

# Syncope after successful implantation of atrioventricular synchronous leadless pacemaker caused by polymorphic ventricular tachycardia



Ahmad Halawa, MD, Martin Aguilar, MD, PhD, Michael O. Sweeney, MD, Sunil Kapur, MD, FHRS

From the Cardiac Electrophysiology Department, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts.

## Introduction

Leadless pacing is an alternative to transvenous single-chamber ventricular pacing (VVI/R) with a favorable safety and efficacy profile in selected patients.<sup>1</sup> Until recently, the Micra (Model MC1VR01; Medtronic Inc, Minneapolis, MN) was the only leadless pacing system approved for clinical use in the United States. The leadless pacemaker is an attractive option for patients with permanent atrial fibrillation who require infrequent ventricular back-up pacing and in those in whom atrioventricular (AV) synchrony is not pursued, especially when vascular access is limited (ie, hemodialysis patients) or the risk of intravascular infection is deemed high. The recently approved Micra AV (Model MC1AVR1; Medtronic Inc) represents a major development in leadless pacing technology. Micra AV uses its accelerometer to sense the atrial mechanical events and deliver leadless AV-synchronous ventricular pacing (VDD).<sup>2</sup> The Micra AV therefore has the potential to provide VDD pacing with a lower rate of pocket/lead-related complications vs transvenous systems. Pacing-facilitated ventricular arrhythmias have previously been reported after Micra implantation.<sup>3–5</sup> Here, we report the first case of bradycardia-dependent polymorphic ventricular tachycardia (PMVT) caused by atrial undersensing in a patient implanted with the Micra AV leadless pacemaker.

## Case report

A 92-year-old woman with history of severe aortic stenosis, hypothyroidism, type 2 diabetes mellitus, and recurrent

urinary tract infections presented to the hospital with progressive fatigue and dyspnea over a 2-week period and found to be in sinus rhythm with complete heart block and stable junctional escape rhythm (Supplemental Figure 1a). Blood biochemistry was within normal limits, as were high-sensitivity cardiac biomarkers and thyroid-stimulating hormone levels. Transthoracic echocardiogram showed mild left ventricular hypertrophy with preserved left ventricular systolic function. Considering her infectious risk and comorbidities but intact sinus node function, she was referred for Micra AV insertion.

The patient was brought to our electrophysiology laboratory in the fasting state. Under conscious sedation, ultrasound-guided right femoral venous access was obtained and upsized to accept the 27 French delivery system. The Micra AV was successfully deployed on the right ventricular mid-septum (Figure 1) using the standard implant technique. After sheath removal, hemostasis was secured with a hemostatic silk suture and manual compression; there were no acute complications. The pacemaker was programmed to VDDR 60–105 beats per minute (bpm) and found to have satisfactory acute implant parameters (impedance 710 W; R wave 8.3 mV; ventricular capture threshold 0.38 V at 0.24 ms). Furthermore, the leadless pacemaker appropriately sensed the atrial mechanical events and delivered AV synchronous ventricular pacing. She was returned to her inpatient bed in stable condition.

Overnight, the patient suffered a syncopal event while supine with seizure-like movements for about 12 seconds correlating with an episode of PMVT (Figure 2); the tachycardia terminated spontaneously. Investigations including blood biochemistry, cardiac enzymes, a transthoracic echocardiogram, and noninvasive ischemia assessment were unremarkable.

We felt that undersensing of the A wave led to paced beat at the lower VVI mode rate followed by premature ventricular contractions (PVCs), which triggered a short-long-short (S-L-S) status leading to the polymorphic arrhythmia. Pacing mode was kept as VDD but the A4 (atrium active contraction) phase sensing property was adjusted, and we added beta

**KEYWORDS** Atrioventricular synchrony; Leadless pacemaker; Polymorphic ventricular tachycardia; Short-long-short; Syncope (Heart Rhythm Case Reports 2020;6:503–506)

**Funding:** This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Conflict of interest: Drs Ahmad Halawa, Martin Aguilar, and Michael Sweeney have no disclosures. Dr Sunil Kapur has received honoraria for lectures from Abbott Inc and Medtronic Inc. Dr Kapur consults for and has received honoraria from Novartis. **Address reprint requests and correspondence:** Dr Ahmad Halawa, Clinical Cardiac Electrophysiology Department, Brigham and Women's Hospital, 3rd Floor, Watkins Cardiovascular Center, Suite C, Carl J. and Ruth Shapiro Cardiovascular Center, 70 Francis Street, Boston, MA 02115. E-mail address: [ahalawa@bwh.harvard.edu](mailto:ahalawa@bwh.harvard.edu).

### KEY TEACHING POINTS

- Micra AV (Medtronic Inc, Minneapolis, MN) is a newly developed VDD pacemaker that can be associated with life-threatening arrhythmias like polymorphic ventricular tachycardia.
- Arrhythmia induction is short-long-short mechanism dependent and atrial undersensing plays an important role in initiating the arrhythmia.
- Noninvasive measurements can be helpful in preventing further arrhythmia.

blockers to her medications. Intervention resulted in a lower sinus node rate, no further PVCs, and better atrial activity sensing (Figure 3).

Repeat ECG showed well-synchronized paced ventricular rhythm (Supplemental Figure 1b) and no further arrhythmia was noted on telemetry monitoring. She recovered and was discharged home to the care of her family.

### Discussion

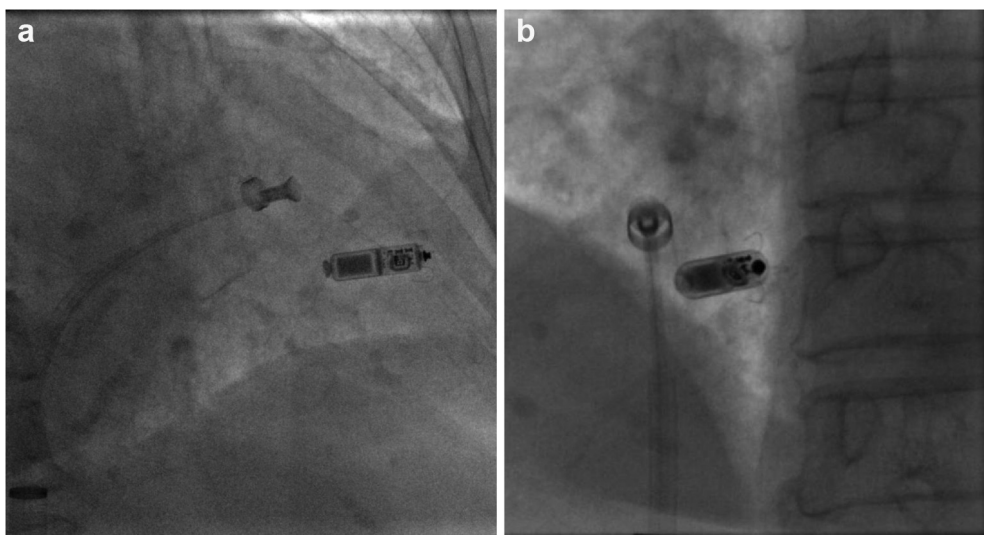
Novel technologies offer new opportunities and challenges. The recently approved Micra AV pacemaker represents a major development in leadless pacing, having the potential of delivering leadless AV-synchronous ventricular pacing (VDD). Here, we report the first case of bradycardia-dependent PMVT caused by atrial undersensing in a patient implanted with the Micra AV leadless pacemaker.

Single-chamber VVI leadless pacing is commonly used in clinical practice and associated with lower pocket- and lead-related complications when compared to transvenous pacing systems.<sup>1,6,7</sup> Until very recently, leadless pacing with the Micra device (Model MC1VR01) was limited by single-

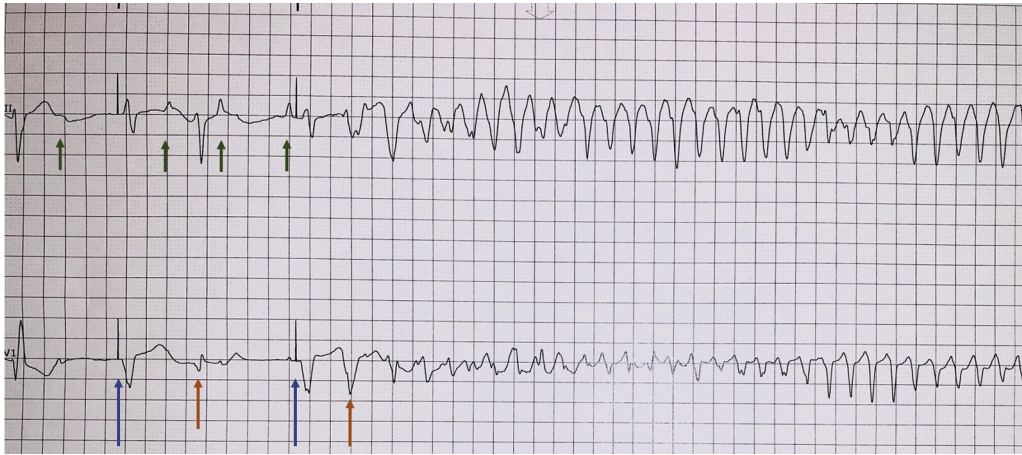
chamber ventricular sensing, making it unsuitable for patients in which AV synchrony is desired. The Micra AV (Model MC1AVR1) uses its accelerometer to sense the atrial mechanical events in sinus rhythm to deliver atrial-triggered ventricular pacing (VVD), thereby preserving AV synchrony. The implant technique and delivery system for Micra AV are the same as for Micra such that Micra implanters can seamlessly opt for the Micra AV system on the basis of the clinical pacing indication. The Micra Atrial tracking using a Ventricular accelerometer 2 (MARVEL 2) study compared leadless VDD vs VVI pacing with the Micra AV in 75 patients with sinus rhythm and complete heart block. Atrioventricular synchrony increased from 26.8% to 89.2% with VVI vs VDD pacing, respectively, and no serious complications were reported.<sup>2</sup> The benefits of atrial-based vs ventricular-based pacing beyond a mild reduction in atrial fibrillation have not been consistently demonstrated, whereas the acute and chronic complication rates are definitely higher with dual-chamber transvenous systems.<sup>8</sup> Nevertheless, the prospect of reliable leadless VDD pacing capabilities will likely significantly expand the indications for leadless pacemakers.

The potential proarrhythmic effect of cardiac pacing is a rare but well-described phenomenon with transvenous pacemakers.<sup>9</sup> Conventional pacing modes such as VVI/R and DDD/R have been observed to facilitate the induction of ventricular tachycardia (VT) and fibrillation (VF). In a combined post hoc analysis of the PainFree Rx II and EnTrust trials, Sweeney and colleagues<sup>10</sup> found that pacing-associated S-L-S sequences were observed in 29.8% of VT/VF events and pacing was adjudicated to be causal in 2.6% to 5.2% of VT/VF episodes. Algorithms to minimize ventricular pacing, such as managed ventricular pacing (Medtronic, Inc, Minneapolis, MN), have also been observed to facilitate and even trigger VT/VF, albeit in an exceedingly small minority of patients.<sup>11,12</sup>

Several hypotheses have been proposed to account for “R-on-T” or S-L-S-mediated PMVT. At a tissue level,



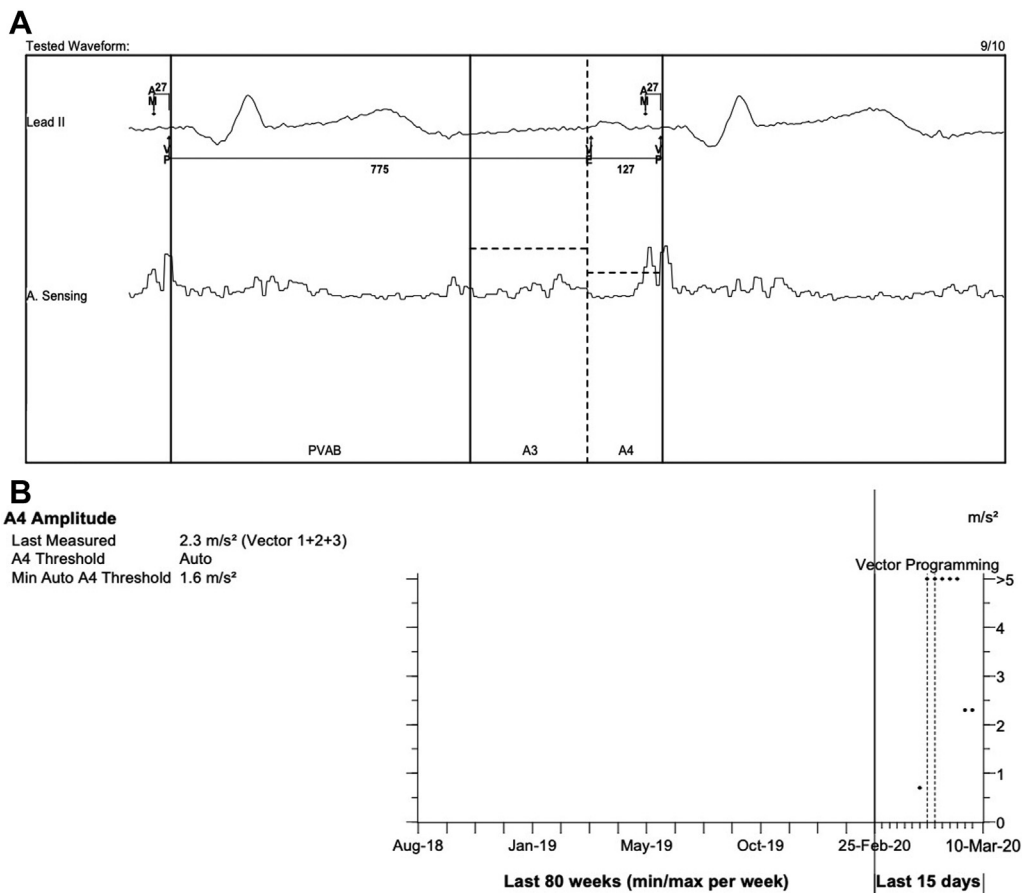
**Figure 1** Thoracic fluoroscopy view depicting the Micra delivery system after deployment of the Micra device (Medtronic, Minneapolis, MN). View is confirming proper placement at the septal aspect of the right ventricle on **a**: standard left anterior oblique view and **b**: right anterior oblique view.



**Figure 2** Telemetry strip showing initiation of polymorphic ventricular tachycardia following a paced beat (blue arrows) after premature ventricular contraction (orange arrows). Dissociated atrial activity is marked with green arrows. Accelerometer sensor failed to detect the mechanical atrial activity and led to premature pacing at the lower programmed rate. Figure shows the short-long-short setup for polymorphic arrhythmia in this patient.

alternations in activation cycle length between paced complexes and PVCs increase the heterogeneity of repolarization, as repolarization is a rate-dependent process, setting the stage for ventricular arrhythmias.<sup>13</sup> An appropriately timed PVC in

the vulnerable period of ventricular repolarization can find parts of the myocardium available for conduction while others may be refractory, leading to functional reentry and PMVT/VF. Fortunately, most patients do not experience



**Figure 3** **A:** The correlation between recorded surface electrocardiogram and accelerometer sensing of ventricular mechanical changes during different phases: A3: passive ventricular filling, A4: active atrium contraction. Shown is a successful electromechanical correlation after adjusting A4 sensing and treatment with beta blockers. PVAB = postventricular atrial blanking period. **B:** Changes in accelerometer sensing threshold over time in this patient. Improper sensing after implantation is noted (threshold > 1.6 m/s<sup>2</sup>). It also shows improvement in the A4 acceleration sensing after adjustments made to sensing parameters and titrating up beta blocker dose over time.



such events, which raises the possibility of subclinical defects in repolarization being unmasked by additional stressors (eg, acute bradycardia, electrolyte abnormalities, ischemia) in those patients that do suffer from S-L-S-mediated PMVT.

Leadless pacemaker-facilitated ventricular arrhythmias have previously been reported with the Micra pacing system.<sup>3–5</sup> Da Costa and colleagues<sup>3</sup> reported a case of VF temporally associated to Micra insertion but without documenting the mechanism of initiation of tachycardia such that a causal relationship cannot be ascertained. More recently, Amin and colleagues<sup>5</sup> and Olsen and colleagues<sup>4</sup> each reported a case of Micra-facilitated sustained ventricular arrhythmias, the former managed with repositioning the Micra and the latter requiring deactivation of the Micra and implantation of a transvenous pacemaker. The number of cases of leadless pacemaker-facilitated or leadless pacemaker-triggered ventricular arrhythmias remains small. However, this may underestimate the true incidence of such events, as the Micra devices are not designed to store arrhythmia events; hence the events could therefore go undetected.

Our case is different and highlights a complication associated with leadless VDD pacing, a novel technology. In our patient, atrial undersensing during VDD pacing led to S-L-S sequences or “R-on-T,” ultimately triggering PMVT. The phenomenon of atrial undersensing leading to PMVT with VDD pacing has previously been described with transvenous pacing systems.<sup>14</sup> The induction of PMVT from an S-L-S sequence is likely a low-probability stochastic event. However, the rate of inappropriate atrial sensing is significantly higher with the Micra AV than with transvenous VDD systems, potentially exposing patients to a higher burden of S-L-S sequences. In fact, in the MARVEL 2 study, AV synchrony was present in 89.2% of patients after a 20-minute rest period but was as low as 69.8% while standing.<sup>2</sup>

Relying on the mechanical forces of the right atrium (RA) leaves this technology vulnerable to RA hemodynamic changes. Enlarged RA, hemodynamic acute shifts, and atrial tachycardia/arrhythmia may compromise the A4 wave sensed by the accelerometer and the device will switch to VVI mode. Therefore, patient selection should consider all the contributing factors.

Further work is needed to ascertain the burden of S-L-S and risk of VT/VF caused by atrial undersensing with the Micra AV pacemaker. Arrhythmia electrogram storage should be within the scope of the available technology and would be very useful in better defining the incidence of atrial undersensing-induced ventricular arrhythmias with the Micra AV.

Elsokkari and colleagues<sup>15</sup> reported a case of PMVT in a patient with complete heart block switched from VDD 50–120 bpm to VVI 50 bpm transvenous pacing. They noted that this change in pacing mode was the functional equivalent of the patient having had *de novo* AV junction ablation. This empiric observation forms the basis for a higher backup pacing rate after AV junction ablation. A similar sequence of events was observed in our patient in that VDD pacing at the sinus rate was replaced by functional VVI pacing at the

lower rate interval because of atrial undersensing. This abrupt change in rate may have acted as an additional stressor to the patient’s ventricular repolarization homeostatic mechanisms. This also highlights the limitations of the accelerometer-based sensing of atrial mechanical events. It is highly likely that this technology will be further improved with subsequent iterations of the device.

## Conclusion

Novel technologies offer new opportunities and challenges. Here, we report the first case of bradycardia-dependent PMVT caused by atrial undersensing in a patient implanted with the Micra AV leadless pacemaker.

## Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2020.05.003>.

## References

1. Reynolds D, Duray GZ, Omar R, et al. A leadless intracardiac transcatheter pacing system. *N Engl J Med* 2016;374:533–541.
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. Da Costa A, Romeyer-Bouchard C, Guichard JB, Gerbay A, Isaaz K. Is the new Micra-leadless pacemaker entirely safe? *Int J Cardiol* 2016;212:97–99.
4. Olsen FJ, Højlund S, Jacobsen MD. Malignant ventricular tachycardia and cardiac arrest induced by a micra™ leadless pacemaker. *J Electrocardiol* 2018;51:1053–1054.
5. Amin AK, Billakanty SR, Chopra N, et al. Premature ventricular contraction-induced polymorphic ventricular tachycardia after leadless pacemaker implantation: a unique adverse effect of leadless pacing. *HeartRhythm Case Rep* 2018;4:180–183.
6. Udo EO, Zuthoff NPA, van Hemel NM, de Cock CC, et al. Incidence and predictors of short- and long-term complications in pacemaker therapy: the FOL-LOWPACE study. *Heart Rhythm* 2012;9:728–735.
7. El-Chami MF, Al-Samadi F, Clementy N, et al. Updated performance of the Micra transcatheter pacemaker in the real-world setting: a comparison to the investigational study and a transvenous historical control. *Heart Rhythm* 2018;15:1800–1807.
8. Healey JS, Toff WD, Lamas GA, et al. Cardiovascular outcomes with atrial-based pacing compared with ventricular pacing: meta-analysis of randomized trials, using individual patient data. *Circulation* 2006;114:11–17.
9. Himmrich E, Przibille O, Zellerhoff C, et al. Proarrhythmic effect of pacemaker stimulation in patients with implanted cardioverter-defibrillators. *Circulation* 2003;108:192–197.
10. Sweeney MO, Ruetz LL, Belk P, Mullen TJ, Johnson JW, Sheldon T. Bradycardia pacing-induced short-long-short sequences at the onset of ventricular tachyarrhythmias: a possible mechanism of proarrhythmia? *J Am Coll Cardiol* 2007;50:614–622.
11. Mansour F, Khairy P. Electrical storm due to managed ventricular pacing. *Heart Rhythm* 2012;9:842–843.
12. Vavasis C, Slotwiner DJ, Goldner BG, Cheung JW. Frequent recurrent polymorphic ventricular tachycardia during sleep due to managed ventricular pacing. *Pacing Clin Electrophysiol* 2010;33:641–644.
13. Roden DM. Taking the “idio” out of “idiosyncratic”: predicting torsades de pointes. *Pacing Clin Electrophysiol* 1998;21:1029–1034.
14. Palanca V, Navarro A, Jiménez J, Quesada A, Morell S, Roda J. Intermittent atrial undersensing in single-lead VDD pacemakers in patients with bradycardia-sensitive repolarization: a possible mechanism for ventricular arrhythmia. *Rev Esp Cardiol* 2010;63:229–232.
15. Elsokkari I, Abdelwahab A, Parkash R. Polymorphic ventricular tachycardia due to change in pacemaker programming. *HeartRhythm Case Rep* 2017;3:243–247.