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Bundle branch reentrant ventricular tachycardia after transcatheter aortic valve replacement

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Introduction

Bundle branch reentrant ventricular tachycardia (BBR-VT) is a well-described entity that accounts for 6% of inducible sustained monomorphic VT during electrophysiology studies.1 BBR-VT is a macroreentrant VT involving the His-Purkinje system and usually requires some form of conduction delay in the His-Purkinje axis.2 BBR-VT is commonly seen in patients with nonischemic dilated cardiomyopathy, myotonic dystrophy,3 hypertrophic cardiomyopathy, and valvular heart disease. Rare case reports have been published involving patients after corrective surgical aortic valve and mitral valve replacement without a clear cause–effect relationship.2 Transcatheter aortic valve replacement (TAVR) procedure is now a well-established percutaneous procedure for patients with symptomatic severe aortic stenosis who are deemed at high risk for a traditional surgical aortic valve replacement. Conduction abnormalities post TAVR have been well described, with high incidence of permanent pacemaker implantation.4 TAVR procedure requiring valvuloplasty and stented valve is likely to affect the conduction system, possibly creating the right milieu for bundle branch reentry. We describe the first case of proven BBR-VT in a patient after TAVR, and its management with catheter ablation.

Case report

A 75-year-old man with hypertension, obesity, insulin-dependent diabetes mellitus, paroxysmal atrial fibrillation, coronary artery disease status post coronary artery bypass graft surgery (26 years ago), and severe aortic stenosis was referred to our institution for TAVR. He was deemed to be at moderate risk for surgical valve replacement (Society of Thoracic Surgeons mortality estimate: 4.3%). His ejection fraction was 50% on an echocardiogram, and a cardiac catheterization prior to valve replacement showed stable coronary artery disease with patent grafts along with a mean transaortic valve gradient of 48 mm Hg. He underwent successful implantation of a 29-mm Edward-Sapien XT aortic prosthesis via the transfemoral approach. His electrocardiogram (ECG) prior to valve replacement showed sinus rhythm with first-degree atrioventricular (AV) block and QRS duration of 120 ms (Figure 1A). A QR pattern in lead V1 was noted along with left axis deviation. An ECG a day after TAVR showed sinus rhythm with a long first-degree AV block and left bundle branch block (LBBB) (Figure 1B). He presented 2 weeks later with palpitations, dizziness, and recurrent presyncope episodes when he was noted to have incessant wide complex left bundle branch (LBB) tachycardia at 200 beats per minute (Figure 1C). QRS morphology of the tachycardia resembled pre-existing LBBB morphology with late precordial R/S transition and QRS axis of 29 degrees. Given highly symptomatic status during the tachycardia, he received multiple synchronized cardioversions and was started on amiodarone infusion. He was taken to the electrophysiology laboratory in fasting state and standard intracardiac catheters were introduced. A long HV interval of 88 ms (Figure 1D) was recorded from the His bundle catheter placed across the tricuspid valve, and 1:1 AV conduction was documented to 620 ms. No ventriculoatrial conduction was noted at baseline or during isoproterenol infusion. Programmed electrical stimulation from the right ventricle (RV) apex using triple extrastimuli was performed, which reproducibly induced ventricular fibrillation requiring external defibrillation. No clinical tachycardia was induced at this study session. Given the presence of coronary artery disease, mildly reduced ventricular function, underlying conduction abnormality, and induction of ventricular fibrillation, a dual-chamber internal cardioverter-defibrillator (ICD) was implanted.

The day after the ICD implant, the patient developed multiple episodes of wide complex tachycardia associated with AV dissociation, some of them pace terminated and...
some requiring ICD shocks. Antitachycardia pacing was always successful in terminating the tachycardia immediately. One of the episodes of VT degenerated into ventricular fibrillation, which required ICD therapy. The patient was taken back to the electrophysiology laboratory while in incessant wide complex tachycardia. Two quadrupolar catheters were advanced to the RV apex and the His bundle area. A decapolar mapping catheter was placed along the RV septum, given the suspicion of a BBR-VT. A right bundle (RB) potential preceding every QRS complex was noted (Figure 2A). Obtaining a His bundle electrogram was challenging during sinus rhythm as well as during tachycardia. Overdrive pacing from the RV apex led to entrainment with manifest fusion and a postspacing interval within 30 ms of the tachycardia cycle length, which supported diagnosis of BBR-VT (Figure 2B). Progressive fusion was demonstrated, with faster overdrive pacing cycle lengths supporting a reentrant mechanism. Spontaneous variation in RB-RB interval preceded changes in R-R interval. A diagnosis of BBR-VT was made with RB being the antegrade limb based on entrainment criteria, presence of prolonged His-Purkinje conduction, and QRS morphology. Radiofrequency ablation using an irrigated-tip catheter was performed during VT, targeting the RB potential. Ablation was performed at 40 W, which led to termination of the VT (Figure 2C-E). Further ablation lesions were delivered targeting the RB, which led to complete AV block and paced ventricular rhythm. Aggressive programmed ventricular stimulation confirmed noninducibility of clinical tachycardia. The patient was discharged home and presented for routine postimplant follow-up after a week. During the visit, it was observed that the patient regained AV conduction with evidence of LBBB on the ECG. During threshold testing of the right ventricular lead, the patient developed an episode of VT, which was easily terminated by antitachycardia pacing (Figure 3A). The patient was brought back to the electrophysiology laboratory and diagnostic maneuvers again confirmed BBR-VT due to recovery of conduction via the right bundle branch (RBB). Repeat ablation of the RB was performed during VT, which led to termination of the VT. Post ablation, the patient developed transient complete AV block followed by antegrade conduction exclusively via the LBB, as evident by the now complete RBB block on ECG (Figure 3B). VT noninducibility was again confirmed and the patient has remained arrhythmia free at 18 months follow-up without need for any antiarrhythmic medications.

Discussion

We report a case of BBR-VT in an elderly patient with a nondilated, normally functioning left ventricle and coronary artery disease after undergoing TAVR procedure for aortic stenosis. To the best of our knowledge, BBR-VT after TAVR has not been reported previously.

BBR-VT is usually described in patients with cardiomyopathy and some form of chronic conduction defect usually associated with a long HV interval. Wide QRS tachycardia with LBBB morphology is the most common form of BBR-VT encountered in clinical practice, in which the RBB serves as the antegrade limb, the left bundle serves as the retrograde limb, and the interventricular septum provides the connecting link. BBR-VT with RBB configuration, where the reverse sequence of activation occurs, is encountered less commonly.

Most of the cases of BBR-VT are seen in patients with structural heart disease, but multiple case reports exist in the literature in patients with structurally normal hearts. BBR-VT has been described secondary to flecainide use, Brugada pattern, and AV block. Conduction abnormalities are common in valvular heart disease owing to associated ventricular dilatation and calcification of the valvular annuli. Anatomic-pathologic studies have revealed that the weakest portion of the LBB system is at the junction of the main His bundle and LBB at the level of pars membranacea and LV summit, where infarction/fibrosis and calcification are seen. We believe that the presence of aortic calcification and stretch induced from a deeply seated TAVR prosthesis worsened the His-Purkinje conduction and generated the right milieu for this form of reentry.

BBR-VT after aortic or mitral valve surgery has been studied by Calambur et al in 31 patients, with 9 cases (30%) documented at a median time of 10 days postoperatively. The patient in this cohort had baseline prolonged PR intervals, long HV interval, and relatively preserved left ventricular function. Our case presented within a week post TAVR procedure, which supports the fact that the valve replacement procedure, either surgically or percutaneously, leads to His-Purkinje system conduction delays that can
A case report of VT originating from the aortomitral continuity region in a patient with transapical TAVR has been reported, but no confirmatory electrophysiology study.

Figure 1  A: Twelve-lead electrocardiogram (ECG) depicting sinus rhythm with first-degree atrioventricular block and a QRS duration of 120 ms. B: Twelve-lead ECG after transcatheter aortic valve replacement demonstrating sinus rhythm with severely prolonged PR interval (340 ms) and left bundle branch block (LBBB), QRS duration (180 ms). C: ECG showing wide complex tachycardia with LBBB morphology, late precordial transition, and normal frontal plane axis at 200 beats per minute. D: Baseline intracardiac measurements reveal HV interval of 88 ms.
was performed in the patient. BBR-VT after TAVR has not been reported to date.

BBR-VT should be in the differential diagnosis of patients undergoing TAVR procedure who present with presyncope or syncope. If patients are noted to have worsening of AV conduction system post TAVR, as evidenced by prolongation of PR interval or the development of bundle branch block, consideration should be given to evaluate for
Figure 2  A: Intracardiac electrograms showing ventriculoatrial dissociation and right bundle (RB) potential preceding each QRS during left bundle branch tachycardia (PR 1–10 refers to decapolar navigation catheter signals across the right ventricle [RV] septum). B: Overdrive pacing from RV apex at 320 ms led to entrainment with manifest fusion and postpacing interval tachycardia cycle length (PPI-TCL) of 17 ms. C: A pre-QRS right bundle (RB) potential is noted on ablation catheter during tachycardia. D: Radiofrequency ablation at the site of recorded RB potential led to termination of ventricular tachycardia. E: Fluoroscopy image (right anterior oblique 29 degrees) showing location of ablation catheter on RV septum and relationship to aortic prosthesis.
Figure 2  Continued
BBR-VT if the patient presents with palpitations or syncope. Consideration should also be given to using short-long-short sequences during programmed electrical stimulation to induce BBR-VT.

Catheter ablation of RBB is curative for typical BBR-VT but can lead to development of complete block or further worsening of AV conduction, often requiring permanent pacemaker implant. Although catheter ablation of LBB has been reported in a patient with baseline LBBB, presence of already prolonged infra-Hisian conduction and aortic prosthesis in our patient justified ablation of RBB for treatment of BBR-VT.

Although ECG of patients presenting with BBR-VT usually shows nonspecific intraventricular conduction delay or typical bundle branch block, the fact that our patient showed AV conduction via the LBB (manifested by complete RBB block on ECG) post ablation points to the concept that conduction in bundle branches is relatively delayed to each other rather than completely interrupted. This concept has been nicely studied by Schmidt et al, where electro-anatomic mapping of left sided His-Purkinje system in patients with LBBB presenting with BBR-VT showed absent conduction via the left anterior fascicle and slowed conduction via the posterior fascicle.

Our case highlights the fact that patients after TAVR can present with sudden presyncope/syncope, which can be

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**Figure 2** Continued

**Figure 3**  
A: Tachycardia noted during internal cardioverter-defibrillator interrogation, which initiated after a short-long-short sequence and was terminated by antitachycardia pacing.  
B: Electrocardiogram showing sinus rhythm with right bundle branch (RBB) block post ablation of RBB, reflecting conduction via the left bundle branch.  
C: Echocardiogram image in parasternal long-axis view showing deep-seated Edward-Sapien 29-mm aortic prosthesis.
attributed to BBR-VT besides AV block. It is likely that the location of aortic prosthesis at the time of deployment plays a role in causing damage to the conduction system. Our patient had the prosthetic valve seated deep in the left ventricular outflow tract, as evident on echocardiogram (Figure 3C).

Conclusions
AV nodal and/or His-Purkinje conduction delay is commonly seen after percutaneous TAVR in patients with severe aortic stenosis, which can create the right milieu for development of BBR-VT. Catheter ablation of the RB is curative in
treatment of BBR-VT but requires permanent pacing support owing to already slowed AV nodal or infra-Hisian conduction. BBR-VT should be considered in the differential diagnosis of patients presenting with palpitations/syncope in the postoperative period after TAVR.

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