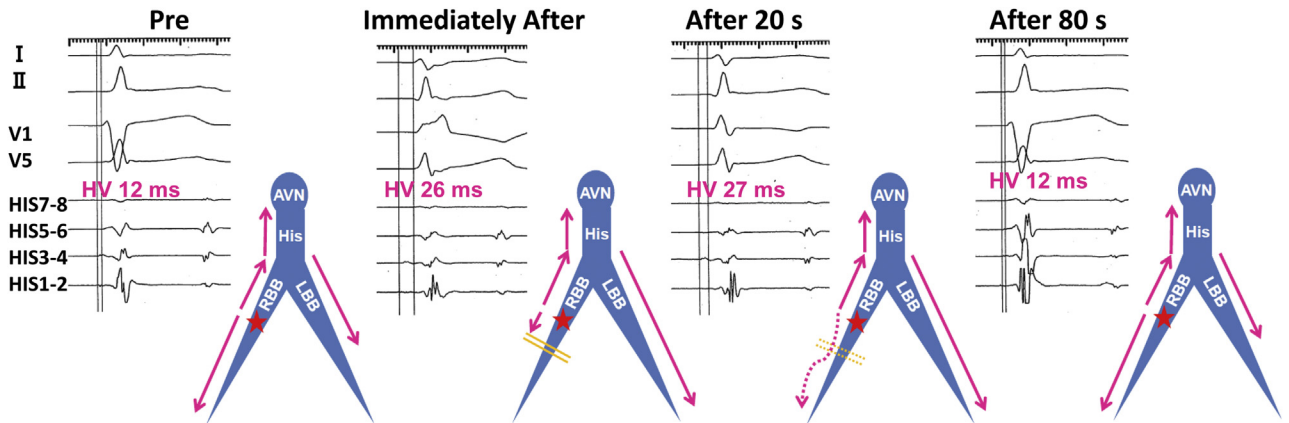

Figure 3 The electrocardiograms and schemas of the His–Purkinje system during ventricular tachycardia (VT) are shown. Immediately after right bundle branch block (RBBB) appeared during sinus rhythm, the QRS morphology of VT changed to RBBB type. After 20 seconds, when RBBB partially improved during sinus rhythm, the QRS morphology of VT changed to that of the sinus rhythm at baseline. After 80 seconds, when RBBB disappeared during sinus rhythm, the QRS morphology of VT returned to the original left bundle branch block configuration. Star, origin of VT; doublet, wounded portion of RBB; arrow, electrical conduction on the His–Purkinje system; dotted arrow, slow electrical conduction. AVN = atrioventricular node; His = His bundle; LBB = left bundle branch; RBB = right bundle branch.

ablation in this case because cryoablation was not yet permitted for use in Japan at that time.

During ablation of VT, if arrhythmia does not automatically appear, then pharmacologic or electrophysiological induction may be attempted. The choices of medications inducing VT include α-adrenergic receptor agonists (eg, phenylephrine), β-adrenergic receptor agonists (eg, isoproterenol), and acetylcholinesterase inhibitors (eg, edrophonium chloride). In addition, RBB-VT is generally suppressed by β-blockers.[3] However, in our case, the β-blocker suppressed sinus rhythm and accelerated VT both in the outpatient clinic and during the electrophysiological study, which was a rare phenomenon. To date, there has been only 1 case report on the induction of ventricular arrhythmia using β-blocker. A 7-year-old female patient who suffered from supraventricular tachycardia twice was orally administered propranolol for prophylaxis. A routine 24-hour ECG monitoring revealed an accelerated idioventricular rhythm mainly at night. After propranolol administration was stopped, a follow-up with repeated 24-hour ECG monitoring revealed the disappearance of accelerated idioventricular rhythm.[8]

There are mainly 3 differential diagnoses of RBB-VT, namely the junctional rhythm with LBBB, the bundle branch reentry with LBBB morphology, and the Mahaim fiber automaticity.[3] The junctional rhythm with LBBB usually has a normal H–V duration and His potential followed by RBB potential. The bundle branch reentry with RBBB has a prolonged H–V duration and His potential followed by RBB potential. In the Mahaim fiber automaticity, the earliest Mahaim potential is usually placed on the tricuspid annulus, and rapid atrial pacing reveals atriofascicular accessory pathway with decremental properties. However, these characteristics were not observed in our case.

When RBBB caused by catheter trauma appeared during VT mapping, we observed an interesting change in the surface 12-lead ECG, which can be explained by the schema of the His–Purkinje system (Figure 3). When RBBB appeared during sinus rhythm, the QRS morphology of VT changed to RBBB type, which suggested that the wounded portion of RBB was situated at the distal portion of the VT origin. At this time, VT excitation was retrogradely conducted on the proximal RBB and then antegradely on the LBB. After 20 seconds, when RBBB partially improved during sinus rhythm, the QRS morphology of VT was similar to that of sinus rhythm at baseline, which suggested that antegrade slow conduction on distal RBB and retrograde conduction on proximal RBB followed by antegrade conduction on the LBB reach the ventricle at the same time as sinus rhythm. After 80 seconds, when RBBB disappeared during sinus rhythm, the QRS morphology of VT returned to the original LBBB configuration.

Conclusion

We report a case of successful ablation of RBB-VT in an 8-year-old female patient. Landiolol infusion induced arrhythmia, and the application of low-energy radiofrequency current for the earliest Purkinje potential eliminated arrhythmia without making RBBB.

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