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CASE REPORT

PERIPHERAL FACIAL PARALYSIS DEVELOPING AFTER A BEE STING: A CASE REPORT

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ABSTRACT

Bee stings usually cause mild local reactions; however, rare neurological complications, including peripheral facial paralysis, may occur. We present a case of peripheral facial paralysis following a bee sting. A 62-year-old male presented with left-sided facial asymmetry two days after being stung in the left infraorbital and preauricular region. Neurological examination revealed left-sided House-Brackmann grade II peripheral facial paralysis without additional neurological deficits, and central causes were excluded clinically. The patient had well-controlled diabetes mellitus. Oral methylprednisolone and supportive therapy were initiated. Significant clinical improvement was observed on day ten, and complete recovery was achieved by the third week. Inflammatory edema, neurotoxic effects, and microvascular ischemia may contribute to the pathogenesis. Bee sting should be considered a rare cause of peripheral facial paralysis, and early corticosteroid therapy may result in complete recovery.

Keywords: Bee sting, case report, corticosteroid, facial palsy, peripheral facial paralysis

INTRODUCTION

Bee stings are highly prevalent insect stings in humans. In the 16-65 year age group, the cumulative lifetime prevalence of bee stings is between 61% and 95% (1). Complications developing after bee stings are classified as early and delayed reactions. While early reactions may range from mild local allergic responses to severe anaphylactic shock, delayed reactions may occur up to ten days later and patients may present with different clinical manifestations depending on the affected system. The clinical course and severity of these reactions may vary depending on the type of immune response and the patient's sensitivity. Various mediators such as histamine, proteases, and thromboxanes play a role in this process. Antihistamines, corticosteroids, adrenaline, and venom immunotherapy, when necessary, are used in treatment (2).

Facial paralysis has a wide etiological spectrum, including infectious, neurological, congenital, neoplastic, traumatic, systemic, and iatrogenic causes (3). Peripheral facial paralysis (PFP) is the most prevalent form of facial paralysis and is caused by the impairment of the facial nerve along its pathway from the brainstem to the peripheral branches. The estimated incidence is 20-30 cases per 100,000 population, with approximately 40,000 new cases each year worldwide (4). Regardless of etiology, the management of facial paralysis requires a multidisciplinary approach, and the prognosis varies depending on the degree of nerve injury (5).

In this case report, a rare case of PFP that developed following a bee sting is presented.

CASE REPORT

A 62-year-old male patient presented with the complaint of deviation in the left half of the face two days after a bee sting. Following the bee sting, the patient applied ice to the affected area to manage localized erythema, increased warmth, edema,

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and tenderness. No medical treatment was administered. According to the patient's history bee stings occurred in the left infraorbital and preauricular region (Figure 1). In the patient's medical history, the patient had no other known history of allergies, there was diabetes mellitus under regular follow-up, and it was stated that glycemic control was regular.

In the otorhinolaryngological examination, otoscopic and nasopharyngeal evaluation were unremarkable. In the neurological examination, findings compatible with left-sided House-Brackmann grade II (Table 1) PFP were observed (6). There was mild weakness in eye closure (Figure 2), flattening of the nasolabial sulcus (Figure 3) and slight weakness to wrinkle the forehead (Figure 4).

No additional neurological deficit was detected. In the clinical evaluation performed to exclude central pathology, no central nervous system findings were observed.

Methylprednisolone (prednol) treatment was initiated at a dose of 1 mg/kg/day (80 mg/day). As supportive treatment, citicoline, benfotiamine (benexol) 2×1, and pantoprazole 1×1 were prescribed. A three-week course of treatment was planned. There was no indication for hospitalization.

During follow-up, significant clinical improvement was observed starting from the tenth day, and complete recovery was achieved at the end of the third week. Informed oral consent was obtained from the patient for the publication of this case report and the accompanying clinical images.

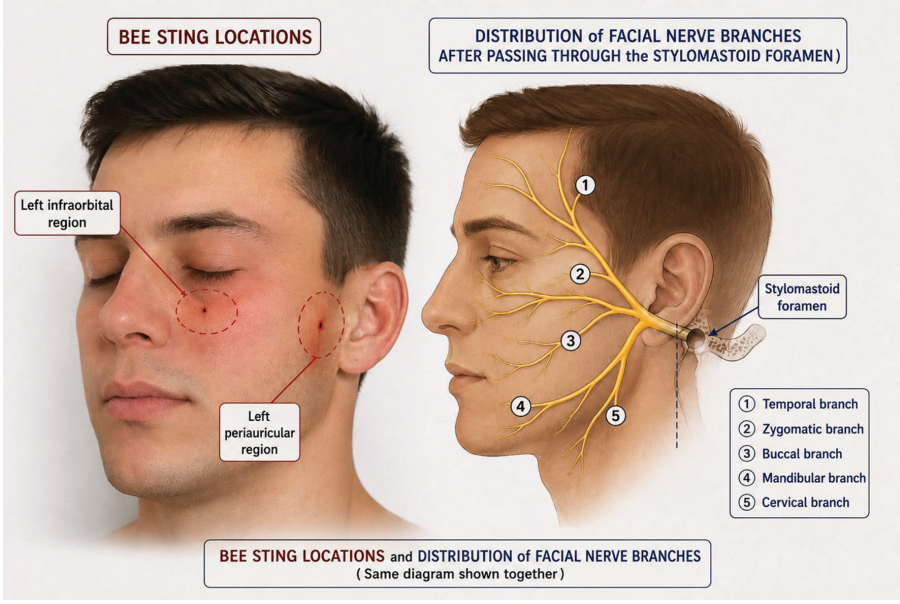

Figure 1: Diagram explaining bee sting locations and the distribution of facial nerve branches (the figure has been generated by ChatGPT on 16.05.2026).

Table 1: The House-Brackmann grading scale (6).

Grade	Description	Gross	At rest	Motion	Estimated function (%)
I	Normal	Normal	Normal	Normal	100
II	Mild dysfunction	Slight weakness noticeable on close inspection, may have very slight synkinesis	Normal symmetry & tone	Forehead: moderate to good function; eye: complete closure w/minimum effort; mouth: slight asymmetry	80
III	Moderate dysfunction	Obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm	Normal symmetry & tone	Forehead: slight to moderate movement; eye: complete closure w/effort; mouth: slightly weak w/ maximum effort	60
IV	Moderately severe dysfunction	Obvious weakness and/or disfiguring asymmetry	Normal symmetry & tone	Forehead: none; eye: incomplete closure; mouth: asymmetric w/ maximum effort	40
V	Severe dysfunction	Only barely perceptible motion	Asymmetry	Forehead: none; eye: incomplete closure; mouth: slight movement	20
VI	Total paralysis	No movement	Asymmetry	No movement	0

w/: with.

Figure 2: Mild weakness in eye closure.

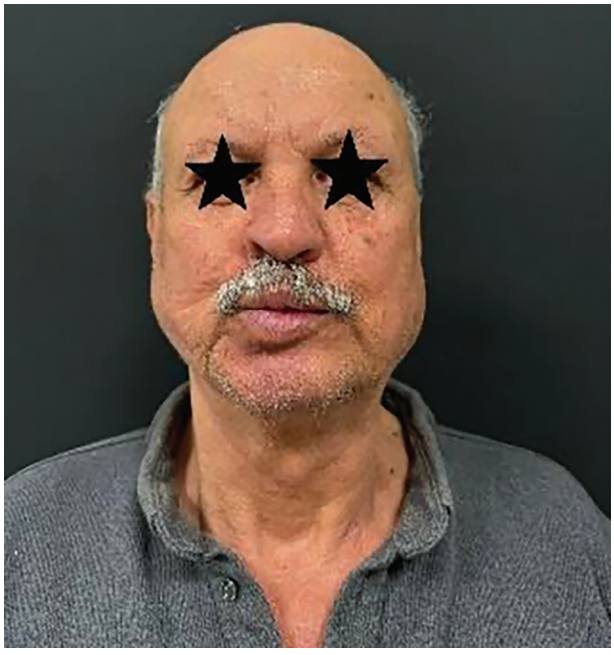

Figure 3: Flattening of the nasolabial sulcus.

DISCUSSION

Both strong allergic reactions and the direct toxic effects of bee venom contribute to the pathophysiology of bee sting injuries. The venom's phospholipase enzyme is the primary source of allergic responses (7). Honeybees, wasps, and hornets are the primary stinging species of bees, which are members of the Hymenoptera order (8). The venom gland is linked to the bee stinger. Toxins from the venom gland are injected into the skin by the stinger during a sting, resulting in either systemic or

Figure 4: Slight weakness to wrinkle the forehead.

local reactions. Honeybee stings usually cause regional edema without a systemic reaction. Venom from all hymenoptera species induce localized pain and swelling, and affected individuals usually notice that they are stung, unlike venomous spider bites. The local reaction includes edema, erythema, and discomfort, represents a non-immunoglobulin E (IgE)-mediated response that resolves spontaneously within 24 hours. The amount of venom in a bee sting is directly correlated with its clinical symptoms, which can be fatal in extreme circumstances. The chemical composition of venom varies among bee species. Wasp venom is alkaline, while honeybee venom is acidic. The stinger withdraws from the human body following a hornet sting, whereas it stays in the body following a honeybee sting. Histamine, various enzymes, formic acid, neurotoxins, and hemolytic toxins are among the complex components of bee venom. Toxic symptoms occur when bee venom enters the human body. The location of the sting, the quantity of venom entering the body, and the existence of an allergic reaction all affect the toxic reaction brought on by bee venom (9, 10).

Although PFP has a broad etiology spanning from infections to injuries and neoplasms, bee sting is a very rarely documented cause (3). Bee stings are a fairly uncommon cause of PFP, even though there is a broad range of etiologies, including diseases, trauma, and tumors (1, 2).

The relationship between the two disorders is supported by the fact that our patient's symptoms developed about 48 hours after the bee sting. The pathophysiology of this condition can be explained by several mechanisms.

Bee venom contains proteases and other bioactive compounds that may directly cause neurotoxic effects (2). Toxic neuropathy can develop because of the sting

site's anatomical proximity to the facial nerve's peripheral branches.

Local inflammatory edema and compression, however, are more likely causes. Neuropraxia may develop from compression of the facial nerve at the stylomastoid foramen exit or peripheral branches due to edema caused by inflammation triggered by IgE-mediated or non-IgE-mediated mediators. Furthermore, ischemia or vascular spasm at the vasa nervorum level may also be considered as a possible explanation.

Our patient's history of diabetes mellitus is also noteworthy. Diabetes mellitus may increase the risk of peripheral nerve damage because of microvascular changes and neuropathy susceptibility (11). This condition may have contributed to a more severe effect of the inflammatory or ischemic process triggered by the bee sting on the facial nerve.

Corticosteroid treatment was initiated in the preliminary period in accordance with Bell's palsy protocols. The three documented cases of bee sting-induced facial palsy differ significantly in terms of corticosteroid treatment. In order to address the systemic toxic and allergic response in the context of multiple organ dysfunction, Li et al. (9) gave intravenous dexamethasone at a fixed dose of 10 mg/day mixed with 5% dextrose in normal saline for 4-5 days, with a maximum duration of 7 days. In accordance with normal Bell's palsy protocols, Arun et al. (12) employed oral prednisolone at a fixed dose of 60 mg/day for the first week, followed by gradual tapering without stating the rate of dose reduction. In the present case, methylprednisolone was initiated at a weight-based dose of 1 mg/kg/day (80 mg/day) and tapered by 10 mg every two days over a total treatment period of three weeks. By using weight-based dosing and a systematic, predetermined tapering schedule, which may enable a more customized corticosteroid tapering strategy, our method was different from both previously reported cases. Methylprednisolone was selected due to its excellent pharmacokinetic profile and low mineralocorticoid activity, which may be especially beneficial for patients with metabolic comorbidities (13). Additionally, our patient's diabetes mellitus is significant since it is linked to chronic nerve ischemia and reduced nerve blood flow, which may have a detrimental impact on the healing and repair processes of the facial nerves (11, 14). All three patients recovered completely or almost completely despite variations in corticosteroid agents, dosage methods, and treatment lengths. This suggests that early corticosteroid therapy administration may be a significant predictor of outcome, independent of the particular regimen used. The rapid and complete recovery observed in the patient suggests that the pathology was at the level of neuropraxia rather than permanent axonal damage.

CONCLUSION

Although rare, bee stings should be considered as a potential trigger associated with the development of PFP. This possibility should be considered especially in patients with microvascular risk factors such as diabetes mellitus. Complete clinical recovery can be achieved with early corticosteroid treatment.

Ethics

Informed Consent: *An informed oral consent was obtained from the patient.*

Footnotes

Conflict of Interest: *The authors declared no conflict of interest*

Author Contributions: *Surgical and Medical Practices: A.K., Concept: İ.G.A., A.B., A.K., Design: İ.G.A., A.B., A.K., Data Collection or Processing: İ.G.A., A.B., A.K., Analysis and/or Interpretation: İ.G.A., A.B., A.K., Literature Search: İ.G.A., A.B., A.K., Writing: İ.G.A., A.B., A.K.*

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