We thank Cervellin et al 1 for their interesting comments on our article on allergic inflammation in acute coronary syndromes. 2 Kounis et al 3 described allergic inflammation to be responsible for acute coronary syndromes, during conditions associated with mast cell activation, including allergic or hypersensitivity and anaphylactic insults. Indeed, eosinophils and mast cells play a key role in the late phase of an allergic response, releasing allergic mediators responsible for coronary vasoconstriction and thrombosis. Autopsy studies demonstrated mast cell infiltration at sites of coronary artery plaque rupture. 4 Moreover, mast cell degranulation was higher at the sites of erosion, suggesting an active role in promoting vasoconstriction and plaque destabilization. 4 Cervellin et al showed elevated troponin levels in patients admitted to emergency department for anaphylactic shock, angioedema, and urticaria, 5 suggesting that coronary arteries could be the primary target of the released mediators during allergic reactions. Allergic inflammation is supposedly initiated by allergens cross-bridging their receptor-bound immunoglobulin IgE antibodies on the mast cell or basophil cell surface, as confirmed by higher IgE levels found during acute coronary syndromes. 6 However, the relationship between ischemic heart disease and allergy/atopy remains a complex issue to be elucidated by larger studies with inhibitors of mast cell degranulation, which might clarify their role in coronary plaque destabilization. The question whether mast cells enter the lesion before or after plaque rupture remains unanswered.

Disclosures

None.

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