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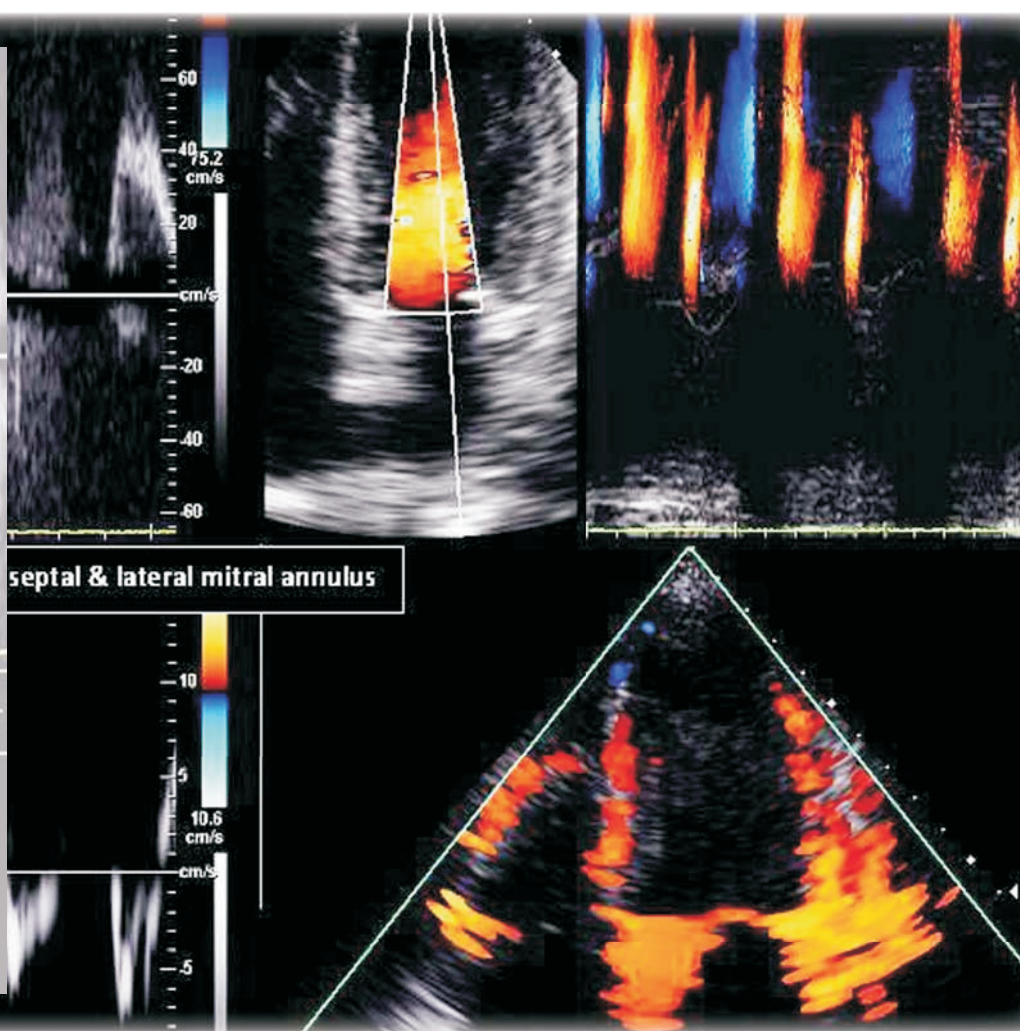
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Manuscript

NSTEMI of Infero-Posterior wall with WPW ECG Pattern-Importance of ST-Segment Depression In Anterior Leads-A Case Report

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ABSTRACT

Wolff-Parkinson-White Syndrome is a type of pre-excitation syndrome and Inferior lead pseudo-infarct Q waves are a common finding in the Wolff-Parkinson-White (WPW) syndrome. Pseudo-infarct Q waves in the inferior leads are associated with positive or isoelectric T waves in a large number of patients. This characteristic Q wave-T wave vector discordance results from secondary repolarisation changes due to altered ventricular activation. As a corollary, the presence of T wave inversion with inferior lead Q waves and a short PR interval is strongly suggestive, but not pathognomonic of inferior ischemia. The Authors describe an interesting case of Non ST-Elevation Myocardial Infarction involving the inferoposterior wall needing percutaneous coronary intervention of a tight proximal to mid Right coronary artery lesion in a patient whose ECG showed WPW pattern with significant ST-Segment depression in anterior leads V1 to V5. Significant ST-Segment depression in anterior chest leads with WPW pattern should not be ignored as secondary or discordant ST-T change. Otherwise, Acute Myocardial Infarction may be missed.

Key words: Wolff-Parkinson-White Syndrome, Non ST-Elevation Myocardial Infarction (NSTEMI), Pseudo-infarct pattern, ST-segment depression, Discordant ST-T segment.

Key Messages: Acute Myocardial Infarction may be missed if significant ST-Segment depression in anterior chest leads are ignored as secondary or discordant ST-Segment shifts in WPW pattern ECGs.

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INTRODUCTION

WPW Syndrome is characterised by presence of an accessory pathway leading to characteristic ECG changes of short PR interval, presence of delta waves, wide QRS complexes, Q waves in inferior leads and Q Wave and T-Wave vector discordance. This leads the clinician to assume these ECG abnormalities as “pseudo-infarct pattern”. Thus, Non ST Elevation Myocardial Infarction can be easily missed in the presence of WPW pattern ECG. A search of literature by the Authors has not revealed any characteristic ECG patterns which can diagnose Acute Non ST Elevation Myocardial Infarction in a patient with WPW ECG pattern at presentation. The Authors here describes a case wherein, based on the clinical presentation and ECG findings of WPW pattern combined with Q waves in inferior leads with “significant” ST-segment depression in anterior chest leads, acute infero posterior wall NSTEMI was strongly suspected. An echocardiogram confirmed involvement of inferoposterior wall. This was followed by coronary angiogram requiring angioplasty and stenting of the Right coronary artery lesion with significant improvement in ECG changes.

Case History

A 62 year female diabetic presented with acute coronary syndrome suggestive of unstable angina and elevated Troponin T levels. ECG showed Type A WPW syndrome with short PR interval, presence of delta waves, wide QRS complexes, Q waves in inferior leads and Q wave and T-wave vector discordance. The ECG also showed significant down sloping ST-segment depression in leads V2 to V5. (Figure 1). Echocardiogram showed regional wall motion abnormalities involving infero-posterior wall of left ventricle and mild LV systolic dysfunction with LVEF of 45%. In view of the clinical presentation, ECG findings, cardiac biomarker elevation and echocardiography findings, patient had coronary angiogram which showed significant 95% long segment stenosis in

proximal to mid Right coronary artery. (Figure 2). An angioplasty with 3.5×33 mm Xience Xpedition stent deployment from proximal to mid RCA with TIMI Grade 3 flow was carried out with good results. (Figure 3). ECG after angioplasty and stenting showed sinus rhythm with significant resolution in ST-segment depression in anterior chest leads V2 to V5. (Figure 4). It was fortuitous to have recorded an ECG immediate post angioplasty in sinus rhythm with complete ante grade conduction through the AV node. If most of the impulses travelling from the atria to the ventricles go through the AV node, then a short PR interval, delta wave, and other findings associated with the WPW pattern will not be seen. Thus, WPW pattern on the ECG may be intermittent.^{1,2,3} ECG recorded after 10 min showed return to WPW pattern, and ST-segment depression in leads V2 to V5 appeared to be significantly resolved compared to the pre angioplasty ECG. (Figure 5).

DISCUSSION

WPW Syndrome was first described in 1930 by Louis Wolff, John Parkinson and Paul Dudley White.⁴ In the normal heart, the atria and the ventricles are electrically isolated, with conduction of electrical impulses from the atria to the ventricles normally occurring via the atrioventricular node (AV) and the His-Purkinje system. Patients with a preexcitation syndrome have an additional pathway, known as an accessory pathway (AP), which directly connects the atria and ventricles, thereby allowing electrical activity to bypass the AV node. Tissue in the accessory pathways, are congenital in origin and result from failure of resorption of the myocardial syncytium at the annulus fibrosis of the atrioventricular valves during fetal development. They typically conduct electrical impulses more quickly than the AV node, resulting in the short PR interval seen on the surface ECG. WPW pattern versus WPW syndrome are two separate terms, distinguished by the presence or absence of

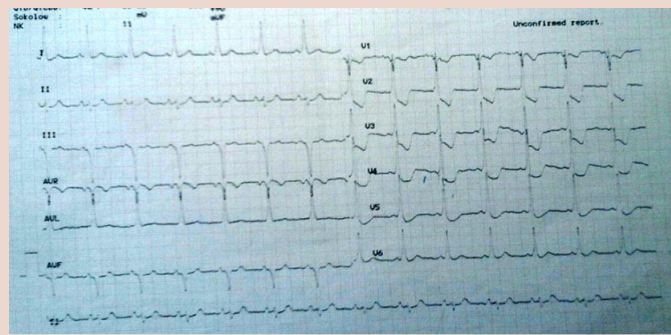


Figure 1: Type A WPW pattern with short PR interval, delta waves, wide QRS, Q in inferior leads and Q wave and T-wave vector discordance and significant down sloping ST segment V2-5

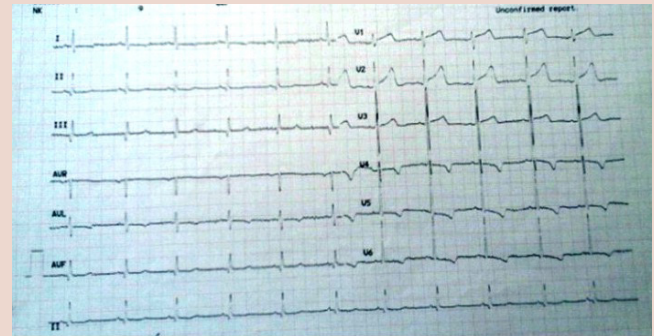


Figure 4: Resolution of ST-depression



Figure 2: RCA Proximal to Mid 95% stenosis

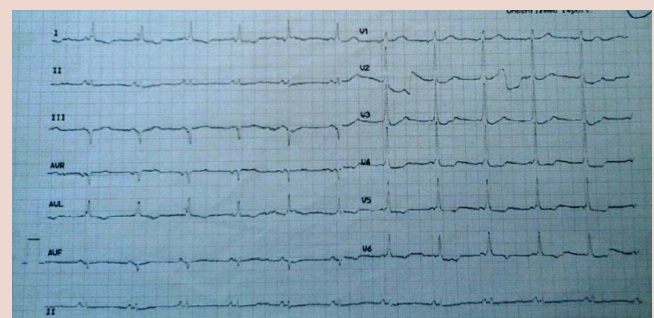


Figure 5: Resolution of ST depression with WPW pattern after 10 min after stenting

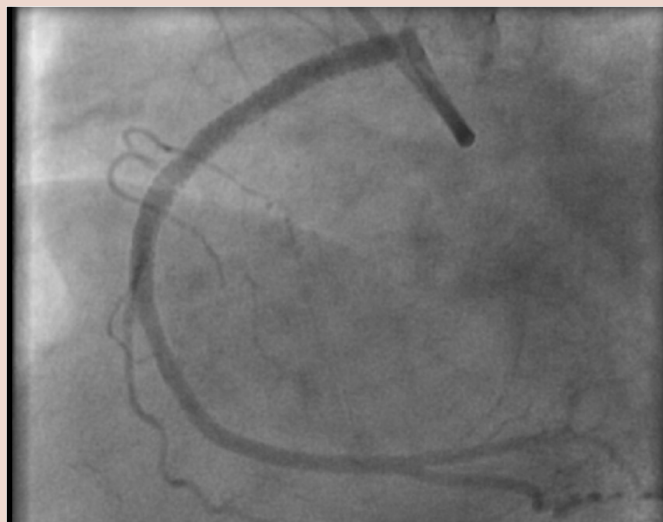


Figure 3: Post stenting

arrhythmias and have been used to describe patients with accessory pathways (AP): The Wolff-Parkinson-White (WPW) pattern is applied to the patient with preexcitation manifest on an ECG in the absence of symptomatic arrhythmias. The Wolff-Parkinson-White (WPW) syndrome is applied to the patient with both preexcitation manifest on an ECG and symptomatic arrhythmias involving the accessory pathway. Both are fairly infrequent, occurring in less than 1 percent of the general population, with the WPW pattern between 10 and 100 times more common than WPW syndrome. The prevalence of a WPW pattern on the surface ECG is estimated at 0.13 to 0.25 percent in the general population.^{1,5,6} Electrophysiologic studies and mapping have shown that accessory atrioventricular pathways may be located anywhere along the AV ring (groove) or in the septum. The most frequent locations are left lateral (50 percent), posteroseptal (30 percent), right anteroseptal (10 percent), and right lateral (10 percent). Many studies have attempted to correlate the site of the accessory pathway with the ECG pattern.^{7,8,9,10} The abnormal sequence of activation which occurs with electrical conduction via the accessory pathway gives rise to an abnormal sequence of repolarization, resulting in ST-T wave abnormalities. The vectors or direction of the secondary ST-T wave changes are usually directed opposite to the vectors of the delta wave and the QRS complex. As a result of the abnormal activation sequence, abnormalities affecting the ventricles, such as ischemia, infarction, hypertrophy, and pericarditis, may not always be reliably diagnosed in the presence of a WPW pattern. The electrocardiographic (ECG) findings in persons with the WPW pattern can be similar to ECG findings seen in other cardiac conditions like Myocardial infarction—A negative delta wave (presenting as a Q wave) may mimic a myocardial infarction pattern. Conversely, a positive delta wave may

mask the presence of a previous myocardial infarction.¹¹ Pseudo-infarct Q waves in the inferior leads are associated with positive or isoelectric T waves in a large number of patients. This characteristic Q wave-T wave vector discordance results from secondary repolarisation changes due to altered ventricular activation. As a corollary, the presence of T wave inversion with inferior lead Q waves and a short PR interval is strongly suggestive, but not pathognomonic of inferior ischemia.¹² In spite of the somewhat variable ECG findings associated with conduction over the accessory pathway, a left lateral pathway generally presents with a pseudo-lateral wall myocardial infarction (MI) pattern (Q wave in leads I and aVL), and a posteroseptal pathway most often presents with a pseudo-inferior wall MI pattern (Q wave in leads II, III and aVF). A review of literature showed a paucity of reported cases of myocardial infarction with WPW syndrome. In a paper by Thomas A. Brackbill *et al*; four cases have been described and they highlight the difficulty encountered in making a diagnosis.¹³ Thus, Non ST Elevation Myocardial Infarction

can be missed in the presence of WPW pattern ECG. There are no characteristic differentiating features reported in the literature, to the best of the authors' knowledge to differentiate pseudo infarct pattern from true infarction in a WPW pattern ECG. This case illustrates the importance of significant ST-segment depression in anterior leads V2 to V5 as a probable marker of infero posterior wall non ST elevation myocardial infarction.

CONCLUSION

ST-segment depression in anterior chest leads with WPW ECG may indicate infero posterior wall non ST elevation myocardial infarction.

ABBREVIATION USED

NSTEMI: Non ST elevation myocardial infarction; **WPW:** Wolff-Parkinson-White; **RCA:** Right coronary artery.

REFERENCES

1. Krahn AD, Manfreda J, Tate RB, Mathewson FA, Cuddy TE. The natural history of electrocardiographic preexcitation in men. The Manitoba Follow-up Study. *Ann Intern Med.* 1992;116(6):456-60.
2. Munger TM, Packer DL, Hammill SC, Feldman BJ, Bailey KR, Ballard DJ. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989. *Circulation.* 1993;87(3):866.
3. Klein GJ, Yee R, Sharma AD. Longitudinal electrophysiologic assessment of asymptomatic patients with the Wolff-Parkinson-White electrocardiographic pattern. *N Engl J Med.* 1989;320(19):1229.
4. Scheinman MM, Rambam. The history of the wolff-Parkinson-white syndrome. *Maimonides Med J.* 2012;3(3):e0019.
5. Cohen MI, Triedman JK, Cannon BC, Davis AM, Drago F, Janousek J. Pediatric and Congenital Electrophysiology Society (PACES), Heart Rhythm Society (HRS), American College of Cardiology Foundation (ACCF), American Heart Association (AHA), American Academy of Pediatrics (AAP), Canadian Heart Rhythm Society (CHRS), Heart Rhythm. 2012;9:1006.
6. Kobza R, Toggweiler S, Dillier R, Abächerli R, Cuculi F, Frey F. Prevalence of preexcitation in a young population of male Swiss conscripts. *Pacing Clin Electrophysiol.* 2011;34(8):949.
7. Milstein S, Sharma AD, Guiraudon GM, Klein GJ. An algorithm for the electrocardiographic localization of accessory pathways in the Wolff-Parkinson-White syndrome. *Pacing. Clin Electrophysiol.* 1987;10(3):555.
8. Fitzpatrick AP, Gonzales RP, Lesh MD, Modin GW, Lee RJ, Scheinman MM. New algorithm for the localization of accessory atrioventricular connections using a baseline electrocardiogram. *J Am Coll Cardiol.* 1994;23(1):107.
9. Reddy GV, Schamroth L. The localization of bypass tracts in the Wolff-Parkinson-White syndrome from the surface electrocardiogram. *Am Heart J.* 1987;113(4):984.
10. Epstein AE, Kirklin JK, Holman VL, Plumb VJ, Kay GN. Intermediate septal accessory pathways: electrocardiographic characteristics, electrophysiologic observations and their surgical implications. *J Am Coll Cardiol.* 1991;17(7):1570-8.
11. Wang K, Asinger R, Hodges M. Electrocardiograms of Wolff-Parkinson-White syndrome simulating other conditions. *Am Heart J.* 1996;132(1):152.
12. Ary L, Goldberger. Pseudo-infarct patterns in the Wolff-Parkinson-White syndrome: Importance of Q wave-T wave vector discordance. *Journal of Electrocardiology.* 1980;13(2):115-8.
13. Brackbill TA, Dove JT, Murphy GW, Barold SS. The Diagnosis of Myocardial Infarction in the Wolff-Parkinson-White Syndrome *Chest.* 1974;65(5):493-9.