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## CASE REPORT

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# A rare case of facial nerve paralysis by apical bee stings

Anagha P Rao, Ishwar S Hasabi, Kalinga B E and Vishwanath Patil

Rao Anagha P. et al. A rare case of facial nerve paralysis by apical bee stings. *Med Toxicol Curr Res* 2024; 7(1):1-3

### ABSTRACT

**Introduction:** Apical bee stings are common scenarios in modern clinical practice in India. The clinical manifestations vary widely from simple allergic reactions to life-threatening anaphylaxis. Despite the large number of notified bee sting cases, among the neurological symptoms, peripheral facial nerve palsy is a rare manifestation.

**Case report:** A 37-year-old lady developed isolated lower motor neuron-type facial paralysis on the sixth day of bee stings, a rare delayed neurological manifestation. After a course of systemic glucocorticoid therapy, she recovered successfully.

**Discussion:** Bee sting manifestations are classified as early or late reactions. IgE-related mediators like histamine, proteases, and thromboxanes facilitate early reactions. Late manifestations are perceived to be non-IgE-mediated immunological responses (type III hypersensitivity) to Hymenoptera venom involving deposition of immune complexes and complement activation. The mainstay therapy involves early and short-term use of steroids to inhibit and reduce the allergic reaction, prevent necrosis of tissues, relieve the stress response to bee venom, and minimize neuritis.

**Keywords:** Bee stings; Facial nerve paralysis; Venom; Steroids

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### INTRODUCTION

Bee stings are common insect bites in modern clinical practice in India. The spectrum of bee sting-related clinical presentation ranges from mild local reactions to life-threatening anaphylaxis [1].

These clinical manifestations are classified as early or late reactions. Early reactions start from a few minutes to a few hours of the bee stings while late reactions usually occur after 6 days-10 days [2]. These reactions are facilitated by IgE-related mediators like histamine, proteases, and thromboxanes [1].

Facial nerve paralysis is an uncommon late neurological complication following bee stings. This case report discusses a young lady who presented with anaphylaxis after bee sting over face and later developed facial nerve palsy with a successful clinical recovery in our hospital.

### CASE PRESENTATION

A 37-year-old lady presented to the emergency department with an alleged history of a swarm of apical bee stings. She had skin itching, swelling, and burning sensation all over the face, head, neck, upper back, and both arms and angioedema within a few hours of bee stings. She was admitted for obvious facial swelling, drooling of saliva,

and threatened airway compromise for close monitoring.

On admission, she had a temperature of 36.8°C, pulse rate of 88 beats/min, respiratory rate of 22 cycles/min, blood pressure of 110/60 mmHg, SpO<sub>2</sub> of 96% on room air, and GRBS of 110 mg/dL. Local examination revealed scattered needle-like wounds over exposed parts of the vertex, occiput, face, neck, upper back, both arms, lower legs, and feet. The wounds over the left half of her face and left ear were extensive. The otorhinolaryngological examination performed was unremarkable. Her systemic examination was unremarkable.

She was promptly treated with IV hydrocortisone, IV chlorpheniramine, IM adrenaline (1:1000), and a course of IV Amoxyclav. Fluid and electrolyte balance were maintained.

Her laboratory results showed leucocytosis of 15,500 cells/dL, absolute neutrophilia of 11,000/dL, and mild anemia of Hb 11.0 g/dL. A routine urine test showed urinary protein +1 with no active urinary sediments. Liver function, renal function, and serum electrolytes were normal. Her ultrasonography of neck and parotid swelling revealed minimal cellulitic changes in both the submandibular and parotid regions with few enlarged lymph nodes in bilateral levels 1a and 1b with no evidence of focal collection or abscess.

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Department of General Medicine, Karnataka Institute of Medical Sciences, Hubballi, India

\*Correspondence: Rao Anagha P, Department of General Medicine, Karnataka Institute of Medical Sciences, Hubballi, India E-mail: [anagharao11@gmail.com](mailto:anagharao11@gmail.com)

Received: 27-Feb-2024, Manuscript No. Pulmtcr-24-6930; Editor assigned: 01-March-2024, PreQC No. Pulmtcr-24-6930 (PQ); Reviewed: 14-March-2024 QC No. Pulmtcr-24-6930 (Q); Revised: 22-March-2024, Manuscript No. Pulmtcr-24-6930 (R); Published: 27-March-2024, DOI No. 10.37532/pulmtcr.2024.7(1)1-3



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With symptomatic supportive therapy, she gradually improved. Her facial cellulitis, neck and arm swelling subsided significantly and her pain resolved.

On the sixth day of admission, she developed facial deviation to the right side and incomplete closure of the left eyelid. Upon examination, she had left-sided lower motor neuron type of facial nerve palsy with left side Bell's phenomenon. Her otoscopic examination was unremarkable. We suspected facial nerve injury secondary to the bee venom. She continued to receive IV steroids for the same and her symptoms gradually improved. She was later discharged and was advised to follow up in the outpatient department Figure 1 and 2.



Figure 1) Loss of prominence of left nasolabial fold.



Figure 2) Bell's phenomenon of left eye

## DISCUSSION

Bees belong to the order Hymenoptera and those which sting, are mainly honey bees, wasps, and hornets [3]. Bee tail stings are connected to the venomous glands. When a person is stung, the toxins in the venomous glands are injected into the skin through the stings, causing local or systemic reactions [4].

Bee venom consists of a complex of chemicals mainly histamine, various enzymes, formic acid, neurotoxin, and hemolytic toxin that affect various tissues [3]. Numerous deaths have been reported after one or more stings, highlighting the lethal effects of envenomation.

The clinical manifestations caused by bee stings are related to:

1. Amount of bee venom entering the human body.
2. Site of the sting.
3. Presence of an allergic reaction [5].

A single bee sting causes only mild local redness, itching, and pain that often heal itself without residual dysfunction. However, if multiple sites are stung, systemic toxicity occurs, predominantly resulting in liver and renal dysfunction, myocardial toxicity, arrhythmias, neuropathies, and encephalopathy (Table 1) [6, 7].

**TABLE 1**  
**List of systemic manifestations of apical bee stings**

<b>Neurological</b>	Peripheral neuritis, Cerebral infarction, Guillain-Barre syndrome, Ocular myasthenia gravis, Optic neuropathy, Encephalopathy
<b>Myocardium</b>	Myocardial infarction, Heart failure, Myocarditis
<b>Musculoskeletal</b>	Rhabdomyolysis, Compartment syndrome
<b>Renal</b>	Acute kidney injury, Acute immune-mediated interstitial nephritis, Acute tubular necrosis, Nephritic syndrome
<b>Hematological</b>	Hemolysis, hemoglobinuria, Coagulopathy, Vasculitis
<b>Others</b>	Liver injury, pulmonary hemorrhage, Multisystemic organ dysfunction

The main pathogenesis behind the clinical severity of a bee sting includes:

1. The allergenic proteins in the bee venom such as phospholipases induce mast cell activation and IgE reaction [8].
2. The vasoactive, inflammatory, and thrombogenic effects are determined by histamine and other newly synthesized mediators namely, apamin, mast cell degranulating peptide, hyaluronidase, acid phosphatase, norepinephrine, and dopamine [8].
3. The neurotoxin of bee venom "melittin" is a peptide with

two disulfide bonds. It has toxic effects on both the central and peripheral nervous systems and is responsible for late neurological manifestations [3].

On the 6<sup>th</sup> day of the bee stings, our patient developed isolated left-sided lower motor neuron-type facial paralysis. As this was a delayed manifestation, it is presumed to be a non-IgE-mediated immunological response (type III hypersensitivity) to hymenoptera venom involving deposition of immune complexes and complement activation [9].

The exact pathogenesis of facial paralysis is unknown. However, possible mechanisms include:

1. The stings were densely distributed in the area of the left facial nerve. Melittin inhibits the activity of Na<sup>+</sup>-K<sup>+</sup>-ATPase on the nerve synapse by inhibiting the nerve-muscle synaptic transmission, causing a weak curare-like effect and impeding the transmission of ganglion to inhibit the peripheral nerve resulting in facial nerve paralysis [9].
2. Bee venom is composed of many inflammatory proteins particularly apamin - a potent neurotoxin known to inhibit calcium-dependent potassium channels in the brain and spinal cord, thereby impeding the transmission of neural signals to peripheral nerves [9].
3. Honey bee venom contains allergenic proteins that induce the production of IgE antibodies. These antibodies cross-react with myelin basic proteins and cause various neurological symptoms, including facial nerve palsy [10].

A similar case reported in Sri Lanka of facial paralysis occurred within 24 hours of the bee sting as reported by Izzathunnisa. Another case reported in Turkey by Yıldız where facial palsy occurred within 2 hours of the bee sting. In contrast, a case reported in China by Tang of facial palsy was delayed until the 7<sup>th</sup> day of the bee sting similar to our case where it manifested on the 6<sup>th</sup> day [2, 3].

Early onset of facial weakness in the above-mentioned former two studies was probably IgE mediated and in our case as well as in that of Tang et al is attributable to non-IgE mediated reactions.

However, the mainstay therapy in all the above studies was an early and short course of systemic glucocorticoids to inhibit and reduce the allergic reaction, prevent necrosis of tissues, relieve the stress response to bee venom, and minimize neuritis.

### CONCLUSION

Bee stings are the most common insect bites manifesting from local reactions to multiple systemic organ dysfunction. Despite the large number of notified bee sting cases, among the neurological symptoms, peripheral facial nerve palsy is a rare manifestation.

This case report adds facial paralysis to the list of clinical manifestations after bee stings that require close observation for carrying out timely intervention, such as in our case, who received timely glucocorticoid therapy resulting in successful clinical recovery.

### CONSENT FOR PUBLICATION

The patient has given consent to publish the case with her photographs.

### AUTHOR CONTRIBUTIONS

The corresponding author wrote the idea for the manuscript and its substance. Co-authors have fully contributed to the idea, design, analysis, and drafting of the case report with the help of the corresponding author.

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