CASE REPORT

Atrial tachycardia with slow pathway conduction mimicking typical atrioventricular nodal reentrant tachycardia

Takumi Yamada*, Jose F. Huizar, Hugh T. McElderry, and G. Neal Kay

Division of Cardiovascular Diseases, Cardiac Rhythm Management Laboratory, University of Alabama at Birmingham, VH B147, 1670 University Boulevard, 1530 3rd Ave S, Birmingham, AL 35294-0019 USA

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A 68-year-old woman with palpitations underwent electrophysiologic testing. During burst atrial pacing the PR interval exceeded the RR interval and induced a supraventricular tachycardia consistent with a typical AV nodal reentrant tachycardia (AVNRT). Radiofrequency ablation of the slow pathway during the tachycardia immediately produced 2:1 AV conduction. After slow AV nodal pathway ablation an atrial tachycardia (AT) remained inducible with the earliest atrial activation around the HB region. Radiofrequency ablation at the site of earliest atrial activation interrupted the AT without AV block. AT originating from the HB region with slow pathway conduction may mimic typical AVNRT.

KEYWORDS
Atrial tachycardia; Slow pathway; Atrioventricular nodal reentrant tachycardia; Radiofrequency catheter ablation

Introduction

Differentiation of atrial tachycardia (AT) from other forms of paroxysmal supraventricular tachycardia during an electrophysiologic study (EPS) is critically important in the era of catheter ablation.1,2 However, the diagnosis of an AT can be especially difficult in the presence of dual atrioventricular (AV) nodal pathways or an AV accessory pathway.3 We report a case of AT with slow pathway conduction mimicking typical AV nodal reentrant tachycardia (AWNRT).

Case report

A 68-year-old woman with a documented narrow complex tachycardia was referred for an EPS and radiofrequency (RF) catheter ablation. Written informed consent was obtained, and the EPS was performed after all antiarrhythmic drugs had been discontinued for at least five half-lives prior to the study. A 6-French decapolar catheter was introduced and positioned into the coronary sinus (CS) via the right common femoral vein. Two 6-French quadrupolar catheters were introduced from the right common femoral vein and placed in the His bundle (HB) region and right ventricular apex for mapping and pacing. During the EPS, burst atrial pacing from the mid-CS demonstrated the presence of a PR interval greater than the RR interval and induced a supraventricular tachycardia, consistent with a typical AVNRT (slow-fast type).4 Atrial extra stimulation was not performed before the ablation because the tachycardia exhibited an incessant form once it was induced by burst atrial pacing. The supraventricular activation was preceded by a HB potential with the earliest atrial activation around the HB region. Radiofrequency ablation at the site of earliest atrial activation interrupted the AT without AV block. AT originating from the HB region with slow pathway conduction may mimic typical AVNRT.

* Corresponding author: Tel: +1 205 975 4724; fax: +1 205 975 4720. E-mail address: takumi-y@fb4.so-net.ne.jp

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stimulation without a jump in the AV conduction and by burst atrial pacing with the disappearance of a PR interval greater than the RR interval. These findings supported a diagnosis of AT. Finally, successful ablation of the AT was achieved in the proximity of the HB region without any evidence of AV block (Figure 2).

Discussion

We demonstrated in the prospective study that the finding of a PR interval that exceeded the RR interval during burst atrial pacing was a reliable indicator of sustained slow AV nodal pathway conduction and inducible AVNRT. We think that this sign may be useful as a method for demonstrating the presence of slow pathway conduction in patients with inducible AVNRT when dual AV nodal physiology cannot be demonstrated by atrial extra stimulation. Additionally, reducing the PR/RR ratio to a value may serve as a useful endpoint during selective slow AV nodal pathway ablation for AVNRT. Therefore, the findings in this case suggested the presence of a dual AV nodal physiology.

To the best of our knowledge, this is the first case report showing that AT originating from the HB region with slow pathway conduction could mimic typical AVNRT. In this case, the anterograde conduction of the fast pathway during the tachycardia was never observed before the catheter ablation. However, 2 to 1 AV conduction via the fast pathway with a longer refractory period was observed after the elimination of the slow pathway, however, the tachycardia cycle length did not change. These findings suggested that the fast pathway was retrogradely activated during the tachycardia before the catheter ablation and accordingly a typical AVNRT might be entrained by the AT. Therefore, before the catheter ablation even entrainment pacing from the right ventricle may not have revealed that the AT was dominant in this case.

In this case, when the first ablation targeting the slow pathway converted the tachycardia from 1:1 to 2:1 AV conduction, the possibility of AVNRT could still not be excluded completely because a 2:1 AV block and even a high degree AV block could be observed during AVNRT. It has been reported that 2:1 AV block during AVNRT is functional and the incidence of reproducible, sustained 2:1 AV block during induced episodes of AVNRT is <10%. In a majority of patients with 2:1 AV block during AVNRT, there is an HB potential visible in the blocked beats, suggesting that the level of the block is within or below the HB. In the minority of patients with 2:1 AV block during AVNRT, there is no HB potential visible in the blocked beats, suggesting that the level of the block is in the junction between the AV node and HB and there is the presence of a lower common pathway. Though in this case, the possibility of AVNRT with a lower common pathway still existed after the first ablation, the resumed EPS provided the definitive diagnosis of AT.

Most efforts should be made before the ablation to obtain a definitive diagnosis of the tachycardia. However, in the complex cases like ours, any diagnostic manoeuvres may not always determine which is the dominant tachycardia, AT or AVNRT. Therefore, it may be most important in the catheter ablation in cases like ours to be willing to reevaluate the first diagnosis when the first ablation is not successful.

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References