



Correspondence

Cardiac anaphylaxis: searching for clarity☆☆☆

To the Editor,

We read the article by Tummala et al [1] with great interest. Apart from the type II Kounis syndrome and iatrogenic administration of excess doses of epinephrine, other mechanisms could contribute to acute ST-segment elevation myocardial infarction in this patient. During anaphylaxis, there will be rapid shift of intravascular fluid into the extravascular compartment, which can trigger the rennin-angiotensin-aldosterone system resulting in compensatory catecholamine release [2]. In addition, histamine could stimulate the release of catecholamines by a direct action on adrenal medulla [3], which initiates the coronary spasm or could rupture the atheroma plaque and causes platelet aggregation in patients with atherosclerotic coronary disease.

Likewise, one cannot disregard the recent systematic reviews and meta-analyses [4,5], which shed light on emerging evidence that not only selective COX-2 inhibitors but also traditional nonsteroidal anti-inflammatory drugs may be associated with atherothrombotic events such as myocardial infarction and stroke. The available evidence points to the fact that diclofenac, in particular, is likely to increase hazard ratio for thrombotic cardiovascular events [6]. Diclofenac inhibits COX-2 to a higher level, which is coupled with an incomplete inhibition of COX-1, which alters the prostacyclin-thromboxane A2 balance, thus causing a prothrombotic situation [7]. The increased cardiovascular risk has been observed in patients with a prior high risk of cardiovascular disease and in previously healthy individuals. In addition, the prolonged systemic hypotension induced by vasoactive and inflammatory mediators released during anaphylaxis probably caused further reduction of the myocardial perfusion, thus favoring in situ thrombus formation and subsequent coronary artery occlusion. In the present case, the patient is at high risk for coronary artery disease in respect with his age, sex, and smoking habit.

We can, therefore, deduce that prolonged coronary vasospasm and thrombotic vascular occlusion on top of the preexisting coronary artery disease might have played a major role in addition to the hypersensitivity reaction in this patient. To overcome drug-induced morbidity and mortality in any case, the prescribers should look into patients' health status and coexisting illness(es), and the status of medicines such as pharmacokinetics, drug interactions, and contraindications. To bring these into reality, the health science education and training as well as accrediting bodies should enforce regular prescription auditing and monitoring. Let our prescriptions be patient centered and value driven.

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Reply to the letter to the editor

To the Editor,

Acute myocardial infarction is multifactorial in the setting of anaphylaxis. The relative risk of low- and high-dose diclofenac without medical prescription causing myocardial infarction is well described [1].

This patient, with moderate Framingham risk score for cardiovascular events over 10 years [2], complained of chest pain immediately