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Revisiting Electrocardiographic Wolff-Parkinson-White Pattern

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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, 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.

Keywords: Wolff-Parkinson-White Syndrome, Wolff-Parkinson-White Pattern, Pre-excitation, Accessory pathway.

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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 have been described as classic for WPW pattern:

1. A wide QRS complex as it is formed by sum of normal ventricular activation and ventricular pre-excitation through the accessory tract [3-8].
2. A slurred ascending limb of the R wave described as the 'delta' wave that occurs due to ventricular pre-excitation [3-8].
3. There is a short PR interval due to early ventricular depolarization that results due to bypassing of the AV nodal delay [3-8].

Refer to figure 1 for an example of an ECG showing WPW pattern in the sinus rhythm.

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 [6-9]. 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 [6,8,9]. 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 [6,8,10,11]. Left axis deviation may be another electrocardiographic finding in WPW pattern [9,11-13].

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 [4,7,14,15].

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 [4,7,8,15-17].

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 [4,8,18,19]. Refer to table 1 below.

Table 1: Types of WPW pattern on ECG [Ref: 4,7,8,14-19]

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.

Paroxysmal atrial tachycardia coexisting with WPW is suggested by the following ECG characteristics:

1. An ECG during the sinus rhythm demonstrating a short PR interval, a delta wave, and a wide QRS complex.

2. Inverted P waves are present suggestive of retrograde atrial activation and conduction.

3. A ventricular rate greater than 200 bpm suggestive of no physiological AV block. [3,5,7,9,11,13]

Commonly, there is anterograde conduction through the AV node returning retrogradely via bypass tract to the atria leading to orthodromic tachycardia resulting in narrow QRS complexes. Less commonly, there occurs anterograde conduction through the bypass tract, returning retrogradely to the atria via normal AV nodal pathway leading to antidromic tachycardia resulting in wide QRS complexes [6,9,11,13,19].

Electrocardiographic mimics

WPW pattern may mimic several other conditions such as:

1. A negative delta wave with ST-T wave changes may appear as myocardial infarction [20,21].

2. If delta wave appears separate from R wave, it may mimic a bundle branch block [6-9,20].

3. A dominant R wave in V1 may appear as a right bundle branch block [5-9,20].

4. Atrioventricular reentrant tachycardia (AVRT) that is antidromic, when conducted via accessory tract anterogradely, may appear as ventricular tachycardia [13,20].

DISCUSSION

WPW syndrome occurs when a patient with preexisting WPW pattern on ECG develop arrhythmias involving the bypass tract with symptoms such as palpitations, chest pain, presyncope, syncope, dizziness, or even sudden cardiac death. Atrioventricular reentrant tachycardia (AVRT) manifesting as a regular narrow complex tachycardia on ECG is a common supraventricular arrhythmia found in WPW syndrome [4,7,9,13,22]. Atrial fibrillation with rapid ventricular rate in WPW syndrome may degenerate to ventricular fibrillation and may lead to sudden cardiac death [8,11,13,23].

Asymptomatic individuals with the WPW pattern on ECG can be evaluated with noninvasive studies such as exercise or procainamide challenge testing or EPS for identification and risk stratification of individuals at a higher risk of sudden cardiac death [4,8,11,13,22-24]. Patients with a history of atrial fibrillation or AVRT plus a shorter refractory period of the accessory pathway have a higher risk of arrhythmias. Patients with a longer refractory period of accessory pathway may have intermittent preexcitation loss which may be depicted as a loss of delta wave on ECG during faster heart rates, and are at a low-risk as the longer refractory period may not conduct atrial fibrillation to degenerate into ventricular fibrillation. As a result, low-risk patients may be managed conservatively [5,11,13,22-24].

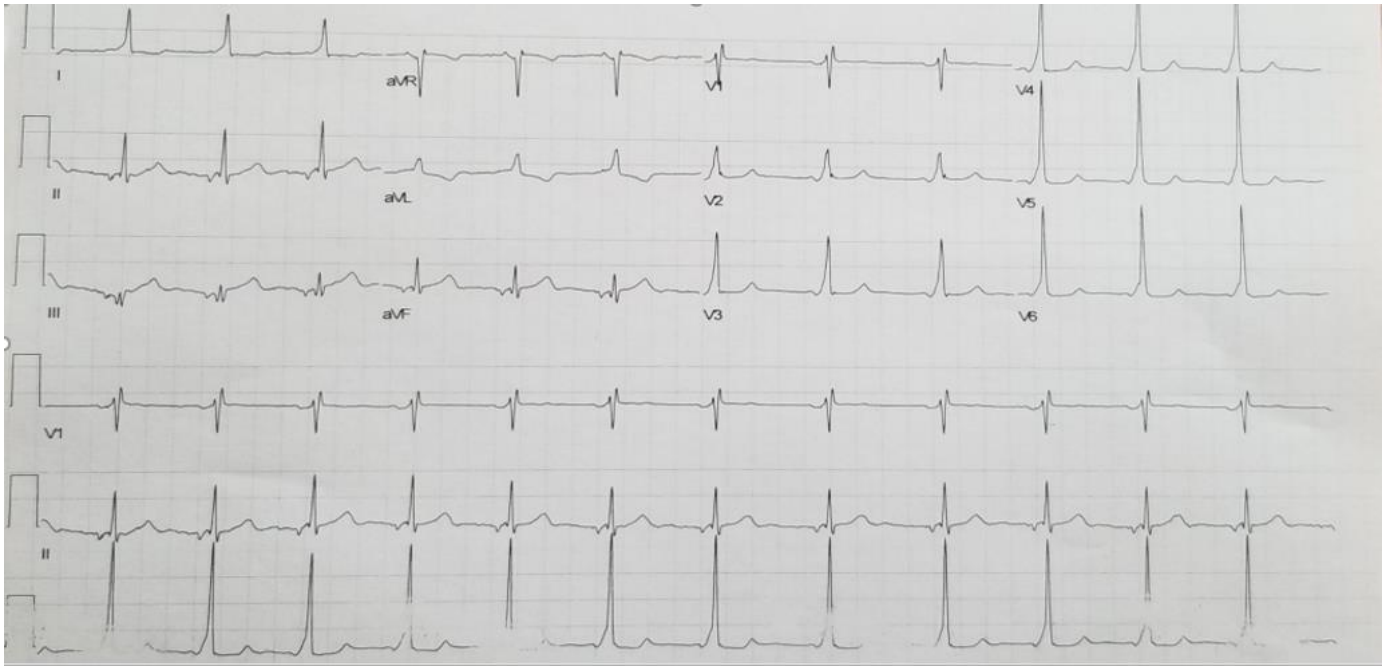

Figure 1: Sinus rhythm with WPW pattern

WPW syndrome predisposes to paroxysmal atrial tachycardia as the accessory pathway leads to formation of reentrant circuit with the regular conduction pathway. Paroxysmal atrial tachycardia coexisting with WPW syndrome suggests a poor prognosis due to risk of degeneration into ventricular tachycardia [7-9,11,13, 23, 25].

EPS are employed to locate the accessory pathways. Radiofrequency ablation of the bypass tract may be helpful for patients with recurrent, frequent, and symptomatic episodes of paroxysmal atrial tachycardia that are refractory to medical management and/or lead to hemodynamic instability. Ablation may be preferred when ECG shows a very short PR interval, or a very short refractory period is noted on an EPS [7-13,22-25].

CONCLUSION

The typical ECG features of WPW pattern include a short PR interval of less than 120 ms, QRS prolongation usually greater than 110 ms, and a delta wave. The immediate occurrence of the QRS complex after the P wave leads to the formation of a delta wave. The ST segment and T wave show discordant changes meaning that the T wave deflection is in an opposite direction to that of major deflection of the QRS complex.

Conflict of interest

The authors declare no conflict of interest.

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REFERENCES

1. Wolff L, Parkinson J, White PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. 1930. Ann Noninvasive Electrocardiol. 2006;11(4):340-353. doi:10.1111/j.1542-474X.2006.00127.x
2. Sethi KK, Dhall A, Chadha DS, Garg S, Malani SK, Mathew OP. WPW and preexcitation syndromes. J Assoc Physicians India. 2007;55 Suppl:10-15.
3. Klein GJ, Gula LJ, Krahn AD, Skanes AC, Yee R. WPW pattern in the asymptomatic individual: has anything changed?. Circ Arrhythm Electrophysiol. 2009;2(2):97-99. doi:10.1161/CIRCEP.109.859827
4. James Kulig and Bruce A. Koplan. Wolff-Parkinson-White Syndrome and Accessory Pathways. 12 Oct 2010https://doi.org/10.1161/CIRCULATIONAHA.109.929372Circulation. 2010;122:e480–e483
5. Kim SS, Knight BP. Long term risk of Wolff-Parkinson-White pattern and syndrome. Trends Cardiovasc Med. 2017;27(4):260-268. doi:10.1016/j.tcm.2016.12.001
6. Hanna Deschamps E, Hanna EB. Atrioventricular Accessory Pathways: Mechanisms, Electrocardiograms, and Associated Arrhythmias. South Med J. 2016;109(10):670-676. doi:10.14423/SMJ.0000000000000538
7. Fox DJ, Klein GJ, Skanes AC, Gula LJ, Yee R, Krahn AD. How to identify the location of an accessory pathway by the 12-lead ECG. Heart Rhythm. 2008;5(12):1763-1766. doi:10.1016/j.hrthm.2008.09.012
8. Rinne C, Klein GJ, Sharma AD, Yee R. Clinical usefulness of the 12-lead electrocardiogram in the Wolff-Parkinson-White syndrome. Cardiol Clin. 1987;5(3):499-509.
9. Jezior MR, Kent SM, Atwood JE. Exercise testing in Wolff-Parkinson-White syndrome: case report with ECG and literature review. Chest. 2005;127(4):1454-1457. doi:10.1378/chest.127.4.1454
10. Chiang CE, Chen SA, Teo WS, et al. An accurate stepwise electrocardiographic algorithm for localization of accessory pathways in patients with Wolff-Parkinson-White syndrome from a comprehensive analysis of delta waves and R/S ratio during sinus rhythm. Am J Cardiol. 1995;76(1):40-46. doi:10.1016/s0002-9149(99)80798-x
11. Lindsay BD, Crossen KJ, Cain ME. Concordance of distinguishing electrocardiographic features during sinus rhythm with the location of accessory pathways in the Wolff-Parkinson-White syndrome. Am J Cardiol. 1987;59(12):1093-1102. doi:10.1016/0002-9149(87)90855-1
12. d'Avila A, Brugada J, Skeberis V, Andries E, Sosa E, Brugada P. A fast and reliable algorithm to localize accessory pathways based on the polarity of the QRS complex on the surface ECG during sinus rhythm. Pacing Clin Electrophysiol. 1995;18(9 Pt 1):1615-1627. doi:10.1111/j.1540-8159.1995.tb06983.x
13. Katritsis DG, Camm AJ. Atrioventricular nodal reentrant tachycardia. Circulation.2010;122(8):831-840. doi:10.1161/CIRCULATIONAHA.110.936591
14. Heikkilä J, Jounela A. Intra-A-type variation of WPW syndrome. Br Heart J. 1975;37(7):767-770. doi:10.1136/hrt.37.7.767
15. Ramachandran S. Wolff-Parkinson-White syndrome. Conversion of type A to type B electrocardiographic changes. Circulation. 1972;45(3):529-533. doi:10.1161/01.cir.45.3.529
16. Gomes JA, Haft JI. Wolff-Parkinson-White syndrome type B with His depolarization occurring after the QRS. Further evidence that WPW-QRS is a fusion beat. Chest. 1975;67(4):445-449. doi:10.1378/chest.67.4.445
17. Takeuchi T, Tomita T, Kasai H, et al. A young patient with atypical type-B Wolff-Parkinson-White syndrome accompanied by left ventricular dysfunction. J Arrhythm. 2015;31(1):50-54. doi:10.1016/j.joa.2014.03.011
18. Haraguchi M, Kondo H, Ishii Y, et al. A case of Type-C Wolff-Parkinson-White syndrome with severe left ventricular dysfunction: Efficacy of catheter ablation. J Cardiol Cases. 2016;15(1):32-35. Published 2016 Nov 14. doi:10.1016/j.jccase.2016.10.002
19. Boersma L, García-Moran E, Mont L, Brugada J. Accessory pathway localization by QRS polarity in children with Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol. 2002;13(12):1222-1226. doi:10.1046/j.1540-8167.2002.01222.x
20. Wang K, Asinger R, Hodges M. Electrocardiograms of Wolff-Parkinson-White syndrome simulating other conditions. Am Heart J. 1996;132(1 Pt 1):152-155. doi:10.1016/s0002-8703(96)90404-4
21. Güler N, Eryonucu B, Bilge M, Erkoç R, Türkoğlu C. Wolff-Parkinson-White syndrome mimicking acute anterior myocardial infarction in a young male patient--a case report. Angiology. 2001;52(4):293-295. doi:10.1177/000331970105200411
22. Gaita, F., Giustetto, C., Riccardi, R. et al. Wolff-Parkinson-White Syndrome. Drugs 43, 185–200 (1992). https://doi.org/10.2165/00003495-199243020-00005
23. Milliez P, Slama R. Le syndrome de Wolff-Parkinson-White [Wolff-Parkinson-White syndrome]. Rev Prat. 2004;54(16):1747-1753.
24. Sharma AD, O'Neill PG. Wolff-Parkinson-White Syndrome. Curr Treat Options Cardiovasc Med. 1999;1(2):117-126. doi:10.1007/s11936-999-0015-7
25. Benson DW, Cohen MI. Wolff-Parkinson-White syndrome: lessons learnt and lessons remaining. Cardiol Young. 2017;27(S1):S62-S67. doi:10.1017/S1047951116002250