



Letter to the Editor

Acute anterior myocardial infarction after multiple bee stings. A case of Kounis syndrome

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Abstract

A 58-year-old man with no history of cardiac diseases or cardiovascular risk factors was stung by honeybees. Soon after, he gradually developed signs of an allergic reaction followed by severe retrosternal pain. Electrocardiographic, echocardiographic evidence and positive biochemical markers were consistent with an acute anterolateral myocardial infarction. Coronary arteriography showed a left anterior descending artery thrombotic lesion. This is a case of Kounis syndrome, which is the concurrence of acute coronary syndromes with conditions associated with mast cell activation including allergic or hypersensitivity reactions as well as anaphylactic or anaphylactoid insults. The clinical implications and pathophysiology of this dangerous association are discussed.

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1. Introduction

Hypersensitivity reactions from hymenoptera stings, mediated through various immunologic pathways, may range from simple urticaria and subcutaneous angioedema to severe anaphylaxis [1]. Cardiac involvement, although rare, constitutes the most severe and life threatening scenario. More specifically, coronary vasospasm and myocardial infarction secondary to allergic reactions have been described since 1991 as the Kounis syndrome [2,3], which has subsequently been associated with several causes which may activate hypersensitivity pathways and induce mast cells degranulation [4]. We report the case of a patient with no history of coronary artery disease, who developed a generalized urticarial reaction, after he was stung several

times by bees, followed by an extensive anterior wall myocardial infarction.

2. Case report

A 58-year-old country man was admitted to our hospital after he was stung on the head and neck several times by honeybees while working on his field. After stinging, he recalled a short-lasting period of dizziness and respiratory discomfort but not loss of consciousness. He was immediately transferred to the hospital for further evaluation and treatment. On arrival, he had a generalized erythematous rash over the face, neck and thorax accompanied by itching. He was afebrile; his blood pressure was 120/80mm Hg and the pulse 90beats/min regular. Examination of the heart, lungs, abdomen and central nervous system revealed no abnormality. He was non-smoker, with no history of dyslipidaemia, hypertension, diabetes mellitus, coronary artery disease or other significant illness. He was asymptomatic in his daily life, with excellent functional capacity. Moreover, he had no

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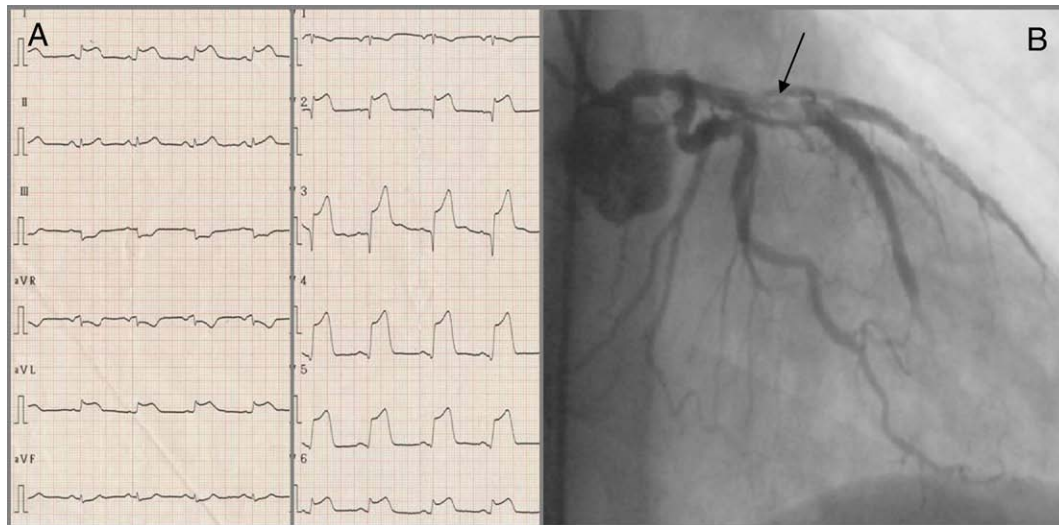


Fig. 1. A. Electrocardiogram: ST segment elevation in I, aVL, V2–V6 with reciprocal ST segment depression in III, aVF leads. B. Coronary angiography: proximal left anterior descending artery thrombotic lesion.

previous history of allergy, rhinitis, bronchial asthma, dermatitis or eczema. However, he recalled that he had been stung, in the past, by honeybees, without any sequelae.

While he was in the emergency department, and before any medication given, he felt unwell, started sweating, became pale and complained of severe retrosternal pain radiating to both arms associated with nausea and vomiting. An electrocardiogram performed immediately revealed elevation of ST segment in leads I, aVL, V2–V6 and ST segment depression in leads III, aVF, compatible with acute anterior myocardial infarction (Fig. 1A). He was given immediately 325mg aspirin per os; and the following medications intravenously: 1g hydrocortisone sodium succinate, 10mg morphine, 50mg diphenhydramin and nitroglycerin infusion of 1.2µg/kg/min. He was transferred to the coronary care unit where he was thrombolysed with tenecteplase shortly after the beginning of the pain. Transthoracic echocardiography revealed severe systolic impairment of the

left ventricle with akinesia of the middle and apical septal and anterior segments and apex. Unfortunately, thrombolysis was not successful and the patient proceeded to rescue percutaneous coronary intervention. Coronary angiography revealed 85% proximal left anterior descending artery thrombotic lesion which was successfully treated with direct stenting (Fig. 1B). Left ventriculography showed a mildly enlarged left ventricle with severely impaired systolic function (EF 25%) and akinesia of the anterolateral wall and apex.

He had an uneventful and uncomplicated hospital follow-up and he was discharged on the seventh day. His medical treatment comprised aspirin, clopidogrel, b-blocker, ACE inhibitor, and statin.

On a follow-up visit, three weeks later, the patient complained of dyspnea on exertion. A new echo study confirmed a severely depressed systolic cardiac function and a pseudonormal pattern of the mitral inflow, while an apical thrombus was visualized in the left ventricle (Fig. 2). Thus,

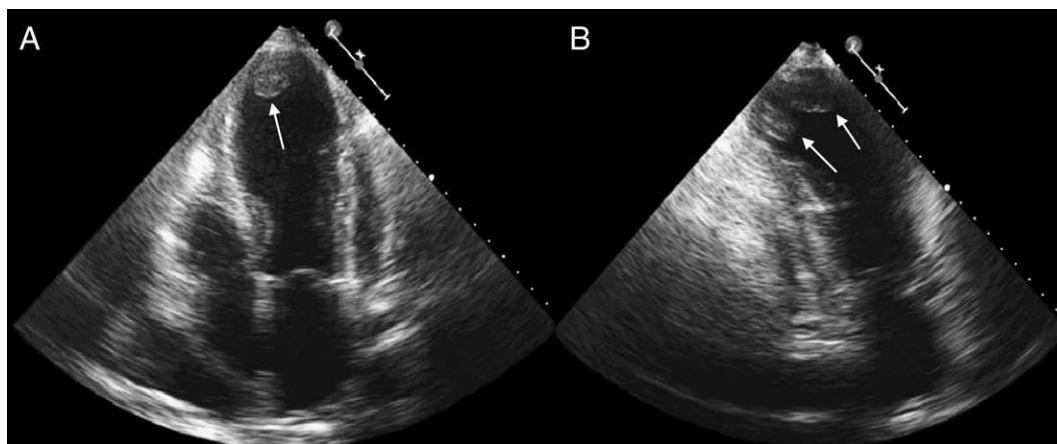


Fig. 2. Four-chamber (A) and two-chamber (B) echocardiographic views: Aneurysmal dilatation of the anterolateral wall and the apex. Apical thrombus in the left ventricle and pericardial effusion.

we set the patient on anticoagulants, furosemide and eplerenone. Upon 24-hour Holter electrocardiographic monitoring, he was found to have single ventricular ectopy and no complex arrhythmias.

At three months' follow-up the patient remains in a stable clinical condition, being in NYHA Class II heart failure, while the thrombus of the left ventricular apex has been fully dissolved.

3. Discussion

Kounis syndrome is the concurrence of acute coronary syndromes with mast cell degranulation induced by allergic or hypersensitivity reactions. It was first described in 1991 as allergic angina and/or allergic myocardial infarction [2]. Symptoms and signs may include chest pain, with/or without raised troponins and cardiac enzymes, dyspnea, faintness, nausea, vomiting, syncope, pruritus, urticaria, diaphoresis, pallor, palpitations, hypotension and bradycardia [4].

There are several causes that have been reported as capable of inducing Kounis syndrome. These include several drugs, a number of conditions and a variety of environmental exposures. The latter include stings by ants, bees, wasps, jellyfishes, as well as grass cutting, poison ivy, latex contact, limpet ingestion, millet allergy, shellfish eating and viper venom poisoning [3,5,6].

Two types of Kounis syndrome have been characteristically described [4]. Type I variant includes patients with normal coronary arteries in whom the acute allergic insult induces either coronary artery spasm leading to unstable angina with normal cardiac enzymes and troponins or coronary artery spasm progressing to acute myocardial infarction. This variant might represent a manifestation of endothelial dysfunction or microvascular angina. Type II variant of Kounis syndrome includes patients with pre-existing, albeit occult, atheromatous disease in whom acute allergic episode can induce plaque erosion or rupture manifesting as an acute myocardial infarction.

Concerning the patient in our case, although he had no significant risk factors and he was asymptomatic in ordinary activities, he had occult atheromatous coronary artery disease. As it is characteristic of type II variant of Kounis syndrome, the acute allergic episode after the bee sting might have probably induced plaque erosion or rupture manifesting as an acute myocardial infarction. Usually, tryptase originating from the degranulation of mast cells, effectively activates the zymogen forms of metalloproteinases such as interstitial collagenase, gelatinase and stromelysin found in atheromatous plaques and plays an important role in plaque erosion or rupture. The ensuing cascade of events is typical of an acute coronary syndrome. Moreover, the clinical course can vary from minimal myocardial damage to extensive myocardial infarction, while the spectrum of complications does not differ from the ordinary coronary events. In our patient, after an extensive anterior wall myocardial infarction, there was a severe impairment of left ventricular function as well as apical thrombus formation. Fortunately, the patient remains in a moderate functional capacity, while the thrombus dissolved readily with the proper treatment.

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