Bundle Branch Reentrant Ventricular Tachycardia With Wide and Narrow QRS Morphology

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Bundle branch reentrant ventricular tachycardia (BBR-VT) is generally recognized as VT with wide QRS complexes that includes the His-Purkinje system and ventricle in its circuit. BBR-VT is well known to be commonly associated with structural heart diseases and left ventricular dysfunction. We report a rare case of BBR-VT displaying wide and narrow type QRS complexes in a patient without obvious structural heart disease or overt ventricular conduction disturbance.

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Case Presentation
A 39-year-old man with episodes of wide QRS tachycardia was referred to our institution for electrophysiological study and catheter ablation. ECG during his clinical tachycardia showed left axis deviation and left bundle branch block (LBBB)–type morphology. Baseline 12-lead ECG showed sinus rhythm with no remarkable abnormalities, except for slight right axis deviation (Figure 1A). Transthoracic echocardiography was also normal, with normal values for left ventricular size (diastolic diameter, 46 mm) and systolic function (ejection fraction, 62%). There was nothing suggestive in the patient’s medical history, such as syncope, nor was there any family history of sudden cardiac death.

Diagnostic electrophysiological study demonstrated a normal A-H interval of 78 ms and an H-V interval of 48 ms during sinus rhythm. Programmed ventricular stimulation with single extrastimulus delivered from right ventricular apex revealed decremental ventriculoatrial conduction as S1S2 interval shortened, and V3 phenomenon was observed by an extra single atrial ectopic beat during VT1. This narrow QRS tachycardia was considered to be BBR-VT because of the same electrophysiological features (1) ≈ (2) as previously described about VT1. VT2 was repeatedly converted to right bundle branch block (RBBB)–type tachycardia (VT3; Figure 1D) without changes in tachycardia cycle length, H-V interval, and activation sequence as VT2 (Figure 3A). VT3 was sustained for several minutes and terminated by terminating ventricular extra beats.

Intracardiac electrograms of all 3 VTs (Figures 2 and 3) demonstrated fractionation of His electrogram preceding ventricular activation. The H-V interval (H1-VH interval described in Figure 3B and 3C) was 68 ms during VT2 and VT3, which was longer than the H-V interval of 48 ms during sinus rhythm.

Radiofrequency (RF) energy was delivered at the anteroseptal area of the right ventricle where right bundle branch (RBB) potential was recorded prominently during sinus rhythm. Ablation resulted in RBBB 5 s after RF without prolongation of H-V interval, and any tachycardia or V3 phenomenon became noninducible by atrial and ventricular stimulation with or without using isoproterenol infusion. During follow-up for 1 year, RBBB persisted, and the patient is free from tachyarrhythmia without antiarrhythmic drugs.

Discussion
As has already been reported previously, BBR-VT is usually seen in the patients with severe left ventricular dysfunction based on underlying structural heart diseases such as dilated cardiomyopathy, ischemic cardiomyopathy, or valvular heart disease. Blanck et al1 reported that 45 of 48 patients with BBR-VT showed structural heart disease and prolonged H-V interval of >60 ms at baseline. They also presented in another report that H-V intervals were prolonged in the 3 patients with BBR-VT without overt structural heart disorder.1 Presentation of BBR-VT in this case is an unusual one because...
of normal structural heart and normal H-V interval of 48 ms during sinus rhythm.

Induction of preceding V3 phenomenon and subsequent VT1 with LBBB-type morphology necessitates a unidirectional retrograde RBB conduction block, slow conduction via retrograde LBB, and recovery of excitability of RBB. These conditions induced reentrant circuit of VT1 with retrograde LBB and antegrade RBB conduction (circuit 1). As a result, an H-V interval of 80 ms (described as H1-VH interval in Figure 2) during VT1 longer than that of sinus rhythm was considered to reflect antegrade RBB conduction. Induction of VT1 was facilitated by the existence of concealed conduction disturbance of antegrade RBB. However, an H-V interval of 48 ms during sinus rhythm must be determined by nearly intact antegrade LBB conduction. This intact LBB conduction property prevented prolongation of H-V interval after RF application at RBB.

VT2 expressed narrow QRS width and QRS morphology similar to that of sinus rhythm. However, VT2 was nothing but BBR-VT incorporating both bundle branches and the His bundle because of the same electrophysiological properties as VT1.4 It was considered that the reentrant circuit of BBR-VT

Figure 1. Twelve-lead ECG of sinus rhythm and ventricular tachycardia (VT). A, Sinus rhythm showed slight right axis deviation. B, VT1 showed left bundle branch block pattern. C, VT2 had narrow QRS complexes and similar morphology to that of sinus rhythm. D, VT3 with right bundle branch block morphology was observed after spontaneous conversion from VT2.

Figure 2. VT1 was induced by programmed ventricular stimulation with a single extrastimulus at an S1S2 interval of 240 ms after a V3 beat. Fractionation of the His electrogram (solid arrows) was observed similar to that during VT2 and VT3, which is demonstrated in Figure 3. H1 represents the initiating point of His amplitude, and Vh and Vr indicate the initiating point of ventricular activation on the HBE and RVA recordings, respectively. Ventriculoatrial activation exhibited Wenckebach conduction property. HBed indicates distal His-bundle electrogram; HBEp, proximal His-bundle electrogram; HRA, high right atrial electrogram; RVA, right ventricular apex electrogram; and VT, ventricular tachycardia.
consisting of retrograde and antegrade bundle branch conduction coexisted with antegrade conduction of both bundle branches during VT2 with narrow QRS complexes. In addition, the fact that antegrade RBBB was manifested but had no other effect on electrophysiological properties of tachycardia when VT2 was converted to VT3 demonstrated that antegrade RBBB conduction was the bystander during VT2 and VT3. Finally, the proposed circuit of VT2 and VT3 included RBB and LBB as retrograde and antegrade limbs, respectively (circuit 2).

Other possible reentrant circuits of VT2 included antegrade activation of the RBB and at least a portion of the LBB and retrograde conduction through a portion of the left posterior fascicle or a fascicle connecting to the proximal right bundle. This is similar to the multiform fascicular VTs involving the left bundle system reported by Sung et al.\(^5\) This explanation satisfies the observation of the bystander antegrade RBBB conduction during VT2 and VT3. Abolition of all the VTs by RF application for the right bundle supports a retrograde limb that connected to the proximal right bundle or His bundle. However, interfascicular reentrant tachycardia involving only fascicles of the left bundle would not be abolished by ablation of the right bundle alone.

Figure 3. Intracardiac electrograms of VT2 and VT3. A, VT2 was spontaneously converted to right bundle branch block-type tachycardia (VT3, asterisk), retaining the same tachycardia cycle length, V-H interval, and activation sequence as VT2. Fractionation of His electrogram as demonstrated in B and C appeared before and after the beats (not shown). B and C, Tracings of VT2 (B) and VT3 (C) in which fractionation of His electrogram was prominently recorded, respectively. H2 represents the terminal point of His amplitude. As described in the text, VT2 and VT3 had the same electrophysiological circuit. Also note that V\(_{RV}\) preceded in VT1 but delayed in VT2 and VT3 compared with VH. HBEd indicates distal His-bundle electrogram; HBEp, proximal His-bundle electrogram; HRA, high right atrial electrogram; RVA, right ventricular apex electrogram; and VT, ventricular tachycardia.
In this case, longitudinal dissociation of His-RBB system seems to be the rational explanation of those electrophysiological findings. Narula reported a concept of asynchronous conduction or longitudinal dissociation of the His bundle based on the observations that His-bundle stimulation at the distal site for the patients with LBBB resulted in eliminating LBBB and making QRS complexes narrow. Our hypothetical mechanisms on VT2 exhibiting narrow QRS complexes can clearly explain the appearance of narrow QRS morphology.

To be remarkable, fractionation or asynchronous conduction of His electrogram preceding ventricular activation on the His-bundle electrogram recording catheter was observed as demonstrated in Figures 2, 3B, and 3C. These findings indicate the existence of localized conduction disturbance and longitudinal dissociation within the His bundle. Conceivable conduction properties during VTs are shown in Figure 4. RBB conduction system was divided into RBB1 and RBB2 fascicles in this figure. As shown in this figure, the conduction over the longitudinally dissociated His-RBB2 system is the bystander of the reentrant circuit of BBR-VT (described as circuit 1 or circuit 2 in Figure 4). This antegrade His-RBB2 conduction via the longitudinally dissociated His-RBB system might play a role in producing narrow QRS complex as shown in Figure 4B; otherwise, it does not participate in determining QRS morphology.

In addition, VT3 was also inducible by atrial burst pacing in this case. Simons et al. reported a case of BBR-VT with LBBB-type QRS morphology only by atrial inputs. There are several other reports describing atrial-induced BBR-VT. In our case, BBR-VT was induced both by atrial and by ventricular pacing. Iesaka et al. reported the similar phenomenon as atrial-induced ventricular ectopic beats in 1983. In this case report, repetitive ventricular beats preceding fractionation of His electrogram were reproducibly induced by both atrial extrastimuli and ventricular stimulation without apparent retrograde atrial activation. They also pointed out longitudinal dissociation within the proximal His-Purkinje system as the explanation of these electrophysiological findings. Based on their hypothesis of longitudinal dissociation of the His-RBB system and asynchronous conduction of the 2 dissociated components, atrial-induced VT3 seems to be explained rationally.

Atrioventricular (A-V) nodal reentrant tachycardia with retrograde Wenckebach conduction in an upper final common pathway and a His-bundle focal tachycardia were alternative explanations that we considered. In both cases, the His bundle would precede each QRS, and H-H oscillations would precede V-V oscillations. Furthermore, the H-V interval could be expected to be similar in narrow QRS and RBBB tachycardia. Absence of discontinuous conduction via the A-V node does not completely exclude the possibility of multiple pathways via the A-V node, and the prolongation of H-V interval during VT2 and VT3 may be caused by the decremental conduction property via the A-V node. However, right bundle ablation could eliminate neither A-V nodal reentrant tachycardia nor a His-bundle focal tachycardia because the originating substrate of these 2 tachycardias should be located at the site above the right bundle. In our case, the right bundle potential was prominently recorded at the successful ablation site, and RF applications at the site resulted in RBBB and eliminated all 3 tachycardias. We carefully ruled out potentially alternative explanations based on the above observations.

A concealed nodoventricular connection in the retrograde direction should be also ruled out. As for this, we thought that if that was the case, (1) H-V interval would not be longer than that during sinus rhythm, and (2) tachycardia cycle length would not show significant differences in 3 types among LBBB, narrow QRS, and RBBB type tachycardia. In our case, tachycardia cycle length was 240 to 260 ms in VT2 and VT3, which was shorter than that in VT1 of 270 to 300 ms.

The electrophysiological mechanism of this case can be summarized as follows. Three types of VTs might be because of BBR-VT based on functional longitudinal dissociation of the His-RBB system associated with latent conduction disturbance within the His bundle, and RF application for right bundle and complete abolition of RBB conduction eliminated all the VTs. The teaching points from our case are as follows: (1) This is the first case report of BBR-VT explainable by bundle branch reentry caused by functional longitudinal dissociation of the His-RBB system. Wide and narrow QRS morphology of BBR-VT can be both rationally explainable using the hypothesis of the longitudinally dissociated His-RBB system and the His-LBB system. (2) The existence of narrow QRS BBR-VT itself and the transition of tachycardia QRS morphology from narrow QRS to RBBB with unchanged fractionated His electrogram and H-V interval imply the bystander nature of the longitudinally dissociated His-RBB system.
Disclosures

None.

References


**KEY WORDS:** bundle-branch reentry; catheter ablation; concealed conduction disturbance; longitudinal dissociation of the His-Purkinje system
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