Medical Image of the Week: Wolff-Parkinson-White Syndrome

Figure 1. Presenting EKG with supraventricular tachycardia at rate of 232.

Figure 2. Post-conversion EKG demonstrating a short PR interval, slurring of the initial QRS upstroke (delta wave), widened QRS, and ST-T repolarization change; characteristic of Wolff-Parkinson-White Syndrome.

A 38-year-old man developed sustained rapid heart rate while rock climbing. The patient reported that he had experienced rare bouts of self-limited palpitations in the past. Blood pressure on arrival to the emergency department was 112/ 65 mm Hg. The patient's initial EKG demonstrated a regular, narrow complex supraventricular tachycardia, with a rate of 232 (Figure 1). Intravenous adenosine was administered with no change in his rate or rhythm. The patient then received amiodarone by intravenous bolus, with subsequent conversion to sinus rhythm (Figure 2).

Wolff-Parkinson-White (WPW) syndrome is a congenital cardiac condition present in approximately 0.15% of the general population. WPW is characterized by the abnormal presence of conduction tissue that creates an accessory atrioventricular pathway and thus potentiates reentrant tachycardia (1). The classic resting EKG findings in WPW are: a shortened PR interval (less than 0.12 seconds), an indistinct initial upslope of the QRS complex (known as the *delta* wave), a widened QRS complex (0.12 seconds or greater), and ST-T repolarization changes (2). In WPW presenting as a narrow complex tachycardia without hypotension, the initial treatment is adenosine or a calcium channel blocker, followed by amiodarone if unsuccessful. If the presenting rhythm is atrial fibrillation, atrial flutter, or an undefined wide complex tachycardia without hypotension, amiodarone is used. A hemodynamically unstable rhythm warrants immediate electrical cardioversion. Definitive evaluation and treatment of WPW requires electrophysiologic mapping and subsequent ablation of the accessory pathway.

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References

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