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CASE REPORT

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A rare outcome following a bee sting: Total alopecia

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Abstract
Following bee stings, multiple case reports have described the onset of autoimmune-related conditions, including myasthenia gravis, nephrotic syndrome, Henoch-Schönlein purpura, myocardial infarction, cerebrovascular events, and localized alopecia, typically within a few days, indicating that bee venom may play a role in triggering autoimmunity. We report a rare case of total alopecia occurring within ten days after a bee sting. While the literature includes one prior case of total alopecia following bee sting-induced anaphylaxis, our case is distinct in that the patient developed total alopecia in the absence of any hypersensitivity reactions, suggesting a potential immunomodulatory or exacerbating effect of the bee sting on the immune system.

Introduction

Bee venom comprises a variety of components, including melittin, peptide 401, phospholipase A2, histamine, dopamine, hyaluronidase, and norepinephrine. While local reactions following bee stings are common, more severe manifestations such as urticaria, angioedema, and anaphylaxis can also occur. Furthermore, case series have indicated that bee stings may act as triggers for systemic and autoimmune diseases.

Alopecia areata is an organ-specific autoimmune disorder that develops as a result of a combination of genetic predisposition and environmental factors. It is frequently associated with other autoimmune diseases.[1,2] The literature reports cases of total alopecia following bee stings as well as localized alopecia resulting from insect stings.[3,4]

Moreover, cases of Guillain-Barré syndrome (GBS) have been reported following venom injection by bees, insects, or snakes, suggesting a potential autoimmune trigger. Cases of myasthenia gravis developing within several days

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following a bee sting, as well as a variant of GBS, specifically Miller Fisher syndrome, manifesting within 3 days after bee stings, have been well-documented in the literature.[5,6] Additionally, nephrotic syndrome and Henoch-Schönlein purpura (HSP) have also been reported after bee stings.[7-9] As an alternative therapeutic approach, bee venom injections have been employed in the management of various forms of arthritis; however, case reports have documented the development of systemic lupus erythematosus following such treatments, as well as GBS after the use of bee venom in acupuncture.[10,11] In the realm of oncological research, the cytolytic activity of bee venom has also been rigorously investigated across diverse cellular models in recent years.[12] These findings provide further support for the hypothesis that bee venom may play a role in triggering autoimmunity, including alopecia.

Case

A 65-year-old male patient with no known history of chronic illness or atopy presented to the immunology and allergy clinic with a 6-month history of generalized pruritus and total alopecia that developed within 10 days following a bee sting. The patient had a history of multiple bee stings but no prior episodes of anaphylaxis, urticaria, angioedema, or other hypersensitivity reactions.

Six months earlier, the patient had been stung by a yellow jackets (*Vespula* spp.) on the nape of the neck, resulting in localized swelling, erythema, and pruritus. The patient who presented to the emergency department was administered intravenous antihistamines and corticosteroids, resulting in a noticeable improvement of local symptoms on the same day. However, 10 days after the sting, the patient developed sudden-onset total alopecia. The patient's medical history was devoid of any evidence of acute stress, medication use, or prior episodes of hair loss.

Laboratory investigations revealed elevated specific immunoglobulin E (IgE) levels to both honeybees (*Apis mellifera*) and yellow jackets (*Vespula* spp.) (Table 1). Over the following months, the patient experienced severe generalized pruritus and an unintentional weight loss of 15 kg, without a corresponding loss of appetite. The patient attributed his symptoms to the bee sting and self-administered antihistamines; however, symptoms persisted.

Given the patient's advanced age, severe pruritus, and markedly elevated total IgE levels, further imaging studies were performed, which revealed no significant abnormalities. A skin biopsy was undertaken to assess the pruritus, but no pathological findings were observed. However, an endoscopic biopsy led to the diagnosis of signet ring cell carcinoma, a subtype of gastric cancer.

Discussion

Type I hypersensitivity reactions, including urticaria, angioedema, and anaphylaxis, represent the most common immune responses associated with bee stings. In our case, although elevated serum levels of specific IgE antibodies to both *A. mellifera* (honeybee) and *Vespula* species were detected, skin prick test results remained negative. Furthermore, the markedly elevated total IgE levels observed in our patient complicates the interpretation of the specific IgE findings, underscoring that the presence of specific IgE alone cannot be considered as definitive evidence of a classical IgE-mediated (Type I) hypersensitivity reaction.

Beyond immediate hypersensitivity, bee stings have been implicated in a wide spectrum of systemic and immune-mediated complications. Reported adverse outcomes include acute kidney injury, GBS, HSP, systemic vasculitis, myocarditis, myocardial infarction, centrilobular hepatic necrosis, acute encephalopathy, cerebral infarction, disseminated intravascular coagulation, and thrombocytopenia.[13,14] These complications suggest that components of bee venom may play a role in triggering aberrant immune responses beyond classical allergy pathways.

In case reports of autoimmune-related conditions following bee stings, the pathogenesis is hypothesized to involve

Table 1 Laboratory test findings.

Test	Results	Reference range
C-reactive protein	16.20 mg/L	0-5
Erythrocyte sedimentation rate	25 mm/h	0-20
Hemoglobin	13.9 g/dL	12.5-17.2
White blood cell count	6.72 x10^9/L	3.6-10.5
Eosinophils	0.73 x10^9/L	0.02-0.5
Platelets	305x 10^9/L	160-400
Thyroid-stimulating hormone	1.97 mU/L	0.55-4.78
Ferritin	13 µg/L	22-322
Stool parasite screening	Negative	-
Indirect hemagglutination	Negative	-
Apis mellifera skin prick test (100 µg/mL)	Negative	-
Vespula skin prick test (100 µg/mL)	Negative	-
Specific IgE - Honeybee venom	6.70 kUA/L	0-0.35
Specific IgE - Wasp venom	9.22 kUA/L	0-0.35
Total IgE	8382.9 IU/mL	0-378

IgE: Immunoglobulin E.

the components of bee venom, T lymphocytes, and cytokine secretion, which mediate immunological disturbances. In our patient, total alopecia developed, and the complex immunological mechanisms underlying the pathogenesis of alopecia remain poorly defined. Studies have initially identified the role of Th1/interferon-gamma (INF-γ) in alopecia, with findings showing a reduction in IFN-γ levels and upregulation of other immune pathways, including T helper 2 (Th2) (IL-4 and IL-13), Th9 (IL-9), phosphodiesterase-4 (PDE4), and Th17/IL-23.[15,16] Given the complexity of the immunopathogenesis of alopecia, clinical research is currently investigating T-cell antagonists, tumor necrosis factor (TNF)-alpha inhibitors, Janus kinase inhibitors, PDE4 inhibitors, and cytotoxic T-lymphocyte-associated protein-4 inhibitors, which target the suppression of the Th1 immune response, inhibition of IFN-γ–mediated signaling, and blockade of costimulatory pathways required for full T-cell activation and are thought to have therapeutic potential.[17]

A review of the existing literature reveals a case in which total alopecia developed following an episode of bee sting-induced anaphylaxis.[4] Acute stress is considered in the differential diagnosis of alopecia. However, no psychiatric evaluation was conducted in our patient for further investigation of cases, which are comparable to other immune-related case reports that developed within days following exposure to bee venom. In our patient, who was diagnosed with gastric cancer simultaneously, it is well established that malignancies can induce alopecia via paraneoplastic effects mediated through the immune system. Following bee stings, an increase in proinflammatory cytokines—particularly interleukins such as IL-1 and IL-8, as well as TNF—has been observed. Moreover, the induction of IL-6 production promotes the differentiation of B lymphocytes into antibody-secreting plasma cells, enhances T-cell proliferation, and facilitates the development of $CD8^+$ T cell–mediated cytotoxicity.[18] These immunological alterations collectively account for the elevation of systemic inflammatory markers. However, it should be emphasized that C-reactive protein, erythrocyte sedimentation rate, and eosinophil counts can rise in several conditions beyond acute systemic inflammation. In this case, the prolonged interval between the bee sting and the clinical findings, together with the persistence of elevated inflammatory markers, eosinophilia, and IgE levels long after the sting, suggests that these abnormalities are more plausibly attributable to malignancy-associated chronic inflammation (paraneoplastic mechanisms) rather than acute inflammation.

Paraneoplastic alopecia arises from complex immunological interactions between tumor-associated antigens and the host immune system. The collapse of hair follicle immune privilege, a well-established mechanism in alopecia areata, has also been described in paraneoplastic cases, leading to perifollicular infiltration of cytotoxic $CD8^+$ T lymphocytes and subsequent premature transition of anagen follicles into the catagen phase.[19] In this context, tumor-driven immune priming may generate autoreactive T cells or autoantibodies directed against shared antigens expressed by both neoplastic tissue and hair follicle structures.[20] Moreover, inflammatory cytokines such as IFN-γ, IL-2, and Th1 chemokines, including CXCL10, known to disrupt follicular immune privilege, may further perpetuate immune-mediated hair loss.[21] These findings suggest that paraneoplastic alopecia arises from the intersection of tumor-induced autoimmunity and localized follicular immune dysregulation. Although cytokine, chemokine, and flow cytometry-based immunological assessments were not performed in this case, it can be postulated that bee venom and paraneoplastic alopecia may activate overlapping immunological mechanisms. Furthermore, when examining case series of bee venom exposure resulting in total alopecia within days, as reported in the literature, our patient's rapid onset of total alopecia occurred within a similarly concise period of 10 days following the bee sting.

There are inherent limitations in elucidating the immunological mechanisms involved in this case. The evaluation of immunological pathways in this case did not include T-cell subset analyses, cytokine measurements, or autoantibody testing. Although elevated bee venom–specific IgE levels were detected, these findings alone cannot definitively establish an IgE-mediated or autoimmune-driven mechanism.

Conclusion

Our patient presents a rare case of total alopecia triggered by an autoimmune reaction following a bee sting. In this case, although bee venom appears to act as both the initiating and accelerating factor in activating the immune response leading to hair loss, the clinical presentation may ultimately reflect the interaction between bee venom–mediated immunomodulation, and paraneoplastic effects may activate overlapping immunopathogenic pathways. Furthermore, a meticulous evaluation of nonhypersensitivity symptoms that emerge as a consequence of immune activation is essential for the timely identification of other potentially concomitant pathologies.

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Author's Contribution

Writing-original draft preparation, C.C.,B.G.A.,S.S., writing-review and editing, supervision, C.C.,B.G.A.,S.S. All authors have read and agreed to the published version of the manuscript.

Conflict of Interest

The authors have no conflicts of interest to declare.

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