Wide QRS Rhythm in a Young Woman with Recurrent Palpitations: What is the Diagnosis?

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Case Representation

An 18-year-old woman was referred for electrophysiologic evaluation of two episodes of rapid regular palpitations that lasted about 1 hour. There was no clinical or echocardiographic evidence of heart disease. Twelve-lead ECG showed a PR interval of 0.12 second, with normal QRS complexes and no delta waves. The tracing shown in Figure 1 was recorded when the patient was lying down at the beginning of the electrophysiologic study, before vascular puncture to introduce electrode catheters into the heart. A regular rhythm at 77/min consisting of a succession of seven wide QRS complexes was recorded following a sinus rhythm at 94/min. The QRS complexes had a left bundle branch block (LBBB) morphology and left-axis deviation.

Figure 1. Twelve-lead ECG tracing recorded during electrophysiologic study before vascular puncture. A stable sinus rhythm (94/min) with normal PR (0.12 s) and normal QRS complexes is followed by an escape rhythm consisting of seven wide QRS complexes with a left bundle branch block-left axis pattern at a rate of 77/min. Note a fusion beat (*) on the next QRS complex before stable sinus rhythm (86/min) resumes.
Figure 2. Twelve-lead ECG tracing recorded about 15 seconds after intravenous bolus administration of 10 mg adenosine triphosphate. After a short sinus rate slowing to 56/min, sinus rate increases and a succession of eight wide QRS conducted beats (79–94/min) is observed. Note the longer PR interval (150 ms) preceding the wide QRS complexes. Also note a fusion beat (*) on the next complex before sinus rhythm with normal PR interval and QRS complexes resumes.

Figure 3. Electrophysiologic recording during right atrial pacing. Shown are ECG leads I, II, III, and V1, as well as intracardiac recordings from the high right atrium (RA), and the distal (Hisd) and proximal (Hisp) His bundle. At a pacing cycle length of 600 ms, a sudden widening of the QRS complex is observed along with prolongation of the stimulus-R interval by 30 ms and shortening of the HV interval from 50 to –20 ms.
Adenosine triphosphate (ATP) test (10 mg injected IV as a rapid bolus before catheters introduction) was performed in an attempt to noninvasively diagnose the mechanism of the palpitations. The tracing obtained during the ATP test is shown in Figure 2. What is the diagnosis of this wide QRS rhythm?

Commentary

There are three possible diagnoses to explain this wide QRS rhythm: (1) a junctional escape rhythm with a phase 4 dependent LBBB; (2) a ventricular escape rhythm originating in the right ventricle; or (3) an escape rhythm originating in a right-sided accessory pathway. The first of these diagnoses seems the less likely due to the presence of a fusion beat observed after the seven beats of the wide QRS rhythm. The ATP test enables further discrimination between the two remaining diagnoses. Following drug injection, QRS complexes identical to those shown in Figure 1 and preceded by sinus P waves with constant PR interval (0.15 s) are transiently observed. Such a finding does not suggest a ventricular origin of the wide QRS rhythm, unless there is perfect synchronization between sinus node activity and a dissociated ventricular rhythm. However, the whole ECG picture is compatible with the existence of a right-sided accessory pathway, latent in sinus rhythm, which becomes manifest during escape activity originating from the pathway (Fig. 1) as well as after ATP-induced AV nodal blockade (Fig. 2). The fact that the right-sided accessory pathway is latent in sinus rhythm and manifests after ATP injection following a slight prolongation in PR interval suggests either an atriofascicular pathway with a long conduction time or a nodoventricular pathway rather than a typical AV pathway. Likewise, an AV accessory pathway with a long conduction time seems unlikely due to the configuration of the wide QRS rhythm, which is that of a typical LBBB and not typical right ventricular preexcitation.

The electrophysiologic study suggested the presence of an atriofascicular pathway with a long conduction time. During high right atrial pacing at a cycle length of 600 ms, a sudden widening of the QRS complex was observed along with prolongation of the stimulus-R interval by 30 ms and shortening of the HV interval from 50 to ~20 ms (Fig. 3). Further increase in the atrial pacing rate resulted in progressive increase of stimulus-R intervals while the QRS complexes during maximal preexcitation remained identical to those observed during the spontaneous wide QRS rhythm and the ATP test. Anterograde block in the atriofascicular pathway was achieved at an atrial pacing cycle length of 250 ms. Non-sustained episodes of AV reentrant antidromic tachycardia (cycle length 280 ms) lasting <30 seconds and involving the accessory pathway in the anterograde direction and the AV node in the retrograde direction were reproducibly induced (not shown). An accessory pathway potential was recorded at the posterolateral part of the tricuspid annulus during both sinus rhythm and short-lasting escape rhythms (75/min) with a morphology identical to the wide QRS rhythm recorded at baseline (Fig. 4). This potential occurred 40 ms before each of the wide QRS complexes. Although one cannot definitely exclude that this ectopic rhythm resulted from catheter-induced trauma to the pathway, it is highly likely that this was the mechanism of the arrhythmia observed in our patient at baseline. A single radiofrequency pulse administered at this site resulted in successful ablation of the pathway. Repeat ATP test after ablation showed transient AV nodal block with no conduction over the accessory pathway or suspicion of dual AV nodal physiology.

References