

Coronary artery dissection, coronary vasospasm, Kounis syndrome, and allergy without cutaneous manifestations

In the intriguing case report by Yoshino *et al.*,¹ a 46-year-old woman who had Kounis syndrome type II 6 years prior as a result of eating momiji manju (steamed bun), a Japanese sweet cake, with skin rash and coronary artery dissection. She experienced again chest pain, without skin rash, associated with ST-segment elevation along with complete atrioventricular block. Intravascular ultrasound revealed again coronary artery dissection with intramural haematoma without any signs of atherosclerotic lesions. The patient had allergic diathesis with history of allergy with skin rash to cefaclor at the age of 30 and to transdermal isosorbide dinitrate patch about 6 months ago. The authors emphasized how unusual it is for two of coronary artery dissection episodes to occur in the same patient, one from an allergic reaction and the other from a non-allergic reaction. Coronary spasm might have been the cause of these episodes.

This report raises important issues concerning coronary artery dissection, coronary vasospasm, and hypersensitivity without skin manifestations:

- (1) According to a recent report² dealing with coronary artery dissection cases, there are CD68⁺ and CD3⁺ protein complexes throughout the adventitial infiltrate, extending into the perivascular adipose tissue and in the media surrounding the dissection plane and haematoma. Furthermore, compared to the same vessel layer in aortic tissues from controls, thoracic artery dissection tissues³ have been found to have significantly more CD68⁺ macrophages, neutrophils, mast cells, and CD3⁺ T lymphocytes in both the medial and adventitial layers. All these immunology cells synergistically play a key role in acute and delayed hypersensitivity events. Regarding mast cells, all that is present in the bloodstream are their precursors, which require several days or weeks to develop into mature cells that are packed with cytoplasmic secretory granules. As a result, the mast cells had to exist at the rupture site prior to the acute event.
- (2) Vasospasm and vasospastic angina in the responsible vessel of coronary artery dissection have been verified by a few studies using the vasoreactivity test. These results suggest that the hallmark of coronary artery dissection may not be coronary epicardial and microvascular vasomotor dysfunction, but rather a complication known as vasospastic angina.⁴
- (3) In a patient with Kounis syndrome after bee sting,⁵ coronary artery dissection was developed without any visible skin symptoms as in the described patient.¹ The lack of skin involvement in acute allergic

or anaphylactic events has been linked to decreased cardiac output caused by volume loss and plasma leakage, which lowers venous return and delays or prevents the released anaphylactic mediators from reaching and acting on the skin.⁵

From the above, it appears that the aetiology of coronary artery dissection includes the same or related causes for its creation. Immunological causes play a major role. In cases of coronary artery dissection, clinicians should take into consideration the possibility of concomitant vasospasm and myocardial infarction due to the need for different clinical management strategies of these cardiac manifestations.

Ethics approval: This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

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Data availability

All data are available from the corresponding author.

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Nicholas G. Kounis^{1*} , and **Virginia Mplani**²

¹Department of Medicine, Division of Cardiology, University of Patras Medical School, Patras 26500, Greece; ²Intensive Care Unit, University of Patras Medical School, Patras, Greece

* Corresponding author. Email: ngkounis@otenet.gr