Coronary artery aneurysm following stent implantation, hypersensitivity and Kounis syndrome

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In the very interesting report published in Interactive CardioVascular and Thoracic Surgery [1] concerning a patient with coronary artery aneurysm occurring late (eight years) after drug-eluting stent implantation, the authors speculate on the pathophysiology of this event, referring to possible local hypersensitivity and chronic inflammation and suggesting that patch reinforcement using BioGlue might be effective for preventing potential rupture.

Coronary aneurysms have been detected as early as three days after drug-eluting, and as late as nine years after bare metal stent implantation, and are usually associated with dangerous stent thrombosis [2]. Some studies have reported that coronary artery aneurysms occurred in 1.25-3.9% of patients after stent implantation [3]. However, recent studies have shown that mast cells contribute importantly to the pathogenesis of abdominal aortic aneurysms and aneurysms of the cerebral arteries. Indeed, ruptured aneurysms had prominently more mast cells stained with monoclonal antibody for anti-mast cells tryptase clone AA1 compared with unruptured aneurysms [4]. The Kounis hypersensitivity-associated acute thrombotic coronary syndrome is the result of the action of inflammatory mediators released locally or in the systemic circulation following mast cell degranulation. Vasospastic angina (type I variant) and acute coronary thrombosis (type II variant) are two variants of this syndrome that have been described so far.

Recently, a type III variant is diagnosed in stent thrombosis when aspirated thrombus stained with Giemsa and haematoxylin-eosin reveals infiltration by mast cells and eosinophils respectively [5]. It has been found that mast cells degranulate when 2000 nearby antibodies attached to their surface are bridged by corresponding antigens and make the critical number of 1000 bridges. The hypersensitivity thrombotic process can also follow activation, by mast cell mediators, of high affinity and low affinity FcγRI, FcγRII, FcδRI and FcδRII receptors situated within the platelet surface. Metal stent components, polymers, and eluted drugs constitute an antigenic complex that applies a continuous and persistent hypersensitivity inflammation on the coronary vessel as long as they last. Mast cell-specific proteases such as chymase and tryptase have been found to induce vascular cell apoptosis thus facilitating adventitial enlargement. All of the above show that stent implantation can promote aneurysm formation.

Therefore, allergy-free stent materials and stents coated with CD34 antibodies that can capture circulating progenitor endothelial cells and increase the acceleration of endothelial coverage, stents coated with NO donors which can decrease platelet adhesion, activation and aggregation, stents able to elute drugs with anti-inflammatory and anti-allergic properties that can abrogate late thrombotic events experimentally, seem to constitute future preventive modalities for coronary aneurysm formation.

Conflict of interest: none declared.

References