ATRIAL FIBRILLATION IN THE WOLFF-PARKINSON-WHITE SYNDROME

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SYNOPSIS

Atrial fibrillation with aberrant ventricular conduction occurring in normal healthy patients with the Wolff-Parkinson-White Syndrome (WPW) is uncommon. This paper describes such a case. The widened QRS complexes during the attack simulates closely ventricular tachycardia with fusion or capture beats. Although 16 cases of ventricular tachycardia associated with WPW syndrome had been reported in the literature by 1966 (Newman, Donoso and Friedberg, 1966), none had shown unequivocal evidence that a correct diagnosis was made (Chung, Walsh and Massie, 1965). It is believed that most of the cases of so called ventricular tachycardias documented so far were actually supra-ventricular tachy-arrhythmias with aberrant conduction (Schamroth, 1971) as is seen in our patient described.

INTRODUCTION

Atrial fibrillation with an aberrant ventricular response is not common in the Wolff-Parkinson-White (WPW) syndrome. Moreover this arrhythmia presents difficulties in diagnosis and is very frequently mistaken for ventricular tachycardia (Hunt and Buckner, 1969).

CASE REPORT

A 43 year old Chinese man was first seen in November 1971 for a sudden attack of palpitations associated with chest pain. Clinical examination showed no abnormalities except for an irregular heart rate of about 280 per minute. The blood pressure was 120/70. The electrocardiogram (ECG) showed an irregular rhythm with no clearly discernable P waves; most of the QRS complexes were widened (Fig. 1). The arrhythmia was thought to be more likely rapid atrial fibrillation with aberrant ventricular conduction rather than ventricular tachycardia.

An intravenous injection of 100 mgms. Antazolin ('Antistin') was given with no effect. He was then given 0·25 mgms. of Digoxin by intramuscular injection followed by 0·25 mgms. of oral Digoxin eight hourly. Continuous electrocardiographic monitoring showed that the patient reverted to sinus rhythm 22 hours after admission to hospital. An ECG done at this stage (Fig. 1) showed classical Wolff-Parkinson-White Syndrome (Type B). A review of the ECG during the attack of tachycardia confirmed that the initial arrhythmia was atrial fibrillation. The QRS complexes during this period showed variation in size and shape but in general resembled the ventricular complexes seen in the ECG which showed the Wolff-Parkinson-White syndrome after reversion of the tachycardia. This variation is due to differences in the degree of fusion. Beats which were conducted primarily through the normal atrioventricular node (e.g. sixth ventricular complex in AVR and sixth ventricular complex in V_6) resembled a 'pure' normally conducted QRS complex. Beats which were identical to the QRS complexes seen after the attack were essentially fusion beats, while those which were bizzare and grossly abnormal (e.g. sixth, ninth and fourteenth beat in LdI) were due to conduction predominantly via the anomalous pathway.

Since the attack of tachycardia, the patient has been maintained on oral Procaineamide. He has been followed up for one year and has had no further episodes of palpitations.

DISCUSSION

In a review of the various types of arrhythmias associated with the Wolff-Parkinson-White (WPW) syndrome, Newman, Donoso and Friedberg (1966) found that 70% of patients had paroxysmal atrial tachycardia, whilst another 10% had unspecified supraventricular tachycardia. Atrial fibrillation was seen in about 16% of patients, but atrial flutter was uncommonly present and accounted for only 4% of all the arrhythmias. Chung, Walsh and Massie

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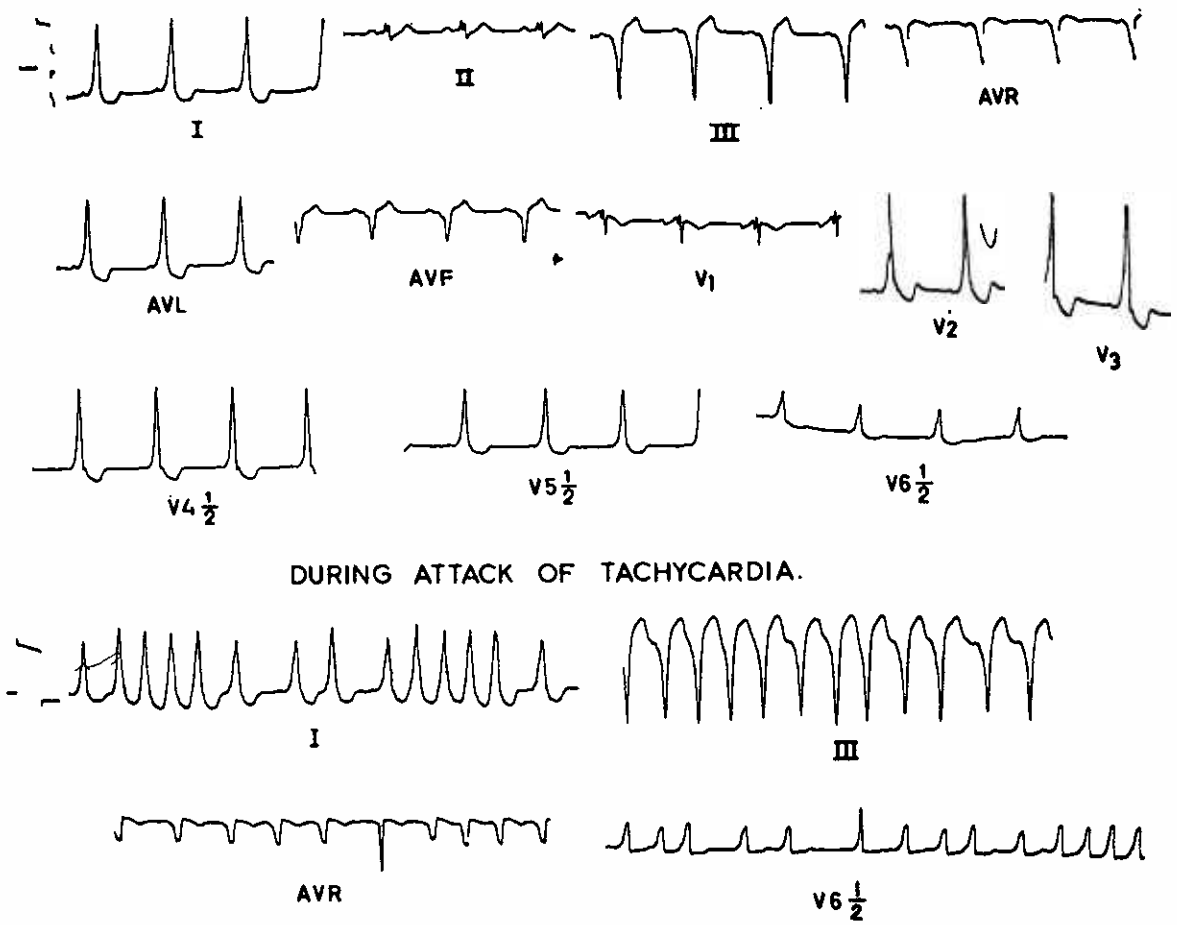

Fig. 1. E.C.G., during the attack of tachycardia (bottom half) and when patient reverted to sinus rhythm (top half) — see text.

(1965) stated that atrial fibrillation often presents in patients with rheumatic heart disease. In this situation, the arrhythmias may be due to the cardiac disease itself. However, atrial fibrillation may also be present in patients with normal hearts as is seen in our particular patient.

The QRS complexes during atrial or junctional tachycardia in the WPW syndrome are normal in the great majority of cases. This is because in atrial or junctional tachycardia in the WPW syndrome, the mechanism of the arrhythmia is that of a reciprocating tachycardia. The supraventricular impulse usually travels down the normal A-V nodal pathway to the ventricles, and back to the atria in a retrograde fashion via the anomalous pathway, thus constituting a circus movement. However, during attacks of atrial fibrillation or flutter, the QRS complexes are often bizzare and widened; this is due to conduction through predominantly the anomalous pathway or to fusion beats which resemble closely the WPW complexes when the patient is in sinus rhythm. Occasionally, normal QRS complexes are also seen when the impulses are conducted predominantly via the normal A-V conducting system. All these three different types of ventricular complexes were present in our patient during the attack of atrial fibrillation. (Chia, 1973).

Since most of the cases of atrial fibrillation and atrial flutter with the WPW syndrome present with widened QRS complexes, difficulties often arise in distinguishing these arrhythmias from ventricular tachycardia. Grossly bizzare looking QRS complexes may be misdiagnosed as runs of ventricular tachycardia and normal and intermediate looking QRS complexes may be misinterpreted as being capture and fusion beats respectively. However, very rapid and irregular ventricular rates of more than 180 per minute and an absence of P waves should always suggest atrial fibrillation due to the WPW syndrome, rather than ventricular tachycardia (Schamroth, 1971).

In a review of the literature, Newman, Donoso and Friedberg (1966) found 16 reported cases of ventricular tachycardia associated with the WPW syndrome. However in 13 of these cases, the ventricular rhythm was completely irregular, and independent atrial complexes, fusion or capture beats were not seen. Chung, Walsh and Massie (1965) were unable to find a single unequivocal case of ventricular tachycardia in the literature, although several such cases have been reported in the past. It is believed that most if not all cases of ventricular tachycardia previously documented were due to a supraventricular arrhythmia with aberrant conduction as seen in our patient.

According to Chung, Walsh and Massie (1965), atrial tachycardia occurs with equal frequency in type A and type B WPW syndrome. However in their series, atrial fibrillation and atrial flutter, were found only in the type A variety. However, at least three of the 5 cases of atrial fibrillation reported by Yahini, Zahavi and Neufeld (1964), the case reported by Hawker (1971) and our present patient all showed WPW syndrome type B. Thus Chung's concept that atrial fibrillation is confined to type A WPW syndrome may not be correct.

Digitalis has not been regarded as the drug of choice in the treatment of tachycardia associated with the WPW syndrome. Most authors prefer using either Procaineamide or Quinidine as the first line of treatment. This is because Digitalis enhances the conduction of the anomalous pathway (Wolff, 1960) whilst depressing that in the normal AV conducting system. However, Digitalis is often effective in abolishing an attack of atrial tachycardia, which is due to a reciprocal mechanism in the WPW syndrome, because of its depressant action on the AV node, thus breaking the circus movement (Schamroth, 1971). In contrast, atrial fibrillation or flutter in the WPW syndrome are not due to a reciprocal mechanism and according to Schamroth (1971) and Newman, Donoso and Friedberg (1966), digitalis is contraindicated. These authors believe that a depression of the normal A-V nodal tissue will only increase conduction through the anomalous pathway which has an immense capacity for rapid conduction giving rise to very fast ventricular rates which may prove disastrous for the patient. Either Procainaemide or Quinidine are the drugs of choice in these two situations. Since attacks of atrial fibrillation in the WPW syndrome are often transitory (Schamroth, 1971), it is likely that the reversion of the arrhythmia to sinus rhythm in our patient 22 hours after admission was spontaneous rather than as a result of the Antazolin or Digoxin which were administered.

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