Journal of Interventional Cardiac Electrophysiology (2018) 52:385–393
https://doi.org/10.1007/s10840-018-0434-z

Bundle branch reentrant ventricular tachycardia: review and case presentation

Jorge Romero[1] · Pasquale Santangeli[2] · Rajeev K. Pathak[3] · Michael Grushko[1] · David Briceno[1] · Roberto Cerrud-Rodriguez[1] · Renato Quispe[1] · Vito Grupposo[1] · Luigi Di Biase[1]

Received: 14 June 2018 / Accepted: 27 July 2018 / Published online: 28 August 2018

Abstract
Bundle branch reentrant ventricular tachycardia (BBRVT) is characterized by a unique, fast (200–300 beats/min), monomorphic wide complex tachycardia (WCT) associated with syncope, hemodynamic compromise, and cardiac arrest. It is challenging to diagnose, requiring a His bundle recording and specific pacing maneuvers. The overall incidence has been reported to be up to 20% among patients with non-ischemic cardiomyopathy (NICM) undergoing electrophysiologic studies. We report a case of BBRVT in a patient with ischemic cardiomyopathy (ICM) presenting as a WCT with recurrent implantable-cardioverter-defibrillator (ICD) shocks. We describe all the characteristic features of BBRVT and discuss its differential. We also discuss the role of ablation for this condition.

Keywords Bundle branch reentrant ventricular tachycardia (BBRVT) · Wide complex tachycardia (WCT) · Implantable cardioverter-defibrillator (ICD) · Ischemic cardiomyopathy (ICM) · Catheter ablation (CA) · Ventricular tachycardia (VT)

1 Introduction

Bundle branch reentrant ventricular tachycardia (BBRVT) was first described by Guerot et al. in 1974 [1]. It is characterized by a unique, fast (200–300 beats/min), monomorphic wide complex tachycardia (WCT) associated with syncope, hemodynamic compromise, and cardiac arrest. It is often challenging to diagnose usually requiring a His bundle recording and specific pacing maneuvers. The overall incidence has been reported to be between 3.5 to 6% of VTs in different series, and up to 20% among patients with non-ischemic cardiomyopathy (NICM) undergoing electrophysiologic studies (EPS) and ablation [2–4].

Herein, we report a case of BBRVT in a patient with ischemic cardiomyopathy (ICM) presenting as a WCT with recurrent implantable-cardioverter defibrillator (ICD) shocks. We will describe all the characteristic features of this arrhythmia and how to differentiate this from a myriad of other rhythm disorders. We will also discuss the utility of radiofrequency ablation for this condition.

2 Case: wide complex tachycardia in ischemic cardiomyopathy

A 52-year-old gentleman from outside the USA with a history of ICM, a left ventricular (LV) ejection fraction of 15–20%, and New York Heart Association Class II symptomatology, who had a single chamber primary prevention ICD placed in 2010, presented with frequent ICD shocks. In 2009, he underwent multi-vessel coronary artery bypass grafting (CABG). A year after ICD implantation, the patient received seven appropriate ICD shocks for episodes of monomorphic VT. He was placed on amiodarone and higher doses of beta-blockers. Since 2012, he experienced a total of 40 episodes of tachyarrhythmias labeled by his device as either SVT or VT for which he has received 28 ICD shocks. His baseline electrocardiogram revealed sinus rhythm, left superior axis, and left bundle branch block (LBBB) of 134-ms duration (Fig. 1). Unfortunately, there were no ECGs of his clinical arrhythmia available for interpretation. Device interrogation showed multiple rapid monomorphic VTs ranging from 188 to 210 bpm and one episode of VT

✉ Luigi Di Biase
dibbia@gmail.com

[1] Division of Cardiology, Montefiore Medical Center, Albert Einstein College of Medicine, 111 East 210th Street, Bronx, NY 10467, USA

[2] Penn Heart and Vascular Center, Perelman Center for Advanced Medicine, East Pavilion, 2nd Floor, 3400 Civic Center Boulevard, Philadelphia, PA 19104, USA

[3] Canberra Hospital, Australian National University, Canberra, Australia

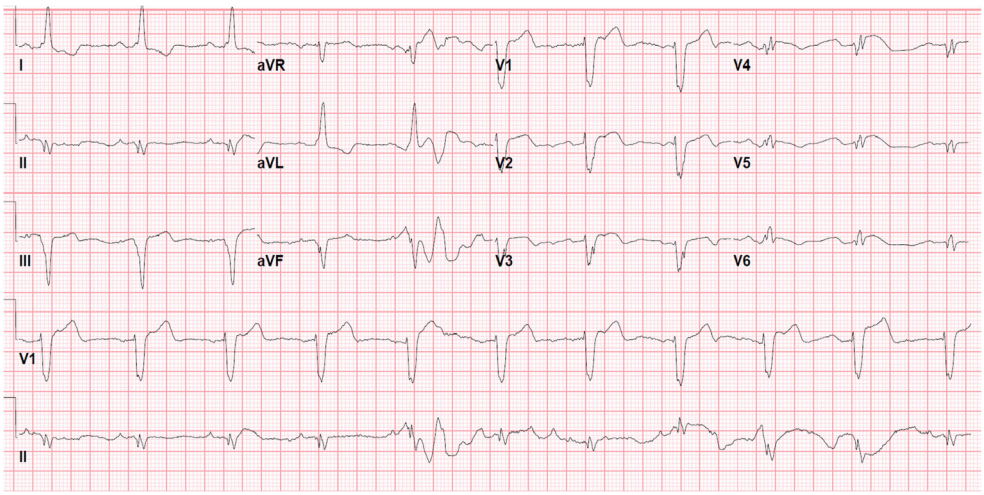

Fig. 1 Baseline 12-lead electrocardiogram. Sinus rhythm, left superior axis, and left bundle branch block (134 ms)

that degenerated into ventricular fibrillation. Several of these episodes were successfully terminated with anti-tachycardia pacing. Due to recurrent ICD therapies despite appropriate heart failure management and mexiletine, amiodarone, and procainamide therapy, the patient was taken to the laboratory for EPS and possible VT radiofrequency ablation.

Given his history of myocardial infarction, severe coronary artery disease with CABG, and resultant depressed LV function, the initial assumption was that there would be appropriate myocardial substrate for reentrant VT. Hence, substrate mapping was initially performed over the LV endocardium. An extensive low-voltage area (less than 1.5 mV) was present in the anteroseptal and inferoseptal walls. This scar extended from base to apex (Fig. 2a–c). The unipolar voltage map also suggested mid-myocardial and epicardial involvement in the same area indicating probable transmural scar from prior myocardial infarctions (Fig. 2d). However, because the VT morphology was unknown, the theoretical possibility of either the primary arrhythmia or a secondary arrhythmia being an SVT (based on the device classifying multiple episodes as SVT), and to define a potential endpoint of ablation, a full EPS was performed before performing an empiric substrate modification of the ventricular scar.

The patient presented to the EP Lab in sinus rhythm and a left bundle branch block (LBBB) with a QRS duration of 130 ms. There was evidence of conduction delay in the His-Purkinje system (HPS) as revealed by a prolonged HV interval

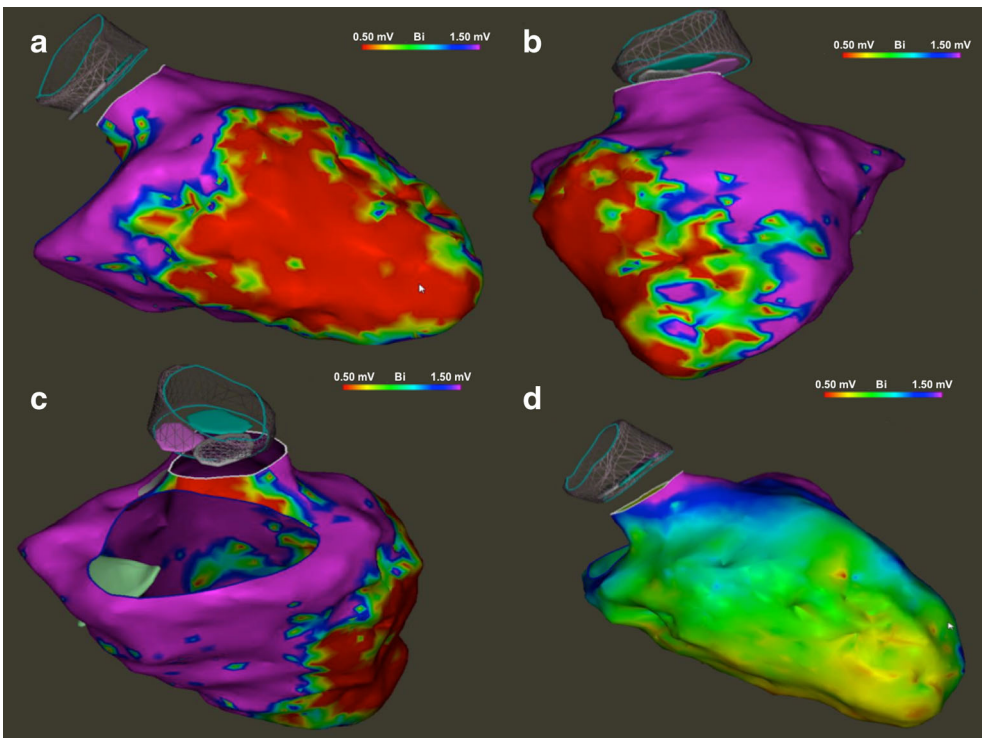

Fig. 2 Electroanatomic (EAM) voltage map of the left ventricle. **a–c** The bipolar volatge amp in septal anterior and inferior views of the scar in the left ventricle. **d** The unipolar voltage map of the left ventricle, suggesting large transmural component of the scar

Fig. 3 Baseline intervals. HV interval was prolonged (75 ms). His His bundle, RV right ventricle

of 75 ms at baseline and during atrial pacing (Fig. 3). There was no evidence of dual AV nodal physiology. There was no baseline pre-excitation or any pre-excitation with differential atrial pacing. Ventricular extrastimulus testing (500/280 ms) from the right ventricular apex repeatedly induced a sustained WCT at 200 bpm with a LBBB morphology, with left inferior axis and a transition at V3 (Fig. 4). Interestingly, the precordial lead morphology was very similar to sinus rhythm.

Though the QRS during WCT looked similar to his baseline LBBB, there was clear evidence of VA dissociation (Fig. 5), strongly suggesting VT instead of either antidromic or orthodromic AV reentrant tachycardia, or SVT with aberrancy such as atrial tachycardia. Likewise, antegrade atrio-fascicular accessory pathway was ruled out. However, junctional tachycardia, AVNRT with AV block, and a nodofascicular pathway could still be possibilities. Interestingly, the His electrogram clearly preceded the QRS during VT, and the HV interval during VT was longer than the HV interval during sinus rhythm (84 vs. 75 ms) (Fig. 5). This finding essentially ruled out myocardial reentry VT, which should also have a negative HV interval. Spontaneous variations in V-V intervals were also *preceded* by similar changes in H-H intervals (Fig. 5). Also, there was rapid intrinsicoid deflection in the right precordial leads, suggesting that the initial ventricular activation occurred through the HPS. These findings are typically observed in BBRVT. The prolongation of the HV interval during BBRVT is thought to be caused by anisotropic conduction seen in the distal His bundle at the upper turnaround point of the tachycardia circuit. Entrainment of the VT by rapid pacing at the RV apex showed manifest fusion with a postpacing interval (PPI) minus tachycardia cycle length (TCL) of 29 ms (Fig. 6), suggesting that the RV apical myocardium is close to the tachycardia circuit and that the mechanism is likely to be BBRVT rather than myocardial reentry. In addition, this would also rule out AVNRT with upper common pathway block and nodofascicular pathways as these would have a long PPI-TCL intervals [5]. We attempted to entrain the tachycardia by atrial pacing to demonstrate concealed fusion but AV conduction limited this maneuver even with the administration of isoproterenol.

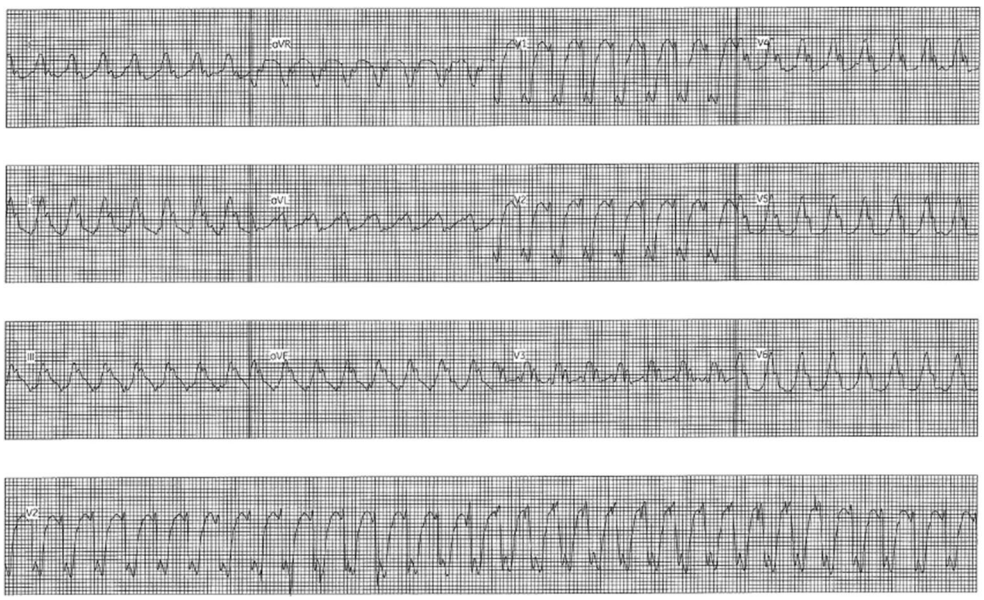

Fig. 4 Twelve-lead ECG of VT induced repeatedly in the EP laboratory. Notice the similar QRS morphology in precordial leads during VT and sinus rhythm. (LBBB morphology with left inferior axis)

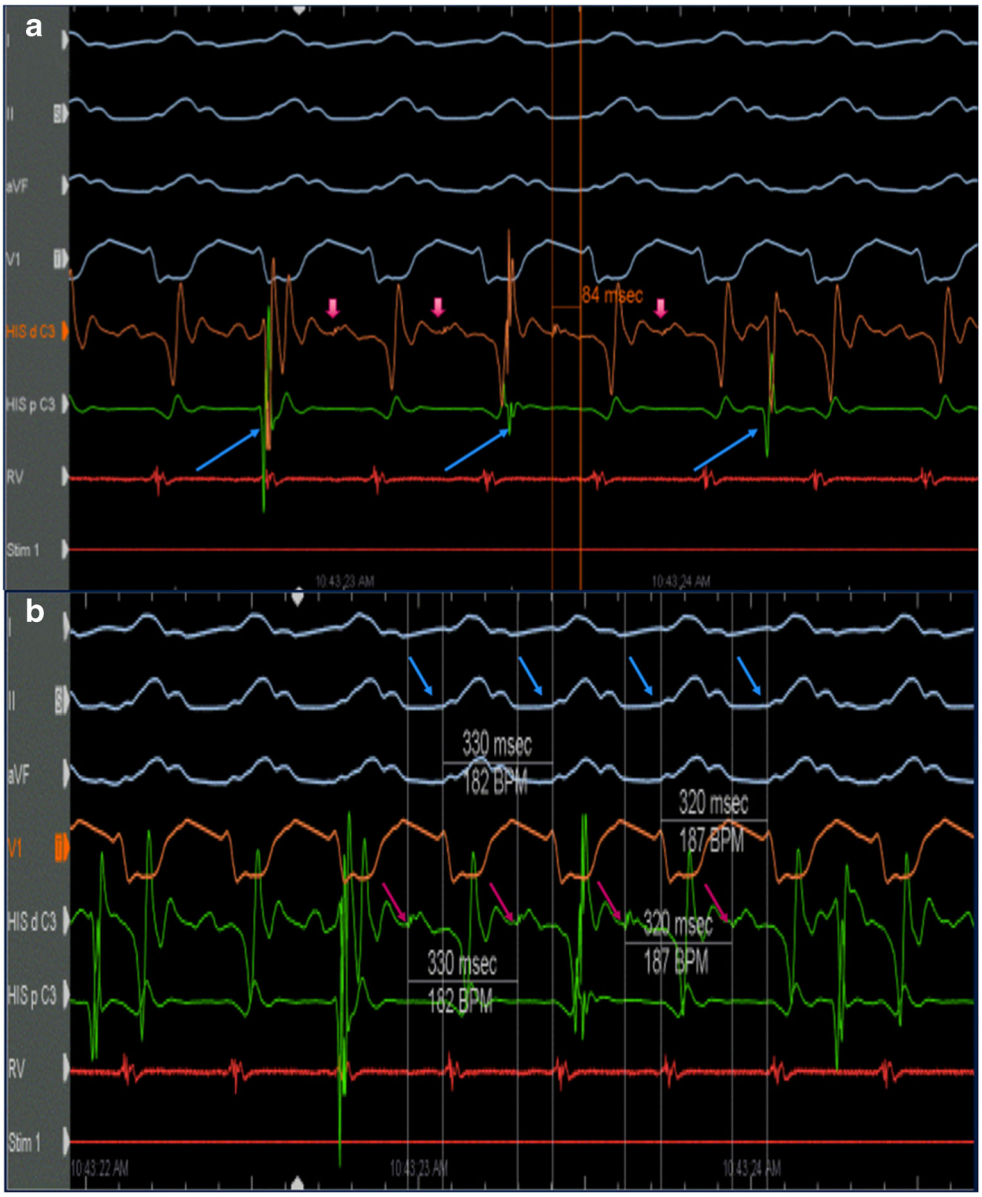

Fig. 5 **a** Ventricular tachycardia with VA dissociation (blue arrows show atrial signal on His channel). Notice that the His signal (pink arrows) precedes the QRS and that the HV interval is longer than in sinus rhythm (i.e.,84 ms vs. 75 ms). **b** During VT, the variations in the V-V interval (blue arrows) are preceded by H-H interval changes (pink arrows). This findings suggests that the HIs bundle is driving the VT

The decision was made to localize and ablate the left bundle branch (LBB) since the patient already had a "complete LBB block," and, in this way, reduce the risk of complete heart block and if unable, to ablate the right bundle branch (RBB). The LBB

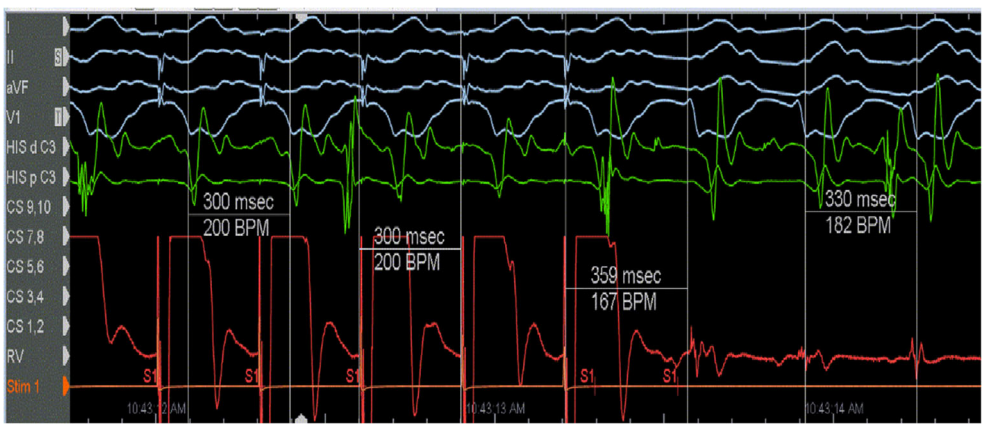

Fig. 6 Entrainment from the RV apex showing manifest fusion but PPI-TCL < 30 ms

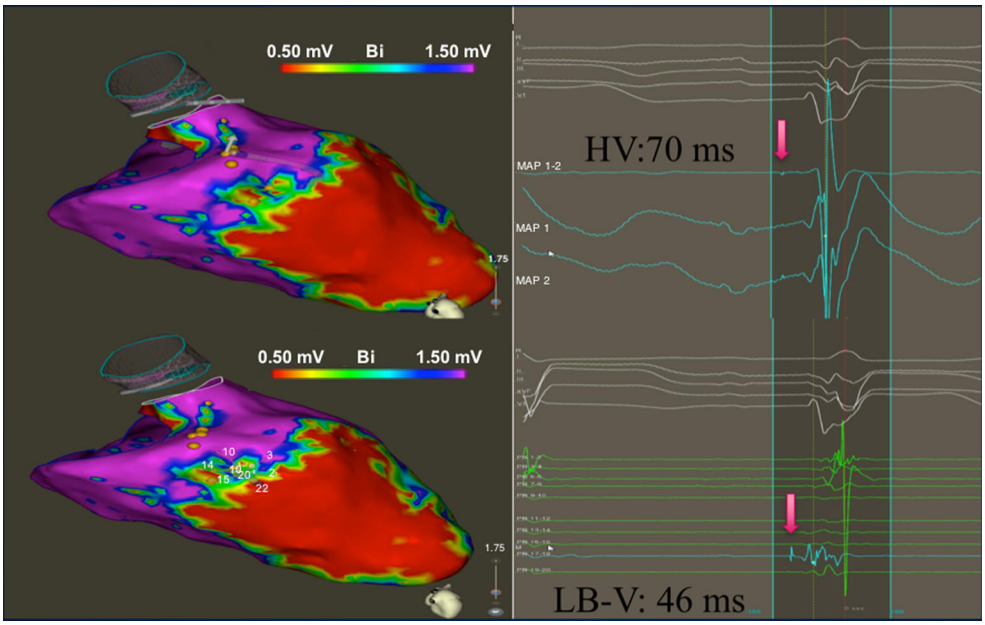

Fig. 7 Electroanatomic mapping illustrating the His bundle location in the distal electrodes of an ablation catheter (top) and the left bundle location (bottom) using a PENTARAY catheter (Biosense Webster, Inc., a Johnson & Johnson company). HV interval 70 ms, LB-V time 46 ms

was localized (Fig. 7; LB-QRS 46 ms) and three non-irrigated radiofrequency lesions were delivered at this location (50 W) achieving an impedance drop of 14 Ω during the third application. QRS duration increased from 132 to 180 ms. The lesion was delivered for 52 s. The arrhythmia was no longer inducible with up to triple extrastimuli from the right and left ventricles (Fig. 8). Of note, it is important to point out that by targeting the LBB, interventricular synchrony may worsen, increasing the need for a CRT-D upgrade.

Subsequently, based on the large septal scar revealed by endocardial voltage mapping, with multiple late potentials and local abnormal ventricular activities (LAVA) (Fig. 9), and the fact that the patient was on three different antiarrhythmic medications, the decision was made to perform substrate modification of the scar. Areas of late potentials and areas of electrically unexcitable scar (pacing threshold greater than 10 mA at 2-ms pulse width) were tagged. Much of the scar region surrounding the midseptum was not electrically excitable. Some sites captured with long delays and QRS morphologies suggesting propagation away through protected channels. Radiofrequency ablation lesions using a 3.5-mm externally irrigated Thermocool Smart Touch catheter (Biosense Webster, Inc. Diamond Bar, CA) were titrated up to a maximum of 50 W over 60–90 s for an impedance drop of up to 18 Ω. Whenever possible, a contact force of greater than 10 g was achieved. A total of 72 lesions in the interventricular septum were delivered for substrate modification (scar dechanneling/core isolation). Radiofrequency ablation performed at likely reentry circuit sites based on substrate mapping rendered the region electrically unexcitable. After ablation, programmed stimulation with up to three tightly coupled extrastimuli during pacing at a drive cycle length of 500 ms was performed with no arrhythmia induction. Patient was discharged 2 days later, off all antiarrhythmic medications. Anticoagulation was administered for 6 weeks postablation. After 18 months of follow-up, the patient has remained asymptomatic and device interrogation revealed no arrhythmia recurrences. Despite LBBB, patient remains NYHA class I and has not required upgrade to CRT-D.

3 Clinical features and comorbidities

Typically, BBRVT has a sudden onset that can lead to hemodynamic instability. Symptoms include dizziness, palpitations, and even more malignant presentations like syncope or sudden cardiac arrest.

Despite being much more common in patients with non-ischemic cardiomyopathy (about one third of inducible sustained VTs) [2], BBRVT occurs in approximately 5% of those with ICM. Underlying disease of the HPS is commonly seen in patients with BBRVT. Dilated cardiomyopathy and prior valve surgery are among the most common predisposing factors. Less frequently, BBRVT has been found in patients with myotonic dystrophy [6–8].

4 Electrophysiological mechanism and diagnostic criteria

BBRVT is a form of a macroreentrant circuit involving the bundle of His, both bundle branches, and the intervening septal/ventricular myocardium. If conduction in the His-Purkinje system (HPS) is prolonged (i.e., drugs, ischemia, fibrosis), sustained reentry utilizing the bundle branches is facilitated. Although LBBB and RBBB are more indicative of conduction slowing rather than complete block in such bundle branches,

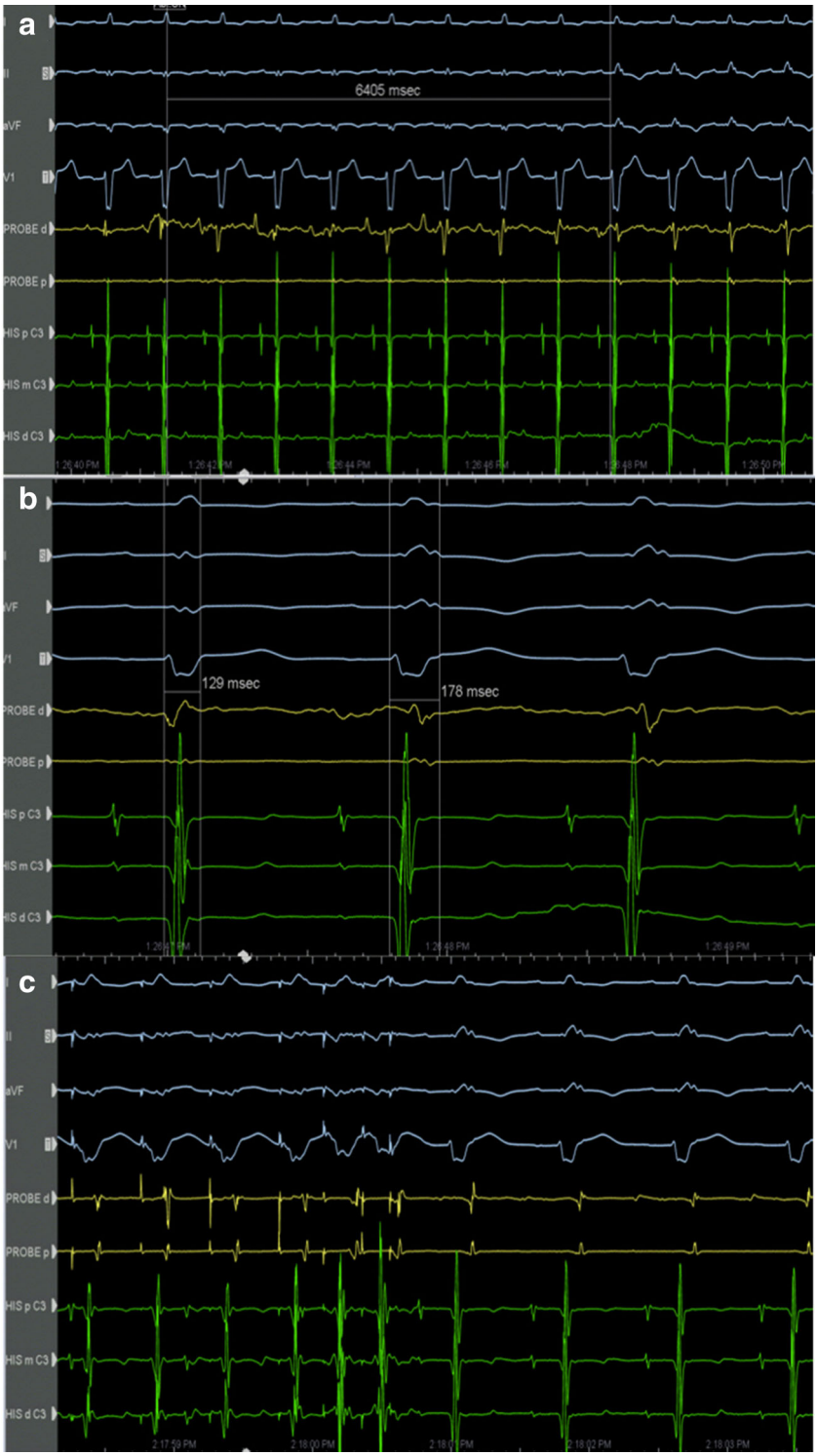

Fig. 8 **a** RF ablation of the left bundle. Notice that the QRS widens from 132 to 184 ms 6 s after starting RFA (25 mm/s). **b** This figure illustrates the change in QRS width at a speed of 100 mm/s. **c** PES from RV apex (500/290/260/200) did not induce tachycardia

there must be a delay or complete anterograde block in at least one bundle branch in order for BBR to occur. Of note, because conduction in BBRVT proceeds in part down the HPS, BBR can present with an identical QRS complex morphology to that in normal sinus rhythm. Although QRS morphology in VT can be identical to that in sinus rhythm [9], not all such VTs are BBR [10]. We believe that, in the case of our patient, his ICM with extensive scar involving the ventricular septum, as well as the additional conduction slowing caused by antiarrhythmic therapy, may have facilitated the appropriate degree of regional/septal conduction slowing necessary to initiate BBRVT.

BBRVT has several modes of spontaneous initiation, though ventricular premature beats are the most common trigger, more often than atrial beats or atrial fibrillation [11].

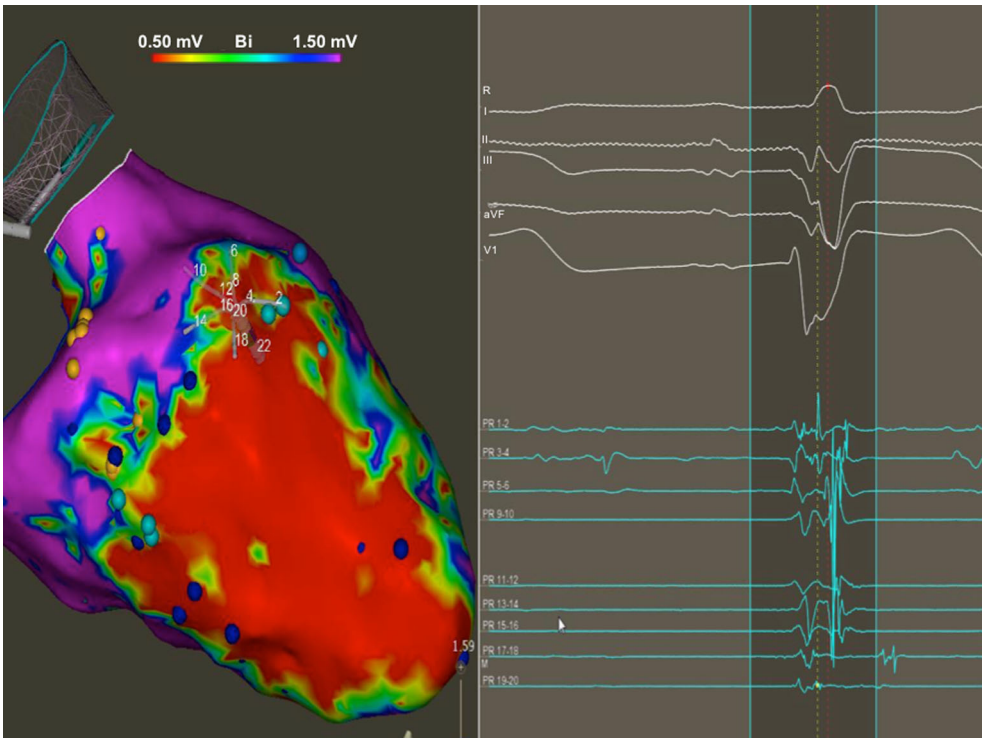

Fig. 9 Local abnormal ventricular activities and late potential within the LV septal scar

Ventricular extrastimuli delivered with short-long-short coupling intervals cause unidirectional block, or sufficient conduction delay in a bundle branch to initiate BBR [12]. Atrial pacing and/or isoproterenol, as well as class IA or IC antiarrhythmics (which may slow conductions in one of the bundles), may also be required to induce BBRVT [13, 14]. Of note, our patient was on mexiletine, which as a class 1B antiarrhythmic, could have also played a role in facilitating BBRVT due to a significant decrease in retrograde refractoriness within the His-Purkinje system [15].

There are three categories of BBRVT as described by Tchou and colleagues (Table 1) [16]. Type A and C are the classic counterclockwise and clockwise BBRVT circuits, respectively. Type B is interfascicular reentry within the LBB fascicles themselves, most commonly seen in patients with anterior wall infarction and left anterior or posterior hemifascicular block.

Patients with BBR commonly have prolongation of the PR interval, QRS duration, and HV interval. In type A BBRVT, activation proceeds anterograde via the RBB, then leftward through the septum, with subsequent retrograde conduction via the LBB up to the septal summit and His bundle, lateral conduction through the His, and reactivation of the RBB ("counterclockwise" reentry). As such, the ECG morphology is a typical LBBB with R-wave transition between leads V4 and V5 [17]. In type C BBRVT, activation proceeds anterogradely via the LBB, then rightward through the septal myocardium, with subsequent retrograde activation via the RBB thereby generating a RBBB morphology ("clockwise" reentry). In type B or the interfascicular form of BBRVT, the VT is initiated when an atrial or ventricular premature depolarization is conducted over the healthy fascicle, leading to QRS inscription, then reenters the blocked fascicle in a retrograde fashion to induce reentrant VT. The upper common pathway is in the fascicles themselves.

Conduction delay is critical for initiation and perpetuation of BBR, and can be present in the connection between the bundles or within the His bundle itself. A greater degree of HV delay is usually found during tachycardia compared to sinus rhythm. One potential explanation for such finding could be related to anisotropic conduction in the distal His bundle. Alternative explanations include progressive distal Purkinje system conduction delay in unhealthy bundles. Rate-related conduction delay between the His bundle and RBB/LBB could be an explanation; however, it is unclear whether the common His bundle is required for BBR propagation, although it is necessary for reentry to occur.

BBR can also occur in patients with normal His-Purkinje conduction during sinus rhythm but impaired at faster rates [18]. This is likely because of the higher turnaround point in the BBR circuit in the proximal portion of His bundle, or in the NH region of the atrioventricular node (transition from the AV node to the His bundle).

The currently accepted criteria for the diagnosis of BBRVT [2, 19] include the following:

1. QRS morphology showing a typical LBBB or RBBB pattern.
2. Onset of ventricular depolarization is preceded by potentials in His-bundle (H), right (RB) or left bundle branch (LB), that follow an appropriate sequence of activation (H-RB-LB) with relatively stable intervals.

Table 1 Types of bundle branch reentry tachycardia

	Type A	Type B (interfascicular tachycardia)	Type C
Anterograde limb	RBB	LAF or LPF	LBB
Retrograde limb	LBB	Contra-left fascicle	RBB
Electrocardiogram morphology	LBBB pattern	RBBB pattern	RBBB pattern

LAF left anterior fascicle, LBB left bundle branch, LBBB left bundle branch block, LPF left posterior fascicle, RBB right bundle branch, RBBB right bundle branch block

3. Variations in the VV interval are *preceded* by similar changes in H-H/RB-RB/LB-LB intervals.
4. Induction of tachycardia during stimulation is dependent on HP conduction delay.
5. Termination of tachycardia is preceded by spontaneous or pacing-induced block in HPS.
6. BBR is non-inducible after successful RBB ablation.

5 Ablation

The typical ablation approach is to target the right bundle branch [6, 8]. As stated above, these patients typically have baseline His-Purkinje disease. Therefore, the incidence of significant conduction impairment necessitating permanent pacemaker placement postablation has been reported in up to 30% of patients [20]. Consequently, it has been suggested that ablation of the left bundle in patients with pre-existing LBBB may decrease the incidence of postablation complete heart block [21].

The optimal ablation approach in patients with BBRVT and large myocardial scars is unknown. In this case, a large septal scar was revealed, and though initially planned for substrate modification, the ablation strategy was modified in view of the diagnosis of BBRVT. The left bundle was targeted to try and minimize heart block and was successfully ablated, rendering the patient non-inducible thereafter even with aggressive ventricular extra-stimulation protocols. Without clear evidence as to the optimal ablation strategy in patients with concomitant scar and BBRVT, given the patients' history, scar homogenization was also performed. There is one caveat about this approach, though, and it is that the risk of complications might outweigh the potential benefits if it is followed in less-experienced centers.

6 Conclusion

BBRVT, though more common in NICM patients, is also seen in ICM patients with scar in the interventricular septum. RFA ablation of the LBB is preferable in patients with underlying LBB disease to avoid the risk of complete heart block. Bundle branch ablation together with substrate modification might be a reasonable ablation option in patients with BBRVT and large scars who are under general anesthesia and multiple AAD to increase long-term success rate.

Compliance with ethical standards

Conflict of interest Dr. Di Biase is a consultant for Stereotaxis, Biosense Webster, Boston Scientific, and St. Jude Medical. Dr. Di Biase received speaker honoraria/travel from Medtronic, Janssen, Bristol Meyers Squibb, EPiEP, and Biotronik. Vito Grupposo is a clinical account specialist at Biosense Webster. The other authors have no disclosures. Rajeev K. Pathak is supported by an Early Career Fellowship from the National Health and Medical Research Council of Australia.

References

1. Guerot C, Valere PE, Castillo-Fenoy A, Tricot R. Tachycardia by branch-to-branch reentry. Arch Mal Coeur Vaiss. 1974;67(1):1–11.
2. Caceres J, Jazayeri M, McKinnie J, Avitall B, Denker ST, Tchou P, et al. Sustained bundle branch reentry as a mechanism of clinical tachycardia. Circulation. 1989;79(2):256–70.
3. Delacretaz E, Stevenson WG, Ellison KE, Maisel WH, Friedman PL. Mapping and radiofrequency catheter ablation of the three types of sustained monomorphic ventricular tachycardia in nonischemic heart disease. J Cardiovasc Electrophysiol. 2000;11(1):11–7.
4. Cantillon DJ, Bianco C, Wazni OM, Kanj M, Smedira NG, Wilkoff BL, et al. Electrophysiologic characteristics and catheter ablation of ventricular tachyarrhythmias among patients with heart failure on ventricular assist device support. Heart Rhythm. 2012;9(6):859–64. https://doi.org/10.1016/j.hrthm.2012.01.018.
5. Merino JL, Peinado R, Fernandez-Lozano I, Lopez-Gil M, Arribas F, Ramirez LJ, et al. Bundle-branch reentry and the postpacing interval after entrainment by right ventricular apex stimulation: a new approach to elucidate the mechanism of wide-QRS-complex tachycardia with atrioventricular dissociation. Circulation. 2001;103(8):1102–8.
6. Cohen TJ, Chien WW, Lurie KG, Young C, Goldberg HR, Wang YS, et al. Radiofrequency catheter ablation for treatment of bundle branch reentrant ventricular tachycardia: results and long-term follow-up. J Am Coll Cardiol. 1991;18(7):1767–73.
7. Blanck Z, Dhala A, Deshpande S, Sra J, Jazayeri M, Akhtar M. Bundle branch reentrant ventricular tachycardia: cumulative experience in 48 patients. J Cardiovasc Electrophysiol. 1993;4(3):253–62.
8. Mehdirad AA, Keim S, Rist K, Tchou P. Long-term clinical outcome of right bundle branch radiofrequency catheter ablation for treatment of bundle branch reentrant ventricular tachycardia. Pacing Clin Electrophysiol. 1995;18(12 Pt 1):2135–43.
9. Olshansky B. Ventricular tachycardia masquerading as supraventricular tachycardia: a wolf in sheep's clothing. J Electrocardiol. 1988;21(4):377–84.
10. Guo H, Hecker S, Levy S, Olshansky B. Ventricular tachycardia with QRS configuration similar to that in sinus rhythm and a myocardial origin: differential diagnosis with bundle branch reentry. Europace. 2001;3(2):115–23. https://doi.org/10.1053/eupc.2001.0151.

11. Blanck Z, Jazayeri M, Akhtar M. Facilitation of sustained bundle branch reentry by atrial fibrillation. J Cardiovasc Electrophysiol. 1996;7(4):348–52.
12. Akhtar M, Denker S, Lehmann MH, Mahmud R. Macro-reentry within the His-Purkinje system. Pacing Clin Electrophysiol. 1983;6(5 Pt 2):1010–28.
13. Simons GR, Sorrentino RA, Zimerman LI, Wharton JM, Natale A. Bundle branch reentry tachycardia and possible sustained interfascicular reentry tachycardia with a shared unusual induction pattern. J Cardiovasc Electrophysiol. 1996;7(1):44–50.
14. Mizusawa Y, Sakurada H, Nishizaki M, Ueda-Tatsumoto A, Fukamizu S, Hiraoka M. Characteristics of bundle branch reentrant ventricular tachycardia with a right bundle branch block configuration: feasibility of atrial pacing. Europace. 2009;11(9):1208–13. https://doi.org/10.1093/europace/eup206.
15. Ruskin JN, Akhtar M, Damato AN, Foster JR. The effect of lidocaine on reentry within the his-Purkinje system in man. Circulation. 1980;62(2):388–400.
16. Tchou P, Mehdirad AA. Bundle branch reentry ventricular tachycardia. Pacing Clin Electrophysiol. 1995;18(7):1427–37.
17. Blanck Z, Akhtar M. Ventricular tachycardia due to sustained bundle branch reentry: diagnostic and therapeutic considerations. Clin Cardiol. 1993;16(8):619–22.
18. Li YG, Gronefeld G, Israel C, Bogun F, Hohnloser SH. Bundle branch reentrant tachycardia in patients with apparent normal his-Purkinje conduction: the role of functional conduction impairment. J Cardiovasc Electrophysiol. 2002;13(12):1233–9.
19. Tchou P, Jazayeri M, Denker S, Dongas J, Caceres J, Akhtar M. Transcatheter electrical ablation of right bundle branch. A method of treating macroreentrant ventricular tachycardia attributed to bundle branch reentry. Circulation. 1988;78(2):246–57.
20. Lopera G, Stevenson WG, Soejima K, Maisel WH, Koplan B, Sapp JL, et al. Identification and ablation of three types of ventricular tachycardia involving the his-purkinje system in patients with heart disease. J Cardiovasc Electrophysiol. 2004;15(1):52–8. https://doi.org/10.1046/j.1540-8167.2004.03189.x.
21. Blanck Z, Deshpande S, Jazayeri MR, Akhtar M. Catheter ablation of the left bundle branch for the treatment of sustained bundle branch reentrant ventricular tachycardia. J Cardiovasc Electrophysiol. 1995;6(1):40–3.