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Brugada-pattern ECG and cardiac arrest in cocaine toxicity: reading between the white lines

A 33-year-old patient presented as an emergency with agitation and central chest tightness. She was diaphoretic and admitted to ingesting 14 g of loosely wrapped cocaine.

Shortly after presentation, she had a generalised tonic-clonic seizure followed by pulseless ventricular tachycardia. Sinus rhythm was restored with a 150-J biphasic DC shock. Post-arrest, she was hypotensive (BP 80/40 mm Hg) and hypercapnic (pCO₂ 18.2 kPa) with a severe acidosis (H⁺ 158 nmol/l), requiring sedation and ventilation.

Twelve-lead ECG showed sinus rhythm, right bundle branch block and coved ST elevation in leads V₁–V₃. Cardiology opinion was sought for consideration of emergency revascularisation. However, the ECG was consistent with Brugada pattern (panel A). Serial ECGs showed these changes to be resolving. The next morning the QRS duration was 88 ms with normalisation of ST segments (panel B).

The patient was extubated after 3 h. She remained stable and took an irregular discharge the following day. Urine toxicology was positive for cocaine. There was no family history of sudden death. She defaulted from follow-up.

Among its many effects on the cardiovascular system, cocaine is known to block cardiac sodium channels.¹ The Brugada syndrome is a genetic disease predisposing to life-threatening ventricular tachyarrhythmia and sudden cardiac death.² Approximately a quarter of cases are due to mutations in the cardiac sodium channel SCN5A, but several non-genetic inducers of Brugada-type ECG are known.² Our case illustrates an alternative diagnosis for acute ST elevation and an association between malignant arrhythmia and cocaine toxicity that may be due to 'poisoning' of the cardiac sodium channels.

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Competing interests None.

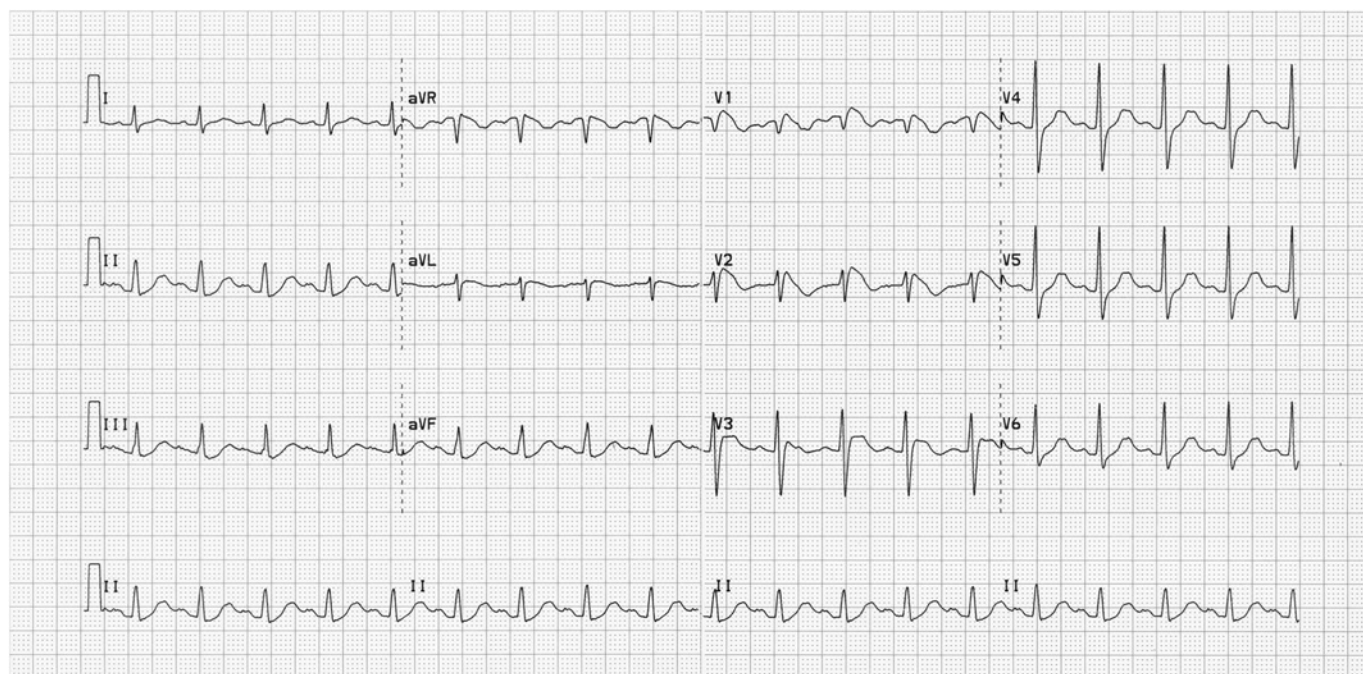
Patient consent Obtained.

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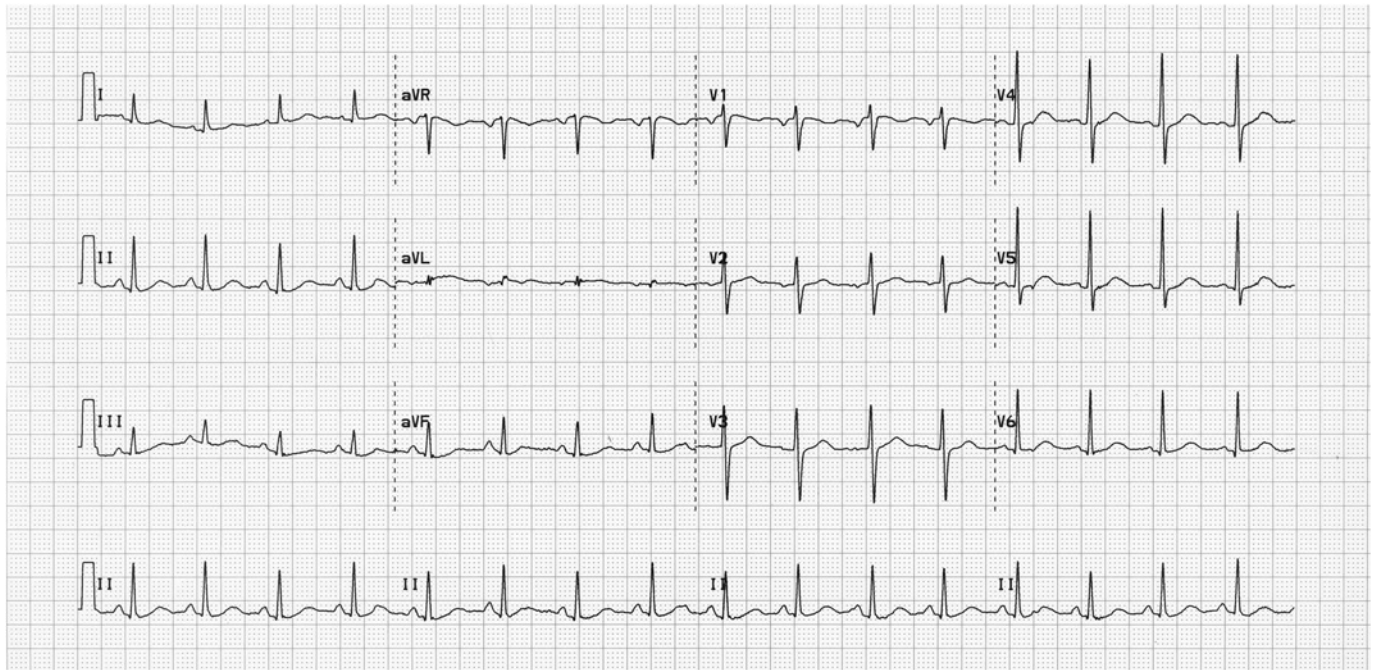
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Panel A

Images in cardiology



Panel B