CASE REPORT

Uncommon Presentation of Drug-refractory Pacemaker-mediated Common Atrioventricular Nodal Reentrant Tachycardia and a Simple Solution by Reprogramming

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Abstract

An 81-year-old woman who had undergone dual chamber pacemaker implantation for sick sinus syndrome was referred to our hospital with drug-refractory common atrioventricular (AV) nodal reentrant tachycardia. Ventricular pacing (Vp) following premature atrial contraction (PAC) with a long AV interval induced ventriculoatrial (VA) conduction, which allowed the tachycardia to be initiated. The sensed AV interval was shortened to 80 ms, allowing Vp during the refractory period of VA conduction. Postventricular atrial refractory period was shortened to 180 ms to sense PACs with short coupling interval. After reprogramming, the suppression of the tachycardia by blocking VA conduction following Vp was confirmed.

Key words: pacemaker, sick sinus syndrome, paroxysmal supraventricular tachycardia, atrioventricular nodal reentrant tachycardia

(DOI: 10.2169/internalmedicine.54.3978)

Introduction

Antegrade slow pathway modification by radiofrequency catheter ablation (RFCA) is an established approach for common atrioventricular nodal reentrant tachycardia (AVNRT) and commonly performed with a high success rate (1). In this case report, we demonstrate instructive recordings from 24-hr ambulatory monitoring of a patient with common AVNRT following DDD pacemaker implantation who had contraindication for RFCA.

Case Report

An 81-year-old woman was referred to our hospital with recurrent palpitations that had worsened over the previous few months. She had undergone dual chamber pacemaker implantation (INSIGNIA Entra DR, Guidant, St. Paul, USA) for sick sinus syndrome 6 years earlier with the following pacing parameters: mode, DDD; lower rate, 60 paces/min; upper tracking rate, 120 paces/min; paced atrioventricular (AV) interval, 250 ms; sensed AV interval, 220 ms; postventricular atrial refractory period (PVARP), 250 ms; and PVARP after premature ventricular contraction, 400 ms. A 12-lead electrocardiogram obtained during palpitations revealed paroxysmal supraventricular tachycardia (PSVT) and a heart rate of 170 bpm (Fig. 1). Electrogram findings at the onset of the tachycardia following spontaneous sinus rate was consistent with common AVNRT with a prolongation in the AV interval following premature atrial contraction (PAC) (Fig. 2). Conversely, 24-hr ambulatory monitoring revealed that the majority of PSVT episodes were consistently triggered by ventricular pacing (Vp) following PACs of much longer coupling intervals than that of the PAC observed prior (Fig. 3A versus Fig. 2). Even with administration of anti-arrhythmic drugs comprising bisoprolol (15 mg daily) and verapamil (240 mg daily), PSVT occurred over 10 times per day (Fig. 3B). Severe comorbidities, including pancytopenia due to multiple myeloma, were involved; therefore, RFCA or increasing the dose of anti-arrhythmic drugs was...
considered inappropriate. Accordingly, the initial management consisted of reprogramming. Fearing that Vp triggered by PAC after a 220-ms AV interval may induce ventriculoatrial (VA) conduction and a subsequent prolongation in the PR interval (Fig. 3A), the mode was changed to AAI. However, no response was achieved and frequent PAC-induced PSVT episodes were observed by the same mechanism (as shown in Fig. 2). The sensed AV interval was then considered inappropriate.
shortened to 80 ms, allowing Vp during the refractory period of VA conduction. PVARP was shortened to 180 ms to sense PACs with short coupling interval. The paced AV interval was not shortened to avoid unnecessary Vp during the basic paced atrial rhythm. After reprogramming, the palpitations disappeared. Suppression of PSVT by blocking VA conduction following Vp was confirmed by 24-hr ambulatory monitoring (Fig. 3C, D). The patient is currently being treated with bisoprolol (5 mg) to maintain basic atrial pacing (Ap) and ventricular sensing (Vs).

Discussion

We demonstrate instructive recordings from 24-hr monitoring of pacemaker-mediated PSVT observed in a patient with sick sinus syndrome which was successfully eliminated by simple pacemaker reprogramming.

The present AVNRT was initiated in two ways, both involving a PAC as the triggering event. The most common circumstance was a longer coupled PAC outside the PVARP, thereby allowing it to be tracked by a Vp after an AV delay of 220 ms, which then allowed AVNRT to be initiated. The second mechanism was a short coupled PAC within the PVARP which was not tracked by the pacemaker but would be followed by a long PR interval due to slow pathway conduction, thus initiating AVNRT.

Generally, algorithms to reduce unnecessary Vp, including long AV interval, search AV interval, and managed Vp, are selected for patients who underwent dual chamber pacemaker implantations because of sick sinus syndrome (2). In the present case, the long AV interval would allow the Vp following PAC to induce VA conduction, which triggered a subsequent prolongation in the PR interval.

Endless loop tachycardia (ELT) can occur in patients with a dual chamber pacemaker. Stimulating and sensing both the atrium and ventricle caused an ELT, which started with a retrograde P-wave occurring after a premature ventricular contraction and was sustained by Vp following P-waves caused by VA conduction (3). When the patient’s heart rate is maintained by atrial sensing (As) followed by Vp at the maximum tracking rate, ELT is suspected. Current pacemakers have functions for the prevention and interruption of ELT that: (i) extend the PVARP following ventricular sensing, in the absence of an atrial event, to avoid tracking a retrograde P-wave following a premature ventricular contraction and (ii) prolong the AV interval or switch the mode from DDD to DDI when the As-Vp rhythm at the maximum tracking rate continues for a specific period. As Vp was involved only at the initiation of tacharyrhythmia in this case, the aforementioned general algorithms to prevent conventional ELT seem to be ineffective for this type of pacemaker-mediated PSVT consistently induced by single Vp.

Although RFCA is a common approach for PSVT (5), the
present case of pacemaker-mediated PSVT responded well to the noninvasive simple approach: shortening of the sensed AV interval and PVARP. This may be achieved by blocking retrograde fast pathway conduction which induces a subsequent prolongation in PR interval (indirect block). Moreover, it would also be effective in preventing spontaneous PSVT by directly blocking the antegrade slow pathway conduction by retrograde collision (direct block). Some pacemaker models use an atrial anti-tachycardia pacing algorithm for supraventricular tachyarrhythmia. However, it cannot be applied to supraventricular tachyarrhythmia of 1:1 ventricular response, including PSVT. Even if such termination algorithms were available, the present method would be superior from a prevention standpoint.

Although rarely combined with AVNRT, the disease process leading to sinus node dysfunction is frequently associated with AV nodal dysfunction (6). Kreiner et al. demonstrated that common AVNRT patients with sinus node dysfunction suffered from significantly more frequent episodes of tachycardia than AVNRT patients without sinus node dysfunction (6). Bae et al. described a patient with AVNRT combined with sick sinus syndrome who was treated simultaneously with RFCA and pacemaker implantation (7). Our case is clinically relevant in that it suggests a simple and noninvasive prevention method for tachycardia episodes in common AVNRT patients with sick sinus syndrome requiring pacemaker implantation.

In summary, we reported a rare case of pacemaker-mediated PSVT. Vp with a long AV interval was involved in the initiation of PSVT. This case demonstrates a potentially useful clinical solution. It may be applied to every patient with common AVNRT in whom a dual chamber pacemaker has been implanted. Furthermore, this case reinforces the importance of precise attention to the initiation of tachycardia, even if narrow QRS regular tachycardia, which is unlikely to be a conventional ELT, is observed during palpitation in patients with dual chamber pacemakers.

The authors state that they have no Conflict of Interest (COI).

Acknowledgement

We would like to thank medical engineers Hirotaka Hoshi, Yasuhiro Kinebuchi, Tatsuya Satoh, and Misato Ueno for their support in device monitoring and reprogramming.

References