Wolff - Parkinson-White syndrome presenting as atrial fibrillation with broad-QRS complexes

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Abstract

The Wolff-Parkinson-White (WPW) syndrome is the commonest form of ventricular pre-excitation and is characterised by the presence of an accessory pathway between atria and ventricles. The term WPW syndrome is applied to patients with both pre-excitation on the ECG and paroxysmal tachycardia. Usually the conducting properties of bypass tracts and the AV node differ, the ventricular response during atrial flutter or fibrillation may be unusually rapid and may cause ventricular fibrillation. Atrial fibrillation (AF) is not an uncommon presentation in emergency department. Moreover, AF associated with WPW syndrome as an underlying condition is also not a rare occurrence; it is seen in 20-25% of WPW Syndrome. Recognition of this condition is very crucial in terms of emergency management. Its early recognition and initial treatment allows rapid restoration to sinus rhythm. Acute management of WPW syndrome with atrial fibrillation with hypotension is DC cardioversion. In haemodynamically stable patients, the drugs of choice are Amiodarone and class Ic anti- arrhythmic agents.

Key words: Paroxysmal tachycardias, pre-excitation, tachycardia.

Introduction

Conduction from the atria to the ventricles normally occurs via the atrio-ventricular node (AV)-His-Purkinje system. Patients with a pre-excitation Syndrome have an additional or alternative pathway, known as an accessory pathway, which directly connects the atria and ventricle and bypasses the AV node. AV conduction through an accessory pathway results in the earlier activation of the ventricles than if the impulse had traveled through the AV node; hence the term preexcitation. Wolff-Parkinson-White syndrome (WPW) is the commonest form of ventricular pre-excitation. It is characterised by the presence WPW pattern of electrocardiographic (ECG) changes and paroxysmal tachycardia. WPW pattern of ECG results due to the presence of an accessory pathway between the atria and the ventricles, which provides an additional route for ventricular activation. This tract avoids the atrio-ventricular node (AVN) hence the term bypass tract, permitting premature ventricular activation. Since the bypass tract does not have the same decremental conducting properties as the AV node, the ventricular response during atrial flutter or

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fibrillation may be unusually rapid and may cause ventricular fibrillation.[1] WPW pattern of ECG features were first described by Wilson in 1915 and then by Wedd in 1921.[2] However, its association with tachyarrhythmia was first recognized by Wolff, Parkinson, and White in 1930 and they have described it as a separate clinical entity. Some patients may remain asymptomatic throughout their lives, others are prone to tachyarrhythmias that may be life threatening. It is seen in around 2.4% of patients presenting with a regular narrow complex tachycardia to the emergency department[3] where it can be successfully treated with the restoration of sinus rhythm. Atrial fibrillation associated with WPW is less common. We, herein report a case of wide-QRS complex tachycardia with haemodynamic stability, which was subsequently diagnosed as WPW syndrome with atrial fibrillation (AF) with antidromic conduction over the bypass tract. In our set-up, when we asked physician doctors and general practitioner in emergency department about the number of cases of WPW syndrome with atrial fibrillation seen by them during their practice revealed answer "NONE" by almost everybody. This interesting clinical presentation which can be disastrous if mismanaged forms the basis of this communication.

Case report

A 35-year housewife presented to emergency with history of palpitation and multiple episodes of vomiting. The patient had reportedly been well until a day earlier, when she began to have sudden onset palpitation during rest. There was no orthopnea, sweating, nausea or vomiting. On examination she was apprehensive. Her pulse was 90 beats per minute, irregularly irregular, and heart rate 278 beats per minute irregularly irregular. Her BP was 100/70 mmHg. Her respiratory rate and jugular venous pulsation were normal. Cardiovascular examination did not reveal any murmur or added sounds. There were no inspiratory crackles at both lung bases. Laboratory tests were performed. The level of creatine kinase and creatine kinase MB were normal, as was the troponin. The level of urea, creatinine, glucose, calcium, electrolytes were normal. Magnesium level couldn't be done. Chest X-ray was also normal. Electrocardiogram showed AF with aberrant conduction (with δ wave) with ventricular rate of 250-278 beats per minute (Figure 1).

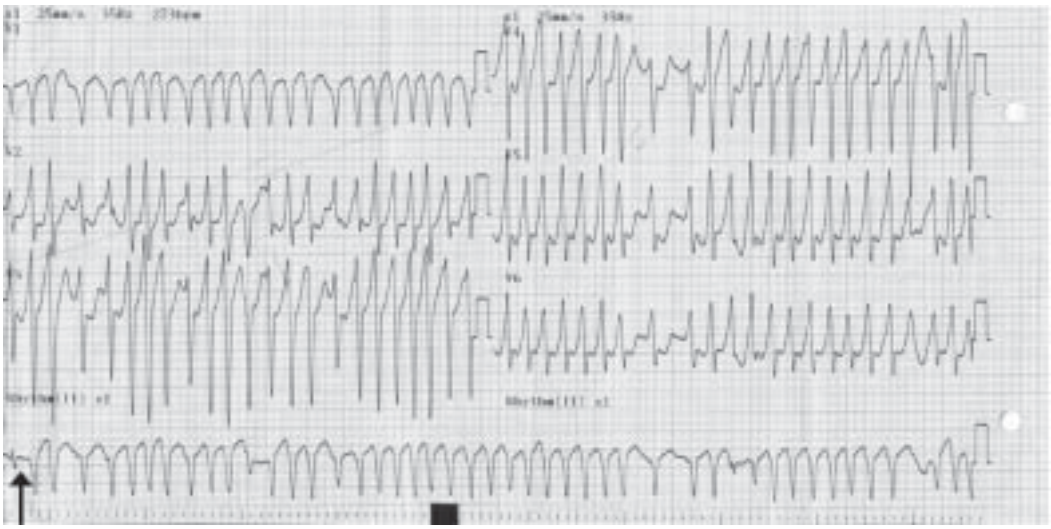

Figure 1: AF in WPW. Note irregularly irregular rhythm with delta wave visible in QRS. Note fast ventricular response and a capture beat in the rhythm-strip (pointed by an arrow).

As she was haemodynamically stable, she was loaded with IV Amiodarone (150 mg over 10 minutes), then 1mg/min for 6 hours and admitted in CCU. Despite the treatment, tachycardia with heart rate of 250-280 beats/min was persisted. She was then given injection Lidocaine 3mg/kg over 20 minutes. After 2-3 min, she went into asystole. She was intubated and resuscitated. Her cardiac activity reverted and showed junctional rhythm with heart rate of 50-60 per minute (Figure 2). Even after the heart rate slowed down, cardiac auscultation did not reveal any added information. She continued to have a junctional rhythm for one day. The next day ECG showed the feature consistent with WPW syndrome (Figure 3). She was weaned off the ventilator and extubated. Echocardiography done after stabilizing the patient was normal.

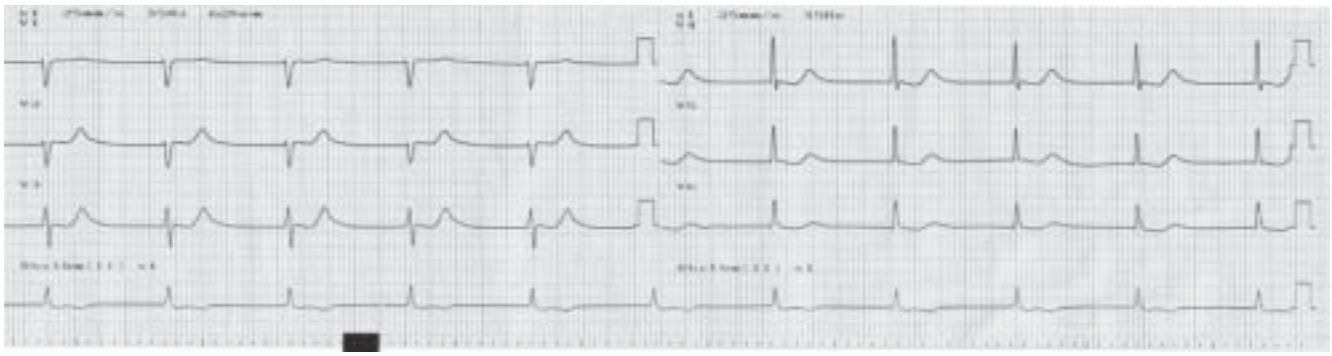

Figure 2: Junctional rhythm of the same patient after lidocaine. Note the normal QRS complexes and the absence of p-wave.

The case was diagnosed as a case of WPW syndrome with atrial fibrillation with fast ventricular rate. The patient was put on oral amiodarone 400 mg twice a day and discharged with the advice for further follow-up for electrophysiological studies and radiofrequency ablation.

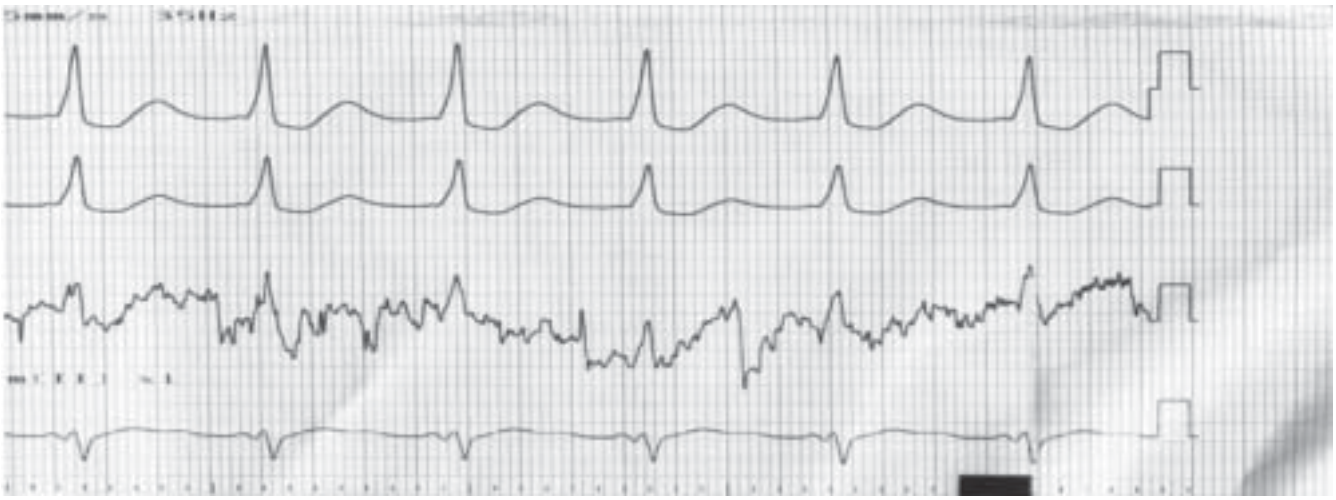

Figure 3: Pre-excitation with shortened PR interval and "delta wave"—that is, slurred upstroke of QRS complex, most visible in rhythm strip.

Discussion

WPW syndrome is characterised by the presence tachyarrhythmia and WPW pattern of ECG. The ECG changes are due to an accessory pathway, usually a bundle of Kent. The classic ECG pattern of WPW syndrome consists of [4] a short PR interval, a slurred, thickened initial upstroke of the QRS complex, which is termed as delta wave, and a slight widening of the QRS deflexion with increased ventricular activation time. In addition to these features there may also be the presence of the secondary ST segment and T wave changes and increased duration of ventricular activation.

WPW syndrome is defined as a clinical entity with symptomatic tachyarrhythmia. It includes a distinct pattern of ECG characteristics which includes: [5]

- Shortened P-R interval of less than 0.12 s
- Slurred slow rising onset to QRS known as the delta wave and
- A prolonged QRS complex more than 0.11 s

The accessory pathway is congenital in origin and results from a failure of complete separation of the atria and ventricles. It consists of a thin filamentous structure situated anywhere along the atrioventricular groove and the left lateral pathway is the most common.[6] In around 10% of cases multiple pathways exist.[7] The incidence of associated congenital abnormalities ranges from 7% to 20%.[5, 8] Tricuspid valve lesions are the most common. The prevalence of WPW syndrome in the population is about 0.3% [5] with an associated risk of sudden death of around 0.5% to 4%. [9]

For reasons that are not clear atrial fibrillation is relatively common (20%) in WPW syndrome compared with the normal population. In atrial fibrillation with pre-excitation activation of the ventricles is predominantly via the accessory pathway (Figure 4D). This causes the expected irregular rhythm but the QRS is widened with a bizarre morphology. Occasional activation of the ventricles occurs via the AVN resulting in a capture beat with a normal QRS (Figure 1). If the accessory pathway is capable of conducting rapidly, potentially life-threatening arrhythmias may result. The ventricular response may be in the region of 180–220 beats/minute.

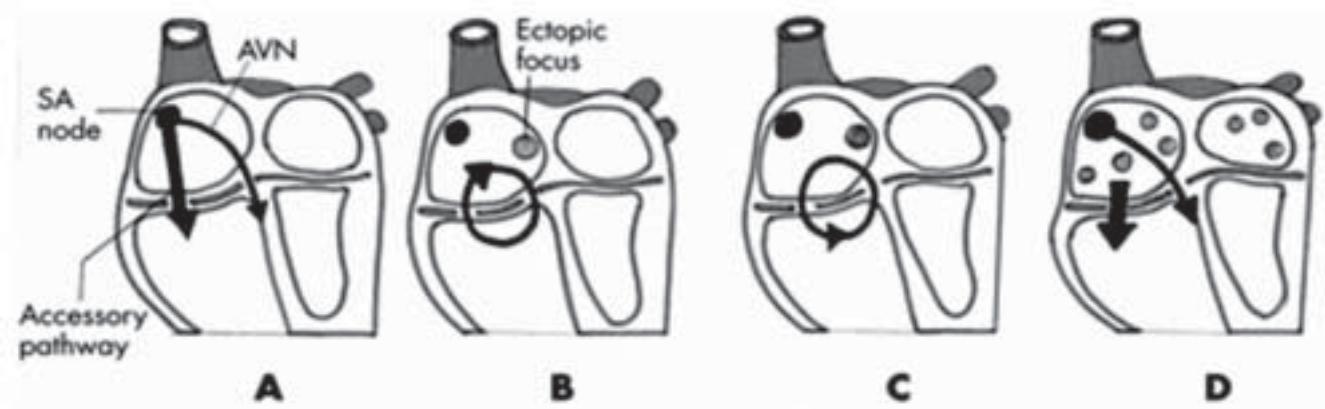

Figure 4: Series of four stylized drawings of the heart. (A) A physiological mechanism of conduction in WPW with accessory pathway conduction (atrioventricular) resulting in the delta wave. (B) Orthodromic conduction in WPW syndrome occurring down AVN and retrogradely up accessory pathway. (C) Antidromic conduction where accessory pathway conducts anterogradely. (D) In AF with pre-excitation activation of the ventricles is predominantly via the accessory pathway.

Rosenbaum and associates first attempted the localisation of bypass tract, separating them into type A – a left bypass tract and type B– a right bypass tract. This is relatively crude and no longer accepted. The bypass tract of the WPW syndrome may be situated anywhere along the AV ring. Ten such locations have been described. More than 90% of the bypass tracts, however, occur at four main sites (Table I). It is evident that the left lateral pathways are the most common (45%), but left anteroseptal pathway is least common (9%). These sites can be fairly accurately localised from the surface ECG by analysis of delta wave and main QRS deflexions (Table II). [4]

Table I: Site of bypass tract and Incidence.[4]

Right lateral	18%
Left lateral	45%
Posteroseptal (right and left)	26%
Anteroseptal (right and left)	09%

Table II: Localisation of the bypass tract from analysis of delta wave axis and polarity of QRS complexes on ECG.[4]

Pathway site	Polarity of main QRS complex			QRS axis	Delta axis
	Lead V1	Lead V2	Lead V3		
Anteroseptal	Negative	Negative	Negative	Normal	Normal
Right lateral	Negative	Negative	Negative	Left	Left
Right posteroseptal	Negative	Positive	Positive	Left	Left
Left posteroseptal	Positive	Positive	Positive	Left	Left
Left lateral	Positive	Positive	Positive	Inferior	Inferior

The most important clinical significance of WPW syndrome is the frequent occurrence of supraventricular tachycardias. The incidence of tachyarrythmias in WPW syndrome is reported to be 40-80%. The most common tachycardia in WPW syndrome is normal QRS complex supraventricular tachycardia (75-80%). Atrial fibrillation is rather uncommon in WPW syndrome (20-25%). [2]

The impulses from the atria are conducted to the ventricles via either:

- both the AV node and accessory pathway producing a broad fusion complex
- or just the AV node producing a narrow complex (without a delta wave)
- or just the accessory pathway producing a very broad 'pure' delta wave

Drugs like Adenosine, Verapamil, Propranolol, and digitalis can terminate the normal QRS complex paroxysmal supraventricular tachycardia (PSVT) with WPW syndrome. However, they do not act on the bypass tract, and drugs like digitalis or intravenous verapamil may even shorten the refractory period of the bypass tract and enhance the ventricular rate thereby placing the patient at increased risk for

ventricular tachycardia (VT). Hence, it is important to use drugs like class Ic anti-arrhythmics and amiodarone, which act on bypass tract as well as AV node, to terminate AF.[1] More recently, Ibutilide has become available as an alternative therapy for preexcitation tachycardia. For most patients with tachycardia, the prognosis is good [10], sudden death occurs rarely, with an estimated frequency of 0.1% in some report.[11]

Significant advances have been made in the field of electrophysiological studies. Radiofrequency ablation has radically changed the management of these patients. The long-term success rates of such procedures are now thought to be approaching 95%. [5] The risk of serious side effects is low and occurs at a rate of less than 1%. [5] Radiofrequency ablation was previously limited to patients with episodes of atrial fibrillation or frequent or disabling symptoms. WPW syndrome is a condition that primarily affects younger people in whom long term anti-arrhythmic prophylaxis is undesirable. There is now evidence to suggest that as its safety improves there may be a place for the risk stratification of all patients. Nevertheless, surgical ablation may be required in the occasional patient in whom catheter ablation fails.

We have reported the above case because WPW syndrome with AF is uncommon and it has a potential to precipitate ventricular fibrillation and sudden cardiac death and catastrophic consequences of using drugs like digitalis, calcium channel blockers, or beta blockers used for AF unassociated with WPW syndrome.

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