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Authors' Response

Sir,

We would like to thank Prof. Kounis et al. for the commentary letter on our case report, A Fatal Case of Anaphylactic Shock During Paragliding, J Forensic Sci. 2012 by Feltracco et al. They suggest considering coronary spasm and concomitant Kounis syndrome (1) as probable cause of anaphylactic death. We have appreciated the reporting of pathophysiological mechanisms involved in systemic vasodilation, reduced venous return, extravascular fluid leakage as well as the details on coronary hypoperfusion and myocardial damage. They conclude that in similar clinical cases, the heart and coronary arteries should always be examined in postmortem screening as they represent one of the primary targets of anaphylaxis.

In this case, we have described that coronary arteries and myocardium were indeed explored on autopsy but significant disease was not discovered. Lack of detection of preexisting atherosclerotic coronary vascular disease and the absence of any implanted intracoronary stent allow therefore to exclude the characteristics of type II and type II variants of Kounis syndrome (1). Signs and symptoms of type I variant (i.e., severe coronary spasm in normal arteries) could have probably occurred in a previously sensitized individual after the bee sting; however, the way the dramatic event occurred and the delay in first rescue arrival obviously precluded any clinical investigation focused on detecting the particular features of "allergic" myocardial infarction. It can be speculated that a simultaneous combination of intense coronary vasoconstriction and severe laryngeal and epiglottic edema, airway edema, and strict bronchospasm very likely occurred.

On ground examination of oral cavity, tongue, and lips by helicopter medical staff, along with the discovery of the bee in his mouth was consistent with an asphyxiating mechanism as the first cause of loss of consciousness and likely of death thereafter. Very likely the marked facial angioedema, massive lingual edema, and severe swelling of oro-pharyngeal mucosa were associated with tightened vocal cords and severe bronchospasm, with no space for alveolar air to enter even under forced insufflations. In this case, sudden brain hypoxia may have occurred prior to the onset of any other symptoms. Nevertheless, an important contribution to the drop in blood pressure and sudden shock could have been determined by severe bradycardia and lethal myocardial ischemia.

However, even if realistically concurrent with the other manifestations of anaphylactic reaction, the possible onset of allergic angina progressing rapidly to acute myocardial infarction and circulatory collapse would have been impossible to recognize in such a setting. Only the absence of electrical activity and no electrocardiographic abnormalities consistent with acute myocardial ischemia were detected at rescue arrival. Even if tested, the cardiac enzymes and troponins would have been questionable and complicated in interpreting.

Hymenoptera sting-induced Kounis syndrome causing unexplained death has already been described (2), but the acute cardiovascular manifestations of anaphylaxis, that is, the rapid onset of shock with peripheral circulatory failure, cannot always be promptly ascribed to a "coronary hypersensitivity" syndrome leading rapidly to myocytes death. For this reason, Kounis syndrome may certainly develop but it might be unrecognized or remain masked.

Physical examination by medico-legal staff only reported massive facial and light systemic swelling; no positive significant findings relevant to the cause of death were identified at autopsy. Serum tryptase measurements would have been useful, as raised serum tryptase level is suggestive of an antemortem anaphylactic event, but unfortunately it was not done.

It is worth noting that at postmortem investigation, remarkable changes in terms of coronary vascular or bronchial tone, or glottis edema, or systemic arteriolar tone, or interstitial blood extravasation, and shift of fluid volume are often lacking in the case of anaphylactic shock (3).

Mast cell infiltration at the site of coronary artery spasm (4) was not searched as no coronary spasm was detected at postmortem examination. As no visible findings of myocardial damage or evident endothelial injury resulted, the signs of recent inflammatory cell response at coronary wall layers were not investigated.

In conclusion, the primary involvement of severe coronary vasoconstriction and concomitant Kounis syndrome, even if not proven on either a clinical or histopathological basis, may have contributed, in association with sudden onset of intolerable hypoxia, to the death of the pilot while still "in midair."

References

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