

Ischemic Stroke After Bee Sting: A Case Report

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Abstract

The global impact of a bee sting extends beyond common perceptions, showing various indications that may range from local reactions to more severe complications. This report details a compelling case involving a 63-year-old male who developed neurological deficits merely six hours after a bee sting. A 63-year-old male patient was brought to the emergency department because of widespread redness, pain and itching at the sting area after a bee sting while pruning roses in the garden. During the follow-up in the emergency room, the patient developed weakness in his right lower/upper extremities. However, the diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle. Various clinical presentations after a bee sting have been described in the literature. Bee sting cases are generally associated with mild and transient local symptoms. Sometimes life-threatening allergic reactions may occur. The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

Keywords: Bee sting, ischemic, stroke

Introduction

The global impact of a bee sting extends beyond common perceptions, showing various indications that may range from local reactions to more severe complications such as vomiting, diarrhea, dyspnea, generalized edema, acute renal failure, hypotension, and collapse (1). The uncommon but documented after-sting complications such as vasculitis, serum sickness, neuritis, and encephalitis often develop within days to weeks following the initial incident (2).

Amid these considerations, the foremost concern is the potential occurrence of anaphylaxis life-threatening reaction with mortality implications. The documented incidence of anaphylaxis (3) resulting from bee stings varies within the range of 0.4% and 5%. Notably, severe allergic reactions leading to mortality typically surface before the age of 20, with a twofold prevalence observed in the male population (4).

While classical local allergic reactions following a bee sting are commonplace, their diagnosis relies heavily on a thorough patient history. In the current case, the medical history and physical examination findings are consistent, pointing to a presentation consistent with a local allergic reaction. Recognizing and understanding such reactions are

crucial for prompt and effective medical intervention.

In addition to the complexity, the neurologic symptoms following bee stings are infrequently documented in the literature, with cases primarily emphasizing encephalitis and acute disseminated encephalomyelitis. The occurrence of a stroke directly related to a bee sting is even rarer, with the precise etiology remaining elusive. This report details a compelling case involving a 63-year-old male who developed neurological deficits merely six hours after a bee sting. The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

Case

A 63-year-old male patient was brought to the emergency department because of widespread redness, pain and itching at the sting area after a bee sting while pruning roses in the garden, with a medical history of hypertension, type 2 diabetes, and coronary artery disease. He denied alcohol or tobacco use.

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Received: 25.02.2024 • **Revision:** 18.03.2024 • **Accepted:** 14.04.2024

DOI: 10.33706/jemcr.1442868

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Cite this article as: Hamdiođlu E, Ataş İ, Yazıcı MM, Bilir Ö. Ischemic Stroke After Bee Sting: A Case Report. Journal of Emergency Medicine Case Reports. 2024;15(2): 50-52

Upon arrival for the ER, the vital signs of the patient were as follows: blood pressure (BP):130/80 mmHg, heart rate (HR) 75 beats/min, respiratory rate (RR) 14/min, fever: 36.4 C° oxygen saturation: 98% in room air. His general condition was alert and oriented, the neurologic examination and, the mental-status examination was normal, Glasgow Coma Scale (GCS) was 15 (E4, V5, M6). In the other physical examinations, there was observed no tachycardia, no tachypnea and no murmur-rubbing-galloping. His respiratory system examination was clear according to the bilateral auscultation. The remainder of his physical examination was normal. After the physical examination, the laboratory tests were requested, and the symptomatic treatment was initiated. During the follow-up in the emergency room, the patient developed weakness in his right lower/upper extremities. In the control neurological examination, his mental status examination was normal, and the GCS score was 15. The patient developed right central facial paralysis, a 3/5 loss of strength in his right upper/lower extremities, and a positive Babinski sign on the right side. As the current clinical presentation changed, a non-contrast brain CT scan was performed, and no acute pathology was detected. However, the diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle (Figure-1). In response to the detection of an acute ischemic lesion in the imaging tests, rt-PA (recombinant human tissue-type plasminogen activator) was administered in accordance with the recommendations of the neurology consultant physician. The patient's relatives were informed about the treatment. However, since it was not accepted, the patient was administered oral (PO) medication, which included 300 mg of acetylsalicylic acid and 0.6 mg of LMWH (Low Molecular Weight Heparin) Enoxaparin. The patient was admitted to the neurology clinic and hospitalized. However, the patient was discharged on the 5th day of the hospital stay.

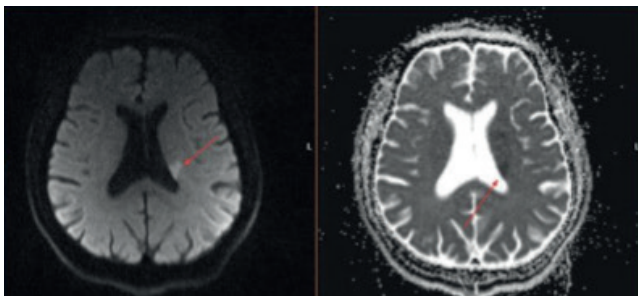


Figure 1. Diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle (red arrow)

Discussion

Various clinical presentations after a bee sting have been described in the literature. Bee sting cases are generally associated with mild and transient local symptoms. Sometimes life-threatening allergic reactions may occur. Classical local allergic reactions occur after bee sting, and the diagnosis depends on the medical history. In our case, the medical history and physical examination findings are consistent with the local allergic reactions. Many bites may cause urticaria and systemic toxicity including cardiovascular and neurologic problems (5). The reactions by bee sting are frequently mediated by IgE, but these reactions may also occur by IgG antibody and IgG venom complex triggered complement activation. The symptoms usually occur within the first few hours, but they can be observed even after hours. The connection between bee stings and strokes is an interesting topic, and there are potential mechanisms through which bee venom might influence the risk of stroke. However, it is important to note that research in this area may be limited, and the information available might not be conclusive.

Pathophysiologically, it is said that several mechanism scan cause this condition. Bee stings may trigger severe allergic reactions (anaphylaxis) in susceptible individuals. Anaphylaxis may lead to hypotension (low blood pressure), which, in turn, may affect cerebral blood flow and increase the risk of ischemic events such as strokes. Bee venom contains various inflammatory mediators, including histamine, thromboxane, and leukotrienes. These mediators may contribute to a hypercoagulable state and potentially promote conditions favorable to strokes. Vasoactive substances in bee venom may influence blood vessel tone and reactivity. Changes in blood vessel dynamics may impact the risk of stroke, particularly if there is a predisposition to vascular events (6-8). According to many studies, patients should be observed for a period of at least 6 hours (9,10). In our case, reactions were observed after the first hour and minimized through the treatment by the medical teams, but at the sixth hour of the follow-up, neurologic symptoms were developed.

Our patient had no previous medical history of epilepsy or cerebrovascular disease and here, we present ischemic stroke after bee sting, which is an extremely rare neurological involvement due to bee sting.

Conclusion

The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory

to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

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