

Unique Ventricular Tachycardia Originating from the Right Bundle Branch

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Ventricular tachycardia (VT), a common arrhythmia, frequently originates from in the right ventricular outflow tract, left ventricular outflow tract, aortic sinus, and left ventricular papillary muscle but infrequently from the His-Purkinje system, whereas the VT stemming from the right bundle branch has rarely been reported. Here we reported a case with of VT originating from the right bundle branch which was subsequently successfully treated with radiofrequency ablation and demonstrated the electrocardiac features of VT using an electrophysiological examination.

Key Words: Catheter ablation • Electrophysiology • Ventricular tachycardia

CASE REPORT

A 23-year-old man was admitted with a 1-year history of paroxysmal palpitations and 2 hours of exacerbation. Echocardiography revealed no structural heart disease, with a left ventricular ejection fraction of 61% and left ventricular end-diastolic diameter of 43.3 mm. Twelve-lead electrocardiography (ECG) performed at admission indicated ventricular tachycardia (VT), deep “S” waves in the QRS complexes of lead V1, and a QRS duration of 108 ms (Figure 1A). Following the intravenous administration of amiodarone, the 12-lead ECG showed sinus rhythm (SR) with a PR interval of 160 ms, QRS duration of 104 ms, and an undeviating electrical axis (Figure 1B).

The patient experienced frequent recurrence of tachycardia despite amiodarone treatment which caused him discomfort, and therefore an electrophysiological study was performed. Written informed consent was

obtained before the electrophysiological study. Diagnostic catheters were positioned in the coronary sinus (CS) and on his bundle (His). A 3.5F steerable saline-irrigated electrode catheter (NaviStar ThermoCool, Biosense Webster, Inc., Diamond Bar, CA, USA) was used for mapping and ablation. Subsequent ECG showed similar tachycardia to the admission ECG (Figure 1C). During tachycardia, the intracardiac electrograms of the proximal CS showed an A-A interval of 734 ms and V-V interval of 624 ms (Figure 2A). His potential (H) was consistently recorded on His catheter and preceded the surface QRS and local ventricular electrogram. In addition, a stable and consistently sharp potential preceding the local ventricular electrogram and surface QRS was detected on the right ventricular septum along the right bundle branch (RBB). Mapping of right ventricle (RV) and right bundle potential (RBP) was obtained using a three-dimensional electroanatomic mapping system (CARTO3, Biosense Webster Inc., Diamond Bar, CA, USA) during tachycardia (Figure 2D). The earliest RBP was recorded at the proximal end of the RBB. At this site, during tachycardia, the H-QRS interval was 9 ms, H-V (local ventricular electrogram) interval was 35 ms, and RBP preceded the surface QRS by 29 ms, His by 20 ms and local ventricular electrogram by 47 ms. In addition, during sinus capture beats, the H-QRS/V interval was 48 ms, and the RBP was later than the His by 20 ms and preceded

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the local ventricular electrogram by 30 ms (Figure 2C). Radiofrequency (RF) energy was delivered at the earliest RBP site during tachycardia with 15 W at 43 °C and water flow velocity of 17 ml per minute. Tachycardia was terminated within 5 seconds, and became a SR with a RBB block (RBBB) pattern in QRS morphology and axis (Figure 2B, 1D). No tachycardia was induced with programmed stimulation, and no palpitations were reported in a follow-up telephone call 2 years later.

DISCUSSION

We report a case of VT originating from the RBB that was successfully treated with RF ablation, and de-

monstrated the electrophysiological features. We consider that the VT in this case was caused by an accelerated idioventricular rhythm originating from the RBB (RBB-AIVR).

In this case, during tachycardia, activation signals from the RBB site of origin not only involved antero-grade propagation to excite the RV but also retrograde propagation to excite His and then left bundle branch (LBB), which in turn induced antero-grade propagation to excite the left ventricle. As a result, RBP was ahead of His by about 20 ms and surface QRS by 29 ms, so that His was ahead of QRS by 9 ms and V by 35 ms (Figure 2C). During the sinus capture beat, the antero-grade propagation of activation signals through His to LBB and RBB excited the left ventricle and the RV, respectively.

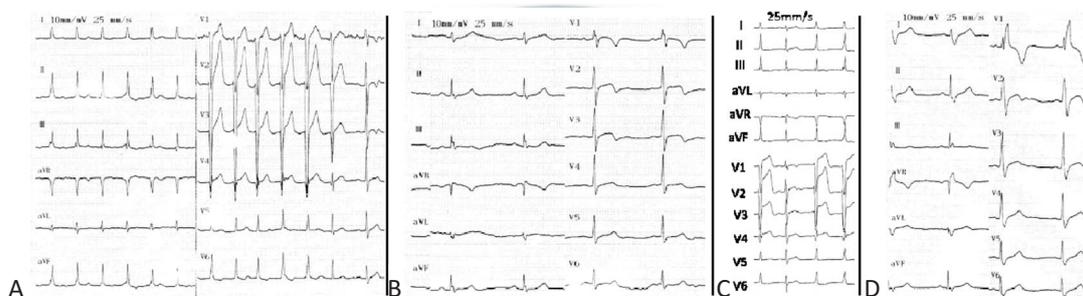


Figure 1. Twelve-lead electrocardiography at admission (A), after the administration of amiodarone (B), during electrophysiological studies (C), and after ablation (D).

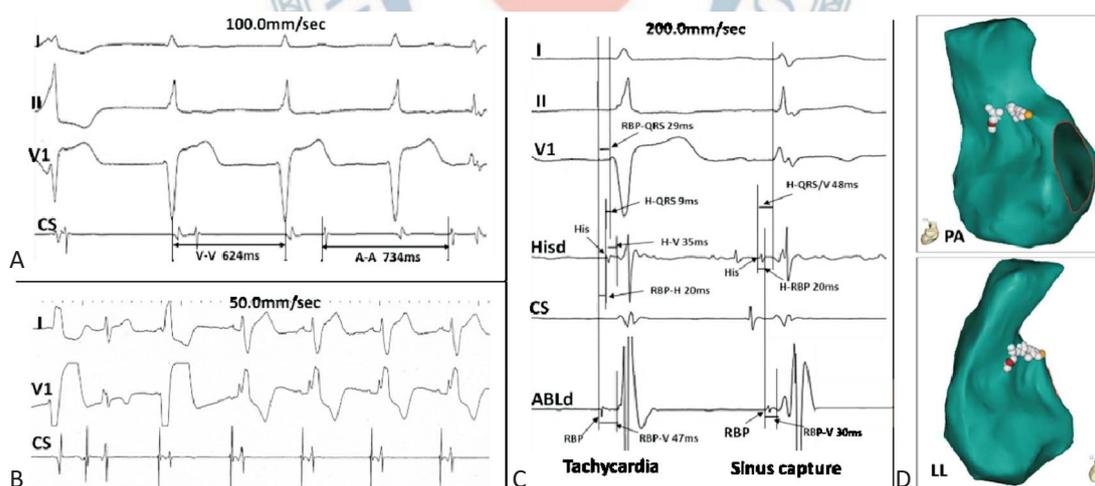


Figure 2. Surface ECG and intracardiac electrograms during tachycardia (A, C) and ablation (B). (A) During tachycardia: V-V = 624 ms, A-A = 734 ms. (B) Tachycardia was terminated and became a sinus rhythm with RBBB during ablation. (C) ABL was positioned at the point where the earliest RBP was recorded. During tachycardia: RBP-H = 20 ms, RBP-V = 47 ms, RBP-QRS = 29 ms, H-QRS = 9 ms, H-V = 35 ms. During sinus capture beat: RBP-V = 30 ms, H-RBP = 20 ms, H-QRS/V = 48 ms. (D) Anatomical map of RV in PA and LL. Yellow dot: point of His; gray dot: point of RBP during tachycardia; red dot: point of the earliest RBP during tachycardia where RF ablation was performed. A, atrial potential; ABL, ablation catheter; CS, coronary sinus; ECG, electrocardiography; H, His potential; His, his bundle; LL, left lateral view; PA, postero-anterior projection; RBP, right bundle potential; V, local ventricle potential.

Subsequently, RBP and His were reversed, and His was ahead of RBP by about 20 ms and QRS/V by 48 ms (Figure 2C). As Durrer et al. reported previously,² interventricular septum activation starts on the left septal surface and proceeds from left to right in an apical-basal direction. The RBP-V interval during tachycardia in our case was 47 ms (Figure 2C), which represented the activation signal conduction time from the RBB site of origin through RBB–His–LBB–left side interventricular septum to the local cardiomyocytes near the RBB site of origin in the right side interventricular septum. The RBP-V interval during the sinus capture beat was 30 ms (Figure 2C), which showed that the local cardiomyocytes near the RBB site of origin in the right side interventricular septum were excited 30 ms later than at the RBB site of origin. The conduction time from His through LBB to the left side interventricular septum and then to the right side interventricular septum near the RBB site of origin was longer than from His directly to the RBB site of origin.

Chen et al. reported eight cases with RBB-AIVR with identical LBB block (LBBB) QRS morphology,³ and the electrophysiological characteristics and ECG features of our case were similar. In addition, Chen et al. reported that persistent RBB-AIVR could be slowed but not terminated by the intravenous administration of amiodarone, propafenone, and metoprolol. However, in our case, VT was terminated by intravenous amiodarone. Because the patient refused long-term amiodarone therapy, and

as he frequently experienced recurrent tachycardia which caused him discomfort, RF ablation therapy was performed. Unfortunately ablation caused permanent complete RBBB.

RBB-AIVR is an uncommon ectopic rhythm. Besides Chen et al.'s eight cases,³ other reports have mainly been case reports.^{4,5} Our case had AIVR originating from the RBB, and we tried to elucidate the relevant electrophysiological mechanism. However, further studies are needed to clarify the mechanism of RBB-AIVR.

REFERENCES

1. Szydło K, Wnuk-Wojnar AM, Trusz-Gluza M, et al. Differentiation of arrhythmia originating from the right or left ventricular outflow tract based on the QRS morphology of premature ventricular beats and duration of repolarisation. *Kardiol Pol* 2013;71:723-9.
2. Durrer D, van Dam RT, Freud GE, et al. Total excitation of the isolated human heart. *Circulation* 1970;41:899-912.
3. Chen ML, Gu K, Yang B, et al. Idiopathic accelerated idioventricular rhythm or ventricular tachycardia originating from the right bundle branch. *Circ Arrhythm Electrophysiol* 2014;7:1159-67.
4. Sternick EB, Gerken LM, Vrandečić M. Appraisal of "Mahaim" automatic tachycardia. *J Cardiovasc Electrophysiol* 2002;13:244-9.
5. Lau EW, Marshall HJ, Griffith MJ. Fascicular tachycardia and parasympole of right bundle branch origin. *Heart Rhythm* 2005;2:994-6.