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Incidental WPW Pattern in a Patient of Acute Coronary Syndrome: Case Report and Review of Literature

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ABSTRACT

Electrocardiographic features of a short PR interval, a delta wave, and a wide QRS complex constitutes a Wolff-Parkinson-White (WPW) pattern. Asymptomatic electrocardiographic findings are defined as a WPW pattern. Symptomatic patients with these electrocardiographic features have WPW syndrome. WPW syndrome may predispose to arrhythmias such as paroxysmal atrial tachycardia, atrial fibrillation and ventricular tachycardia. Patient's with WPW syndrome at risk for sudden cardiac death. It is important to recognize the common electrocardiographic characteristics of WPW pattern. The advent of electrophysiological studies (EPS) and radiofrequency ablation has revolutionized the management of WPW syndrome. We are presenting an incidental case report of WPW pattern in a middle aged female with acute coronary syndrome.

Keywords: Wolff-Parkinson-White Syndrome, Wolff-Parkinson-White Pattern, Pre-excitation, Accessory pathway, WPW pattern in ACS

1. INTRODUCTION

Wolff, Parkinson, and White described a patient series in the year 1930 who suffered from paroxysms of tachycardia with classic electrocardiographic findings which has been described as WPW pattern. [1] It is a congenital abnormality in the cardiac conduction system. WPW pattern is a ventricular pre-excitation entity wherein an accessory bypass tract known as the bundle of Kent serves as the connection between the atrial to the ventricular myocardium bypassing the atrioventricular (AV) node. [2, 3] When electrical impulses are conducted via this accessory tract, it leads to premature ventricular activation described as pre-excitation. Following the pre-excitation, an electrical impulse is conducted via the usual conduction system. [2-4] WPW presence may predispose to paroxysmal supraventricular tachycardia in which a reentrant circuit is formed from the bypass tract and the normal conduction pathway. [3, 5]

1.1 Electrocardiographic features

The following electrocardiogram (ECG) features (Figure 1) have been described as classic for WPW pattern:

- A wide QRS complex as it is formed by sum of normal ventricular activation and ventricular pre-excitation through the accessory tract. [3-5]
- A slurred ascending limb of the R wave described as the 'delta' wave that occurs due to ventricular pre-excitation. [3-5]
- There is a short PR interval due to early ventricular depolarization that results due to bypassing of the AV nodal delay. [3-5]

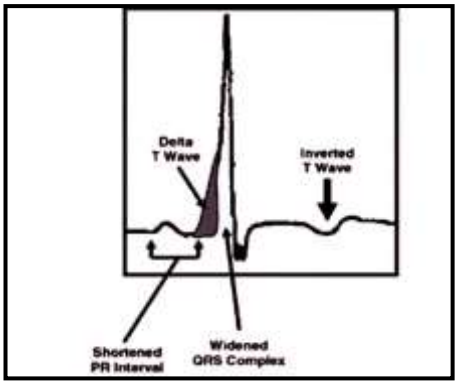

Fig. 1 - Classical WPW ECG pattern
1.Short PR interval
2.Delta wave - slurred upstroke of QRS complex
3.Wide QRS complex

Rapid conduction of the electrical impulses through the accessory tract leads to pre-excitation resulting in a short PR interval. T wave inversion and ST segment depression occurs due to the QRS complex abnormality. [6] T wave is inverted in the leads that show a wide and positive deflection. T wave inversion suggests abnormal repolarization due to ventricular pre-excitation. [6] Usually, the transition of R/S ratio from less than 1 to greater than 1 is expected to occur between leads V3 and V4. Early transition occurs when the R/S ratio is greater than 1 in V2 and may be seen with WPW pattern. [6] Left axis deviation may be another electrocardiographic finding in WPW pattern. [7]

Based on the direction of the accessory pathway, three QRS morphologies are described in the WPW pattern as follows:

- Type A WPW- left septal connection: positive QRS complexes in all the precordial leads. This may mimic posterior wall myocardial infarction or right bundle branch block. [4].
- Type B WPW- right sided connection: negative QRS complexes in lead V1 and positive QRS complexes in lead V6. This may mimic left ventricular hypertrophy or left bundle branch block. [4]
- Type C WPW- left lateral connection: positive QRS complexes in lead V1 and negative QRS complexes in lead V6. This may mimic right ventricular hypertrophy. [4] Refer to table 1 below.

Table 1 - Types of WPW pattern on ECG [4]

Type A WPW	**Positive QRS complexes in all the precordial leads**
Type B WPW	Negative QRS complexes in V1 and positive QRS complexes in V6.
Type C WPW	Positive QRS complexes in V1 and negative QRS complexes in V6.

Multiple pathways have been located for the conduction of WPW pattern (Figure 2) [4]

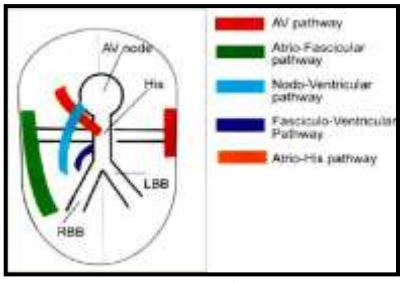

Fig. 2 - Various types of accessory pathways

2. CASE PRESENTATION

A middle aged healthy looking pleasant lady (Figure 3) presented to us with classical anginal pain occurring frequently at rest and moderate effort for last 3-4 months. She had consulted several local medical practitioners who had dismissed her symptoms to be related "Gas" and anxiety.

Fig. 3 - Photograph of the patient

Couple of ECG'S were also conducted during these visits (Figure 4,5) and each of them were suggestive of ECG findings of WPW pattern Type B (Short PR interval, Delta wave in L1, aVL, V4, V5, V6-slurring of upstroke of QRS complex, QRS complex 116 mmHg). Her problems had escalated for last 3-4 days.

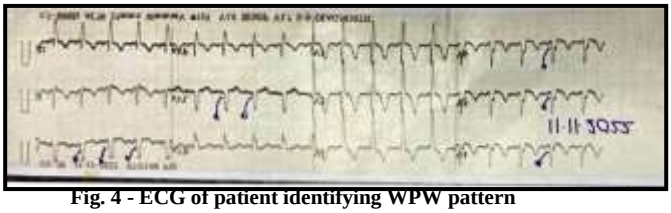

Fig. 4 - ECG of patient identifying WPW pattern
1. Short PR interval (< 120 msec)
2. Delta wave in L1, AVL, V4, V5, V6 - slurring of upstroke of QRS complex
3. QRS complex > 110 msec

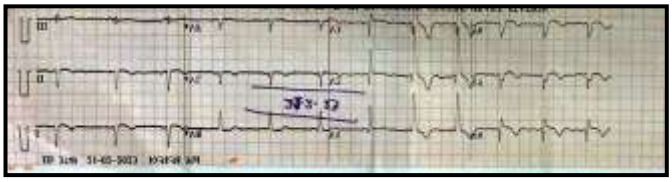

Fig. 5 - ECG of incidental WPW pattern co-existing with acute coronary syndrome dated 21.2.2023

On clinical examination she was calm and composed with a pulse rate of 91/min, regular, BP was 120/90 mmHg in the right upper limb in sitting position, SpO2 was 96% at room air. Cardiovascular, respiratory and abdominal examination were unremarkable.

3.Investigations

Test name	**Result**	**Bio Ref. Range**	**unit**
Hemoglobin	14.4	11.0 -14.0	g/dl
TLC (Total Leukocyte Count)	6,800	4,000-11,000	/cumm
DLC (differential leukocyte count)			
Neutrophil	55	40.0-70.0	%
Lymphocytes	34	20.0-45.0	%
Eosinophils	6	1.0-6.0	%
Monocytes	5	00.0-10.0	%
Basophils	0	00.0-02.0	%
Platelets	1.52	1.5-4.0	lac/cumm
C-reactive protein High sensitive	2.23	0.00-6.00	mg/dl
Serum creatinine	0.82	0.40-1.50	mg/dl
Serum uric acid	4.0	3.5-7.3	mg/dl
Blood sugar R	102	70.0-170.0	mg/dl
HBA1C	4.5	0.0-6.0	%
T3	1.63	0.5-2.5	ng/ml
T4	9.40	5.0-12.5	mcg/dL
TSH	1.53	0.3-6.0	µlU/m
Trop T	0.016	0.00-0.010	ng/ml
Serum Sodium	143.9	135.0-150.0	mmol/L
Serum Potassium	3.38	3.50-5.50	mmol/L
Lipid Profile, serum			
Serum Cholesterol	198.0	130.0-230.0	mg/dL
Serum Triglyceride	105.0	70.0-190.0	mg/dL
Serum Low Density Lipoprotein	126.0	<130.0	mg/dL
Serum High Density Lipoprotein	51.2	35.0-75.0	mg/dL
****Trop T was borderline (0.016 ng/ml)**			

ECG done at our centre was similarly consistent with WPW pattern. Color Echocardiography identified a moderate sized area of regional wall abnormality in the apical, mid septum, LV apex and apical anterior wall of left ventricle perfused by left anterior descending artery (LAD). We planned a TMT which disclosed significant horizontal downsloping ST depression in L_2, L_3, aVF V_5, V_6 with mild ST elevation in AVR (Figures 6a, 6b, 6c). These signs are strongly suggestive of inducible ischemia. There was accompanying classical anginal pain at peak exercise. Hence the patient was referred to a tertiary care medical institute for Coronary Angiography and if required necessary revascularization. In the interim phase she was advised to continue with medical treatment. Regarding the incidental presence of WPW pattern we decided not to refer the patient for Electrophysiology study and subsequent ablation of the accessory

pathway, because the occurrence of sudden death in asymptomatic WPW pattern is extremely rare and argues for a non-interventional approach. [8]

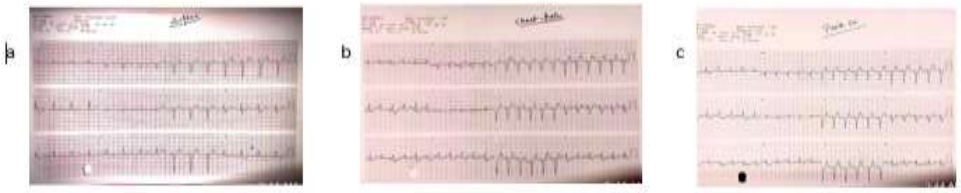

Fig. 6 - (a) Supine resting ECG of patient prior to TMT; (b) Patient experienced classical anginal pain on TMT with significant horizontal downsloping depression in L2, L3, AVF, V5, V6 and mild ST elevation in Lead AVR; (c) At peak exercise on the TMT, even though there was changes of inducible ischemia, however, there was no change in WPW pattern and moreover delta wave did not disappear.

3.1 Medical Treatment

Bisoprolol (1.25) BD
Nitrolong (2.6) BD
Ranozex (500) TDS
Novastat (20) ┐
Deplatt A (75/75)) ┘ After

DISCUSSION

The WPW pattern is applied to the patient with pre-excitation manifest on an ECG in the absence of symptomatic arrhythmia. The WPW syndrome is applied to the patient with both pre-excitation manifested on the ECG and symptomatic arrhythmia involving the accessory pathway. Although the majority of patients with pre-excitation remain asymptomatic throughout their lives, sudden death has been reported with ventricular fibrillation being the usual mechanism.[9] EKG of patient with pre-excitation may mimic other conditions, including myocardial infarction, ventricular bigeminy, accelerated idioventricular rhythm, or electrical alternans.[9-10] in 1976, Ruskin et al. reported that among the 44 patients with WPW syndrome referred to their institution, 70% had EKG findings simulating a Q wave myocardial infarct pattern.[11] Guler et al. described a patient who was successfully resuscitated from ventricular fibrillation and whose initial EKG showed ST-segment elevation in precordial leads, suggesting an acute anterior myocardial infarction.[9] Poh et al. Also described a similar case of WPW but with early repolarization changes in the EKG which could mimic myocardial infarction. [12]

In our case with WPW pattern ECG, the diagnosis of acute coronary syndrome was suspected by the presence of classical anginal pain, together with a borderline positive Trop T and on color echocardiography presence of regional wall motion abnormality in left anterior descending artery territory.

4.1 WPW ECG pattern- Review of Literature

In 1921, a phenomenon of "intraventricular block and a PR interval of 0.08 ms" in a 19-year-old patient with paroxysms of tachycardia was first described by A.M. Wedd.[13] It was in 1930, when Louis Wolff, John Parkinson, and Paul D. White together described a set of eleven cases of an electrocardiographic pattern consisting of a "functional bundle branch" and a "short PR interval" in the healthy young people with paroxysms of tachycardia.[1] However, it is not uncommon to incidentally find a Wolff-Parkinson-White (WPW) pattern in a routinely performed electrocardiography (ECG) without any arrhythmia. WPW pattern is a ventricular pre-excitation wherein an accessory bypass tract known as the bundle of Kent serves as the connection between the atrial to the ventricular myocardium bypassing the atrioventricular (AV) node. When electrical impulses are conducted via this accessory tract, it leads to premature ventricular activation described as pre-excitation. [14]

In the absence of a documented tachyarrhythmia or related symptoms, the characteristic ECG findings alone are referred to as the WPW pattern. [15]

4.2 Anatomy of the accessory pathways

The accessory pathway (AP), which leads to pre-excitation, results from a developmental failure to eradicate the remnants of the atrioventricular connections present during cardiogenesis. It also results from the anomalous myocardial tissue spanning the fibrinous bridges between the atria and ventricles.[1] Morphologically, accessory pathways are the strands of normal myocardium that bridge the AV groove at any point around the annulus fibrosus on either side of the heart except that portion of the mitral valve annulus between the right and left fibrous trigones. [16] The locations of the accessory pathways are regionalized to the left-free wall (58%), posterior septal (24%), right free-wall (13%), and anterior septal (5%) sites, respectively. [17] The accessory pathways usually exhibit rapid and non-decremental conduction and have a longer effective refractory period (ERP) than that of the AV node. The atrial and ventricular insertions of accessory pathways (free-wall APs) are located between the valve annulus and the atrial and ventricular epicardial reflections, respectively. [18] The posterior septal pathways are located within the pyramidal space, bounded anteriorly by the insertion of the atrial extension of the membranous septum into the right fibrous trigone and posteriorly by the epicardium overlying the crux of the heart. The lateral boundaries are formed by divergent walls of the left and right atria. Within this space is the AV nodal artery, the tendon of Todaro, epicardial fat, and the proximal portion of the coronary sinus. The AV node and its proximal penetrating bundle lie within the triangle of Koch, immediately adjacent to the pyramidal space. The anterior septal pathways are located just anterior to the AV node and pass through the fat pad between the right and left fibrous trigones and the insertion of the right coronary artery into the AV groove. This path lies anterior to the membranous portion of the interatrial septum and is bounded by the pericardial reflection of the ascending aorta and medial wall of the right atrium. [19] The accessory pathway present in the WPW pattern is capable of conducting in both antegrade and retrograde directions that eventually begets a re-entrant supraventricular tachycardia (SVT). [20] The orthodromic SVT, in which the anterograde conduction to the ventricles is through the AV node/His bundle, and the retrograde conduction to the atria is through the accessory pathway, accounts for 90% of arrhythmia. This is also known as a concealed accessory pathway in which there is no pre- excitation in the ECG but the patients are prone to develop orthodromic SVT. The antidromic SVT occurs in 10% of patients with the WPW syndrome in which the anterograde limb is the accessory pathway, and the retrograde limb is either the normal conduction system or a second accessory pathway. This is also known as a manifest accessory pathway in which there is pre-excitation in the ECG and the patients are prone to develop antidromic SVT. [10]

It is a well-known fact that the accessory pathway allows for rapid ventricular conduction in the presence of atrial fibrillation, which, in turn, might degenerate into ventricular fibrillation with the hemodynamic collapse that leads to sudden cardiac death (SCD).

The characteristics of various accessory pathways are depicted in Table 2

Table 2 - Types of accessory pathways and their ECG features.

Pathways	PR interval	Delta wave
Atrioventricular (Kent)	Short	Present
Atrionodal (James)	Short	Absent
Atriohisian (Brechenmaker)	Short	Absent
Atriofascicular (Mahaim)	Normal	Present
Nodofascicular (Mahaim)	Normal	Present
Fasciculoventricular	Normal	Present
Nodoventricular	Normal/Decreased	Present

5.Clinical Presentation

In the absence of any symptoms, a WPW pattern may be an incidental finding in the routinely performed ECG. In those patients who are symptomatic, arrhythmias, predominantly in the form of AV re-entrant tachycardia (AVRT) and atrial fibrillation (AF) are the commonest presentations. AVRT is the most common arrhythmia accounting for 95% of re-entrant tachycardias and AF has been estimated to be present in one-third of cases of WPW syndrome. Ventricular fibrillation is the dominant cause of SCD in patients with WPW syndrome. [2] Commonly, there is anterograde conduction through the AV node that returns retrogradely via a bypass tract to the atria leading to an orthodromic tachycardia that would result in narrow QRS complexes. Less commonly, there occurs anterograde conduction through the bypass tract, returning retrogradely to the atria via the normal AV nodal pathway leading to antidromic tachycardia resulting in wide QRS complexes. [21]

5.1 Localisation of accessory pathway in the ECG

Several algorithms have been developed and validated. We present a simple method of localizing the accessory pathway in the ECG as shown in Table 3. [22]

Table 3 - ECG localization of accessory pathways.

I. aVL	II, III, aVF	QRS axis	Precordial leads (QRS polarity)	Pathways
Positive	Negative	0° to-30°	V1: Negative V2-V3: Positive	Right Posteroseptal
Positive	Negative	-30° to-60°	V1-V3: Negative	Right Lateral
Positive	Negative	-60° to-90°	V1-V3: Positive	Left Posteroseptal
Negative	Positive	+90° to-120°	V1-V3: Positive V5-V6: Negative	Left Lateral
Positive	Positive	Normal	V1-V3: Negative	Anteroseptal

5.2 Natural history of WPW pattern

The prevalence of WPW is estimated to be 1-3 in 1000 individuals based on large-scale population-based studies. [23] Among the first-degree relatives following an index case of WPW, the incidence was found to be 5.5 in 1000 individuals.[24] On a resting ECG with a WPW pattern, approximately 65% of adolescents and 40% of adults over 30 years are asymptomatic.[25] The majority of patients with WPW patterns have a structurally normal heart. However, it can occur commonly in the presence of Ebstein's anomaly and hypertrophic cardiomyopathy, and uncommonly in the presence of cardiac rhabdomyoma. In symptomatic patients with palpitations and pre-syncope, arrhythmias in the form of atrioventricular reciprocating tachycardia (AVRT) and atrial fibrillation (AF) are most commonly encountered.

Rapid conduction of AF over the accessory pathway resulting in ventricular fibrillation (VF) is rare but unfortunately may be the first manifestation of WPW syndrome.[26] Various studies have estimated the overall lifetime risk of SCD in asymptomatic WPW patients to be 3% - 4%, with the most cases between ages 10 and 40 years [27] and SCD has been found to be the first presentation in 65% of patients with asymptomatic WPW pattern.[26] Of note, asymptomatic WPW patterns are also being increasingly identified in children during routine screening and investigations for an unrelated illness. Hence, the accurate identification of high-risk features for SCD can help prevent this dreaded outcome.

5.3 Risk stratification in an asymptomatic WPW pattern

The main purpose of risk stratification in asymptomatic patients with a WPW pattern is to identify which individuals are at an increased risk of lethal arrhythmia and SCD.

- **Patient history:** On clinical evaluation, the high-risk features include male sex, familial WPW syndrome (autosomal dominant, chromosome 7, PRKAG2 gene mutation), WPW pattern detected in the first two decades of life, history of

atrial fibrillation and arrhythmic symptoms like syncope, and presence of congenital heart disease, especially, Ebstein's anomaly. Also, high-risk occupations such as those of pilots, bus drivers, and athletes should be given a special priority. [15]

- **Non-invasive testing:** In asymptomatic patients with WPW patterns, non-invasive tests like 12-lead ECG, ambulatory ECG monitoring, and exercise stress test (EST) are considered safe and should be done.[29]

The intermittent loss of pre-excitation during sinus rhythm on serial ECGs or ambulatory monitoring confers a low risk for cardiac arrest.[30] The appearance of different pre-excited morphologies on the ECG or ambulatory monitoring denotes a higher risk for SCD.[31]

The effect of sympathetic stimulation on the accessory pathway refractoriness and AV nodal conduction affects the delta wave behavior during exercise. The exercise induced rapid AV nodal conduction may mask persistent pre-excitation and portends a low risk for VF. [32]

5.4 Pharmacological challenge test: No longer used

5.5 Invasive Electrophysiological (EP) testing

When the non-invasive tests are inconclusive regarding the anterograde conduction of AP, an invasive EP study should be considered. The review of literature in context to invasive EP study and management of asymptomatic ECG WPW pattern is beyond the scope of this manuscript.

The recommended management guidelines according to the American College of Cardiology/American Heart Association (ACC/AHA) for asymptomatic adults with a WPW pattern in ECG are summarized in Table 4. [15]

Table 4 - Asymptomatic Patients with Pre-Excitation: Recommendations

Class of Recommendations	Recommendations
Class I	Abrupt loss of conduction over a manifest pathway during EST in sinus rhythm, intermittent loss of preexcitation during ECG, and ambulatory monitoring are all useful tests in order to identify patients at low risk of SCD.
Class IIa	An EP study is reasonable to risk-stratify for arrhythmic events.
	Catheter ablation of the accessory pathway is reasonable if an EP study identifies a high risk of arrhythmic events, including rapidly conducting pre-excited AF.
	Catheter ablation of the accessory pathway is reasonable in asymptomatic patients if the presence of pre-excitation precludes specific employment (such as with pilots).
	Observation, without further evaluation or treatment, is reasonable in asymptomatic patients with pre-excitation.

Association between asymptomatic Wolff-Parkinson-White (WPW) syndrome and sudden cardiac death (SCD) has been well documented. Such asymptomatic patients should be subjected to different non-invasive and invasive tests to classify them as low or high risk for SCD. Regular cardiac follow-up, reassurance, and proper counseling are the mainstay of therapy for low-risk patients whereas radiofrequency ablation (RFA) of the accessory pathway is the definitive therapy for high-risk patients. Further studies are needed to identify predictors for future cardiac events and to assess whether medical intervention can reduce overall risk.

6. CONCLUSION

Although it is common for WPW to simulate myocardial infarction on ECG through ST-segment elevation and Q wave presence. However, no reliable algorithm exists for making an ECH diagnosis of non ST-segment elevation myocardial infarction in the presence of WPW pattern. Similarly, currently non-invasive modalities have limitations in detecting jeopardized myocardium. It acute or hyper an acute injury is suspected; the patient should be

emergently referred for coronary angiography.

Our patient was managed adequately and is doing well on medical therapy while wailing for coronary angiogram. The incidental WPW pattern on ECG passes of difficult management dilemma.

Declaration by Authors

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