Acute increase in pacing capture threshold and impedance post–leadless pacemaker implant with spontaneous resolution

Jian Liang Tan, MD, Andrew E. Epstein, MD, FHRS, Timothy M. Markman, MD, FHRS

From the Section of Electrophysiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

Introduction

Leadless pacemakers (LPMs) have been increasingly used owing to advantages compared to traditional transvenous pacemakers, including longer battery life in patients with minimal pacing requirements, lower risk of lead-related complications, and a negligible risk of infection.[1,2]

The Micra AV (Medtronic Inc, Minneapolis, MN) LPM has 4 nitinol tines that are separate from the sensing and pacing electrode and are used to anchor the device in the right ventricular myocardium.[3] This design allows for low pacing thresholds as with traditional transvenous leads. During the implantation procedure, multiple repositioning attempts may be required to achieve an optimal pacing threshold prior to device release. Device parameters are often serially measured during the procedure to assess the device stability and performance. A capture threshold of ≤1.0 V at a 0.24 ms pulse width is a valuable target to achieve at initial implantation and subsequent follow-ups for optimal battery longevity.[4] Acute change in device parameters after deployment may require immediate attention owing to the potential for device dislodgement or malfunction.[2,4] Here we report a case of acute transient elevation of pacing capture threshold and impedance following implantation of a Micra AV LPM that subsequently improved without intervention.

Case report

A 23-year-old man with no other significant past medical history underwent LPM implantation (Micra AV; Medtronic Inc, Minneapolis, MN) for infrequent paroxysmal atrioventricular block. The LPM was successfully implanted in a septal position at the first deployment using a standard approach including femoral venous access with 3000 IU of heparin given following sheath insertion. Once the LPM was deployed to the interventricular septum, a pull-and hold test was performed, which confirmed fixation of all 4 tines. Device parameters were interrogated after deployment (Figure 1A), after the pull-and-hold test, and again following release of the device. All parameters were within manufacturer-recommended ranges during these evaluations (pacing threshold 0.75 V at a 0.24 ms pulse width, impedance 670 Ω, R wave was 3.0 mV). Final programming was set to VVI mode with a lower rate limit of 40 beats per minute. Intracardiac echocardiography was used during the LPM implantation. The LPM was seen well seated in the ventricular septum and the proximal end of the LPM was below the tricuspid leaflet. No complication was observed after sheath removal.

When the device was checked 1 hour after implant, the capture threshold and pacing impedance had increased (pacing threshold 1.63 V at a 0.24 ms pulse width, impedance 900 Ω) with adequate and stable R-wave sensing of 3.4 mV. The pacing threshold returned to <1.0 V at a 0.24 ms pulse width; the impedance was consistent and <600 Ω with a stable R-wave amplitude when interrogation was performed in the standing position. A chest radiograph showed appropriate device positioning (Figure 1B). A decision was made

KEY TEACHING POINTS

- Acute change in device parameters after leadless pacemaker (LPM) deployment may occur even if optimal device parameters were obtained at LPM implantation and all 4 tines were confirmed fixated to the myocardium under fluoroscopy.
- Careful assessment and management of LPM parameters after implantation is essential.
- Conservative management and close monitoring for the trend of the LPM parameters are reasonable considerations in patients with acute elevation of pacing threshold and impedance after LPM implantation when stable R-wave sensing is present.

KEYWORDS Leadless pacemaker; Capture threshold; Impedance; Device thrombus; Cardiac implantable electronic device
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Address reprint requests and correspondence: Dr Timothy M. Markman, Department of Electrophysiology, Hospital of the University of Pennsylvania, Pavilion Level 2, One Convention Avenue, Philadelphia, PA 19104. E-mail address: timothy.markman@pennmedicine.upenn.edu.

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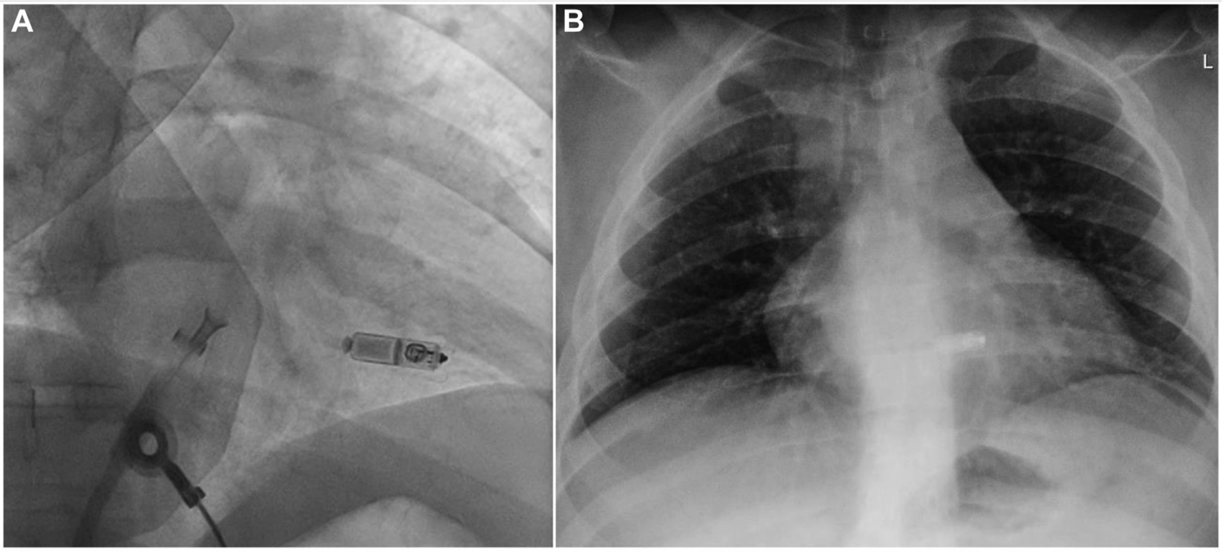

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Time	Capture Threshold, V at (ms)	Impedance, Ω	Sensed R wave, mV	Battery Life, years
At implant	0.75 (0.24)	670	3.0	>10
Post-implant	0.88 (0.24)	680	3.2	>10
1-hour later	1.63 (0.24)	900	3.4	>10
3-hour later	1.88 (0.24)	890	3.5	>10
6-hour later	1.88 (0.24)	964	18.8	>10
Day 1	1.63 (0.40)	700	>20	5.1
Day 2	1.63 (0.40)	650	>20	4.9
Day 4	1.63 (0.40)	640	>20	6.1
Day 6	1.25 (0.40)	540	>20	>10
Day 9	1.50 (0.40)	520	>20	>10
Day 12	0.88 (0.40)	550	>20	>10
Day 18	0.88 (0.40)	530	>20	>10
Day 31	0.75 (0.40)	490	>20	>10
Day 58	0.63 (0.40)	610	>20	>10

Figure 1 **A:** Right anterior oblique view of the position of leadless pacemaker implant. **B:** Chest radiograph with stable position of the leadless pacemaker. **C:** Trends of the leadless pacemaker parameters.

to closely monitor the LPM parameters. The final pacing output was set at 4.4 V at a 0.40 ms pulse width, which correlated with an estimated battery life of 4.8 years.

Frequent remote monitoring was performed after discharge. Trends for the measured LPM parameters at supine position are shown in Figure 1C. R-wave sensing improved rapidly, followed by improvement in the impedance and finally the pacing threshold over the course of 2 weeks. He remained in sinus rhythm and required minimal ventricular pacing (<0.1%). After 2 weeks, the pacing output was reprogrammed to 2.1 at a 0.4 ms pulse width. Capture management remained as adaptive with amplitude safe margin of +1.00 V, which correlated to an estimated battery life of >10.0 years.

Discussion

This case highlights an uncommon course in LPM parameters after initial device implantation. A previous study reported that elevated pacing threshold after LPM implant may require LPM retrieval and reimplantation in 1.3% of the patients.[1] In our case, an optimal pacing threshold and impedance were obtained at implantation. However, an acute rise in those parameters was noted after implantation. These acute changes initially led to concern for dislodgement or instability of the device, potentially related to interaction with the tricuspid valve apparatus. This was felt to be less likely for several reasons, including excellent fixation of all 4 tines to the myocardium during implantation and stability of the device on postimplant radiography. The excellent and consistent R-wave sensing and elevated impedance also argued against dislodgement or instability. Furthermore, intracardiac echocardiography was used to guide the placement of the LPM, which had clearly demonstrated the device to be placed beyond the tricuspid leaflets and without any mechanical interaction between the device and the valve apparatus.

Other possible explanations for an elevated pacing threshold, such as tine fractures, have been reported.[5–7] This has been observed rarely and often leads to pacing failure owing to instability of the device. LPMs with broken tines often present with normal R-wave sensing and impedance but intermittent pacing failure despite programming of the LPM to the maximum pacing output. Fractured tines often require replacement of the device. In our case, all device parameters including sensing, impedance, and pacing threshold improved slowly over the course of 2 weeks. Therefore, tine fracture would not have explained the trend of LPM parameters seen in our case.

Though the precise mechanism of acute increase in threshold and impedance is unknown in our case, we suspected acute thrombus may have developed between the tip of the LPM and the myocardium, which self-resolved over time. Increased LPM pacing thresholds have been reported previously and are associated with a higher implant threshold or a lower pacing impedance.[8] Hagiwara and colleagues[9] reported a case of presumed thrombus on day 3 after LPM implantation with an acute drop in sensed R wave corresponding to an elevation of pacing impedance and pacing threshold, both of which improved without any intervention. We also hypothesize that the positional changes were consistent with a thrombus between a portion of the device and the myocardium that was limiting contact only in certain positions. This may also explain the differences in sensing noted in our case, where there was no acute reduction in sensed R-wave amplitude and pacing impedance was only mildly increased. Since a standard heparin protocol was used in our case, it remains unclear why thrombus formation would have occurred in this case. It is possible that a small gap may have been present between the distal end of the LPM electrode and the myocardial surface. This potential gap might create an area of sluggish blood flow that could serve as a nidus for clot formation adjacent to 1 aspect of the device. This layer of thrombus formation could also potentially explain the trend in elevated impedance and capture threshold after LPM implantation.

Conclusion

This case highlights the importance of evaluating LPM parameters after implantation. An acute increase in the pacing threshold and impedance may occur after implantation. Although unstable lead position is a consideration, acute thrombus may explain abrupt rise in pacing threshold and impedance with stable sensing. Conservative management with close monitoring for the trend data might be reasonable in these patients.

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