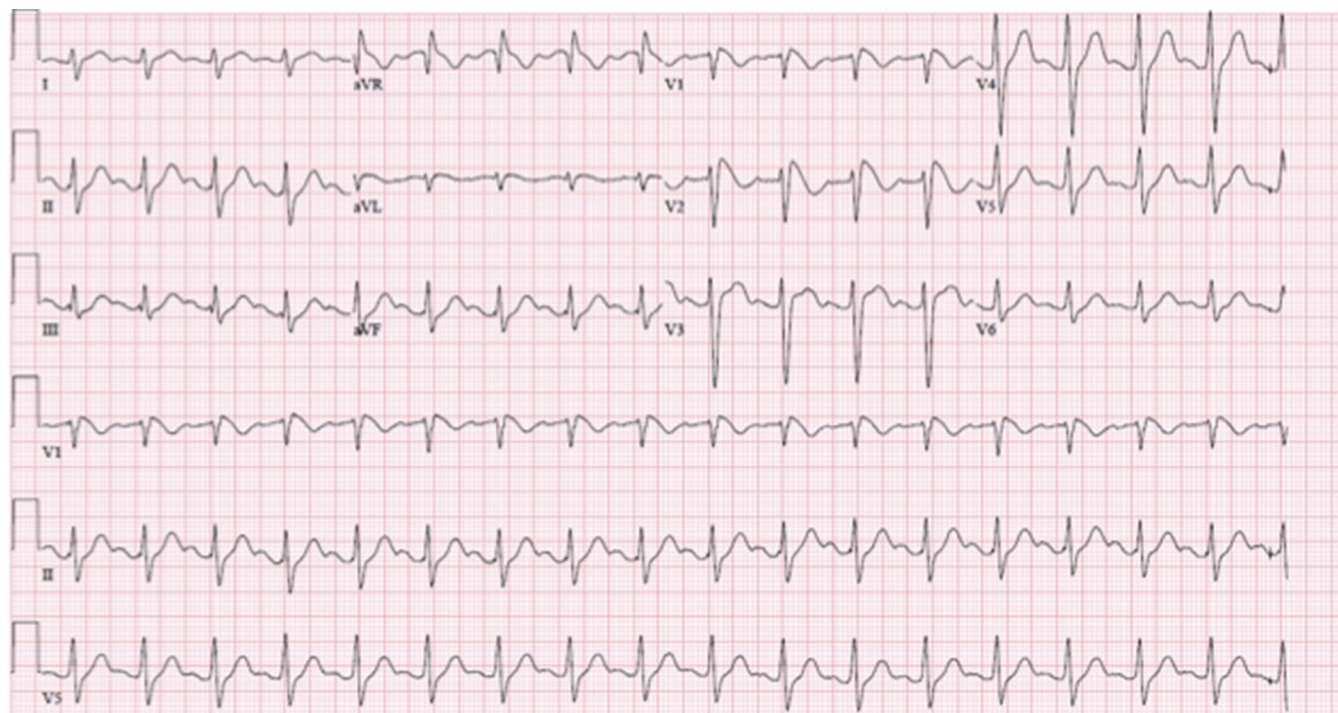




0196-0644/\$-see front matter

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**Figure 1.** Initial electrocardiogram.

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## CASE PRESENTATION

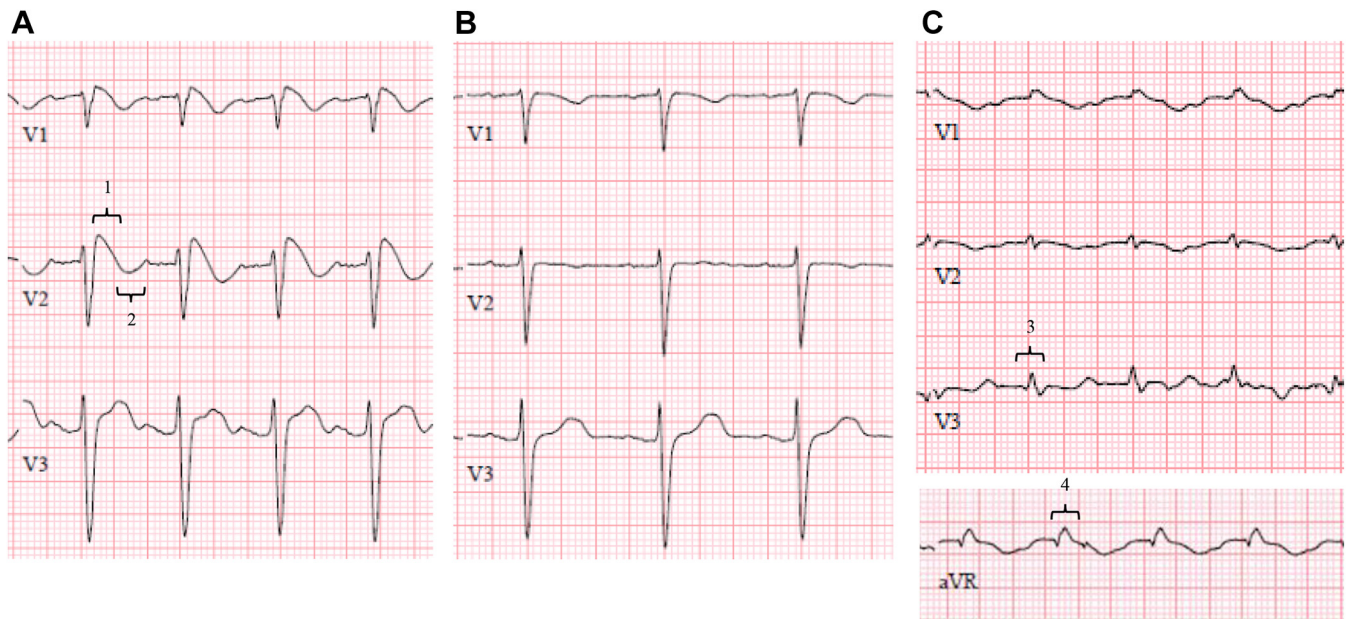
A 47-year-old white man was brought to the emergency department (ED) by ambulance after being found unresponsive by his wife. He was accompanied by empty bottles of amitriptyline and cyclobenzaprine.

On physical examination, the patient's vital signs were remarkable for a blood pressure of 94/55 mm HG, pulse rate of 105 beats/min, respiratory rate of 18 breaths/min, and temperature of 35.7°C (96.3°F). Physical examination revealed an obtunded mental status, normal cardiopulmonary examination, and dry skin. Laboratory study results were normal: glucose 117 mg/dL; negative salicylate, acetaminophen, and ethanol concentrations; and a negative urine toxicology screen. An initial ECG (Figure 1) was obtained.

What is the diagnosis?

*For the diagnosis and teaching points, see page 560.*

*To view the entire collection of ECG of the Month, visit [www.annemergmed.com](http://www.annemergmed.com)*



**Figure 2.** A, Initial ECG demonstrating a type 1 Brugada pattern. This pattern is defined by <sup>1</sup>coved ST-segment elevation greater than or equal to 2 mm, followed by <sup>2</sup>T-wave inversion in greater than 1 of the right precordial leads (V1 to V3). B, Repeat ECG obtained after 300 mEq total of sodium bicarbonate. Note the disappearance of the coved ST segments in V1 and V2, as well as subtle QRS-interval shortening. C, Representative ECG from tricyclic antidepressant overdose without induced type 1 Brugada pattern. This ECG was obtained from a separate patient also presenting with tricyclic antidepressant overdose. Note the sinus tachycardia, <sup>3</sup>QRS prolongation, and <sup>4</sup>prominent R wave in aVR that are typical ECG features of tricyclic antidepressant overdose. Compared with B, this ECG does not demonstrate coved ST-segment elevation followed by T-wave inversion in the right precordial leads, which are typical of the type 1 Brugada pattern.

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## PEARLS

In young patients presenting with syncope, chest pain, palpitations, or exercise intolerance, the diagnosis of hypertrophic cardiomyopathy should be considered.

ECG findings suggestive of hypertrophic cardiomyopathy include T-wave inversions, ST-segment depressions, pathologic Q waves (typically in the inferior or lateral leads), conduction disturbances, left ventricular hypertrophy, left atrial enlargement, and rhythm abnormalities.

When the diagnosis is considered according to ECG findings and history, transthoracic echocardiogram with Doppler is the key diagnostic test for assessment of hemodynamic abnormalities.

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## ECG OF THE MONTH

*(continued from p. 552)*

### ECG FINDINGS

Figure 1 demonstrates sinus tachycardia, right axis deviation, a high-amplitude R wave in aVR, and QRS-interval widening, typical features of tricyclic antidepressant overdose. Figure 1 also demonstrates coved ST-segment elevation in V1 and V2, consistent with a type 1 Brugada ECG pattern (magnified in Figure 2A).

### CLINICAL COURSE

The patient was intubated for airway protection. After the first ECG was obtained, multiple boluses of sodium bicarbonate were administered to treat QRS-interval widening in tricyclic antidepressant overdose. Continuous telemetry demonstrated stepwise narrowing of the QRS interval with each successive sodium bicarbonate bolus and resolution of the type 1 Brugada pattern, demonstrated on a subsequent ECG (Figure 2B). The patient was admitted to the ICU and eventually endorsed overdosing on both amitriptyline and cyclobenzaprine in a suicide attempt.

## REVIEW OF CRITICAL TEACHING POINTS

Tricyclic antidepressant overdose results in sodium channel blockade, producing the classic ECG findings of right axis deviation and QRS-interval prolongation shown in Figure 1. Sinus tachycardia is also typical of tricyclic antidepressant overdose because of muscarinic blockade. Cyclobenzaprine is structurally similar to amitriptyline and exhibits a similar toxidrome in overdose.<sup>1</sup>

In addition to the classic ECG findings of sodium channel blocker overdose (Figure 2C), there is also a pronounced type 1 Brugada pattern in leads V1 and V2, characterized by downward-sloping ST-segment elevation and T-wave inversion. Although ST-segment changes have been reported in sodium channel blocker overdose,<sup>2</sup> the ST segments demonstrated here are more typical of type 1 Brugada pattern because of their coved downward-sloping appearance. Despite the appearance of a type 1 Brugada ECG pattern, this patient's clinical presentation was most consistent with tricyclic antidepressant overdose, and management proceeded accordingly.

The Brugada syndrome is associated with sudden cardiac death in young and otherwise healthy adults because of inherited dysfunction in the myocardial sodium channel and related proteins.<sup>3-5</sup> Type 1A and 1C antiarrhythmics are known to unmask type 1 Brugada ECG pattern in patients with underlying Brugada syndrome and are used diagnostically in electrophysiology studies. A number of other agents known to block sodium channels (eg, tricyclic antidepressants, tramadol, propranolol, diphenhydramine, cocaine) have been reported to produce a Brugada ECG pattern that resolves after administration of sodium bicarbonate.<sup>6,7</sup> Inducible Brugada ECG patterns have also been reported in the setting of fever, thyroid storm, and profound electrolyte derangement, such as hyponatremia and hyperkalemia.

The incidence of Brugada ECG pattern after tricyclic antidepressant overdose is rare, with 1 study finding that 2% of patients presenting with tricyclic antidepressant ingestion developed Brugada ECG pattern. This study did find an increased risk of adverse outcomes in patients with ingestion and Brugada pattern, such as seizure and hypotension, although no tricyclic antidepressant overdose-associated Brugada ECG pattern resulted in death or dysrhythmia.<sup>6</sup> Although it remains unclear whether tricyclic antidepressant-induced Brugada ECG pattern indicates underlying Brugada syndrome, these ECG findings should prompt ED providers to perform a thorough evaluation of the presenting events and relevant family history, as well as seek consultation with an electrophysiologist to determine the need for further evaluation.

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