
Atrial fibrillation with Wolff-Parkinson-White syndrome

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Citation

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Abstract

The management of atrial fibrillation (AF) with Wolff-Parkinson-White syndrome (WPWS) is complex and can result in significant morbidity and mortality if not correctly recognized and treated appropriately. We report the case of a 25-year-old male who presented to the Emergency Department (ED) with palpitations; his ECG revealed WPWS with AF. He underwent successful synchronized cardioversion and subsequent catheter ablation of the accessory bypass tract.

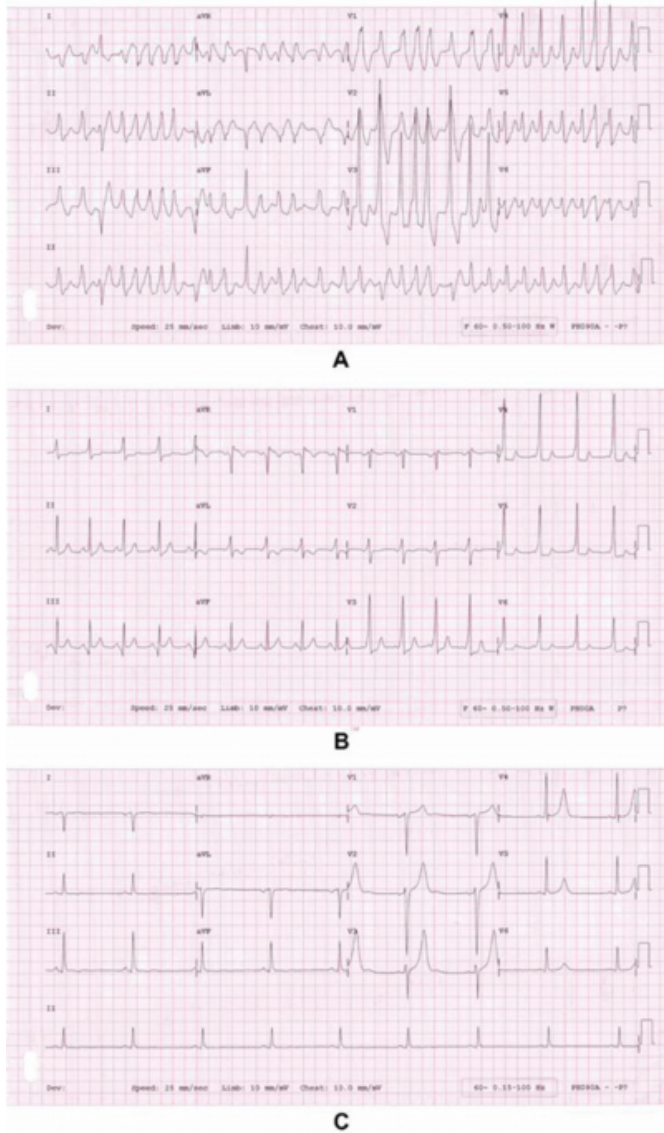
CASE REPORT

A 25-year-old male presented to the emergency department complaining of palpitations associated with chest pressure, shortness of breath and sweating. He was in no acute distress, but had a rapid, irregular pulse. Vital signs revealed a pulse of 207 beats/minute with a blood pressure of 110/70 mm Hg. A 12-lead ECG (Figure, panel A) demonstrated an irregular, wide QRS complex tachycardia, with delta waves (leads V₁-V₄). These findings were consistent with atrial

fibrillation (AF) in the setting of Wolff-Parkinson-White syndrome (WPWS). He was considered to be relatively stable, so underwent synchronized cardioversion following procedural sedation. A repeat ECG (Figure, panel B) demonstrated a normal sinus rhythm, shortened PR intervals, delta waves, and minimally widened QRS complexes consistent with WPWS. He was admitted to the hospital and underwent catheter ablation of an accessory atrioventricular (AV) bypass tract. A post-ablation ECG demonstrated resolution of the delta waves (Figure, panel C).

Figure 1

Figure: 12-lead ECGs from a 25-year-old male with palpitations demonstrating WPWS with AF (panel A), following synchronized cardioversion with conversion to normal sinus rhythm (panel B), and after catheter ablation for WPWS (panel C)



DISCUSSION

WPWS was first described in 1930 by Louis Wolff, Sir John Parkinson, and Paul Dudley White. ¹ WPWS is defined by the presence of an accessory pathway (AP) and a predisposition to the development of supraventricular tachyarrhythmias. ² 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. ³ Electrocardiographic

findings in WPWS include a shortened PR interval (less than 0.12 seconds), slurring and slow rise of the QRS complex (delta wave), a widened QRS complex (greater than 0.12 seconds), and ST-T wave changes generally directed opposite the QRS complex. ^{2,4} Although the incidence of dysrhythmias in WPWS is rare, approximately 80% of dysrhythmias are AV reentrant tachycardias; 15-30% atrial fibrillation (AF) and 5% atrial flutter. ⁵ Clues in the ECG suggesting WPWS and AF include rhythm irregularity, a rapid ventricular response (often with R-R intervals approaching 300 beats/min), and wide, bizarre QRS complexes. ²⁴

The goal of treating WPWS with AF is to restore hemodynamic stability by prolonging the anterograde refractory period of the AP relative to the AVN. If WPWS with AF is treated by drugs that prolong the AVN refractory period (e.g., calcium-channel blockers, beta-blockers, digoxin, adenosine), the rate of conduction through the AP may increase, which may degenerate to ventricular fibrillation (VF). ⁴ Unstable patients with WPWS and AF should receive immediate electrical cardioversion. ² Stable patients can be chemically cardioverted with IV procainamide (17 mg/kg, not to exceed 1000 mg or 30 mg/min). ⁶ Long considered second-line in the treatment of WPWS with AF ⁵, amiodarone should be used with caution due to its ability to cause ventricular rate acceleration and degeneration into VF in these patients. ² Ibutilide is considered an alternative agent (1 mg over 10 minutes), although it has numerous side effects. ⁶ Cardiology consultation with consideration for radiofrequency mapping and ablation should occur for patients presenting to the ED with AF in the setting of WPWS.

References

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