A case of ventricular-atrial conduction near tachycardia rate via a concealed atrioventricular accessory pathway located anterior to the right pulmonary vein

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Introduction

Intermittent rate-dependent retrograde conduction over a concealed accessory pathway (AP) is a rare occurrence. Although APs are widely distributed in the mitral and tricuspid valve annuli, cases of successful ablation procedures at unusual sites, such as the left coronary cusp and middle cardiac vein, have previously been reported. In our case, the atrial attachment end of the AP, which exhibited unique conduction properties, was found in an atypical location.

Case report

An 88-year-old woman who complained of palpitations and dizziness was admitted to our hospital after an emergency at another hospital. The patient had been experiencing symptoms since a young age and was on medication. The patient presented with heart failure due to persistent tachycardia and complained of dizziness following a long pause; the tachycardia terminated spontaneously. Tachycardia recurred immediately after a long pause.

Two types of tachycardia were noted on the surface electrocardiogram: a narrow QRS complex and a wide QRS complex with a left bundle branch block. We observed Coumel phenomenon, in which the heart rate decreased from 169 beats per minute (bpm) to 160 bpm when transitioning from a narrow QRS to a wide QRS with the left bundle branch block. No pre-excitation was observed on the surface electrocardiogram during sinus rhythm. Therefore, we decided to treat the patient with temporary cardiac pacing for the prolonged pauses, followed by medication. Tachycardia disappeared after the placement of temporary pacing, and the patient's subjective symptoms and heart failure improved drastically. Echocardiography performed after improvement of heart failure showed normal cardiac function.

We obtained written informed consent for an electrophysiological study (EPS) and catheter ablation. The EPS was performed under mild sedation, using a 20-pole electrode catheter with 12 electrodes directly inserted into the coronary sinus (CS) and 8 electrodes located in the right atrium and inserted into the CS from the right atrium, a 4-pole catheter placed in the apex of the right ventricle (RV), and an 8-pole catheter positioned in the bundle of His region.

Based on the EPS findings (Table 1, Figures 1 and 2, Supplemental Figures 1 and 2), we determined that atrioventricular conduction occurred through the atrioventricular node (AVN). We diagnosed that ventricular-atrial (VA) conduction at RV pacing of $\leq$110 bpm was through the AVN, compared with that at RV pacing of $\geq$130 bpm, which was through the AP. Additionally, AP conduction was observed when adenosine triphosphate (ATP) was administered during RV pacing at 100 bpm. These VA conduction findings are summarized in Supplemental Figure 3. However, we could not evaluate the AP's decremental conduction property.

KEY TEACHING POINTS

- Pacing close to the clinical tachycardia rate may be effective if no accessory pathway (AP) is observed in a routine electrophysiological study.
- Adenosine triphosphate administration may help in such cases.
- Consider the possibility that the atrial attachment end of the AP is located far from the mitral or tricuspid annulus.

KEYWORDS Accessory pathway; Rate-dependent retrograde conduction; Atrioventricular reentrant tachycardia; Supraventricular tachycardia; Adenosine triphosphate

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Table 1 Electrophysiological study findings

Sinus rhythm and AV conduction	• Baseline sinus rhythm of 64 bpm • AV conduction with a decremental conduction property • No jump-up phenomenon
VA conduction	• RV pacing at 70 bpm presenting with a Wenckebach-type VA block. • RV pacing at 100 and 110 bpm presenting a 2:1 VA conduction, with the earliest atrial activation site of the His bundle region (Figure 1A-1 1A-2). • RV pacing at 130 and 150 bpm resulting in a 1:1 VA conduction, with the earliest atrial activation site of the proximal CS (Figure 1B-1 and 1B-2). • RV pacing at 160 and 170 bpm presenting a 2:1 VA conduction with the earliest atrial activation site of the proximal CS (Figure 1C-1 and 1C-2). • Extrastimulation from the RV showing a slight prolongation of the stimulus-A interval from 212 ms at a basic drive CL of 400 ms to 234 ms when the coupling interval is 360 ms but the VA block is at a coupling interval of 350 ms. • ATP 20 mg administration during RV pacing at 150 bpm with no block in VA conduction. • ATP 20 mg administration during RV pacing at 100 bpm with transient 1:1 VA conduction and the earliest atrial activation site at the proximal CS (Figure 2). • Evaluation of para-Hisian pacing not done because of simultaneous VA capture and tachycardia induction.
SVT	• Atrial rapid pacing and program stimuli from the distal CS easily induce SVT with a CL of 390 ms. • During SVT, recording of the earliest atrial activation at the proximal CS, and spontaneous termination of tachycardia with a VA block. • Premature ventricular beats with RV pacing during antegrade refractoriness of the His bundle terminate the SVT. • Postpacing interval for entrainment pacing from the RV apex is tachycardia CL + 30 ms.

ATP = adenosine triphosphate; AV = atrioventricular; bpm = beats per minute; CL = cycle length; CS = coronary sinus; RV = right ventricle; SVT = supraventricular tachycardia; VA = ventricular-atrial.

We created an atrial activation map using a remote magnetic navigation system (Navistar RMT ThermoCool™; Biosense Webster, Inc, Irvine, CA) during RV pacing at 150 bpm. The earliest activation site in the left atrium (LA) was anterior to the right pulmonary vein, far from the mitral annulus. We also created an activation map for the right atrium; however, activation in the LA occurred earlier. Ventricular or AP potentials were not found at the earliest activation site of the LA; however, AP conduction disappeared immediately after delivery of radiofrequency energy (Figure 3). After ablation, we administered ATP 20 mg during RV pacing at 100 bpm, resulting in a VA conduction block (Supplemental Figure 4).

Six months after the procedure, the patient experienced no recurrent palpitations or dizziness.

Discussion

To the best of our knowledge, this is the first report of a rate-dependent concealed AP connected to the anterior part of the right pulmonary vein.

Similar to this case, several cases of AP with rate-dependent VA conduction have been reported. The causes of this rare phenomenon are considered catecholamine-induced changes in the AP refractory period, pacing site–dependent conduction block, supernormal retrograde conduction, bradycardia-dependent block,[1] and phase 3 or phase 4 retrograde block.[2] In our case, further differentiation was difficult because we did not use catecholamines, change the RV pacing site, slow down the sinus rhythm, or evaluate VA conduction at slower rates before radiofrequency energy delivery.

The appearance of the AP after ATP administration could be associated with inhibition of retrograde AVN conduction, ATP directly acting on the AP to improve its conduction, or endogenous "rebound" catecholamines released because of ATP-induced hypotension promoting AP conduction. In this case, AVN conduction was initially weak, which may be associated with improved AP conduction owing to ATP. Additionally, although differentiating these atrial waveforms observed after ATP administration (Figure 2) from accelerated sinus rhythm was challenging, we believe that these waveforms were most likely mediated by AP, based on comparison with the waveforms during sinus rhythm (Supplemental Figure 1) and other tracings.

The sites where the AP connects to the atrium are widely distributed in the mitral and tricuspid annuli.[3] There are case reports in which the AP was ablated at the left coronary cusp[4,5] or middle cardiac vein with satisfactory efficacy.[6] Although further evaluation is difficult because CS angiography was not performed in this case, the CS–ventricular AP formed by the CS myocardial coat is widely connected to the left and right atria,[7] and the anatomy of the CS is highly variable,[8] which may explain some of these atypical attachment ends of the APs, including the present case.

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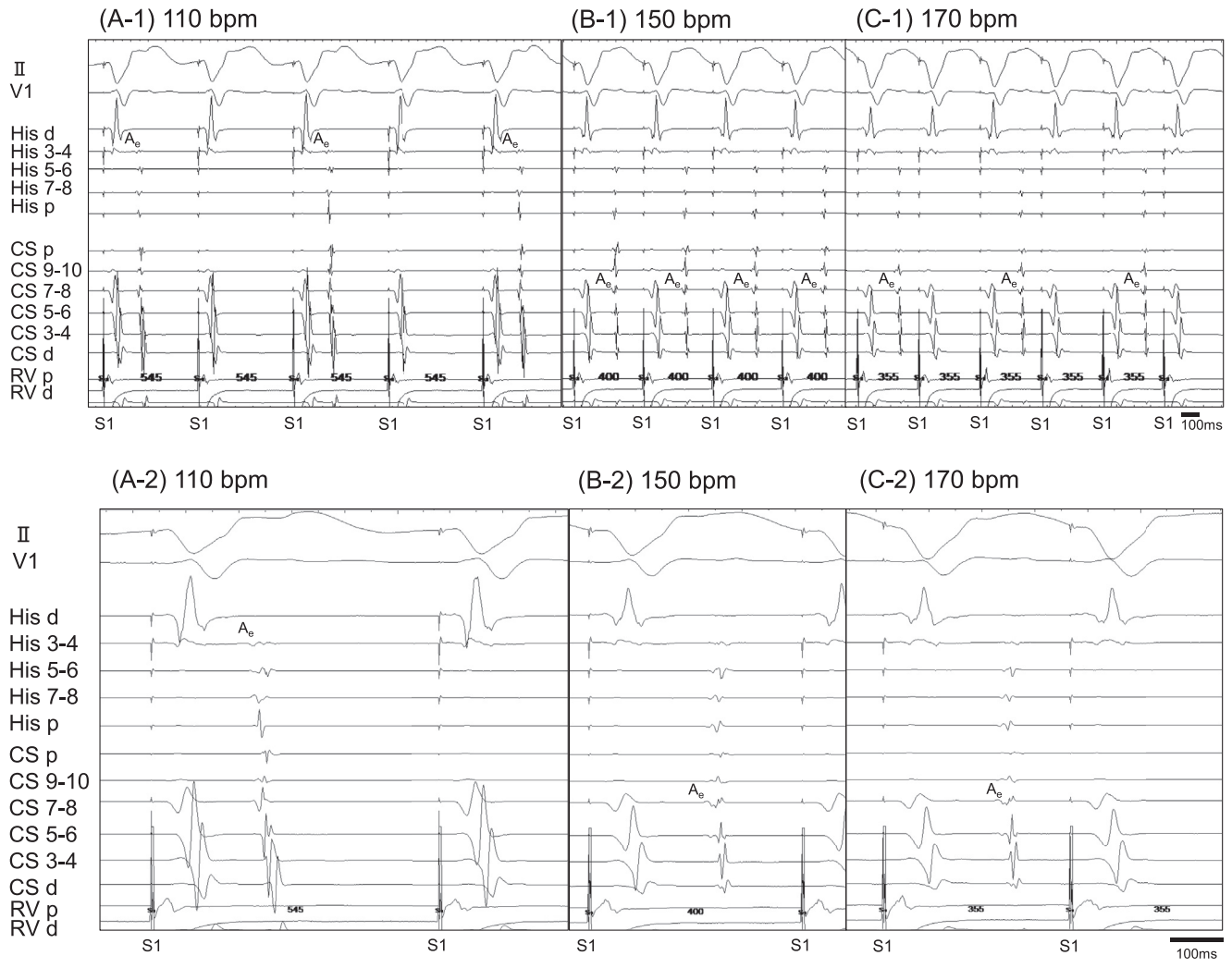

Figure 1 Ventricular pacing during sinus rhythm. **A-1:** During ventricular pacing at 110 beats per minute (bpm), 2:1 ventricular-atrial (VA) conduction is shown, with the earliest atrial activation (A_e) in the His bundle region (paper speed 50 mm/s). **B-1:** During ventricular pacing at 150 bpm, 1:1 VA conduction is shown, with the A_e in CS 7–8 (paper speed 50 mm/s). **C-1:** During ventricular pacing at 170 bpm, 2:1 VA conduction is shown, with the A_e in CS 7–8 (paper speed 50 mm/s). **A-2, B-2, C-2:** Magnified waveforms of A-1, B-1, and C-1, respectively (paper speed 200 mm/s). CS = coronary sinus; d = distal; His = His bundle; p = proximal; RV = right ventricle; S1 = stimulus artifact.

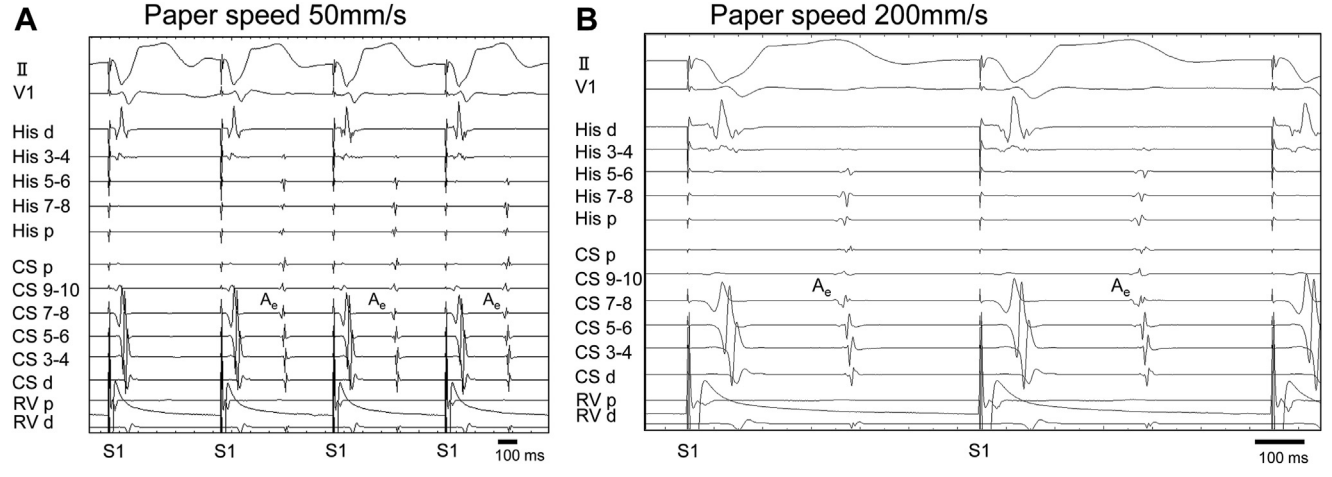

Figure 2 Adenosine triphosphate (ATP) administration during right ventricle (RV) pacing. **A:** When ATP 20 mg is administered during RV pacing at 100 beats per minute, 1:1 ventricular-atrial conduction is transiently observed, and the earliest atrial activation site (A_e) is coronary sinus (CS) 7–8 (paper speed 50 mm/s). **B:** Magnified waveform of A (paper speed 200 mm/s). d = distal; His = His bundle electrogram; p = proximal; S1 = stimulus artifact.

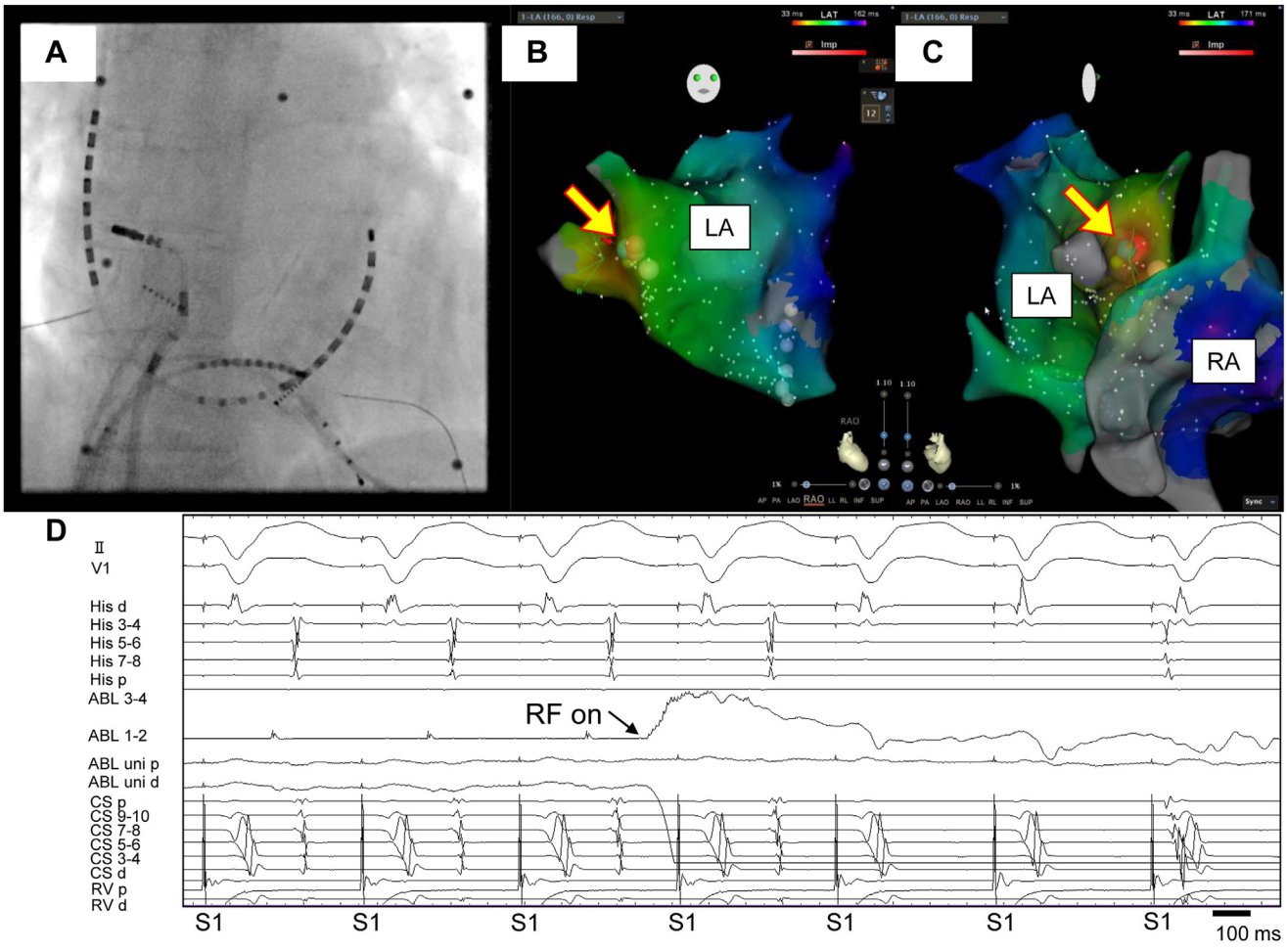

Figure 3 Earliest atrial activation site. **A:** Ablation catheter is placed at the earliest activation site in the left atrium (LA). **B, C:** Atrial activation map during right ventricle pacing at 150 beats per minute. The earliest activation site is anterior to the right pulmonary vein (*yellow arrow*). **D:** Successful radiofrequency (RF) ablation. Ventricular or accessory pathway (AP) potentials are not found in the earliest activation site of the left atrium (LA). The AP conduction is blocked immediately after delivering RF energy. Paper speed 100 mm/s. ABL = ablation catheter; CS = coronary sinus; d = distal; His = His bundle; p = proximal; RA = right atrium; RV = right ventricle; S1 = stimulus artifact.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.06.001.

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