In response to: Anaphylaxis during cardiac surgery for hypertrophic cardiomyopathy: pathophysiologic and therapeutic considerations

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We are most appreciative for the comments from Dr. Nicholas G. Kounis [1] and his colleagues regarding our recent publication describing the challenging case of anaphylactic shock in patients suffering from hypertrophic obstructive cardiomyopathy (HOCM) scheduled for surgical treatment, namely myectomy [2]. Their letter raises several important concerns regarding anaphylaxis in anaesthesia and particularly in patients with HOCM. Before we address each point, we would like to mention that several topics discussed by authors were mentioned in our case report but could not be described in detail due to space limitations. In this context, several comments made by Dr. Kounis provide excellent complimentary information to our case reports.

The authors of this letter have indicated that the diagnosis of anaphylaxis may be difficult while the patient is under general anaesthesia and subsequently during emerge from anaesthesia [3]. In the described case, patient presented several symptoms typical for severe anaphylactic reaction: arterial hypotension, high airway pressures, airway swelling and skin rash. Additionally, after initiating a cardiopulmonary bypass which stabilized patient hemodynamic collapse, we obtained a blood sample to test for mast cell tryptase. The test came positive, confirming the occurrence of severe anaphylactic reaction. Finally, after recovery following cardiac surgery, the patient was referred to an allergologist and additional skin tests confirmed a strong sensitivity to chlorhexidine.

Concerns were also raised about the role of anaesthetic drugs in the development of anaphylaxis. Indeed, several medications used to induce and maintain general anaesthesia have been described as strong allergens. The most common are non-depolarizing muscle relaxants and antibiotics [2, 3]. Dr. Kounis and colleagues have suggested that salbutamol could aggravate hemodynamic collapse; this is an excellent point as this beta mimetic certainly causes tachycardia, which can worsen hemodynamic compromise in patients suffering from HOCM. Additionally, they suggested that preservatives present in ampoules of epinephrine (for example, sulphites) may also contribute to allergic reactions. We checked the contents of epinephrine ampoules used in our hospital and they are preservative-free. Finally, the authors of the letter asked the question about possibility of Kounis syndrome, which is defined as the concurrence of acute coronary syndromes with conditions associated with mast cell activation, involving interrelated and interacting inflammatory cells, and including allergic or hypersensitivity and anaphylactic or anaphylactoid insults [5–7]. Again, it is an excellent point and all anaesthesiologists should be aware of this phenomenon. However this diagnosis was unlikely in the presented patient since a transesophageal echocardiography demonstrated excellent contractility of both ventricles without any features of regional wall motion abnormalities.

Dr. Kounis and colleagues pointed out another important genetic association — HOCM and atopic sensitivity. This is again an excellent point and one which alludes to our conclusions that patients suffering from this rare genetic disorder (HOCM) should be treated in tertiary referral centres, which can offer extracorporeal support in cases of hemodynamic collapse induced by anaphylactic reaction including Kounis syndrome.

In summary, we would like to thank again Dr. Kounis and his colleagues for their excellent comments. Without doubt, they have highlighted several challenges facing anaesthesiologists who are looking after patients suffer-
ing from HOCM. Additionally, their letter alludes to the importance of multidisciplinary cooperation, which always benefits the perioperative care of complex surgical patients suffering from this disease [8].

References:

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