Latent pre-excitation: exposure of anterograde accessory pathway conduction during atrial fibrillation

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SUMMARY In three patients anterograde conduction across a previously undocumented accessory pathway was exposed by the development of atrial fibrillation. This unusual presentation illustrates the possible unmasking of the Wolff-Parkinson-White syndrome by other factors in patients who have not previously demonstrated the potential for anterograde accessory pathway conduction.

Atrial fibrillation is a known but uncommon feature of the Wolff-Parkinson-White syndrome. Classically patients who develop this arrhythmia have overt pre-excitation on the resting 12 lead electrocardiogram. In some individuals, however, conduction across the accessory pathway may not be seen during sinus rhythm. Under certain circumstances—for example with delayed atrio-ventricular nodal conduction or after premature atrial stimuli—anterograde accessory pathway conduction may become evident. We report three patients in whom pre-excitation was not visible on the resting electrocardiogram but when atrial fibrillation was seen anterograde accessory pathway conduction was exposed, with rapid ventricular rates.

Case reports

CASE 1

A 25 year old man who presented with a seven year history of palpitation had experienced several episodes of near syncope. The history and physical examination were otherwise normal.

Although the resting electrocardiogram was normal on most occasions, a single recording showed an atrial extrasystole with a pattern suggesting pre-excitation. There was a rapid ventricular

response (about 200/min) with broad QRS complexes consistent with anterograde conduction across an accessory pathway during paroxysmal atrial fibrillation (fig 1).

At electrophysiological study there was no pre-excitation in control recordings but incremental right atrial pacing at rates above 120/min and pacing from the coronary sinus at rates faster than that of the sinus node produced evidence of pre-excitation. The accessory pathway was localised to the left lat-

Fig 1 Twelve lead electrocardiogram and rhythm strip showing considerable pre-excitation during atrial fibrillation with no delta waves in sinus rhythm.

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eral wall of the heart. At operation, it was interrupted by endocardial dissection.¹ During follow up (six months) there has been no recurrence of arrhythmia.

CASE 2

A 47 year old woman with mitral stenosis and aortic regurgitation who had previously undergone closed mitral valvotomy presented with a two year history of recurrent palpitation. The resting electrocardiogram showed no evidence of pre-excitation but a tracing during paroxysmal atrial fibrillation showed a rapid ventricular response (about 280/min) with broad QRS complexes reflecting anterograde conduction across an accessory pathway (fig 2). At electrophysiological study a left lateral accessory pathway was demonstrated. At operation the accessory pathway was ablated with a cryoprobe²; at the same time the mitral valve was repaired and considerable left atrial enlargement was confirmed. Nine years later she remains symptom free with a normal resting electrocardiogram and no evidence of recurrent mitral valve disease.

CASE 3

A 23 year old woman had a nine year history of regular palpitation. Resting electrocardiograms were normal but pre-excitation became apparent during paroxysmal atrial fibrillation (ventricular rate 240/min). Electrophysiological study showed enhanced atrioventricular nodal conduction with a short AH interval (55 ms) which did not increase significantly during rapid atrial pacing³ though pre-excitation then became obvious. At operation a left lateral accessory pathway was ablated by endocardial dissection.¹

Discussion

The Wolff-Parkinson-White syndrome is characteristically associated with reentrant atrioventricular

tachycardia. Atrial fibrillation and flutter occur less frequently—in 10–32% of cases.⁴ The potential importance of atrial arrhythmias depends on the rapidity of anterograde conduction of atrial impulses across the accessory pathway to the ventricles, and the possibility that this might precipitate ventricular fibrillation.⁵ In the Wolff-Parkinson-White syndrome anterograde conduction via the accessory pathway is usually evident in sinus rhythm because there is simultaneous conduction down the atrioventricular node, producing the classic fusion complex. Even in the absence of overt pre-excitation an accessory pathway may only be capable of retrograde conduction—and thus of maintaining reentry atrioventricular tachycardia (so-called concealed pre-excitation).⁶

Our patients failed to display resting pre-excitation despite the presence of pathways capable of rapid anterograde conduction. Factors that may be relevant to this were the left lateral site of the accessory pathway in all three patients, the increased left atrial size in the second patient, and accelerated atrioventricular nodal conduction in case 3. Thus in all three patients the distance between the sinus node and the area of atrial input into the accessory pathway may have been sufficient to favour conduction via the atrioventricular node. The relative conduction time between the sinus node and the accessory pathway was even greater in case 2 because of left atrial enlargement and in case 3 accelerated conduction via the atrioventricular node probably made the use of the accessory pathway even less likely.

Owing to several spatial and electrical factors a normal electrocardiogram may be seen even though there is an accessory pathway capable of anterograde conduction. In principle, if the time taken for propagation of a sinus impulse to and across the accessory pathway is greater than the time taken for that impulse to propagate to the ventricles via the atrioventricular node pre-excitation may not be seen. Such conditions may occur with a left lateral pathway,⁷ delay in intra-atrial conduction time,⁸ or increased left atrial size. Alteration in pacemaker site to an area further from the accessory pathway⁹ or slow conduction across the accessory pathway¹⁰ may also be important. Evidence that contrary autonomic influence on accessory pathway and atrioventricular nodal conduction may also have a role comes from the finding that carotid sinus pressure can enhance and atropine diminish the degree of pre-excitation.¹¹ Similarly, if enhanced atrioventricular conduction or an atrionodal bypass tract is also present,¹² the sinus impulse arriving at the accessory pathway may find the ventricles already depolarised, or the accessory pathway may have been rendered refractory by concealed retrograde conduction into it from the atrioventricular node.¹³ Conversely, the slowing¹¹ or the removal^{8 14} of atrioventricular nodal conduction may expose anterograde conduction across an

Fig 2 Twelve lead electrocardiogram and rhythm strip showing pre-excited ventricular complexes during atrial fibrillation. There are no delta waves on the resting electrocardiogram but P mitrale is present.

accessory pathway.

The onset of atrial fibrillation may disturb this balance of relative anterograde conduction, the homogeneous origin of atrial impulses cancelling out intra-atrial differences in conduction time. Yahini and his colleagues¹⁵ reported on one patient with paroxysmal atrial fibrillation but no pre-excitation in sinus rhythm, who was otherwise indistinguishable from five others with atrial fibrillation and the Wolff-Parkinson-White syndrome. In other cases⁸ ¹⁰ coexistent mitral valve disease was present or the distance between the sinus node and the accessory pathway was thought to be responsible for lack of resting pre-excitation in sinus rhythm.¹⁶

Where the QRS complex is normal in sinus rhythm but there is documented supraventricular tachycardia and suspicion of an accessory pathway or when anterograde conduction is abolished by ajmaline or procainamide,¹⁷ the patient is conventionally regarded as being at low risk of rapid conduction across the accessory pathway in atrial fibrillation. Drug-induced abolition of resting pre-excitation has been thought to preclude the development of dangerously high ventricular rates in response to atrial fibrillation.¹⁷ Such tests have limitations,¹⁶ however, and in one patient pre-excitation was abolished by ajmaline but at electrophysiological study the ventricular rate during atrial fibrillation was more than 300/min. Reflex changes associated with the flushing and dizziness after intravenous ajmaline and procainamide may increase anterograde atrioventricular nodal conduction and mask, rather than block, anterograde conduction in the accessory pathway.

The term "latent pre-excitation" has been used previously to describe the absence of expression of pre-excitation, whatever the cause, in the presence of an accessory pathway.⁶ It might be useful to restrict it to cases in which anterograde conduction is usually masked but which may become manifest under certain circumstances, as opposed to those in which anterograde transmission across an accessory pathway is impossible at all times (concealed), owing to the presence of permanent unidirectional block or an extremely long anterograde refractory period.

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