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Acute coronary syndrome triggered by honeybee sting: a case report

S N Murat, B B Karasu, R Akdemir, H Kilic, E Ornek, O Ozcan

A 56-year-old previously healthy man was admitted to our hospital with a severe chest pain that started shortly after being stung by a honeybee. He did not have any allergic symptoms and there was no cutaneous or respiratory evidence of allergy on physical examination. Electrocardiographic and biochemical markers were consistent with an acute coronary syndrome. Coronary angiography demonstrated a significant thrombotic lesion in the right coronary artery. Here, a rare and unexpected event is presented, the development of acute coronary syndrome after a bee sting.

Exposure to bee stings is common worldwide; however, the development of acute coronary syndrome secondary to bee stings is a rarely reported event. This phenomenon was first described by Kounis and Zavras¹ as allergic angina in 1991. It is defined as the occurrence of acute coronary syndrome induced by the inflammatory mediators released through mast cells during allergic or hypersensitivity reactions.^{1,2} However, the exact mechanism triggering the development of myocardial infarction after a bee sting is not clearly established. Here, we present a patient with acute coronary syndrome triggered by a honeybee sting.

CASE REPORT

A 56-year-old previously healthy man was admitted to the emergency department of our hospital with a sudden onset of central, crushing chest pain radiating to both arms. He mentioned that he was stung on his neck by a honeybee 30 minutes before the onset of his chest pain. On admission, he was pale and diaphoretic. His blood pressure was 140/90 mm Hg and the pulse was regular with a rate of 80 beats per minute. He was not dyspnoeic and there was no cutaneous or respiratory evidence of allergy.

He was a cigarette smoker without any history of coronary artery disease, hypertension, diabetes mellitus or dyslipidaemia. He was asymptomatic in his daily life, with a normal functioning capacity and was not on regular medication. He did not have a known allergic disease and did not recall any previous history of a bee sting or drug-induced allergy.

A 12-lead ECG revealed ST segment depression in leads V4–V6. Following first-line medication with aspirin and clopidogrel, he was immediately transferred to the coronary care unit. He was treated with anti-ischaemic and anticoagulant medication. The results of blood examination revealed increased levels of

troponin and cardiac enzymes confirming the diagnosis of acute coronary syndrome; creatinine kinase-MB was 93 IU/l (reference 0–25 IU/l) and troponin was 0.92 µg/l (reference <0.01 µg/l). The blood eosinophils and total IgE were within normal limits.

He was taken to the coronary angiography laboratory and selective coronary angiography was performed. The right coronary artery was found to be totally occluded with a thrombotic lesion just distal to the conus branch. The left circumflex artery was diffusely diseased and there were non-critical lesions in the left anterior descending artery. The lesion in the right coronary artery was crossed with a floppy guidewire and a 3.0 × 20 mm stent was implanted successfully after a predilatation procedure (fig 1).

He had an uneventful and uncomplicated follow-up period; he was discharged on the fourth day at the hospital. Three weeks

later, in a follow-up visit, he was doing well and had a normal functioning capacity.

DISCUSSION

The mechanism triggering the development of myocardial infarction after a bee sting is not clearly established. It can be a part of a wide spectrum of allergic manifestations ranging from urticaria to anaphylaxis or just an independent phenomenon related to the direct cardiotoxicity of venom constituents.²

Mast cells are found in most parts of the human body including the heart and vessels. With an allergic insult, mast cells become activated and release their pre-formed granules into the circulation. These granules contain a number of compounds such as histamine, tryptase, chymase and platelet-activating factor. Upon degranulation, mast cells continue to undergo some ultrastructural changes, and by the metabolism of arachidonic acid some additional mediators such as leukotrienes and thromboxane are also released.^{1,2}

These mediators have been shown in many clinical and experimental studies to induce coronary artery spasm and/or myocardial infarction. It is known that histamine is a powerful vasoconstrictor. It can constrict coronary arteries via the H1 receptors situated in the coronary arteries. Moreover, histamine can activate and potentiate platelet aggregation, induce the expression of tissue factor, which is the main enzyme in the coagulation process.³ Another mediator, platelet-activating factor can cause myocardial ischaemia and

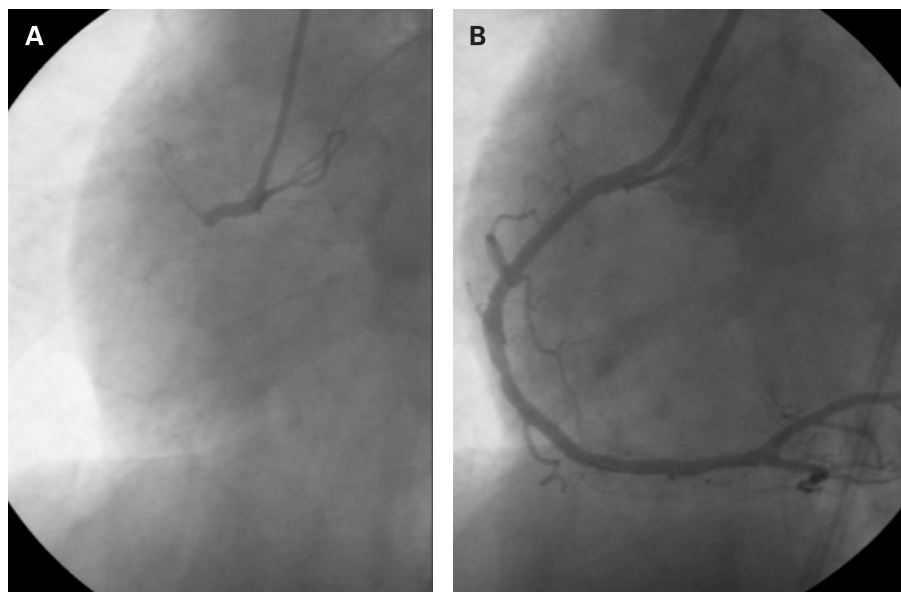


Figure 1 Coronary angiography showing (A) occlusion of the proximal right coronary artery and (B) stenting with procedural success.

produce ST segment changes on the ECG. Leukotrienes are powerful arterial vasoconstrictors; thromboxane is a potent mediator of platelet aggregation and has vasoconstriction properties.³

However, it was also shown that venom itself contains many vasoactive and thrombogenic mediators such as histamine, epinephrine, dopamine, 5-hydroxy tryptamine, leukotriene, thromboxane and phospholipase A. These venom constituents have physiological effects that are the same or similar to mediators released from mast cells during allergic reactions. With regard to the

mechanism, venom has the capability to cause acute coronary syndrome by its active constituents directly acting on the coronary vasculature without causing mast cell degranulation.⁴

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Images in emergency medicine

Inadvertent cannulation of the C6 transverse foramen during central venous catheterisation

A 37-year-old man on warfarin presented to the emergency department with reduced conscious level following a fall. A computed tomography scan of the brain revealed an acute subdural haematoma. Before transfer to a neurosurgical unit, a right internal jugular central venous catheter was uneventfully inserted under ultrasound guidance. Postoperatively he remained sedated and ventilated. Before lightening of sedation, a cervical spine computed tomography scan was performed, which demonstrated the catheter to have traversed the right C6 transverse foramen (fig 1). Aspiration of blood and blood gas analysis confirmed the venous placement of the catheter. The catheter was removed without complication. Cannulation of the vertebral vein in the right C6 transverse foramen has been reported previously in a 13-year-old boy, in whom the position of the catheter remained undetected for 5 months.¹ Excess lateral rotation of the head at the time of insertion may have contributed to the unusual location of the catheter.

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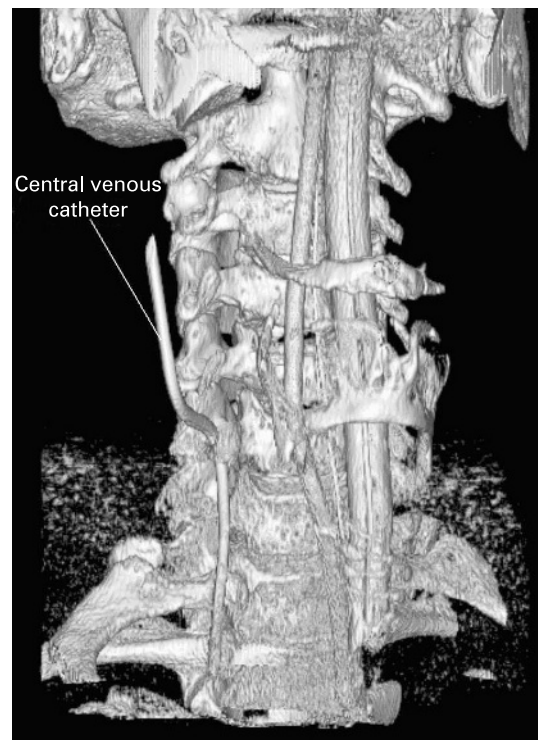


Figure 1 Three-dimensional computed tomographic reconstruction demonstrating the central venous catheter traversing the right C6 transverse foramen.

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