



## Case Report

## Coronary PCI revascularization novel treatment of bundle branch reentrant ventricular tachycardia



Sonia Marrakchi (MD)<sup>a,b,\*</sup>, Lobna Laroussi (MD)<sup>a</sup>, Emna Bennour (MD)<sup>b</sup>, Ikram Kammoun (MD)<sup>a</sup>, Salem Kachboura (MD)<sup>a</sup>

<sup>a</sup> Cardiology Department, Abderrahman Mami Hospital Ariana, Tunis, Tunisia

<sup>b</sup> University Manar II – Medical University of Tunis, Tunis, Tunisia

## ARTICLE INFO

## Article history:

Received 12 April 2019

Received in revised form 15 July 2019

Accepted 19 July 2019

## Keywords:

Bundle branch reentrant ventricular tachycardia

Coronary revascularization

Ischemia

Syncope

## ABSTRACT

Bundle branch reentrant ventricular tachycardia (VT) is a form of macroreentrant tachycardia. Although infrequent in occurrence, this arrhythmia presents with serious clinical manifestations and has potential for cure by catheter ablation.

We report a case of bundle branch reentrant VT with ischemic source. Revascularization of culprit coronary artery was another means to treat VT.

**<Learning Objective:** The role of coronarography exploration in patients with bundle branch reentrant tachycardia and a history of coronary artery disease before right bundle branch ablation with risk of conduction disorders.>

© 2019 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

## Introduction

Bundle branch reentrant ventricular tachycardia (BBR-VT) is a form of macroreentrant tachycardia involving the bundle of His, both bundle branches, and the ventricular myocardium in the circuit. BBR-VT is seen in about 6% of all patients with inducible sustained monomorphic VT who undergo programmed electrical stimulation for VT induction [1]. It generally occurs in the background of dilated cardiomyopathy, prior valve surgery, or other cardiac disease with an underlying His-Purkinje system (HPS) disease. Clinically, BBR-VT usually results in marked hemodynamic compromise and often presents with syncope, presyncope, or sudden cardiac arrest [1].

Although infrequent in occurrence, this arrhythmia presents with serious clinical manifestations and has potential for cure by catheter ablation [1].

We report a case of BBR-VT with ischemic source.

## Case history

A 50-year-old male with hypertension, hyperlipidemia, diabetes, asthma, pacemaker VVI Saint Jude Medical ZEPHYR XL SR SSIR (St Jude Medical, St Paul, MN, USA) implanted for conduction disease with syncope and long HV in electrophysiology test

without VT response to programmed electrical stimulation during study. His pacemaker was programmed VVI 40–130.

He had a history of percutaneous coronary intervention (PCI) revascularization due to significant stenosis in the middle of the left anterior descending artery by bare metal stent in another hospital. He was admitted for recurrent syncope caused by wide QRS tachycardia detected by telemetry of his pacemaker.

Physical examination revealed normal blood pressure of 120/80 mmHg on both arms, pulse was 64 bpm with a regular rhythm. Chest and abdomen investigation was unremarkable. Extremities were warm and well perfused, with normal range of motion and no edema. The patient had no cognitive defects

Clinical blood, thyroid, and coagulation profiles were normal. Biochemistry panel revealed hyperlipidemia and modestly elevated creatine kinase. Troponin test was negative. HbA1c level was 6.1%. B-type natriuretic peptide level was less than 10 pg/mL.

Electrocardiogram (ECG) at rest displayed sinus rhythm 65 bpm, PQ interval was 220 ms, QRS 120 ms, and right branch block and left anterior hemibranch block.

24-h ECG monitoring registered two episodes of non-sustained supraventricular arrhythmias and no significant pauses (Fig. 1). Echocardiography revealed a preserved left ventricular ejection fraction (LVEF) (50%), without wall motion abnormalities, and no valvular disease.

Electrophysiology study showed delayed conduction in HPS. We recorded decremental conduction properties in anterograde and retrograde conduction. AH interval duration was 66 ms, His interval duration was 26 ms, Wenckebach point anterogradely

\* Corresponding author at: 8, Rue Zohra Touati El Manar 1- 2092, Tunis, Tunisia.  
E-mail address: [sonia.marrakchi@fmt.utm.tn](mailto:sonia.marrakchi@fmt.utm.tn) (S. Marrakchi).

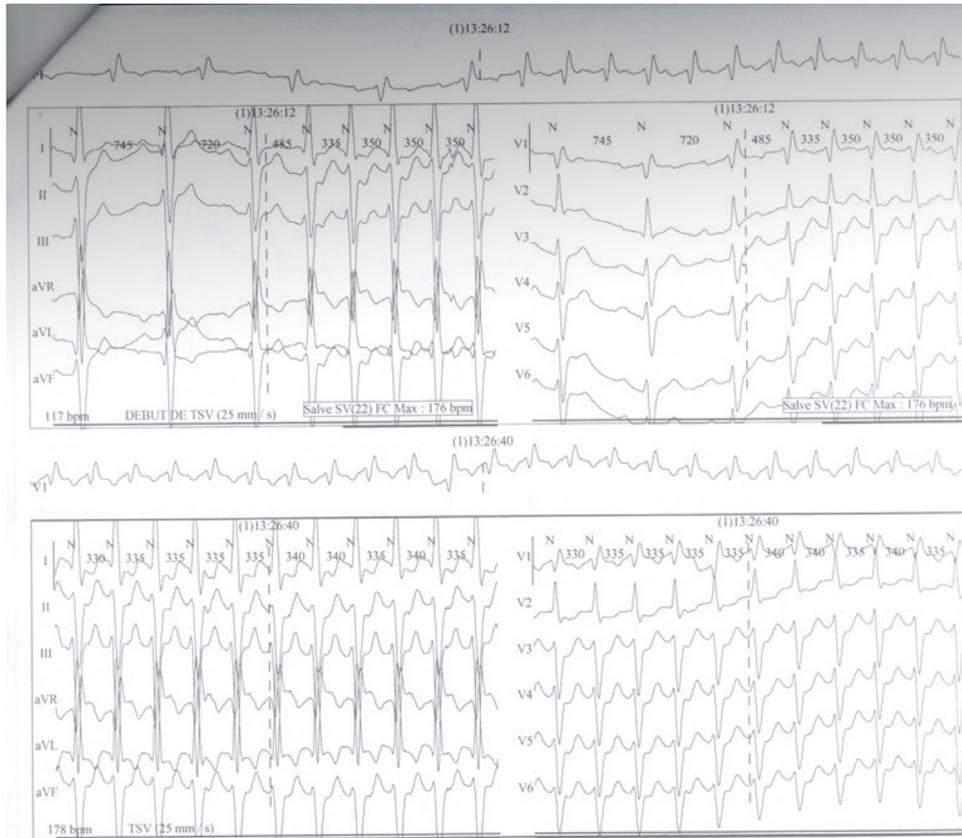


Fig. 1. Wide tachycardia with abrupt onset at 170 bpm.

and retrogradely was 280 ms, atrioventricular node refractory period was 290 ms. HV interval duration was 100 ms (Fig. 2). Programmed electrical stimulation from the right ventricle apex using one extrastimulus was performed, which reproducibly at 600 340 ms induced BBR-VT with a rate of 170 bpm and RBBB morphology with anterograde conduction over LBB exactly from left posterior hemibranch and retrograde conduction over RBB (Fig. 2). HV interval duration in VT was 120 ms. All episodes of VT were successfully terminated by burst pacing.

The following are the currently accepted diagnostic criteria for BBR-VT:

- the QRS morphology of the tachycardia shows the same QRS in sinus rhythm;
- the onset of ventricular depolarization is preceded by His-bundle (H) and stable HV;
- spontaneous variations in V–V intervals are preceded by similar changes in H–H intervals;

- the induction of tachycardia during programmed stimulation is consistently dependent on achieving a critical conduction delay in the HPS;
- the tachycardia termination is preceded by a spontaneous or pacing-induced block in the HPS.

Another characteristic feature of classic BBR-VT is that the HV during tachycardia is longer than HV during sinus rhythm. This prolongation of the HV interval during tachycardia is speculated to be caused by anisotropic conduction seen in the distal His bundle at the upper turn around point of the tachycardia circuit (Fig. 2).

VT morphology was RBBB morphology with anterograde conduction over LBB and retrograde conduction over RBB because QRS of VT were similar to QRS in sinus rhythm but it was also possible the anterograde conduction was over posterolateral hemibranch and the retrograde conduction were over RBB or over anterolateral left hemibranch. We suspected an interfascicular VT in our patient. In fact, both diastolic and presystolic Purkinje

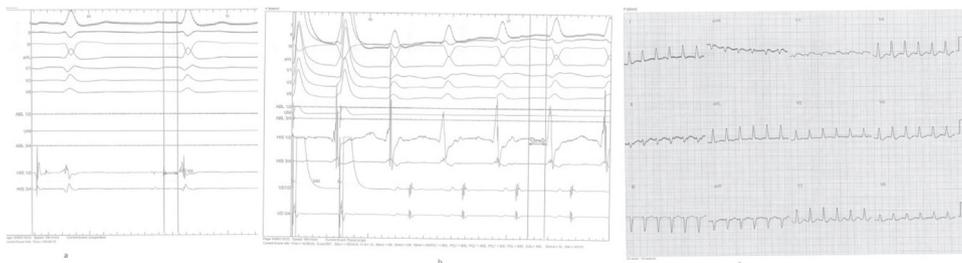


Fig. 2. (a) HV interval duration was 100 ms at base. Long HV 120 ms (Fig. 2b) in wide QRS tachycardia at 170 bpm (Fig. 2c).

potentials were sequentially recorded. During the VT, the late Purkinje potentials observed in sinus rhythm were recorded in front of VT. Induction of VT occurred after progressive retrograde conduction delay on increasing the prematurity of the extra-

stimulus (Fig. 3). Anterograde conduction occurred exclusively over the left posterior fascicle, which caused identical QRS morphology during sinus rhythm and VT. During VT, the left anterior fascicle was used retrogradely. The usual target for bundle

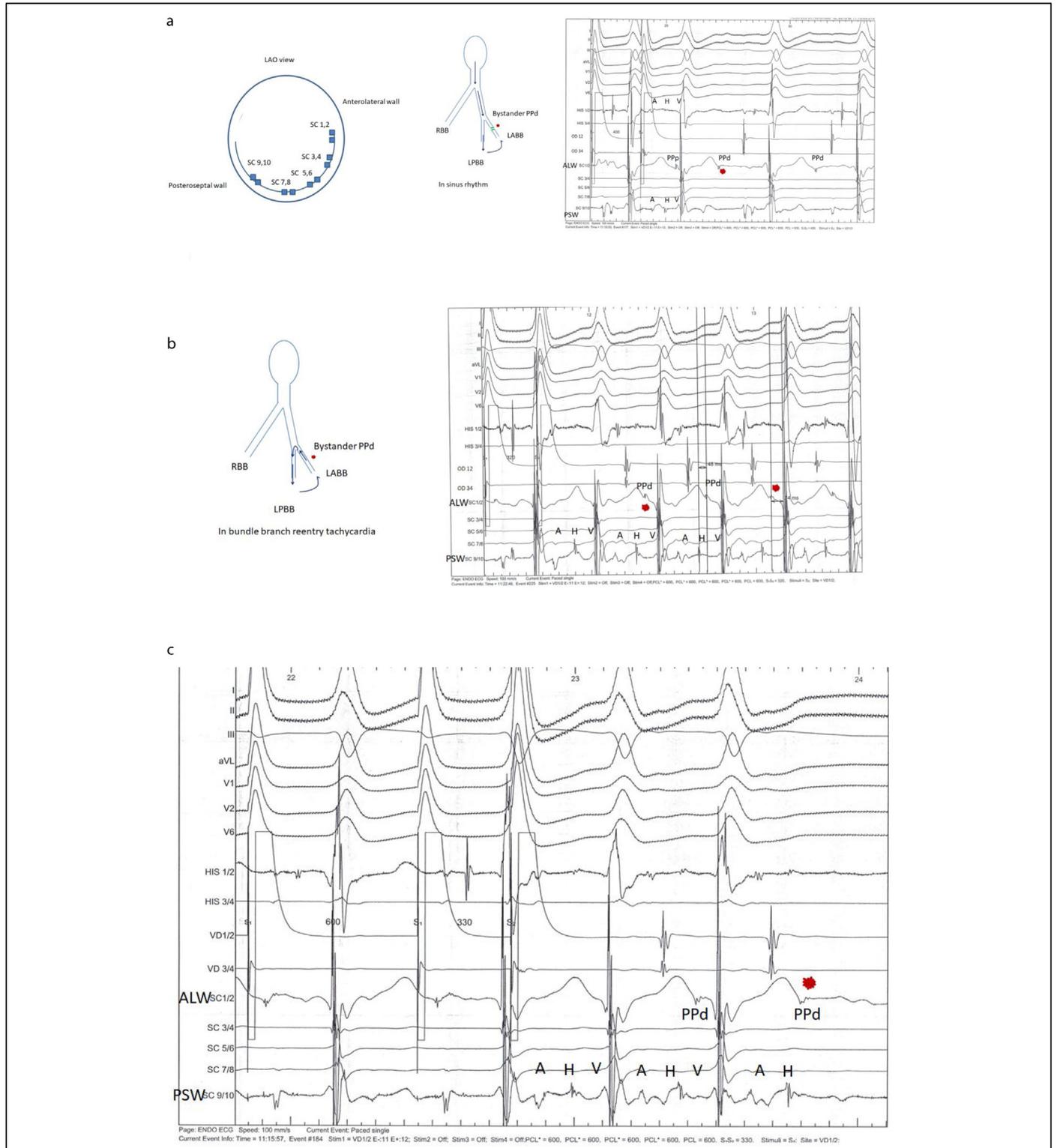


Fig. 3.

Suspected mechanism of interfascicular ventricular tachycardia (a) left anterior oblique (LAO) projections of Localizer of decapolar catheter in left ventricular in fluoroscopy. Purkinje potential distal recorded later in front of anterolateral wall. This was probably LABB(\*). (b) Intracardiac electrograms during the ventricular tachycardia: intracardiac recording and schematic representation of the hypothesized mechanism during reentrant ventricular tachycardia (VT) originating from left posterior purkinje fibers. In inter fascicular tachycardia, PPd represented LABB and was a bystander of tachycardia. This preceded 48 ms the tachycardia (c) Arrest of nonsustained VT. PPp: Purkinje Potential presystolic, PPd: Purkinje Potential diastolic, A: atrial electrocardiogram, H: his electrocardiogram, V: ventricular electrocardiogram, LABB: left anterior bundle branch, LPBB: left posterior bundle branch, RBB: right bundle branch, ALW: anterolateral wall, PSW: posteroseptal wall.

branch reentry ablation, the right bundle, did not participate in the reentrant circuit. While performing left ventricular endocardial mapping, VT was interrupted when positioning the catheter on left posterior fascicle. For these reasons, we supposed an interfascicular tachycardia but usually in interfascicular tachycardia HV in VT was shorter than in rhythm sinus. In our case, the HV during tachycardia is longer than HV during sinus rhythm. For this reason, we did not conclude it was interfascicular tachycardia but BBR-VT.

Considering structural heart disease and HPS involvement the decision was to refrain from radiofrequency ablation of conduction in the right bundle branch after coronary PCI.

Coronary angiography showed a permeable stent in anterior interventricular artery and significant stenosis in right coronary artery CDIII segment and in posterior interventricular ostium. This artery was revascularized by stent bare metal omega 2.5 × 20 mm without scintigraphy because this right coronary artery lesion was symptomatic.

A second electrophysiology study after angioplasty did not show a VT inducible like the first electrophysiology study and we did not record a late potential of fascicle branch. Electrophysiological parameters of conduction system were the same as the first electrophysiology study (AH was 62 ms, HV was 100 ms, and PRNAV 290 ms). Programmed right ventricular stimulation was performed using a maximum of two extrastimuli at two different cycle lengths 600 ms and 400 ms from the right ventricular apex and the outflow tract. Isoproterenol administration was not used to induce the VT because the patient had coronary disease and isoproterenol administration was not required before revascularization to induce the VT. Pacing from the right ventricle entrained or terminated the VT the first time. Unfortunately, we did not analyze retrograde conduction proprieties in RBBB.

The patient was discharged on conventional medical treatment. A 10-month follow-up was remarkable for absence of recurrent syncope and coronary angiography showed a permeable stent in both arteries.

## Discussion

In this case, BBR-VT was reported in a patient with preserved LVEF and with ischemic source. In fact, revascularization of culprit coronary artery disease was another means to treat VT.

BBR-VT 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 HPS is prolonged (i.e. drugs, ischemia, fibrosis), sustained reentry utilizing the bundle branches is facilitated [2].

Detailed knowledge of the underlying arrhythmogenic substrate is an essential step in the development of a successful ablation strategy in patients undergoing ablation for VT [3]. In patients with structural heart disease, VTs are commonly identified as ischemic or nonischemic in origin. The distribution of an arrhythmogenic substrate in non-ischemic cardiomyopathy (NICM) is often distinct from ischemic cardiomyopathy in that there is predilection for the perivalvular and basilar region as well as the epicardium [4]. It is feasible that patients with coronary artery disease may have a cardiomyopathy and VT origin from a separate non ischemic process. This is important because mapping and ablation guided by coronary anatomic abnormalities and/or prior infarct locational one in this group of patients can be misleading. To date, there have been limited data on VT incidence and substrate pattern in this group of patients.

Many studies [5–7] indicate that ischemia due to proximal left anterior descending artery disease or right coronary artery disease can affect the left posterior fascicle. This finding supports the validity of the location of coronary artery disease yielding the substrate for left posterior Purkinje reentry, in which the successful

ablation site was located at the posterior midseptal wall in these studies.

Earlier studies showed that subendocardial Purkinje fibers over the infarcted region remain almost structurally intact and electrophysiologically viable even after an extensive myocardial infarction [8,9]. This probably can be attributed to adequate nourishment by blood within the left ventricular cavity, abundant glycogen and sparse myofibrils, and less oxygen use than by ventricular myocardium.

In experimental studies, surviving Purkinje fiber exhibited decreased resting potential, action potential amplitude, and maximum depolarization velocity, and increased action potential duration, leading to spontaneous diastolic depolarization or reentry within the subendocardial Purkinje network of the infarct region [5,8].

The typical ablation approach in BBR-VT is to target the right bundle branch. As stated above, these patients typically have baseline His-Purkinje disease. Therefore, the incidence of significant conduction impairment necessitating permanent pacemaker placement post ablation has been reported in up to 30% of patients [10]. In our case, we did not make any ablation but dilated right coronary stenosis led to cure of the VT. In fact no VT was reproducibly induced by programmed stimulation.

## Limitations

Electrophysiology study after PCI was incomplete. There was no long-term follow-up (only 10 months).

## Conclusion

BBR-VT, although more common in NICM patients, is also seen in ICM patients. Our case was the first case of BBR-VT with the treatment of culprit coronary artery curing this tachycardia type. This new finding of treatment was preferable to right bundle branch ablation with conduction impairment risk.

## Conflict of interest

No conflict of interest.

## References

- [1] Balasundaram R, Rao HB, Kalavakolanu S, Narasimhan C. Catheter ablation of bundle branch reentrant ventricular tachycardia. *Heart Rhythm* 2008;5 (6 Suppl):S68–72.
- [2] Romero J, Santangeli P, Pathak RK, Grushko M, Briceno D, Cerrud-Rodriguez R, et al. Bundle branch reentrant ventricular tachycardia: review and case presentation. *J Interv Card Electrophysiol* 2018;52:385–93.
- [3] Aldhoon B, Tzou WS, Riley MP, Lin D, Callans DJ, Hutchinson MD, et al. Nonischemic cardiomyopathy substrate and ventricular tachycardia in the setting of coronary artery disease. *Heart Rhythm* 2013;10:1622–7.
- [4] Soejima K, Stevenson WG, Sapp JL, Selwyn AP, Couper G, Epstein LM. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: the importance of low-voltage scars. *J Am Coll Cardiol* 2004;43:1834–42.
- [5] Hayashi M, Kobayashi Y, Iwasaki YK, Morita N, Miyachi Y, Kato T, et al. Novel mechanism of postinfarction ventricular tachycardia: originating in surviving left posterior Purkinje fibers. *Heart Rhythm* 2006;3:908–18.
- [6] Frink RJ, James TN. Normal blood supply to the human his bundle and proximal bundle branches. *Circulation* 1973;47:8–18.
- [7] Papa LA, Scariato A, Gottlieb R, Duca P, Kasparian H. Coronary angiographic assessment of left posterior hemiblock. *J Electrocardiol* 1983;16:297–301.
- [8] Friedman PL, Stewart JR, Wit AL. Spontaneous and induced cardiac arrhythmias in subendocardial Purkinje fibers surviving extensive myocardial infarction in dogs. *Circ Res* 1973;33:612–26.
- [9] Fenoglio Jr JJ, Albala A, Silva FG, Friedman PL, Wit AL. Structural basis of ventricular arrhythmias in human myocardial infarction: a hypothesis. *Hum Pathol* 1976;7:547–63.
- [10] 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.