Wolff-Parkinson-White syndrome: atrial fibrillation as the presenting arrhythmia

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SUMMARY Atrial fibrillation was identified as the initial arrhythmia complicating the Wolff-Parkinson-White syndrome in ten (9%) of 108 patients. Despite initially rapid ventricular responses in seven, long term survival and control of arrhythmia were excellent on medical treatment. Whereas symptom free patients with the pre-excitation syndrome who have additional underlying disease predisposing to atrial fibrillation may need detailed electrophysiological study, a more conservative approach is suitable for the typical symptom free individual.

Atrial fibrillation is a potentially serious complication of the Wolff-Parkinson-White syndrome and may degenerate into ventricular fibrillation.^{1,2} When spontaneous atrial fibrillation occurs reentrant atrioventricular tachycardia has usually already been confirmed or there is a history of palpitation.^{3,4}

We studied ten patients in whom atrial fibrillation was the first arrhythmia to develop. We report their clinical presentation, electrophysiological characteristics, and long term follow up.

Patients and methods

Between 1976 and 1984 electrophysiological investigation of 108 patients with the Wolff-Parkinson-White syndrome was undertaken. Ten patients were identified in whom the first episode of palpitation was shown to have been atrial fibrillation or flutter. In each case it was established that there had been no history of palpitation, dizziness, syncope, or other symptoms suggesting arrhythmia. No patient was receiving antiarrhythmic medication. The mode of clinical presentation, the appearance of the presenting electrocardiogram, age, sex, symptoms, and the presence or absence of organic heart disease were analysed. All patients underwent invasive electrophysiological study according to our protocol.⁵

A further 17 patients referred for investigation had atrial fibrillation as the presenting arrhythmia but in

these there was a history suggesting previous arrhythmia.

Results

There were six male and four female patients (mean age 40 (range 16-61) years). A bimodal distribution was apparent—five were <33 and the remainder were >47 years (table 1).

CLINICAL FEATURES

When atrial fibrillation developed all had palpitation, three had near-syncope, and three had syncope. Three patients had a previous history of chronic angina pectoris and one had diabetes, hypertension, and a long history of alcoholism.

ELECTROCARDIOGRAPHIC FINDINGS

Electrocardiographic tracings recorded at the time of presentation were available in all but one patient and showed variable patterns of conduction over the accessory pathway. The range of mean ventricular responses during atrial fibrillation at initial presentation was 125-310 beats/minute. In the five patients aged 49-61 years the range of mean ventricular responses was 125-240 beats/minutes, whereas in those aged ≤33 the range was 270-310 beats/minute. The patient in whom the initial electrocardiogram was not available (patient 10) was semiconscious at admission to hospital and required immediate direct current cardioversion for an extremely rapid and irregular ventricular rhythm.

ELECTROPHYSIOLOGICAL FINDINGS (TABLE 2)

In six patients the accessory pathway was found to be on the left free wall and in three it was posteroseptal;

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Table 1 Summary of clinical findings in ten patients in whom atrial fibrillation was the first arrhythmia

Patient No	Age at presentation (y)	Sex	Symptoms	Other cardiac disease	Follow up (yr)
1	58	M	Palpitation	Coronary heart disease	9
2	49	M	Palpitation	Coronary heart disease	6
3	33	M	Palpitation + presyncope	None	3
4	24	F	Palpitation	None	9
5	61	F	Palpitation + presyncope	Coronary heart disease	7
6	56	F	Palpitation + presyncope	None	11
7	24	M	Palpitation + syncope	None	10
8	19	M	Palpitation	None	9
9	59	M	Palpitation + syncope	Coronary heart disease, diabetes mellitus, hypertension	5
10	16	F	Palpitation + syncope	None	7

Table 2 Electrophysiological findings in all patients

Patient No	Site of accessory pathway	AP ERP (ms)	Mean AF rate (beats/min)		SRR (ms)	
			At EP study	Clinically	At EP study	Clinically
1	Left free wall	230	230	200	200	210
2	Left free wall	> SCL	140	125	520	300
3	Posteroseptal	230	245	270	210	160
4	Posteroseptal	260	173	270	220	180
5	Left free wall	280	200	180	240	230
6	Anteroseptal	290	240	260	200	180
7	Posteroseptal	230	300	305	170	180
8	Left free wall	230	300	310	200	180
9	Left free wall	290	200	240	290	190
10	Left free wall	< ARP	300	—	200	—

AP ERP, accessory pathway effective refractory period; AF, atrial fibrillation; EP, electrophysiological; SRR, shortest RR interval; ARP, atrial refractory period; SCL, sinus cycle length.

it was anteroseptal in the other patient. The anterograde effective refractory period of the accessory pathway ranged from 230 to 290 ms in eight patients; in two it could not be measured owing to atrial refractoriness in one (patient 10) and because it was longer than the spontaneous sinus cycle length in the other (patient 2). All patients had inducible sustained orthodromic reentrant atrioventricular tachycardia. In two this degenerated spontaneously into atrial fibrillation while in the remainder the arrhythmia was induced by rapid atrial stimulation. Atrial fibrillation was sustained during electrophysiological study until sinus rhythm was restored, either pharmacologically or electrically in nine. Reversion to sinus rhythm was spontaneous in the tenth. The range of mean ventricular rates during atrial fibrillation was 140–300 beats/minutes with shortest RR intervals ranging from 170 ms to 520 ms.

FOLLOW UP

Patients were followed for 3–11 (median eight) years. Two have died. One (patient 2), who was treated with verapamil and quinidine, had remained free of arrhythmia until he died from carcinoma of the lung five years later. Patient 9 remained free of arrhythmia for four years and died of acute myocardial infarction that was confirmed at necropsy. Patient 8 declined treatment and continues to have reentrant atrioven-

tricular tachycardia but not atrial fibrillation. Patient 7 takes medication erratically and has palpitation. The arrhythmia responsible has not been identified. Patient 5 is well on verapamil and quinidine; patient 4 requires varying regimens and, though he still has paroxysmal reentrant atrioventricular tachycardia, there has been no more atrial fibrillation. Three patients (cases 1, 6, and 10) are on amiodarone and have no symptoms; patient 3, also on amiodarone, has episodes of paroxysmal atrial fibrillation but with slower ventricular rates than he had originally (for example 120 v 270 beats/minute).

Discussion

Reentrant atrioventricular tachycardia is the most common arrhythmia in the Wolff-Parkinson-White syndrome.⁶ Although atrial flutter is rare, atrial fibrillation is seen in 10–32% of patients⁴ and a rapid ventricular response to this may cause not only serious haemodynamic deterioration but also ventricular fibrillation.^{1,2}

Atrial fibrillation may develop for a number of reasons but the coexistence of other forms of heart disease makes it more likely.³ Sick sinus syndrome,⁷ cardiomyopathy, rheumatic heart disease,⁸ and coronary heart disease can all be contributing factors but they are not necessarily prerequisites for the

appearance of arrhythmia, as can be seen from our patients. Atrial or ventricular extrasystoles, reentrant atrioventricular tachycardia,^{3,4} and atrial or ventricular pacing, especially in the setting of intra-atrial conduction disturbance,⁴ can all precipitate atrial fibrillation.

In our study, atrial fibrillation was the initial arrhythmia in 9% of patients. In other patients with the Wolff-Parkinson-White syndrome a history of palpitation may be present and the same symptoms may be reproduced by the induction of atrial fibrillation at electrophysiological study. It may, therefore, be the first arrhythmia in a higher proportion of patients with pre-excitation but not be identified as such.

In older patients, for reasons that are unclear, the ventricular response was slower. The higher incidence of coexisting disease may, however, have provided the mechanism for initiation of the arrhythmia.

In younger patients, extremely rapid ventricular rates were seen and one required direct current cardioversion. In a retrospective study Klein *et al* showed that rapid ventricular responses to atrial fibrillation were associated with ventricular fibrillation.² The degree to which induced atrial fibrillation is predictive of ventricular fibrillation has not, however, been prospectively evaluated. A rapid ventricular response to atrial fibrillation may be a highly sensitive marker for the development of ventricular fibrillation but it may be non-specific and other factors are known to have a role including digitalis¹ or other drugs.⁹ Despite this, our median follow up over eight years suggests that such patients have a good medium term survival when they are medically managed. Furthermore, treatment was successful in preventing atrial fibrillation and in controlling the ventricular response if it recurred. Surgical ablation was advocated in only one patient but he declined it. He now has fewer and milder attacks. The other patients have opted for medical management despite the satisfactory long term results of operation.¹⁰

These findings counterbalance those of Milstein *et al* who found that 17% of symptom free patients with the Wolff-Parkinson-White pattern had "potentially lethal ventricular rates during induced atrial fibrillation",¹¹ a figure which is clearly in excess of sudden death rates in this syndrome.

The role of electrophysiological study in the symptom free patient with resting pre-excitation remains unclear.¹² The risk of rapidly conducted atrial fibrillation is small and although a previous history of reentrant tachycardia has been thought to be an important risk factor for the development of atrial fibrillation our results indicate that such a history may not always be present.

It has been suggested that electrophysiological study may be important even in symptom free individuals¹¹ but identification of those at high risk of development of atrial fibrillation, such as those with mitral valve disease or Ebstein's anomaly, may allow more appropriate selection of patients in such studies. The presence of rapid ventricular rates during induced atrial fibrillation in many patients who are usually symptom free¹¹ emphasises the necessity of interpreting these values in the context of other predictors of high risk. A prospective study of pooled electrophysiological and epidemiological data from several centres may help to elucidate the natural history of atrial fibrillation in the Wolff-Parkinson-White syndrome and its relation to sudden death.

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