A 51-year-old adopted white female was admitted to the intensive care unit with hepatic encephalopathy secondary to an upper gastrointestinal hemorrhage on a background of alcohol-induced chronic liver disease. She was hemodynamically stable (blood pressure 119/64 mm Hg) but pyrexial (39.0°C) and in atrial fibrillation, with a poorly controlled ventricular rate (Figure 1). Her regular medications included 200 mg spironolactone and 40 mg furosemide once daily. Her renal function and acid-base status were normal. A transthoracic echocardiogram revealed a structurally normal heart. As part of her initial treatment, an intravenous amiodarone infusion was commenced that not only restored sinus rhythm within 12 hours but also, interestingly, unmasked the classic features of type 1 Brugada syndrome (ST-segment elevation in leads V1 to V3, which descended with upward convexity into inverted T waves) (Figure 2). Discontinuation of the infusion led to a resolution of these electrocardiographic changes (Figure 3).

Brugada syndrome is an autosomal-dominant ion channel disorder that predisposes individuals with structurally normal hearts to ventricular arrhythmias and sudden cardiac death. The classic ECG phenotype is often concealed in affected individuals, and genetic testing is limited to approximately 20% of cases with mutations in the SCN5A gene affecting the α-subunit of the cardiac sodium channel. In clinical practice, therefore, the diagnosis of Brugada syndrome is largely based on pharmacological provocation tests using class Ia antiarrhythmic drugs, notably flecainide, ajmaline, and procainamide, which block sodium ion channels and unmask the key features of Brugada ECG phenotype. Treatment with nonantiarrhythmic agents such as tricyclic antidepressant drugs, lithium, and cocaine, as well as increased body temperature, may also lead to unmasking of the Brugada ECG phenotype. To our knowledge, however, this is the first ever reported case of unmasking the Brugada ECG phenotype as a result of amiodarone therapy. Amiodarone is predominantly a potassium ion channel–blocking agent (Vaughan Williams class III) but has been shown in vitro to have sodium ion channel–blocking properties, especially in the acute phase of its administration.1,2 This provides a plausible scientific basis for this observation, as well as another potential mechanism for fatal ventricular arrhythmias in conjunction with amiodarone.

This case illustrates that antiarrhythmic drugs, despite their various classifications, are a rather heterogeneous group of compounds commonly affecting more than one cardiomyocyte ion channel. The mechanisms underlying the antiarrhythmic and proarrhythmic effects of these agents may therefore be more diverse than expected in a clinical setting.

Disclosures
None.

References
Figure 1. Twelve-lead ECG undertaken before amiodarone infusion demonstrates persistent atrial fibrillation with a poorly controlled ventricular rate.
Figure 2. Twelve-lead ECG undertaken 12 hours after initiation of the amiodarone infusion demonstrates restoration of sinus rhythm and unmasking of type 1 Brugada ECG phenotype.

Figure 3. Twelve-lead ECG shows resolution of Brugada phenotype once amiodarone had been discontinued.
Unmasking of the Brugada Syndrome Phenotype During the Acute Phase of Amiodarone Infusion
Gideon Paul, Shamil Yusuf and Sanjay Sharma

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