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Case Report

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An Interesting Case of Wolff-Parkinsin-White Syndrome with Pre-excited Atrial Fibrillation

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Abstract

Background: Atrial fibrillation with accessory pathway can present with confounding ECG findings leading to inaccurate diagnosis sometimes leading to fatal outcomes. Treatment with AV nodal blockers is contra-indicated in pre-excited atrial fibrillation as it can lead to fatal ventricular arrythmia.

Case Presentation: A 72-year-old female presenting with acute onset palpitations, chest discomfort, shortness of breath and light-headedness with similar past episodes was initially diagnosed to have atrial fibrillation. An ECG after metoprolol administration revealed the features of pre-excitation and Wolff- Parkinson-White syndrome. A repeat episode of tachyarrhythmia was terminated with electrical cardioversion and patient was followed-up in cardiology for radiofrequency ablation.

Conclusion : A bizarre ECG with irregular wide complex tachycardia with QRS of varied shape and amplitude and sustained rates surpassing 200 beats per minute, suspicion of WPW syndrome with pre- excited AF should be considered. It is difficult to distinguish from polymorphic ventricular tachycardia, although electrical cardioversion is the primary therapy when hemodynamically unstable.

Keywords: Atrial fibrillation; Pre-excitation; WPW syndrome; Accessory pathway; Wide complex tachycardia

Abbreviations: ECG: Electrocardiogram; WPW Syndrome: Wolff-Parkinson white syndrome; BPM: Beats Per Minute; AP: Accessory Pathway AF: Atrial Fibrillation; AV blockers: Atrio-ventricular Blockers

Background

Wolff Parkinson-white syndrome can lead to a variety of dysrhythmias from severe bradycardia to fatal tachycardias. Atrial impulses can directly trigger the ventricular myocardium through the accessory pathway (AP) in the Wolfff-Parkinson-White syndrome (WPW), which is thought to affect 0.1–0.3% of the population. The electrocardiographic triad of WPW consists of a PR interval less than 0.12 seconds, a slurred and slow-rising initial QRS complex (delta wave), a widened QRS complex with a total duration greater than 0.12 seconds, and secondary repolarization changes reflected as ST segment-T wave changes that are typically directed in the opposite direction of the major delta wave and QRS complex.

These electrocardiographic changes must be observed alongside a recognised dysrhythmia in order to make the diagnosis of WPW [1].

If the pathophysiology of WPW is taken into account, these

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electrocardiogram (ECG) alterations are simple to understand. The AP conducts an impulse produced in the atria quickly and non-decrementally. Before the AV node activates the His-Purkinje system, this impulse enters the ventricle and starts to depolarize a section of the ventricular myocardium. This region of the ventricular myocardium, which depolarizes earlier than expected, first generates the delta wave, which then merges with the QRS complex to produce a barely expanded QRS complex. Strands of myocardium were seen within the typically insulating fibrous AV annulus, suggesting that accessory routes probably arise during embryologic development as a result of improper AV ring development [2].

The majority of the time, the APs conduct impulses in a non-decremental fashion, which means they are unable to decrease the quantity of impulses they send to the ventricles per unit of time. In contrast, the AV node's decremental conduction only permits a limited number of atrial impulses to reach the ventricles each period. The electrophysiologic characteristics of APs appear to be influenced by age, autonomic stage, anatomic location, and pharmaceutical effect, and they differ greatly from person to person [3].

This case report focuses on the diagnosis and management of pre-excited atrial fibrillation in a patient with Wolff-Parkinson-White syndrome.

Clinical Presentation

A 72-year-old female presented, with palpitations, left-sided chest discomfort, shortness of breath, and dizziness for 16 hours. She described three similar episodes over past one week, each starting suddenly at rest, lasting for three to four minutes and subsiding spontaneously. There was no prior structural heart disease. On arrival, she had an irregular pulse of 170 beats per minute (bpm), blood pressure of 122/84 mm Hg, respiratory rate of 18 per minute, and spO2 of 98% on room air. She was conscious and oriented. Strong palpable pulses felt in all her limbs, no pedal oedema or jugular venous distension. The cardiovascular examination was notable for irregular tachycardia with a variable S1 without any added heart sounds. Lungs were clear to auscultation bilaterally and abdomen was soft, nontender, and nondistended.

The initial ECG obtained in the emergency revealed an irregular wide-complex tachycardia, absence of P waves, QRS complexes of varying amplitude and morphology and ventricular rate of up to 300 bpm. The upstroke of some QRS complexes appeared slurred (Figure 1).

Patient was diagnosed to have Wolff-parkinson-white syndrome with pre-excited atrial fibrillation.

Because of the initial interpretation of the rhythm as atrial fibrillation, the patient was given IV metoprolol 5mg for 2

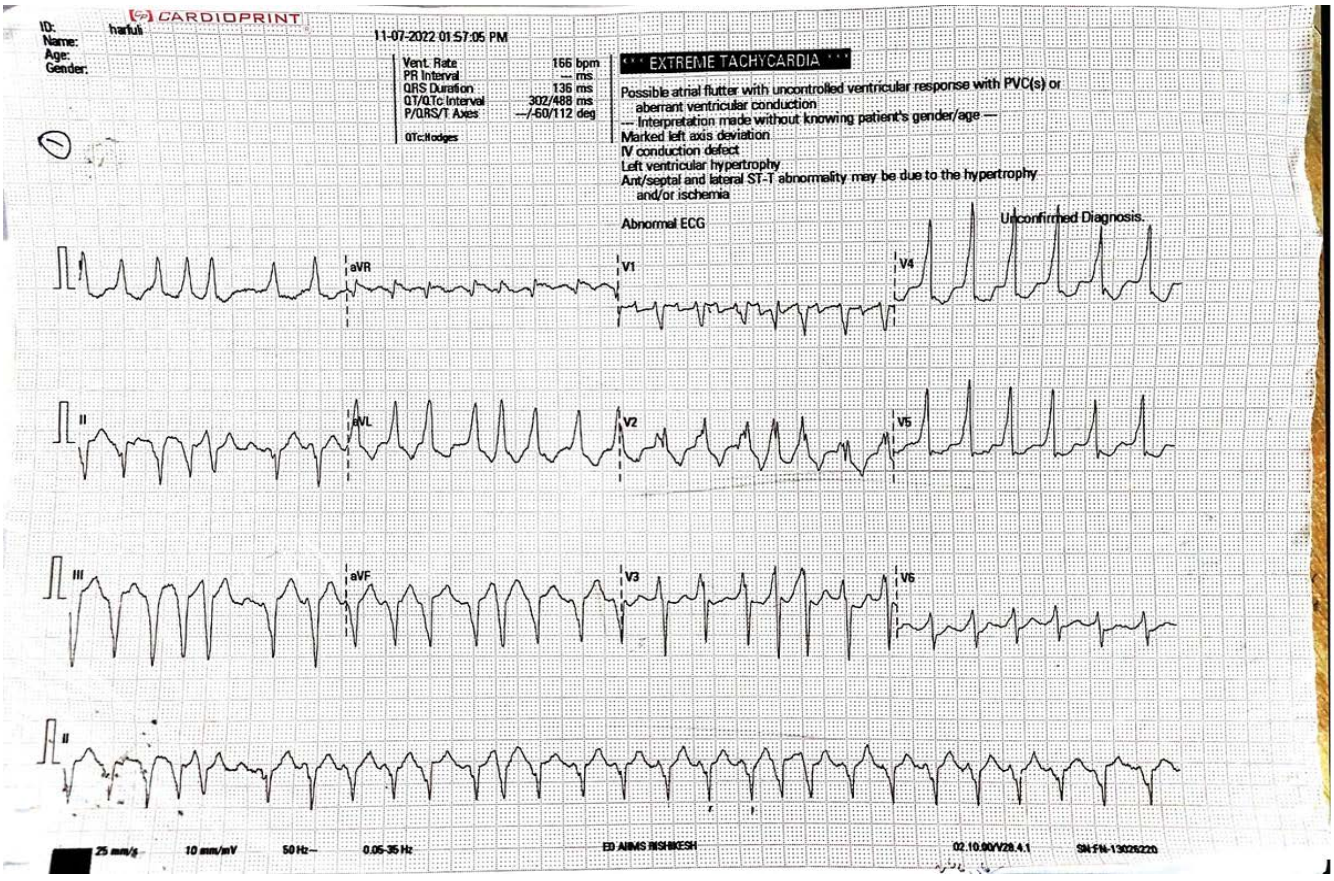

Figure 1: Initial ECG obtained at presentation.

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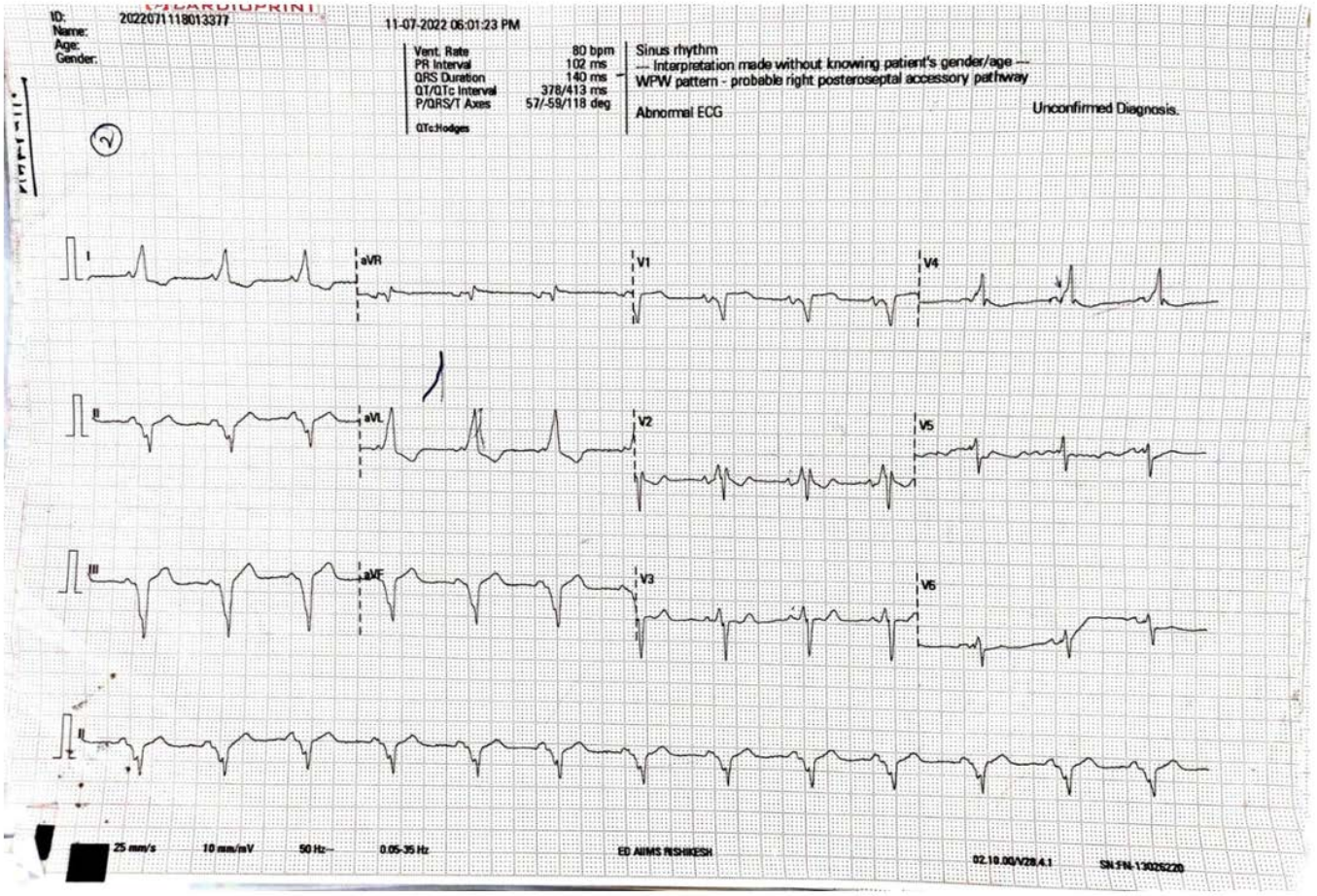

Figure 2: ECG after metoprolol showed normal sinus rhythm, heart rate of 84 bpm with the preexcitation pattern of positive delta waves in the anterolateral leads (I, aVL, and V4-6) and negative delta waves in inferior leads (II, III and aVF) and short PR interval of 102 milliseconds.

doses five minutes apart. The ECG after giving metoprolol showed normal sinus rhythm, heart rate of 84 bpm with the preexcitation pattern of positive delta waves in the anterolateral leads (I, aVL, and V4-6), negative delta waves in inferior leads (II, III and aVF) and short PR interval of 102 milliseconds. (Figure 2) Troponin I was negative. Formal 2D echocardiography revealed left ventricular ejection fraction of 55%, no valvular lesions, mild mitral regurgitation and trace tricuspid regurgitation. After 8 hours patient again developed palpitations and left sided chest discomfort, pulse rate was 154 bpm, irregularly irregular and a blood pressure of 116/82 mm of Hg in right arm supine position. ECG was suggestive of atrial fibrillation with fast ventricular rate (Figure 3). Due to non-availability of procainamide or ibutilide, we went ahead with synchronised DC cardioversion. Post cardioversion ECG showed normal sinus rhythm (Figure 4). Patient was followed-up in cardiology for radiofrequency ablation.

Discussion

Atrial fibrillation (AF) with an accessory pathway can present with confounding electrocardiographic findings, possibly leading to inaccurate diagnoses and treatment that may be fatal.

The atrial rate can increase to more than 300 impulses per minute during pre-excitation AF, concealing delta waves, which are the main electrocardiographic sign of Wolff-Parkinson-White (WPW) syndrome. The AV node often blocks the majority of these impulses because of decremental conduction, an innate repolarization feature that allows the node to conduct more slowly when it receives faster signals. An auxiliary pathway, on the other hand, permits 1:1 conduction at ventricular rates of about 300 bpm without having such a built-in delay [4,5].

Pre-excited AF should not be treated with AV nodal blockers (e.g., adenosine, calcium channel blockers, beta-blockers, and amiodarone) since atrial impulses would then preferentially conduct down the accessory pathway in an antidromic direction. As a result, the rhythm may continue to deteriorate and enter ventricular fibrillation, a rhythm that may be fatal. On the other hand, AV nodal blockers like adenosine or diltiazem are first-line treatments for patients with narrow QRS atrioventricular re-entrant tachycardia. In certain situations, they can stop the tachycardia by inhibiting the orthodromic re-entrant circuit and restore sinus rhythm [6,7].

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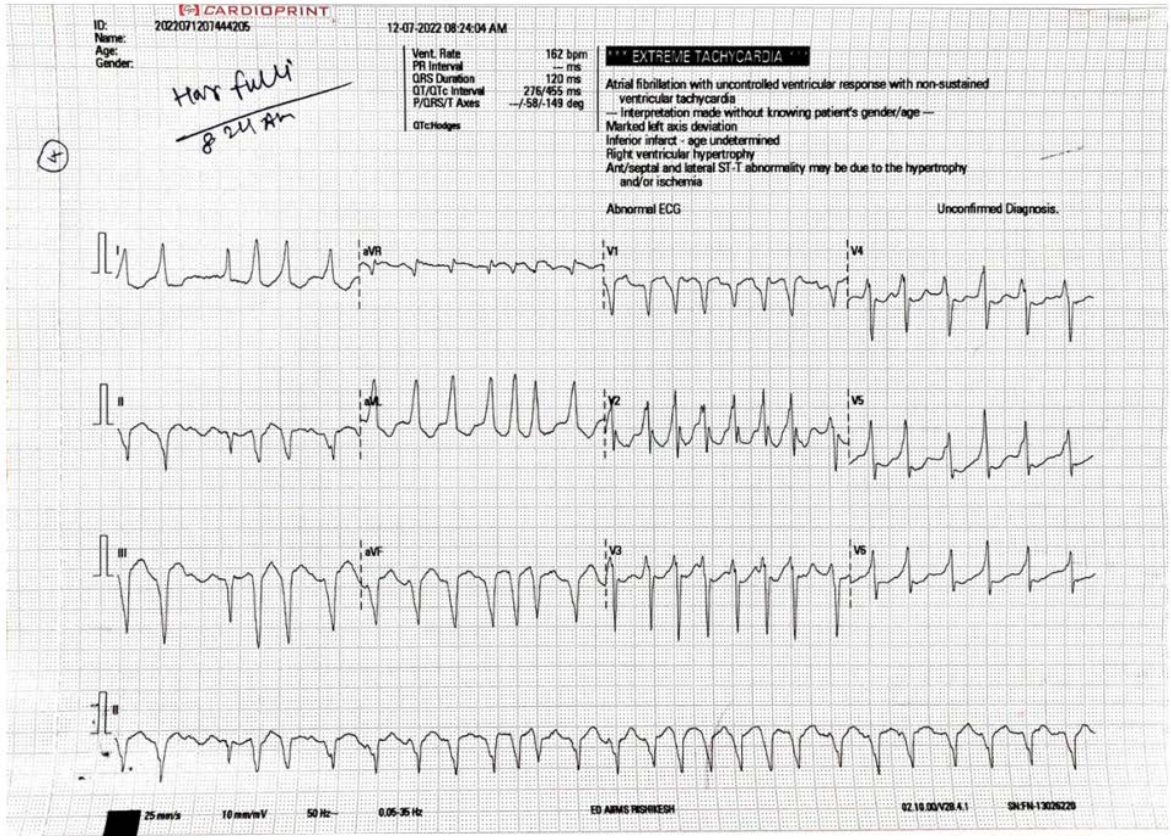

Figure 3: ECG after 8 hours was again suggestive of atrial fibrillation with fast ventricular rate.

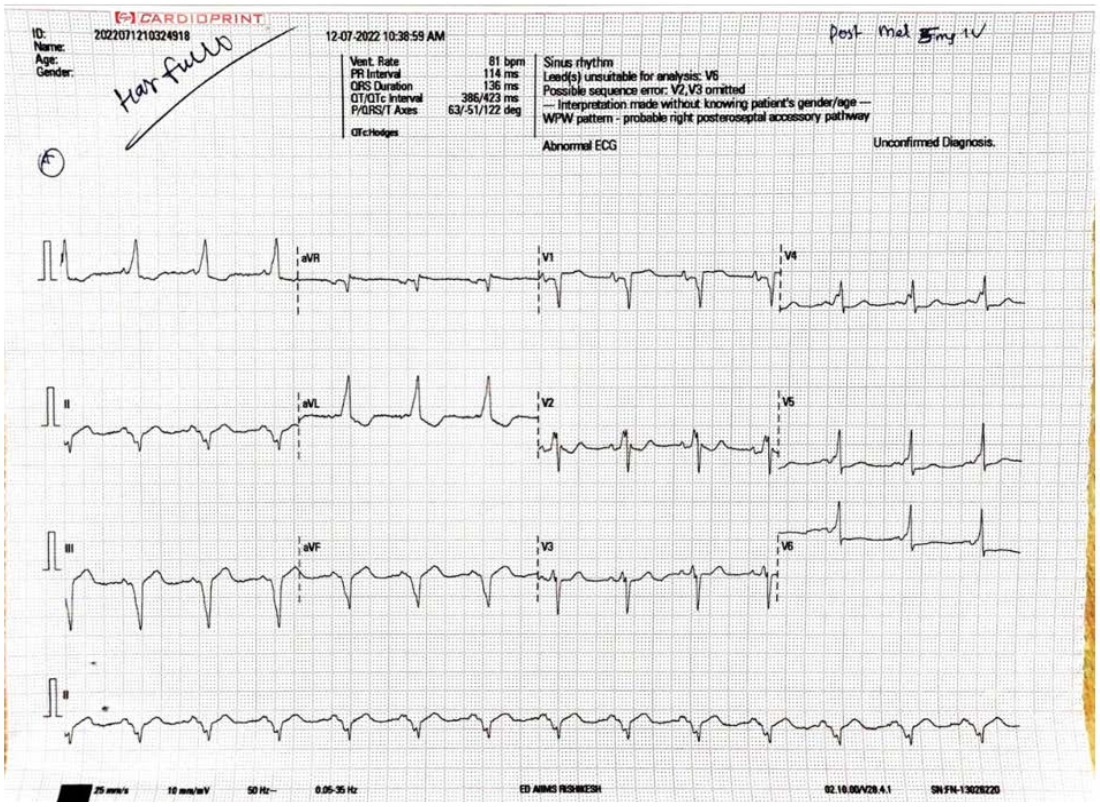

Figure 4: ECG post DC cardioversion showing sinus rhythm at 81 beats per minute.

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Thus, irregular wide complex tachycardia with QRS of varied shape and amplitude and sustained rates surpassing 200 bpm is the key to recognising WPW syndrome with pre-excited AF. Procainamide or ibutilide may be beneficial in reducing conduction velocity of the accessory pathway if the patient's blood pressure is normal. This rhythm can be difficult to distinguish from polymorphic ventricular tachycardia, although electrical cardioversion is the primary therapy for both in the setting of hemodynamic instability. Radiofrequency ablation is the definitive therapy for pre-excited AF in WPW syndrome for the prevention of recurrent arrhythmias [6,7].

Conclusion

A bizarre ECG with irregular wide complex tachycardia with QRS complexes of varied shape and amplitude, and sustained rates surpassing 200 bpm should be suspected as WPW syndrome with pre- excited AF. Although, it can difficult to distinguish from polymorphic ventricular tachycardia, electrical cardioversion is the primary therapy when hemodynamically unstable.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the Declaration of Helsinki. The collection and evaluation of patient's information was done with the approval and according to the guidelines of Institute Ethics Committee, All India Institute of Medical Sciences Rishikesh. Written informed consent was obtained from the patient's husband for collection, processing and publication of all the information collected for the case report.

Consent for publication: Informed consent was obtained from the patient's husband.

Availability of data and materials: Not Applicable

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