Coronary stents attract like magnet inflammatory cells and induce stent thrombosis and Kounis syndrome

Dear Editor,

Patients with implanted stents who develop an allergic reaction elsewhere in the body from various different causes are prone to develop intrastent thrombosis. Stent thrombosis has been associated with allergic symptoms such as glottis edema, cold sweat, and tongue enlargement following a flaxonate/propyphenazone administration a week after stent implantation. [1] Acute myocardial infarction in the stented area coincided with allergic reactions following intravenous administration of the non-anionic contrast material iopromide during a routine excretory urography.[2] Late drug eluting stent thrombosis defined as a type III variant of Kounis syndrome[3] has occurred following an allergic reaction to non-steroidal anti-inflammatory agent acemetacine.[4] Intrastent thromboses have also been reported following insect and larvae sting-induced allergic reactions.[5] Even allergies to clopidogrel,[6] the drug given to prevent stent thrombosis, has induced stent thrombosis. All above reports concerned patients who were receiving multiple medications following stent implantation. Therefore, one can assume that stents, like magnets, attract inflammatory cells and constitute the area of possible mast cell and platelet activation.

In the report by Isik et al.[7] a 65-year old atopic, hypertensive and hypercholesterolemic patient with a bare metal stent implantation who was taking aspirin, clopidogrel, statin, and angiotensin converting enzyme inhibitor developed an allergic reaction following a wasp sting in his face. Sixty minutes later he developed an acute myocardial infarction complicated with ventricular tachycardia and was found to have his bare metal stent totally thrombosed. This report raises some important issues concerning the etiology, pathophysiology, prognosis, prevention of stent thrombosis, and management of patients with stent implantation. The described patient was taking four different drugs, all of which are known to have antigenic properties. Furthermore, the implanted bare metal stent is made from stainless steel which consists of nickel, chromium, manganese, titanium and molybdenum. These agents can join forces in order to degranulate mast cells and release mediators. It is known that mast cell surface brings 500,000 to 1 million IgE molecules and degranulation occurs when 2,000 of these molecules make 1,000 bridges using antigens of different specificities as it happens in the stented patients.[8] Furthermore, a subset of platelets contains both high (FCεRI) and low (FRεRII) affinity IgE receptors[9] and these receptors are activated by antigens of different specificities in order to induce platelet aggregation and thrombosis.

Ideally, in the described patient, thrombus aspiration during angiography and staining with hematoxylin-eosin for eosinophils and Giemsa for mast cells would have confirmed the diagnosis for Kounis syndrome type III.[10] In order to predict and prevent allergy associated stent thrombosis, complete histories of allergies and hypersensitivities to any drug, condition, or environmental exposure for patients who are going to have stent insertion should always be taken. Fortunately, new generation stent manufacturing companies have already emphasized these cautions and precautions in information sheets enclosed in the commercial stent packages.[11]

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References
Authors reply

Dear Dr. Kounis,

Thank you for your attention to our letter, in which we presented a late bare-metal stent (BMS) thrombosis in a patient following wasp sting. A BMS had been applied to a left anterior descending critical lesion nine months ago. The patient was known previously to have honeybee venom allergy. To our knowledge, this represents the first case of total occlusive late stent thrombosis (ST) in a BMS following wasp sting.

Kounis et al. [1] had reported the relation between ST and allergic reaction in different case reports, and they also mentioned our case report. Kounis et al. had previously defined late drug-eluting ST, which is a variation of type 3 Kounis syndrome. As the authors stated in previous cases and also in our case, the most important defect is that the patient is exposed to multiple allergens when the ST occurs. At this time, the thrombus material is not stained, and as a result, type 3 Kounis syndrome is not confirmed. This raises multiple questions. Whether the thrombotic process that occurs is a result of a single or multiple allergen(s) is not known exactly. Nonetheless, in our case, we believe that the wasp sting was the probable cause of the ST. Firstly, the patient had used drugs regularly prior to the ST development, and little time had passed between the sting and the ST. Secondly, phospholipase A1 has a considerable role in thrombosis, and wasp venoms harbor a higher amount of phospholipase A1 than honeybee venoms.[2]

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