Case Report
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A rare neurological manifestation of seizure in a case of anaphylaxis following a hornet sting: case report and literature review

Eşek arısı sokmasına bağlı anafilaksi vakasında nadir bir nörolojik bulgusu olarak nöbet: olgu sunumu ve literatür incelemesi

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Abstract
Anaphylaxis is a severe, life-threatening allergic reaction that can present with a variety of systemic manifestations. Neurological symptoms such as seizures or seizure-like activity are uncommon in anaphylaxis. We present the case of a healthy young male with no history of seizures who experienced anaphylaxis following a hornet sting, manifested by urticarial rashes, facial flushing, and generalized pruritus. Interestingly, he developed seizure-like activity, characterized by focal jerky movement of the left upper limb with impaired consciousness as reported by eyewitnesses. This case highlights the atypical presentation of neurological manifestations of anaphylaxis, discusses its potential pathophysiological mechanisms, and reflecting individualized management approach in relation to established guidelines.

Keywords: Anaphylaxis, wasp sting, hornet sting, seizure, neurological manifestations.

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Öz
Anafilaksi, çeşitli sistemik belirtilerle ortaya çıkabilen, şiddetli ve yaşamı tehdit eden bir alerjik reaksiyondur. Nörolojik semptomlar, örneğin nöbetler veya nöbet benzeri aktiviteler, anafilakside nadir görülür.
Bu yazıda, eşek arısı sokması sonrası anafilaksi gelişen, nöbet öyküsü olmayan sağlıklı bir genç erkek olgusu sunulmaktadır. Hastada ürtiker döküntüleri, yüzde kızarıklık ve yaygın kaşıntı şeklinde belirtiler gelişmiştir. İlginç bir şekilde, görgü tanıklarının aktardığına göre, bilinç bozukluğu ile birlikte sol üst ekstremitede fokal sarsıntılı hareketlerle karakterize nöbet benzeri bir aktivite gözlemlenmiştir.
Bu olgu, anafilaksinin atipik nörolojik belirtilerle seyredebildiğini vurgulamakta ve bu semptomların olası patofizyolojik mekanizmaları üzerine tartışma sunmaktadır.

Anahtar kelimeler: Anafilaksi, yaban arısı sokması, eşek arısı sokması, nöbet, nörolojik bulgular.

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Introduction

Anaphylaxis is a severe, life-threatening systemic hypersensitivity reaction characterized by the rapid onset of multiple organ involvement, often requiring immediate medical intervention. Common triggers include food, insect venom, medications, and latex, leading to widespread immune activation and inflammatory mediator release [1].

The pathophysiology primarily involves the activation of mast cells and basophils through immunoglobulin E (IgE)-mediated pathways. Upon allergen exposure, cross-linking of IgE antibodies bound to mast cells leads to the degranulation of inflammatory mediators, including histamine, leukotrienes, and prostaglandins, which drive the clinical symptoms [2].

Histamine increases vascular permeability, leading to edema and hypotension, while leukotrienes contribute to bronchoconstriction and airway compromise [3].

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Additionally, non-IgE-mediated mechanisms, such as direct mast cell activation by drugs (e.g., opioids, radiocontrast agents), can also trigger anaphylaxis [4].

Hornet venom, a potent allergen, contains vasoactive amines, mast cell degranulating peptides, and neurotoxins that can induce an exaggerated immune response in susceptible individuals [5].

Clinically, anaphylaxis presents with a spectrum of manifestations that can involve the skin (urticaria, angioedema), respiratory system (bronchospasm, stridor, airway edema), cardiovascular system (hypotension, syncope), and gastrointestinal tract (nausea, vomiting, diarrhea) [6].

Neurological manifestations, though less commonly reported, may include dizziness, confusion, and even seizure-like activity, often attributed to hypoxia or hypotension [7].

Systemic inflammation and immune-mediated neuronal effects may contribute to neurological symptoms. Additionally, direct neurotoxicity from venom components warrants further study [8]. Reported neurotoxic effects include encephalopathy, hemorrhagic strokes, encephalitis, and grand mal seizures. However, delayed presentations suggest a secondary antigen-triggered autoimmune process in some cases [9].

While cardiovascular and respiratory symptoms are the hallmark features, neurological manifestations are exceedingly uncommon.

In this report, we describe a rare presentation of anaphylaxis in a previously healthy young male who developed seizure-like activity following a hornet sting, emphasizing the need for heightened awareness of atypical neurological symptoms in anaphylaxis.

Case report

A 34-year-old male with a prior history of anaphylaxis secondary to a hornet sting two years earlier presented to the emergency department following another hornet sting on his left forearm. Within minutes, he developed urticarial rashes, facial flushing, and generalized pruritus. Approximately 30 minutes after the sting, witnesses observed focal jerky movements of the left upper limb with transient loss of consciousness lasting about one minute. Upon arrival, he was fully alert and hemodynamically stable (BP 130/80 mmHg, HR 86 bpm, SpO2 99% RA). Physical examination revealed facial flushing and urticarial rashes without airway or cardiovascular compromise.

Laboratory investigations and CT brain were normal. The patient was treated with intravenous hydrocortisone (200 mg) and chlorpheniramine (10 mg). Intramuscular adrenaline was withheld due to the absence of other systemic compromise and the need to exclude neurological causes of seizure. He was closely monitored in the resuscitation area and later admitted for observation. His symptoms resolved without recurrence, and he was discharged well.

Discussion

Anaphylaxis is a systemic hypersensitivity reaction that can rapidly progress to life-threatening complications involving multiple organ systems [1]. While cardiovascular and respiratory symptoms are the hallmark manifestations, this case highlights the rare occurrence of seizure-like activity in anaphylaxis, likely due to hypoxia, hypotension, and/or direct neurotoxic effects of hornet venom [2]. Several mechanisms could explain the seizure-like activity observed in this case:

1) Cerebral Hypoxia

Severe hypotension and impaired cerebral perfusion during anaphylaxis can result in transient cerebral hypoxia, which may manifest as confusion, loss of consciousness, or seizure-like activity. The rapid onset of systemic vasodilation and increased vascular permeability, primarily driven by histamine and leukotrienes, leads to hypoperfusion of the brain [8].

2) Mast Cell Mediator Effects on the Central Nervous System (CNS)

Histamine and tryptase, which are released during mast cell degranulation, have been implicated in neurological hyperexcitability. Histamine receptors (H1 and H3) in the brain

influence neurotransmission and may contribute to seizure-like activity [4]. Furthermore, increased permeability of the blood-brain barrier (BBB) during systemic inflammation may allow immune mediators to enter the CNS, exacerbating neuroinflammation and neuronal dysfunction [10].

3) Direct Neurotoxic Effects of Hornet Venom

Hornet venom contains a mixture of mastoparan, neurotoxins, and vasoactive amines, which can disrupt neuronal function and induce neuroinflammation [5]. The mechanism of neurotoxicity following Hymenoptera envenomation is believed to be secondary to direct effects of toxins including low-molecular-weight kinins leading to neuronal membrane irritability [9].

A variety of neurological manifestations have been reported following anaphylaxis due to Hymenoptera stings, with ischaemic stroke having the highest prevalence. A literature report stated that 26 cases of cerebral infarction following a bee or wasp sting since the initial index case occurred in 1962 [11].

Table 1 highlights the most recent reported case representing each distinct neurological diagnosis, providing an overview of the spectrum of central nervous system involvement associated with such reactions.

Table 1. Spectrum of neurological complications of Hymenoptera sting

Author & Year	Age / Gender	Type of Sting	Neurological manifestations	Neurological diagnosis
Du et al., (2017) [12]	66/F	Wasp	Agitation, generalized seizures with loss of consciousness	Posterior reversible encephalopathy syndrome (PRES)
Maramattom, (2021) [13]	48/M	Wasp	Headache, disorientation & seizures	Allergic encephalitis
Si et al., (2022) [14]	47/M	Wasp	Blurred vision, and weakness in the extremities & epileptic seizures	Acute posterior circulation toxic encephalopathy
Li et al., (2023) [15]	60/M	Wasp	Headaches	Subarachnoid haemorrhage (SAH)
Zhang et al., (2024) [16]	59/M	Wasp	Right hemiparesis	Cerebral venous sinus thrombosis (CVST)
Kaur et al., (2024) [17]	26/M	Wasp	Bilateral upper & lower limb weakness	Acute inflammatory demyelinating polyneuropathy (AIDP)
Tomic et al., (2024) [18]	70/M	Hornet	Parkinsonism	Rapid onset parkinsonism
Thavara et al., (2025) [19]	49/M	Wasp	Right hemiparesis	Ischaemic stroke

Management considerations

Anaphylaxis can rapidly progress to life-threatening complications, and early administration of intramuscular adrenaline is the cornerstone of treatment.

However, this case presented an unusual diagnostic challenge due to the presence of seizure-like activity without hemodynamic instability, necessitating a cautious and individualized management approach.

In this case, intramuscular adrenaline was withheld due to the atypical presentation of seizure-like activity, which is a rare manifestation of anaphylaxis. The occurrence of a focal seizure in the absence of cardiovascular or respiratory compromise raised the possibility of an alternative or concurrent neurological pathology. This consideration was further supported by the fact that the patient had experienced a prior anaphylactic episode following a hornet sting without any neurological involvement. Although immediate intramuscular adrenaline is recommended as first-line therapy in anaphylaxis, it can acutely raise blood pressure and cerebral perfusion. In the presence of an undiagnosed intracranial hemorrhage, it could theoretically worsen the bleeding. Therefore, a cautious approach was adopted, which prioritize stabilization, neurological assessment and brain imaging to exclude other potential causes of seizure before attributing the event solely to anaphylaxis.

International guidelines, including those from the World Allergy Organization (WAO, 2020) [20], the American Academy of Allergy, Asthma & Immunology (AAAAI, 2020) [21], and the European Academy of Allergy and Clinical Immunology (EAACI, 2021) [22], uniformly emphasize early intramuscular adrenaline administration as the first-line therapy for anaphylaxis, regardless of hemodynamic or respiratory compromise. However, these recommendations are largely based on classic presentations involving airway compromise, hypotension or circulatory collapse.

In this case, the patient exhibited only cutaneous manifestations in the absence of other systemic involvement, mainly respiratory, cardiovascular or gastrointestinal manifestations. The presence of a transient seizure-like episode, which is an uncommon neurological manifestation in anaphylaxis, warranted further evaluation to exclude alternative etiologies such as intracranial pathology or metabolic disturbances. Excluding this neurological event, the remaining clinical features were consistent with a mild-to-moderate systemic allergic reaction rather than a severe anaphylactic episode. Therefore, a cautious approach with close monitoring was considered appropriate while diagnostic clarification was underway.

This case underscores the importance of individualized clinical judgment when managing atypical presentations of anaphylaxis. While adherence to established guidelines remains essential, clinicians must also consider alternative diagnoses and the potential risks of therapy in complex scenarios, particularly when neurological symptoms predominate. The favorable outcome in this patient, who remained hemodynamically stable throughout observation, illustrates that a cautious, evidence-informed approach can be appropriate in selected cases.

On the other hand, although the patient remained hemodynamically stable, delayed administration of adrenaline could theoretically worsen outcomes if hypotension or airway compromise had developed. Nevertheless, this report reinforces that deviations from standard management should always be cautiously justified and discussed within the context of current clinical guidelines, as summarized in Table 2.

Table 2. Comparison of international anaphylaxis guidelines and current case management

Guideline / Source	Recommended Management
WAO (2020) [20]	IM adrenaline 0.3-0.5 mg as first-line treatment for all suspected anaphylaxis
AAAAI (2020) [21]	Adrenaline is mandatory first line; adjuncts such as antihistamines and corticosteroids are secondary
EAACI (2021) [22]	Early IM adrenaline even in mild cases without hypotension or airway symptoms
Current Case Reflection	IM Adrenaline was withheld, only antihistamine & corticosteroid given. Watch and observe approach

Consequently, clinicians should be aware of possible neurological manifestations following hornet stings, as venom components may directly affect the nervous system. Future investigations are needed to better characterize the short- and long-term neurological outcomes of severe anaphylactic reactions and to explore the role of adjunctive treatments addressing neuroinflammatory mechanisms.

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