A Case of Adenosine Sensitive Atrial Tachycardia Originating from the His-Bundle Region Successfully Ablated from the Non-coronary Aortic Cusp

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The case of a patient with narrow QRS tachycardia is herein presented. During an electrophysiological study, this tachycardia was diagnosed as a focal atrial tachycardia with a high sensitivity to adenosine-5'-triphosphate (2 mg), and originating from near the His-bundle region. We could not ablate the earliest activation site in the right atrium because of the simultaneous recording of the His potential. The radiofrequency applications in the right atrium around the earliest activation site failed to terminate the tachycardia. Subsequent attempt of radiofrequency application in the non-coronary aortic cusp, where the earliest atrial activation without His potential was obtained, successfully eliminated the tachycardia without any atrioventricular nodal injury.

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Key words: ATP sensitive atrial tachycardia, Catheter ablation, Non-coronary aortic cusp

Introduction

Focal atrial tachycardias (ATs) commonly originate from the crista terminalis, the tricuspid or mitral annulus, pulmonary veins and septal region. In previous reports, the prevalence of AT originating from the His-bundle region was estimated to constitute 0–13% of all ATs. Although most of these cases can be successfully ablated in the right atrium without atrioventricular node (AVN) injury, in some patients it is difficult to ablate in the right atrium because of an inordinately close proximity of the target site to the AVN or the His-bundle. We herein report a case of adenosine-5'-triphosphate (ATP) sensitive AT originating from the His-bundle region, which was successfully ablated in the non-coronary aortic cusp (NCC) without any complications.

Case Report

A 55-year-old female with a 5-year history of palpitation was referred to our hospital to undergo catheter ablation for tachycardia resistant to anti-arrhythmic drug treatments. A surface electrocardiogram (ECG) during palpitation showed a regular narrow QRS tachycardia with a heart rate of
150 bpm which was reproducibly terminated by a 10 mg of intravenous administration of ATP. During the tachycardia, a small negative wave just after the QRS complex was observed in leads II, III, aVF (Figure 1B). A chest radiography showed no cardiomegaly (cardiothoracic ratio 48%), and echocardiography showed a normal heart with a left ventricular ejection fraction of 65%. The 12-lead ECG during sinus rhythm demonstrated a lack of pre-excitation (Figure 1A).

Following informed consent from the patient, the electrophysiological study and catheter ablation were performed. All antiarrhythmic drugs were discontinued for at least five half-lives prior to the procedure. Three catheters were placed at the high right atrium, the right ventricle, and the His-bundle region via the right femoral vein approach. A 5-F decapolar catheter was introduced and positioned at the coronary sinus via the right subclavian vein. Bipolar and unipolar electrograms were filtered at 30–400 and 0.05–400 Hz, respectively. At the beginning of the procedure, the sinus rhythm cycle length (CL) was 735 ms with an atrio-His interval of 100 ms and a His-ventricle interval of 55 ms. There was no retrograde ventriculoatrial conduction observed during the pacing from the right ventricle. Program stimulation from the high right atrium revealed a dual pathway of the AVN with a jump-up phenomenon. The clinical regular narrow QRS complex tachycardia was reproducibly induced by a burst stimulation from the high right atrium (pacing CL: 400 ms) (Figure 2A). The intracardiac recordings demonstrated that the earliest atrial activation potential preceding the P wave by 30 ms was observed in the His-bundle recording. The tachycardia was sustained despite the demonstration of AV block (Figure 3A), and no relationship was observed to exist between the atrio-His interval and the A-A interval. An intravenous bolus injection of small doses (2 mg) of ATP reproducibly terminated the tachycardia without involving AV block (Figure 2B). Combining three findings together, we diagnosed this tachycardia to be focal AT originating from near the His-bundle region with high sensitivity to ATP.

A 4 mm tip 7-F catheter (RF Marinr, Medtronic CardioRhythm, San Jose, CA, USA) was used for mapping and ablation. Although detailed mapping in the right atrium showed the earliest activation potential preceding the P wave by 40 ms at the His-bundle region, a His potential was simultaneously recorded and the uni-polar electrocardiograms of ablation catheter did not show a QS pattern at this site. Three radiofrequency (RF) applications with a

Figure 1
(A) Surface 12-lead electrocardiogram (ECG) in sinus rhythm with a heart rate of 77 bpm. (B) Surface 12-lead ECG during tachycardia with a heart rate of 150 bpm. The negative P wave in II, III, aVF leads and the positive P wave in I, aVL leads is shown immediately after QRS (arrows). I, II, III, aVL and aVF represent recordings from 12-lead surface ECG.
target temperature of 50 degrees and a power of 30 watts around this region (anterior, superior, and posterior side of the His-bundle region) without His potential failed to terminate the tachycardia.

We subsequently mapped the aortic root through the retrograde approach. The local atrial activation in the NCC did not show the His potential and preceded the P wave by 50 ms with a QS pattern in a unipolar electrogram of the ablation catheter (Figure 4C). Angiography using a 5-F pigtail catheter demonstrated that the ablation catheter was located on the NCC adjacent to the His-bundle electrode. RF energy delivery at this site with a target temperature of 50 degrees and a power output limit of 30 watts (commenced with 15 watts) resulted in the termination of this tachycardia after 9.5 seconds of RF application without any findings of junctional rhythm or AV block. Although multiple AV conducting pathways persisted after a single RF application from the NCC, no arrhythmia was inducible by program stimuli from the atrium. No complications occurred either during or after the ablation procedure. The patient had no recurrence of tachycardia during the 6-month observation period without any antiarrhythmic drug treatment.

**Discussion**

In this case, the tachycardia was diagnosed as adenosine sensitive focal AT originating from the His-bundle region based on the following findings: 1) the earliest atrial potential was recorded near the His-bundle region; 2) no retrograde AVN conduction was observed during the pacing from the right ventricle; 3) tachycardia was sustained despite the appearance of AV block; and, 4) tachycardia was reproducibly terminated by a bolus injection of small doses (2 mg) of ATP.

The adenosine sensitive intra-atrial reentrant tachycardia within the AVN or its transitional tissues was first described by Iesaka et al. In canine and swine hearts, the atrial cells along the AV junction,
including the slow pathway region, exhibited node-like electrophysiological characteristics and were highly sensitive to adenosine.\textsuperscript{8}) It is likely that such peri-nodal atrial cells were involved in the genesis of AT in this case. In general, as has been previously described,\textsuperscript{5,9}) the ablation at the earliest activation site near the His-bundle region in the right atrium can eliminate this type of AT without injuring AVN. However, in our case it was impossible to identify the site of earliest atrial activation without showing the His potential in the right atrium, and a subsequent attempt of ablation in the NCC successfully eliminated this tachycardia. This might suggest that the origin of AT was possibly the epicardial side of the His-bundle region as has been described by Ouyang et al.\textsuperscript{10}) On the other hand, as for the ATP sensitivity of AT which could be ablated in the NCC, there have been only a few descriptions in previous reports.\textsuperscript{11,12}) Our case is unique in that ATP sensitivity was much higher than previous reported cases (2 mg vs 5 and 12 mg).

The safety of ablation in the NCC for the AT originating near the His-bundle region has been described elsewhere. Tada et al first described the successful ablation in the NCC without any complications,\textsuperscript{11}) and then Ouyang et al reported ATs successfully ablated in the NCC with no damage to AVN.\textsuperscript{10}) However, special care is recommended to prevent not only AV block but also the formation of thrombus and injury to aortic valves as described in the previous reports.\textsuperscript{13}) As for the RF applied in the NCC, either aortography or intracardiac echocardiography was recommended in order to confirm the position of the ablation catheter before the RF applications;\textsuperscript{14}) in addition, it was recommended that the RF energy delivery should be stopped if AT was not terminated after 10 seconds to avoid impairing the AVN and the aortic valve.\textsuperscript{10}) In our case, we performed the RF applications after aortography, and could terminate AT after 9.5 seconds without any complications.

\textbf{Figure 3}

(A) Intracardiac electrogram during the tachycardia is shown. The tachycardia continued despite the Wenckebach block in the atrioventricular conduction (arrow). The earliest atrial activation potential was observed in His 1-2 (dotted line). The atrial local (A-A) interval did not correlate with the atrio-His interval during tachycardia. The sequence of the atrial activation is constant regardless A-A interval variability. (B) Surface 12-lead electrocardiogram (ECG) during tachycardia with atrioventricular block. The ECG demonstrates the P wave to be negative in leads II, III and aVF, and positive in leads I and aVL during tachycardia.

H: His potential, other abbreviations as in Figure 1, 2.
Several previous reports investigated the predictor of the origin of AT near the His-bundle region. Tang et al analyzed the P wave morphology, positive P-wave in lead V1, to predict that the left atrial focus was 93% in sensitivity and 88% in specificity.\(^\text{15}\) Chen et al reported a biphasic P wave in lead V1 and a positive P wave in inferior leads in AT from the non-coronary aortic cusp,\(^\text{7}\) while Ouyang et al\(^\text{10}\) and Saumya et al\(^\text{16}\) described the tendency of positive P waves in lead I and aVL, and biphasic P wave in lead V1. On the other hand, Marrouche et al reported that the P wave morphology did not predict that the AT originated from the left septum.\(^\text{17}\) This present case showed positive P waves in leads I and aVL, negative P waves in leads II, III and aVF and a biphasic P wave in lead V1 (Figure 3B). The morphologies of the P waves in lead I, aVL and V1 in our case were considered to be consistent with the findings of previous studies, whereas those in the inferior leads differed from those described by Chen et al. Thus it seems difficult to predict the focus of AT originating near the His-bundle region by the P wave morphology alone from the surface ECG. We were, therefore, interested in other methods of detection for which the approach was most suitable for AT originating near the His-bundle region before the ablation procedure.

References
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