



# A Revealing Form of Injury

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## Vignette

A 34-year-old man with a history of depressive illness is brought to the emergency department because of ingestion of an unknown quantity of amitriptyline. On initial assessment, he is drowsy, but co-operative and denies chest pain or any other cardiac symptoms. His cardiovascular exam, with the exception of an intermittent sinus tachycardia, is normal. An electrocardiogram (ECG) is obtained (Figure 1).

## Questions

- 1. What ECG abnormality is shown?**
- 2. What is the likely diagnosis?**

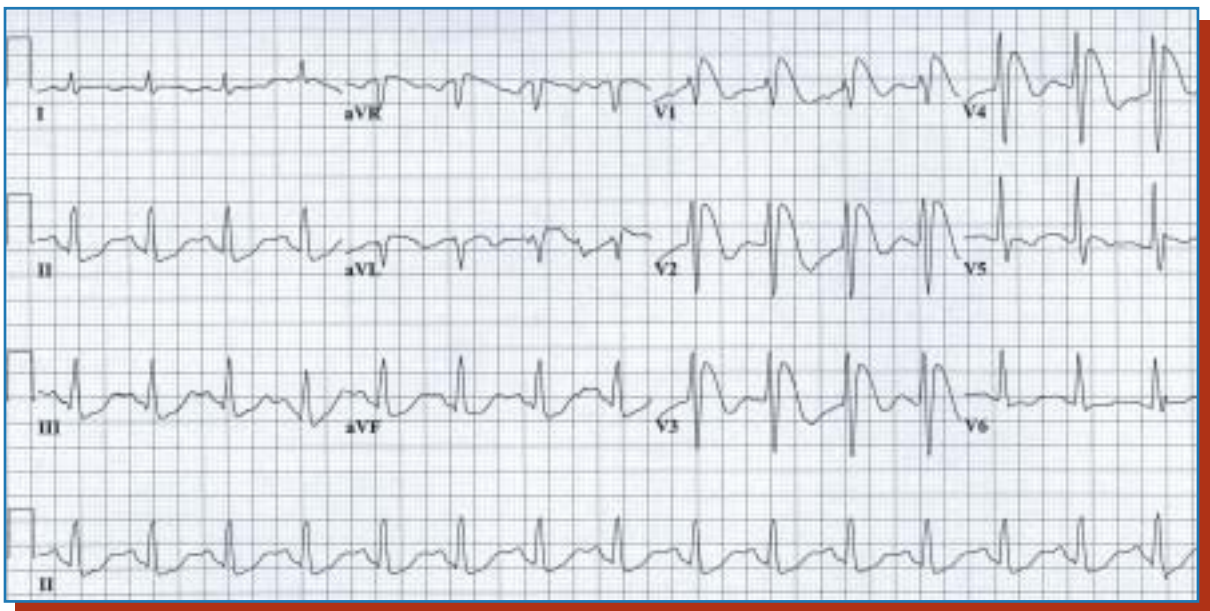


Figure 1. ECG upon presentation.

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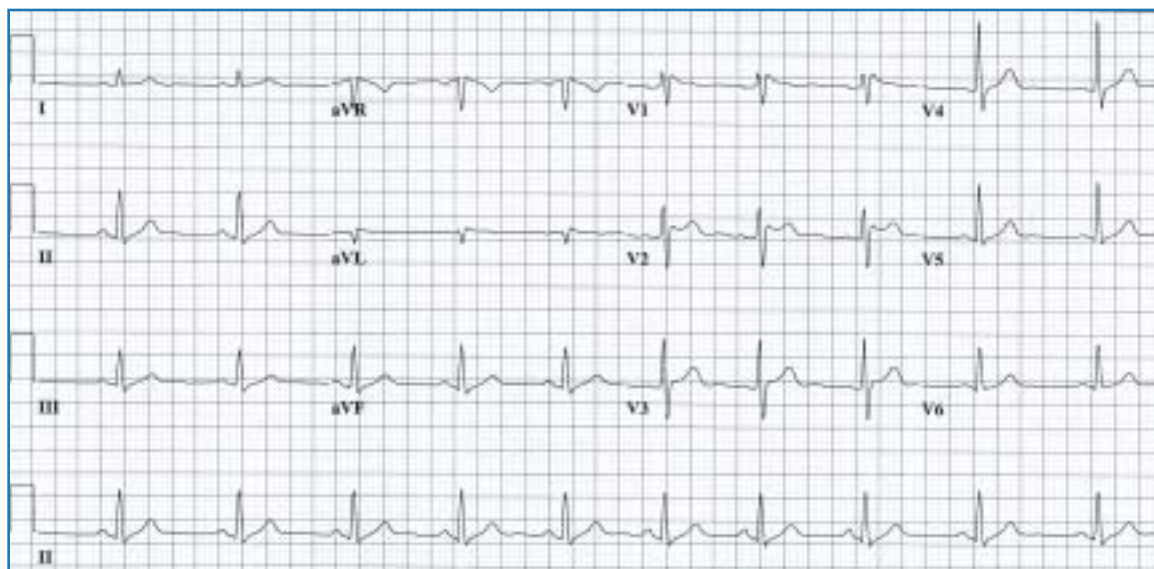


Figure 2. ECG two years earlier.

## Answers

**1.** The ECG shows sinus rhythm at 95 beats per minute. The QRS duration appears somewhat prolonged, although precise measurement is difficult. The striking abnormality is the very abnormal repolarization pattern in leads  $V_1$  to  $V_5$ . There is nearly 10 mm of ST-segment elevation in these leads, raising concern about the possibility of an acute anteroapical myocardial infarction (MI). But the ST-segment configuration is a little unusual (note the elevated J point and unusually rounded shape) and the patient is relatively young, with no cardiac symptoms.

**2.** Because of concern about the possibility of a clinically “silent” or atypical acute MI, an emergency two-dimensional echocardiogram was performed, showing no evidence of left ventricular wall motion abnormality. Fortunately, the hospital records contained an ECG recorded in the same patient two years earlier (Figure 2). There is an RSR pattern and unusual concave-upwards ST segment elevation in leads  $V_1$  and  $V_2$ . While this may be seen as a normal variant due to early repolarization, the changes seen on the ECG recorded during the tricyclic overdose are very suggestive of Brugada syndrome.

Brugada syndrome is characterized by an abnormal ECG (varying degrees of right bundle branch block with associated elevation of the ST segment and/or J point in leads  $V_1$  to  $V_3$ ) and a predisposition to sudden cardiac death due to idiopathic ventricular fibrillation. The condition is not common, although estimates of prevalence are hampered by lack of uniform criteria for diagnosis; not all individuals with a Brugada-type ECG are at risk for serious arrhythmia.

The mechanism of Brugada syndrome is related to abnormalities in the cardiac sodium channels, leading to changes in the configuration of the action potential in epicardial cells and a transmural voltage gradient manifesting as an “injury current” on the surface ECG. These changes can be exaggerated or unmasked by the administration of sodium channel blocking drugs, such as procainamide. Tricyclic drugs are also sodium channel blockers, hence the dramatic ST-segment changes seen in this patient.

The tricyclic drug was discontinued and the patient was referred to an electrophysiologist for further evaluation. **Dx**