

Premature ventricular contraction–induced polymorphic ventricular tachycardia after leadless pacemaker implantation: A unique adverse effect of leadless pacing



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Introduction

The MICRA transcatheter leadless pacing system (Medtronic, Minneapolis, MN) is a revolutionary shift in pacemaker technology.¹ The leadless design allows pacing therapies to be offered through a minimally invasive transcatheter approach via the femoral vein to patients in whom traditional implants would have posed either excessive technical or excessive infection risks. As with any novel technology, the possibility of unique complications must be thoroughly described. We demonstrate a case of recurrent premature ventricular contraction (PVC)-induced polymorphic ventricular tachycardia (PMVT) after MICRA leadless pacemaker deployment, which resolved with extraction and reimplantation at a different right ventricular site.

Case report

A 74-year-old patient with ankylosing spondylitis, on long-standing immunosuppression with infliximab, and with remote history of coronary artery bypass grafting with preserved left ventricular ejection fraction presented with recurrent syncope. Inpatient telemetry and baseline electrocardiogram demonstrated sinus bradycardia with a normal PR interval and right bundle branch block with QRS duration of 160 ms. Bedside right carotid sinus massage resulted in a 6-second sinoatrial arrest and reproduced his clinical symptoms. The remainder of his physical examination revealed a chronic non-healing left lower extremity skin ulcer, which was thought to be the nidus of recurrent bacteremia, requiring previous hospital-

izations on a number of occasions and prompting chronic wound care and recurrent antibiotic therapy. Based on a high risk of infectious compromise of a traditional transvenous system, a leadless pacemaker was recommended.

The initial leadless MICRA implant was uncomplicated, with a high right ventricular septal position targeted (Figure 1). The device was delivered on the first attempt with initial R waves of 20 mV and an impedance of 1110 ohms and capture of 0.25 mA at 0.24 ms. In the perioperative period the patient demonstrated episodes of PMVT. The next day interrogation revealed high ventricular rate counters and telemetry demonstrated short-long-short sequences of PVC-induced PMVT (Figure 2) associated with near-syncope and dizziness.

The corrected QT (QTc) interval was noted to be between 460 and 500 ms at baseline and immediately after pacemaker implantation. The patient was not on any QT-prolonging medications. In light of a pre-existing right bundle branch block with QRS duration of ~160 ms, the QTc was considered unchanged from prior to the implantation of the pacemaker. Laboratory evaluation revealed normal serum electrolytes, TSH, and free T4 levels and troponin levels. Telemetry demonstrated frequent unifocal PVCs of left bundle, leftward axis morphology. These were not present prior to the pacemaker implantation. The PVC-induced PMVT occurred at a back-up pacing rate of 50/min instigated by a “short-long-short” sequence (Figure 2). The QT interval of the paced beat was noted to be as long as 600 ms (Figure 2). Spontaneous PVCs after a paced complex resulted in PMVT (R-on-T phenomenon). The initial approach was to increase the basal pacing rate to 80 beats per minute to eliminate this phenomenon. This strategy was unsuccessful in reducing ventricular ectopy and the patient continued to have PMVT. The patient’s history of remote cardiac bypass raised concern for cardiac ischemia and prompted a left heart catheterization, which demonstrated no new obstructive coronary artery disease or disruption of the prior grafts after

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KEY TEACHING POINTS

- Leadless pacemaker implantation will result in unique complications distinct from traditional device implantation. Ventricular arrhythmias arising following implantation of a leadless pacing system should be recognized as potentially secondary to the implant position of the device.
- Leadless pacing systems have no ability to store ventricular high-rate electrograms. Episodes of ventricular high rates should raise concern for ventricular arrhythmias in these patients.
- Extraction of leadless devices is safe and effective. An experienced extraction team should be available at centers implanting leadless pacing devices.

the pacemaker implantation. Close observation revealed spontaneous PVCs similar to the paced QRS morphology (Figure 3). At this point the device was considered to be causative of the arrhythmia.

The right femoral vein was re-accessed with ultrasound guidance, and a 23 French Micra introducer sheath (Medtronic, Minneapolis MN) was advanced under fluoroscopic guidance to the right atrium and a new MICRA was delivered at the mid-septal right ventricular location with sensing of 16.7 mV and capture of 0.5 mV at 0.24 ms. An angiogram of the 2 devices in place reveals the high septal positioning of the index device (Figure 1). Following successful deployment of the second device, an Amplatz 4 French 10-mm Gooseneck Snare (Covidien/Medtronic, Plymouth, MN) was advanced through the delivery sheath and positioned over the retrieval pin on the previously implanted MICRA. The cup was then advanced to the pacemaker, and the device was captured and retrieved. There were no cardiac or vascular complications.

The patient was discharged 48 hours later, with no recurrent spontaneous ventricular ectopy, with a wearable defibrillator (LifeVest, Zoll, Pittsburgh, PA) and was seen at 30-day follow-up, with pacemaker interrogation revealing no ventricular high-rate events, at which time the LifeVest was discontinued.

Discussion

The MICRA leadless pacemaker has been demonstrated as an effective and safe alternative to single-chamber transvenous pacing systems, with over 99% of devices successfully implanted with limited complications.¹ Observed complications include device dislodgement, cardiac perforation, and high capture thresholds. These complications are well established and occur with lower frequency than in historical transvenous pacing systems.¹ Nonetheless, our experience with leadless pacemakers continues to grow and, as in the case of any new technology, unforeseen consequences can and will occur. To our knowledge, the MICRA leadless

pacemaker has been extracted only for systemic infection and loss of capture.^{2,3} Our case illustrates the first published report of PMVT associated with the MICRA leadless pacemaker prompting system extraction and reimplantation with resolution of PMVT.

Conservative basal pacing rates resulted in short-long-short ventricular excitation sequences, during which spontaneous PVCs occurring immediately after the paced complex induced PMVT. This is a well-described cause of torsades de pointes.⁴ The underlying mechanism is thought to be marked temporal dispersion of refractoriness during prolonged QTc, increasing vulnerability to early depolarization by PVCs inducing PMVT.⁴ Pacemaker function was normal and undersensing was not observed. Increases in baseline pacing rates lessened the density of events but did not eliminate PVC-induced PMVT.

The exact underlying mechanism for the observed proarrhythmic effect is unclear, but is most consistent with irritation of the right ventricular myocardium at the site of MICRA pacemaker implantation. Right ventricular irritation has been previously described in both retained fragments of pacemaker leads and loops of device leads in the right ventricle or right ventricular outflow tract.^{5,6} This is further supported by unifocal PVCs, which were noted after MICRA implantation, that were similar to the paced QRS complex. This phenomenon could only be rectified with extraction and reimplantation of the MICRA to a new position in the right ventricle, resolving both spontaneous unifocal PVCs and PVC-induced PMVT.

Conclusion

Our case report demonstrates that local irritation to the right ventricular myocardium from a leadless pacemaker can occur, and clinicians should be aware of this potential novel adverse effect of this new technology. We think that this phenomenon is unique and perhaps more common with a



Figure 1 Fluoroscopic image depicting high septal location of index leadless device and relative position to newly placed leadless pacemaker.

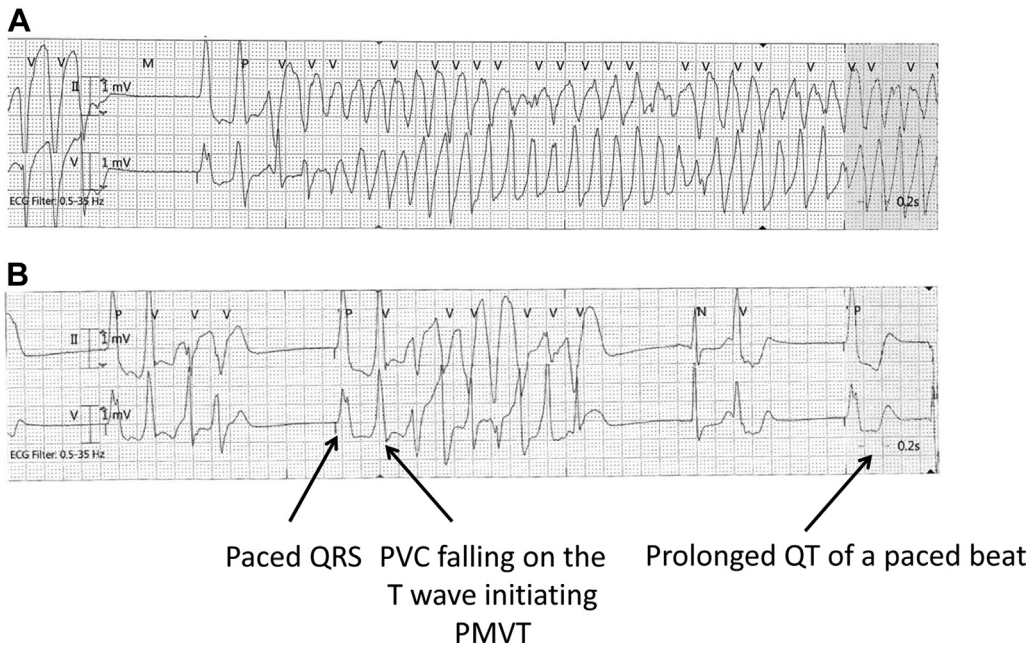


Figure 2 A, B: Telemetry strips depicting “short-long-short” sequence and R-on-T phenomenon causing spontaneous premature ventricular contraction (PVC)-induced polymorphic ventricular tachycardia (PMVT).

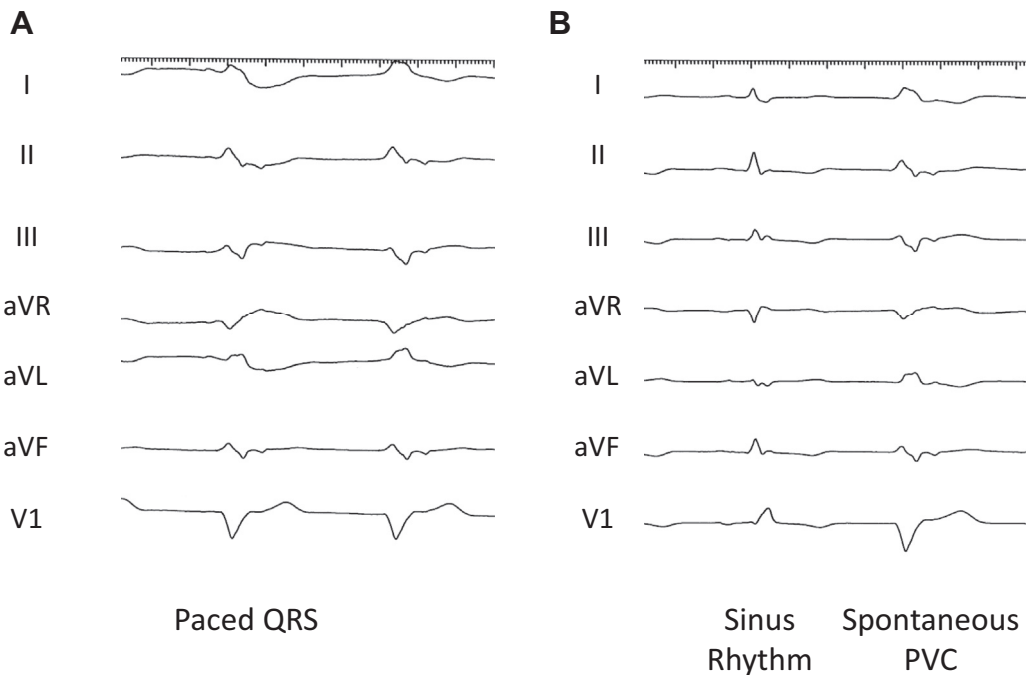


Figure 3 The QRS morphology of a paced beat (A) is similar to that of a spontaneous premature ventricular contraction (B). PVC = premature ventricular contraction.

leadless pacemaker than with a conventional transvenous pacemaker, owing to difference in design, with an overall larger device hardware profile than the conventional pacemaker lead, and to technique of implantation, with forward pressure on the device to deploy multiple tines into the trabecula of the myocardium.

**Appendix
Supplementary data**

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2018.01.006>.

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