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

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A B S T R A C T

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. Herein we are presenting an incidental case report of WPW pattern in a middle aged female with acute coronary syndrome with exhaustive literature review.

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]

Electrocardiographic features

The following electrocardiogram (ECG) features (Figure 1) have been described as classic for WPW pattern: Bulleted lists may be included and should look like this:

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

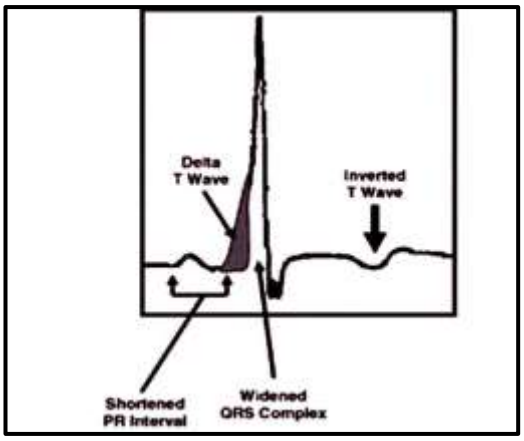

Fig. 1 - Classical WPW ECG pattern

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 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:

1. 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].
2. 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]
3. 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.

All tables should be numbered with Arabic numerals. Every table should have a caption. Headings should be placed above tables, left justified. Only horizontal lines should be used within a table, to distinguish the column headings from the body of the table, and immediately above and below the table. Tables must be embedded into the text and not supplied separately. Below is an example which the authors may find useful.

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]

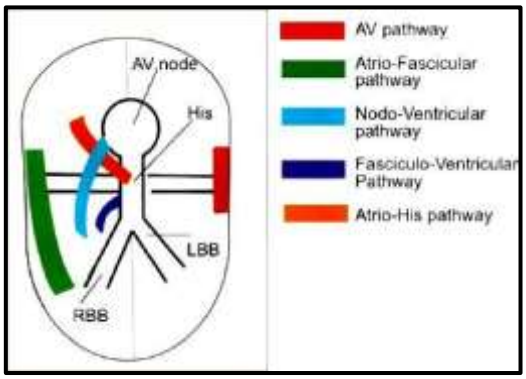

Fig. 2 - Various types of accessory pathways

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 to "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 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.

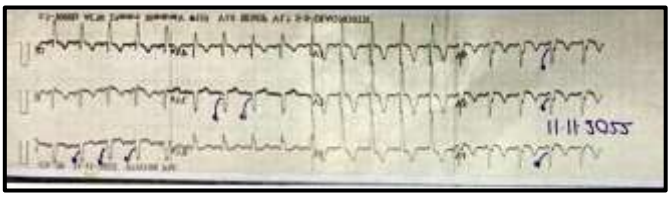

Fig. 4 - ECG of patient identifying WPW pattern

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

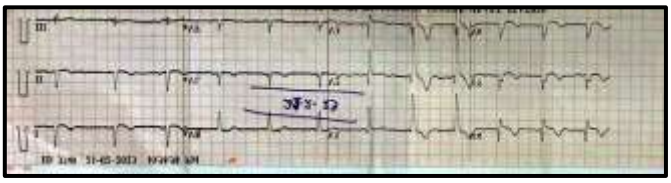

Fig. 5 - ECG of incidental WPW pattern co-existing with acute coronary syndrome dated 21.2.2023

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.

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	3.86	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). There was normal left ventricular dimensions with normal left ventricular ejection fraction (LVEF). LVEF was 57%. We planned a TMT which disclosed significant horizontal downsloping ST depression in L2, L3, aVF V5, V6 with mild ST elevation in AVR (Figure 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 centre 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. [2, 3]

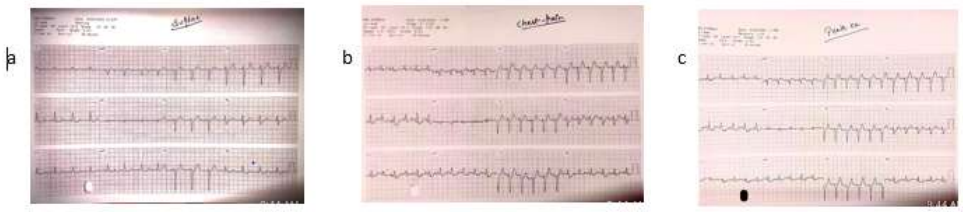

Fig. 6 - (a) Supine resting ECG of patient prior to TMT; (b) Patient experienced classical anginal pain on TMT with significant horizontal downsloping depression in L2, L3, AVF, V5, V6 and mild ST elevation in Lead AVR; (c) At peak exercise on the TMT, even though there was changes of inducible ischemia,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

1.1 Medical Treatment

- Bisoprolol (1.25) BD
- Nitrolong (2.6) BD
- Ranozex (500) TDS
- Novastat (20)
- Deplatt A (75/75))] After Dinner

2. Discussion

The WPW pattern is relatively common and found in the range of 2-4 individuals per 1000, [8, 9] the great majority will not be aware of the issue unless it is discovered incidentally. The lifetime risk of mortality related to this in asymptomatic individuals can never be accurately known but has been estimated to be in the range of 1 per 1000 (0.1% annual risk). [9] This small risk of sudden death is thought to be in the first part of life, with the majority of patients identified between the ages of approximately 10-40 years. The small hearts of normal infants and small children are perhaps more resistant to sustained atrial fibrillation (AF) or ventricular fibrillation (VF), whereas the risk in older individuals who have not had arrhythmia is attenuated by the tendency of accessory pathways to lose arrhythmic functionality over time. [9]

Interestingly, on a resting ECG with a WPW pattern, approximately 65% of adolescent and 40% of adults over 30 years of age are asymptomatic. [9] Notably, majority of patients with WPW pattern have a structurally normal heart. Conspicuously it may occur alongwith Ebstein's anomaly, hypertrophic cardiomyopathy and cardiac rhabdomyoma.

The WPW pattern is defined as pre-excitation manifest on an ECG in the absence of symptomatic arrhythmia and WPW syndrome is characterised by both pre-excitation manifested on the ECG and symptomatic arrhythmia involving the accessory pathway (AP). 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.[10] ECG of patient with pre-excitation may mimic other conditions, including myocardial infarction, ventricular bigeminy, accelerated idioventricular rhythm, or electrical alternans.[10-11] in 1976, Ruskin et al. reported that among the 44 patients with WPW syndrome referred to their institution, 70% had ECG 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.[10]

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 there was presence of regional wall motion abnormality in left anterior descending artery territory.

2.1 WPW ECG pattern- Review of Literature

Solitary Wolff-Parkinson-White (WPW) pattern ECG without any accompanying arrhythmia is not uncommon routinely and generally accessory pathway of bundle of Kent connects the atria with ventricles by circumventing or bypassing the atrio-ventricular node (AVN). [12] Wolff Parkinson and White in 1930, explicitly illustrated in eleven healthy young adults ECG pattern of a functional bundle branch block and a short PR interval associated with episodes of fast heart rate. The conglomeration of such remarkable symptoms and ECG signs were famously coined WPW syndrome. [1] When the patients possess these distinctive ECG features without any obvious symptomatology, then they are designated as WPW pattern. [13]

During early cardiogenesis in the fetus there is development failure to abolish the remnants of atrio-ventricular connections, which leads to the formation of accessory pathway (AP). [14] Morphologically, accessory pathways (APs) are the stands 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. [15] The locations of the accessory pathways are distributed to the left-free wall (58%), posterior septal (24%), right free-wall (13%), and anterior septal (5%) sites, respectively. [16] 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. [17] The accessory pathway present in the WPW pattern is capable of conducting in both antegrade and retrograde directions that ultimately leads to a re-entrant supraventricular tachycardia (SVT). 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 with a narrow QRS. [18] 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. [19]

In the presence of atrial fibrillation accessory pathway causes rapid ventricular conduction which might degenerate into ventricular fibrillation with hemodynamic collapse that may lead to sudden cardiac death (SCD). [19, 20] The peculiar features of multiple accessory pathways are elucidated in Figure 7.

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

Fig. 7 - Distinguishing ECG features of accessory pathways.

Incidental WPW pattern in routine ECG is generally benign. In symptomatic patients arrhythmias 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]

A simple method of localizing the accessory pathway in the ECG is shown in Figure 8.[21]

I. aVL	II, III, aVF	QRS axis	Precordial leads (QRS morphology)	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

Fig. 8 - Localisation of accessory pathways on ECG

Unfortunately VF may be the first manifestation of WPW syndrome, even though it is rare. [21] Overall lifetime risk of SCD in asymptomatic WPW patients is 3-4% [20] and SCD was found to be the first presentation in 65% of these asymptomatic patients. [21] Saliently in children asymptomatic WPW pattern is being increasingly demonstrated incidentally on routine ECG. Therefore rapid identification of high risk patients is crucial for the prevention of the dreaded SCD.

In asymptomatic patients with a WPW pattern, risk stratification is done with the sole intention of early detection of serious ventricular arrhythmia leading of SCD.

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. [13]

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.[22]

The intermittent loss of pre-excitation during sinus rhythm on serial ECGs or ambulatory monitoring confers a low risk for cardiac arrest. [23] The appearance of different pre-excited morphologies on the ECG or ambulatory monitoring denotes a higher risk for SCD. [24] 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. [25]

Pharmacological challenge test is not being used currently. Inconclusive non-invasive studies brings into contention the definitive EP study. The review of literature in context to invasive EP study and management of asymptomatic ECG WPW pattern is beyond the scope of this manuscript. However, the guidelines for management of asymptomatic WPW pattern adults according to the American College of Cardiology/American Heart Association (ACC/AHA) are enumerated in Figure 9. [13]

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.

Fig. 9 - ACC/AHA guidelines for management of asymptomatic ECG pattern

3. 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 ECG diagnosis of non ST-segment elevation myocardial infarction in the presence of WPW pattern. Currently non-invasive modalities have limitations in detecting jeopardized myocardium. If acute or hyperacute 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 in this patient is a difficult management dilemma in the presence of acute Coronary Syndrome.

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.

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