



Case Report

Epinephrine-induced myocardial infarction in severe anaphylaxis: is nonselective β -blockade a contributory factor?

Abstract

Epinephrine-induced myocardial ischemia in the setting of anaphylaxis is a rare event and is postulated to be due to coronary artery spasm. We report the case of a 43-year-old woman who presented to the emergency department with an anaphylactic reaction triggered by flucloxacillin. She was treated with intramuscular epinephrine, following which she developed ischemic chest pain and electrocardiographic changes, associated with troponin elevation. Subsequent coronary angiography demonstrated normal coronary arteries. In this case report, we discuss the potential role of prior nonselective β -blockade with propranolol in predisposing such patients to ischemic cardiac events following treatment with epinephrine.

A 43-year-old woman was admitted via the emergency department of our hospital with sudden onset of tongue and periorbital swelling. She had recently commenced a course of oral flucloxacillin for cellulitis of the left hand. She had a history of migraine and dyspepsia. Regular medications included propranolol, lansoprazole, and fexofenadine. She had no known traditional risk factors for coronary artery disease.

A diagnosis of anaphylaxis was made. She was first treated with intravenous hydrocortisone and chlorphenamine. Blood pressure (BP) was initially normal, but despite this treatment, she became profoundly hypotensive with a BP reading of 73/43 mm Hg. This prompted the intramuscular administration of 0.5 mg epinephrine (0.5-mL 1:1000 solution). There was inadequate clinical response to the first dose, and so, a second 0.5-mL dose was administered. This resulted in normalization of BP and halted progression of the facial edema.

Despite this clinical improvement, within the next 5 minutes, the patient subsequently complained of typical ischemic chest discomfort. An electrocardiogram (ECG) demonstrated 1-mm ST-segment depression in the anteroapical chest leads (Fig. 1A). The pain and ECG changes resolved spontaneously after approximately 30 minutes. Laboratory investigations performed 6 hours later revealed an elevated high-sensitivity troponin I level of 530 ng/L (upper limit of normal 30 ng/L). Other routine investigations were unremarkable, except for an elevated C-reactive protein (49 mg/L). She was given loading doses of aspirin and clopidogrel on suspicion of an acute coronary syndrome. Her condition remained stable with no further cardiac symptoms. After 48 hours, she underwent diagnostic coronary angiography. This demonstrated angiographically normal coronary arteries (Fig. 1B–C). Left ventriculography revealed normal left ventricular systolic function.

Taking account of her normal coronary anatomy, it was presumed that the chest pain, ECG changes and troponin elevation were due to coronary artery spasm induced by epinephrine. As such, she fulfilled the criteria for diagnosis of type 2 myocardial infarction (MI), that is, MI

secondary to ischemia due to imbalance between oxygen demand and supply [1], as opposed to coronary plaque rupture. Antiplatelet therapy was discontinued, and the patient was discharged from hospital. She was issued with an epinephrine auto-injector (EpiPen, Meda Pharmaceuticals Ltd, Bishop's Stortford, UK) in case of further anaphylaxis.

Myocardial infarction associated with anaphylaxis—sometimes referred to as “Kounis syndrome”—has been reviewed extensively [2]. More specifically, however, cases of epinephrine-induced MI and unstable angina in the setting of anaphylaxis have only been reported sporadically in the literature [3–6]. These cases have occurred largely in patients with normal coronary arteries, and the postulated mechanism is via coronary artery spasm.

In our case report, the patient was taking 80 mg of modified-release propranolol daily for the prophylaxis of migraine. This is of particular interest as treatment with propranolol has been reported to be associated with an increase in the risk of severe anaphylaxis [7], and more pertinently, nonselective β -blockade (ie, blockade of both β_1 - and β_2 -adrenoceptors) has been postulated to mediate coronary vasoconstriction via unopposed α -adrenoceptor agonism in the absence of the vasodilatory effects of β_2 -adrenoceptor stimulation [8]. Indeed, previous experimental work in humans has demonstrated that epinephrine can trigger coronary spasm in known sufferers of Prinzmetal's (variant) angina [9,10] and that this effect may be inhibited by α -blockade [9] and exacerbated by prior treatment with propranolol [10].

We believe that this is the first report of epinephrine-induced MI in the setting of anaphylaxis in a patient treated with a nonselective β -blocker. Epinephrine-induced type 2 MI is a rare event; however, emergency physicians and cardiologists should be alert to the possibility of ischemic cardiac complications in patients treated with nonselective β -blockers who present with severe anaphylaxis.

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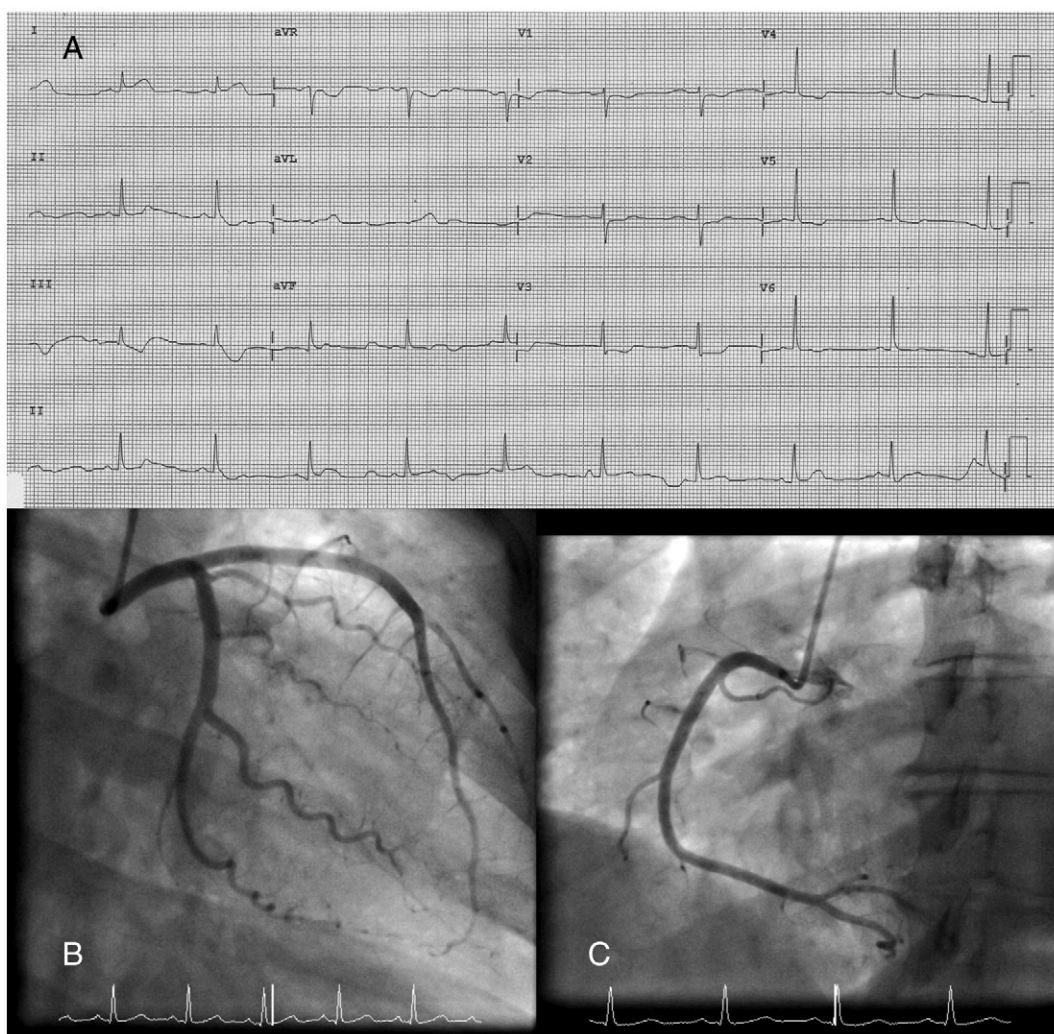


Fig. 1. Panel A, Twelve-lead ECG obtained during chest pain demonstrating ST-segment depression in the anteroseptal leads. Panels B and C, Coronary angiogram demonstrating unobstructed left (panel B) and right (panel C) coronary arteries.

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