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Authors

Feng, Zekun
Marcus, Gregory M
Badhwar, Nitish

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Case Report

Mechanically induced electrical storm as a complication of cardiac resynchronization therapy: A case report☆

Zekun Feng [a,*], Gregory M. Marcus [b], Nitish Badhwar [c]

[a] California Pacific Medical Center, San Francisco, CA, USA
[b] University of California, San Francisco Medical Center, San Francisco, CA, USA
[c] Stanford Medical Center, Palo Alto, CA, USA

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ABSTRACT

Background: Cardiac resynchronization therapy (CRT) has been shown to improve both the functional status and mortality of heart failure patients with left bundle branch block. Multiple recent studies suggest several mechanisms for proarrhythmia associated with CRT device.
Case summary: A 51-year-old male with symptomatic non-ischemic cardiomyopathy and no previous history of ventricular arrhythmias underwent placement of a biventricular cardioverter-defibrillator. The patient developed sustained monomorphic ventricular tachycardia (VT) soon after implantation. The VT recurred despite reprogramming to right ventricular only pacing. The electrical storm resolved only after a subsequent discharge from the defibrillator caused inadvertent dislodgement of the coronary sinus lead. No recurrent VT occurred throughout 10-years follow up after urgent coronary sinus lead revision.
Discussion: We describe the first reported case of mechanically induced electrical storm due to the physical presence of the CS lead in a patient with a new CRT-D device. It is important to recognize mechanical proarrhythmia as a potential mechanism of electrical storm, as it may be intractable to device reprogramming. Urgent coronary sinus lead revision should be considered. Further studies on this mechanism of proarrhythmia are needed.

Introduction

Cardiac resynchronization therapy (CRT) has been shown to improve both the functional status and mortality of chronic heart failure patients with left bundle branch block in large, randomized controlled trials [1,2]. However, evidence in recent case reports and cohort studies suggest left ventricular (LV) epicardial or right ventricular (RV) pacing in a CRT device may be proarrhythmic [3–7]. The proposed mechanisms include pacing into regions of arrhythmic substrate leading to scar related reentry, and LV epicardial pacing causing reversal of ventricular activation and prolongation of transmural dispersion of repolarization (TDR) [8]. Reprograming pacing sequence or inactivation of pacing has led to successful termination of ventricular arrhythmia in these cases.

Mechanically induced proarrhythmia related to the presence of an RV lead has recently been described [9,10]. We report a novel case of mechanically induced electrical storm in a patient with a newly implanted CRT defibrillator (CRT-D) device, which persisted after device reprogramming, but inadvertent coronary sinus (CS) lead dislodgement led to complete cessation of ventricular tachycardia (VT). This case contributes to the current literature by providing evidence and discussion of management strategies for non-pacing related mechanically induced etiology of VT storm as a complication of CRT.

Case presentation

A 51-year-old man with a history of hypertension, paroxysmal atrial fibrillation (AF), and non-ischemic cardiomyopathy presented for elective implantation of a biventricular implantable cardioverter defibrillator (or CRT-D device). He exhibited New York Heart Association Class III symptoms despite goal directed medical therapy with carvedilol 25 mg twice daily, lisinopril 40 mg once daily, and furosemide 20 mg once every other day. He described

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* Corresponding author. California Pacific Medical Center 1101 Van Ness Ave, 11th Floor, Suite 1120, San Francisco, CA, 94109, USA.
E-mail address: fengz1@sutterhealth.org (Z. Feng).
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intermittent palpitations associated with documented episodes of AF, for which he was prescribed amiodarone 200 mg daily. He had no previous history of presyncope, syncope, chest pain, or documented ventricular arrhythmia. His physical exam demonstrated displaced point of maximal impulse and otherwise unremarkable. His electrocardiogram (ECG) demonstrated normal sinus rhythm with left atrial enlargement, a prolonged PR interval, and left bundle branch block with QRS duration of 226 ms (Fig. 1A). An echocardiogram revealed severely depressed left ventricular systolic function, and an equilibrium radionuclide angiogram confirmed the presence of left ventricular dyssynchrony.

A CRT-D device was implanted, with leads positioned via the left axillary vein. A bipolar LV lead was placed into a large posterolateral branch of the CS, with adequate sensing and pacing thresholds achieved. Post implant ECG shows biventricular pacing and chest radiograph (CXR) shows posterolateral position of the CS lead (Fig. 1B and C). Defibrillation threshold testing was successfully performed at 25 J and 20 J.

During recovery in the post anesthesia care unit, several episodes of non-sustained VT with a right bundle-branch morphology, superior axis, and cycle length of 280 ms, consistently initiating on the T wave of a right bundle-branch morphology premature ventricular complex (PVC) with an inferior axis, were noted on telemetry (Fig. 2A). Two sustained episodes of rapid monomorphic VT subsequently occurred, both resulting in successful defibrillation by the implanted device (Fig. 2B). Device interrogation

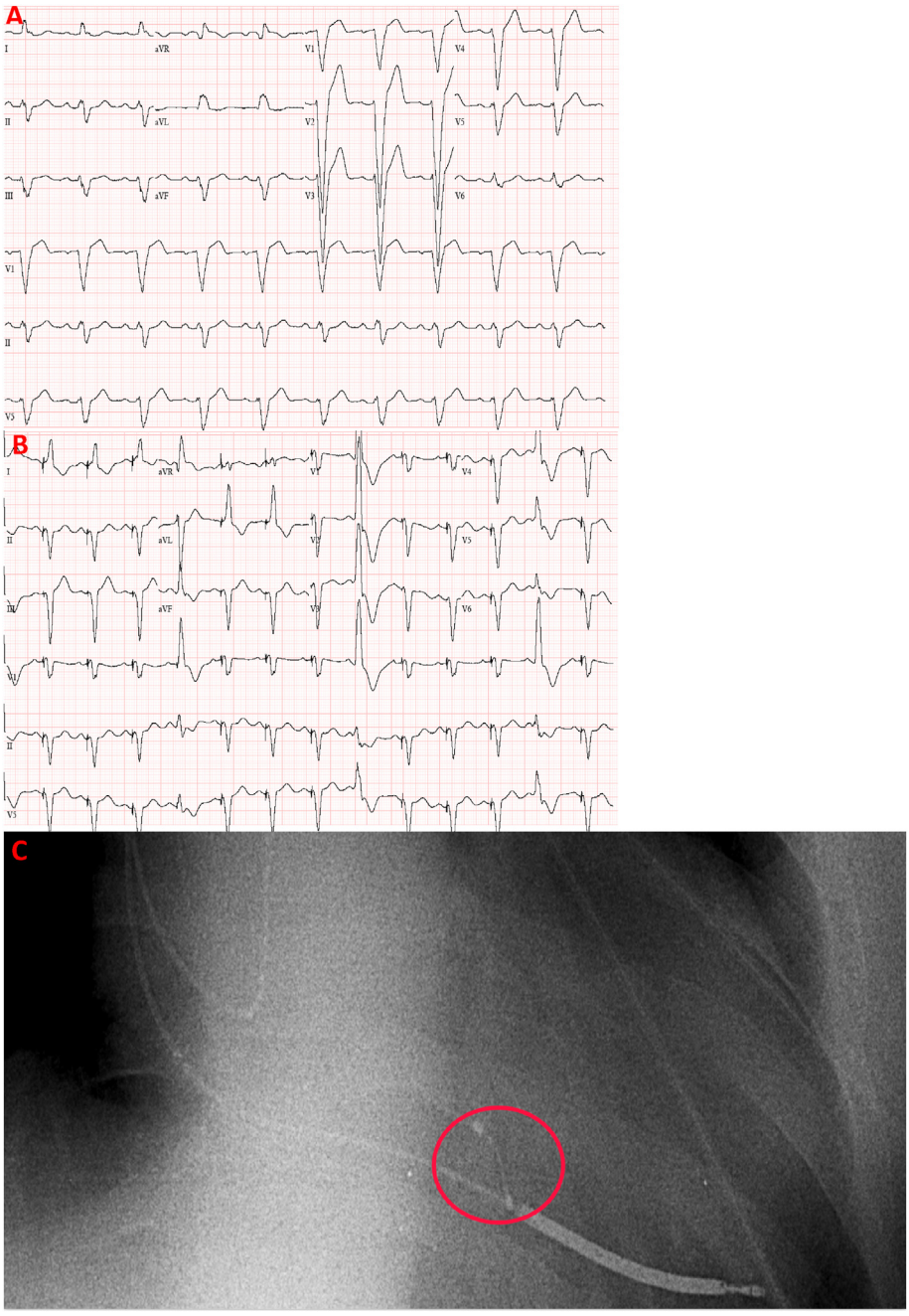

Fig. 1. **(A)** Baseline ECG showing LBBB with QRS of 226 ms. **(B)** Electrocardiogram after CRT-D implantation showing biventricular pacing and intermittent PVC. (C) Chest radiograph demonstrating posterolateral position of the coronary sinus lead.

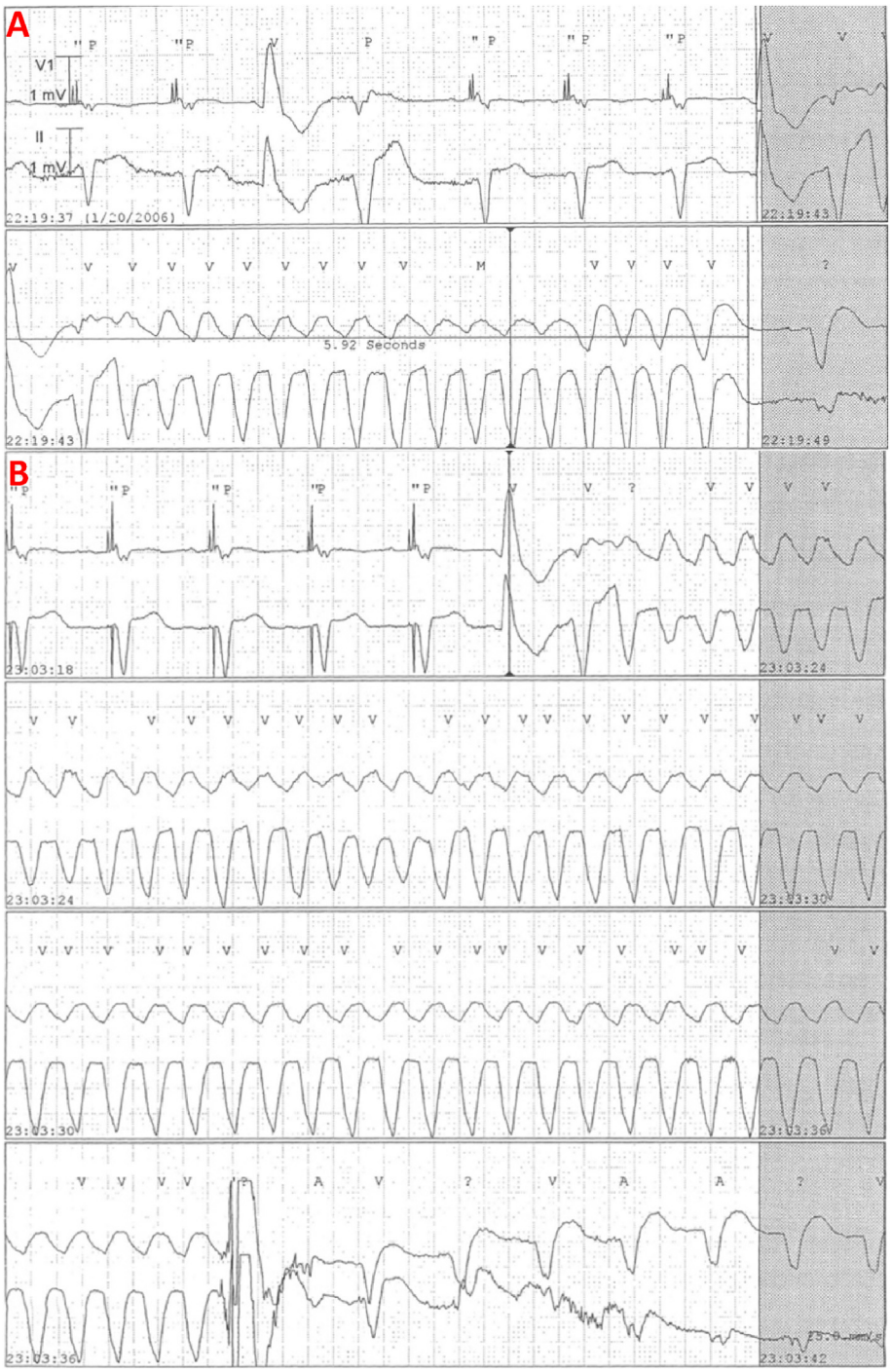

Fig. 2. (A) Telemetry post CRT-D implant: Continuous rhythm strip recorded from V1 (top) and lead II (bottom) positions, demonstrating non-sustained rapid VT initiated immediately after a PVC. **(B)** Continuous rhythm strip recorded from V1 (top) and lead II (bottom) positions, demonstrating sustained monomorphic VT initiated immediately after a PVC and terminated with an energy discharge from the implantable cardioverter-defibrillator.

revealed no change in lead sensing or thresholds. Left ventricular pacing alone was associated with ventricular couplets, while right ventricular pacing alone did not produced ectopy. Given LV pacing was a potential culprit for recent episodes of VT, the patient's device was subsequently reprogrammed to RV pacing only, with a back-up rate of 40 beats per minute.

A third episode of sustained monomorphic VT with the same mode of initiation, morphology, and cycle length occurred approximately 2 h later and was successfully treated with device defibrillation. Immediately after this episode, the CS lead would no longer capture, even at maximum output. An ECG shows absence of biventricular pacing (Fig. 3A). A CXR showed the CS lead had moved medially (presumably during the most recent defibrillation from the device), dislodged from its original position (Fig. 3B).

The patient experienced no further episodes of VT despite the absence of any changes in antiarrhythmic drug or catheter ablation-based therapies. The previously apparent triggering PVC continued to be observed (Fig. 3C). Two days later, the CS lead was successfully

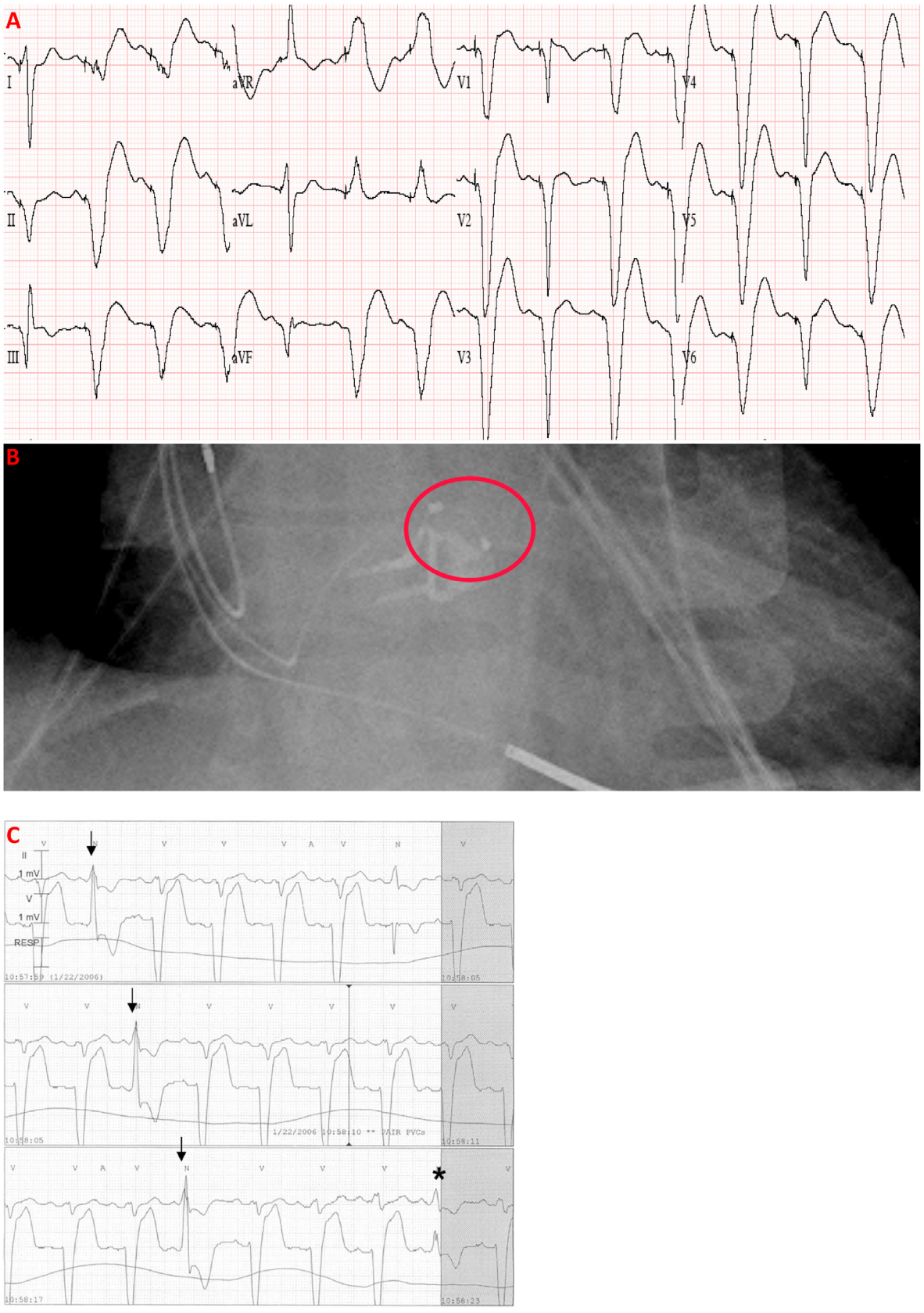

Fig. 3. **(A)** Electrocardiogram demonstrating loss of biventricular pacing after dislodgement of CS lead. **(B)** Chest radiograph demonstrating CS lead dislodgement. **(C)** The same PVC is intermittently seen (arrows) interpolated within the patient's native sinus rhythm with LBBB; a fusion beat is also shown (*). Despite persistence of these premature beats, no further ventricular arrhythmia was observed.

repositioned into an anterolateral branch. Follow up ECG and CXR confirms biventricular pacing with CS lead in the anterolateral position (Fig. 4A and B). The patient has had no further episodes of ventricular tachyarrhythmias with regular follow-up at 10-years.

Discussion

We describe a case in which recurrent, malignant VT developed soon after placement of a CRT-D device. The ventricular arrhythmia persisted after reprogramming to right ventricular pacing, excluding left ventricular pacing as the culprit source. Since the recurrent VT resolved only after the CS lead was dislodged from its original position after the third defibrillation, we believe the VT was related to mechanical irritation from the CS lead. Despite the persistence of an apparent triggering PVC (observed before and after implantation of the device), no sustained VT episodes occurred after dislodgement and urgent CS lead revision.

Experimental animal studies showed that epicardial LV pacing may be proarrhythmic by prolonging the QT interval and increasing the TDR, increasing the propensity to polymorphic VT [11,12]. One clinical case reported that LV pacing only resulted in polymorphic VT in a patient with a non-ischemic cardiomyopathy [13]. Two clinical cases of monomorphic VT induced by LV pacing and/or biventricular pacing have been described in patients with ischemic cardiomyopathies, each with a previous history of VT [14,15]. In these two cases, rather than affecting TDR, the initiation of these monomorphic arrhythmias has been attributed to the propagation of depolarization in a particular (in fact, opposite) direction, facilitating unidirectional block around a pre-existing scar, and therefore increasing the propensity towards re-entry [14,15].

Mechanically induced proarrhythmia is a newly described mechanism of ventricular arrhythmia. Ventricular tachycardia due

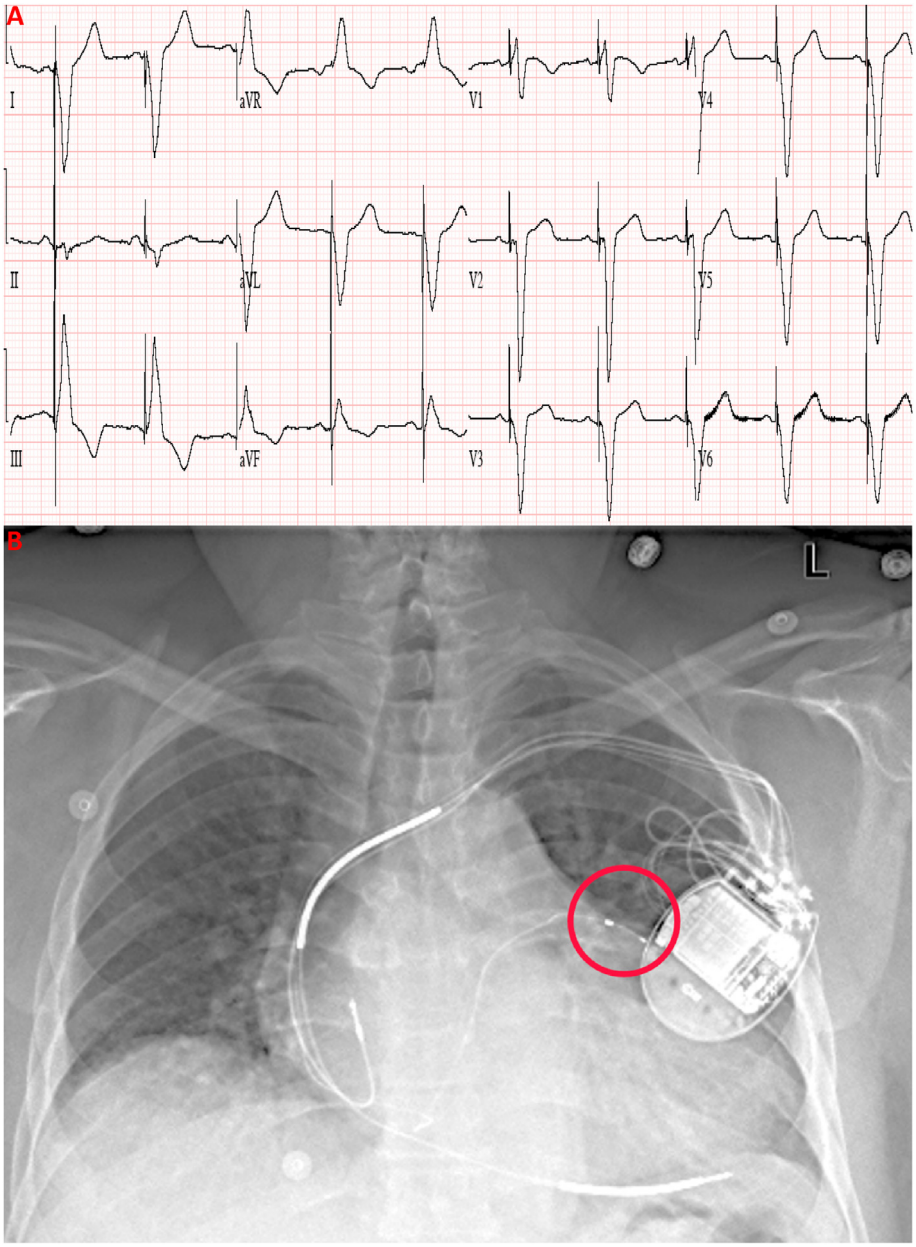

Fig. 4. **(A)** Electrocardiogram showing biventricular pacing after CS lead revision. **(B)** Chest radiograph demonstrating CS lead in the new anterolateral position.

to endocardial implantable cardioverter defibrillator or inactive pacemaker leads has been reported [9,10]. In these cases, spontaneous VT had a similar QRS morphology to that of the paced rhythm from respective leads, and lead extraction led to spontaneous resolution of recurrent VT. Another case reported late onset VT due to an abandoned surgically epicardial pacemaker lead placed prophylactically in a patient who underwent ventricular septal defect repair [16]. Electrophysiology study identified the earliest site of activation to be exactly opposite of the abandoned epicardial lead. Mapping showed normal voltage signals, suggesting the origin of VT was also not endocardial.

This is the first reported case in which a mechanically induced monomorphic VT due to a coronary sinus lead in a particular position has been described in a patient with non-ischemic cardiomyopathy. Presumably, the mechanical irritation of the lead was sufficient to change the ventricular conduction properties and/or repolarization properties of the myocardium such that the PVC became a trigger for malignant ventricular arrhythmias. The main limitation of our case is that we cannot reliably predict when and which patients are likely to experience this complication. However, we contribute data to this rare mechanism of VT storm and described management strategies which differ from traditional VT storm management.

Conclusion

We describe the first reported case of mechanically induced electrical storm due to the physical presence of the CS lead in a patient with a new CRT-D device. It is important to recognize mechanical proarrhythmia as a potential mechanism of electrical storm, as it may be intractable to device reprogramming. Urgent coronary sinus lead revision should be considered.

Statement of consent

The authors certify that they have obtained patient informed consent for publication.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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