CASE REPORT

Spontaneous automaticity of an atriofascicular accessory pathway

Santosh Kumar Dora1*, Jaganmohan A. Tharakan2, Ajithkumar Valaparambil2, Narayanan Namboodiri2, Krishnakumar Nair2, and Thomas Peter3

1 Division of Cardiology, Department of Medicine, Cedars-Sinai Medical Center, 8700 Beverly Boulevard, Room 5353, Los Angeles, CA 90048, USA; 2 Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala 695011, India; and 3 Cedars-Sinai Medical Center, Los Angeles, CA 90048, USA

Received 24 November 2004; accepted after revision 30 June 2005; online publish-ahead-of-print 5 January 2006

In a 12-year-old girl with history of recurrent palpitation, an ambulatory 24 h Holter electrocardiogram showed a wide QRS complex rhythm with atrioventricular dissociation. During an electrophysiology study, an atriofascicular pathway was diagnosed with an inducible antidromic atrioventricular re-entrant tachycardia. At slower heart rates, the patient had a wide QRS complex escape rhythm similar to the tachycardia and the pre-excited QRS complex morphology. This indicates the presence of pacemaker-like cells in the atriofascicular accessory pathway giving rise to the wide QRS complex escape rhythm at a slower heart rate.

KEYWORDS
Accessory pathway; Automaticity; Mahaim fibre; Atriofascicular pathway

Introduction

The atriofascicular accessory pathway is a rare conduit for accessory pathway mediated atrioventricular reciprocating tachycardia. It has decremental antegrade conduction properties like the atrioventricular (AV) node. Histologically, this type of accessory pathway has AV node like morphology and pacemaker cells have been identified in it.1 It has been seen that successful radiofrequency (RF) ablation of an atriofascicular accessory pathway is usually associated with accelerated automatic rhythm originating from the Mahaim fibres.2,3 This may indicate the presence of pacemaker cells in the atriofascicular accessory pathway. In this case report, we describe spontaneous automaticity of the atriofascicular accessory pathway, which again provides electrophysiological evidence of pacemaker cells in the atriofascicular pathway.

Case report

A 12-year-old girl with a history of recurrent episodes of palpitation for the last 3 years was referred for further evaluation. The episodes of palpitations were of sudden onset and occasionally associated with dizziness and blurring of vision. A 12 lead electrocardiogram taken during one such episode of palpitation showed a wide QRS complex tachycardia at a rate of 214 per minute with LBBB morphology and leftward axis deviation (axis +15°). An ambulatory 24 h Holter electrocardiogram performed elsewhere showed intermittent wide QRS complex rhythm with AV dissociation suggestive of slow ventricular tachycardia (Figure 1). The electrocardiogram at rest did not show any pre-excitation or characteristics of arrhythmogenic right ventricular dysplasia. There was no family history of sudden cardiac death or any cardiac disorder. An electrophysiological study was scheduled to determine the mechanism of the tachycardia.

Three quadripolar diagnostic catheters were positioned at right atrial appendage, His bundle region, and right ventricular apex, respectively, via right and left femoral veins. A wide QRS complex rhythm with isorhythmic atrioventricular dissociation was noted at rest (Figure 2, panel 1). The QRS complex morphology of this wide QRS rhythm due to automatic activity was similar to the ventricular pre-excitation following atrial stimulation (Figure 2, panel 2) and the antidromic tachycardia (Figure 2, panel 3). The occurrence of this rhythm was probably due to abnormal/spontaneous automaticity from the accessory pathway. Incremental atrial stimulation showed gradual shortening of the HV interval with the appearance of ventricular pre-excitation. The antegrade conduction was decremental in nature. The pre-excited QRS morphology indicated the presence of the accessory pathway to be at the right-free wall. Ventricular stimulation showed earliest atrial activity at the His bundle region due to retrograde conduction via the AV node (Figure 3, panel 2) and not via the right-free wall accessory pathway. The delivery of a late atrial extrastimulus during tachycardia when the AV node was refractory...
Figure 1  The Holter electrocardiogram shows periods of wide QRS rhythm with AV dissociation suggestive of slow ventricular tachycardia.

Figure 2  Wide QRS escape rhythm during electrophysiological study (panel 1, paper speed 25 mm/s). Intracardiac atrial electrogram inside the QRS complex indicating isorhythmic AV dissociation. The wide QRS complex morphology is similar to that during atrial pacing with pre-excited QRS (panel 2, paper speed 50 mm/s) and during antidromic AVRT (panel 3, paper speed 50 mm/s). HRAD, high right atrium distal; HBEP, His bundle electrogram distal; RVA, right ventricular apex.
Figure 3  Late atrial extrastimulus during the AV node refractory period advances ventricular activity indicating antegrade conduction via atriofascicular rather than a nodofascicular accessory pathway (panel 1, paper speed 50 mm/s). Ventricular pacing shows earliest atrial activity at the His bundle rather than at the right atrial-free wall indicating retrograde conduction via the AV node (panel 2, paper speed 100 mm/s). Abbreviations for intracardiac electrogram are the same as in Figure 2. HBEP, His bundle electrogram proximal.

Figure 4  A tiny high frequency Mahaim potential seen during sinus rhythm via the mapping catheter positioned at 9 o’clock on the tricuspid annulus (panel 1, paper speed 100 mm/s). Appearance of automatic Mahaim accelerated rhythm during radiofrequency ablation (panel 2, paper speed 25 mm/s). Abbreviations for the intracardiac electrogram are the same as in Figure 2. RFD, radiofrequency distal.
advanced ventricular activity without advancing atrial activity in the His bundle electrogram indicating atriofascicular rather than a nodofascicular connection of the accessory pathway (Figure 3, panel 1). All of the previous electrophysiological features suggested the presence of a solely antegrade conducting accessory pathway with decremental properties, situated at the right-free wall, giving rise to both automatic rhythm and antidromic supraventricular tachycardia. An SR 0 sheath was positioned in the right atrium via the right femoral vein, and a 7F RF catheter (BARD Stinger, D-curve) was passed via the sheath and the lateral tricuspid annulus was mapped. At 9 o’clock on the tricuspid annulus, a high frequency low amplitude discrete potential suggestive of a Mahaim potential was obtained (Figure 4, panel 1). RF ablation was performed at that site at a target temperature of 70°C for a period of 90 s. Early during RF ablation, there was a brief burst of accelerated wide QRS complex rhythm similar to that of the tachycardia (Figure 4, panel 2) followed by resumption of sinus rhythm. Subsequently, there was no further conduction via the accessory pathway and the tachycardia was not inducible by routine stimulation protocols.

Discussion

Spontaneous automatic activity originating from an atrioventricular accessory pathway is extremely rare. Although pacemaker-like cells have been identified in atriofascicular accessory pathways, which are a variant atrioventricular accessory pathway, automatic activity originating from this bundle is rare. However, accelerated automatic rhythm resembling the QRS morphology of the tachycardia is commonly seen during RF ablation of the atriofascicular accessory pathway. It may be due to heat-related stimulation of the pacemaker cells present in the atriofascicular accessory pathway. This property is similar to the accelerated junctional rhythm commonly seen during RF ablation of the slow pathway in atrioventricular nodal re-entrant tachycardia confirming AV nodal like properties of Mahaim fibres. Electrophysiologically, the existence of pacemaker cells in the atriofascicular accessory pathway is best proved by the escape rhythm. In this case report, the wide QRS complex rhythm was shown with AV dissociation at a time when the sinus rate was slower. The QRS morphology of this rhythm was similar to that of ventricular pre-excitation during atrial pacing and antidromic AVRT. Therefore, it was likely that this rhythm was originating in the Mahaim fibre. In addition, an accelerated rhythm with the same morphology was seen during RF ablation of the accessory pathway at a region where the Mahaim potential was obtained. This confirms that the presence of subsidiary pacemaker cells in the atriofascicular accessory pathway. Abnormal automaticity in the atriofascicular accessory pathway resulting in a wide QRS tachycardia also has been described. Sternick et al. have recently reported that spontaneous automaticity in the atriofascicular accessory pathway may be present in as many as 12.5% of cases. The rarity of spontaneous automaticity in the atriofascicular accessory pathway may be due to overdrive suppression of the subsidiary pacemaker cells by the sinus rhythm.

Conclusion

In addition to an accelerated automatic wide QRS complex rhythm commonly seen during RF ablation of the atriofascicular accessory pathway with the same morphology as during antidromic AVRT, a similar automatic wide QRS complex rhythm was seen at rest. The occurrence of the abnormal/spontaneous automaticity at rest from the accessory pathway indicates the electrophysiological evidence of pacemaker cells in the Mahaim fibre.

References