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Case Report

Bundle branch reentry: A rare mechanism of ventricular tachycardia in endomyocardial fibrosis, without ventricular dilation

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ARTICLE INFO

Article history:
Received 20 December 2015
Accepted 7 February 2016
Available online 14 April 2016

Keywords:
Endomyocardial fibrosis
Bundle branch reentry
Ventricular tachycardia

ABSTRACT

Introduction: Bundle branch reentry as a mechanism of ventricular tachycardia (VT) in endomyocardial fibrosis (EMF) is not described.

Case report: A 52-year-old woman with left ventricular (LV) EMF had VT needing cardioversion. She had mitral regurgitation and left bundle branch block, but no LV dilation or heart failure. During electrophysiological study, clinical VT could be easily induced, and it was confirmed to be bundle branch reentrant VT (BBRVT). She was treated with ablation of the right bundle branch.

Conclusion: BBRVT can occur in EMF even without cardiac dilatation. Its recognition is important, as radiofrequency ablation can be curative.

1. Introduction

Ventricular tachycardia (VT) is rarely encountered in endomyocardial fibrosis (EMF) and only isolated case reports exist.[1,2] Bundle branch reentrant VT (BBRVT) is uncommon in nondilated hearts. BBRVT as a mechanism of VT in EMF is unknown. We describe a case of BBRVT with left ventricular (LV) EMF and a nondilated LV as the underlying substrate.

2. Case report

A 52-year-old postmenopausal diabetic woman had long standing exertional dyspnea with recent worsening, and had moderate mitral regurgitation (MR) but no heart failure on evaluation. She had a monomorphic VT of left bundle branch block (LBBB) morphology, at 187 min^{-1} with syncope, which was cardioverted with 200 J biphasic DC shock. She also

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http://dx.doi.org/10.1016/j.ihj.2016.02.005

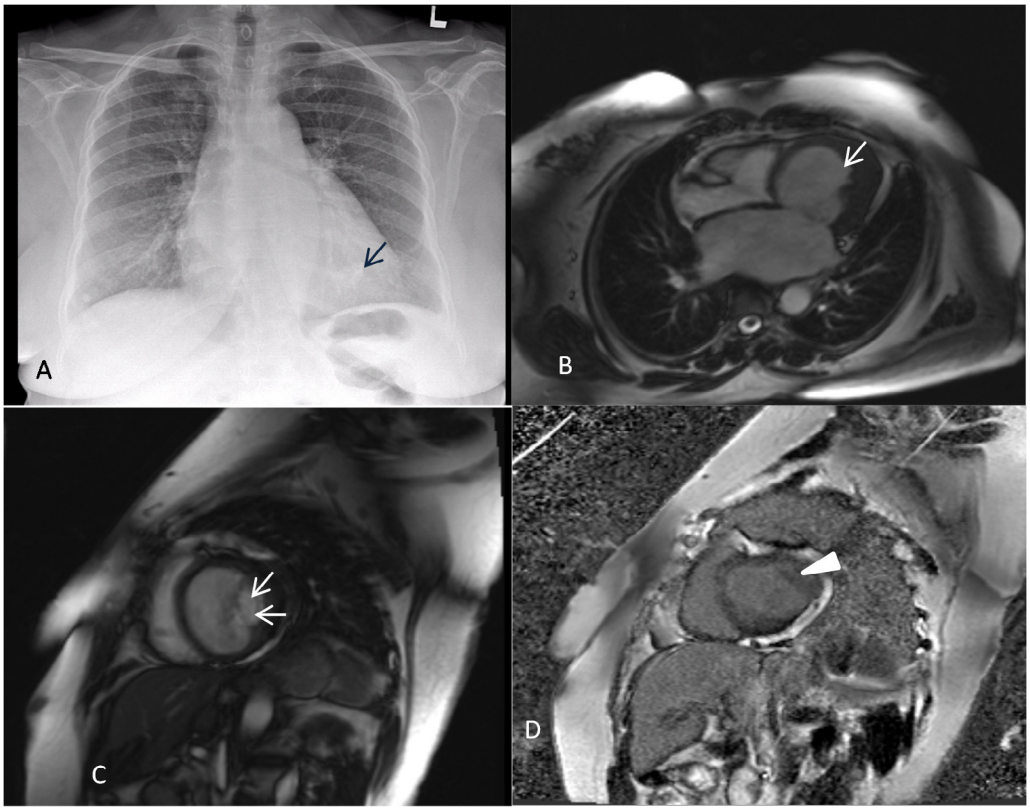

Fig. 1 – Chest X-ray PA view (A): Calcific spot (black arrow); Cardiac MRI: (B,C) Cine CMR imaging – shows normal-sized LV with obliteration of apex (white arrow in B) and calcified thrombus (double arrow in C). LA dilatation, and normal RA/RV noted. (D) Postcontrast imaging (PSIR sequences at 20 min) – shows subendocardial gadolinium enhancement (arrow head) of the same regions.

has a history of paroxysmal atrial fibrillation (AF). Chest X-ray showed calcification of cardiac apex. Baseline electrocardiogram (ECG) showed sinus rhythm, PR interval of 180 ms, LBBB, and QRS duration of 150 ms. Echocardiogram showed normal-sized left ventricle with ejection fraction (EF) 57%. There was calcification of LV endocardium at mid and apical levels. Right ventricle (RV) was normal. She had moderate (3+) MR. Cardiac magnetic resonance imaging (CMR) confirmed LV apical obliteration with chronic calcified thrombus and underlying fibrosis showing gadolinium enhancement (Fig. 1). LV apex was obliterated with retraction toward base. Cine sequences showed global LV hypokinesia. There was plastering of posterior mitral leaflet to the posterolateral LV wall, with moderate MR, left atrial enlargement and subendocardial enhancement. Hemogram, and renal and liver functions were normal. Coronary angiogram showed normal coronaries. LV angiogram showed EF of 55%, calcific and obliterated apex, and moderate MR. RV angiogram was normal. Cath study showed normal filling pressures and mild pulmonary arterial (PA) hypertension (mean PA pressure – 23 mm Hg). Electrophysiology study (Fig. 2) was done with three standard quadripolar diagnostic catheters that were placed in high right atrium, His bundle region (His), and right ventricular apex. Baseline HV interval was prolonged (72 ms). Ventricular pacing showed central decremental VA conduction with VA Wenkebach block occurring at 600 ms. A single programmed electrical stimulation (PES) from RV apex induced clinical VT. Tachycardia was initiated with VH jump and HV during tachycardia was more than that during sinus rhythm. There was VA dissociation and the QRS morphology was similar to clinical tachycardia (LBBB with left axis deviation). HV interval was 102 ms during VT. Timed His-refractory ventricular extrastimulus showed advancement of His and V with resetting of tachycardia. Entrainment from RV apex showed a short post-pacing interval with concealed fusion. All the features were consistent with a counter-clockwise bundle branch reentrant tachycardia (BBR-VT). Left bundle potentials could not be recorded and patient was having basal LBBB. Right bundle branch (RBB) mapping during sinus rhythm showed RB-V duration of 30 ms and RV apex EGM was earlier than surface QRS. Right Bundle ablation was done in sinus rhythm using RFA (4 mm tip Bard stinger D curve catheter, 60 W, 55 degree, temperature control; IBI 1500T RF generator), and resulted in prolongation of HV to 92 ms, and change in QRS. Postablation, there was no VT inducible, but atrial tachycardia could be induced. The atrial tachycardia appeared to be originating from the left atrium and subsided spontaneously in 15 s. Precaution was taken for emergency pacing, as development of complete heart block was likely with RBB ablation. She underwent a single-chamber (VVI) permanent pacemaker in view of prolonged HV interval, after 4 days. The lead parameters were normal (R wave 15 mV, threshold 0.75 V @0.4 ms, impedance 513 Ω). At 18 months of follow-up, the device interrogation

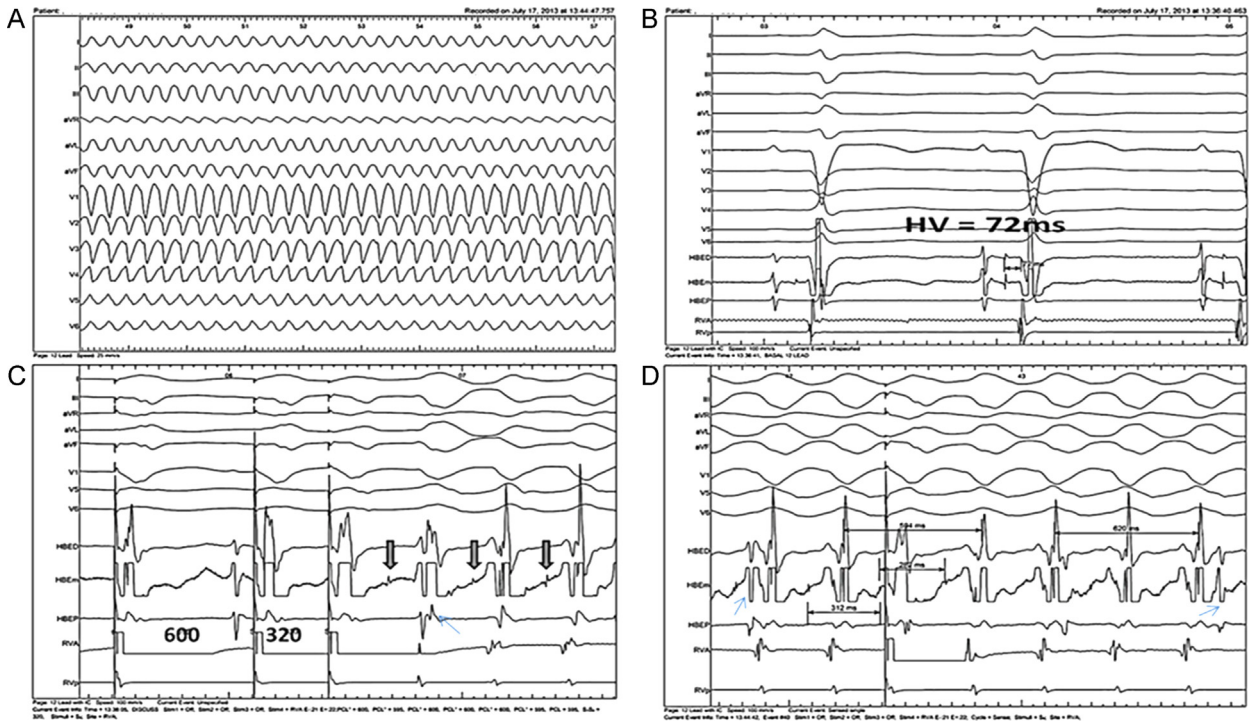

Fig. 2 – HBEp, HBEm, and HBEd represent proximal, mid, and distal His bundle electrogram, respectively, RVA and RVp represent RV distal and proximal electrograms, respectively. (A) Is at paper speed of 25 mm/s while other tracings are at 100 mm/s. (A) 12-lead ECG of the VT of LBBB morphology and left superior frontal axis. (B) The baseline intracardiac signals. Sinus cycle 784 ms, AH interval 116 ms, and HV interval of 72 ms were observed. (C) Induction of VT with PES at 600–320 ms from RV apex. The thick arrows mark the His potential, which is preceding every V with a HV interval of 102 ms. The thin arrows indicate atrial signal that is dissociated from the V. (D) Advancement of H by a single extrastimulus from RV apex. Tachycardia cycle length is 310 ms. The interval encompassing the extrastimulus is 594 ms showing the advancement of V. This is preceded by advancement of H as marked in the figure. The H–H interval during tachycardia is 312 ms and has shortened to 282 ms with the extrastimulus. The thin arrows indicate atrial signals that are dissociated from V. Abbreviations used are same as in previous figures.

showed no episodes of ventricular high rate events. But, during this period, she had an episode of AF with fast ventricular rate necessitating cardioversion. Pacemaker threshold and other parameters have remained in normal range.

3. Discussion

BBRVT usually occurs in patients with structural heart disease like cardiac dilation due to DCM, coronary artery disease, and valvular heart disease.[3] EMF as the underlying cause of BBRVT has not been reported so far.

Prolongation of baseline HV interval, conduction delay in His-Purkinje system without complete bundle branch block, and intramyocardial conduction delay are the factors facilitating sustained clinical BBRVT.[4] The longer distance between the bundle branches in a dilated heart also favor BBRVT in dilated hearts. However, BBRVT has also been reported in nondilated hearts with structural heart disease and even with structurally normal hearts but with infra-Hisian conduction delay.

Our patient had a normal-sized left ventricle with EMF. The RV was not involved with EMF. She had infra-Hisian conduction system involvement as evident in the LBBB and prolonged HV interval. The lesions in EMF consist of fibrosis of the endocardium and underlying myocardium and results in valve regurgitation, restrictive hemodynamics, and heart failure. The involvement of conduction system of the heart by the fibrotic process may be responsible for the conduction delay in this case. Atrial arrhythmias in EMF are common and studied.[5] The atrial dilation may have formed the substrate for atrial arrhythmias in our patient. It is interesting that in spite of the fibrous involvement of myocardium, VT is extremely uncommon in EMF. Occurrence of VT is not described in any of the large series, but only in a few case reports, on this condition.[1,2]

BBRVT may be a mechanism of VT in patients with EMF even when there is no cardiac dilatation. The importance of recognition of BBRVT lies in the fact that RBB ablation has a curative role. The procedure is simple and long-term results are excellent.[3]

Conflicts of interest

The authors have none to declare.

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