Wolff - Parkinson - White Syndrome (A Case Report)

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A case of W P W Syndrome in a known hypertensive who developed an episode of supra ventricular tachycardia simulating ventricular tachycardia is reported. Pitfalls in the diagnosis of WPW Syndrome and problems of differential diagnosis of pseudo-ventricular arrhythmia when the patient is acutely ill are high lighted.

The Wolff-Parkinson-White Syndrome is recognised electrocardiographically rather than clinically and is characterised by short PR interval and prolonged QRS duration in frequent association with episodes of paroxysmal tachycardia. Originally termed Bundle branch block with short PR duration[20] it is also known as Preexcitation syndrome[14], Bundle of Kent Syndrome[19] and accelerated nodal conduction.[15] Wilson[18] (1915) first described the electrocardiographic findings in W-P-W Syndrome and later isolated cases were reported by Wedd[17] (1921), Bain and Hamilton[3] (1929) and Hamburger[9] (1929). The incidence has been variably reported between 0.1 to 2.4 percent. Averil,[2] on a large survey of asymptomatic flying personnel reported an incidence of 0.16 percent.

The clinical importance of this syndrome lies in its frequent association with attacks of spontaneous paroxysmal tachycardia which may occur in about 70 percent of cases[21]. The paroxysmal tachycardia is usually a rapid, regular supraventricular tachycardia, much less frequently it is an atrial flutter or fibrillation[13]. This is usually well tolerated but occasionally syncope may result during such attacks. Therefore, the recognition of this syndrome assumes special importance amongst aircrews. Rarely ventricular fibrillation may occur accounting for sudden death in these cases[5]. The electrocardiographic pattern during an episode of supraventricular tachycardia may simulate ventricular tachycardia. It becomes imperative to distinguish true ventricular tachycardia from supra ventricular tachycardia due to aberrent conduction both from therapeutic and prognostic points of view. Similarly to an unwary observer it may mimic bundle branch block, myocardial infarction or ventricular hypertrophy.

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We report a case of W.P.W. Syndrome in a known hypertensive who presented with congestive cardiac failure and supraventricular tachycardia simulating ventricular tachycardia.

Case Report

P. J., a male patient aged 40 years was admitted to the Air Force Hospital, Bangalore on 15th August, 1972 with the complaints of breathlessness, palpitations, restlessness and progressive swelling over legs, face and abdomen. Past history revealed him to be a case of long standing hypertension with congestive cardiac failure (since 1968) for which he was taking anti-hypertensive (Aldomet) and was on maintenance doses of Digoxin. Also he gave history of attacks of palpitation with marked weakness and headache since past 10-12 years which appeared at intervals of 4 to 6 months and continued from one day to 3-4 days.

Physical examination on admission revealed, an averagely nourished patient with marked restlessness and dyspnea. There was rise in JVP (6 cms), pitting edema over legs with puffiness of face, enlarged (6 cms) and tender liver, indicating congestive cardiac failure. BP was 216/160 mm Hg and heart rate 146/minute and regular. Heart was enlarged and a grade 3 systolic murmur was detected over apex conducted to axilla and along the left sternal border. Fine crepitations were detected over both lung bases and apart from slight mental confusion no overt neurological deficit could be detected. An electrocardiogram taken on 15th August, 1972 (Figure 1) was interpreted as indicative of I. H. D. The patient was diagnosed as a case of hypertension with I.H.D. and congestive cardiac failure and treated with bed rest, digoxin, aminophylline, Lasix, salt restriction and Aldomet

An electrocardiogram repeated on 16th August, 1972 at 1030 hours (Figure 2) revealed paroxysmal tachycardia with a regular ventricular rate of 188/minute and

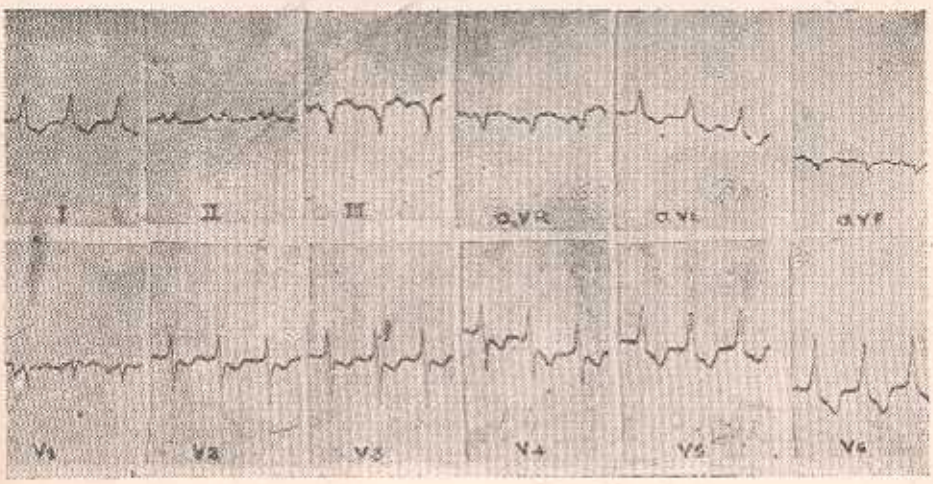

Fig. 1

wide bizzare QRS complexes with absent P waves. Patient at this stage was asymptomatic and otherwise quite comfortable. Blood pressure was maintained around 118/116 mm Hg. Pulse was 188/min regular but feeble. Other clinical findings did not show any change.

Investigations

Blood Hb : 12.0 gms%
T L C . 8,800/Cmm.
DLC : P 78, E 1, L 20, M 1%
E S R : 9 mm fall after one hour
Urine RE : NAD
Blood sugar : 93 mgm%
Blood urea : 10 mgm%
Serum Cholesterol : 154 mgm%
Serum Uric acid : 5.2 mgm%
Serum sodium : 145 m Eq/litre
Serum Potassium : 4.5 m Eq/litre

X-ray chest showed cardiomegaly, C T ratio 16.5 : 30, with unfolding of Aorta and congestion of lung fields.

The clinical picture, in spite of the bizzare ominous looking electrocardiogram, did not show any significant deterioration. The notches on broad QRS complexes especially in V4—V6 suggested hidden P waves. Carotid massage was given but proved ineffective. A possibility of digoxin toxicity was considered in view of the patient having been on maintenance doses of digoxin for the past 4 years. Digoxin was, therefore, withdrawn and Lignocaine given intravenously 100 mgm stat. Lignocaine was later continued by I.V. drip 1 mg/minute. After about 3 hours the patient reverted to sinus rhythm and a careful analysis of ECG revealed short PR interval (0.08 sec), prolonged QRS duration (0.12 sec) with delta waves in almost all leads, consistent with a classical W-P-W. Syndrome (Fig. 3). Patient was treated with anti-hypertensives, diuretics and digoxin (which was restarted) and he made an uneventful recovery and was discharged after 4 weeks stay in the hospital.

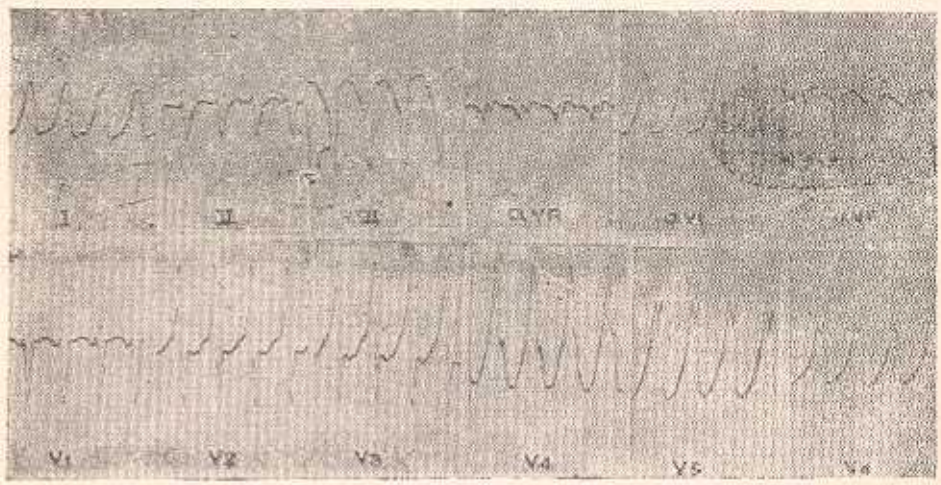

Fig. 2

Discussion

The classical electrocardiographic criteria in W-P-W. Syndrome comprise of a short PR interval which usually is between 0.08 and 0.10 sec, a wide QRS complex between 0.11 to 0.14 sec and a slow upstroke of the initial QRS complex in certain leads, called the delta wave. The P. J. Interval (Sum of PR and QRS intervals) approximates 0.25 sec or less in almost all instances. The electrocardiographic pattern may be continuous or intermittent. Further depending upon the Vector Orientation the W-P-W. electrocardiogram has been divided into two groups, A and B. Group A is characterised by tall R waves and no S waves in right precordial leads, Q or QS complexes in inferior leads. In Group B there are tall R waves in left precordial leads and QS or rS pattern in V1 and V2 and frequently Q or QS in leads III and avF. The case reported belongs to Group B type. In view of the marked ST-T wave changes in the anterior and lateral leads an erroneous diagnosis of ischemic heart disease was made in this case. Master's exercise tests usually reveal false positive results and are therefore of no significance in W-P-W. Syndrome[8]. Association of W.P.W. Syndrome with Congenital Heart disease particularly in Ebsteins Anomaly of tricuspid valve[7], its frequent presence in infants and children and its familial occurrence[12] in certain cases support the concept of congenital nature of the electrocardiographic abnormality. Detection of organic heart disease with W. P. W. Syndrome is generally coincidental[20] but there are reports of its associations with coronary heart disease, hypertensive heart disease[10], Rheumatic Valvular diseases and Thyrotoxicosis. In the case reported there was incidental presence of hypertensive heart disease. As yet, however, there is no proof to suggest any causal relationship of W-P-W. Syndrome with any acquired heart disease.

The precise mechanism responsible for the W. P. W. Syndrome is not clear. Whereas Wolff et al[20] considered the electrocardiographic changes to be due to excessive vagal tone, others (Wood et al[22]) postulated the existence of an aberrent

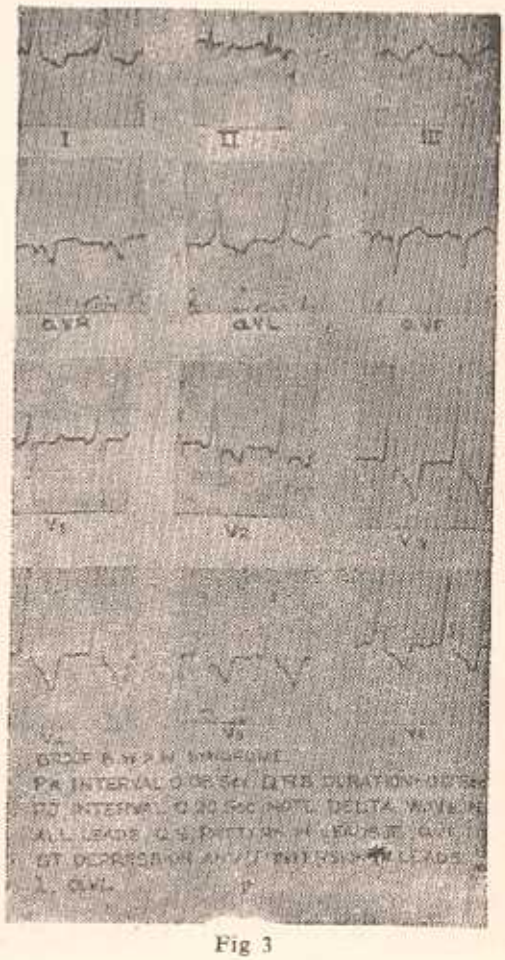

Fig 3

pathway between the atria and ventricles to be responsible. Subsequently this theory received considerable attention when a structure similar to the 'bundle of Kent' was demonstrated at autopsy[12]. The anomalous pathway is considered responsible for the attacks of paroxysmal tachycardia by facilitating retrograde conduction of the impulses into the atria. Prinzmetal and associates[15] believe that disturbances in the AV node are responsible for the accelerated conduction and postulated that the nodal abnormality occasionally acts as an ectopic focus giving rise to various supraventricular arrhythmias.

In view of the erroneous diagnosis of ischemic heart diease, a problem in differential diagnosis of the dysrhythmia occurred when the electrocardiogram (Fig. 2) showed wide and bizzare QRS complexes with absence of P waves. This suggested a possibility of ventricular tachycardia but the clinical status of the patient was re-assuring. A carotid massage was given which proved unsuccessful and since the diagnosis was not very clear, digoxin was with-held and Lignocaine used which effectively terminated tachycardia. Use of Lignocaine in successfully aborting episodes of supraventricular tachycardia with WPW Syndrome have been reported by Dye[6] (1969). Herrmann et al[11] emphasise that extremely rapid ventricular rates (over 200) where the clinical picture does not warrant alarm inspite of the bizzare and wide QRS complexes, should suggest pseudo-ventricular (supraventricular) tachycardia in a patient with accelerated A-V conduction and false bundle branch block. When, however, a proper diagnosis cannot be made it is prudent to resort to DC Conversion[1] or use procaineamide[11] or Lignocaine[6] rather than digitalis for terminating the tachyrhythmia[1]. Congestive cardiac failure following prolonged paroxysms of tachycardia though rare has been reported[4]. In our case the episode of paroxysmal tachycardia aggravated the existing congestive failure. The prognosis is generally good but on rare occasions death may result from altered haemodynamics of sustained tachyrhythmias especially in an individual with cyanotic heart disease[1]. Sudden death due to ventricular fibrillation has also been documented[3].

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