Tricuspid regurgitation complicating leadless pacemaker implantation: Surgical intervention for pacemaker removal and tricuspid valve replacement

Vikas Kilaru, DO, MS,[1] Alexander Adams, DO,[2] Priyadarshini Dixit, MD,[2] Joon Ahn, MD, FACC, FHRS[2]

From the [1]Northeast Georgia Medical Center, Graduate Medical Education, Gainesville, Georgia, and [2]Georgia Heart Institute, Northeast Georgia Health System, Gainesville, Georgia.

Introduction

Leadless pacemakers (LPs) represent a revolutionary advancement in cardiac pacing, offering a number of advantages over conventional transvenous devices. Many of these advantages lie in the fact that the absence of a pacemaker generator and pacing lead may result in less pocket and device infections, venous thrombosis and occlusion, and tricuspid valve regurgitation (TR) and dysfunction. We present a case illustrating an uncommon major complication of severe TR after LP implantation necessitating surgical intervention.

Case Report

An 85-year-old female patient was referred to the structural heart clinic for evaluation of severe TR after she was noted to have worsening dyspnea and exercise intolerance 6 weeks after atrioventricular node ablation and implantation of a Medtronic Micra LP. Medical history included heart failure with preserved ejection fraction, coronary artery disease, chronic kidney disease, and nonvalvular persistent atrial fibrillation. She had clinical signs of right-sided heart failure with hepatic congestion of the liver, ascites, and peripheral edema. Transthoracic echocardiogram (TTE) obtained 6 weeks post pacemaker implantation during hospitalization for decompensated heart failure showed severe TR with a central regurgitant jet, which had significantly worsened from moderate severity on TTE obtained 6 months before device implantation (Supplemental Video 1). The TTE also showed severe right atrial and right ventricle (RV) dilatation with reversal of hepatic vein flow. Routine device checks were performed and right ventricular pacing was found to be 93% and 80.6% at 2 weeks and 3 months after placement, respectively.

Transesophageal echocardiogram with 3-dimensional imaging was performed to further assess the tricuspid valve, which showed poor coaptation of the septal and posterior leaflet in 3- dimensional imaging of the tricuspid valve,

KEY TEACHING POINTS

- It is essential to recognize that although leadless pacemakers offer advantages over traditional pacemakers, they can still cause significant complications, such as tricuspid regurgitation. Clinicians should be aware of and able to recognize signs of dysfunction post implantation, as early detection and intervention can significantly improve patient outcomes.
- When selecting candidates for leadless pacemaker implantation, it is important to consider individual patient factors that may predispose them to complications. Thorough preimplantation assessment and selection criteria can help minimize risks. Post implantation, follow-up protocols should be implemented to promptly address any emerging issues, thus optimizing patient safety and device efficacy.
- The management of tricuspid regurgitation caused by leadless pacemaker implantation often requires a multidisciplinary approach. Collaboration among cardiologists, cardiothoracic surgeons, and electrophysiologists is crucial to evaluate the extent of the complication, plan for the optimal route of pacemaker removal, and potentially perform tricuspid valve replacement, ensuring comprehensive patient care.

KEYWORDS Leadless pacemaker; Pacemaker complication; Tricuspid valve regurgitation; Tricuspid valve repair; Surgical pacemaker explanation; Pacemaker implantation; Structural heart disease

(Heart Rhythm Case Reports 2025;11:108–110)

Address reprint requests and correspondence: Dr Vikas Kilaru, Northeast Georgia Medical Center, Graduate Medical Education, 6305 New Holland Way NE, Gainesville, GA 30501. E-mail address: vikas.kilaru@nghs.com.

https://doi.org/10.1016/j.hrcr.2024.10.017

causing severe TR (Supplemental Video 2) with an effective regurgitant orifice area of 1.55 cm^2 and vena contracta of 0.72 cm. The patient underwent coronary computed tomography angiography to assess the anatomy of the tricuspid valve for evaluation of percutaneous vs surgical treatment options. Computed tomography angiography showed evidence of the Micra LP causing interference with the function of the anterior tricuspid valve leaflet, resulting in malcoaptation of the leaflets (Figures 1 and 2). The pacemaker was noted to be implanted in a superior location on the anterior right ventricular free wall within the right ventricular outflow tract, which led to an abnormal interaction with the sub-valvular anterior tricuspid valve apparatus. The distance between the proximal end of the LP and the tricuspid valve annulus was noted at 3.8 mm. Imaging demonstrated that the anterior leaflet of the tricuspid valve was being drawn superiorly and anteriorly with ventricular systole restricting its motion, resulting in malcoaptation with the septal and posterior leaflets, leading to severe TR. Direct mechanical damage to the subvalvular apparatus of the tricuspid valve was determined to be the main mechanism of her worsening TR and was most likely irreversible.

A multidisciplinary discussion was held with the structural heart and cardiothoracic surgery teams who concluded that the best option for the patient would be explantation of the LP and repair of the tricuspid valve via surgical intervention. She subsequently underwent successful tricuspid valve replacement with a 33-mm Medtronic Mosaic mitral bioprosthetic valve (as it was deemed intraoperatively unsuitable for repair) and removal of the LP. The pacemaker was noted to be located in the body of the RV along the anterior septum with no evidence of bleeding along the RV free wall after removal.

Postoperatively, the patient required temporary ventricular pacing from her epicardial pacing wires and briefly from placement of a transvenous pacing catheter due to atrial fibrillation with slow ventricular response, but had recovery of atrioventricular conduction within 5 days postoperatively with intermittent rapid ventricular conduction. Her hospital course was complicated by respiratory failure requiring reintubation due to congestive heart failure and pleural effusion requiring chest tube placement, aggressive diuresis, and intermittent inotropic support. She did not require re-implantation of a permanent pacemaker. Post-implantation 30-day TTE demonstrated a normal functioning bioprosthetic tricuspid valve with mild TR.

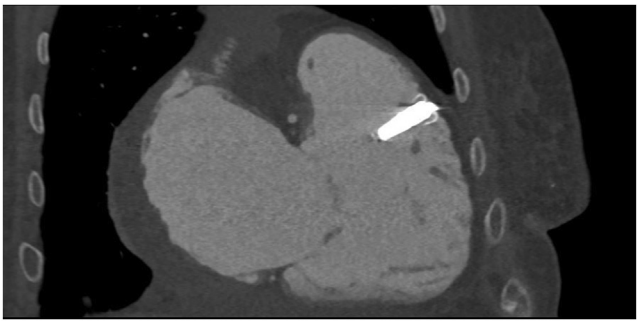

Figure 1 Computer tomography angiogram showing the leadless pacemaker in the right ventricular outflow tract.

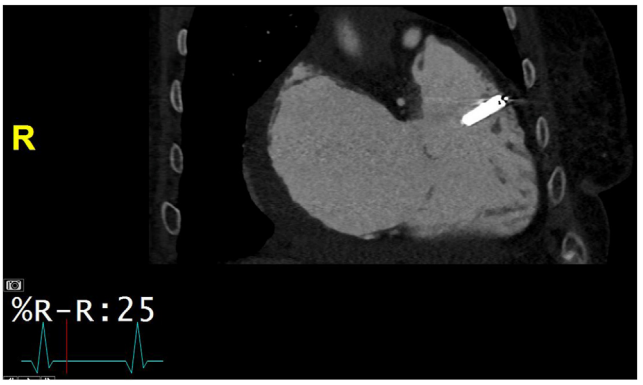

Figure 2 Leadless pacemaker interference with anterior cusp of tricuspid valve during systole.

Discussion

Leadless pacing represents an innovative method of pacing the heart, offering several advantages over traditional transvenous pacemakers. Benefits of LPs include reduced risk of infection, hematoma, vein thrombosis, skin erosion, pneumothorax, lead dislodgment, and tricuspid valve impingement and perforation.[1] Currently 2 US Food and Drug Administration–approved LPs exist, Micra and Aveir (Abbott), and have been approved for bradycardia indications to provide single-chamber right ventricular pacing.[2] Studies have reported successful implantation rates >99%, with complication rates of 3.4% for the Micra pacemaker and a successful implantation rate of 98.3% with a complication-free rate of 90.3% at 90 days.[3,4]

Tricuspid valve dysfunction after implantation of a traditional transvenous pacemaker is well documented and LP therapy was developed to help remedy this common complication. However, tricuspid valve dysfunction after LP implantation is increasingly being recognized. One study reported worsening tricuspid valve function in up to 43% of patients at 12 months after LP implantation.[5] The causes of these findings are potentially multifactorial. One explanation is that the septal location of implantation—the recommended site of implantation for the Medtronic Micra device—is more proximal to the valve, resulting in higher risk of interaction with the subvalvular apparatus and higher rates of TR compared with apical implantation. Multiple redeployments of the device during the implantation process may also contribute to injury to the valve and subvalvular apparatus. In addition, RV pacing has been found to be an independent risk factor for development of TR due to asynchronous activation of the RV leading to alterations in RV dimensions, annular dilatation, and subsequent TR.[6]

Our case highlighted the importance of optimal LP device implantation to avoid potentially injurious interactions with the tricuspid valve. The implantation of the device into a high anterior location within the right ventricular outflow

tract led to its interaction with the subvalvular apparatus and worsening TR. Contrast was used to visualize the interventricular septum during implantation and a septal location was confirmed before advancement of the delivery sheath. During advancement of the sheath, we suspect that the tip may have been directed more anteriorly and superiorly and inadvertently into the anterior free wall of the right ventricular outflow tract. The device was deployed only once without repositioning. Furthermore, pre-existing severe RV dilatation may have contributed to a leftward shift of the heart, which can lead to more anteriorly directed sheath and device positions. In addition, our patient underwent atrioventricular nodal ablation and required a high percentage of RV pacing and the resulting RV dyssynchrony may have also contributed to worsening TR.

The mechanisms behind TR after LP implantation are complex and not fully understood. Initial beliefs that TR was due mainly to electrode impairment of valve closure have evolved to include the possibility that crossing the tricuspid valve at any point could lead to clinically significant damage to the valve, chordae tendineae, and papillary muscle.[5] Accordingly, implanting LPs further from the valve into a more apical location seems to decrease rates of tricuspid valve dysfunction. Although septal positioning reduces QRS duration and surgical complications like myocardial perforation and pericardial effusion, it is also linked to increased TR rates, posing a clinical challenge. The presence of pre-existing tricuspid valve disease may also be an independent risk factor for worsening TR, which requires further study.

To address these issues, various approaches are being explored. One existing safety mechanism involves using guiding sheaths over the LP until it is safely positioned in the ventricle, protecting the valve. In addition, the use of right ventriculography has been found to improve accuracy of LP placement and reduce fluoroscopy time, making it useful for difficult anatomy. The use of 3-dimensional echocardiography alongside fluoroscopy has also been beneficial in cases of anatomic variations.[6,7]

Conclusion

This case highlighted an uncommon but significant complication of worsening TR due to LP implantation. Although LPs offer several advantages over traditional devices and are likely to see increased use, their potential to cause TR through interaction with the tricuspid valve is a concern. This case emphasizes the need for heightened vigilance post implantation and the importance of multidisciplinary collaboration in managing such complications. Future studies are essential to identify optimal strategies for preventing and managing these issues, aiming to enhance safety and improve patient outcomes in LP implantations.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: The authors have no conflicts of interest to disclose.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.10.017.

References

1. Ngo L, Nour D, Denman RA, et al. Safety and efficacy of leadless pacemakers: a systematic review and meta-analysis. J Am Heart Assoc 2021;10:e019212. https://doi.org/10.1161/JAHA.120.019212.
2. Steinwender C, Khelae SK, Garweg C, et al. Atrioventricular synchronous pacing using a leadless ventricular pacemaker: results from the MARVEL 2 study. JACC Clin Electrophysiol 2020;6:94–106.
3. Reynolds D, Duray GZ, Omar R, et al. A leadless intracardiac transcatheter pacing system. N Engl J Med 2016;374:533–541.
4. Knops RE, Reddy VY, Ip JE, et al. A dual-chamber leadless pacemaker. N Engl J Med 2023;388:2360–2370.
5. Beurskens NEG, Tjong FVY, de Bruin-Bon RHA, et al. Impact of leadless pacemaker therapy on cardiac and atrioventricular valve function through 12 months of follow-up. Circ Arrhythm Electrophysiol 2019;12:e007124. https://doi.org/10.1161/CIRCEP.118.007124.
6. Yarmohammadi H, Batra J, Hennessey JA, Kochav S, Saluja D, Liu Q. Novel use of 3-dimensional transthoracic echocardiography to guide implantation of a leadless pacemaker system. HeartRhythm Case Rep 2022;8:366–369.
7. Mattson AR, Zhingre Sanchez JD, Iaizzo PA. The fixation tines of the Micra leadless pacemaker are atraumatic to the tricuspid valve. Pacing Clin Electrophysiol 2018;41:606–1610.