Oral amiodarone provoking inferior ST elevation and unmasking Brugada-like electrocardiogram feature

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A woman presented with broad complex tachycardia. She was converted to sinus rhythm with intravenous amiodarone and continued on oral amiodarone. The amiodarone was stopped 3 weeks later as she was pregnant. Electrocardiogram (ECG) then revealed coved-type ST elevation in C1, suggestive of Brugada syndrome, and widespread inferior ST elevation. Electrocardiogram several months later showed resolution of inferior ST elevation.

Case Report

A 29-year-old Asian woman presented with palpitation and was found to be in haemodynamically stable monomorphic broad complex tachycardia (Figure 1A). There was no previous syncope, fever, or family history of sudden death. Intravenous amiodarone converted her to sinus rhythm. Electrocardiogram (ECG) post-conversion was suspicious but not diagnostic of Brugada syndrome (Figure 1B). She was discharged home on oral amiodarone which was stopped 3 weeks later when she was found to be pregnant. Electrocardiogram after discontinuation of amiodarone revealed coved-type ST elevation in C1-2 that was more diagnostic of Brugada syndrome and widespread inferior ST elevation (Figure 1C). Cardiac magnetic resonance imaging showed a structurally normal heart. Electrocardiograms several months later and post-natural labour showed resolution of inferior ST elevation but persistence of ST elevation in C1-2 (Figure 1D).

Following childbirth, she underwent an electrophysiological study which showed an increased HV (110 ms), and no supraventricular tachycardia. After 25 mg of intravenous flecainide, she developed ventricular tachycardia that degenerated into ventricular fibrillation (VF), requiring multiple electrical shocks. A cardiac resynchronization therapy defibrillator device was subsequently implanted. Genetic testing was considered but not carried out as it was felt that her case was not typical Brugada syndrome, hence too low a yield. A year later, she experienced appropriate implantable cardioverter defibrillator shock while overseas. A month after returning to the UK, she presented to a local hospital with intractable VF and passed away.

Brugada syndrome, a predominantly autosomal-dominant condition that predisposes to sudden cardiac death, is associated with various channelopathies, with mutations affecting sodium channel being the commonest.1 There is only one other reported case of Brugada syndrome unmasked by amiodarone,2 which also noted slight ST elevation inferiorly. This may be related to the ability of amiodarone to inhibit the sodium channel even though its main mechanism of action is via potassium channel blockade. In the

Figure 1
(A) Monomorphic broad complex tachycardia of left bundle branch block pattern (28 April 2008).
(B) Electrocardiogram after conversion to sinus rhythm with minimal ST elevation in C1-2 (29 April 2008).
(C) Post-oral amiodarone, there were significant ST elevations in precordial and inferior leads (19 June 2008). Amiodarone was discontinued around 18 May 2008. Electrocardiogram recorded on 1 June 2008.
present case, amiodarone was initially thought to have unmasked the Brugada features on the ECG, but the ST elevation in the right precordial leads persisted even after amiodarone had been discontinued for 9 months. However, amiodarone did appear to have caused marked inferior ST elevation in the patient, which subsequently resolved. There were other reported cases of Brugada syndrome displaying inferior ST elevation on the ECG, but with no link to amiodarone. Different channelopathies, especially in the presence of conduction disease and amiodarone, may result in unusual ECG appearances, as possibly in this case. It is increasingly recognized that several drugs can induce Brugada like ECG or cause arrhythmia in patients with Brugada syndrome, and should be avoided in these patients (www.brugadadrugs.org). Amiodarone use in Brugada patients is controversial, and was subsequently avoided in this patient even after childbirth.

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References