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Extended bipolar left ventricular pacing as a possible therapy for late electrical storm induced by cardiac resynchronization therapy

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Abstract Although cardiac resynchronization therapy (CRT) has become a well-established treatment option for patients with drug-refractory severe systolic heart failure, there has been some evidence of adverse proarrhythmic events. We report a case of a patient with ischemic cardiomyopathy who underwent CRT with a defibrillator for primary prevention of sudden cardiac death. Two years after the implantation, the patient presented with electrical storm, which was completely terminated by modifying the left ventricular (LV) pacing configuration from true to extended bipolar LV pacing. We discuss the possible pathophysiologic mechanisms of this phenomenon through a brief review of the literature on CRT-induced proarrhythmia.

Keywords: CRT; Proarrhythmia; VT storm

Introduction

Over the last years cardiac resynchronization therapy (CRT) has become an established treatment for patients with severe systolic heart failure, refractory to optimal medical therapy. Several multicenter trials showed clear benefits of CRT in reducing mortality and morbidity and improving symptoms, exercise tolerance, left ventricular (LV) reverse remodeling and quality of life [1]. However, despite the hemodynamic and antiarrhythmic benefits of cardiac resynchronization therapy, there has been evidence of adverse proarrhythmic events [2,3]. We report the case of a patient with severe ischemic cardiomyopathy who developed VT storm as a late pro-arrhythmic effect of true bipolar LV pacing. The incessant VT resolved completely with extended bipolar LV pacing.

Case presentation

An 84-year-old man was admitted to our hospital with drug-refractory congestive heart failure (CHF). He had a previous medical history of ischemic cardiomyopathy, percutaneous coronary intervention (PCI), diabetes and paroxysmal atrial fibrillation. His electrocardiogram (ECG) revealed sinus rhythm and a left bundle branch block (LBBB) QRS morphology with a duration of 130 msec. The patient had a functional status of New York Heart Association (NYHA) class III and severe LV dysfunction with an ejection fraction (LVEF) of 30%. He had no history of ventricular tachycardia (VT) or resuscitated sudden cardiac death.

The patient underwent cardiac resynchronization therapy device with a defibrillator (CRT-D) (Medtronic Model DTBA2QQ Viva Quad XT CRT-D). An active-fixation bipolar atrial lead (Medtronic CapSureFix Novus MRI 5076 52 cm) was placed in the right atrial appendage, an active-fixation bipolar DF4 right ventricular defibrillator lead (Medtronic Sprint Quattro Secure 6947 M MRI 62 cm) was placed in the right ventricular apex and a quadripolar LV lead (Medtronic Attain Performa 4598 88 cm IS4) was implanted in a posterolateral vein of the coronary sinus. The procedure was uncomplicated and good pacing parameters of all the leads were achieved. The device was programmed to track the atrium and biventricularly pace the ventricle with an atrio-ventricular (AV) delay of 130 msec, a ventriculo-ventricular (VV) delay of 50 msec with LV preactivation, using true bipolar LV distal pacing (distal-1 to mid-2: D_1-M_2) with three zones programmed: i) VT(1) rate 150 bpm for monitoring only, ii) VT(2) rate 188 bpm, therapies: ATP 3 x burst, 3 x ramp, 4 × 35 J, iii) VF zone rate 222 bpm, therapies: ATP during charge, 6 × 35 J.

After a 13-month follow-up the patient remained arrhythmia-free and biventricularly paced. Unfortunately, he then developed persistent atrial fibrillation with a rapid

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ventricular response despite optimal medical therapy. In view of the fast conducted atrial fibrillation and the resulting low biventricular pacing percentage, the patient underwent atrioventricular junction ablation.

Two years after his CRT-D implantation, he was admitted to hospital with slow incessant monomorphic VT at a rate of 130 bpm (Fig. 1). The chest x-ray film confirmed stable lead positions. The device interrogation confirmed lead parameters to be stable and within normal limits. The transthoracic echocardiogram (TTE) revealed no improvement in LV systolic function compared to the TTE before the CRT implantation. After multiple attempts at manual ATP therapy, the VT was eventually terminated and the patient was discharged on high dose oral amiodarone and the VT zone was programmed to 122 bpm. However, 48 h later the patient was readmitted with multiple episodes of sustained and non-sustained VT which did not respond to intravenous amiodarone or manual ATP therapies (Fig. 1B and C). We therefore decided to proceed to VT ablation. During the device reprogramming to facilitate the VT ablation, we observed that cessation of biventricular pacing (by programming only to RV pacing) completely suppressed VT. A resumption of LV pacing resulted in immediate VT recurrence. This pattern was also observed when LV pacing was configured from distal-1 to mid-3 (D_1-M_3) with VT reactivation and immediately suppressed when LV pacing was stopped. This configuration was repeated multiple times resulting each time immediately to VT recurrence. At this point LV pacing induced proarrhythmia was considered as the trigger factor of the VT storm. Further configurations were assessed with pacing from the proximal LV electrodes instead of the distal electrodes. Specifically, the device was reprogrammed from true distal LV bipolar pacing (D_1-M_2) to extended LV bipolar pacing (proximal 4 to RV coil: P_4-RV) with good LV lead parameters and complete resolution of the VT. Having eliminated VT, we decided to withhold VT ablation due to his comorbidities and his high risk for general anesthesia. No further complications were noted over 2-month follow-up.

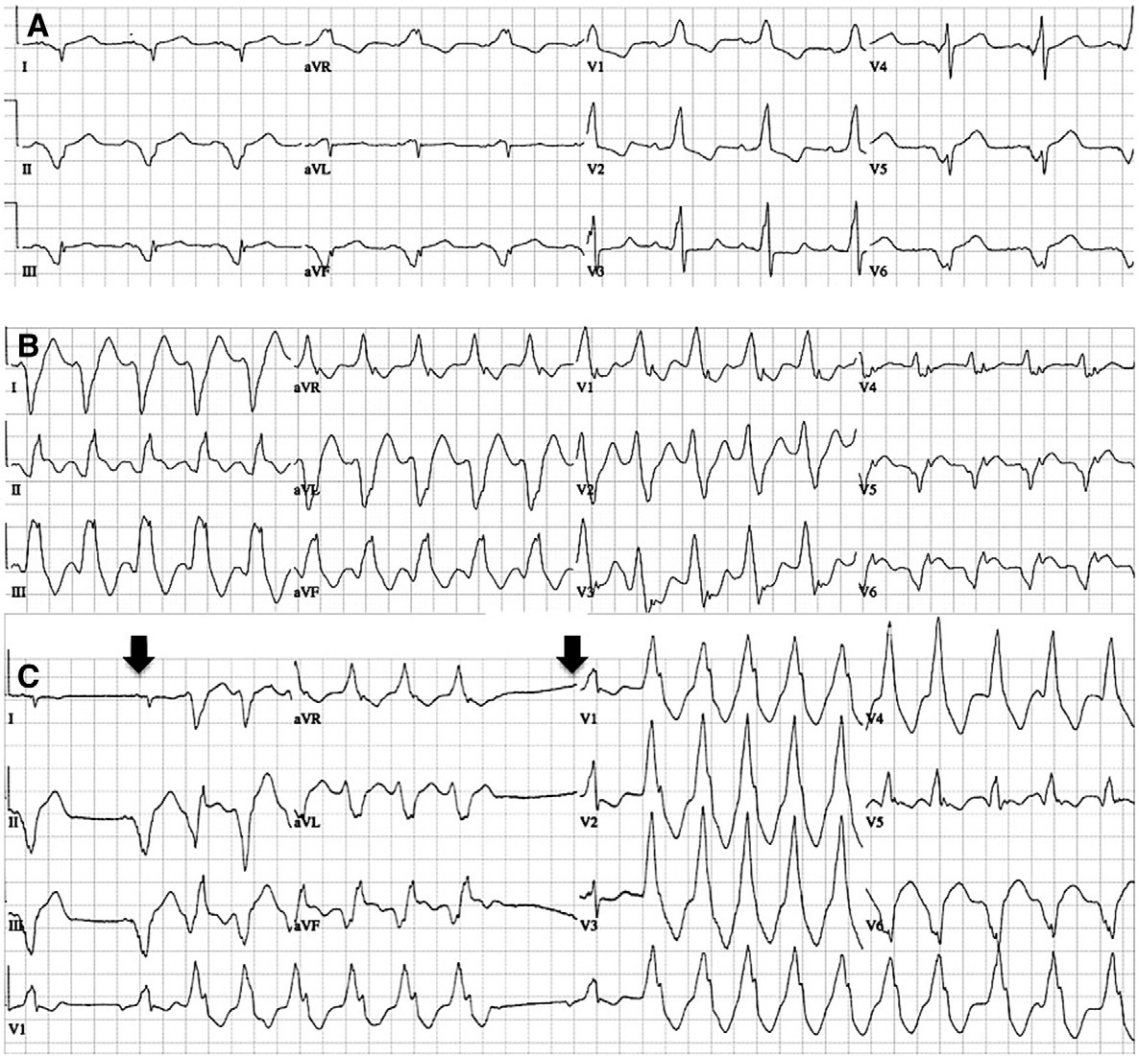

Fig. 1. Twelve-lead electrocardiogram (panel A) during LV pacing with an AV delay of 130 ms and VV delay of 50 ms with LV preactivation. Panel B shows sustained VT and panel C nonsustained VT triggered by LV pacing (arrows) during VT storm 2 years after CRT-D implantation. LV = left ventricular, AV = atrio-ventricular, VV = ventriculo-ventricular, RV = right ventricle, VT = ventricular tachycardia.