QT interval prolongation and torsade de pointes induced by left ventricular pacing rescued by His bundle pacing

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Introduction

Cardiac resynchronization therapy (CRT) has been proven to be a beneficial therapeutic tool for appropriately selected patients with heart failure. CRT can improve hemodynamics, exercise capacity, quality of life, and survival.[1] The CARE-HF study demonstrated that CRT could reduce the risk of all-cause death in patients with heart failure and cardiac desynchrony.[1] However, it did not show a reduction in the risk of sudden death by CRT.[1]

A potential proarrhythmic effect of CRT has been hypothesized and supported by several case reports of ventricular arrhythmia following CRT implant.[2–6]

His bundle pacing has been shown to have the potential to induce cardiac resynchronization[7] with improvement in echocardiographic and functional status compared to biventricular (BiV) pacing in a blinded crossover trial.[8] Reversal of left bundle branch block when pacing slightly distally and with higher output was also identified and reported.[9] No untoward arrhythmogenic effects induced by His bundle pacing have been reported.

We describe a case of a patient with nonischemic cardiomyopathy (NICMP) that developed prolonged QT interval and torsade de pointes (TdP) episodes after CRT implantation, which resolved with His bundle pacing.

Case report

A 77-year-old woman with NICMP, severe left ventricular (LV) systolic impairment, and complete left bundle branch block presented for CRT evaluation. Past medical history included hypertension, hyperlipidemia, type 2 diabetes mellitus, and paroxysmal atrial fibrillation. A CRT defibrillator had been implanted in 2015 (Device: model no. Viva Quad XT CRT D DTBA1QQ; RA lead: model no. 5076 CapSure-Fix Novus; RV lead: model no. 6935M; LV lead: model no. 4298 Attain Performa MRI; Medtronic, Minneapolis, MN) in another facility. The LV lead had been noted to have dislodged and a few months following implantation it was turned off owing to failure to capture the LV. The atrial and right ventricular leads were performing well.

On follow-up, the patient developed progressively symptomatic heart failure and was referred for LV lead revision.

She was brought to the laboratory and underwent successful removal of the old LV lead. A coronary sinus venogram showed a suitable LV vein and a quadripolar LV lead (model no. 429888) was inserted without difficulty. The best LV capture threshold achievable was 2.5 V at 1.0 ms with the LV 2–3 electrode vector and that was accepted. The device generator was not changed.

The patient recovered well immediately after the procedure and was admitted for overnight observation. Overnight she sustained several episodes of dizziness and near syncope. Device interrogation showed short runs of polymorphic VT (Figure 1).

No previous VT episodes had been ever recorded through the lifetime of the device. Surface electrocardiogram showed pronounced QTc prolongation with LV and BiV pacing compared to baseline (QTc 479 ms for intrinsic conduction, 678 ms for LV-only pacing, 682 ms for BiV pacing, Figure 2). Changing the V-V delay did not significantly change the QTc. The patient had no known genetic channelopathy or long QT syndrome or any drug exposure that prolonged the QT. We therefore surmised the polymorphic VT might be caused by LV pacing. Other LV configurations were checked and had either high thresholds or no significant change in the QTc.

Based on these findings a decision was made to revise the procedure and add a His lead. Given the ideal LV lead location, we chose to leave the LV in place and connected it via a Y-connector (Lead adaptor, model number BIS/IS-15; Oscor Inc, Palm Harbor, FL). The IS4 pin of the quadripolar LV lead was inserted into 1 of the IS1 inputs of the Y-adaptor (the ring one). Only the distal electrode made a connection,

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KEY TEACHING POINTS

- Cardiac resynchronization therapy (CRT) pacing may induce prolongation of the QTc interval.
- Pacing-induced prolongation of QTc was found to be related to sustained ventricular tachycardia in a patient with CRT.
- His bundle pacing can be used as a rescue in cases with prolonged QT and arrhythmia after CRT implantation.

but electrodes 2, 3, and 4 were deep enough in the housing of the Y-adaptor to be insulated. Silicone was used to seal the connection.

The IS1 pin of the His lead (Medtronic; model number 3830 SelectSecure) was inserted into the other IS1 input of the Y-adaptor (the tip electrode). The Y-connector was connected to the IS1 LV port of a new implantable cardioverter-defibrillator generator; thus with different implantable cardioverter-defibrillator programming we could choose to pace unipolar LV or unipolar His.

The threshold for selective His-bundle capture was 4.5 V at 1.0 ms, impedance 285 ohms. However, satisfactory QRS narrowing and reduction of QTc prolongation was achieved with nonselective His pacing with lower outputs. The absolute capture threshold was down to 0.75 V at 1.5 ms. With nonselective His capture, the QRS was narrower (128 ms) than with intrinsic conduction or with BiV pacing and the QTc was 470 ms. (The QRS duration was 144 ms with BiV pacing and 150 ms in intrinsic conduction. The QTc duration was 550 ms with His pacing compared to 682 ms with BiV pacing.) We programmed the device at 3.25 V at 1.5 ms.

On subsequent follow-up, 12 months later, the patient had freedom from arrhythmias and her symptoms were improved. LV ejection fraction was improved from an estimated 30%–34% before the procedure to 50%–55% a few months after.

The His lead threshold remains stable, 0.75 V at 1.5 ms, and the device battery remaining longevity was 4.8 years.

Discussion

CRT has become an established adjunctive treatment to optimal pharmacological therapy for select heart failure patients with advanced congestive heart failure, diminished LV function, and intraventricular conduction delay. The benefits of CRT are well established and include improvements in hemodynamics, exercise capacity, quality of life, and survival.[1] A decrease in the frequency of ventricular arrhythmia was also described.[10–12] However, evidence reported in the literature suggests that LV pacing can potentially provoke arrhythmia.[2–6] Tachyarrhythmia was observed as

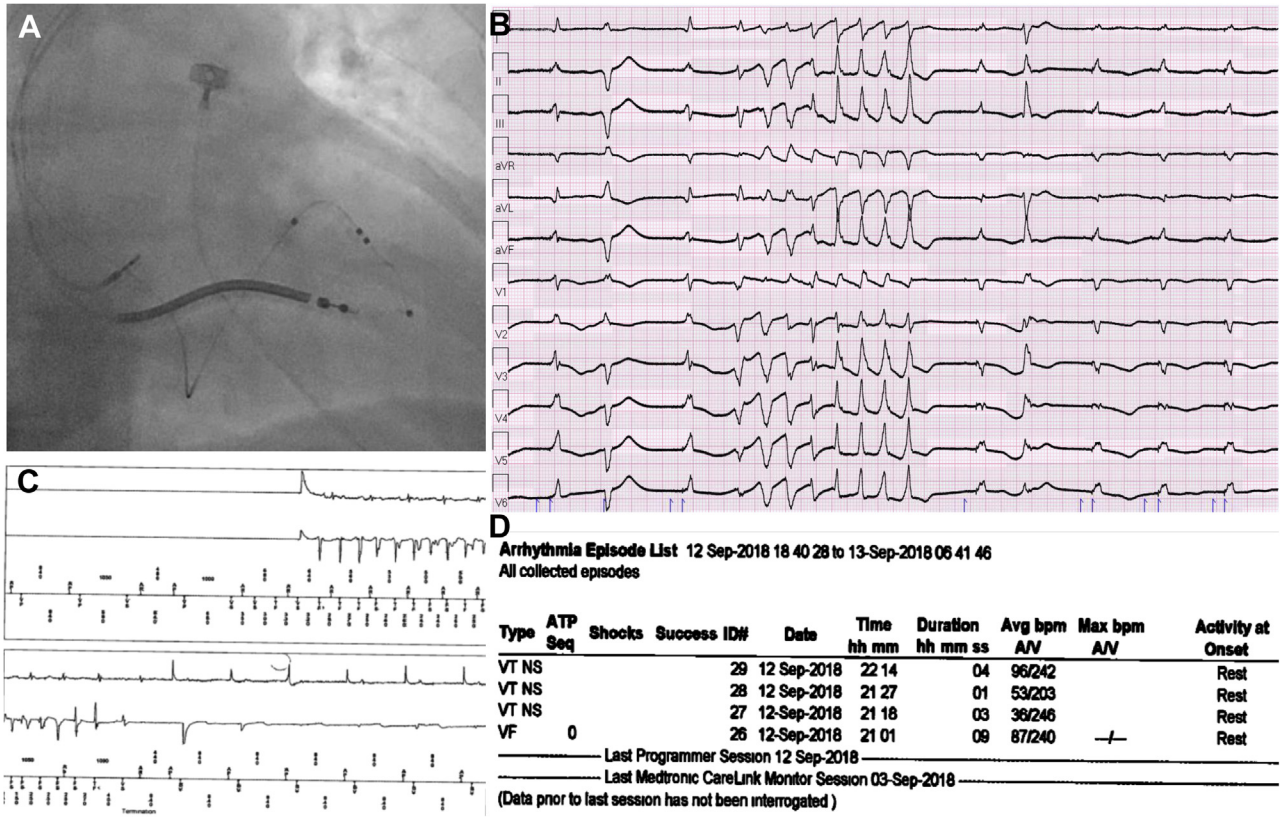

Figure 1 Polymorphic ventricular tachycardia (VT) after left ventricle (LV) lead revision. **A:** Lead position at the end of the procedure, right anterior oblique projection: right atrium lead, right ventricle lead, and new LV lead. **B:** Electrocardiogram after LV lead revision with prolonged QTc, 682 ms, and nonsustained VT. **C:** Device interrogation showing nonsustained VT. **D:** Arrhythmia episode list after LV lead revision procedure.

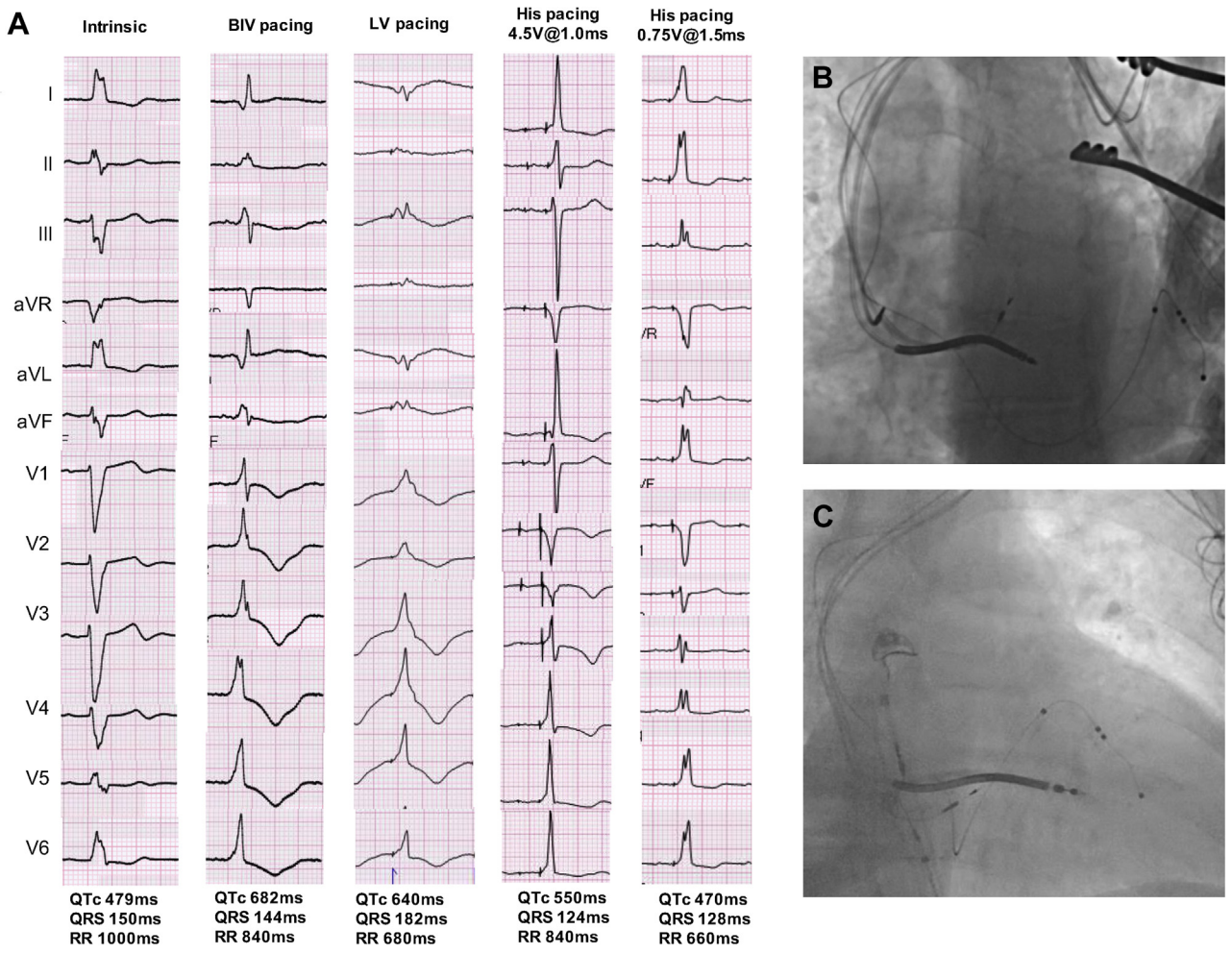

Figure 2 His bundle pacing improves the QT prolongation. **A:** The 12-lead electrocardiograms demonstrated the change in the QTc. Intrinsic QTc was 479 ms; with biventricular (BIV) pacing, 682 ms; with left ventricle (LV) pacing only, 678 ms; with nonselective His pacing at higher output, 550 ms; and with nonselective His pacing at lower output, 470 ms. **B:** His lead positions in left anterior oblique projection. **C:** His lead positions in right anterior oblique projection.

monomorphic VT, polymorphic VT, TdP, or ventricular fibrillation.[5]

Medina-Ravell and colleagues[2] first reported the potential mechanisms of CRT-induced arrhythmia in 2003. In both human cases and animal experiments, BiV and LV epicardial pacing were found to be associated with ventricular arrhythmia, including R-on-T extrasystoles and TdP. The mechanism was broadly attributed to altered ventricular repolarization, including prolongation of the QT and JT intervals, and increased transmural dispersion of repolarization (TDR). Reentry was another proposed mechanism responsible for monomorphic VT in reported series of cases.[5]

Normally, ventricular depolarization spreads from endocardium to epicardium. However, because of the increased abundance of I_{to} channels in the epicardium, the action potential is shorter in the epicardium, which repolarizes earlier and causes the direction of repolarization to be from the epicardium to the endocardium.[13] Thus, the electric vectors of depolarization and repolarization are identical, causing the QRS complex and the T wave to have the same polarity in general.

LV epicardial pacing via the coronary sinus route may result in reversal of the direction of the depolarization, resulting in prolonged QT and JT intervals and TDR. This was verified by experimental study reported by Fish and colleagues,[13] who suggested that epicardial activation of LV wall prolongs the QT and the TDR intervals. Prolongation of ventricular repolarization time may enhance myocardial vulnerability toward early afterdepolarizations and initiation of TdP.

Management of this problem may be challenging. Cessation of LV pacing can result in arrhythmia termination. However, discontinuing resynchronization pacing can cause the loss of the proven benefits of CRT. In the past, the majority of such patients were given antiarrhythmic drugs in order to safely reintroduce biventricular pacing.

Other options could be using alternative ways to achieve ventricular resynchronization by epicardial LV lead placement via thoracoscopy or video-assisted thoracoscopic procedure, placing the lead across the interatrial or interventricular septum into the LV endocardium or using the novel WiSE-CRT system (EBR Systems, Sunnyvale, CA) incorporating an LV endocardial microelectrode

powered by ultrasound energy.[14,15] These options are associated with technical challenges, additional procedural risks, and need for systemic anticoagulation.

Recently, His bundle pacing has been shown to have the potential to induce cardiac resynchronization with improvement in echocardiographic and functional status in comparison with biventricular pacing in a blinded crossover trial.

We describe a case of a patient with NICMP with no history of arrhythmia that developed prolonged QT and TdP episodes immediately after CRT implantation. We added a His lead that resulted in QRS narrowing and QT prolongation reduction, with freedom from further arrhythmias on follow-up.

Conclusion

His bundle pacing can be used as a rescue in cases with prolonged QT and arrhythmia after CRT implantation.

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