Bundle branch reentry: A novel mechanism for sustained ventricular tachycardia in Chagas heart disease

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Introduction

Bundle branch reentrant ventricular tachycardia (BBR-VT) is a unique type of VT that involves the right and left bundle branches and the ventricular septum as components of a macroreentrant circuit, and classically has been described in individuals with cardiomyopathy and some degree of His-Purkinje system disease.[1,2]

Among patients with Chagas heart disease (ChHD), some form of His-Purkinje system disease is commonly seen, clinically manifesting as intraventricular conduction delay,[3–5] which can create the appropriate milieu for reentry within the bundles. However, BBR has not been described thus far as a potential mechanism underlying sustained VT in this clinical setting.

We herein report the first case of proven BBR-VT in a patient with ChHD. The patient presented with a rapid, hemodynamically unstable wide complex tachycardia, subsequently experienced multiple shocks from the implantable cardioverter-defibrillator, and ultimately was treated with ablation of the right bundle branch (RBB).

Case report

A 42-year-old man without any medical history had a sudden-onset episode of palpitations and collapsed. Upon presentation to the emergency room the patient was unresponsive, with hemodynamic instability owing to a wide complex tachycardia with heart rate of approximately 200 beats per minute (bpm), requiring prompt termination by a direct current shock. During subsequent hospital work-up, the patient was diagnosed with Chagas disease by 2 positive serologic tests (complement fixation / immunofluorescence tests). The baseline 12-lead electrocardiogram (ECG) during sinus rhythm showed a prolonged PR interval (240 ms) and intraventricular conduction delay resembling left bundle branch block (LBBB) with left-axis deviation and QRS duration of 130 ms. Transthoracic echocardiogram revealed severe left ventricular enlargement (diastolic diameter, 73 mm), and left ventricular ejection fraction of 25% with an inferolateral akinesia and left ventricular apical dyskinesia. Coronary angiography was normal. The patient underwent a biventricular implantable cardioverter-defibrillator (ICD) placement and was discharged on optimized treatment for heart failure and on amiodarone. Within 2 months, he began experiencing frequent shocks from the ICD caused by episodes of sustained VT at cycle lengths of 300–320 ms (rates of 187–200 bpm), and he was referred to our institution for an electrophysiologic study (EPS) and for attempting radiofrequency catheter ablation of a presumptive myocardial scar–related VT. The EPS was carried out with the patient sedated and biventricular ICD was reprogrammed to a backup pacing mode. During EPS, baseline intervals in sinus rhythm showed a normal atrial-His interval of 110 ms, a prolonged His-ventricular (HV) interval of 80 ms, and QRS duration of 130 ms (Figure 1). Programmed ventricular stimulation with up to 3 extrastimuli delivered from right ventricular (RV) apex induced a sustained monomorphic LBBB VT with a rate of 190 bpm (Figure 2A). The typical LBBB morphology of VT resembled the nonpaced sinus rhythm QRS morphology. During VT, atrioventricular (AV) dissociation was observed (Figure 2A), and early activation of the RV apex was documented; onset of VT was preceded by His-bundle activation, and the HV interval during VT was longer (95 ms) than the HV interval in sinus rhythm; and spontaneous variations in V-V intervals were preceded by similar changes in H-H intervals, indicating that His-Purkinje activation drives the VT (Figure 2B). Further maneuvers, such as entrainment from RV, were not attempted because of hemodynamic instability during VT. The aforementioned findings strongly suggested that the mechanism of the induced VT was BBR, and the RBB was targeted for ablation. Radiofrequency energy was delivered at the anterior septum of the RV, where the proximal RBB potential was recorded in sinus rhythm (RBB-V interval of 65 ms; Figure 3A), and resulted in development of right

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KEY TEACHING POINTS

- Bundle branch reentrant ventricular tachycardia (BBR-VT), a unique form of reentrant VT involving the His-Purkinje system, generally occurs in patients with structural heart disease and associated His-Purkinje conduction disease
- Patients with Chagas heart disease (ChHD) usually display evidence of His-Purkinje system disease, clinically manifesting some degree of intraventricular conduction delay, most commonly right bundle branch block, left anterior fascicular block, and, less commonly, left bundle branch block, ultimately creating the appropriate milieu for development of BBR. However, BBR-VT has never been described in patients with ChHD.
- Our case highlights BBR as a potential mechanism underlying sustained VT in ChHD, which can be cured with catheter ablation and should be considered in the differential diagnosis of ChHD patients presenting with arrhythmic events.

bundle branch block (RBBB) on surface ECG, prolongation of QRS from 130 to 150 ms (Figure 3B), increase of HV interval from 80 to 95 ms, and disappearance of the RBB potential at the ablation site (Figure 3C). An atypical RBBB ECG pattern with absent or minimal S waves in leads I and aVL, also referred to as bilateral bundle-branch delay/block, was noted after RBB ablation (Figure 3B).[6] After ablation, repeat ventricular stimulation showed no inducible ventricular arrhythmias and the patient's ICD was reprogrammed to atrial sensed biventricular pacing. After over 4 years of follow-up no arrhythmias were detected by the ICD, and ultimately the patient underwent heart transplantation owing to worsening heart failure.

Discussion

We describe a unique case of BBR-VT in a patient with ChHD, who experienced multiple shocks from a recently implanted ICD and was ultimately treated with radiofrequency ablation of the RBB. To the best of our knowledge, BBR-VT has never been described in the setting of ChHD.

Myocardial scar–reentrant VT is the most common type of sustained VT within the population of ChHD,[7,8] and the reported patient was referred to our institution for attempting catheter ablation of a presumptive scar-related VT. Unexpectedly, during programmed ventricular stimulation a sustained LBBB-type wide QRS tachycardia resembling the nonpaced sinus rhythm QRS morphology was induced and diagnosis of BBR-VT was established with usual criteria[1,2]: (1) the 12-lead ECG morphology of the VT exhibited a typical LBBB pattern, and AV dissociation was observed; (2) onset of VT was preceded by His-bundle activation, and the H-V interval during VT was longer than the H-V interval in sinus rhythm; (3) spontaneous variations in V-V intervals were preceded by similar change in H-H

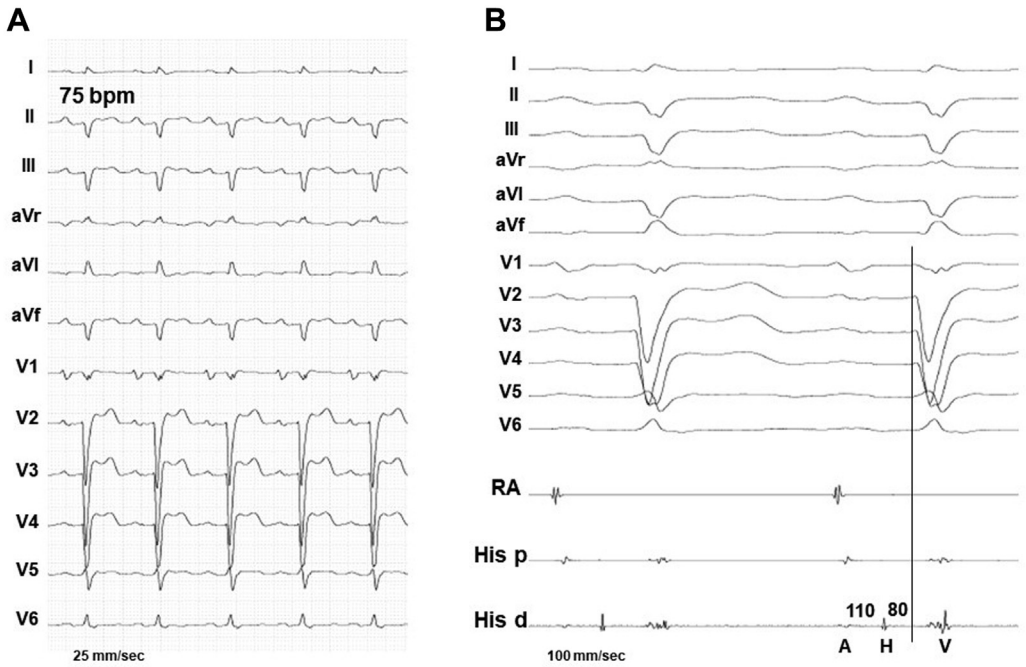

Figure 1 **A:** Twelve-lead electrocardiogram (ECG) showing sinus rhythm with prolonged PR interval (240 ms) and intraventricular conduction delay resembling left bundle branch block with left axis deviation. **B:** Baseline intracardiac recordings showing a normal atrial-His (AH) interval of 110 ms, a prolonged His-ventricular (HV) interval of 80 ms, and QRS duration of 130 ms. Displayed from top to bottom are ECG leads and intracardiac recordings from the right atrium (RA) and His-bundle proximal (His p) and distal (His d).