FOCUS ON ALLIED HEALTH PROFESSIONALS

An unusual case of atrial pacemaker syndrome

Heather M. Ross, PhD, DNP, FHRS,*†‡ Laura J. Kawasaki, MSN,* Claudia Williams, BSN‡

*From the *Arizona State University Edson College of Nursing and Health Innovation, Phoenix, Arizona,*
†Arizona State University School for the Future of Innovation in Society, Tempe, Arizona, and
‡HonorHealth Cardiac Arrhythmia Group, Scottsdale, Arizona.

Introduction

A small-stature 23-year-old woman (body surface area 1.67 m^2, body mass index 34.1) required pacemaker implantation for profound sinus node dysfunction (resting rate 18 beats/min) after sinus node modification. With potential future imaging needs, a dual-chamber magnetic resonance imaging–compatible system was implanted (Medtronic Revo pacemaker, CapSureFix MRI 5086 leads [7F], Dublin, Ireland). Postimplantation, she developed intermittent ipsilateral arm edema, rubor, and paresthesias. Doppler studies revealed no thrombus, but sluggish flow through a small-caliber subclavian vein. Symptoms persisted on aspirin and clopidogrel. Warfarin was avoided owing to occupational hazards. Given normal atrioventricular (AV) node function with stable conduction at pacing to 130 beats/min, the ventricular lead was extracted to enhance subclavian vein patency and relieve arm symptoms (Figure 1a, C), which resolved completely.

Case report

Shortly following lead extraction, the patient developed palpitations, chest discomfort, lightheadedness, and presyncope lasting seconds to hours. Device interrogation demonstrated normal atrial lead function with reasonable rate histogram (Figure 1b), suggesting appropriate rate response with AAIR programming and no recorded atrial arrhythmias (Figure 1a). Occasional intrinsic atrial activity was evidenced by <100% atrial pacing (Figure 1a, D), consistent with known atrial tachycardia treated with class Ic antiarrhythmics (flecainide 50 mg twice daily) and AV nodal blocking agents (metoprolol succinate 25 mg twice daily, diltiazem ER 120 mg daily). Medication uptitration over 6 weeks (flecainide 100 mg twice daily, metoprolol succinate 50 mg twice daily, and diltiazem ER 120 mg daily) reduced arrhythmia without complete resolution (Figure 1a, E) and increased fatigue.

KEYWORDS Pacemaker syndrome; Atrial tachycardia; Single-chamber pacemaker; Pacemaker clinic; Allied professional
(Heart Rhythm Case Reports 2023;9:64–65)

Funding Sources: None. Disclosures: The authors have no conflicts to disclose. **Address reprint requests and correspondence:** Dr Heather Ross, P.O. Box 876002, Tempe, AZ 85287-6002. E-mail address: hmross1@asu.edu.

Three months later, the patient's symptoms worsened, negatively impacting activity (Figure 1a, F). She experienced transient lightheadedness during pacemaker interrogation; real-time intracardiac electrograms demonstrated brief atrial tachycardia with conducted atrial signals in blanking and refractory periods and atrial pacing dyssynchronous to ventricular activity, consistent with undersensed atrial tachycardia in a single-chamber atrial pacemaker. Radial pulse was weak and rapid, consistent with pacemaker syndrome, the symptomatic manifestation of decreased cardiac output and nonphysiologic intracardiac pressures from pacing-induced A-V dyssynchrony. A smaller-diameter ventricular lead (Medtronic CapSure Sense 4076 [5.3F]) was implanted to restore dual-chamber pacing, with complete symptom resolution and no recurrent arm edema or paresthesia.

Discussion

This case demonstrates an unusual occurrence of pacemaker syndrome in an atrial pacemaker. Although pacemaker syndrome is well known, it is typically associated with asynchronous ventricular pacing stimuli.[1] There is a single case report of pacemaker syndrome during sinus tachycardia in minimum ventricular pacing mode.[2] To our knowledge, there has not been another case reported of pacemaker syndrome related to atrial pacing. The possibility of atrial-mediated pacemaker syndrome is important to consider in patients with sinus node dysfunction, intact AV node function, and known atrial arrhythmias. Atrial signals in blanking and refractory periods may not be captured with intracardiac electrogram recordings for transient arrhythmias that do not trigger a saved electrogram, and are unlikely to be evident on 12-lead electrocardiogram, making diagnosis difficult without reproducible symptoms during device interrogation.

Conclusion

Particularly in regulatory climates requiring direct justification for dual-chamber pacemaker implantation, patients with atrial tachycardias may be at risk of developing pacemaker syndrome with single-chamber atrial devices. Moreover, vein size and lead diameter should be considered when planning dual-chamber pacemaker implantation using larger-caliber leads. Finally, the entire heart rhythm care team must be familiar with device implantation techniques

https://doi.org/10.1016/j.hrcr.2022.11.012

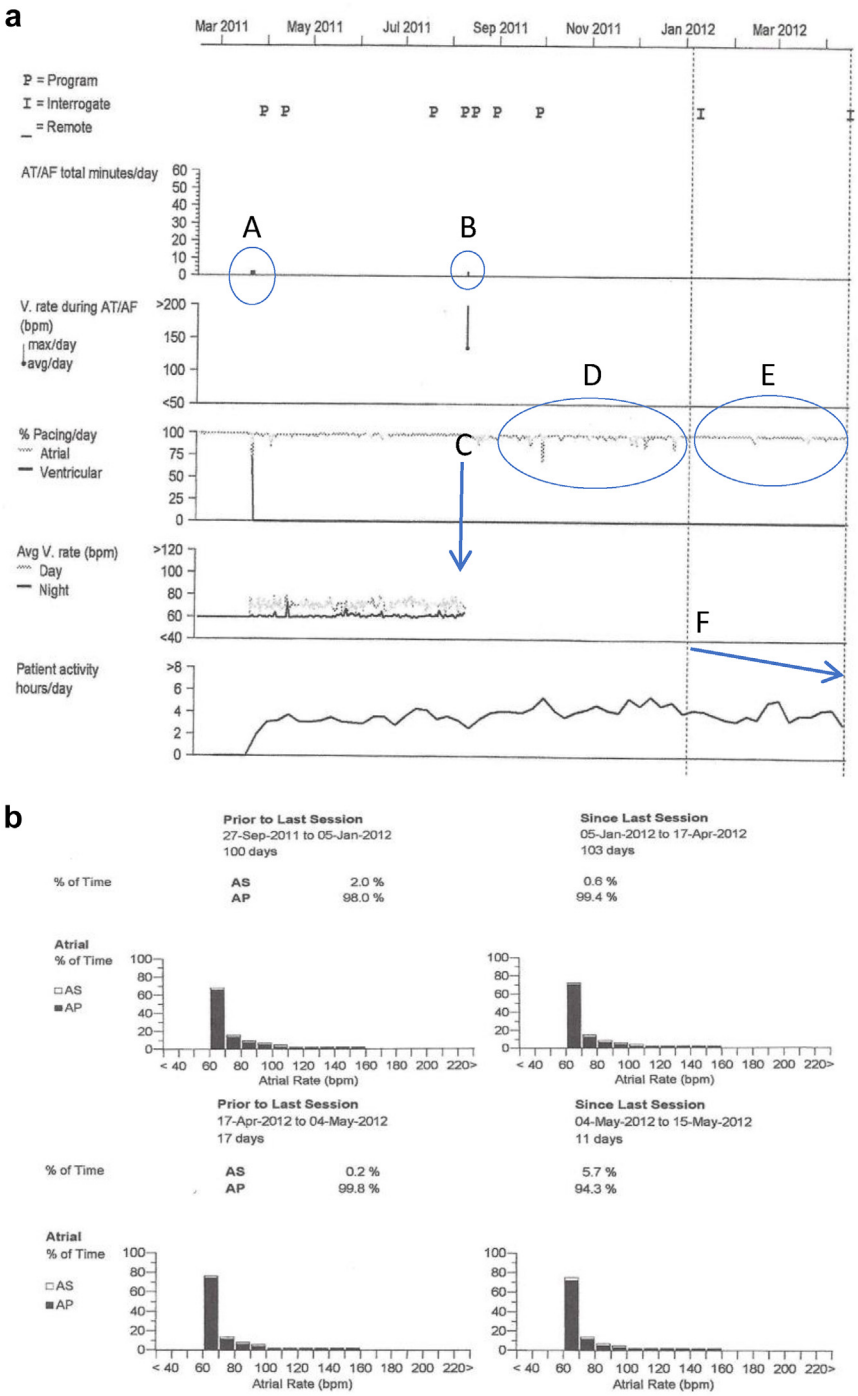

Figure 1 **a:** Cessation of recorded atrial tachycardia episodes (A, B) following ventricular lead explant (C). Atrial arrhythmia episodes postexplant shown by reduced pacing percentages (D) and attenuated with medication uptitration (E), though activity levels were impacted (F). **b:** Reasonable chronotropic profile with reduced atrial arrhythmia burden after medication uptitration in January 2012.

and current technologies to diagnose and manage rare and complex patient needs, particularly when diagnosis requires direct observation during interrogation. For clinical settings where device interrogation may be performed separately from clinical evaluation, correct diagnosis may depend on the ability of the pacemaker clinic staff to recognize and report unusual findings.

References

1. den Dulk K, Lindemans FW, Brugada P, Smeets JL, Wellens HJ. Pacemaker syndrome with AAI rate variable pacing: importance of atrioventricular conduction properties, medication, and pacemaker programmability. Pacing Clin Electrophysiol 1988;11:1226–1233.
2. Pascale P, Pruvot E, Graf D. Pacemaker syndrome during managed ventricular pacing mode: what is the mechanism? J Card Electrophysiol 2009; 20:574–576.