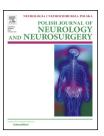


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# Letter to the Editor

# Subarachnoid hemorrhage occurring after bee sting



To the Editor.

Bee venom by virtue of containing enzymes, biogenic amines, lipids, amino acids may cause local and systemic complaints and sudden deaths. Sobotka et al. analyzed bee venom and found four major proteins: hyaluronidase, phospholipase A, melittin and apamin. Individuals who are honeybee sensitive are also very sensitive to phospholipase A [1]. Also it was discovered that the allergen named Apim6, causes strong IgE and T cell reaction [2].

A 68-year-old male patient was brought to our hospital due to loss of consciousness, tonic-clonic convulsion and respiratory distress after bee sting from the dorsal surface of the patient's foot. His relatives denied a history of any allergy and asthma, chronic disease, drug intake, and head trauma. The patient was unconscious and was having difficulty in breathing. His score on the Glasgow Coma Scale was 4. Respiratory acidosis was detected in his arterial blood gas analysis and he was connected to a mechanical ventilator. His initial vital signs were as follows: arterial blood pressure: 85/62 mmHg; heart rate: 115/min; temperature: 36.8 °C. Hematologic, biochemical and cardiac examinations were performed and investigations revealed normal, except white blood cell count: 14.9 K/ $\mu$ L (4–10), and urea (65 mg/dL) levels. The cranial tomography showed widespread subarachnoid hemorrhage (SAH) between cerebral sulcus, in the ventricular system and in the cisterns. The patient was diagnosed as SAH due to exposure to bee stings. Severe electrolyte and laboratory (urea, creatine) imbalance, metabolic acidosis developed on the second day and the patient was taken to the dialysis treatment after nephrology consultation. The patient died due to multiple organ failure and brain herniation fourth day.

Studies reported that systemic reactions occur in 0.66–3.3% [3–5] of the general population. Anaphylaxis cases after a bee sting is around 0.4–0.5% [6]. Anaphylactic shock, acute kidney failure, acute pulmonary bleeding, acute myocardial infarction, atrial fibrillation and acute hemorrhagic pancreatic are also other extraordinary and more serious complications that can be encountered [7]. Also convulsions, optic neuritis, exacerbation of multiple sclerosis, brachial plexus block, hypoxic brain injury, encephalomyelopolyradiculoneuritis,

cerebral hemorrhage and hemorrhagic vasculitis are some of the neurological complications reported in association with possible bee sting [8].

A person consists of a variety of immune reactions as a result of bee stings, depending on his/her immunologic structure. Venom hyper-sensitivity, immunologic mechanisms (IgE-mediated or non-Ig-E mediated venom allergy), can arise out of immunologic mechanisms but also can come about due to non-immunologic mechanisms which are responsible for some of the reactions to bee stings [9].

Bee venom includes histamine, thromboxane, leukotriene and other vaso-active and inflammatory mediators. Systemic immune reaction will cause vasoconstriction after a bee sting. In addition, a neuropharmacological (sympathetic) mechanism of endothelial permeability involving the cerebral vasculature with a concurrent systemic thrombogenic or immune response has also been postulated [10]. Mellitin, phospholipase, hyaluronidase are the main toxic proteins. The most fatal part of the poison is phospholipase A2. This is an anti-coagulant that prevents the blood from coagulating and also causes the production of cortisol, which is a stress hormone [11]. In addition to that, it is a venom component that dilates the hyaluronidase blood capillaries, which cause more inflammation.

We could not carry out angiographic imaging as the condition progressed rapidly, and that the clinical situation was not well and that he developed acute kidney failure. For this reason, we cannot say if there is an aneurismal bleeding or not. Even if there is an aneurismal bleeding, we believe that systematic anaphylactic reaction caused the progress of the condition. We think that one or more of toxic proteins in content of venom, which can cause capiller dilatation, increase in endothelial permeability and trigger mechanisms related to anticoagulant effect, are associated with SAH, although sufficient explanatory data in the literature does not exist. Although the evidence is not conclusive, the findings point to physio-pathological mechanisms.

### **Author contributions**

Concept – HHK, AUU; Design – HHK, AUU; Supervision – HHK, AUU; Resource – HHK, AUU; Materials – HHK, AUU, MA, ABE;

Data Collection and/or Processing – HHK, AUU, MA, ABE; Analysis and/or Interpretation – HHK, AUU, MA; Literature Search – HHK, AUU, MA, ABE; Writing – HHK, AUU; Critical Reviews – HHK, AUU, MA.

#### Conflict of interest

None declared.

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#### **Ethics**

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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