CASE REPORTS

Unmasking of left free wall ventricular preexcitation by His bundle ablation

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Abstract

His bundle ablation was performed in a 48 year old man with drug refractory orthodromic atrioventricular reentrant tachycardia and paroxysmal atrial fibrillation. Reentry was caused by a left free wall concealed accessory pathway. Interruption of the His bundle by low energy direct current shock (25 J) was quickly followed by anterograde conduction via the accessory pathway, with various forms of advanced block. The combined effect of unfavourable anterograde conduction (prolonged anterograde effective refractory period and decremental conduction), the left lateral location of the accessory pathway and intra-atrial conduction delay on one side, and normal conduction via the atrioventricular node and His bundle with retrograde concealed conduction into the accessory pathway on the other side resulted in masking of the anterograde conduction capability of the anomalous pathway. This observation does not seem to invalidate the choice of His bundle ablation in similar cases.

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The existence of an atrioventricular accessory pathway capable of only retrograde conduction was postulated in 1971¹ and subsequently confirmed by clinical and intracardiac electrophysiological studies.² Because of the absence of anterograde conduction over the accessory pathway ventricular preexcitation does not become manifest during sinus or other atrial rhythm and the existence of the accessory pathway remains concealed in the surface electrocardiogram. To date there have been reports of three cases of the permanent form of junctional reciprocating tachycardia in which ventricular preexcitation via a concealed posteroseptal accessory pathway became apparent after His bundle ablation.^{3,4}

We here report a case in which ventricular preexcitation via a left free wall accessory pathway was unmasked after direct current shock ablation of the His bundle for intractable orthodromic atrioventricular reen-

trant tachycardia and atrial fibrillation. The mechanisms that might have led to concealment of the accessory pathway and the potential clinical implications are discussed.

Case report

A 48 year old man was referred to our institute for catheter ablation of drug refractory orthodromic atrioventricular reentrant tachycardia and paroxysmal atrial flutter and atrial fibrillation. The patient had had symptoms for about eight years. Eight trials of antiarrhythmic drugs, alone or in combination, had failed or had had to be discontinued because of side effects. Physical examination and routine non-invasive laboratory tests were normal. The electrocardiogram at rest showed sinus rhythm with an intra-atrial conduction defect characterised by widened (120 ms) notched P waves, but no signs of ventricular preexcitation. The PR interval during sinus rhythm measured 160 ms with an AH interval of 75 ms and an HV interval of 55 ms in the His bundle electrogram. Several paroxysms of regular narrow QRS complex tachycardia and atrial flutter and fibrillation had been recorded. Holter recordings showed that atrial flutter and fibrillation were initiated by premature atrial complexes and not by deterioration of regular narrow QRS tachycardia. The diagnosis of an atrioventricular reentrant tachycardia through a left free wall accessory pathway was based on the following observations: (a) during tachycardia RP' was shorter than P'R (b) atrial activation was eccentric in a left caudal to cranial direction as shown by inverted P' waves in electrocardiographic leads I, II, and III⁵ and distal coronary sinus activation preceding proximal coronary sinus and low right atrial septal activation in the intracardiac electrograms, and (c) evidence on Holter recording of shortening of the tachycardia cycle length by 40 ms after the disappearance of left bundle branch aberrancy.²

Inability of the accessory pathway to conduct in an anterograde direction was postulated because there was no evidence of ventricular preexcitation in any of the available resting and ambulatory electrocardiograms recorded in the eight years of follow up

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or during paroxysms of atrial fibrillation with RR intervals ranging from 300 to 1000 ms. However, the effects of carotid sinus massage and isoprenaline (isoproterenol) on anterograde accessory pathway conduction had not been evaluated.⁶ During the pre-ablation electrophysiological study, including decremental atrial pacing and the atrial extrastimulus method, no preexcitation was observed.

A single low energy direct current shock (25 J) was administered through a special low energy ablation catheter (4 mm distal electrode) that was positioned in the proximal His bundle area according to the electrophysiological criteria and technique reported by Gallagher *et al.*⁷ Persistent complete interruption of the His bundle was achieved and this was shortly followed by anterograde conduction over the accessory pathway. The characteristics of the surface electrocardiogram were consistent with maximal preexcitation of the ventricles via a left lateral bypass (monophasic slurred positive QRS complexes in lead V1 with marked right axis deviation) (fig A). The P- δ interval of the conducted P waves measured 160 ms. At a P width of 120 ms,

this indicates delayed conduction over the accessory pathway. One to one anterograde conduction was later followed by 2:1 conduction over the accessory pathway depending on the duration of the RP interval. The shortest RP interval at which conduction was observed was 520 ms, corresponding to a sinus cycle length of 680 ms. During longer periods of 2:1 preexcitation the conduction pattern was further complicated by sudden failure of anterograde conduction. This was preceded by a 40 ms increment in the P- δ interval of the conducted P waves (fig B), indicating a superimposed type I block in the accessory pathway. The resultant pauses in the ventricular response were terminated by narrow His bundle escapes (R') and an escape rhythm resulting in atrioventricular dissociation (fig B and C).

Such complex patterns with apparent unexpected failure of preexcitation are best explained by assuming two levels of block in the accessory pathway: (a) a proximal level of block which is governed by a considerably prolonged effective refractory period, in which all P waves occurring at an RP interval

Figure Representative panels of electrocardiogram leads V1 and I recorded after His bundle ablation. (A) 1:1 ventricular preexcitation at an average cycle length of 700 ms. (B) Transition of 2:1 block into a more advanced degree of block in the accessory pathway, preceded by a 40 ms increment in the P-delta interval of alternately conducted P waves, indicating type I block. (C) Perpetuation of atrioventricular dissociation ascribed to a complex interplay between concealed anterograde conduction of alternate P waves superimposed on a basic 2:1 block in the accessory pathway, and concealed retrograde conduction of His bundle escapes. Probable atrial fusions (Fp) are diagnosed on the basis of their timing and the retrograde conduction characteristics of the accessory pathway as seen in panel D. (D) Complete anterograde block after ajmaline with preserved retrograde conduction over the accessory pathway (arrow). Numbers above the tracing: in (A) and (B) indicate PP intervals. In panel C and D these numbers represent P-R' intervals. A, atria; AV, His bundle area and accessory pathway. For clarity, in the ladder diagrams anterograde conduction has been indicated only for the accessory pathway because all P waves are assumed to block in the ablated His bundle. V, ventricles; P', premature atrial complex. Paper recording speed 25 mm/s.

of 520 ms or less are blocked. P waves occurring outside this interval may excite the ventricles or undergo decremental conduction and be blocked at a more distal level in the accessory pathway (attempted 2:1 preexcitation). Concealed anterograde conduction into the distal level of block will tend to maintain the basic 2:1 conduction pattern across the proximal region, thus allowing for a sufficiently long pause in the ventricular excitation pattern to enable a His bundle escape. (b) Once a His bundle escape occurs the conduction pattern is further complicated by concealed retrograde conduction into the accessory pathway. This may cause perpetuation of anterograde block and maintenance of atrioventricular dissociation (fig C).

Whereas anterograde conduction over the accessory pathway was unstable and could already be completely blocked by intravenous (5 mg) ajmaline,⁸ retrograde conduction of His bundle escapes over the anomalous connection were preserved and occurred whenever the PR' interval was 360 ms or more (fig D).

Because accessory pathway conduction remained unstable, a backup VVIR pacemaker was implanted. During a follow up of 24 months the patient remained symptom free.

Discussion

In this patient two criteria determined that His bundle ablation rather than accessory pathway ablation would be used. First, Holter recording showed that paroxysms of atrial flutter and atrial fibrillation were triggered by atrial premature complexes and did not result from deterioration of orthodromic circus movement tachycardia. Second, the accessory pathway was assumed to be incapable of anterograde conduction. Ablation of the accessory pathway would only cure the atrioventricular tachycardia, but would not protect the ventricles against rapid ventricular responses during atrial fibrillation with conduction over the normal pathway. In contrast, His bundle ablation was expected to achieve both goals by creating a complete atrioventricular block. Much to our surprise, interruption of the His bundle was followed by 1:1 preexcitation and various forms of advanced block in the accessory pathway.

This observation accords with reports by Przybylski *et al*⁶ and Critelli *et al*^{3,4} that concealment of an accessory pathway does not necessarily indicate that anterograde conduction is not possible. Several mechanisms have been proposed to explain the presence of unidirectional anterograde block in an anomalous atrioventricular connection.^{1,9-12}

In the present case, the absence of preexcitation before His bundle ablation can be explained by a combination of mechanisms. At relatively rapid sinus rates (cycle lengths <680 ms) the considerably prolonged anterograde effective refractory period of the anomalous bundle prevented preexcitation. At lower rates conduction over the accessory pathway was unfavourably influenced by the left lateral location of the anomalous bundle,

the observed intra-atrial conduction delay, and the prolonged anterograde conduction time (P-delta interval of 160 ms) with decremental characteristics. In contrast, conduction over the atrioventricular node-His bundle axis was unimpaired as shown by the relatively short AH interval and normal HV interval during sinus rhythm. The combined effect must have favoured conduction via the normal pathway with concealed retrograde activation of the anomalous bundle by the sinus impulse. These effects were abolished by His bundle ablation, leading to unmasking of the accessory pathway at relatively long diastolic intervals.

Stable anterograde conduction via a previously concealed accessory pathway may obviate the need for backup pacing after His bundle ablation.⁴ Since the conduction capability of concealed bundles is probably limited⁶ the risk of rapid ventricular rates with conduction over the anomalous pathway during atrial fibrillation is probably small or absent. Thus, as in the present case, ablation of the His bundle seems to be an acceptable treatment for drug refractory symptomatic atrial and atrioventricular reentrant arrhythmias, with backup pacing to cover for the unstable anterograde conduction characteristics of the accessory pathway. We chose VVIR pacing rather than DDD pacing because of the occurrence of paroxysmal atrial fibrillation, apparently unrelated to the atrioventricular reentrant tachycardias. In doing so, we accepted the potential disadvantage of VVI pacing with persistent retrograde atrial activation via the accessory pathway on haemodynamic function.

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