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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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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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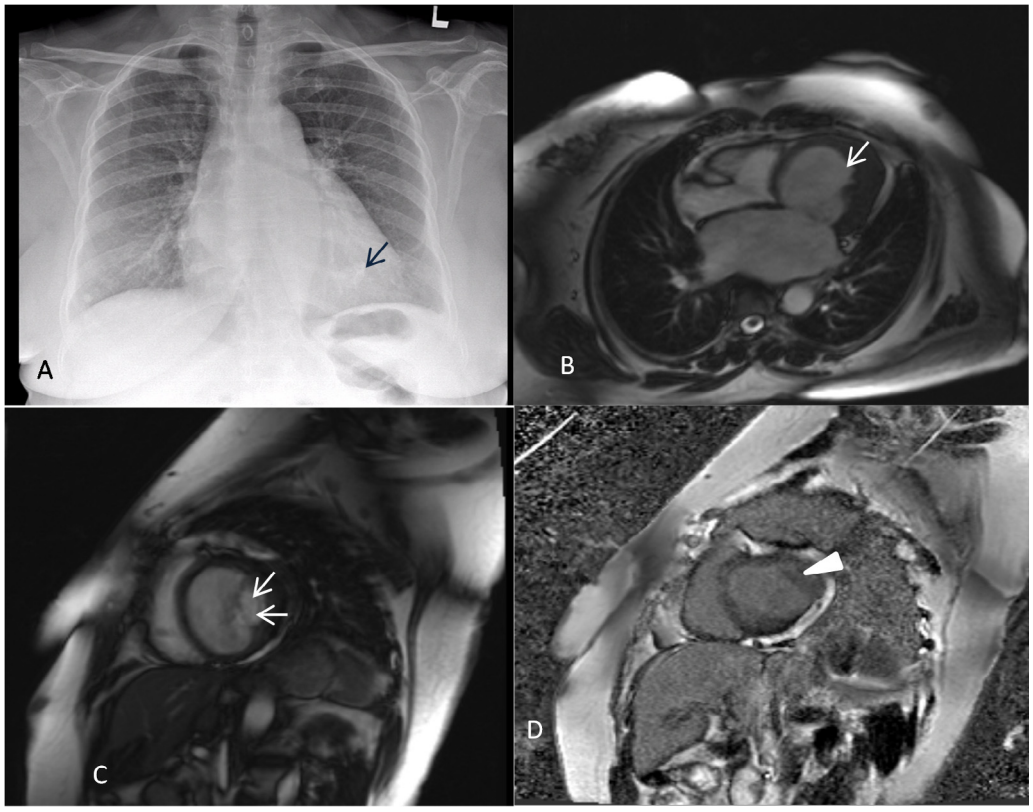

Fig. 1 – Chest X-ray PA view (A): Calcific spot (black arrow); Cardiac MRI: (B,C) Cine CMR imaging – shows normal-sized LV with obliteration of apex (white arrow in B) and calcified thrombus (double arrow in C). LA dilatation, and normal RA/RV noted. (D) Postcontrast imaging (PSIR sequences at 20 min)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

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