

Authors' response

We are grateful to Dr Kounis and Dr Soufras for their thoughtful insight into the mechanisms of anaphylactic shock and their evaluation of our article entitled "Major contribution of vasospasm-induced coronary blood flow reduction to anaphylactic ventricular dysfunction assessed in isolated blood-perfused rat heart" [1].

As Dr Kounis and Dr Soufras pointed out, the current consensus on the mechanisms of anaphylactic shock may be that anaphylactic hypotension is caused primarily by reduction in effective circulating blood volume which results from systemic vasodilatation with the peripheral pooling and increased vascular permeability with a shift of intravascular fluid to the extravascular space [2]. We have further suggested that anaphylactic hypotension involves hepatic venoconstriction, as well as blood pooling, and extravasation in splanchnic organs [3, 4]. Reduced venous return and resultant depression of cardiac output would cause coronary hypoperfusion, myocardial damage, and ventricular dysfunction.

Nevertheless, we agree with Dr Kounis and Dr Soufras in that myocardial ischemia induced by coronary spasm is involved in the pathogenesis of anaphylactic shock. In our most recent study [1], we specifically addressed the question of whether the cardiac failure during anaphylaxis is primarily due to coronary vasoconstriction-induced myocardial ischemia or the direct negative inotropic effect of chemical mediators, using the method of the imposed constriction of coronary artery to reproduce the reduction in coronary blood flow during rat cardiac anaphylaxis. Consistent with the concept of 'Kounis syndrome', our study suggests that anaphylactic ventricular dysfunction is attributed mainly to vasoconstriction-induced coronary flow reduction and resultant myocardial ischemia. As pointed out by Dr Kounis and Dr Soufras, other experimental and clinical studies also suggest the involvement of myocardial ischemia via coronary vasoconstriction and ventricular contractile failure in the pathogenesis of anaphylactic shock [5, 6]. Chemical mediators released not only locally from immunologically activated cardiac mast cells

but also from tissues other than the heart would cause a marked constriction of coronary vessels, cardiac arrhythmias, and negative inotropism [7, 8]. In anaphylactic shock, therefore, myocardial dysfunction due to vasospasm-induced coronary flow reduction manifesting as 'Kounis syndrome' should always be considered.

We believe that both vascular dysfunction to decrease circulating blood volume and coronary spasm-induced myocardial dysfunction underlie the development of anaphylactic shock. As Dr Kounis and Dr Soufras suggested, further experimental and clinical studies are necessary to determine the importance of myocardial ischemia via coronary spasm and effects of chemical mediators for development of anaphylactic shock, as well as to propose new therapeutic approach including the protection of cardiac tissue.

Conflict of interest: none declared

References

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