



Bee Sting-induced Status Epilepticus: Case Report

Arı Sokmasına Bağlı Gelişen Status Epileptikus: Olgu Sunumu

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ABSTRACT

A bee sting can provoke a spectrum of clinical symptoms. Status epilepticus, though rare, is a serious and potentially life-threatening complication associated with bee stings, which can be fatal if not treated promptly. This case report describes an adult patient who developed status epilepticus following a bee sting. The individual presented to the emergency department with generalized swelling and erythema after the sting and experienced a seizure within minutes. When seizures could not be managed with diazepam and levetiracetam, intravenous midazolam was administered at a dose appropriate for general anesthesia, leading to the patient's admission to the intensive care unit. It is essential to remember that status epilepticus, though rare, can develop as a consequence of a bee sting. Physicians should closely monitor patients in the emergency department for four to 12 hours to facilitate the early detection of potential complications.

Keywords: Bee sting, status epilepticus, emergency department

ÖZ

Arı sokması, çeşitli klinik semptomların ortaya çıkmasına neden olabilir. Status epileptikus, nadir görülmesine rağmen arı sokmalarıyla ilişkili ciddi ve potansiyel olarak yaşamı tehdit eden bir komplikasyondur ve zamanında tedavi edilmezse ölümcül olabilir. Bu olgu sunumu, arı sokması sonrası status epileptikus gelişen bir yetişkin hastayı anlatmaktadır. Hasta, arı sokmasının ardından genel şişlik ve eritem ile acil servise başvurmuş ve dakikalar içinde nöbet geçirmiştir. Nöbetler diazepam ve levetirasetam ile kontrol altına alınamadığında, genel anestezi için uygun bir dozda intravenöz midazolam uygulanmış ve hasta yoğun bakım ünitesine kabul edilmiştir. Status epileptikusun nadir de olsa arı sokmasının bir sonucu olarak gelişebileceği unutulmamalıdır. Hekimler, olası komplikasyonların erken tespitini kolaylaştırmak için acil servisteki hastaları dört ila 12 saat boyunca yakından izlemelidir.

Anahtar Kelimeler: Arı sokması, status epileptikus, acil servis

INTRODUCTION

Bee stings can result in a wide range of clinical presentations, from mild to severe. Factors such as the patient's age, underlying health conditions, and the number of stings can affect these presentations¹. Clinically, reactions are categorized as local inflammatory responses (e.g., pain, swelling, itching, rash), allergic reactions (e.g., urticaria, angioedema), anaphylactic

shock, or systemic toxic effects (e.g., myocardial injury, hypertension, liver damage, rhabdomyolysis, hemolysis, coma, and acute renal failure)². Neurological symptoms, though rare, may also occur after bee stings³. These symptoms can arise without accompanying systemic reactions, vary in severity, and often pose diagnostic challenges. Here, we present a case of status epilepticus following a bee sting.

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Received: 21.11.2024 **Accepted:** 23.02.2025 **Publication Date:** 04.03.2026

Cite this article as: Çatal Y, Genç S. Bee sting-induced status epilepticus: case report. Nam Kem Med J. 2026;14(1):100-102

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CASE REPORT

A 50-year-old male patient presented to the emergency department with erythema and swelling in the abdominal and dorsal regions following multiple bee stings. He denied experiencing dyspnea, abdominal pain, chest pain, nausea, or vomiting. His past medical history was unremarkable, with no chronic conditions, previous surgeries, or regular medication use. Upon admission, his vital signs were as follows: body temperature 36.5 °C, heart rate 112 beats per minute, blood pressure 126/79 mmHg, respiratory rate 19 breaths per minute, peripheral oxygen saturation 99%, and blood glucose 84 mg/dL. Physical examination revealed multiple areas of erythema and swelling in the abdomen and back, consistent with bee stings, while the systemic examination showed no abnormalities. During the physical examination, the patient suddenly developed jaw locking, altered consciousness, and generalized muscle contractions, consistent with a generalized tonic-clonic seizure. Airway, breathing, and circulation were promptly evaluated, and oxygen therapy was initiated via mask along with continuous monitoring. Immediate treatment was provided for both anaphylaxis and the seizure. The patient received intramuscular 0.5 mg adrenaline, intravenous (IV) diazepam at a dose of 0.15 mg/kg, and IV corticosteroids. As the seizure continued, additional doses of adrenaline and diazepam were administered. Due to the inability to control the seizure, an IV loading dose of 20 mg/kg levetiracetam was administered. The patient was deemed to be in status epilepticus after failing to stabilize following levetiracetam administration and was subsequently intubated. An IV bolus of midazolam was administered at a dose of 0.1 mg/kg, followed by a continuous IV infusion at 0.1 mg/kg/hour. The administration of midazolam resulted in the cessation of the patient's seizures. Comprehensive laboratory evaluations, including a complete blood count, biochemical panel, coagulation parameters, and venous blood gas analysis, were conducted. The laboratory findings revealed leukocytosis ($18.62 \times 10^3/\text{mL}$) and lactic acidosis (pH: 7.22, lactate: 8.1 mmol/L), while other parameters remained within normal limits: creatinine 1.1 mg/dL, blood urea nitrogen 11 mg/dL, sodium 138 mmol/L, potassium 3.6 mmol/L, calcium 8.4 mg/dL, magnesium 2.5 mg/dL, glucose 122 mg/dL, and hemoglobin 14 g/dL. The patient's electrocardiogram demonstrated a normal sinus rhythm. To exclude differential diagnoses, non-contrast cranial computed tomography and diffusion-weighted magnetic resonance imaging were performed, both of which revealed no intracranial pathologies. An electroencephalogram conducted 48 hours after admission showed normal results, with no evidence of epileptiform activity. The patient was extubated six days after being admitted to the intensive care unit for status epilepticus and was subsequently discharged in good health.

DISCUSSION

Status epilepticus is characterized by the occurrence of two or more consecutive seizures lasting more than 30 minutes, with incomplete recovery of consciousness between seizures⁴. Potential etiologies for status epilepticus include cerebrovascular diseases, central nervous system infections, neurodegenerative disorders, intracranial tumors, cortical dysplasias, head trauma, poisonings, autoimmune disorders, metabolic disturbances, and mitochondrial diseases⁵. Notably, bee stings are not classified as one of these etiological factors.

The management of status epilepticus begins with a stabilization phase, which focuses on ensuring airway patency, as well as adequate respiration and circulation. The subsequent medical treatment is divided into three phases. The first phase is initiated when the seizure duration reaches 5 minutes, with benzodiazepines (midazolam, lorazepam, or diazepam) recommended as the first-line therapy. The second phase begins at the 20-minute mark, with treatment options including fosphenytoin, valproic acid, and levetiracetam. The third phase is initiated when the seizure duration reaches 40 minutes, at which point either the second-line treatment is repeated, or anesthetic doses of thiopental, midazolam, pentobarbital, or propofol are administered⁶.

Various clinical symptoms can arise following a bee sting. In a multicenter study conducted by Xie et al.⁷ which examined 1091 cases of bee stings, it was reported that all patients exhibited a local inflammatory reaction, with 22% experiencing anaphylactic shock, 23% demonstrating two or more organ dysfunctions, and 1.2% presenting with seizures. The literature also includes various case reports of seizures triggered by bee stings^{8,9}. However, the development of status epilepticus has only been reported in a single case, which was associated with allergic encephalitis¹⁰. To the best of our knowledge, this is the first reported case of status epilepticus induced by bee venom without an underlying allergic or encephalitic process. This case highlights the potential neurotoxic effects of bee venom in the central nervous system, suggesting a direct role in the pathogenesis of status epilepticus. These findings underscore the need to consider a broader spectrum of neurological complications associated with bee stings than previously recognized.

Cases involving the central or peripheral nervous system related to bee stings have been documented, including encephalitis, acute inflammatory polyradiculoneuropathy (Guillain-Barré syndrome), acute ischemic stroke, seizures, and intracranial hemorrhage^{9,11-13}. The proposed pathological mechanisms underlying these cases include vasoconstriction and platelet aggregation triggered by exposure to bee venom, direct neurotoxic effects leading to neuronal membrane damage, and delayed autoimmune mechanisms initiated by antigen exposure^{3,14}.

Bee venom comprises at least 18 components, including peptides (melittin, apamin, and adolapin), enzymes (phospholipase A2 and hyaluronidase), biologically active amines (histamine and epinephrine), and non-peptides (lipids, carbohydrates, and free amino acids)¹⁵. These components contribute to the local and systemic reactions observed following a bee sting. Melittin, which constitutes 52% of the peptides, plays a pivotal role in causing prolonged pain, hyperalgesia, and local inflammation¹⁶. Hyaluronidase, accounting for 1-3% of the peptides, facilitates the spread of inflammation by dilating capillaries; histamine, which comprises 0.5-2%, is implicated in allergic reactions; and dopamine and norepinephrine, making up 1-2%, contribute to increased heart rate⁶.

Certain components of bee venom, such as apamin and phospholipase A2, exhibit neurotoxic properties affecting receptors, carriers, and ion channels in neural tissue¹⁷. Florea et al.¹⁸ reported that bee venom induces widespread and irreversible structural changes in both neurons and glial cells in a dose-dependent manner, resulting in cellular death. Gandolfo et al.¹⁹ demonstrated that experimental injection of the phospholipase A2 component of bee venom into mice led to the emergence of neurotoxic symptoms, including stereotypic movements and seizures, which could not be effectively managed with standard antiepileptic medications, gamma-aminobutyric acid receptor blockers, or calcium channel blockers. Similarly, in our case, seizures could not be controlled with conventional antiepileptic therapies.

CONCLUSION

This case highlights status epilepticus as a rare but serious complication of bee stings. Clinicians should remain vigilant for life-threatening conditions such as status epilepticus and anaphylaxis, in addition to the more common local reactions. Therefore, patients should be closely monitored for four to 12 hours after a bee sting, depending on their risk factors, to ensure early detection of potential complications.

Ethics

Informed Consent: Informed consent form was obtained from the patient.

Footnotes

Authorship Contributions

Concept: Y.Ç., Design: Y.Ç., S.G., Data Collection or Processing: Y.Ç., S.G., Analysis or Interpretation: Y.Ç., S.G., Literature Search: Y.Ç., S.G., Writing: Y.Ç., S.G.

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

Financial Disclosure: The authors declared that this study received no financial support.

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