associated with ERO but it has a minor influence and is not a determinant of mitral valvular tenting. Lastly, regional dyssynchrony is an independent predictor of the degree of FMR in patients with non-ischaemic but not in patients with ischaemic LV dysfunction.

Authors' affiliations

E Agricola, M Oppizzi, M Pisani, A Meris, A Margonato, Division of Noninvasive Cardiology, San Raffaele Hospital, IRCCS, Milan, Italy
M Galderisi, Department of Clinical and Experimental Medicine, Federico II University of Naples, Naples, Italy
C Pappone, Electrophysiology Unit, San Raffaele Hospital, IRCCS, Milan, Italy

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IMAGES IN CARDIOLOGY

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Amiodarone induced skin necrosis

A 61-year-old man was admitted with an acute inferior non-ST elevation myocardial infarction. On day 3, he was noted to be pulseless and in ventricular fibrillation. He was electrically defibrillated to sinus rhythm and administered a 300 mg bolus of intravenous amiodarone. The attending medical team prescribed a further 900 mg intravenous amiodarone infusion over 23 hours via a peripheral cannula. Twenty-four hours after starting the infusion, the patient developed a dark discolouration of the right forearm, oedema of the limb and tense blisters (see panel). The attending dermatologist diagnosed skin necrosis secondary to amiodarone extravasation. There was no evidence of compartment syndrome. The case was discussed with the plastic surgeons who decided not to proceed to skin grafting because of his acute myocardial infarction. In addition, the pharmaceutical company advised the use of hot or cold packs and topical steroid cream.

To our knowledge this is the first case report to highlight the direct serious toxic effects of amiodarone on the skin and soft tissues. The Committee for the Safety of Medicines has only two entries for amiodarone extravasation injury and nine for injection site reactions. However, extravasation is not uncommon with peripheral vein infusions and care should be taken to minimise the risk of tissue necrosis by only administering amiodarone infusions via a central vein.

The toxic effects of the amiodarone infusion are due to the low pH of the solution (between pH 3.5 and 4.5) and the additives polysorbate and benzyl alcohol.

S J Russell
S Saltissi

stuandlou@doctors.org.uk