Bee sting induced facial nerve palsy

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Abstract

Bee stings are one of the common clinical scenarios in day-to-day clinical practice in Sri Lanka. The spectrum of bee sting-related complications can vary from simple allergic reactions to life-threatening conditions and sometimes death. Anaphylaxis, cardiogenic shock, respiratory arrest, acute renal failure, vasospasm-related ischaemia, and acute pulmonary oedema are some of the life-threatening conditions. Neurological manifestations such as acute cerebral infarction, acute demyelinating polyneuropathy, encephalitis, transverse myelitis, and optic neuritis are also reported. We report a rare presentation of unilateral facial nerve palsy following bee stings in a 63-year-old woman, who fully recovered with treatment.

Key words: bee sting, peripheral facial paralysis, histamine, venom, steroids

Introduction

Bee stings are a frequent presentation in clinical practice in Sri Lanka. The complications following bee stings could be broadly divided into early and late reactions.[1,2,3] Early reactions can vary from simple allergic reactions to anaphylactic shock. Late reactions can be delayed up to 10 days and their manifestations could vary according to the system involved. The clinical outcome and the extent of the reactions differ with IgE levels. Some are IgE-dependent and others are IgE-independent reactions.[1,4,5] IgE-related mediators like histamine, proteases, and thromboxanes play a role in IgE-dependent reactions. Antihistamines, steroids, adrenaline, and venom immunotherapy are widely used treatment options.

Case presentation

A 63-year-old woman was admitted to the emergency department with a complain of deviation of the face to the left side for one-day duration. She had multiple bee stings the previous day while working in the garden. She developed itching, pain, and swelling on the right side of the face and around the ears soon after. Immediate medical advice was sought at the local hospital as an outpatient where she was given antihistamines and analgesics. The following day she noticed drooling from the right side of the mouth and deviation of the mouth to the left side. Her ear pain and facial edema on the right side persisted despite treatment.

There were no features of limb weakness, numbness, slurring of speech, headache, or diplopia. She denied any recent infections, falls, or trauma. She had no other comorbidities including diabetes or hypertension.

On admission, pulse rate was 80 beats per minute, blood pressure was 120/70 mmHg and respiratory rate was 18 per min. The pupils and the optic fundi were normal. There was a lower motor neuron type facial nerve palsy on the right (Figure 1). All other cranial nerves, upper limbs, lower limbs, and cerebellar examination were normal. Examination of the ear was normal except for mild edema with redness around the right ear. There was no rash suggestive of herpes zoster, particularly in the external auditory canal or face. The rest of the clinical examination including the cardiovascular, respiratory, abdomen, and muscular skeletal systems were normal.

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Figure 1. Right lower motor neuron facial nerve palsy with the deviation of mouth.

Her investigations including full blood count, ESR, CRP, HbA1c, and fasting blood sugar were normal. Serum creatinine, serum electrolytes, and serum calcium were within normal limits.

There were no abnormalities noted in the ECG. Non-contrast CT brain was normal without any ischemic or hemorrhagic changes specifically in the pontine area. The pure tone audiogram and tympanogram were normal.

She was started on oral prednisolone 60mg daily and tapered off over a period of two weeks with a combination of antihistamines and analgesics. Physiotherapy was initiated with Transcutaneous Electrical Nerve Stimulation (TENS) and eye protection. She improved gradually and she was discharged on the third day of admission with the plan to review her as an outpatient. She made a complete recovery in 4 weeks.

Discussion

Bee venom consist of a complex of chemicals that affect various tissues. Numerous deaths have been reported after one or more stings, highlighting the lethal effects of envenomation.

The allergic mechanisms due to bee stings are brought on by pollen adhering to the bee, sensitization to allergens present in the bee, and the chemicals of the venom. These are the three main elements that define the severity of the clinical effects of a bee sting in a patient.[4] Bee venom contains allergenic proteins including phospholipases that cause mast cell activation by inducing an IgE reaction.[5] The vasoactive, inflammatory, and thrombogenic effects are determined by histamine and other newly synthesized mediators including apamin, melittin, mast cell degranulating peptide, hyaluronidase, acid phosphatase, norepinephrine, and dopamine.[5]

Acute reactions typically start between minutes to hours in 76% to 96% of envenomation. However, a small percentage of people could encounter various delayed reactions that happen days to weeks after the event.[1,2] The acute complications can range from a straightforward simple allergic reaction to life-threatening anaphylactic shock. Serum sickness, renal involvement, neurological symptoms, hepatic dysfunction, and delayed hypersensitivity phenomena are a few of the uncommon, delayed reactions reported in the literature.[3,6-9]

Several case reports of neurological manifestations, including peripheral neuritis, cerebral infarction, Guillain-Barre syndrome, ocular myasthenia gravis, optic neuropathy, and encephalopathy have been described in the literature despite the rarity of these symptoms.[2,3,6-11] A rare symptom of bee stings is isolated facial nerve paralysis, which was previously reported in Turkey.[12] No cases have been documented in Sri Lanka yet, and to our knowledge, this may be the first case report. Twenty-four hours after being stung by a bee, our patient developed isolated lower motor neuron-type facial paralysis. The most likely mechanisms in our patient is underlying neuritis brought on by local toxic effects of venom as well as the hypersensitivity reactions.[4,10-11]

Steroids and antivirals are recommended to treat primary facial paralysis. Considering the secondary

causes of peripheral facial paralysis, eliminating the underlying cause is the primary management strategy. In addition to steroids our patient was treated with antihistamines to decrease IgE-mediated histamine production. In four weeks, she made a complete recovery without any further neurological deficits.

Conclusion

Each year clinicians deal with a significant number of cases of bee stings in Sri Lanka. Local responses are frequently encountered in these patients at the time of presentation. Systemic signs and symptoms, including anaphylaxis, also occur frequently. Neurological symptoms are extremely rare. Peripheral facial nerve palsy is a rare presentation following bee stings that can be treatable. In cases of bee stings, treating clinicians should be mindful of the uncommon but potential neurological complications and their consequences.

Author declaration

Conflict of interest

There are no conflicts of interest.

Consent for publication

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

Author contributions

The idea for the manuscript and its substance were written by the corresponding author. 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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