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Case Report

A case of premature ventricular contractions, ventricular tachycardia, and arrhythmic storm induced by right ventricular pacing during cardiac resynchronization therapy: Electrophysiological mechanism and catheter ablation



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ABSTRACT

A 77-year-old man with ischemic cardiomyopathy and a cardiac resynchronization therapy-defibrillator (CRT-D) device came to our attention due to incessant ventricular tachycardia and multiple implantable cardioverter defibrillator (ICD) shocks. An electrocardiogram showed non-sustained monomorphic ventricular tachycardias (NSVTs) constantly occurring after each biventricular stimulation. During an electrophysiological study, NSVTs reproducibly recurred only after right ventricular (RV) pacing; LV pacing did not induce any NSVTs.

The activation map was consistent with a localized reentry at the interventricular septum, and a double exit; at the LV exit site, a single radiofrequency energy application immediately interrupted the occurrence of the NSVTs.

Current evidence supports LV pacing to be pro-arrhythmogenic in few CRT patients. This unusual case shows that RV pacing during CRT could produce frequent ventricular arrhythmias and arrhythmic storm. Catheter ablation can be considered an effective therapeutic option, especially when CRT maintenance is highly advisable.

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1. Introduction

Left ventricular pacing (LVP) has been recognized as a rare trigger of ventricular arrhythmias after cardiac resynchronization therapy (CRT) [1]. In general, such events arise early after implantation, while a late occurrence has been reported only once, in a patient with ischemic cardiomyopathy suffering from LVP-induced ventricular tachycardia (VT) storm 7 years after CRT initiation [2].

The pathophysiology of LVP-induced VTs remains controversial. The leading theory relies on enhanced transmural dispersion of repolarization during LVP [3]; recently, a reentrant mechanism involving an epicardial scar was demonstrated in a small group of patients with early electrical storm after CRT onset, in which catheter ablation resulted in complete resolution of the condition and safe CRT resumption [4].

We describe a case of right-ventricular pacing (RVP)-induced VTs along with the possible mechanism of the arrhythmia, eventually

causing an arrhythmic storm, in a patient with previous myocardial infarction and CRT.

2. Case report

A 77-year-old man with ischemic dilated cardiomyopathy and a previous anterior myocardial infarction came to our attention due to incessant ventricular tachycardia and multiple implantable cardioverter defibrillator (ICD) shocks.

The patient was affected by dilated cardiomyopathy with an apical aneurysm, severe left-ventricular (LV) systolic impairment, a complete left bundle branch block (LBBB), a chronic total occlusion of the left descending coronary artery, and severe stenosis of the right coronary artery. An atrioventricular defibrillator had been implanted in 2006 following resuscitation from a cardiac arrest, with a passive fixation, dual-coil implantable cardioverter defibrillator (ICD) lead (Sprint Fidelis[®], Medtronic Inc., Minneapolis, MN, US) positioned at the mid-apical right ventricular (RV) septum.

The subsequent implant follow-up was uneventful. Elective replacement of the device was performed in 2009 and again in March 2014; the leads were performing well and were regularly

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connected to the new device (VIVA XT CRT-D, Medtronic Inc., Minneapolis, MN, US); the ICD programming was left unchanged.

A few weeks after the last replacement, multiple ICD shocks occurred. A device interrogation revealed non-sustained fast monomorphic ventricular tachycardias (NSVTs) incessantly recurring after a few biventricular paced beats; ICD therapies with anti-tachycardia pacing (ATP) and direct current shocks (DCSs) were either ineffective or inappropriately delivered after spontaneous interruption.

After the initial evaluation, which excluded acute ischemia, electrolyte imbalance, and other common transient precipitating factors, stabilization was achieved with sedation and intravenous amiodarone. Subsequently, a recurrence of slower, short NSVTs occurred. Of note, each biventricular stimulation was reproducibly followed by three monomorphic ventricular complexes (Fig. 1A).

The NSVTs showed an LBBB morphology with superior and right axis deviation and negative concordance in the precordial leads, which is indicative of inferoapical septal origin. Suspecting a pacing-induced triggered mechanism, biventricular pacing (BiVP) was temporarily suspended, which was followed by the immediate cessation of the NSVTs. Subsequently, RVP only, LVP only, or BiVP were unable to reinduce the NSVTs.

A VT ablation procedure was scheduled. The procedure was conducted in a fasting state under general anesthesia with mechanical ventilation. The presence of intraventricular thrombus was excluded using echocardiography. The pre-specified procedural end-points were the non-inducibility of sustained monomorphic VTs and substrate modification by means of the elimination of late potentials (LPs). As previously described [5], an LP was defined as an isolated bipolar potential occurring after the end of the surface QRS (either spontaneous or paced), which was separated from the main ventricular potential by an isoelectric tract.

A quadripolar mapping catheter was placed along the RV septum, and a 3.5 mm bidirectional open irrigated mapping catheter (ThermoCool SF[®], Biosense Webster, Johnson's & Johnson's, Diamond Bar, CA, US) was inserted retrogradely into the LV. An electroanatomical substrate map created using the CARTO3[®] system (Biosense Webster, Johnson's & Johnson's, Diamond Bar, CA, US) was obtained during constant sinus-triggered LVP (via the

LV device lead). Pacing was necessary due to the absence of a spontaneous ventricular rhythm; LVP was preferred to enhance delayed septal activation. Thresholds for the bipolar substrate map were set at 1.5 mV and 0.5 mV to discriminate respectively healthy tissue and “dense” scar, as is typically set up for ventricular substrate mapping.

A large area of the anterior, septal, and apical scar was reconstructed without evident LPs. The RV substrate map revealed low amplitude potentials at the apical septum, again without LPs (Fig. 2a, b).

Inducibility was tested with programmed LV stimulation first, without the induction of any sustained arrhythmias. RV pacing was then initiated, but the induction protocol was immediately stopped since a regular recurrence of the NSVTs following one or two paced complexes appeared, with the same morphology that had been encountered clinically.

Changing the site of RV septal pacing did not influence the NSVT occurrence or morphology, despite a different interval between the spike and NSVT onset; reproducibility was demonstrated by temporary switching to LV stimulation (Fig. 1B). Notably, the ICD bipolar morphology (tip-ring) of the NSVTs closely matched the clinical VTs (see Supplemental material, SM1).

A biventricular activation map of the induced NSVTs was then performed, which confirmed a centrifugal activation originating from the infero-apical interventricular septum to both ventricles (see Fig. 2c) without significant anticipation on both sides of the septum. The best pacemap QRS concordance was 11/12 on both sides of the septum (a discordant V1 is evident; see Supplemental material, SM2); a unipolar electrogram was also suboptimal for ectopic origin at the same sites.

Initial ablation attempts of a putative intramural source of NSVTs, where the best pacemap or best unipolar electrograms were present, failed to interrupt the arrhythmias, despite the high power and irrigation rate on both the RV and the LV sides of the septum.

Further careful mapping at the LV exit allowed to find a single site showing a late potential after a paced RV complex, followed by a low-amplitude, highly fragmented diastolic potential occurring 90 ms before the NSVT onset (Fig. 3). At this site, a single radiofrequency (RF) energy application interrupted the occurrence of

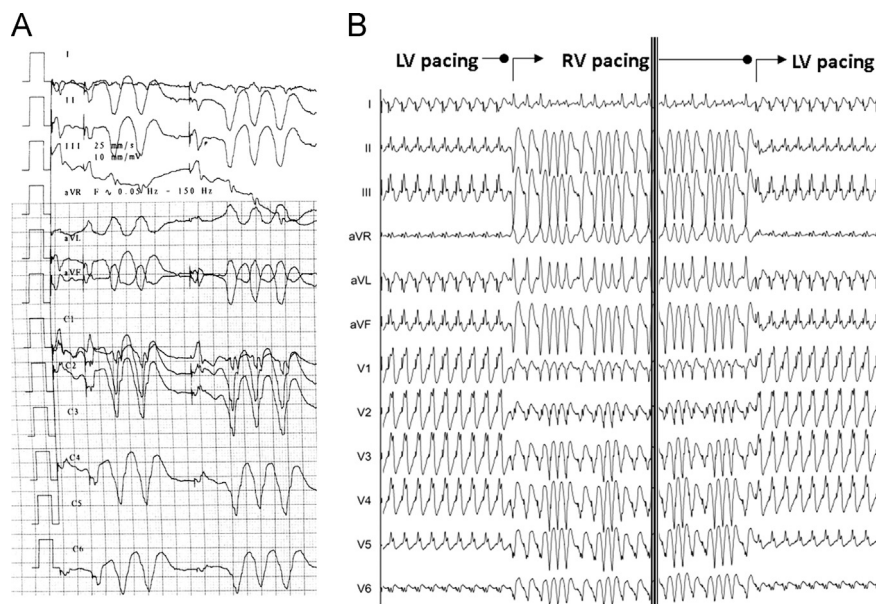


Fig. 1. (A) A 12-lead surface electrocardiogram showing biventricular pacing-induced non-sustained ventricular tachycardias (NSVTs). (B) Demonstration of the NSVTs' dependency on right ventricular pacing (RVP) during an electrophysiological study. The shift from left ventricular pacing (LVP) to RVP is followed by the immediate induction of NSVTs; the NSVTs are suddenly abolished after the pacing site has been shifted again to LVP.

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