Letter to Editor

Hymenoptera Stings, Anaphylactic Shock and the Kounis Syndrome

Dear Editor,

Hymenoptera venoms contain various peptide and protein components, most of which can act either as allergens or as toxins. These components can induce coronary artery spasm and cardiac anaphylactic shock via IgE and direct mast cell activation, respectively. It has been estimated that about 1500 stings would be required to release a lethal dose of venom for a non-allergic adult who weighs 70 kg and that about 40 deaths a year are attributed to hymenoptera stings.[1]

In the very important report[2] published in the *North American Journal of Medical Science*, a patient was stung by multiple wasps and developed type I variant of Kounis syndrome with anaphylactic shock and myocardial ischemia. Treatment with two liters of normal saline, adrenaline, hydrocortisone, and anti-histamines did not have any immediate effect, and the patient recovered in a later stage with vasopressors and myocardial infarction protocol therapy.

This report raises some important issues concerning the pathophysiology of anaphylactic shock and the prediction of severe anaphylaxis and Kounis syndrome following hymenoptera stings. It is generally believed that, during anaphylactic shock, systemic vasodilatation, increased vascular permeability with plasma volume extravasation, and reduced venous return contribute to depression of cardiac output with subsequent coronary hypoperfusion and myocardial damage. This is why the treatment is fluid repletion and adrenaline administration. However, experimental and clinical evidence indicates that the human heart can be the primary target of anaphylaxis. In experimental anaphylaxis with ovalvumin-sensitized guinea pigs, it was shown that within three min after the antigen administration, the cardiac output decreased by 90% with rising of left ventricular end-diastolic pressure and arterial blood pressure indicating pump failure. Contemporarily, electrocardiographic recordings uniformly showed signs of acute myocardial ischemia.[3] The blood pressure started declining steadily after four min. The conclusion was that, "the idea that the registered anaphylactic damage might be due to peripheral vasodilatation can be definitely excluded." In addition, the rapid increase in left ventricular end-diastolic pressure suggests that decreased venous return and volume loss due to an increase of vascular permeability are unlikely to be the primary causes of the documented depression of cardiac output and blood pressure." In the clinical setting, patients with anaphylactic cardiac shock[4] do not always respond to intravenous fluid administration and anti-allergic therapy but require myocardial infarction treatment as it happened with the published report.[2] Differentiating global myocardial hypoperfusion from a primary cardiac myocardial suppression due to mast cell mediator action is clearly challenging. Combined myocardial suppression and peripheral vasodilatation, perhaps, occur simultaneously. Patients prone to develop immediate and severe anaphylactic reaction to hymenoptera stings have found to have raised baseline tryptase.[5] Such patients have clonal mast cell disorder either systemic mastocytosis or monoclonal mast cell activation syndrome. One can, therefore, wonder if these patients have KIT mutations that lower the threshold for severe anaphylaxis and they respond vigorously in order to develop Kounis syndrome. KIT is the mast receptor for the stem cell factor that is essential for mast cell development, proliferation, survival, adhesion, and homing.

In conclusion, in any case of anaphylactic cardiac shock manifesting with symptoms and signs of Kounis syndrome, combined treatment of anaphylaxis and acute myocardial infarction seems to be mandatory.

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