Ventricular tachycardia exacerbated by left bundle branch area pacing

Tanyanan Tanawuttiwat, MD, MPH, FHRS,* Eric Kellett, MD,*
Mithilesh K. Das, MBBS, MBA, FHRS,* Lukas J. Fore, BSN, RN, CEPS,†
John M. Miller, MD, FHRS*

*From the *Division of Cardiovascular Medicine, Indiana University, Indianapolis, Indiana, and †Biosense Webster, Inc, Irvine, California.*

Introduction

Since 2017, after Huang and colleagues[1] suggested direct capture of the left bundle branch (LBB) by placing the lead deep inside the proximal interventricular septum, left bundle branch area pacing (LBBAP) has gained popularity owing to its easier application, lower and stable pacing thresholds, higher sensing amplitude, and more stable lead position, when compared with His bundle pacing. Here, we describe a rare case of ventricular tachycardia (VT) that developed after implantation of an LBBAP lead and necessitated both catheter ablation and lead removal.

Case report

A 52-year-old woman with a history of Hodgkin lymphoma and chest radiation therapy presented with syncope due to intermittent complete heart block. A year earlier, she had a transcatheter aortic valve replacement (TAVR). Echocardiogram revealed normal left ventricular (LV) function (ejection fraction of 66%) and a normal gradient across the TAVR aortic valve. She therefore underwent dual-chamber pacemaker placement with LBBAP using the 3830 SelectSecure™ lead and C304 SelectSite™ catheter (Medtronic, Minneapolis, MN). LBB capture was confirmed as per previously described criteria.[1,2] The device was programmed in AAI-DDD mode with unipolar ventricular pacing (Figure 1A). She experienced a second episode of syncope a month after pacemaker placement; this correlated with sustained VT on device interrogation (Figure 1B). She had frequent premature ventricular complexes (PVC) with QRS morphology that were similar to her baseline QRS, but no further VT occurred in hospital (Figure 1A and 1B). Given her high ventricular pacing percentage, she had a system upgrade to a cardiac resynchronization therapy (CRT) system with a new right ventricular (RV) defibrillator lead at the apex and preexisting LBBAP and right atrial leads. The device was programmed in DDD mode (LV to RV delay 80 ms).

Two months after device upgrade, she presented to the emergency department with chest pain. Two more VT episodes, which were terminated by antitachycardia pacing (ATP), were noted during device interrogation (Figure 1B). At this time, the patient was pacemaker dependent with ventricular escape <40/min. She was diagnosed with non-ST elevation myocardial infarction (high-sensitivity troponin 388 ng/L [normal, <55 ng/L]). A drug-eluting stent was implanted after coronary angiography revealed 80% distal left main artery stenosis. During 2-month follow-up after revascularization, she had a few instances of lightheadedness associated with VT episodes that were terminated with ATP.

KEY TEACHING POINTS

- Proarrhythmic effects from ventricular pacing are rare and under-recognized. We report the first case of left bundle branch area pacing (LBBAP) lead–induced ventricular tachycardia (VT).
- Several reports of VT caused by ventricular pacing have been treated by turning pacing off, reducing pacing output to a subthreshold level, and reprogramming right and left ventricular timing in patients with cardiac resynchronization therapy. Our case necessitated LBBAP lead removal and catheter ablation.
- There are some unique characteristics of LBBAP lead, including lead depth in the interventricular septum, pacing location at the left bundle branch region, and possible effects of septal contraction or hinge at the insertion site, that could theoretically be proarrhythmic. However, the data on long-term outcomes of the LBBAP in terms of proarrhythmic effects remain limited.

KEYWORDS Left bundle branch area pacing; Conductive system pacing; Cardiacphysiologic pacing; Ventricular tachycardia; Ventricular arrhythmias
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Address reprint requests and correspondence: Dr Tanyanan Tanawuttiwat, 1800 N Capitol Ave, Indianapolis, IN 46202. E-mail address: ttanawu@iu.edu.

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Metoprolol succinate was increased from 25 mg to 100 mg orally daily.

A few months later, she arrived at the hospital with multiple VTs. These VTs were terminated with ATPs but reinitiated after 4–5 A-V sequential pacing beats, leading to her being shocked by the device. She received a 150 mg intravenous bolus dose of amiodarone at the emergency department. After the device was set to only RV pacing, the VT did not recur while the patient was in the hospital. However, she experienced additional episodes of VT, terminated with implantable cardioverter-defibrillator (ICD) shocks after discharge from hospital. Several device programming changes, including LBBAP tip pacing only, LBBAP ring pacing only, and simultaneous RV and LBBAP pacing, were attempted, but her arrhythmia burden was unchanged. The LBBAP pacing lead impedance and threshold were steady, with no significant changes since implant, and the QRS morphology of LV pacing only remains unchanged, indicating that a perforated septum as a cause of VTs is improbable. She underwent electrophysiologic study (EPS), attempting to induce VT. Up to triple extrastimuli from the RV lead, the LBBAP lead, and a catheter at the RV apex and RV outflow tract during moderate sedation did not induce VT. She was discharged with metoprolol 150 mg once daily and mexiletine 200 mg twice a day.

Two weeks after hospital discharge, she had episodes of dizziness and was found to have incessant, nonsustained VT. The VT burden was decreased with programming to RV pacing only at 40/min, but nonsustained VT resumed at a lower rate of 60/min (Figure 1C). However, she was pacemaker dependent and was not able to tolerate a rate of 40/min. She then underwent a second EPS under moderate sedation, but ventricular arrhythmias were again not inducible. An RV voltage map (775 points, with 3.5-mm, deflectable irrigated catheter [Thermocool SmartTouch; Biosense Webster]) showed normal voltage throughout, with no areas of scar. Given the similar QRS morphology of her nonsustained VT and the paced complex of the LBBAP ring electrode to the RV coil (Figure 1C), radiofrequency ablation was done under fluoroscopic guidance at the RV insertion site of the LBBAP lead (Figure 2A). LV mapping was not done because of the patient's history of TAVR, noninducible arrhythmias, and LBB morphology in lead V_1 during nonsustained VT (Figure 1C). She continued metoprolol and mexiletine at discharge.

She experienced more VT episodes requiring ICD shocks a week following the second EPS. After 2 failed attempts to induce VT, the occurrence of VT after the LBBAP lead implant, and unsuccessful attempts to reduce VT with device reprogramming, we decided to remove the LBBAP lead. She underwent LBBAP lead removal and placement of a new bipolar LV lead at the posterolateral branch of the coronary sinus. Soon after the procedure, VT returned, and she was started on amiodarone. A few VT episodes occurred during oral amiodarone loading, but VT burden was significantly decreased afterwards. However, she experienced extreme fatigue while using amiodarone and could not tolerate the medication.

She therefore underwent a third EPS with attempted ablation. Ventricular extrastimuli failed to induce sustained arrhythmias. However, with quadruple extrastimuli, reproducibly initiated single complexes of a right bundle branch (RBB) superior axis PVC (Figure 1C), similar to her initial PVC prior to CRT-defibrillator upgrade (Figure 1A). Pace mapping was performed, and a 12/12 lead match was obtained at the mid-LV septum (Figure 2B). Ablation was performed in the area using an irrigated catheter, with 5 applications of 50 W covering 1.1 cm^2 (3 applications lasting 100 seconds and 2 lasting 60 seconds) was performed. Amiodarone was discontinued after the EPS. When anatomical maps from the last 2 EPS were combined using the coronary sinus locations for registration, the proximity of both ablations suggested the site of the second ablation was at the site of the tip electrode of the previously implanted LBBAP lead. No VT or nonsustained VT recurrence was found at the 10-month follow-up since the second ablation (Figure 3).

Discussion

There have been multiple reports on the proarrhythmic effects of cardiac resynchronization therapy, single- and dual-chamber pacemakers/ICDs.[3–6] In 2003, Himmrich and colleagues[3] described episodes of VT with onset after a single visible and effective pacemaker stimulus as "pacemaker-induced ventricular tachycardia/fibrillation (PIT)" in ICD patients with VVI backup pacing programming. The authors demonstrated in their randomized crossover study that sustained ventricular arrhythmias no longer occurred after the pacemaker backup feature was deactivated, implying that stimulated impulses were responsible for inducing the VT / ventricular fibrillation and that the arrhythmia did not occur independent of ventricular stimulation. In their study, all episodes occurred after a pause of 1200 ms, suggesting a possible mechanism of a short-long-short sequence resulting in dispersion of the ventricular refractory period, which is regarded as a prerequisite of ventricular arrhythmia. Additional postulated mechanisms of proarrhythmia in patients with pacemakers/ICDs included local irritability (early occurrence) and/or fibrosis (late occurrence).[5] Irritability is supported by a relatively higher incidence of VT and ventricular fibrillation in the first week after transvenous and epicardial ICD implantation, whereas local fibrosis is a theoretical mechanism that could occur at any time after lead implantation.[5] Another mechanism that has been hypothesized is functional conduction slowing during ventricular pacing, supported by a higher degree of ventricular electrogram fractionation. Slow conduction may coexist with unidirectional conduction block and may result in emergence of reentry.[4] Additionally, pacing within or very close to the region of slow conduction responsible for the clinical VT circuit in patients with ventricular substrate/scar may have a

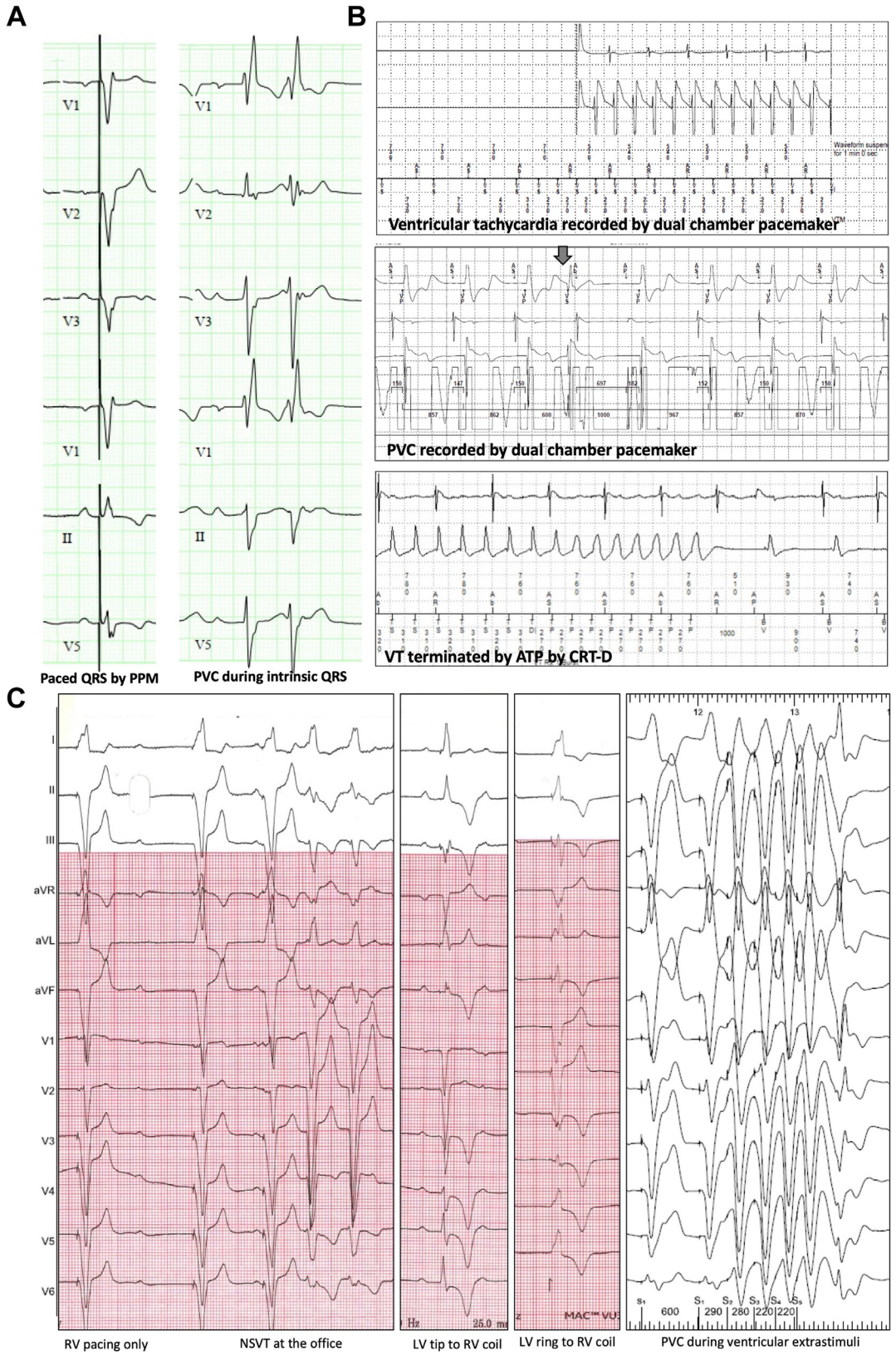

Figure 1 **A:** Unipolar left bundle branch area pacing (LBBAP), intrinsic QRS complex, and premature ventricular complex (PVC) morphologies during the patient's second admission with syncope. **B:** An intracardiac electrogram recorded from the pacemaker and implantable cardiac resynchronization therapy defibrillator (CRT-D) device. Far-field morphologies of the ventricular tachycardia (VT), PVC, and ventricular pacing via LBBAP are demonstrated. **C:** The QRS morphology of right ventricular (RV) pacing only, nonsustained VT, LBBAP tip–to–RV coil pacing, LBBAP ring–to–RV coil pacing, and PVC following quadruple extrastimuli.

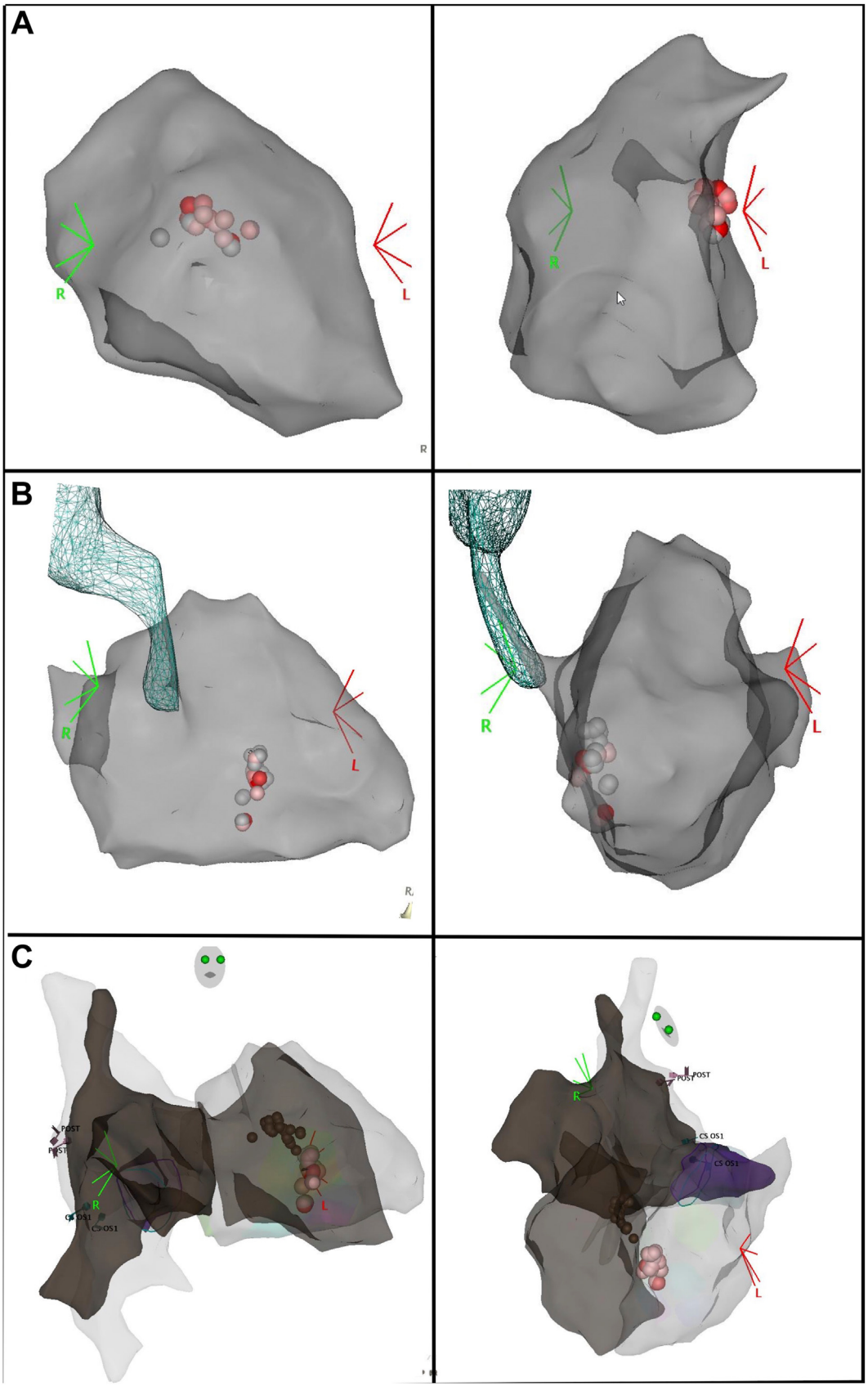

Figure 2 **A:** Location of the radiofrequency (RF) applications at the right ventricle during the first ablation procedure by fluoroscopic guidance at the left bundle branch area pacing (LBBAP) lead ring electrode. **B:** Location of the ablation lesions in the left ventricle based on pace mapping. **C:** Proximity of RF applications from the 2 procedures by combining anatomical maps registered on the coronary sinus location. This suggests the locations of RF applications at the second procedure were at or near the tip electrode of the implanted LBBAP lead.

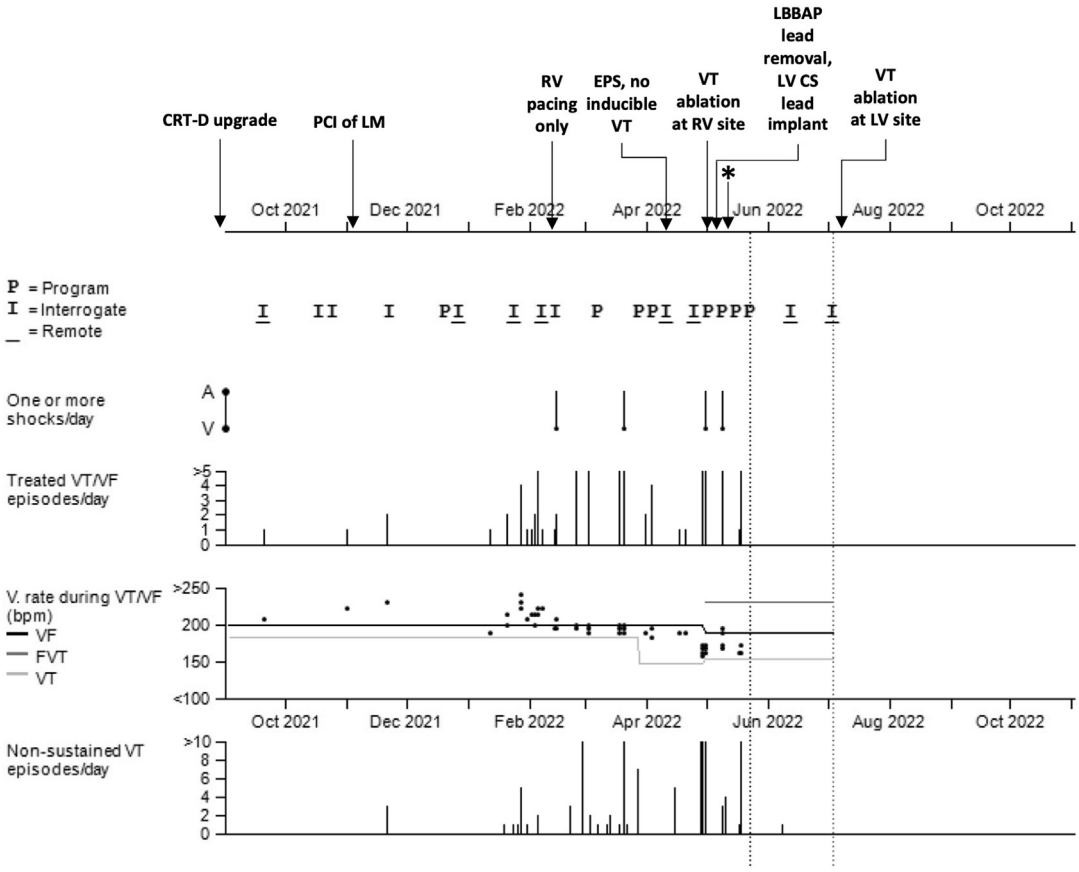

Figure 3 Ventricular tachycardia burden recorded by the implantable cardiac resynchronization therapy defibrillator (CRT-D) device and timeline of treatment (* represents the time that the patient was started on amiodarone).

proarrhythmic effect.[4] This mechanism was previously described as "pace-map induction" of VT.[7]

In our patient, VT occurred shortly after LBBAP lead implant. The PVC morphology during the initial admission with VT was similar to the baseline intrinsic QRS morphology and suggested its origin was in or close to the conduction system (Figure 1A). In retrospect, this PVC morphology was comparable to those induced by quadruple extrastimuli (Figure 1C) that were used for pace mapping during successful ablation. Owing to the PVC morphology of RBB block in V_1, irritability from the septal movement at the RV insertion site of the LBBAP lead is unlikely. All her far-field VT morphology recorded by the device is the same on all treated and nonsustained VT episodes, before and after ablation at the RV septum. The LBB morphology changing to RBB morphology (Figure 1C) could be from minimal changes in exit site after ablation that are not detected by far-field morphology or incorrect precordial lead positioning in the setting of morbid obesity and the patient's position at the clinic. The patient's history of chest radiation and distal left main coronary artery disease suggest the possibility of ventricular arrhythmia substrates within her ventricular septum. We initially hypothesized that the LBBAP pacing lead may be pacing at or near a critical region of slow conduction. Since VT episodes continued after reprogramming to RV pacing only and LBBAP lead removal, the mechanism of VT in our case is due not to pace-map induction alone but also to fibrosis as a component of arrhythmia substrate after lead implant. Although several reports of VT induced by ventricular pacing were resolved by turning pacing off, decreasing pacing output to a subthreshold level, and reprogramming RV and LV timing in patients with CRT, our case required lead removal as well as catheter ablation. While LBBAP has emerged as a new physiological pacing modality, proarrhythmic effects from placing the lead via a transventricular septal approach and pacing near or at the conduction system remains unknown.

Conclusion

We describe the first case of LBBAP lead–induced ventricular tachycardia. High suspicion of LBBAP lead as a cause of VT should be raised when the clinical VT/PVC morphology resembles the baseline paced QRS complex. Turning pacing off, lowering the pacing output to a subthreshold level, and reprogramming RV and LV timing in patients with CRT have all been reported to manage ventricular pacing–induced ventricular tachycardia and were attempted in our case. Nonetheless, LBBAP lead removal and catheter ablation were ultimately required. Further research on proarrhythmic effects of LBBAP is warranted.

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References

1. Huang W, Su L, Wu S, et al. A novel pacing strategy with low and stable output: pacing the left bundle branch immediately beyond the conduction block. Can J Cardiol 2017;33:1736.
2. Jastrzebski M, Burri H, Kielbasa G, et al. The V6-V1 interpeak interval: a novel criterion for the diagnosis of left bundle branch capture. Europace 2022; 24:40–47.
3. Himmrich E, Przibille O, Zellerhoff C, et al. Proarrhythmic effect of pacemaker stimulation in patients with implanted cardioverter-defibrillators. Circulation 2003;108:192–197.
4. Hayase J, Khakpour H, Shivkumar K, Bradfield JS. Right ventricular lead proarrhythmia: a novel intervention for an under-recognized phenomenon. HeartRhythm Case Rep 2018;4:50–53.
5. Germano JJ, Reynolds M, Essebag V, Josephson ME. Frequency and causes of implantable cardioverter-defibrillator therapies: is device therapy proarrhythmic? Am J Cardiol 2006;97:1255–1261.
6. Guerra JM, Wu J, Miller JM, Groh WJ. Increase in ventricular tachycardia frequency after biventricular implantable cardioverter defibrillator upgrade. J Cardiovasc Electrophysiol 2003;14:1245–1247.
7. Tung R, Mathuria N, Michowitz Y, et al. Functional pace-mapping responses for identification of targets for catheter ablation of scar-mediated ventricular tachycardia. Circ Arrhythm Electrophysiol 2012;5:264–272.