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Case Report

Two Simultaneous Ventricular Tachycardias in a Structurally Normal Heart: A Case Report and Literature Review

Muhammad Tariq Shakoor[1]*, Khawar Masood[2] and Mathias Stoenescu[3]

[1]*Hospitalist, Baptist Health Medical Center, Little Rock, AR, USA*

[2]*Cardiology Fellow, Baystate Medical Center, Springfield, MA, USA*

[3]*EP Cardiologist, Baystate Medical Center, Springfield, MA, USA*

***Corresponding Author:** Muhammad Tariq Shakoor, Hospitalist, Baptist Health Medical Center, Little Rock, AR, USA.

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Abstract

Bundle branch reentrant ventricular tachycardia and idiopathic fascicular tachycardia are rare types of ventricular tachycardia. A 71-year-old female with history of recurrent syncopal episodes thought to be secondary to tachy-brady syndrome status post pacemaker placement presented with syncope. Her device interrogation showed six episodes of ventricular tachycardia. Baseline EKG showed sinus rhythm with left bundle branch block. EP study was done which was suggestive of BBRVT with IFT. Coexistence of BBRVT and IFT has always been reported in the setting of structural heart disease. Our case is unique because the patient had no evidence of structural heart disease.

Keywords: *Bundle Branch Reentrant Ventricular Tachycardia (BBRVT); Idiopathic Fascicular Tachycardia (IFT); Ventricular Tachycardia (VT)*

Introduction

Bundle branch reentrant ventricular tachycardia (BBRVT) and idiopathic fascicular tachycardia (IFT) are rare types of ventricular tachycardia (VT) which are usually associated with structural heart disease and can present with syncope and sudden cardiac death. They have characteristic EKG changes on electrophysiology study and are both amendable to radiofrequency ablation. We report a unique case of BBRVT and IFT in a patient without any evidence of structural heart disease who underwent successful ablation for both tachycardias. To the best of our knowledge, this is the first case report of these two tachycardias in a patient without structural heart disease. We have also included a detailed literature review of these two rare tachyarrhythmias.

Case Presentation

A 71-year-old female with past medical history of atrial fibrillation and recurrent syncopal episodes which were thought to be secondary to tachy-brady syndrome status post single lead VVI pacemaker placement one month prior to presentation, now presented with another syncopal episode. Patient was watching television at home when she experienced palpitations and passed out. Patient regained consciousness before EMS arrived. Patient was in no apparent distress and was alert and oriented. Vital signs showed heart rate of 105 beats per minute (bpm), blood pressure 155/58 mm of Hg, respiratory rate 16/min and oxygen saturations of 98% on room air. Her general physical examination was essentially normal. Her basic laboratory data including CBC, BMP, magnesium, troponin I and TSH was with in normal limit. Admission EKG revealed atrial fibrillation with rapid ventricular response at 112 bpm with premature ventricular complexes, left axis deviation, QRS duration of 136 msec and QTc of 554 msec (Figure 1).

Figure 1: *Admission EKG showing atrial fibrillation with rapid ventricular response with PVCs.*

Patient subsequently had another syncopal episode in the ED and was found to be in broad complex tachycardia. Rhythm strips showed monomorphic ventricular tachycardia (VT) but the same run showed 2 different axis with the initial run of VT at 240 bpm and a subsequent one at 300 bpm. Patient was resuscitated, cardioverted and started on amiodarone drip. Patient converted to normal sinus rhythm. She was transferred to coronary care unit (CCU) for further assessment and management. On arrival to the CCU, the patient's device was interrogated which showed 6 episodes of ventricular tachycardia at rates between 270 to 380 beats per minute, all of which occurred on the day of admission. 2-D echocardiogram showed normal myocardial thickness with ejection fraction of 55% to 65%, no regional wall motion abnormalities and an elevated pulmonary artery systolic pressure at 45 - 50 mm of Hg.

An EP study was planned and performed next day. Patient's baseline EKG showed sinus rhythm, basic cycle length 934 msec, PR interval 218 msec, QRS width 122 msec, QT interval 472 msec, AH interval 118 msec, and HV interval 58 msec (Figure 2, 3).

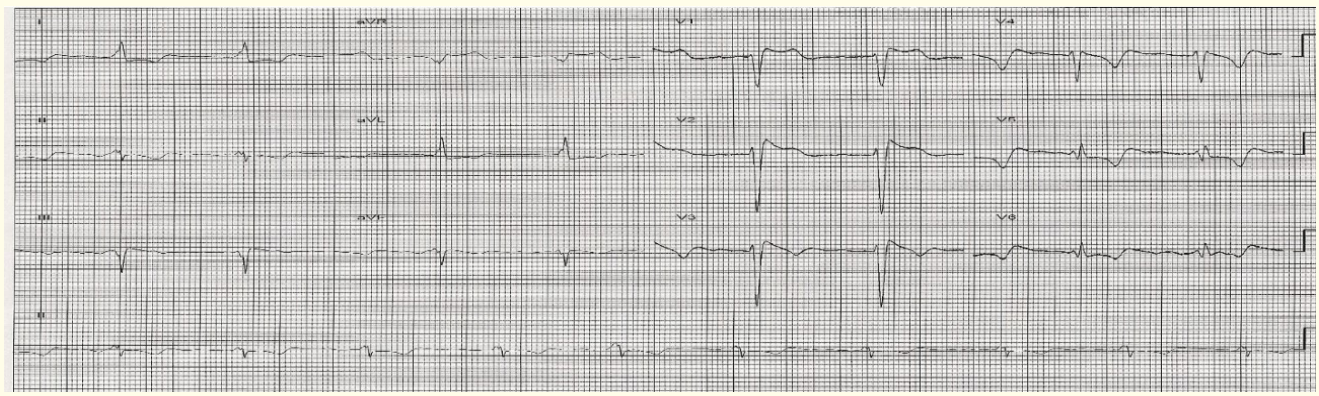

Figure 2: *Baseline EKG before procedure.*

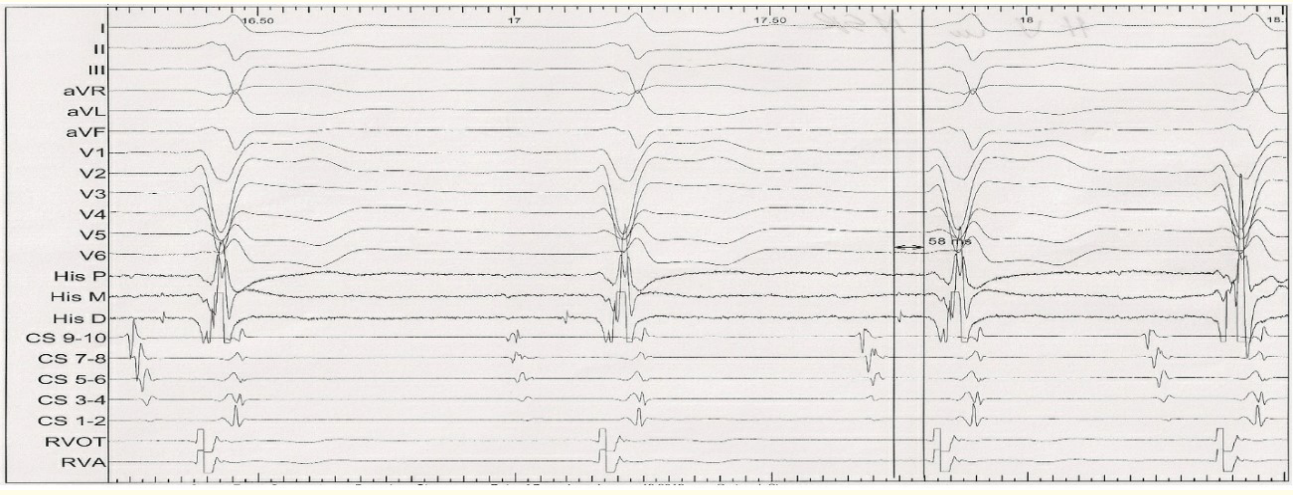

Figure 3: *HV interval 58 msec in normal sinus rhythm.*

Incremental ventricular pacing revealed absence of ventriculo-atrial conduction. Triple ventricular extra-stimuli induced wide complex tachycardia with a cycle length of 252 msec, which was terminated by ventricular pacing as the patient did not tolerate the rhythm. The tachycardia had LBBB morphology with leftward axis, suggestive of bundle branch reentrant tachycardia (Figure 4).

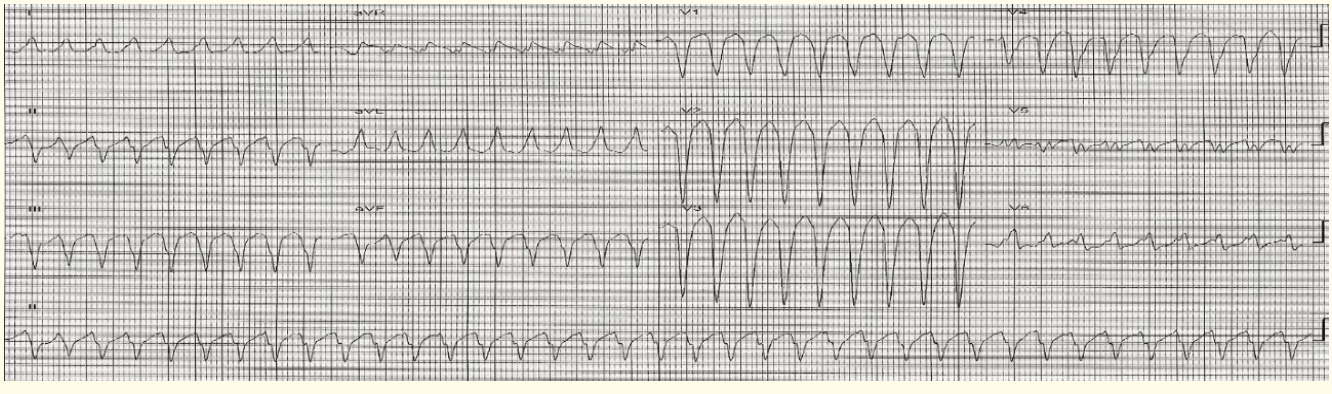

Figure 4: *Bundle branch reentrant VT.*

All beats were preceded by an H spike, with an HV interval that was longer than that during sinus rhythm (Figure 5).

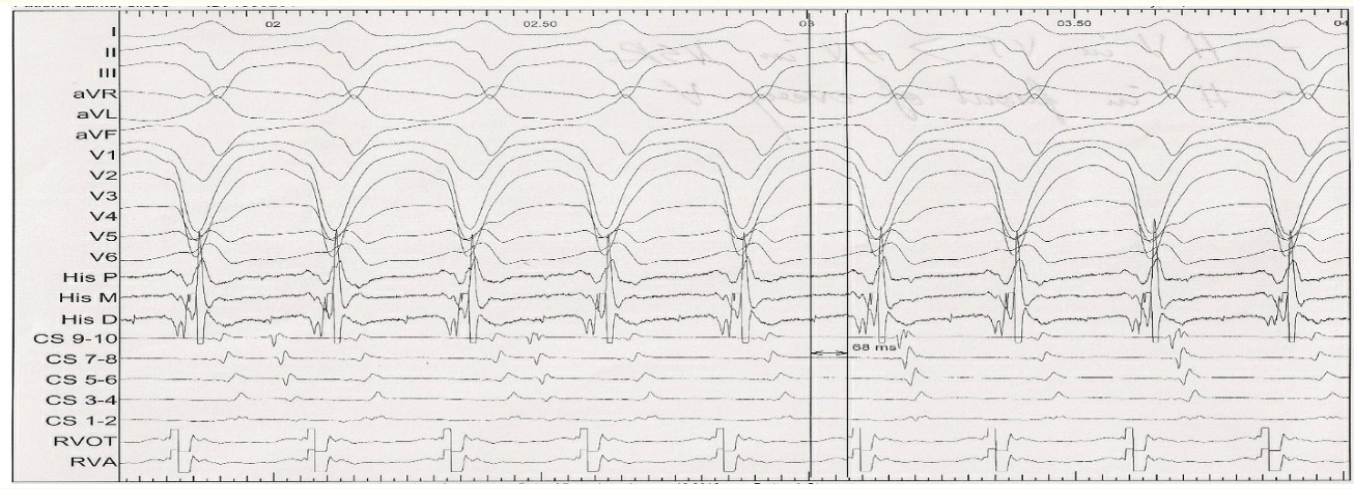

Figure 5: *HV in BBRVT longer than during NSR.*

Right bundle branch was localized and ablated. This resulted in normal sinus rhythm with right bundle branch block(RBBB) morphology (Figure 6). We were still able to induce the same tachycardia, so we did a proximal right bundle ablation which successfully stopped the BBRVT.

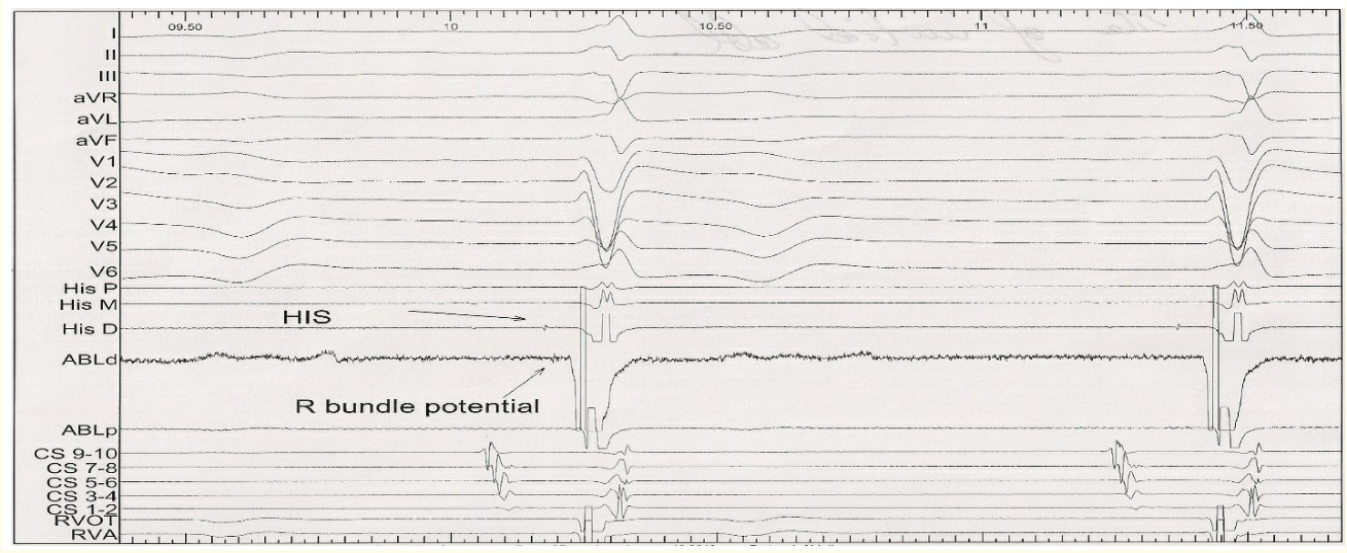

Figure 6: *Showing site of initial right bundle branch ablation; His potential preceding the RB potential.*

During observation period, a second wide QRS tachycardia with a cycle length of 290 msec was induced. There was clear AV dissociation and RBBB morphology with rightward axis. This was suggestive of idiopathic fascicular tachycardia exiting from the left anterior fascicle (Figure 7).

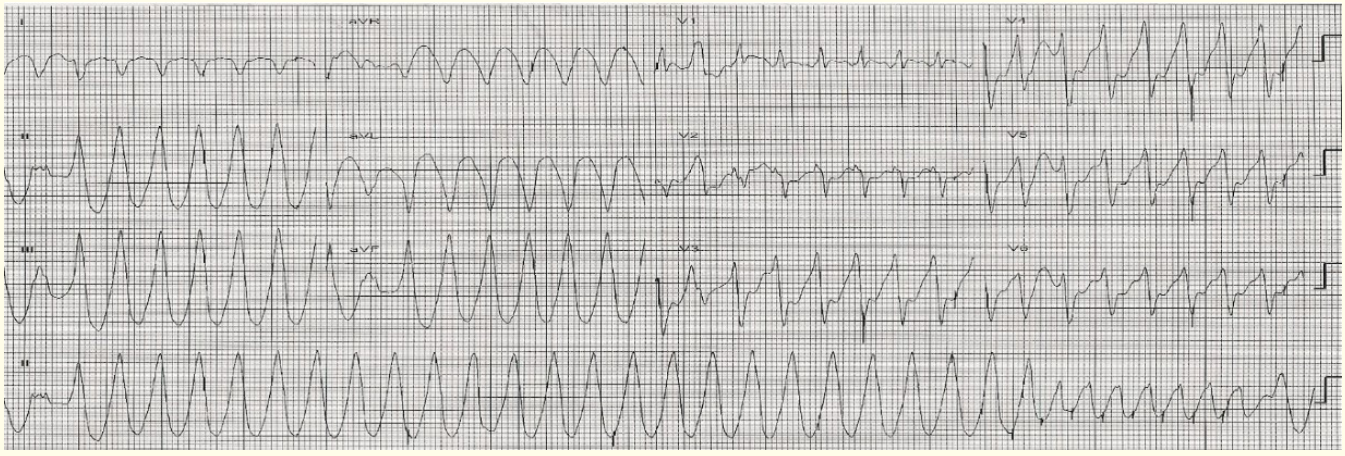

Figure 7: *Idiopathic fascicular tachycardia.*

Patient did not tolerate this rhythm and became hypotensive. Burst pacing was done to terminate the arrhythmia. Cardiac mapping could not be attempted due to patient's labile hemodynamics. We empirically ablated the mid to inferior septum which resulted in resolution of the fascicular tachycardia.

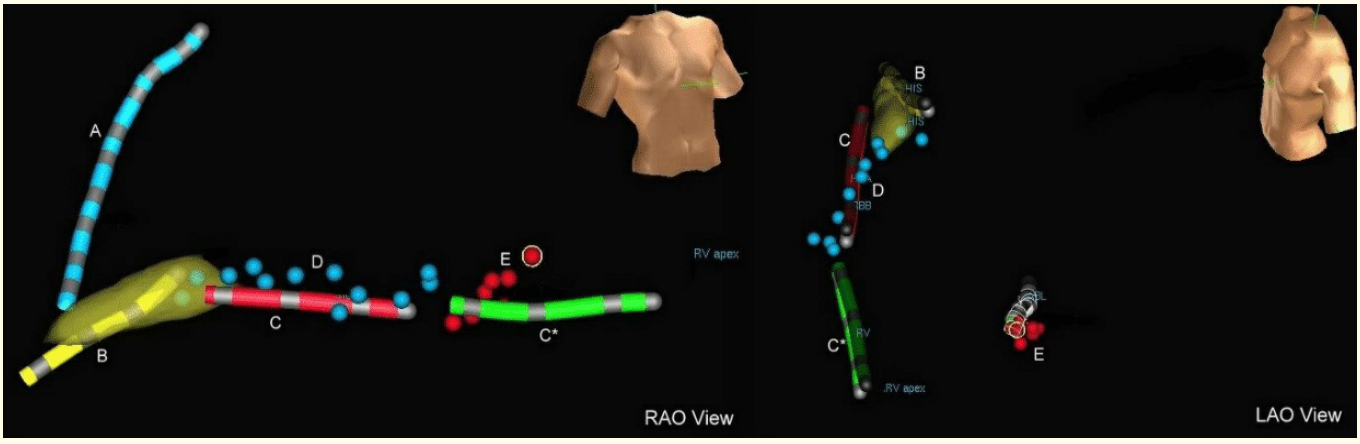

Figure 8: *3Dmapping using NavX system. 'A' representing the coronary sinus catheter; 'B' representing His catheter; 'C' and 'C*' showing RV catheter at two different positions; 'D' representing RB ablation with blue dots representing the line of ablation; 'E' representing the linear septal ablation for IFT.*

Patient was observed for 30 minutes on and off isoproterenol, neither of the tachycardia could be induced. Patient was discharged home over the next 24 hours. She has been followed up in the EP clinic over the last one year and has been doing well without any further palpitations or syncopal episodes.

Discussion

BBRVT and IFT are types of idiopathic fascicular tachycardia which use His-Purkinje system (HPS) for the tachycardia circuits. BBRVT uniquely uses right bundle for antegrade conduction and left bundle branch as retrograde conduction circuit. BBRVT can be very rapid (often > 200 bpm) and can results in presyncope, syncope and sudden cardiac death. It is usually seen in patients with underlying structural heart disease and conduction system impairment and is a relatively rare arrhythmia [1-3]. Dilated cardiomyopathy has been implicated as risk factor for BBRVT with non-ischemic cardiomyopathy more commonly associated with BBRVT than ischemic [4,5]. Mitral or aortic surgery can facilitate BBRVT due to close proximity of HPS to the valves [6]. It has also been reported in patients with Brudaga electrocardiographic pattern, Ebstein anomaly, hypertrophic cardiomyopathy and Becker muscular dystrophy and myotonic myocardial dystrophy [7-11].

Characteristic changes seen on EKG of BBRVT include LBBB morphology, prolongation of HV interval during VT when compared with sinus rhythm and changes in the H-H interval preceding the changes in the V-V interval. Typical LBBB appearance is due to antegrade conduction down the right bundle and resulting delay in depolarization of the left ventricle [12]. RBBB appearance with retrograde conduction up the RB can also occur. A narrow complex pattern at baseline has also been reported suggesting functional conduction delay as mechanism of bundle branch reentry [13]. Mean electrical axis is usually about +30º and the PR interval may be normal or prolonged. It must be understood that the presence of true bundle branch block at baseline precludes this arrhythmia but an EKG pattern of bundle branch block may not be a true marker of complete conduction block (like in our case) [14]. Treatment of choice for BBRVT is ablation of right bundle which has been compared and found superior to pharmacologic therapy [1,15]. Up to 30% of patients need placement of permanent pacemaker depending on the extent of underlying conduction system impairment. Patient with advanced heart failure may be considered for implantation of cardiovertor-defibrillator with or without cardiac resynchronization therapy [16].

IFT is also known as fascicular tachycardia or idiopathic left ventricular tachycardia is characterized by relatively narrow complexes (120 - 140 msec) and RBBB morphology. It is usually described in young adults without any structural heart disease [17]. But it has been reported with giant cell myocarditis and after ablation of right bundle for BBRVT [18,19]. It is classically terminated by calcium channel blocker like verapamil and it usually does not respond to adenosine [20,21].

There are three types of fascicular tachycardia:

- Left posterior fascicular VT which has a RBBB morphology and left axis deviation (most common form).
- Left anterior fascicular VT which has RBBB pattern and right-axis deviation (uncommon form, our patient had this form).
- Upper septal fascicular VT which has a narrow QRS and a normal axis (rare) [22].

HV interval is usually shorter by more than 40 msec when compared with sinus rhythm in patients with IFT. This is in contrast to BBRVT where the HV interval is longer with VT than sinus rhythm. In BBRVT with RBBB morphology, the His potential precedes the LB potential while in IFT the LB potential precedes the His potential [23]. A high frequency potential of short duration called Purkinje potential or P potential can be recorded in sinus rhythm and during the tachycardia in IFT. Termination of IFT with vagal maneuvers, verapamil, propranolol and adenosine have been reported [20,21]. Radiofrequency ablation remains the treatment of choice and has high success rates [24,25].

Conclusion

Coexistence of BBRVT and fascicular tachycardia has been reported in the literature before but it was in the setting of structural heart disease including dilated cardiomyopathy, myotonic dystrophy and ischemic heart disease. Our case is unique because the patient did not have any evidence of structural heart disease as evidenced by a normal echocardiogram and normal troponins with arrhythmia. It is difficult to say that the patient's syncopal episodes before presentation to our hospital were in fact secondary to sick sinus syndrome or they were episodes of VT. In conclusion BBRVT and IFT can coexist in patients without structural heart disease.

Disclosure

I, Muhammad Tariq Shakoor, being corresponding author solemnly declare that I don't have any financial relationships or conflicts of interest regarding the content herein.

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