Radiofrequency Ablation of a Right Atriofascicular Mahaim Fiber and Two Contralateral Left Free-wall Accessory Pathways

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Summary

We report a rare combination of a right atriofascicular Mahaim fiber and two left-sided atrioventricular accessory pathways in a 57-year-old female presenting with an antidromic atrioventricular reciprocating tachycardia. Radiofrequency ablation was first targeted at the left lateral accessory pathway that served as the retrograde limb of the tachycardia. After elimination of the left lateral pathway, a bystander left posterolateral pathway was detected, and it too was successfully ablated. Although no tachycardia was inducible, the Mahaim pathway was ablated because of its short effective refractory period. A discrete Mahaim potential recorded at the right atrial free-wall successfully guided the ablation (Jpn Heart J 1999; 40: 481–487).

Key words: Accessory pathways, Mahaim fiber, Radiofrequency ablation

Mahaim fiber is a distinct bypass tract exhibiting a unique antegrade and decremental conduction property.1) When an antidromic atrioventricular reciprocating tachycardia is utilizing the Mahaim pathway as the antegrade limb, the atrioventricular node or, less commonly, an atrioventricular accessory pathway (AP) is incorporated as the retrograde limb. In the latter setting, the Mahaim fiber is associated with a single ipsilateral or contralateral AP.2–6) Hluchy et al recently reported an unusual combination of two ipsilateral right APs and a right atriofascicular Mahaim fiber.6) In this report, we present the exceptional coexistence of a right atriofascicular Mahaim fiber with two contralateral left-sided APs in a patient with an antidromic atrioventricular reciprocating tachycardia. Both left-sided APs (the culprit left lateral AP and a bystander left posterolateral AP) and the Mahaim fiber were successfully ablated using radiofrequency (RF) current.

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**Case Report**

A 57-year-old female presented with a 3-year history of episodic palpitations associated with dizziness and chest pain. At the last attack, ECG documented a wide QRS complex tachycardia (heart rate 150/min) with left bundle branch block morphology and normal axis. An intravenous injection of 6 mg adenosine terminated the tachycardia, following which repeat ECG showed a narrow QRS complex without ventricular preexcitation. There was no evidence of structural heart disease by physical examination, chest roentgenography, or echocardiography.

**Electrophysiologic study:** After obtaining informed consent, the electrophysiologic studies were performed using standard protocol. During sinus rhythm, the QRS duration was 80 msec, the AH 98 msec, and HV 43 msec. During incremental atrial pacing, ventricular preexcitation with the earliest site of ventricular activation was registered at the right ventricular apex with a discernible right bundle potential (Figure 1). The right bundle branch activation was recorded before the His bundle potential which became gradually merged into the ventricular electrogram (Figure 1). Decremental conduction between the atrial stimulus and the right bundle potential was demonstrated (Figure 1). A clinical tachycardia with a cycle length (CL) of 420 msec and a QRS axis of 40° was induced at an atrial paced CL of 360 msec. The retrograde atrial activation sequence was eccentric with the earliest activation inscribed at the mid-coronary sinus electrogram. A single ventricular extrastimulus delivered during the His bundle refractory period advanced subsequent atrial activation and reset the tachycardia. A late right atrial extrastimulus delivered 20 msec after onset of the septal atrial electrogram advanced subsequent ventricular activation by 13 msec with the same preexcited QRS morphology (Figure 1).

During sinus rhythm a single atrial extrastimulus of 280 msec coupled to a driving CL of 600 msec also induced the tachycardia. The effective refractory period of the atriofascicular accessory pathway was 270 msec at the same driving CL. During ventricular overdrive pacing, the retrograde atrial activation sequence was identical to that during tachycardia (Figure 2). At a ventricular paced CL of 480 msec, second degree block occurred in the concealed retrograde AP. Using a single ventricular extrastimulus coupled to a driving CL of 600 msec, the effective refractory period of the retrograde accessory pathway was 480 msec. The tachycardia was not induced by ventricular stimulation.

**Radiofrequency ablation:** A deflectable 7F quadripolar catheter (Webster) was used for mapping and temperature-guided RF ablation. During constant overdrive ventricular pacing (600 msec), within 9 seconds (30 Watt, 56°C) of single RF pulse application, loss of VA conduction via the left lateral AP was
Figure 1. A: Rapid atrial pacing at a cycle length of 350 msec results in ventricular preexcitation, progressive conduction delay between atrial stimulus and right bundle potential, and reversed activation sequence of right bundle branch and His bundle. The sixth atrial stimulus fails to capture the atrium (arrow in HRA). Top to bottom: surface ECG leads I, aVF, V1, and intracardiac electrograms from high right atrium (HRA), distal (CSd) and proximal coronary sinus (CSp), right bundle branch (RB), and distal (HISd), middle (HISm), and proximal His bundle (HISp). B: A late atrial extrastimulus delivered 20 msec after onset of the septal atrial electrogram (arrowhead in HISd) during tachycardia advances subsequent ventricular activation by 13 msec without a change in preexcited QRS morphology. A2-S = septal atrial electrogram to extrastimulus interval; A2 = local high right atrial capture from extrastimulus.
Figure 2. Mapping and ablation of two atrioventricular accessory pathways (A, A', B and B') and Mahaim fiber (C and C'). During mapping of left-sided atrioventricular pathways, a decapolar catheter (inter electrode spacing of 2-10 mm) with its distal (CS1,2) and proximal (CS9,10) electrodes, respectively, positioned at distal coronary sinus and coronary sinus orifice, is used as a reference. S = stimulus artifact; ABL = ablation; M = Mahaim potential; arrow = the earliest atrial activation at the successful ablation site; arrowhead = loss of Mahaim potential. Other abbreviations are as in Figure 1. See text for discussion.
observed (Figure 2). A booster RF was delivered for 30 seconds during sinus rhythm. Repeat ventricular pacing revealed a second eccentric retrograde atrial activation sequence with the earliest site of activation in the proximal coronary sinus. During constant ventricular pacing, RF current was applied, and the left posterolateral AP was eliminated in 1 second with loss of VA conduction (Figure 2). A booster RF application was delivered for 30 seconds. After ablation, repeat electrophysiologic study was performed with and without isoproterenol infusion (2 μg/min). The shortest atrial paced CL in maintaining 1:1 antegrade conduction over the atriofascicular pathway was 290 msec (270 msec with isoproterenol), and the effective refractory period of the pathway at a driving CL of 600 msec was 260 msec (260 msec with isoproterenol). With or without isoproterenol infl-
sion, no evidence of retrograde AP conduction or tachycardia induction was observed.

Follow-up study 4 days later showed complete elimination of the left-sided APs. During sinus rhythm a discrete Mahaim potential was identified at the lateral atrial aspect of the tricuspid annulus (Figure 3). During incremental atrial pacing, progressive conduction delay between the atrial electrogram and the Mahaim potential was demonstrated (Figure 3). Although no tachycardia was inducible, ablation of the atriofascicular pathway was attempted because of its short effective refractory period. During constant atrial pacing (600 msec), within 3.5 seconds of RF ablation (45 Watt, 45°C), conduction block over the Mahaim pathway with loss of ventricular preexcitation and abolition of the Mahaim potential developed. A booster RF application for 30 seconds was delivered during sinus rhythm. Repeat atrial stimulation with and without intravenous isoproterenol (2 μg/min) infusion documented elimination of the Mahaim pathway.

**DISCUSSION**

The characteristics of the atriofascicular pathway in this case fulfilled all the following electrophysiologic properties of Mahaim fiber: 1) decremental anterograde conduction, 2) preexcitation during atrial pacing with QRS morphology of left bundle branch block pattern, and 3) participation as the anterograde limb in antidromic atrioventricular reciprocating tachycardia.\(^\text{3,8,9}\) The right atrial free-wall origin of this pathway was determined by delivering a late atrial extrastimulus after onset of the septal atrial electrogram during the tachycardia, and a resultant observation that the extrastimulus advanced the following ventricular activation without alteration in the preexcited QRS morphology.

The concealed left lateral AP was readily identified during ventricular pacing and antidromic tachycardia. A bystander concealed left posterolateral AP was detected after successful ablation of the first AP, and was also eliminated with RF ablation. Failure to detect the second AP before elimination of the first AP was probably due to inherent limitations of endocardial mapping, pathway trauma during catheterization or concealed conduction.\(^\text{10,11}\) The lack of participation of the second AP in the antidromic tachycardia may be explained by repetitive concealed conduction into that pathway. Although no tachycardia was inducible at repeat electrophysiologic study, it was thought to be prudent to ablate the Mahaim fiber because of its short effective refractory period. Successful RF ablation of this pathway was guided by a discrete Mahaim potential at the lateral atrial aspect of the tricuspid annulus.
REFERENCES


