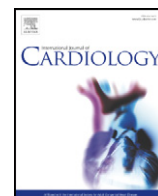




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Letter to the Editor

An extremely rare trigger of Kounis syndrome: *Actinidia chinensis*

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A 23 year old male was admitted to our department for typical ischemic chest pain. His physical examination revealed diffuse erythema and rash on his face, neck and bilateral upper extremities with pruritus. He has a history of oral allergy syndrome caused by kiwi. His blood pressure was recorded as 110/70 mm Hg with a heart rate of 86 bpm. Electrocardiogram (ECG) showed ST segment elevation in DII, DIII and aVF leads (Fig. 1). He has no cardiovascular risk factors and medication usage. His allergic symptoms and chest pain started after ingestion of a piece of kiwifruit. He was transferred to the coronary care unit. Transthoracic echocardiography revealed preserved left ventricle systolic functions and coronary angiography showed normal coronary arteries (Fig. 2A–B). On laboratory tests, eosinophil ratio (19%), troponin I level (3.1 ng/ml) and the value of total IgE (386 IU/ml) were significantly increased. Type 1 Kounis syndrome (KS) was considered in view of the clinical history and relationship with kiwi. Treatment was started with parenteral corticosteroids, intravenous volume replacement and antihistamines.

The “allergic angina” which is known as Kounis syndrome was firstly described in 1991 by Kounis and Zavras as the “concurrence of angina pectoris progressing to myocardial infarction with an immediate type hypersensitivity reactions associated with a massive mast cell activation” [1]. It has been postulated that the release of various vasoactive inflammatory mediators (histamine, leukotrienes and chemokines) secondary to mast cell degranulation is the main pathophysiologic factor of the vasospasm of coronary arteries, atheromatous plaque rupture and thrombus formation. The trigger may be chemicals, foreign bodies, environmental exposure, stent implantation and various drugs as well as intravenous anesthetics, contrast media, antineoplastics, analgesics and antibiotics [2–5]. In addition, the KS secondary to kiwifruit allergens has been identified in literature [6]. Actinidine (Act d1), a thaumatin-

like protein (Act d2) and kiwellin (Act d5) are major allergic proteins of kiwi (*Actinidia chinensis*).

KS has been categorized into three variants: the first variant (type 1) describes patients with normal coronaries who do not report pre-existing cardiovascular risk factors predisposing toward coronary artery spasm following exposure to an allergic insult and release of vasoactive mediators. The acute allergic reaction induces a coronary vasospasm with ST changes in the ECG and increased cardiac enzymes and troponins. The second variant (type 2) is observed in patients with pre-existing atherosclerotic disease. The vasospasm induced by release of the allergic mediators may predispose the erosion or the rupture of the atheromatous plaque, evolving more frequently toward an acute myocardial infarction. Recently a third variant (type 3) includes patients with drug eluting stent thrombosis in whom eosinophils and mast cells were isolated in aspirated thrombus [7].

In the treatment of type 1 KS, intravenous glucocorticoids may alleviate hypersensitivity-induced vasospasm safely. H1 and H2 antihistamines were also recommended to relieve the symptoms. Type 2 KS should be managed as an acute coronary syndrome procedure with glucocorticoids and antihistamines. Epinephrine may aggravate the myocardial ischemia and prolong QT interval and arrhythmias by inducing mast cell degranulation. For that reason, epinephrine use is controversial in KS. In type 3 KS, the interventional procedures of stent thrombosis should be performed.

We aimed to clarify the consideration and management of KS whose diagnosis and treatment require scrutinized evaluation. When allergic symptoms lead to acute coronary events, the possibility of KS should be kept in mind and related agents should be identified to avoid recurrent acute coronary syndrome.

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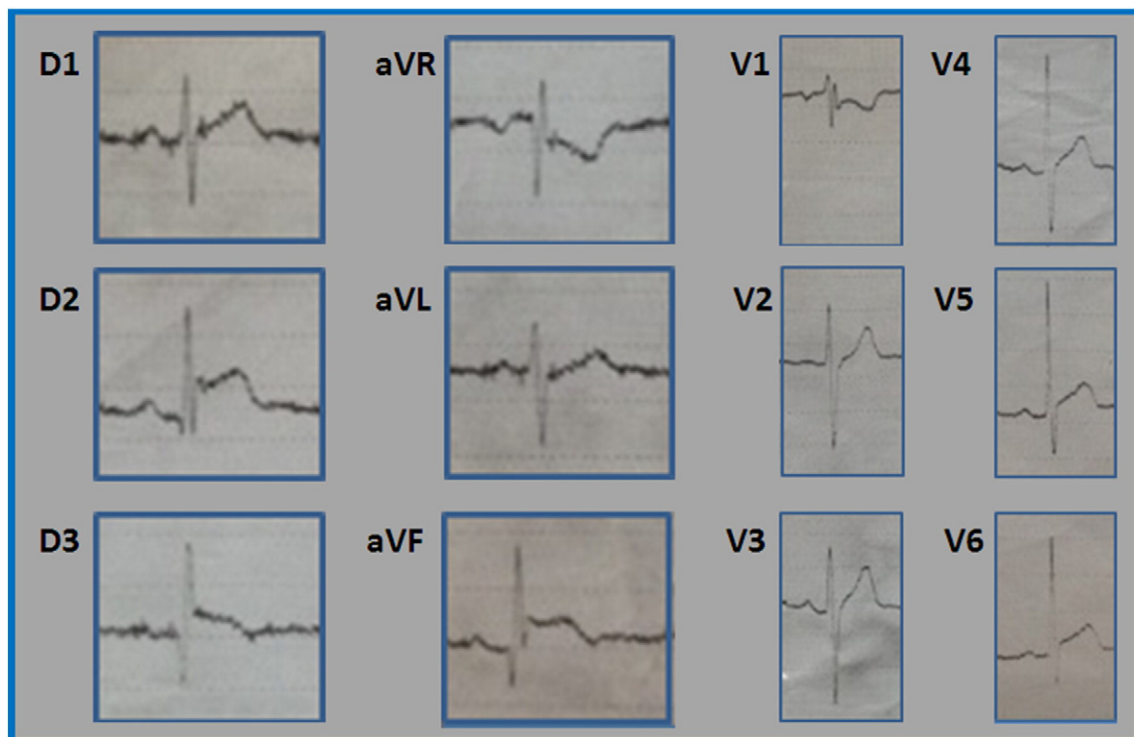


Fig. 1. ST elevations in DII, DIII and aVF leads.

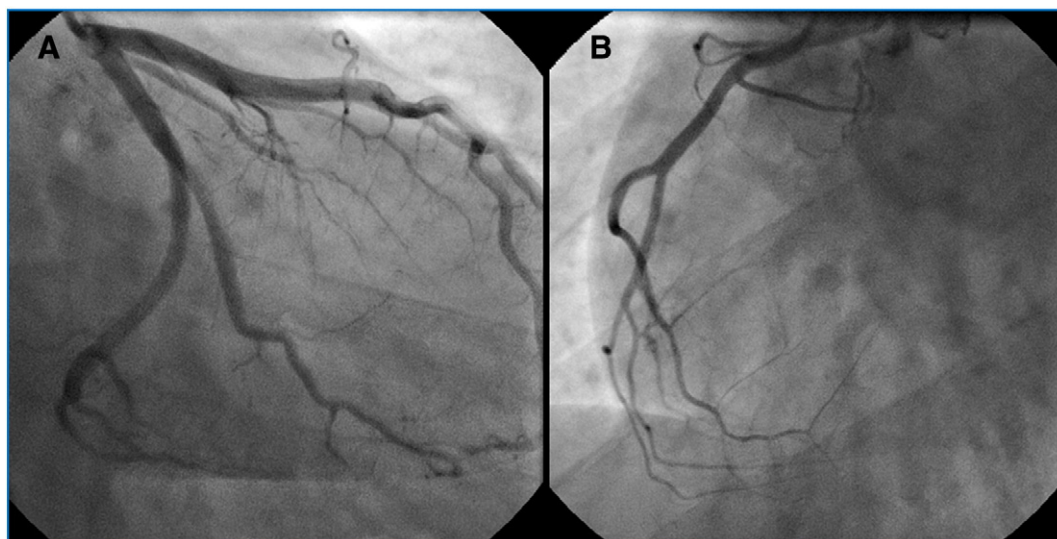


Fig. 2. A–B: On coronary angiogram, normal coronary arteries were seen.