Case Reports

Transient 2:1 Atrioventricular Block with Peri–Conduction System Pacing After Leadless Pacemaker Implantation

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Abstract

This report discusses a case of transient 2:1 atrioventricular block with conduction system pacing 4 hours after leadless right ventricular pacemaker implantation in a 19-year-old patient with a history of cardioinhibitory syncope and asystole cardiac arrest but without preexisting atrioventricular block. The atrioventricular block was resolved spontaneously. Pacing morphology was suggestive of right bundle branch pacing. Neither 2:1 atrioventricular block nor conduction system pacing has previously been a reported outcome of right ventricular leadless pacemaker implantation. The report demonstrates that conduction system pacing with leadless devices is achievable. Further study of techniques, limitations, and complications related to intentional right ventricular leadless conduction system pacing is warranted.

Keywords: Electrophysiology; pacemaker, artificial; heart conduction system; atrioventricular block

Case Report

Presentation

A 19-year-old female patient with patent ductus arteriosus status following coil occlusion and an epicardial single-chamber pacemaker placed for cardioinhibitory syncope without a history of preexisting atrioventricular (AV) block presented with a pacemaker with a high pacing threshold and output at its elective replacement indicator. She underwent the transcatheter placement of a Micra VR (Medtronic) leadless pacemaker (LPM) with a ventricular pacing and ventricular sensing with inhibition (VVI) setting as well as the removal of her epicardial generator. The LPM was initially deployed in the superior right ventricular septum with adequate tine positioning, but it was then displaced backward, toward the tricuspid valve, and retrieved back into the device delivery system. A new system was deployed, and the LPM was placed in the mid-distal right ventricular septum (Fig. 1). The device demonstrated adequate sensing, pacing threshold, and impedance. Cineangiography was performed, demonstrating that at least 2 tines were in contact. No intraoperative or immediate postoperative conduction disturbance was identified. Postoperative chest radiography demonstrated that the LPM was in the appropriate position (Fig. 2).

Four hours postoperatively, the patient developed an intermittent second-degree type 1 and 2:1 AV block with narrow-complex backup pacing (QRS duration <120 ms), with a similar axis to sinus rhythm (Fig. 3). Neither the AV block nor the rate-dependent bundle-branch block was identified on the preoperative 24-hour Holter monitor. The remainder of her vital signs were within normal limits. She did not have lightheadedness, fatigue, chest pain, or dyspnea. Examination revealed normal cardiac auscultation, except for regular bradycardic rhythm. She tolerated

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ambulation without abnormal symptoms, albeit without improvement of AV conduction. Echocardiography did not demonstrate any structural abnormality or pericardial effusion. Device interrogation revealed appropriate sensing, impedance, and threshold.

Medical History

Coil occlusion of patent ductus arteriosus was performed when the patient was 5 years old. Three months later, a single-lead epicardial pacemaker was placed for recurrent cardioinhibitory vasovagal syncope with symptomatic bradycardia. At 9 years old, she developed lead fracture and presented with asystole cardiac arrest. She underwent lead and generator exchange at that time without subsequent recurrence of syncope. During that procedure a pericardial effusion was identified and resolved without intervention on serial imaging.

Differential Diagnosis

No metabolic or structural cardiac abnormalities to explain the AV block were identified. Given the temporal association to LPM implantation as well as the absence of a preoperative AV block on Holter monitoring, this was suspected to represent a procedural sequela. The device's implantation in the right ventricular septum likely placed it in sufficient proximity to the conduction system to cause the AV block and narrow-complex pacing. The AV block may have occurred as a result of mechanical disruption of conduction at the implantation site. Device redeployment may have contributed to

Key Points

- A transient delayed-onset AV block after right ventricular LPM implantation is possible, and prolonged observation is reasonable. In the case this report discusses, no medical therapy or device repositioning was required.
- Right bundle pacing produces more delayed left ventricular activation than His bundle pacing, with comparable capture thresholds and clinical outcomes.
- Conduction system pacing is achievable with right ventricular LPMs, though it warrants further study.

Abbreviations and Acronyms

AV	atrioventricular
LPM	leadless pacemaker

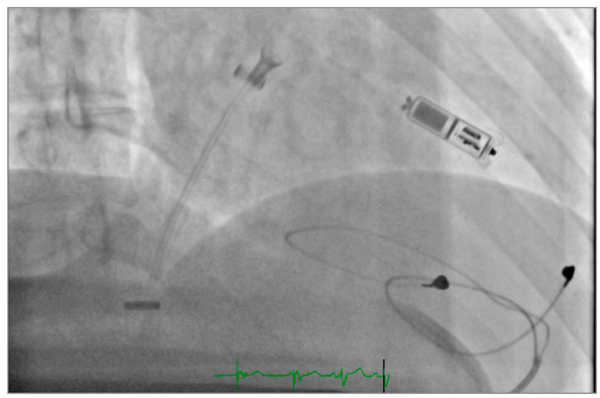

Fig. 1 Postdeployment intraoperative fluoroscopy in the right anterior oblique position demonstrates the leadless pacemaker's position in the mid-distal right ventricular septum.

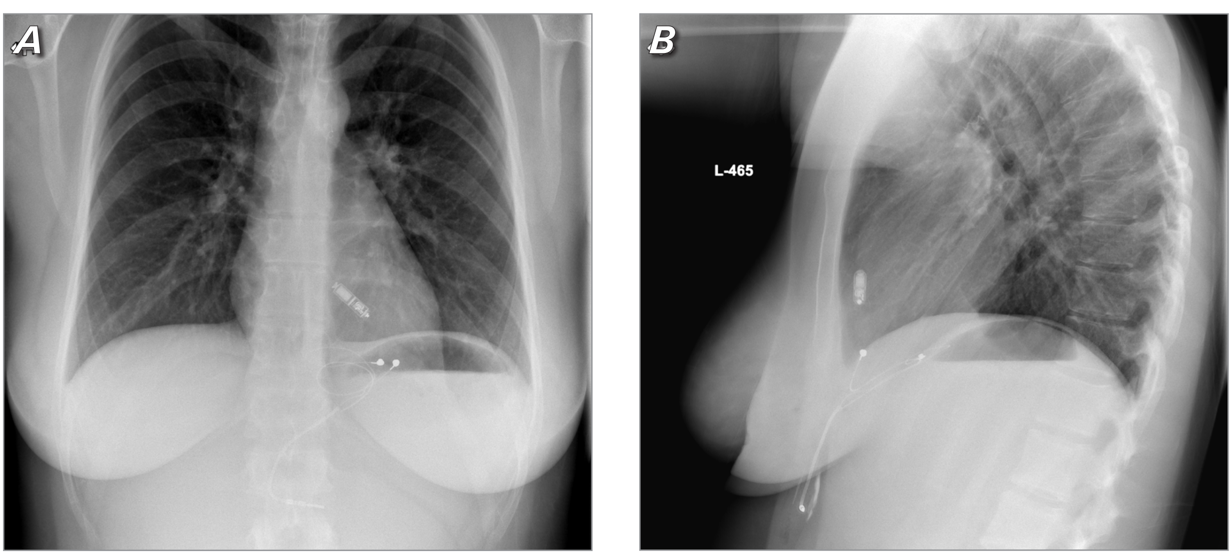

Fig. 2 Postimplantation chest radiographs show (**A**) posterior-anterior and (**B**) lateral views of the leadless pacemaker's position in the mid-distal right ventricular septum.

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Fig. 3 Electrocardiogram tracings. **A**) Holter monitor tracing obtained the evening after device implantation demonstrates a 2:1 AV block; (**B**) postimplantation telemetry tracing demonstrates the AV block and successful backup pacing, with QRS complexes of a similar electrical axis to native sinus conduction[a]; (**C**) the QRS duration of the paced complex measures 119 ms by caliper on telemetry.

AV, atrioventricular.

[a] The lead labeled V_1 was misplaced and likely represents V_6.

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localized, procedure-related trauma, edema, or bleeding at the device tines, thereby contributing to conduction system disturbance.

Thrombus formation is an alternative explanation and has been described as a complication of LPM implantation.[1] The delayed nature of the AV block could also be explained by delayed thrombus formation. Device position changes related to patient movement out of the recumbent position postoperatively could also explain delayed onset. The gradual pattern of resolution of the phenomenon and persistence of the AV block at night argue against this etiology.

Appropriately timed para-Hisian ventricular ectopy could create 2:1 conduction impairment by rendering the ventricle refractory to conduction at regular intervals. Ventricular ectopy triggered by LPM implantation has been reported.[2] In the previous case, however, ectopy was identified in the perioperative period and required device explant for resolution, whereas no ectopy was identified in this patient.

Technique

The AV block was resolved without intervention, with the patient's heart rate gradually increasing from 50/min to between 75/min and 90/min in sinus rhythm. The patient was discharged home in stable condition after 24 hours of observation.

Latest Follow-Up

At 1-year follow-up, the patient continued to demonstrate narrow-complex pacing (Fig. 4) and low pacing burden (<0.1%), as expected.

Discussion

A transient complete AV block immediately after LPM implantation that resolved within seconds to a few hours was identified in 2.9% of patients in early studies.[3] Neither delayed-onset 2:1 AV block nor conduction system pacing has been a previously reported outcome of right ventricular LPM implantation. The possible etiologies of an AV block associated with LPM implantation discussed in early studies included bleeding, thrombus formation, edema, ventricular ectopy, and positional changes. Each of these etiologies would suggest a different treatment paradigm if refractory AV block were to develop. Given the spontaneous resolution of the AV block in this case and the patient's overall stable course, no specific treatment was required. There is insufficient evidence to suggest specific therapeutics for similar cases.

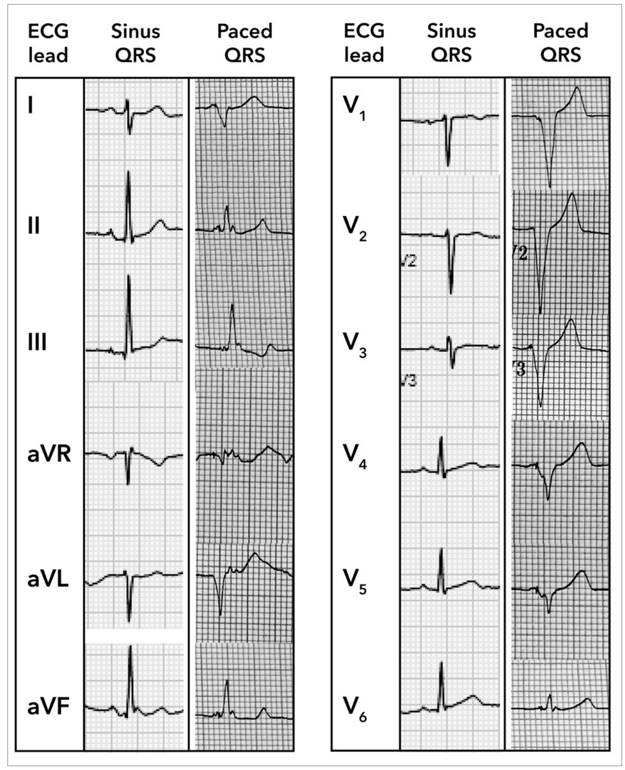

Fig. 4 Paced QRS complexes at 1-year follow-up are comparable to native sinus conduction.

ECG, electrocardiogram.

Permanent right ventricular LPMs are well suited to the needs of young patients with congenital heart disease because of the elimination of pacing leads, which are the primary drivers of complications, including infection and venous thromboembolism. A wide-complex paced rhythm with left bundle-branch block morphology is typically produced after LPM implantation in the right ventricular septum as a result of pacemaker capture of the right ventricular myocardial cells, not the initiation of depolarization in the His-Purkinje system. Narrow-complex paced rhythm with a similar morphology and similar axis to sinus rhythm, as seen in this case, suggests pacing of the conduction system.

Compared with sinus rhythm, the paced rhythm demonstrates a delay in left ventricular activation, with a relatively wider QRS complex, a smaller R wave, and a deeper S wave in lead V_1. Based on this morphology, it may be postulated that right bundle-branch pacing was achieved in this case. Right bundle-branch pacing produces more delayed left ventricular activation than His bundle pacing, with comparable capture thresh-

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olds and clinical outcomes.[4] Ventricular ectopy arising from the septum can produce QRS complexes similar to those seen in this case. Activation of the ventricular septum may result in narrow QRS complexes without conduction system capture as a result of concurrent leftward and rightward ventricular activation; however, ventricular septal ectopy is more likely to demonstrate early precordial transition and an RS complex in lead V_1, neither of which was present in this case. The late precordial transition and QS morphology in lead V_1 is more consistent with para-Hisian activation.[5] Some degree of fusion of the conduction system and septal pacing may be present. Assessment of morphologic differences at different pacing outputs can help differentiate pure conduction system pacing from fusion with septal activation; however, such an assessment was not performed in this case.

Most LPMs are not intended to accomplish narrow-complex pacing. Patients with an indication for cardiac resynchronization therapy typically require transvenous pacing leads. Compared with transvenous pacing, pacing-induced cardiomyopathy is less common but still occurs with leadless systems.[6] Wireless left ventricular pacing using the WiSE CRT System (EBR Systems, Inc) device is an emerging alternative to cardiac resynchronization therapy via transvenous coronary sinus lead. This device features a wireless endocardial left ventricular electrode that detects the pacing impulse from a co-implanted right ventricular device to produce ventricular synchrony.[7] The right ventricular device used with the WiSE CRT System is typically a traditional pacing lead; however, a fully leadless system using a right ventricular Micra device has been reported.[8]

As opposed to multidevice solutions and the attendant risk of multiple procedures, leadless conduction system pacing offers the promise of ventricular synchrony delivered in a single device. Temporary left bundle area pacing has been shown to be technically feasible using the WiSE CRT System electrode, and leadless left bundle area pacing is an area of ongoing exploration.[9] Though the safety profile of right ventricular LPMs is well established, there is more uncertainty regarding the safety of WiSE CRT System implantation because of the risk of accessing arterial circulation and the substantial rate of pericardial effusion observed in early studies.[10] Although this patient had a low pacing burden, the ability to safely achieve conduction system pacing with right ventricular leadless devices is promising for patients with high pacing burdens.

One challenge in successful conduction system pacing is anatomical variation in the His-Purkinje system. An advantage of right bundle-branch pacing is that, unlike the His bundle and the left bundle, the right bundle has a predictable course with low anatomical variability. The anatomical course of the right bundle branch is straight, passing through the muscular portion of the interventricular septum, then entering the base of the medial papillary muscle before distributing into Purkinje fibers to supply the right ventricle.[11] Targeting pacemaker placement in the midseptum superior to the base of the medial papillary muscle may facilitate consistent and intentional capture of the right bundle branch. Electrophysiologic parameters to distinguish His bundle and right bundle capture have been previously reported.[4]

A transient, delayed-onset AV block after right ventricular LPM implantation is possible, and prolonged observation is reasonable. In this case no medical therapy or device repositioning was required. Narrow-complex pacing with leadless devices is also achievable. Further study of techniques, limitations, and complications related to intentional right-ventricular leadless conduction system pacing is warranted.

Article Information

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