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Kounis Syndrome And D-Dimer Elevation After Bee Sting

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ABSTRACT

Bee stings can cause a wide variety of clinical presentations, from a simple local lesion to anaphylaxis. Kounis syndrome is the development of acute coronary syndrome (ACS) with activation of mast cells associated with allergy, hypersensitivity or anaphylactic reactions. It has a large clinical spectrum from chest pain to AMI and that accompanies a subclinical, clinical, acute or chronic allergic reaction. Medications, foods, insect bites, bee stings and intracoronary stent placement may set off the allergic reaction. In this case, we presented a 50-year-old male patient with Kounis syndrome, which is one of the rare complications of bee sting, and our patient had elevated blood D-Dimer level, a first in the literature.

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INTRODUCTION

Bee stings are quite common, especially in beekeepers. Bee stings can cause local symptoms such as skin redness, swelling, itching to very serious conditions such as dyspnea, cough, hypotension, acute myocardial infarction (AMI), arrhythmia, anaphylaxis and death. More serious consequences occur in those who are allergic to bee venom [1,2]. In the treatment, cold application, oxygen, antihistamine, steroids and adrenaline in case of anaphylaxis can be applied [3]. Kounis syndrome is the development of acute coronary syndrome (ACS) with activation of mast cells associated with allergy, hypersensitivity or anaphylactic reactions. It has a large clinical spectrum from chest pain to AMI and that accompanies a subclinical, clinical, acute or chronic allergic reaction. Medications, foods, environmental factors (such as insect bites, bee stings, pollen, latex contact) and intracoronary stent placement may set off the allergic reaction [4,5]. We presented a Kounis syndrome case with D-Dimer elevation, which is a first in the literature.

Case Report

Our patient in this case was 50 years old and presented to the emergency department with complaints of respiratory distress and chest pain. The general condition of the patient was moderate to poor, his appearance was pale, his blood pressure was 70/50, his pulse was 80 beats/minute, his temperature was 37.4 degrees, and his respiratory rate was 23/minute. On physical examination of the patient, minimal rhonchi were heard in the lung, other than that, physical examination findings were normal. The patient did not describe an allergy or disease history, stated that he had been stinged by bees before, but he did not have any complaints.

In the first laboratory examinations of the patient, leukocyte count: 13200 (4-10x10^3), troponin T (TnT): 75ng/L (0-14 ng/L), creatine kinase muscle brain (CKMB): 6.0 ug/L (0-5 ug/L), and D-Dimer: 875 ng/ml (0-232 ng/ml). The control values of the patient who was observed in the emergency department at the fourth hour were TnT: 52 ng/L, CKMB: 2.6 ug/L, D-Dimer: 425 ng/ml. At the eighth hour, TnT: 12 ng/L, CKMB: 2.2 ug/L and D-Dimer: 200ng/ml were found. Normal sinus rhythm was observed in the second hour electrocardiogram (ECG) of the patient who had ST depression in the first ECG (Figure 1).

The patient's other laboratory tests were normal. In the patient who was thought to have Kounis syndrome, D-Dimer elevation and dyspnea also suggested pulmonary embolism.

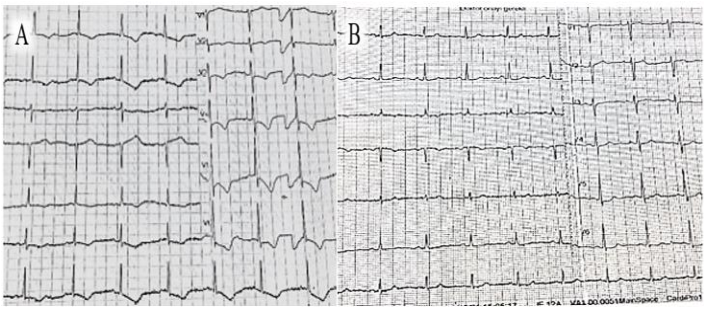

Figure 1: Patient's ECGs (A) after bee sting and (B) after medical treatment

A decrease in D-Dimer level was observed with clinical improvement during the observation period of the patient, who was initially thought to have Kounis syndrome. Our patient, who developed dyspnea at her first admission, was immediately administered 0.5 mg of adrenaline intramuscularly for anaphylaxis. Subsequently, 3 L/hour oxygen was started, 1 ampoule of antihistamine, 1 mg/kg prednisolone and 1 ampoule of pantoprazole were administered intravenously. Inhaled salbutamol was given. Our patient was followed up for 24 hours with fluid support. The patient, who was clinically relieved and his vital signs were stable, was discharged with recommendations and planned outpatient control.

DISCUSSION

Bee stings can cause local reactions such as sudden pain, swelling and exacerbation reaction that resolve within a few hours, as well as life-threatening upper respiratory tract edema, vomiting, diarrhea, rhabdomyolysis, intravascular hemolysis, renal failure and rarely neurological reactions including paralysis [6]. There are studies showing that an allergic reaction defined as Kounis syndrome, is characterized by AMI clinic with increase in CKMB and TnT due to bee sting [7]. In our case, CKMB and TnT elevation were present after the bee sting. Although the pathophysiology of this reaction is discussed, it is thought to be associated with mast cell activation, which causes allergy, and ACS. It is believed to be an ACS due to coronary artery vasospasm or atherosclerotic rupture resulting from this reaction [8,9]. In some clinical and laboratory studies, some cytokines and chemokines that are involved in arachidonic acid metabolism and released have been accused of causing coronary artery spasm and AMI. Two types of Kounis syndrome have been identified in which the coronary arteries are normal and the coronary arteries are atheromatous [10,11]. ACS is a thrombotic coronary artery disease with or without ST-segment elevation, inclusive myocardial infarction and angina and diagnosed by ECG and cardiac enzymes such as troponin [12]. ACS is an important cause of morbidity and mortality and accounts for the majority of deaths from coronary heart disease [13]. Our patient with ECG changes was followed up with the diagnosis of Kounis syndrome causing reversible coronary artery spasm and was discharged with recovery.

In our case, D-Dimer elevation was also present in addition to the typical laboratory findings of Koinus syndrome. D-Dimer is formed as a result of the destruction of the fibrin clot by plasmin. There are studies showing that although D-Dimer is a marker that increases in some physiological conditions, it is most frequently increased in thromboembolic events and is an important mortality indicator [14]. In the literature, there are publications showing that D-Dimer elevation is also associated with infection, cancer, heart failure, and anemia in addition to thromboembolism [15]. There were no cases in the literature showing D-Dimer elevation in Kounis syndrome. Our case is a first in the medical literature with this feature, and we think that D-Dimer increases secondary to cardiac vasospasm.

The most common cause of death due to bee stings is respiratory and cardiac causes. In these patients, an airway should be established first. If cardiopulmonary collapse is present, intubation should be performed, and if laryngeal edema has developed, tracheostomy should be performed and oxygen therapy should be initiated. If there is shock, volume replacement should be performed with 25-50 ml/kg or more colloid solution. In case of anaphylaxis, adrenaline (0.5 ml intramuscularly in adults, 0.01 ml/kg intramuscularly in children) is applied. Adrenaline and steroid therapy can be used in bronchospasm. Intravenous aminophylline and inhaled salbutamol are other options. If the patient has angioedema but no hypotension, adrenaline can be applied subcutaneously [16-18]. The medical treatment we applied was the recommended treatment protocol. The rapid response to the treatment shows that the appropriate treatment protocol is applied.

CONCLUSION

Many clinical conditions may develop after a bee sting, from simple local effects to anaphylactic shock or Kounis syndrome causing cardiac findings. Therefore, when a bee sting comes, systemic effects should be taken into consideration, if necessary, it should be kept under observation for a while, necessary tests should be ruined, and in cases with serious complications, early treatment should be started promptly.

Ethical approval

Clinical approval was obtained from the department of emergency medicine in Niğde Ömer Halisdemir Training and Research Hospital, along with the patient's informed consent, to write this article.

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CONFLICT OF INTEREST

There is no conflict of interest between the authors of this article.

Informed Consent

Informed consent was obtained from the patient described in this case report.

Authorship Contributions

ÖY is the doctor of the case. All authors worked together in the literature review and preparation of the article. MCA is the corresponding author.

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