



Letter to the Editor

Kounis syndrome presenting as ST-segment elevation myocardial infarction following a hymenoptera (bee) sting

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Abstract

We described a patient presenting with acute myocardial infarction after sustaining a hymenoptera (bee) sting. Cardiac catheterisation confirmed significant intra-coronary thrombosis in the left anterior descending artery with minimal underlying plaque disease. A unifying diagnosis of Kounis syndrome secondary to hymenoptera envenomation was made. This is followed by a brief review of the pathophysiology of acute myocardial infarction in the context of Kounis syndrome.

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A 35-year-old Afro-Caribbean gentleman presented with central crushing chest pain. He had no past medical history or cardiac risk factors and had an excellent exercise tolerance. He was taking no regular medications and there was no history of allergy. However, around 6 h prior to presentation he mentioned having sustained a bee sting.

On physical examination he was sweaty but haemodynamically stable. The remainder of the examination was normal. A 12-lead electrocardiogram showed anterior ST-segment elevation and hyperacute T waves.

A diagnosis of acute anterior ST-segment elevation myocardial infarction was made and following first line medical therapy including pre-treatment with aspirin 300 mg and clopidogrel 300 mg, he was transferred for emergency primary percutaneous coronary intervention (PCI).

Selective coronary angiography was performed via the right radial artery access route and demonstrated extensive thrombus in the mid-left anterior descending (LAD) artery with sluggish contrast flow distally (Fig. 1). The remaining coronary arteries were normal.

Using a 6 French XB3.5 (Cordis) guide catheter, a Balanced Middle Weight (BMW) wire was passed across the lesion. An abciximab infusion was commenced of which half of the initial bolus was given directly into the left coronary system. Sequential inflations using Voyager™ (Abbott) 2.0 × 20 mm and Maverick® (Boston scientific) 3.5 × 20 mm balloons were used to disperse the thrombus. This resulted in reperfusion, however considerable thrombus remained. The abciximab infusion was continued and a further angiogram was performed 48 h following presentation to ascertain whether there was underlying coronary atheromatous disease. This demonstrated prompt flow along the LAD with a single ulcerated plaque in the proximal vessel. This was confirmed by intravascular ultrasound. A Driver (Medtronic) 4.0 × 24 mm stent was deployed with an excellent angiographic result (Fig. 2). The patient was subsequently discharged home without complication.

A unifying diagnosis of Kounis syndrome secondary to hymenoptera (bee) envenomation was made.

Hypersensitivity reactions can occur in response to a variety of stimuli including drugs, environmental exposures and autoimmune disease. Such reactions, including hymenoptera stings, can range from localised inflammation and urticaria to severe anaphylaxis [1].

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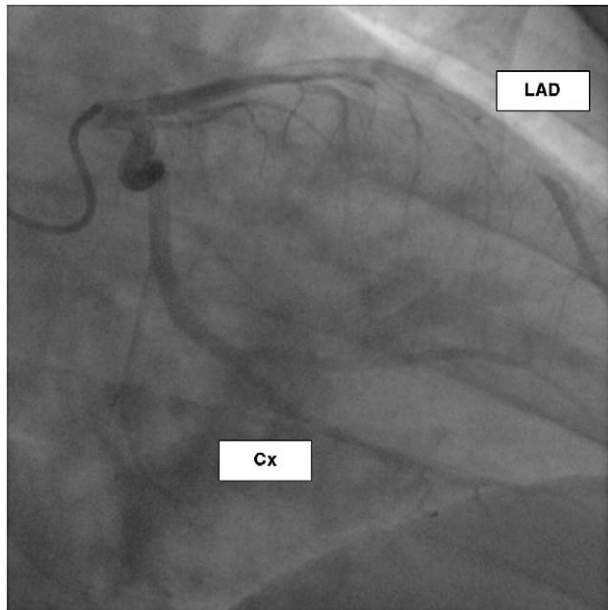


Fig. 1. Right anterior oblique (RAO) view of left coronary system. Unobstructed flow of contrast seen down left main stem (LMS) and circumflex (Cx) coronary arteries. Extensive thrombus visible within the left anterior descending (LAD) coronary artery.

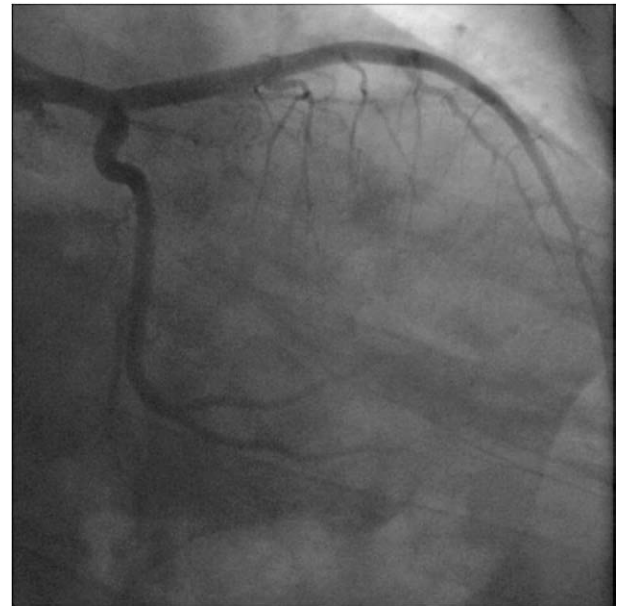


Fig. 2. Right anterior oblique (RAO) view of left coronary system following coronary angioplasty demonstrating excellent angiographic result.

A rare manifestation of such reactions is that of angina and acute coronary syndromes and this is referred to as the Kounis syndrome [2,3].

Two variants of the Kounis syndrome have been described. Type I refers to patients with normal coronary arteries who develop coronary vasospasm as part of the hypersensitivity response. The clinical presentation is usually of angina, but if the response is prolonged progression to myocardial necrosis and infarction can occur. It is hypothesised that type I Kounis syndrome is a manifestation of endothelial dysfunction (2,3). Type II Kounis syndrome occurs in patients with atheromatous coronary artery disease whereby the hypersensitivity reaction induces plaque erosion and rupture leading to the clinical manifestation of acute myocardial infarction [2,3].

The inflammatory response is essentially mast cell driven with the release of several vasoconstricting and collagen

degrading compounds including histamine, platelet activating factor and neutral proteases (tryptase and chymase). The latter activate metalloproteinases in atheromatous plaques and play an important role in their erosion and rupture [3,4].

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