CASE REPORT

Atrioventricular accessory pathway with anterograde decremental conduction property

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SUMMARY

We report a case of atrioventricular (AV) accessory pathway with anterograde decremental conduction property. The δ wave polarity suggested the presence of a right posteroseptal accessory AV pathway. During atrial pacing, Wenckebach-type AV block over the accessory pathway was observed with prolongation of the local AV conduction time without the change in QRS morphology. No retrograde ventriculoatrial conduction was observed. During mapping of the right midseptal area, mechanical conduction block at the level of the proximal input to the accessory pathway was induced repeatedly by catheter manipulation. Radiofrequency energy delivery eliminated the accessory pathway. In this case only the bump phenomenon was the best marker of successful ablation.

BACKGROUND

In 1937, Mahaim and Winston¹ described tracts connecting the atrioventricular (AV) node and ventricular myocardium as well as discrete connections from the fascicles to the ventricles. Recently, several reports suggest that most of Mahaim fibres are actually decrementally conducting AV or atriofascicular connections that have a proximal origin in the atrial muscle rather than the AV node.^{2–5} The resting ECG in patients with Mahaim fibres is usually normal. But in this case, fully pre-excited QRS morphology was observed at resting ECG. We present a case of AV accessory pathway with anterograde decremental conduction property which was successfully eliminated by radiofrequency ablation.

CASE PRESENTATION

The patient is a 31-year-old woman referred for evaluation of ECG abnormalities. She experienced episodes of palpitations in her childhood. The 12-lead ECG performed on admission showed the presence of a δ wave with a PR interval of 0.10 s and QRS duration of 0.15 s, indicating typical Wolff-Parkinson-White (WPW) syndrome. δ wave polarity (positive in leads I, aVL and V1 to V6, negative in leads II, III and aVF) suggested the presence of a right posteroseptal accessory AV pathway (figure 1). Physical examination, chest radiograph and echocardiography revealed no underlying heart disease. Body surface maps showed that the location of the initial minimum by a time criterion, 40 ms after onset of the QRS complex, was C3. This suggests the accessory pathway was located in the right posterolateral area and that the location of the initial minimum by an amplitude criterion, -0.15 mV, was M1, indicating that the accessory pathway was located in the left posteroseptum⁶

(figure 2). After obtaining written informed consent, a standard diagnostic electrophysiological study was performed.⁷ Three quadripolar catheters and one decapolar catheter were introduced percutaneously and positioned at the high right atrium (HRA), His bundle recording region (HBE), right ventricular apex (RVA) and within the coronary sinus (CS) under fluoroscopic guidance. Bipolar intracardiac electrograms were filtered at 30–400 Hz and displayed simultaneously with a multichannel oscilloscope (EP Lab, Quinton, Seattle, Washington, USA). Real-time recordings were obtained with paper speeds of 50–200 mm/s, and data were recorded on magnetic disc. Programmed stimulation was performed with an isolated constant-current source (RMC-200, Nihon Koden, Tokyo, Japan). Stimuli were delivered as rectangular pulses of 2 ms duration at two times diastolic threshold. Basic intervals revealed a sinus cycle length of 764 ms and an AV interval of 46 ms at the HBE. No visible His bundle potentials could be recorded because of almost fully pre-excited QRS morphology. Ventricular potentials at HBE and CS were relatively earlier than that at RVA. This finding means that the distal insertion of the accessory pathway connected to the ventricular myocardium rather than to the fascicle. No retrograde ventriculoatrial conduction was observed either via accessory pathway or via AV node during premature ventricular extrastimulus or constant ventricular pacing, even in the setting of isoproterenol infusion. Incremental pacing from HRA at a cycle length of less than 450 ms showed decremental AV conduction properties, with a relatively long stimulus-QRS (St-QRS) interval. The QRS morphology was not changed at any pacing cycle length despite the presence of a Wenckebach block. During atrial pacing at a cycle length of 333 ms, a Wenckebach-type AV block over the accessory pathway was observed with progressive prolongation of the local AV conduction time without the change in QRS morphology (figure 3). Note the atrial electrogram at HBE was divided into two components, and the interval between them was progressively prolonged. The AV conduction curve at a basic cycle length of 600 ms and an S1S2 interval of 300 ms was discontinuous, with a discrete jump in AV conduction time of 76 ms. From these findings this accessory pathway had a Mahaim fibre physiology and may locate to the septal area because a presumed Mahaim potential was observed at HBE during constant atrial pacing.

TREATMENT

No circus movement tachycardia using this accessory pathway was induced. Because she was a physician and an athlete, the effective refractory period

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Figure 1 Twelve-lead ECG showing sinus rhythm, a short PR interval with a δ wave.

of this accessory pathway was short, transient atrial fibrillation was noted on the Holter monitoring, and several reports indicate that surgical or catheter ablation of accessory pathways often reduces the occurrence of atrial fibrillation,^{8–9} radiofrequency catheter ablation was performed. Meticulous mapping at the septal area was performed using a steerable ablation catheter with a 4 mm distal electrode. During mapping of the right mid-septal area, mechanical conduction block at the level of the proximal input to the accessory pathway was induced repeatedly by catheter manipulation. At this site, a local electrogram showed an AV interval of 43 ms without a PQS pattern on the unipolar recording method (figure 4). At this site, temperature-controlled radiofrequency energy was delivered at 50°C. During radiofrequency energy delivery, an accelerated junctional rhythm occurred, followed by a His to atrial block, prompting the immediate discontinuation of energy delivery within 15 s. Thereafter, the AH interval was 116 ms and the HV interval was 44 ms during a sinus cycle length of 844 ms. Programmed stimulation could not induce pre-excitation during either baseline conditions or isoproterenol infusion. The AV conduction curve showed no discontinuities during atrial extrastimulus

analysis. The atrial electrogram taken at HBE during constant atrial pacing showed single potential.

OUTCOME AND FOLLOW-UP

The patient remained free of symptoms over a 12-year follow-up period.

DISCUSSION

The possible anatomic conditions of Mahaim-like pre-excitation from an accessory pathway ablated at the right midseptal annulus are considered following three courses¹⁰: (1) the accessory pathway originates from the compact AV node, distal to the zone of slow conduction and inserted distally at the ventricular aspect of the midseptal annulus (true nodoventricular fibre); (2) the accessory pathway is separated from the compact AV node throughout its course; the pathway has inherent decremental anterograde conduction owing to a proximal node-like structure and a subannular ventricular insertion (AV pathway with Mahaim-like pre-excitation); (3) the accessory pathway is a common fast-conduction pathway, and decremental anterograde conduction is because of its upper insertion being located within the transitional area of the AV nodal posterior input; the absence of retrograde accessory pathway conduction is secondary to a conduction mismatch at the accessory pathway-atrial interface (true AV midseptal pathway with an unusual upper insertion site; figure 5). In our case, the bypass tract may be AV accessory pathway with Mahaim-like pre-excitation located at the mid-septal region; the decremental anterograde conduction was because of its upper insertion being located within the transitional area of the AV nodal posterior input.

Accelerated junctional rhythm during slow pathway ablation of AV nodal re-entrant tachycardia is presumably caused by stimulation of the AV junction by radiofrequency energy delivery to the posterior atrionodal inputs to the AV node.^{11–13} In other words, applications of radiofrequency energy that result in adequate heating of a specific portion of the atrionodal fibres will result in junctional rhythm. In our case, radiofrequency energy delivery at the mid-septal region where the bump phenomenon¹⁴ was observed caused an accelerated junctional rhythm. This finding suggests that a specific fibre which was ablated by radiofrequency energy should input to the AV node, and therefore, this fibre may originate from the AV node. At the site of ablation, the unipolar recordings did not show a perfect PQS pattern. Such electrographic features may be explained by the fact that ventricular insertion site of septal pathways are often situated deep beneath the endocardium, so that the distance between the catheter electrode on the endocardium and

Figure 2 Body surface electrogram. Upper panel: the location of the initial minimum by a time criterion, 40 ms after onset of the QRS complex, is C3. Lower panel: the location of the initial minimum by an amplitude criterion, -0.15 mV, is M1.

Figure 3 Surface ECG leads II, aVF and V1 and intracardiac electrograms are shown during constant atrial pacing at a cycle length of 333ms. Wenckebach-type atrioventricular (AV) block over the accessory pathway was observed with progressive prolongation of the local AV conduction time (AV) without the change in QRS morphology. Note the atrial electrogram at the His bundle recording (HBE) was divided into two components and the interval between them (AA) was progressively prolonged. CSd indicates distal coronary sinus electrogram; CSp, proximal coronary sinus electrogram; HBE d, distal HBE; HBE p, proximal HBE, RV, right ventricular electrogram, St-QRS, stimulus artefact to QRS interval.

the accessory fibre is relatively longer even when the endocardial electrode is right on site.¹⁵

Grogin *et al*¹⁶ reported six patients with Mahaim tract conduction. One of these patients showed an AV nodal re-entrant tachycardia with Mahaim bystander conduction, which was abolished by a single application of radiofrequency energy to the midseptal region (slow pathway ablation). They concluded that the Mahaim tract originated from the slow pathway responsible for AV node re-entry. In our case, the accelerated junctional

rhythm was observed during ablation at the mid-septal lesion, and the slow pathway conduction disappeared after ablation, suggesting the Mahaim fibre may take-off from the slow pathway as the case that Grogin *et al* reported.

It is not possible for our case to completely deny the existence of true nodoventricular Mahaim fibre, because the presumed Mahaim potential recorded at His bundle electrogram shown in figure 3 have disappeared after radiofrequency energy delivery to the mid-septal region where the bump phenomenon was observed.

Figure 4 Left panel: Preablation ECGs obtained during sinus rhythm. Surface ECG leads II, aVF and V1 and six endocardial recordings. Manipulation of the ablation catheter at the midseptal area results in loss of pre-excitation in the first beat because of mechanically-induced conduction block in the accessory fibre. Note that the unipolar recordings of the ablation catheter (uni) do not show a PQS pattern in the last beat during anterograde accessory fibre conducted, suggesting that the ventricular insertion site of the accessory pathway is far from the site of the distal electrode of the ablation catheter. Right panel: Radiographs illustrating catheter positions at the successful ablation site. The tip of the ablation catheter (arrow) is placed at the site of the accessory fibre atrial insertion, in the midseptal region of the tricuspid annulus. Also shown are quadripolar catheters in the high right atrium (HRA) and the bundle of His (HBE) and a decapolar catheter in the coronary sinus (CS). Top, 30° right anterior oblique view; bottom, 60° left anterior oblique view. CSos indicates ostium of CS; HBE d, distal HBE; HBE p, proximal HBE.

Figure 5 Upper panel: true nodoventricular fibre. Middle panel: atrioventricular pathway with Mahaim-like pre-excitation. Lower panel: true atrioventricular midseptal pathway with unusual upper insertion site. atriov. indicates atrioventricular; AV, atrioventricular; nodov., nodoventricular; os, ostium; RA, right atrium; RV, right ventricle.

Contributors All the authors have significantly contributed to the design and performance of the study and the analysis of the data.

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ There is no case that has been clinically demonstrated the true nodoventricular Mahaim fibre.
- ▶ Resting 12-lead surface electrocardiogram showed fully pre-excited QRS morphology.
- ▶ Electrophysiological study made diagnosis as atrioventricular accessory pathway with anterograde decremental conduction property.
- ▶ Bump phenomenon was the only optical marker to locate the ablation site.
- ▶ Presumed Mahaim potential recorded at the His bundle electrogram have disappeared after radiofrequency ablation.

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