

WPW Syndrome – ECG Manifestations

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A 21-year-old male presented with history of recurrent episodes of palpitations that had sudden onset, lasted few minutes to hours and used to subside suddenly and spontaneously. The ECG during the episode has never been recorded. The patient is absolutely asymptomatic in between the episodes and his baseline 2D echocardiography is normal. The ECG recorded at baseline is shown in Figure 1. What is the ECG diagnosis?

The ECG shows regular sinus rhythm at 92/min with a wide QRS following each P-wave after a very short PR interval. The initial part of the QRS shows slurring known as the delta wave. These features are diagnostic of ventricular preexcitation due to an atrioventricular accessory pathway (AP) and result from earlier activation of some part of the ventricles (preexcitation) than that would have occurred by the AV node–His–Purkinje system. The interesting feature of this ECG is

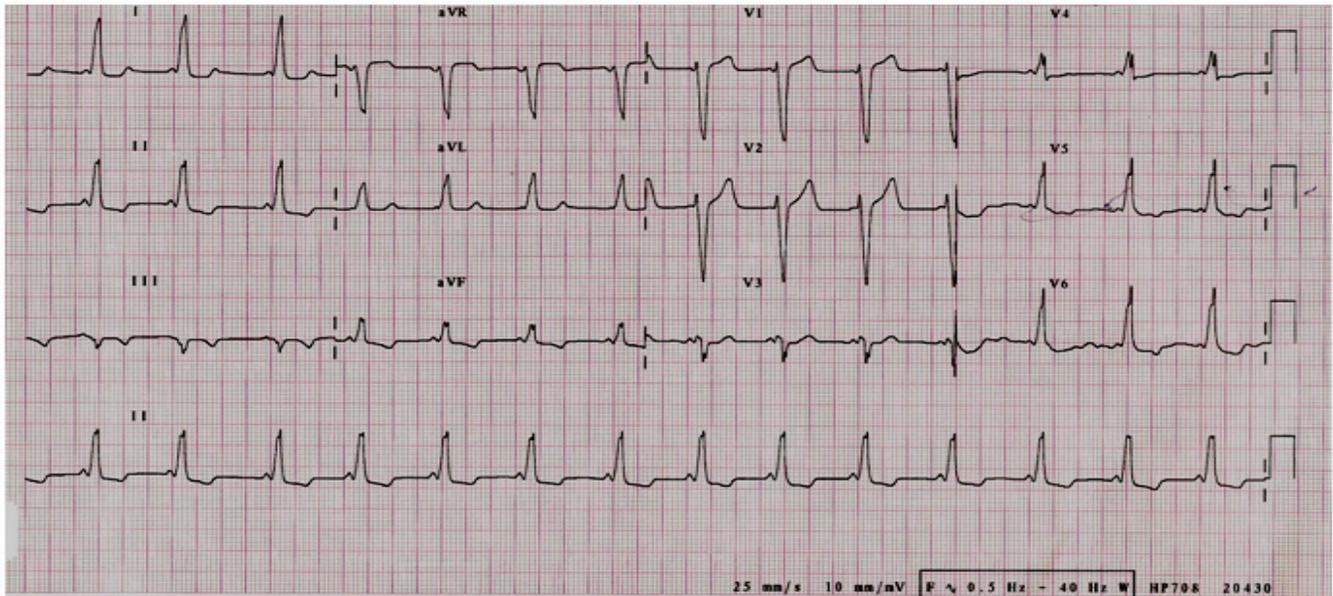


Figure 1. Twelve-lead ECG of the patient.

markedly short PR interval so much so that the P-wave itself is truncated. Hence, the ECG can be confused with junctional rhythm and isorhythmic AV dissociation. However, the presence of delta wave and a wide QRS complex clinches the diagnosis. A junctional beat will produce a narrow QRS complex provided there is no

bundle branch block. A junctional beat arises in the AV junction (below the atria) and hence, even in patients with WPW syndrome (atrioventricular AP), has narrow QRS complex since the AP has no contribution in activation of the ventricles.

The term Wolff-Parkinson-White (WPW) syndrome is used when tachyarrhythmia/s due to the AP are present in addition to the ventricular preexcitation. The patients who do not have symptoms of tachyarrhythmias but only show preexcitation pattern on the ECG should preferably be referred to as having asymptomatic ventricular

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preexcitation. These APs are usually present in patients with structurally normal hearts, though association with few heart diseases like Ebstein's anomaly, hypertrophic cardiomyopathy, and mitral valve prolapse also exists.

The atrioventricular APs, also known as Bundles of Kent, usually connect the atria and the ventricles at the mitral or tricuspid annulus. These provide an additional electrical connection between the atria and the ventricles apart from the normal AV node–His–Purkinje system and result in earlier depolarization of that part of the ventricle where they insert. In contrast to the AV node, these APs have the capability to conduct electrical impulses very rapidly either antegradely (from atrium to ventricle) or retrogradely (from ventricle to atrium) or in both directions. Most APs conduct in both directions and only antegradely conducting pathways are rare. The APs that conduct only retrogradely do not produce ventricular preexcitation and are known as *concealed APs*.

The degree of ventricular preexcitation during sinus rhythm depends on three factors (1):

1. Distance between the sinoatrial node and the atrial end of the AP – shorter the distance, more is the preexcitation.
2. Conduction time through the AV node–His–Purkinje

system – enhanced AV nodal conduction results in less preexcitation whereas slow AV nodal conduction produces more preexcitation.

3. Conduction time through the AP – slow conducting AP produce minimal preexcitation.

The preexcitation in cases of right free wall AP is marked since the atrial end of the AP is close to the sinus node and so the atrial impulse reaches quickly from the sinus node to the AP as in the present case. In contrast, in left free wall pathways, the AP lies in the lateral mitral annular region far away from the sinus node, resulting in very little or inapparent preexcitation.

Many algorithms have been proposed (2,3) to ascertain the location of the AP from the surface ECG based on the major delta wave and QRS forces in leads V1, V2, inferior leads and leads I and aVL. In general, a positive delta wave and QRS in lead V1 indicates a left-sided AP as also a negative QRS in lead I and aVL. On the other hand, a negative delta and QRS in lead V1 suggests a right-sided AP. Negative delta and QRS complexes in all three inferior leads (II, III and aVF) indicates a posteroseptal location of the AP. However, since the QRS during sinus rhythm in a patient with WPW syndrome is a fusion complex and is not totally preexcited, these predictions



Figure 2. Twelve-lead ECG showing narrow QRS tachycardia in initial half with P-waves in the ST segment seen best in leads III and aVL. The tachycardia terminates with VA block and sinus rhythm with ventricular preexcitation due to right free wall AP resumes in the latter half.

of AP localization are not always accurate. Furthermore, presence of multiple APs, conduction abnormalities and structural heart disease may render ECG localization of APs very difficult or almost impossible.

The arrhythmias that can occur in patients with WPW syndrome include the following:

1. Orthodromic reciprocating tachycardia (ORT). It is the most common type of arrhythmia in patients with WPW syndrome and presents as a type of PSVT. In this arrhythmia, the reentrant circuit travels from atria to ventricles over the normal AV node–His–Purkinje system and retrogradely from ventricles to the atria over the AP. Since the antegrade conduction is over normal His–Purkinje system, the ventricles are activated normally and hence it presents like a narrow QRS tachycardia (see Fig. 2). ORT can also occur in patients with concealed APs that have no preexcitation on baseline ECG.
2. Antidromic atrioventricular reentrant tachycardia (AVRT). It is a very uncommon form of arrhythmia wherein the reentrant circuit is reverse of ORT, that is, the antegrade conduction is over AP and retrograde conduction is over the AV node–His–Purkinje system. The ventricular activation in this tachycardia is sequential – the ventricle (left or right)

where the AP is inserted gets activated first followed by activation of the other ventricle. This sequential activation of the two ventricles results in wide QRS complexes during this arrhythmia that can be difficult to differentiate from ventricular tachycardia on ECG (Fig. 3). Fortunately, this comprises only 5% of all arrhythmias in patients with WPW syndrome.

3. Atrial fibrillation (AF). AF occurs more commonly in patients with WPW syndrome than those without APs, though AP itself does not have any direct role in initiation or maintenance of AF. However, when it occurs it can cause very rapid ventricular rates in WPW syndrome. The ECG shows a wide QRS tachycardia that is very fast, has broad complexes with varying QRS durations (normal narrow and with varying degrees of fusion) and is irregular. Rarely, due to very high ventricular rates, it can degenerate in to ventricular fibrillation and cause sudden death. Hence, patients with WPW syndrome carry a small but definite risk of sudden cardiac death (Fig. 4).

In addition to the above arrhythmias, a patient with WPW syndrome may have any other type of supraventricular tachyarrhythmias (like atrial flutter, atrial tachycardia, etc.) that in the presence of AP will present as a wide QRS preexcited tachycardia.

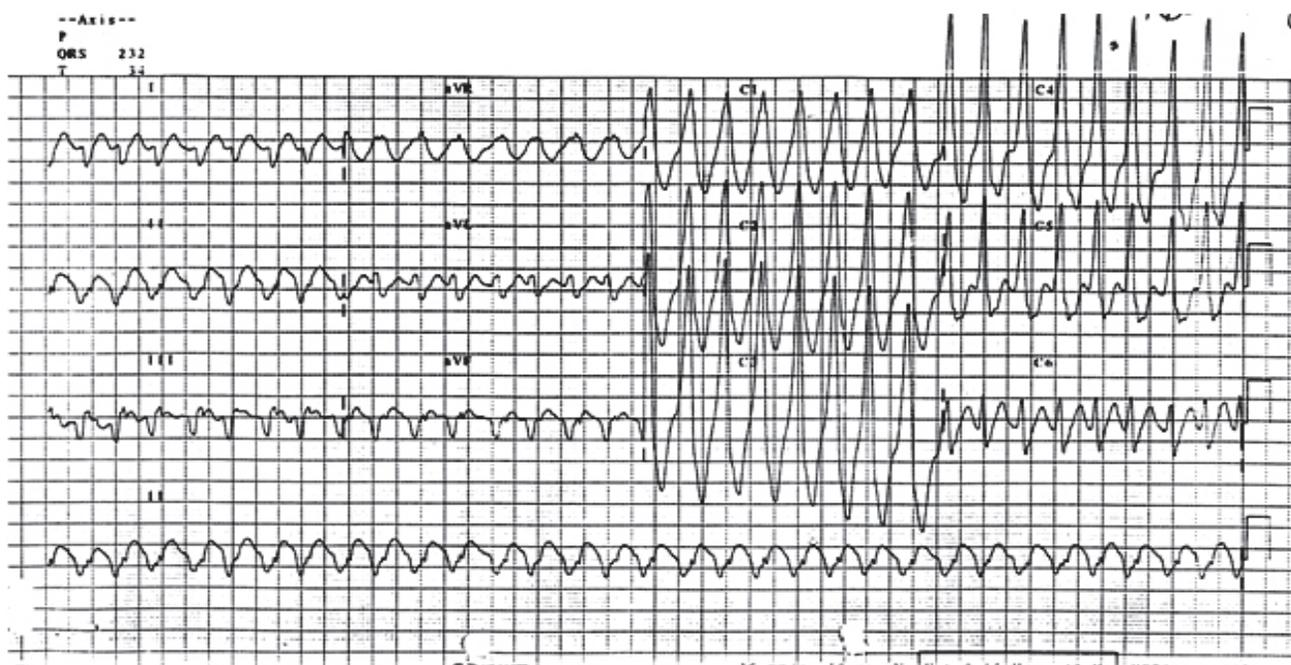


Figure 3. Wide QRS tachycardia due to antidromic AVRT in a patient with left free wall AP.

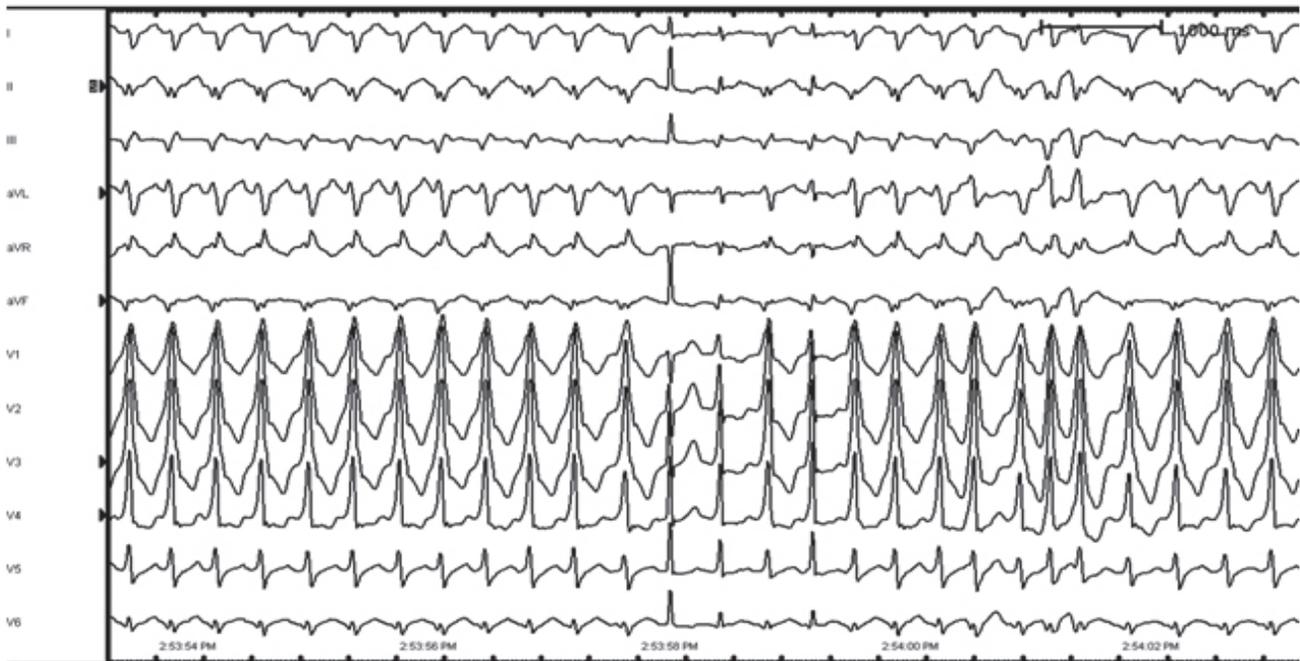


Figure 4. Preexcited atrial fibrillation in a patient with left free wall AP.

Conflicts of Interest:

None

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