Intraatrial conduction block in the right posteroseptal region after failed accessory pathway ablation—Importance of delineation of three-dimensional pathway geometry

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Introduction

Although catheter ablation has become routine treatment for accessory pathway (AP)-mediated tachycardias,[1,2] the procedure still fails in a minority of cases. One of the causes of failure is incomplete mapping resulting in inaccurate pathway localization. We report a case of atrioventricular reentrant tachycardia (AVRT) using a right posteroseptal AP that recurred following prolonged ablation attempts that led to local tissue injury and low-amplitude electrograms as well as intra-atrial conduction block in an area adjacent to the atrial insertion of the AP.

KEY TEACHING POINTS

- Prolonged radiofrequency applications during catheter ablation of accessory pathways can significantly alter the atrial activation pattern, making further diagnostic and therapeutic efforts much more difficult.
- Iatrogenic block can occur after accessory pathway ablation.
- This case underscores the importance of precise localization of accessory pathways to maximize the chances of a successful ablation at the initial procedure.

Case report

Clinical history

The patient is a 25-year-old woman who presented after an episode of syncope, which occurred after sudden onset of palpitations. She had minimal warning symptoms and sustained facial injuries as a result of the syncopal episode. She was hospitalized, at which time an electrocardiogram revealed ventricular preexcitation and an echocardiogram revealed a structurally normal heart. Given this presentation a decision was made to proceed with electrophysiology study and catheter ablation. The procedure was performed at an outside hospital.

Description of prior ablation

At initial electrophysiology study, the patient was found to have a right posteroseptal AP. The ensuing procedure lasted approximately 6 hours and the pathway was described as difficult to define and ablate. Using an 8 mm catheter, extensive radiofrequency (RF) ablation was performed in the right posteroseptal region of the right atrium close to the tricuspid annulus. Preexcitation recurred after the ablation procedure and the patient was started on flecainide and metoprolol. Following discharge, she experienced recurrent episodes of highly symptomatic supraventricular tachycardia. Given this presentation, the patient was referred to our center for a redo procedure after a failed initial RF ablation attempt.

Electrophysiology study

Baseline electrocardiogram showed ventricular preexcitation with delta waves that were negative in the inferior leads, positive in lead I, and isoelectric in lead V_1, consistent with the prior diagnosis of right posteroseptal AP (Figure 1A).[3] Quadripolar electrode catheters were positioned at the high right atrium (HRA), His-bundle area, and right ventricular apex. A steerable decapolar electrode catheter was placed in the coronary sinus (CS). Baseline intracardiac conduction intervals were AH 76 ms, HV 16 ms (Figure 1B). During

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ventricular pacing the earliest retrograde atrial activation was recorded on the HRA catheter, consistent with retrograde conduction via a right-sided AP (Figure 1C). The earliest retrograde atrial activation recorded on the CS catheter was at the proximal CS. During ventricular pacing, retrograde conduction occurred via both the AP (HRA activated early) and atrioventricular (AV) node (persistent VA conduction after retrograde block in the AP was achieved with single ventricular extrastimulus). Para-Hisian pacing demonstrated a fusion/AP pattern (Figure 1D). The stimulus-A interval recorded from the HRA catheter was similar during the beats with and without His capture, suggesting that the atria were activated by the AP throughout the tracing. With loss of His capture there was a change in the atrial activation sequence, indicating loss of retrograde atrial activation via the AV node. Therefore, the atria were activated by both the AV node and the AP (fusion) during His capture and by only the AP during loss of His capture. There was 1:1 VA conduction down to a paced cycle length of 290 ms. During programmed atrial stimulation the antegrade effective refractory period of the AP was 290 ms at a drive cycle length of 400 ms. During induced atrial fibrillation the shortest preexcited R-R interval was 277 ms. There was no evidence of dual AV node physiology before or after ablation.

Tachycardia characteristics

Supraventricular tachycardia at a cycle length of 397 ms was reproducibly induced with ventricular pacing at a cycle length of 380 ms (Figure 1C). The electrocardiogram revealed a narrow complex short RP tachycardia. While there was a degree of QRS morphology variability likely owing to intraventricular conduction delay, all QRS complexes of the tachycardia were less than 100 ms in duration. The ventriculoatrial activation patterns during ventricular pacing and during tachycardia were similar, suggesting retrograde conduction via the AP in both. The initial AH interval at tachycardia initiation was long, likely because of concealed retrograde conduction in the AV node during the ventricular pacing train.

During tachycardia the AH interval was 129 ms and the HV interval was 57 ms. Premature ventricular complexes delivered during tachycardia at a time of His bundle refractoriness resulted in atrial preexcitation and advancement of the next ventricular complex, confirming the presence and participation of the AP in the tachycardia circuit (Figure 2A).

Mapping and ablation

We used a 4-mm deflectable-tip ablation catheter for both mapping and ablation, which were guided by 3-dimensional Carto electroanatomic mapping (Biosense Webster, Diamond Bar, CA). Atrial activation sequence mapping was performed during tachycardia. The earliest retrograde atrial electrogram (Figure 2C) was mapped to the right posteroseptal area (approximately 5 o'clock on the tricuspid annulus). Mapping was difficult given the very low-amplitude electrograms seen in this area, likely owing to previous ablation lesions.

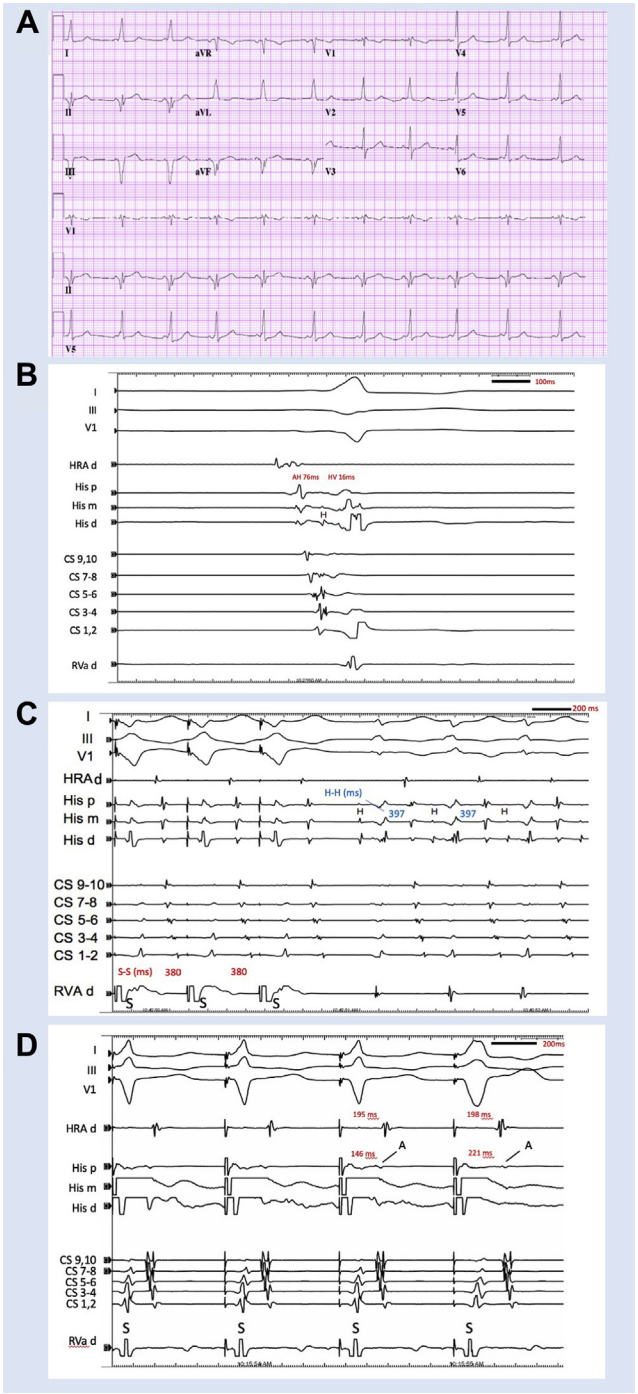

Figure 1 **A:** Baseline electrocardiogram. **B:** Intracardiac electrogram at baseline. **C:** Induction of supraventricular tachycardia with ventricular pacing. **D:** Para-Hisian pacing demonstrating a fusion/accessory pathway pattern. CS 1–10 = coronary sinus catheter from the distal to the proximal pole; H = His deflection; His d = His bundle electrogram distal; His m = His bundle electrogram mid; His p = His bundle electrogram proximal; HRA d = high right atrium distal bipole; RVA = right ventricular apex.

In addition, just medial to the area of earliest atrial electrogram, in the vicinity of the ostium of the CS, the atrial electrograms were much delayed, occurring later than the HRA, His, and CS, suggesting conduction block across the posteroseptal region. A recording with the ablation catheter straddling the area of conduction block is shown in Figure 2B, and the

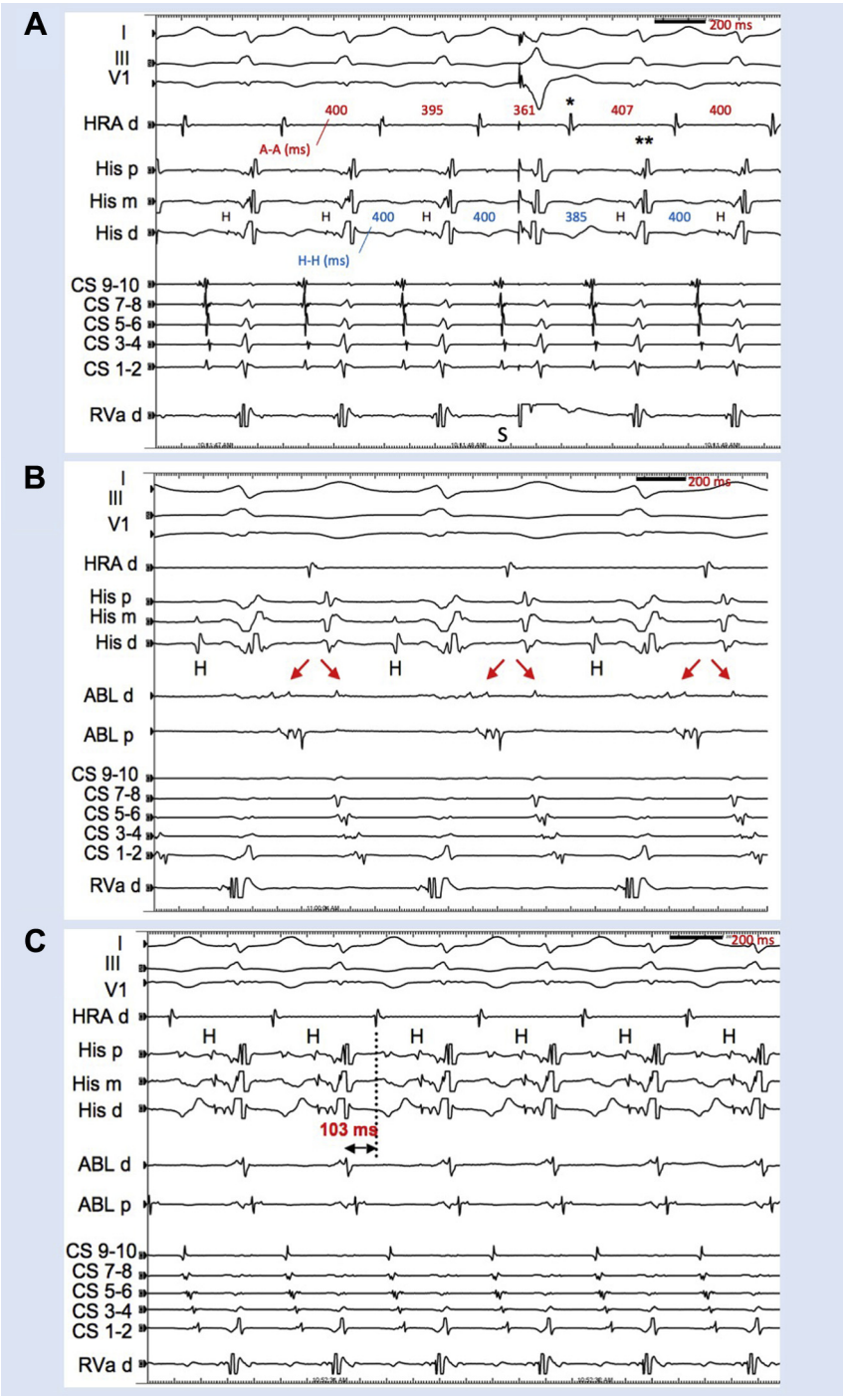

Figure 2 **A:** Premature ventricular complex delivered during tachycardia at a time of His bundle refractoriness resulted in atrial preexcitation (*asterisk*) and also advancement of the next ventricular complex (*double asterisk*), confirming the presence and participation of the accessory pathway in the tachycardia circuit. **B:** Ablation catheter slightly medial to the area of earliest atrial electrogram. Multiple early potentials and 1 delayed (100–110 ms) potential on the proximal ablation channel. **C:** Ablation catheter at the site of earliest atrial electrogram during tachycardia. Delivery of radiofrequency ablation at this site terminated the tachycardia and abolished accessory pathway conduction. Abl d = ablation distal; Abl p = ablation proximal; CS 1–10 = coronary sinus catheter from the distal to the proximal pole; H = His deflection; His d = His bundle electrogram distal; His m = His bundle electrogram mid; His p = His bundle electrogram proximal; HRA d = high right atrium distal bipole; RVA = right ventricular apex.

corresponding fluoroscopic image is shown in Figure 3A. The distal bipolar electrogram recorded on the ablation catheter depicts multiple early components likely originating in areas adjacent to the AP, and a late component generated from tissues situated on the medial side of the zone of block, which is later than the atrial electrograms on the HRA, His, and CS proximal channels. The late component occurred 100–110 ms later than the early components. This activation pattern was due to the zone of conduction block (or very slow conduction) situated medial to the atrial insertion site of the AP

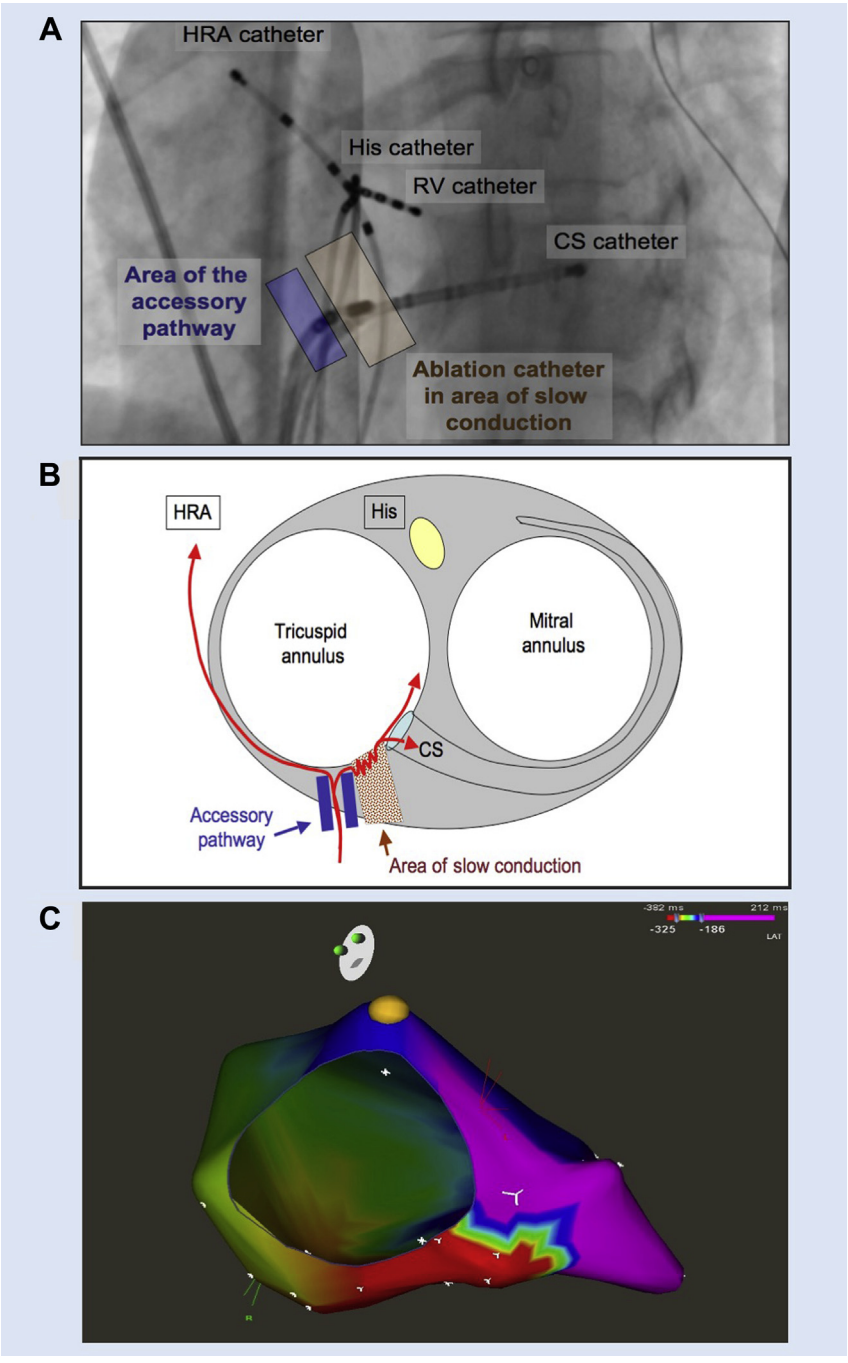

Figure 3 **A:** Fluoroscopy image with catheter positions during the study. The ablation catheter is positioned in the region of conduction block. **B:** Diagram depicting the likely activation pattern during tachycardia. **C:** Activation map during atrioventricular reentrant tachycardia using the right posteroseptal pathway. Arrowhead marks the area of conduction block.

(Figure 3B) that prevented (or slowed) the direct propagation toward the CS and His, so that the retrograde atrial activation during tachycardia started at the AP insertion site, progressed upwards toward the HRA, and later reached the His and CS. RF ablation was delivered in the area of earliest atrial electrogram during tachycardia and resulted in termination of tachycardia. A total of 11 RF applications (total RF time: 7.4 minutes) were delivered in this area.

Following the ablation, both antegrade and retrograde AP conduction were abolished. Postablation the HV interval was 48 ms, and AV Wenckebach block was noted at a paced cycle length of 310 ms. Retrograde VA Wenckebach block occurred at a paced cycle length of 650 ms. CS pacing revealed significant delay in medial-to-lateral conduction across the right posteroseptal area: 100–120 ms delay from CS 9–10 to the ablation catheter positioned lateral to atrial insertion of the AP. This delay was comparable to the delay in the lateral-to-medial direction noted earlier in the study during tachycardia (Figure 3C). In addition, pacing laterally from the atrial insertion of the AP revealed a 120 ms delay

to CS 9–10. There was therefore bidirectional block/delay across the right posteroseptal area.

Follow-up

The patient was seen in the arrhythmia clinic 1 month after the ablation. She was feeling well, with no recurrent symptoms. The electrocardiogram revealed sinus rhythm with normal conduction intervals, and no delta wave.

Discussion

Primary failure of RF ablation and recurrence of AP conduction occur more frequently in patients with right-sided pathways. Unstable local electrograms and prolonged time to block are independent predictors for recurrence.[4] In a consecutive series of 619 patients undergoing catheter ablation of an AP, Morady and colleagues[5] identified several predictors of failed ablations or recurrence of AP conduction. Even though the septal AP location was not associated with a particularly high risk of ablation failure, inaccurate mapping of the AP was one of the predictors. In our patient the AP was not accurately localized at the first electrophysiology procedure and extensive ablation was delivered slightly medial to the AP location. This led to significant reductions in the amplitude of the local electrograms and to conduction block that caused major changes in the atrial activation sequence, making further attempts at pathway localization difficult. Even though local conduction delay does not occur frequently after RF ablation, there are reports of iatrogenic block in patients who underwent ablation for left-sided APs. A report by Bortone and colleagues[6] described previous ablation for concealed left-sided AP creating iatrogenic block responsible for a split retrograde atrial activation pattern during orthodromic AVRT. The AP was ablated at the site with the shortest interval between the ventricular signal and the earliest component of the retrograde atrial activation, as was done in our patient during the second procedure. Meticulous mapping during AVRT in patients with an unusual retrograde atrial activation pattern was the strategy that finally allowed accurate localization and eventual elimination of the AP. In a retrospective analysis of 159 patients who underwent RF ablation of a left free-wall AP, mitral isthmus conduction block was observed following RF delivery in 6.9% of cases.[7] This was evidenced by a sudden change in retrograde left atrial activation sequence despite persistent AP conduction. In some patients, reversal of eccentric atrial activation during orthodromic AVRT falsely suggested the presence of a second (septal) AP.[7] In our patient the abnormal atrial activation sequence was noted after attempts to ablate a right-sided AP in an area of thick myocardial wall. The likely explanation for creation of this line of block in this unusual location is the extensive amount of ablation that was performed using an 8 mm ablation catheter. It is likely that mapping prior to ablation was insufficient. Ablation with an 8 mm catheter led to extensive tissue injury medial to the AP, making subsequent efforts to map and ablate more difficult. It is difficult to ascertain whether the conduction across the right posteroseptal area was completely abolished (conduction block) or significantly delayed (extremely slow conduction). The facts that the local double potential interval was >100 ms and the apparent conduction time across the area with CS pacing was >100 ms are suggestive of bidirectional conduction block,[8] although extremely slow conduction cannot be excluded with certainty.[9]

Significant changes in the atrial activation sequence have been noted not only after AP ablations in the right posteroseptal region. Complete isolation of the CS has been described after an attempt to ablate an atrial tachycardia originating in the area of the CS ostium.[10]

In summary, this case underscores the importance of precise localization of APs to maximize the chances of a successful ablation at the initial procedure, and the fact that prolonged radiofrequency applications can significantly alter the atrial activation pattern, making further diagnostic and therapeutic efforts much more difficult.

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