Case Report

Wide-QRS Tachycardia Inducible by Both Atrial and Ventricular Pacing

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Key words:
Antidromic atrioventricular re-entrant tachycardia, Mahaim pathway.

We describe an interesting case of an atriofascicular re-entrant tachycardia due to a Mahaim pathway. The differential diagnosis is discussed and a review of the relevant literature is presented.

Manuscript received:
June 3, 2008;
Accepted:
October 20, 2008.

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Mahaim pathways were first described in the 1930s. Here we describe a case where an accessory Mahaim pathway participated in atriofascicular re-entrant tachycardia.

Case presentation

A 62-year-old woman with a history of recent onset paroxysmal tachycardias was referred for electrophysiological study (EPS). Initial tachycardia episodes were documented when she was in her twenties. Episodes responded to Valsalva manoeuvres, although interruption with adenosine or verapamil was necessary on two previous occasions. Physical examination was unremarkable, echocardiography revealed a structurally normal heart, and resting ECG was normal (Figure 1). During the EPS a wide-QRS tachycardia (Figure 1) was easily inducible by both atrial (Figure 2) and ventricular pacing (Figure 3). Tachycardia interruption could also be accomplished by atrial and ventricular pacing, as well as with intravenous adenosine.

The easy manipulation of the tachycardia by both atrial and biventricular pacing virtually ruled out atrial and ventricular tachycardia. The differential diagnosis in this case included atrioventricular nodal re-entrant tachycardia (AVNRT) with aberrant ventricular conduction due to bundle branch block or a bystander accessory pathway, and antidromic atrioventricular re-entrant tachycardia (AVRT) due to an accessory pathway. Atrial retrograde activation was concentric and relatively prolonged (118 ms) at the His bundle electrode, thus excluding slow-fast AVNRT. Since the small potential indicated in Figures 2 and 3 (arrows) most probably represented a His bundle potential, if this tachycardia were AVNRT we would be dealing with a slow-slow form that was conducted to the ventricles with left bundle branch block or through a bystander accessory pathway. However, during EPS, changes of atrioventricular conduction with varying degrees of aberration and decremental conduction were observed spontaneously and on atrial pacing (Figure 4). During sinus rhythm, the ECG was at times suggestive of pre-excitation due to a right-sided accessory pathway (Figure 4). The presence of intermittent pre-excitation on resting ECGs, as well as the reproduction of this pattern by atrial pacing at a sufficiently high rate, argued in favour of the presence of an accessory pathway. The LBBB pattern at full pre-excitation and the response to atrial pacing were suggestive of a Mahaim atriofascicular or nodoventricular pathway.[1]

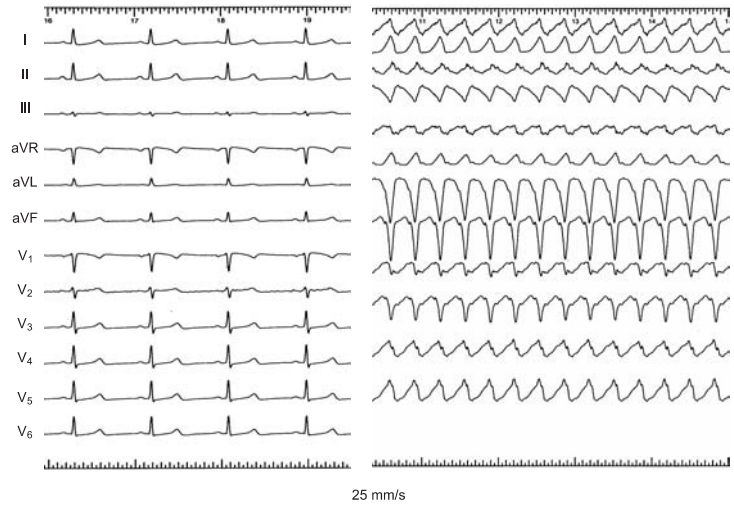

Figure 1. Twelve-lead ECGs during sinus rhythm (left panel) and tachycardia (right panel). I, II, II, aVR, aVL, aVF, V_1 to V_6 – surface ECG leads.

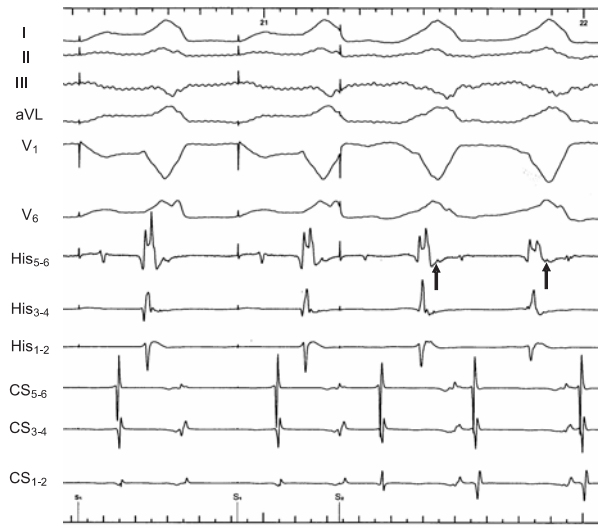

Figure 2. Induction of tachycardia by atrial pacing. During constant atrial pacing at a cycle length of 500 ms the surface ECG is pre-excited. The last extrastimulus at 320 ms induces maximum pre-excitation, thus indicating conduction along the Mahaim fibre. The impulse activates the His retrogradely (arrows) through the right bundle branch and then, through the slow pathway of the atrioventricular node, is conducted to the atria. I, II, III, aVL, V_1, V_6 – surface ECG leads; His – His bundle; CS – coronary sinus.

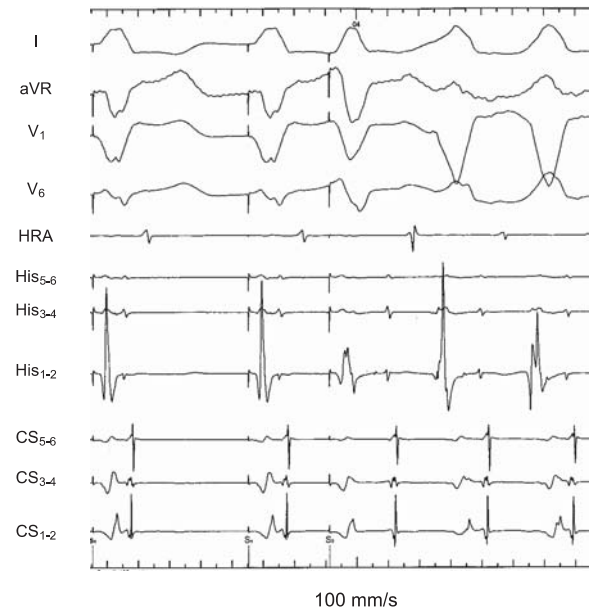

Figure 3. Induction of tachycardia by ventricular pacing. During constant ventricular pacing at a cycle length of 500 ms retrograde atrial activation is concentric through the atrioventricular node. The last extrastimulus at 260 ms results in prolonged retrograde conduction that returns antegradely via the Mahaim pathway. The next beat is a typical antidromic re-entry beat, with the His bundle (arrow) being activated retrogradely through the right bundle branch and then through the slow pathway of the atrioventricular node (as indicated by the prolonged activation time) to the atria. I, aVR, V_1, V_6 – surface ECG leads; HRA – high right atrium; His – His bundle; CS – coronary sinus.

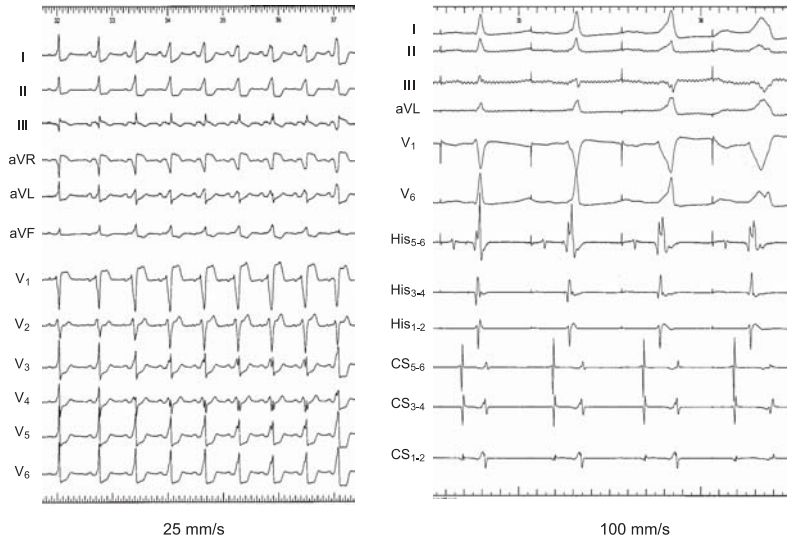

Figure 4. Left panel: Spontaneous pre-excitation during sinus rhythm. Right panel: Varying degrees of pre-excitation during atrial pacing. Normal atrioventricular nodal conduction (first beat), fusion between nodal and Mahaim conduction (next two beats) and full left bundle branch block appearance due to conduction over the Mahaim pathway (last beat) are depicted. Abbreviations as in Figures 2 and 3.

Differentiation between AVNRT with the bystander Mahaim pathway and antidromic AVRT in this setting can be achieved by atrial extrastimulation.[2] Atrial extrastimuli delivered during tachycardia after recording of the His bundle atrial electrogram (Figure 5) advanced the next pre-excited ventricular, His bundle, and atrial electrograms by 22 ms, without changing the retrograde atrial activation sequence. This argued in favour of a macro-re-entrant circuit with the Mahaim as the antegrade limb and the AV node as the retrograde limb of the tachycardia. The inscribed potential on the His bundle electrode in Figures 2 and 3, therefore, represents a His electrogram that is activated retrogradely through the right bundle branch. Subsequent retrograde atrial activation is most probably accomplished through the slow pathway of the AV node, judging from the prolonged activation sequence and the site of earliest activation that was identified in the posterior septum at the anatomic site of the right inferior nodal extension.[3,4] In Figure 4, right panel, the first beat is conducted normally through the AV node and the His bundle. The next two beats represent fusion beats between AV nodal and Mahaim conduction, and the last beat is fully pre-excited, suggesting that the inscribed His potential is due either to delayed activation of the His bundle, or to retrograde activation of the His bundle via the right bundle branch. Application of the St. George's algorithm to the fully pre-excited ECG suggested a right lateral pathway.[5]

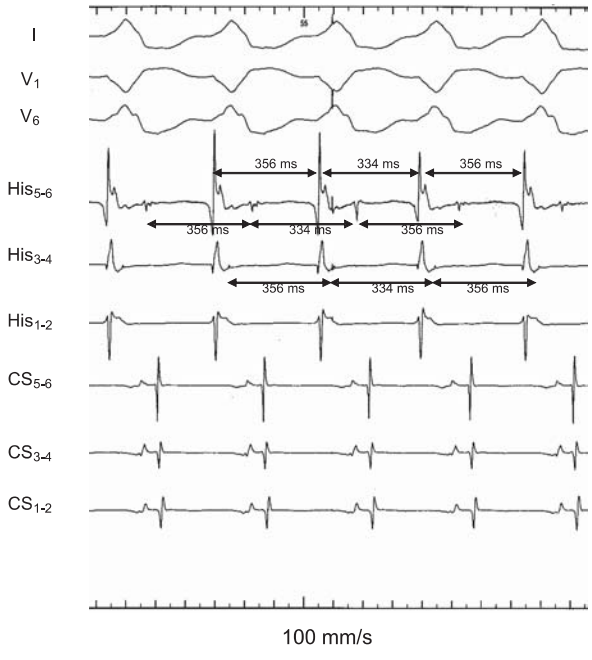

Figure 5. Resetting of tachycardia by atrial extrastimuli. Please note advancement of ventricular, retrograde His, and atrial electrograms by 22 ms without affecting retrograde activation sequence. I, V_1, V_6 – surface ECG leads; His – His bundle; CS – coronary sinus.

The anterior tricuspid annulus was mapped during atrial pacing and full pre-excitation and a distinct Mahaim potential followed by an early ventricular electrogram was recorded (Figure 6, left panel, arrow). However the ablation electrode position was not stable, as documented by the inconsistently recorded pathway potential and ventricular electrograms. Radiofrequency current delivery at this site resulted in a transient loss of pre-excitation, which reappeared following the cessation of current delivery. The lady underwent a repeat procedure the next day. A long SR0

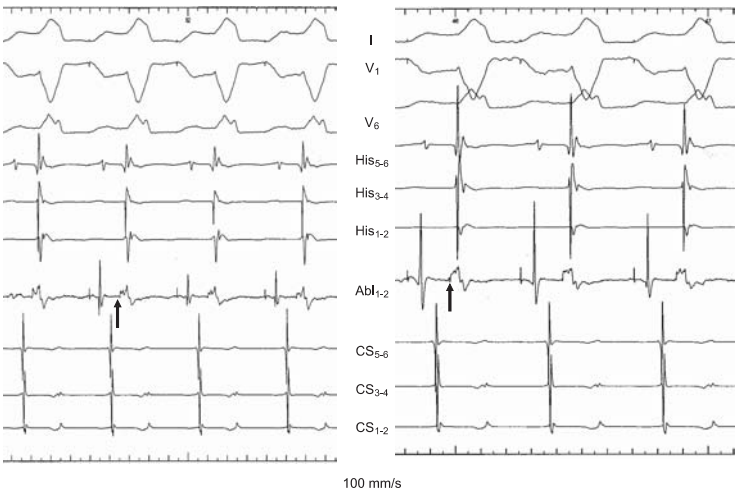

Figure 6. Mapping of the tricuspid annulus with the ablation catheter during atrial pacing. Left panel: A possible Mahaim potential (arrow) followed by a relatively early ventricular electrogram is recorded. Catheter instability, reflected in inconsistent electrogram recording, would not allow permanent loss of pre-excitation. Right panel: Recording of a Mahaim potential (arrow) and earliest ventricular activation at the anterior tricuspid annulus during atrial pacing and maximum pre-excitation. Radiofrequency current delivery at this point resulted in permanent loss of pre-excitation within 5 s. I, V_1, V_6 – surface ECG leads; Abl – ablation catheter; His – His bundle – CS: coronary sinus.

sheath was used through the right femoral vein, in order to ensure ablation catheter stability, and mapping was resumed. A consistent Mahaim potential followed by a continuous ventricular electrogram was recorded (Figure 6, right panel). Radiofrequency current delivery resulted in loss of pre-excitation (time to block 5 s) and rendered the tachycardia non-inducible. The lady was discharged in normal sinus rhythm on the following day.

Discussion

Antidromic atrioventricular re-entrant tachycardias due to accessory pathways are clinically documented in 5% of patients with the Wolff-Parkinson-White syndrome, but may be induced in 10% of patients during an EPS.[6] In the 1930s, Mahaim and co-workers described tracts connecting the AV node to the bundle branches or ventricular myocardium, bypassing the His bundle.[7] These pathways are characterised by decremental conduction that results in a gradual increase of the atrioventricular interval simultaneously with the development of left bundle branch block.[8] We know now that most of them actually connect the atrium to the fascicles by crossing the tricuspid annulus rather than the septum (atriofascicular pathways), although true nodoventricular Mahaim pathways also exist.[1,8,9] Mahaim conduction is usually antegrade only, but concealed fibres have also been described.[10] Their recognition is necessary in order to guide precise mapping. The differentiation of an antidromic macro-re-entrant circuit from AVNRT with the Mahaim pathway acting as a bystander fibre is of paramount importance for successful ablation. In the case of AVNRT, ablation of the slow pathway may render the tachycardia non-inducible despite the persistence of pre-excitation. In antidromic AVRT, however, the Mahaim fibre has to be ablated for abolition of the tachycardia. Atriofascicular Mahaim fibres are usually ablated during atrial pacing that results in maximum pre-excitation on the anterior tricuspid annulus. In this setting, the use of supportive long sheaths that stabilise the ablating catheter may be very helpful. For the ablation of nodoventricular fibres ablation lesions target the area between the slow pathway and the mid-septum. In antidromic tachycardias mapping of the retrograde atrial activation is of no use for ablation purposes.

References

1. Grogin HR, Lee RJ, Kwasman M, et al. Radiofrequency catheter ablation of atriofascicular and nodoventricular Mahaim tracts. Circulation. 1994; 90: 272-281.
2. Katritsis DG, Camm AJ. Classification and differential diagnosis of atrioventricular nodal re-entrant tachycardia. Europace. 2006; 8: 29-36.
3. Katritsis DG, Ellenbogen KA, Becker AE, Camm AJ. Retrograde slow pathway conduction in patients with atrioventricular nodal re-entrant tachycardia. Europace. 2007; 9: 458-465.
4. Katritsis DG, Becker A. The atrioventricular nodal reentrant tachycardia circuit: a proposal. Heart Rhythm. 2007; 4: 1354-1360.
5. Xie B, Heald SC, Bashir Y, et al. Localization of accessory pathways from the 12-lead electrocardiogram using a new algorithm. Am J Cardiol. 1994; 74: 161-165.
6. Packer DL, Gallagher JJ, Prystowsky EN. Physiological substrate for antidromic reciprocating tachycardia. Prerequisite characteristics of the accessory pathway and atrioventricular conduction system. Circulation. 1992; 85: 574-588.

7. Mahaim I, Benatt A. Nouvelles recherches sur les connexions supérieures de la branche gauche du faisceau de His-Tawara avec cloison interventriculaire. Cardiologia. 1938; 1: 61-76.
8. Mahaim I. Kent's fibers and the A-V paraspecific conduction through the upper connection of the bundle of His-Tawara. Am Heart J. 1947; 33: 651-653.
9. Kriatselis C, Gohl K, Jessl J, Gottwik M, Karabinos I. Mahaim fibre coexisting with anomalous origin of the left main coronary artery from the right aortic sinus and an interarterial course. Hellenic J Cardiol. 2007; 48: 302-305.
10. Hluchy J, Schlegelmilch P, Schickel S, Jörger U, Jurkovicova O, Sabin GV. Radiofrequency ablation of a concealed nodoventricular Mahaim fiber guided by a discrete potential. J Cardiovasc Electrophysiol. 1999; 10: 603-610.