

EXPERT COMMENTARY

Managing Syncope After Transcatheter Aortic Valve Replacement: More than Meets the Eye

KEYWORDS. Bundle branch reentrant tachycardia, syncope, transcatheter aortic valve replacement.

ISSN 2156-3977 (print)
ISSN 2156-3993 (online)
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Rhythm Management

Drs. Vijayaraman and Young comment

In this issue of *The Journal of Innovations in Cardiac Rhythm Management*, Kocherla et al.¹ describe an interesting and educational case of bundle branch reentrant tachycardia (BBRT) in a patient with transcatheter aortic valve replacement (TAVR). Historically, BBRT has been described in patients with dilated cardiomyopathy: Caceres et al.² in 1989 reported a 6% incidence of BBRT among patients with ventricular tachycardia (VT) over an eight-year period. Of note, 85% of their patients with BBRT had a history of syncope and all patients had underlying conduction disease with prolonged H–V intervals. Subsequent reports have suggested that BBRT can occur in patients with valvular heart disease, especially after corrective surgery in the setting of underlying conduction disturbances.³ TAVR has become an established therapeutic option not only in patients with severe aortic stenosis at high risk for adverse surgical outcomes but also in patients at low risk.⁴ Conduction abnormalities including left bundle branch block (LBBB), right bundle branch block (RBBB), and complete heart block occur at significantly higher rates in patients undergoing TAVR relative to surgical valve replacement.⁵

Given the increasing volumes of TAVR and the high incidence of conduction abnormalities recorded post-TAVR, the case described by Kocherla et al. raises several important questions and concerns in this population as follows.

Who is at high risk for bundle branch reentrant tachycardia?

Kocherla et al.'s case differs from the classic description of BBRT in that the patient's left ventricular function was

normal. However, the patient in this case did have syncope—presumably from the BBRT. So far, to our knowledge, only three cases of BBRT have been described post-TAVR.^{1,6,7} All three reported cases occurred in the setting of underlying conduction disease post-TAVR. Further, of these three cases, two had RBBB and left anterior hemiblock and one demonstrated alternating BBB, all had residual atrioventricular (AV) conduction at the time of BBRT, and two had permanent pacemaker implantation at the time of BBRT. Prior reports have observed BBRT in up to 30% of patients with inducible VT following valve surgery.⁴ Of note, 40% of patients in this study had normal left ventricular (LV) function. The incidence of RBBB in patients with BBRT after valve surgery was 40% as compared with 6% among patients with dilated cardiomyopathy and BBRT.⁴ Based on these observations, normal LV function or RBBB do not preclude the development of BBRT in patients after TAVR. When patients develop RBBB following TAVR, it is likely that there is additional injury to the left bundle branch that causes significant conduction delay between the fascicles, increasing the susceptibility of reentry in these patients.

What is the true incidence of bundle branch reentrant tachycardia?

While it is difficult to assess the true incidence of BBRT post-TAVR, our suspicion is that this is likely to be higher than that observed following surgical aortic valve replacement. The incidence of conduction abnormalities post-TAVR is as high as 65%. While the incidence of sudden death was reported to be 1% to 1.8% at two years post-TAVR,⁸ ventricular arrhythmias occur at much high rates in patients with LBBB post-TAVR.⁹ Careful monitoring of patients and the evaluation of stored events in

patients with pacemakers are likely to shed additional light in this regard.

What is the potential time frame in which bundle branch reentrant tachycardia can occur?

In previous observations of VT after valve surgery, BBRT was most likely to occur within the first one to four weeks when compared with the very late occurrence of myocardial VT.³ Surgical trauma-induced conduction delays and hyperadrenergic states following surgery are potential substrate/triggers for BBRT. In three cases of BBRT following TAVR, VT occurred at three, five, and 14 days following the procedure.^{1,6,7} It is likely that a similar kind of vulnerability exists in patients undergoing TAVR.

Can bundle branch reentrant tachycardia occur in patients with complete atrioventricular block following transcatheter aortic valve replacement?

None of the cases reported had complete heart block post-TAVR. However, BBRT had been reported in a patient with complete heart block eight years after surgical aortic valve replacement who was successfully mapped and ablated.¹⁰ In the majority of patients with BBB occurring after TAVR, conduction delay occurs primarily in the main His bundle. Even if complete interruption of conduction occurs in the proximal His bundle, the distal his bundle–left bundle–right bundle junction may effectively function as the turnaround site for reentry. Is conduction delay/block occurring at the main His bundle during TAVR or is surgical aortic valve replacement alone an adequate substrate to develop BBRT? Does the patient need additional conduction delay in the peripheral bundle branches to facilitate unidirectional conduction block and reentry? We recently reported that the site of block is at the intra-Hisian level in three-fourths of patients with infranodal AV block.¹¹ It is possible that, if the conduction disease extends into the His bundle–bundle branch junction, then reentry is unlikely to occur. In our experience, permanent His-bundle pacing was feasible in 63% of 46 patients with AV block post-TAVR, suggesting that the distal His bundle is intact in these patients.¹² If so, the true incidence of BBRT may be much higher than thought and under-recognized. Further research into the mechanisms of BBRT in patients with conduction disease post-TAVR is warranted.

What is the ideal treatment for bundle branch reentrant tachycardia?

It has been well-established that BBRT can be successfully ablated by targeting the right bundle branch. In patients with normal LV function and BBRT, the overall survival rate is much higher than in those with underlying cardiomyopathy following the catheter ablation of BBRT. In patients with underlying cardiomyopathy and BBRT, often, myocardial VT coexists and implantable defibrillators (ICDs) are likely to provide additional protection. In the absence of underlying cardiomyopathy or

inducible myocardial VT, ICD implantation may not be necessary.

What type of pacing should be considered?

A significant number of patients undergoing TAVR have left ventricular hypertrophy, left ventricular diastolic and/or systolic dysfunction, and heart failure symptoms. Right ventricular pacing in such patients places them at an increased level of risk for the onset or worsening of heart failure. In a recent study of 1,629 patients undergoing TAVR, it was demonstrated that patients requiring permanent pacemakers showed a heightened risk for heart failure hospitalization and lesser LV ejection fraction improvement relative to patients without pacemakers.¹³ Importantly, this effect was more pronounced in patients with reduced LV ejection fraction pre-TAVR.¹⁴ It is likely that, in patients with reduced LV function requiring permanent pacemakers, cardiac resynchronization therapy utilizing biventricular pacing or conduction system pacing may improve clinical outcomes. Even in the absence of underlying LV dysfunction, conduction system pacing (His-bundle pacing or left bundle branch pacing) is more likely to preserve ventricular synchrony when compared with right ventricular pacing and to reduce future adverse clinical outcomes.¹⁵ In our experience, His bundle pacing is more successful in patients with SAPIEN valves (Edwards Lifesciences, Irvine, CA, USA) than CoreValve systems (Medtronic, Minneapolis, MN, USA), while left bundle branch pacing is feasible in most patients post-TAVR.¹²

How should we approach syncope in patients after transcatheter aortic valve replacement?

The recent description of BBRT in patients post-TAVR highlights the importance of establishing an accurate diagnosis regarding the etiology of syncope. While the majority of patients who present with syncope have demonstrable conduction disease in the form of RBBB, LBBB, or intermittent AV block, few may present with normal QRS intervals. In patients who present with high-grade or complete AV block with correlating symptoms, AV block is the most likely etiology of syncope and electrophysiology study in this context may not be necessary. However, in patients without demonstrable AV block with correlating symptoms, electrophysiology study may be warranted. The presumption of AV block as the cause of syncope in patients with underlying LBBB or RBBB may not be accurate. Patients with normal QRS and slightly prolonged P–R intervals may still have His-Purkinje disease and are likely to have substrate for AV block or BBRT. The completion of a thorough and complete electrophysiology study including isoproterenol challenge may be warranted to exclude the possibility of BBRT in these patients.

While BBRT is not a very common occurrence, we may see the incidence increase in the face of expanded indications for TAVR, especially in the setting of RBBB or LBBB

following TAVR. An electrophysiology study to accurately diagnose the etiology of syncope is warranted in patients post-TAVR. The treatment of BBRT should be the ablation of the right or left bundle branch. If this ablation results in heart block or if the patient has significant His–Purkinje disease, the pursuit of biventricular pacing or conduction system pacing, especially if the LV function is reduced, should be considered. If the ablation is successful, LV function is normal, and no sustained myocardial VT is induced, ICD implantation is generally not necessary.

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Dr. Vijayaraman reports he is a speaker for, researcher for, receives fellowship support from, and is a consultant to Medtronic and a consultant to Biotronik and Abbott. Dr. Young reports no conflicts of interest for the published content.

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Drs. Rao and Tung consider

TAVR is emerging as a leading treatment option for both high- and low-risk surgical candidates who suffer from severe aortic stenosis.^{1–3} While heart block is the common sequelae and concern after valve deployment, Kocherla et al.⁴ present a case in which the predominant arrhythmia was VT arising within the conduction system. After undergoing TAVR, the patient was observed to show alternating bundle branch with high-degree AV block and underwent dual-chamber pacemaker implantation. Three days after the placement of a pacemaker and discharge from the hospital, she returned with syncope and was found to have VT with device-confirmed ventriculoatrial dissociation suggestive of BBRT. The authors present an excellent discussion of classical criteria to confirm bundle branch reentry (BBR).

BBRT is a macro-reentrant circuit with anterograde activation typically progressing down the right bundle branch and retrograde conduction occurring through the left branch,⁵ which is typified by a LBBB pattern similar or identical to baseline QRS. BBRVT clinically presents as syncope or sudden cardiac death and commonly develops in patients with structural heart disease such as ischemic or nonischemic cardiomyopathy, valvular heart disease, or myotonic dystrophy.⁶ It is important to note that the majority of patients who undergo permanent device implantation after TAVR are prescribed pacemakers, which are not protective against VT. BBRVT requires a critical level of conduction disease and slowing within the normally rapid conduction system, allowing for unidirectional block and an excitable gap. The prolongation of proximal His–V shown in this case is the classical setup of requisite conduction delay needed for reentry. In all reentrant arrhythmias, a central theme of obligate slow conduction is ubiquitous (eg, slow–fast atrioventricular nodal reentry tachycardia, BBR to initiate atrioventricular reentrant tachycardia, scar-related VT with delayed and late potentials as a result of deceleration).

Considering the two approved valves (ie, SAPIEN from Edwards Lifesciences, Irvine, CA, USA and CoreValve or

Evolut R from Medtronic, Minneapolis, MN, USA), the self-expanding mechanism of the latter has been reported in the Ambulatory Electrocardiographic Monitoring for the Detection of High-degree AV Block in Patients with New-onset Persistent LBBB After TAVR (MARE) trial⁷ to accompany greater numbers of patients with persisting LBBB both at one year and overall due to the ongoing radial force projected onto the aortic annulus. In general, however, reports of tachyarrhythmias were rare, only occurring in 14% of the study population. With both types of valves, impingement on the root in turn injures and creates necrosis in the His bundle and proximal branching bundle on the basal LV septum, resulting in conduction delay (ie, intermittent bundle branch and BBR). Calcification and stretch from the prosthetic valve in addition to prior conduction disease (long P–R interval) in the patient likely attributed to her developing both brady- and tachyarrhythmia.⁸

Can this unusual sequela be predicted before transcatheter aortic valve replacement?

As expected, the presence of preexisting RBBB and long H–V interval are predictive of subsequent higher-degree infra-Hisian AV block.⁹ This patient was noted to have a narrow QRS complex with a P–R prolongation of 212, which is presumably a reflection of AV nodal disease with a long A–H interval. However, it is possible that preexisting H–V disease may have been present, though knowledge of this is unlikely to change the management decision with respect to the risk for BBRVT. One could question whether there was any validity to conducting an electrophysiological study at the time of pacemaker implantation to exclude VT and BBR, based on this report.

We agree with the authors' decision not to upgrade to an ICD after successful ablation of the right bundle. As BBR is a unique form of VT that is highly amenable to catheter ablation, ICD implantation in the absence of established primary prevention criteria or inducible myocardial VT is not warranted.¹⁰ The establishment of RBBB after ablation in the setting of LBBB without complete heart block nicely highlights that the vast majority of patients do not have "true" complete conduction block within the left bundle but, rather, show intraventricular conduction delay or intact retrograde conduction without antegrade conduction. A recent study by Chen et al. reports six patients who underwent electroanatomic mapping prior to ablation to demonstrate the nonstructural etiology of BBRT.¹¹ In the case described by Kocherla et al., the authors primarily focus on the etiology of the BBRVT from the TAVR valve; however, electroanatomic mapping could have provided additional insight on any structural abnormalities and enhanced the accuracy of termination.

In summary, the authors present a unique case that beautifully illustrates both a common and uncommon arrhythmia presentation after TAVR. It should serve to remind us that conduction slowing is the classical prerequisite for BBR and to consider tachycardia etiologies along with anticipated bradycardia in patients who suffer recurrent arrhythmic symptoms after TAVR.

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The authors report no conflicts of interest for the published content.

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