

Adenosine-sensitive atrial tachycardia originating from the anterior mitral annulus



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Introduction

Adenosine-sensitive atrial tachycardia (AT) has a variable location of origin, and the tachycardia origin sometimes shifts to another site after ablation. We present a case of adenosine-sensitive AT that originated from the anterior mitral annulus (MA) but showed a shift in tachycardia origin after ablation. The ablation was successful in suppressing AT by understanding a full picture of the circuit. Precise mapping is important because, in some cases, ablation of the earliest activation site modifies only its exit and does not modify the critical slow conduction zone in adenosine-sensitive AT.

Case report

A 49-year-old man presented to our hospital for catheter ablation of paroxysmal supraventricular tachycardia. Twelve-lead electrocardiography (ECG) revealed an episode of long RP, narrow QRS regular tachycardia with a heart rate of 170 bpm. The P-wave morphologies on the 12-lead ECG during the tachycardia were positive in leads II, III, aVF, and V₁ (Figure 1). After obtaining written informed consent from the patient, a cardiac electrophysiological study was performed. Multielectrode catheters were placed at the high right atrium (HRA), His bundle, coronary sinus, and right ventricular apex. The tachycardia (cycle length 390 ms) was reproducibly induced by ventricular electrical stimulus with a V-A-A-V sequence. Earliest atrial activation was observed at the His-bundle electrode (HBE). The ventriculoatrial intervals on the first return beat of the entrained tachycardia were variable by >14 ms.¹ An intravenous 2-mg bolus of adenosine 5'-triphosphate during the tachycardia lengthened the tachycardia cycle length (TCL) and reproducibly terminated the tachycardia without atrioventricular (AV)

KEY TEACHING POINTS

- Ablation at the earliest activation site modifies only its exit and cannot modify the critical slow conduction zone in adenosine-sensitive atrial tachycardia (AT).
- Precise entrainment mapping around the AT exit reveals the relationship between its critical slow conduction zone and exit.
- Precise entrainment mapping enables us to image the most ideal ablation site even if the AT shows a shift in tachycardia origin.

block. Based on these findings, the tachycardia was diagnosed as an adenosine-sensitive AT.

Activation mapping in the right atrium (RA) using a high-density multipolar electrode mapping catheter (PentaRay, Biosense Webster, Diamond Bar, CA) during the tachycardia revealed that the earliest RA activation was at the septal RA, but the postpacing interval (PPI) at the earliest site was 130 ms longer than the TCL. The PPI at the noncoronary aortic cusp was 60 ms longer than the TCL. Activation mapping in the left atrium performed using the PentaRay revealed earliest atrial activation at the 10 o'clock position on the MA and a centrifugal pattern from that site. The PPI TCL at that site was 20 ms, and the activation time at the site was 70 ms earlier than that at the earliest right activation site. Entrainment pacing at 1 o'clock on the MA revealed that the PPI TCL was 25 ms. Atrial electrograms recorded at the HRA, HBE, and PentaRay 1/2, 3/4, 7/8, 11/12, 15/16 up to 19/20 were captured orthodromically with a long conduction interval and the same morphologies as those during the tachycardia (Figure 2). Other electrograms, captured directly or antidromically, showed different morphologies from those recorded during the tachycardia. Thus, this pacing site was indicated proximal to the slow conduction zone, and the entrance of the tachycardia circuit was located between this pacing site (1 o'clock on the MA) and the earliest activation site (10 o'clock on the MA).

KEYWORDS Activation mapping; Adenosine-sensitive atrial tachycardia; Atrial tachycardia; Entrainment mapping; Mitral annulus (Heart Rhythm Case Reports 2018;4:542-544)

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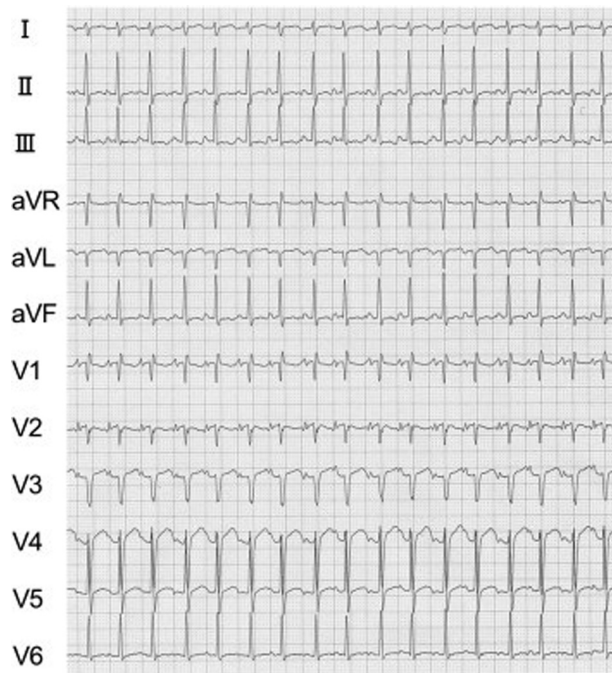


Figure 1 Twelve-lead electrocardiography revealed an episode of long RP, narrow QRS regular tachycardia with a heart rate of 170 bpm. The P-wave morphologies during the tachycardia were positive in leads II, III, aVF, and V₁.

Radiofrequency (RF) energy was delivered 10 o'clock on the MA, which was the earliest activation site during AT. The ablation catheter potential followed onset of the P wave by 20 ms. The unipolar potential revealed that the QS pattern and

AT changed their sequence during ablation, and this change was sustained. Activation mapping performed conventionally with an ablation catheter from 9 o'clock to 1 o'clock on the MA revealed earliest atrial activation at 11 o'clock on the MA. The ablation catheter potential followed onset of the P wave by 20 ms, and the unipolar potential revealed the QS pattern at that site. RF energy delivered to 11 o'clock on the MA during AT terminated the AT 1.2 seconds after ablation was started. No arrhythmia was inducible by programmed stimulus thereafter, and the patient had no recurrence of tachycardia during the 1-year observation period, without any antiarrhythmic drug.

Discussion

In 1997, Iesaka and colleagues² reported the existence of adenosine-sensitive reentrant AT that did not involve an AV nodal pathway. The exact location of the AT origin has been found to vary considerably,³ but an AT originating from the MA is relatively rare. Yamabe and colleagues⁴ reported that adenosine-sensitive AT was organized as reentry involving the verapamil-sensitive slow conduction zone, with its entrance and exit at different distinct locations. They demonstrated that RF energy delivery to the proximal to the earliest activation site (entrance of slow conduction zone of reentrant circuit) could terminate adenosine-sensitive AT originating from the RA.

This case showed that the entrance of the tachycardia circuit existed between 10 o'clock and 1 o'clock on the MA, and the site at 1 o'clock on the MA was proximal to the critical slow conduction zone (Figure 3). Considering that the entrance

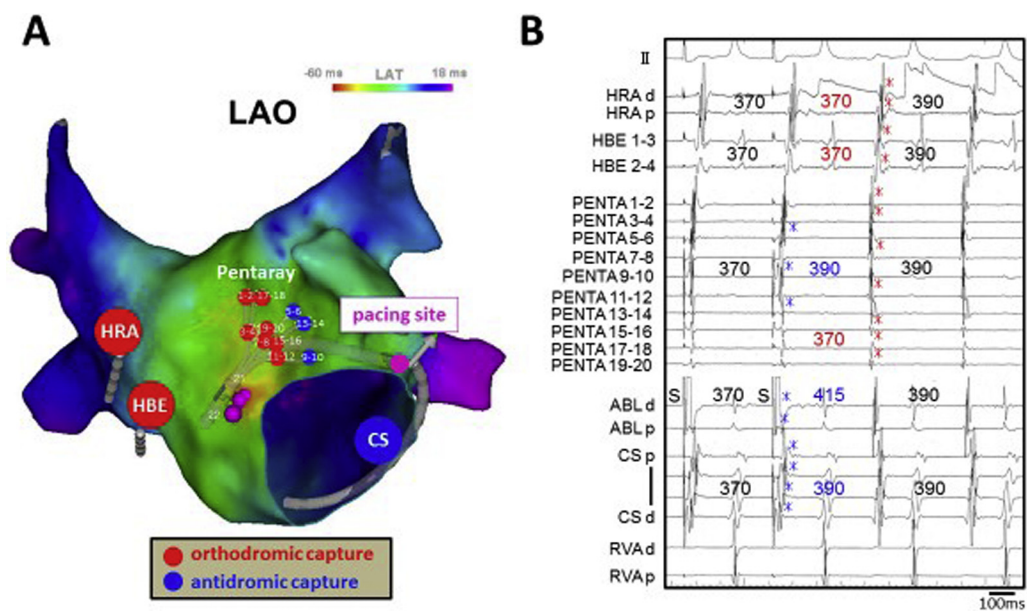


Figure 2 **A:** Catheter position when entrainment pacing was performed from the site at 1 o'clock on the mitral annulus (MA). Activation mapping revealed earliest atrial activation at 10 o'clock on the MA and a centrifugal pattern from that site. The catheter electrodes encircled by red circles were captured orthodromically and those encircled by blue circles were captured antidromically. **B:** Intracardiac electrograms. Red and blue asterisks indicate the last electrograms captured using the last pacing stimulus. Catheter electrodes indicated with red asterisks were captured orthodromically and those indicated with blue asterisks were captured antidromically. ABL = ablation; CS = coronary sinus; HBE = His-bundle electrogram; HRA = high right atrium; LAO = left anterior oblique; LAT = local activation time; RVA = right ventricular apex.

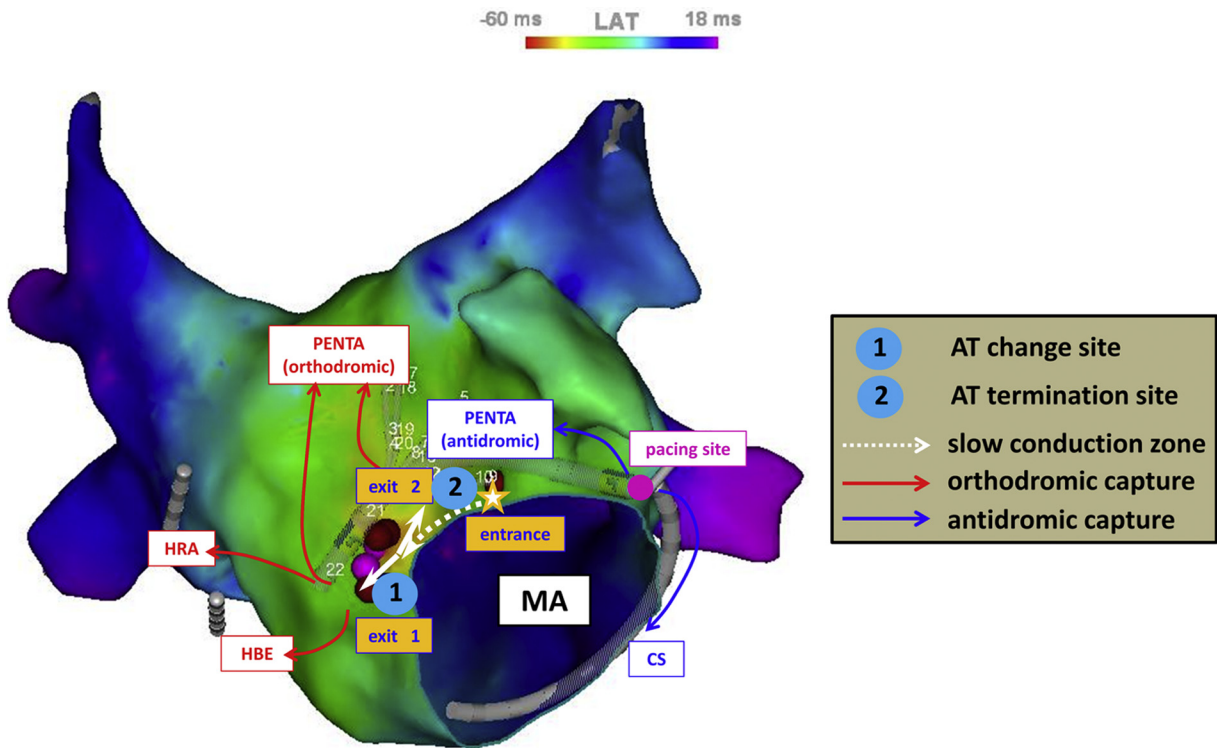


Figure 3 Supposed conduction pathway and capture style of each electrode (orthodromically or antidromically) when entrainment pacing was performed from the site at 1 o'clock on the mitral annulus (MA). A critical slow conduction zone existed between the exit and 1 o'clock on the MA (pacing site). After radio-frequency energy was delivered to exit 1, it was suspected that the atrial tachycardia (AT) had changed its exit to another one (exit 2). AT was terminated within 1.2 seconds after ablation was started at exit 2. It was believed that ablation at the site that successfully terminated the AT modified not only the exit but also the critical slow conduction zone near the ablation site, and this contributed to AT termination. CS = coronary sinus; HBE = His-bundle electrogram; HRA = high right atrium; LAT = local activation time.

existed on the septal site of the PentaRay electrodes (around 12 o'clock on the MA), which were captured antidromically and on the lateral site of the exit 1 (around 11 o'clock on the MA), the entrance existed around 11–12 o'clock on the MA. After RF energy was delivered to exit 1, it was suspected that the AT had changed its exit. It was supposed that ablation at the site that successfully terminated the AT modified not only the exit but also the critical slow conduction zone near the ablation site, and this contributed to AT termination. Koyama and colleagues⁵ reported that verapamil-sensitive AT originating from the RA often showed a shift in tachycardia origin to another site. In this case, the AT originated from the MA and similarly showed a slight shift in tachycardia origin; therefore, precise mapping was useful for AT termination. Unfortunately, we did not perform entrainment pacing at 10 o'clock on the MA after delivering RF energy to exit 1. If this had been performed, we could have understood a full picture of the circuit more clearly. It is assumed that ablation of the earliest activation site modifies only its exit and cannot modify the critical slow conduction zone in adenosine-sensitive AT. Precise entrainment mapping around the AT exit reveals the relationship between its critical slow conduction zone and exit. By performing ablation for AT, we can avoid the AV node and image the most ideal ablation site even if AT shows a shift in tachycardia origin. In our case, if we delivered RF

energy to the entrance of the slow conduction zone first, AT would terminate without changing its origin.

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