Circulation

CASES AND TRACES

Wide QRS Complex Tachycardia

What the Algorithms Fear

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ECG CHALLENGE

A 70-year-old man was admitted to the emergency department for several hours of palpitations, dizziness, and dyspnea. The patient had a history of hypertension, diabetes mellitus, dyslipidemia, chronic obstructive pulmonary disease, and paroxysmal atrial fibrillation. His medications included eprosartan, amlodipine, atorvastatin, sitagliptin, indacaterol, and acenocoumarol. On arrival, his heart rate was 170 bpm and blood pressure was 110/50 mm Hg. The following 12-lead ECG was obtained (Figure 1).

Based on the ECG, what is the most likely diagnosis?

Please turn the page to read the diagnosis.

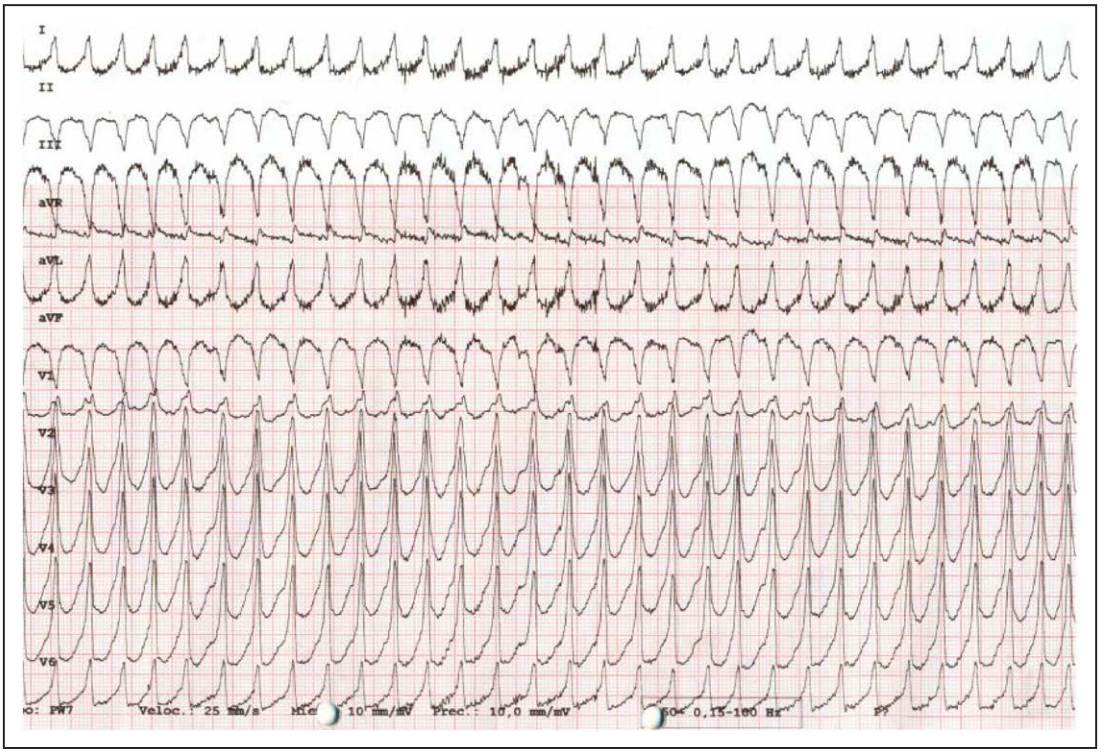

Figure 1. ECG obtained at admission in the emergency department.

RESPONSE TO ECG CHALLENGE

The ECG in Figure 1 shows a wide QRS complex tachycardia at 170 bpm. Wide QRS complex tachycardia can be originated by 3 main mechanisms[1]:

1. Ventricular tachycardia (VT).
2. Supraventricular tachycardia (SVT) with an aberrant conduction attributable to a preexisting bundle-branch block or functional bundle-branch block induced by the fast heart rate.
3. SVT with an atrioventricular conduction over an accessory pathway.

The ECG at admission showed several criteria used in common algorithms to diagnose VT: positive concordance with a lack of RS complexes in V_1 to V_6 leads, initial R wave in V_1 and in aVR leads, Q wave nadir time >50 ms in lead II, initial and terminal ventricular activation velocity ratio <1. In our patient, a clinical history of bronchospasm limited the use of adenosine; however, Valsalva maneuvers had no effect on the tachycardia. For all these reasons, our most probable diagnosis was VT, and an infusion of procainamide was started. A second ECG was performed after a few minutes (Figure 2). Ventricular activity was alternating wide QRS complexes and narrow complexes, at 170 bpm and at 85 bpm, respectively; the wide QRS complexes were similar to those of the ECG at admission. Moreover, a regular atrial activity at 340 bpm was detectable. These findings were consistent with an atrial flutter intermittently conducted to the ventricle through an accessory pathway and the atrioventricular node, with a 2:1 and a 4:1 atrioventricular conduction, respectively. The following day, after a washout period of procainamide, a preexcited sinus rhythm was documented in a new ECG (Figure 3). During the stay, the patient underwent an electrophysiology study in which a posterior left-sided accessory pathway was located and ablated successfully.

There are numerous algorithms and criteria for the diagnosis of VT.[2] However, as shown in our case, SVT with anterograde conduction via an accessory pathway shares many electrocardiographic characteristics with VT. It is difficult to distinguish VT from preexcited SVT because, in both cases, ventricular activation begins outside the normal conduction system. Algorithms to differentiate VT and SVT have a high sensitivity, but in general, their validation studies excluded patients with accessory pathways. Accordingly, despite the usefulness of these algorithms, to reach the right diagnosis, it is mandatory to search for preexcitation features in the sinus rhythm ECG.

Guidelines suggest procainamide and amiodarone as agents that could be used for the acute therapy for stable VT. These drugs can be effective both for the treatment of VT and for preexcited SVT. However, in sustained monomorphic wide QRS complex tachycardia, procainamide showed a higher proportion of tachycardia termination and less hypotension than amiodarone.[3] Moreover, the elimination half time of amiodarone is much longer than that of procainamide (up to 180 days versus up to 4 hours, respectively), and this may limit the prompt detection of preexcited signs when sinus rhythm is restored.

Our case remarks the limitation of ECG algorithms to differentiate VT and preexcited SVT. These algorithms may orient the initial diagnosis, but an accessory pathway

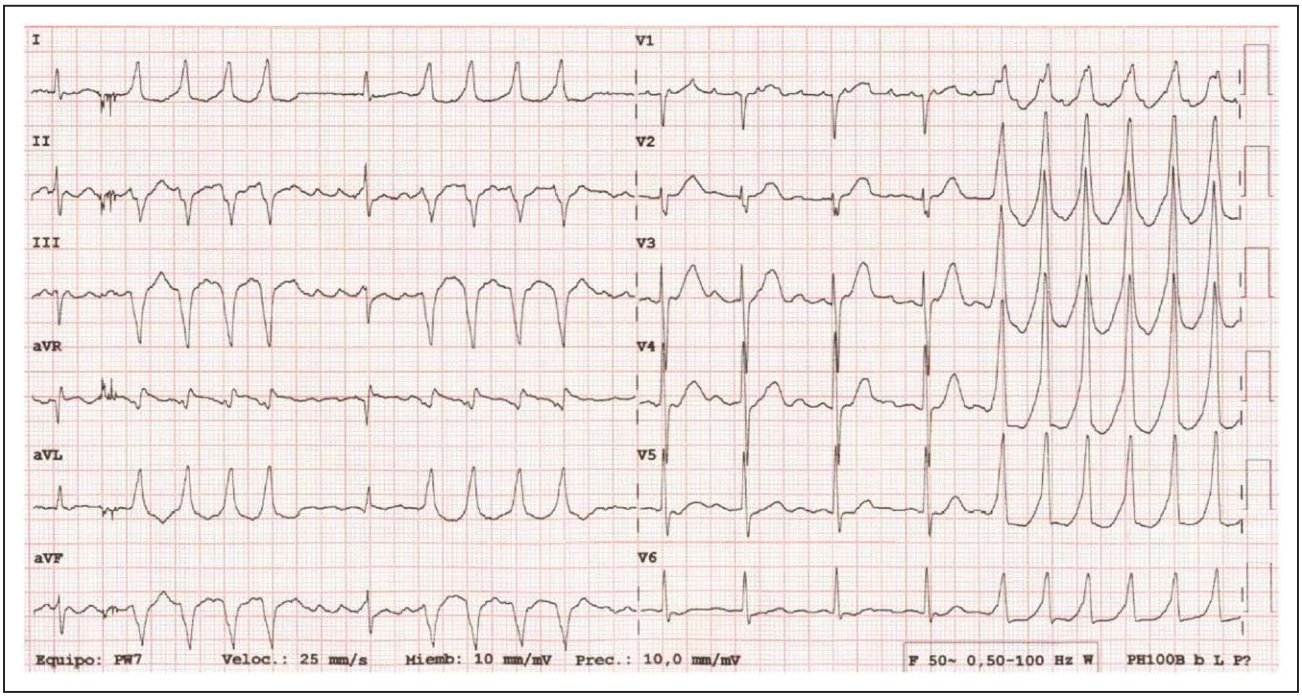

Figure 2. ECG at the beginning of intravenous procainamide administration.

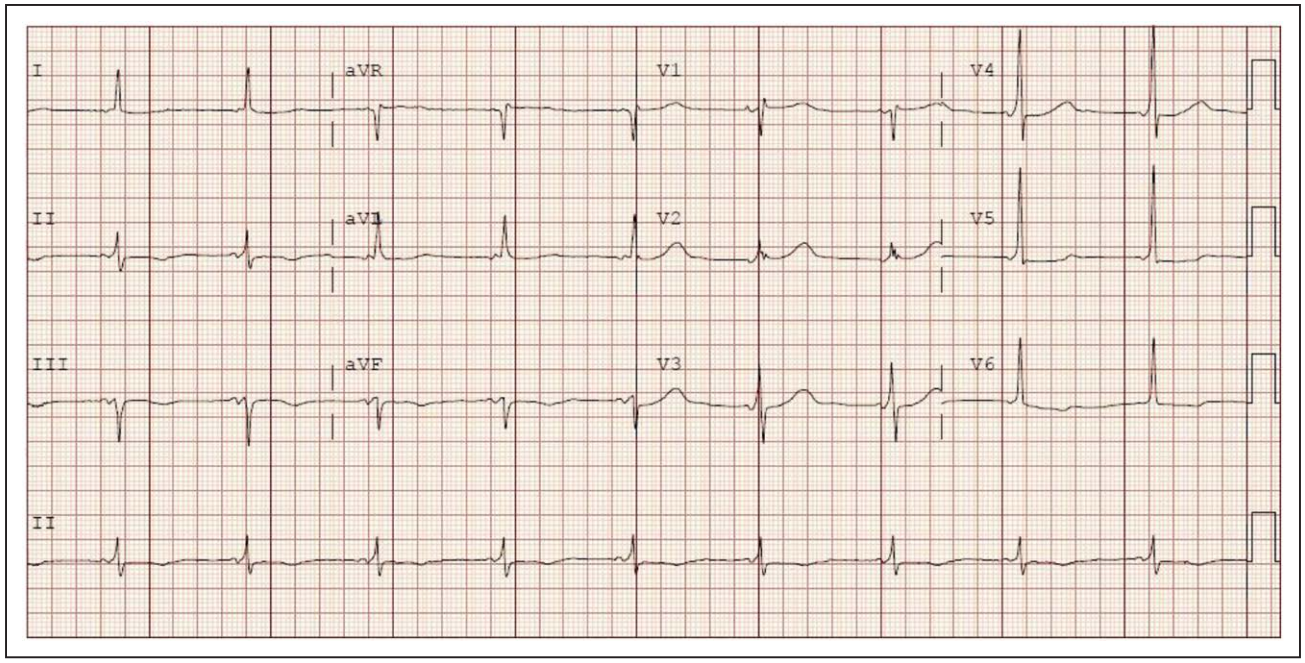

Figure 3. ECG in sinus rhythm with preexcitation.
Short PR interval and delta waves are observed.

should always be ruled out by the analysis of sinus rhythm ECG after a washout period of antiarrhythmic drugs.

Disclosures

None.

ARTICLE INFORMATION

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