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CASE REPORT / *OLGU SUNUMU*

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Encephalitis and Toxic Hepatitis Caused by Bee Sting: An Unusual Case Report

Arı Sokmasına Bağlı Ensefalit ve Toksik Hepatit: Alışılmadık Bir Olgu Sunumu

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ABSTRACT A bee sting can be a serious problem that affects people all over the world. Several clinical manifestations of bee sting have been described elsewhere. Furthermore, local allergic reactions are more common, causing pain, redness, and swelling of soft tissue within a few hours. In some cases, severe neurological deficits that can lead to death have been reported. According to the literature, the onset of neurological symptoms can range from 30 seconds to 96 hours. Herein, we present the case of a 48-year-old man who developed allergic encephalitis and toxic hepatitis as a result of multiple bee stings.
Keywords: Bee sting, bee venom, encephalitis

ÖZ Arı sokması, dünya çapında görülen ciddi bir tıbbi durumdur. Arı sokması sonrası gelişen çeşitli klinik tablolar tanımlanmıştır. Lokal alerjik reaksiyonlar daha yaygındır ve birkaç saat içinde yumuşak dokuda ağrı, kızarıklık ve şişmeye neden olabilir. Nadiren ölüme yol açabilen ciddi nörolojik defisitler bildirilmiştir. Literatürde bildirildiği üzere nörolojik semptomların başlangıcı 30 saniye ile 96 saat arasında değişmektedir. Burada, birden çok arı sokmasına bağlı gelişen alerjik ensefalit ve toksik hepatit ile başvuran 48 yaşında bir erkek hastayı bildiriyoruz.
Anahtar Kelimeler: Arı sokması, arı zehiri, ensefalit

Introduction

Various clinical manifestations after a bee sting have been described in the literature. Bee stings often cause local allergic reactions. However, various systemic involvements can result in serious complications (1). Anaphylaxis is a serious systemic involvement that causes sudden death. Anaphylactic shock, acute kidney failure, acute pulmonary bleeding, acute myocardial infarction, atrial fibrillation are other unusual systemic manifestations that can occur. Also, there have been prior reports of neurological reactions including epileptic seziures, optic neuritis, exacerbation of multiple sclerosis, brachial plexopathy, acute axonal polyneuropathy, and cerebrovascular disease. The clinical signs of neurological involvement associated with bee sting vary depending on underlying immunological, ischemic or toxic mechanisms (2). Here, we present a case with encephalitis complicated with toxic hepatitis, which is an extremely rare neurological involvement due to bee sting.

Case Report

A 48-year-old male patient was brought to the emergency department as he experienced a sudden loss of consciousness soon after stung by a bee while working in the rural area. His family history revealed subjects with similar systemic reactions after a bee sting. His father died due to systemic complications after bee sting during the follow-up in the intensive care unit. The patient's vital signs were a temperature of 36.7 °C, blood pressure of 130/90 mmHg, and respiratory rate of 16/min. The physical examination revealed localised allergic reaction findings suggesting multiple bee stings at the neck and left arm. On the neurological examination, the patient exhibited

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reduced consciousness with stupor. He had dysarthric speech. Bilateral pupils were of normal size and reacting to light. Neck rigidity and Kerning's sign were positive. The horizontal saccadic eye movements were slow, and partial gaze restrictions were noted. He had quadriparesis with brisk deep tendon reflexes and Babinski sign on the right. Cardiac and respiratory examinations were normal. The biochemistry and hemogram tests were within normal limits. The cerebrospinal fluid (CSF) examination revealed higher protein levels (79 mg/dl). The opening pressure was within normal limits. The CSF colour was bright, and there were no cells. Magnetic resonance imaging showed T2W and FLAIR images hyperintense lesions involving lateral temporal lobes bilaterally suggesting cortical oedema (Figure 1). Electroencephalography was unremarkable. The patient was transferred to the intensive care unit. The patient was diagnosed as encephalitis due to exposure to bee stings. He was treated by antihistaminics, high dose corticosteroids (1 mg/kg/day) and antibiotics. However, his clinical findings showed progression he developed vegetative state and complicated with gastrointestinal haemorrhage on the third day of the follow-up. The massive increase in serum liver enzymes (AST: 880 U / L, ALT: 2200 U / L, GGT: 430 U / L) and abnormal coagulation tests (PTT: 34 sec, APTT: 61 sec, INR: 2.6) were observed. The patient died despite the intervention and supportive treatments.

Discussion

Local allergic reactions due to bee sting in the form of pain, redness and swelling are self-limiting. Sometimes it may represent severe clinical findings. Among these, the most known is anaphylactic shock, with severe clinical conditions such as myocardial infarction, acute pulmonary oedema, gastrointestinal haemorrhages, and acute organ dysfunctions (1,2). Rare neurological clinical findings include ischemic stroke, polyneuropathy, parkinsonism, encephalitis, acute disseminated encephalomyelitis, Guillain-Barré syndrome and toxic encephalopathy (2,3).

The amount of venom injected with the sting of a single bee is 0.33 mg. If more than one bee stings, the amount of venom entering the systemic circulation increases.

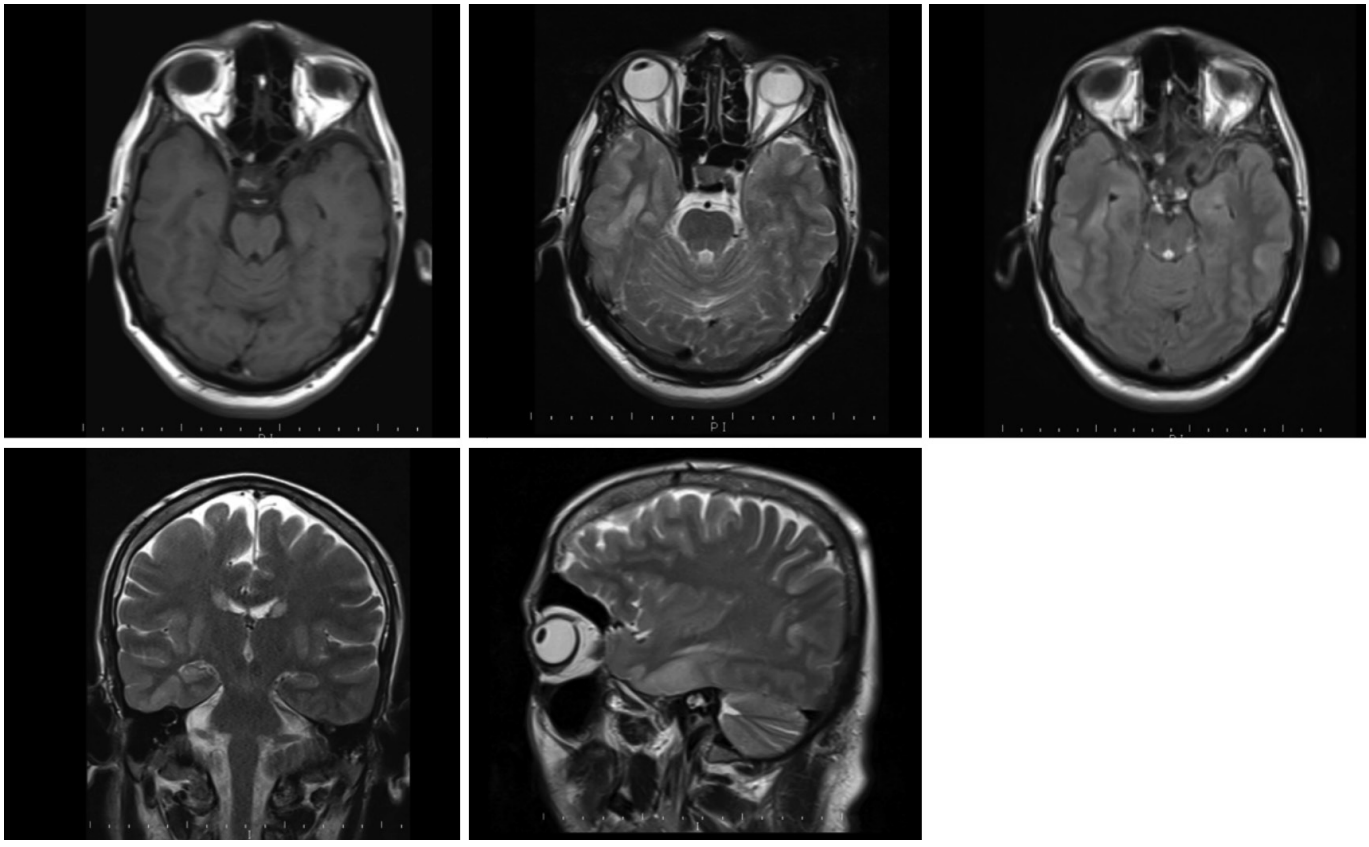

Figure 1. T2W and FLAIR hyperintense, T1W hypointense lesions involving lateral temporal lobes bilaterally suggesting cortical oedema

In our patient, it can be thought that with the sting of more than one bee, more venom enters the systemic circulation and the severity of the clinical picture is related to this condition. Bee venom contains various amines and enzymes (4). More immunological reactions are induced compared to the immune sensitivity of individuals. Our patient had previous exposure to the wasp venom and was likely sensitized. We speculate that previous sensitization to venom cross-reacted with neuronal structures, resulting in the development of encephalitis. Specific IgE antibodies are bound to high-affinity IgE receptors on the surface of mast cells in individuals who become sensitive to the venom of the bee after a bee sting. These surface antibodies that encounter antigen initiate signal transmission by forming bridging. Microflames move the granules towards the microtubules or plasma membrane. These granules are released out of the cell by exocytosis. Various mediators and cytokines are released at different times as a result of the activation of the mast cell. Ready-to-release mediators; Proteases such as histamine, tryptase, chymase, cathepsin G, carboxypeptidase, acid hydrolases and heparin. The mediators that can be released in the early and late stages are leukotrienes B4 and C4, prostaglandin D2, platelet-activating factor (PAF), thromboxane B2, and adenosine. Tumour necrosis factor-alpha, transforming growth factor-beta, and granulocyte-monocyte colony-stimulating factor are released in the late period. Among these products, in the first few minutes, pre-synthesized mediators such as mainly histamine, tryptase, heparin, chymase and newly created mediators such as leukotrienes, prostaglandins, PAF; IL-4 is released at the third hour and IL-13 later. Early released substances are responsible for the vascular manifestations of anaphylaxis, and ischemic stroke, which can be seen as neurological involvement, is a result of this mechanism. Substances released in the late period are responsible for immunological inflammation symptoms (5,6).

Possible mechanism mentioned in neurological involvement; Although Guillain-Barré syndrome is directly associated with immunological damage as in encephalitis and encephalomyelitis and encephalopathies, it may also result from the direct interaction of enzymes and amines such as phospholipases, hyaluronidase, histamine, serotonin, dopamine, norepinephrine, and acetylcholine receptors (7). In the literatüre, there have been only 4 reports of a bee sting-induced allergic encephalitis; 2 in Russia, 1 in India and 1 in USA (8-11). Clinical presentation in one of the reports included headache, generalized seizures and response to steroids was observed (10). The other case with similar complaints, had to be treated by multiple anticonvulsants adding to steroids because of the refractory gelastic seizures (11). In our case, seizures were not observed. Similar to the case of Shasaitov et all., the clinic manifestations of our case developed by more than one bee stings (8). In this case, unlike the other case reports, there were systemic and neurological involvements that occurred as a result of different mechanisms related to multiple bee sting. It is thought that causes of the death are the encephalitis by the immunological mechanisms and secondary coagulation factor deficiency based on toxic hepatitis directly caused by bee venom. Our case is precious as it is a demonstrative presentation showing that neurological and systemic involvement due to bee sting develops with many different mechanisms.

Ethics

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: Ö.Ö., A.H.A., Design: Ö.Ö., A.H.A., Literature Search: Ö.Ö., Writing: Ö.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

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