

ECG Diagnosis: Wolff-Parkinson-White Syndrome

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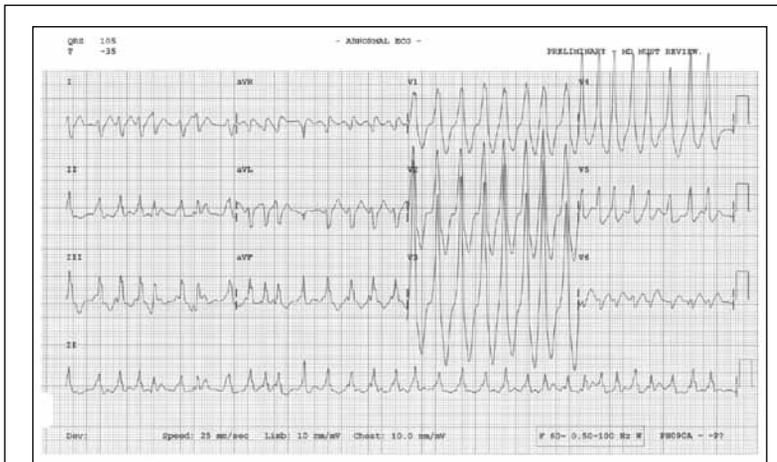


Figure 1. 12-lead ECG in a man, age 33 years, with palpitations, chest discomfort, and shortness of breath

Figure 1 demonstrates an irregular, rapid ventricular rate (rate 190) with wide QRS complexes and delta waves, consistent with atrial fibrillation in the Wolff-Parkinson-White syndrome.

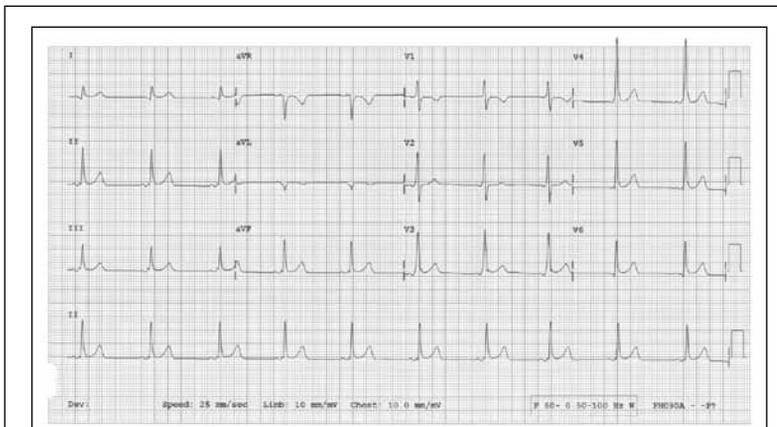


Figure 2. 12-lead ECG from same patient two days earlier

Figure 2 (same patient two days earlier) reveals findings consistent with Wolff-Parkinson-White syndrome: shortened PR interval, slurring and slow rise of the QRS complex (delta wave) and widening of the QRS complex.

Wolff-Parkinson-White Syndrome (WPWS) is defined as the presence of an accessory pathway (AP) and has a predisposition to the development of supraventricular tachyarrhythmias. Conduction over an AP circumvents conduction delay occurring within the atrioventricular node (AVN), which leads to early eccentric activation of the ventricles and fusion complexes.¹ If WPWS with atrial fibrillation (AF) is treated by drugs that prolong the AVN refractory period (eg, calcium-channel blockers, beta-blockers, digoxin, adenosine), the rate of conduction through the AP may increase and degenerate to ventricular fibrillation (VF).² Unstable patients with WPWS and AF should receive immediate electrical cardioversion.³ Stable patients can be chemically cardioverted with IV procainamide. Amiodarone should be used with caution due to its ability to cause ventricular rate acceleration and degeneration into VF.³ Ibutilide is considered an alternative agent, although it has numerous side effects.¹ Cardiology or electrophysiology consultation with consideration for radiofrequency mapping and ablation should occur for patients presenting with AF in the setting of WPWS. ♦

References

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