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Forensic Evaluation of Blindness Following Ocular Bee Sting: A Case Report and Literature Review

Yongping Wei[1], Xian Ju[2], Di Liang[2], Cenxuelun Zhang[1]*

[1]Judicial Expertise Center, The First Affiliated Hospital of Guilin Medical University, Guilin 541000, Guangxi, China
[2]School of Forensic Medicine, Shanxi Medical University, Jinzhong 030600, Shanxi, China

**Corresponding author: Cenxuelun Zhang, 771947458@qq.com*

Abstract: This report presents a forensic evaluation of a case involving blindness (visual acuity grade 5) following a bee/wasp sting to the left eye. Through systematic analysis of the patient's multiple hospital admissions, postoperative follow-up data, and a review of the pathological mechanisms of ocular injury caused by bee venom, this study comprehensively assesses the injury characteristics, treatment course, and visual outcomes. Bee venom induces severe complications such as corneal damage, uveitis, cataract, and secondary glaucoma through multiple mechanisms including direct cytotoxicity, immune-inflammatory responses, and enzymatic hydrolysis. Despite interventions including anterior chamber irrigation, phacoemulsification with intraocular lens implantation, and antiglaucoma surgery, the affected eye ultimately lost light perception. Forensic examination confirmed the absence of light perception in the left eye and abnormal visual pathway function, consistent with clinical observations. According to the relevant Chinese disability assessment standard (JR/T 0083-2013, Article 4.2.2), the injury was classified as grade 7 disability. This study provides an in-depth discussion of the mechanisms and key forensic identification points in bee-sting-induced blindness, offering a scientific reference for similar forensic clinical cases.

Keywords: Forensic clinical medicine; Bee sting; Ocular injury; Blindness; Disability assessment

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1. Introduction

Hymenoptera insects cause injuries to over 100 million people worldwide annually, with bee/wasp stings accounting for nearly 50% of cases [1–3]. Most stings occur on exposed skin of the head, face, and limbs. Mild cases may present with local redness, swelling, and pain, while severe cases can lead to anaphylaxis, multiple organ dysfunction syndrome, and even death [4–9]. Isolated ocular bee/wasp stings are relatively rare. Ocular morbidity depends on the interaction between the nature and virulence of the venom components and the

subsequent inflammatory response in the patient, with symptoms ranging from mild irritation to severe visual loss. The outcome of ocular stings varies based on the affected ocular structures and underlying injury mechanisms. Treatment depends on the mechanism of injury: retained stingers are usually surgically removed, while acute or chronic tissue reactions are managed medically. Although ocular injuries are common in forensic clinical practice, cases involving bee/wasp stings are seldom reported. This article reviews a case of blindness following conjunctival bee/wasp sting and discusses key forensic identification points related to ocular bee/wasp sting injuries.

2. Case history

On November 24, 2024, the examinee was stung in the left eye by a toxic bee/wasp while working on a mountain and subsequently sought hospital treatment. The case was referred to our center for assessment of disability degree related to personal insurance claims.

3. Medical record summary

3.1. Summary of admission and discharge within 24 hours at a county-level hospital

Admitted on November 24 and discharged the following day.

3.1.1. Chief complaint

Left eye pain and blurred vision for over 2 hours after being stung by a toxic bee/wasp while working on a mountain. Symptoms included left eye pain, photophobia, lacrimation, difficulty opening the eye, blurred vision, accompanied by dizziness, nausea, and chills without vomiting.

3.1.2. Specialist examination

Left eyelid swelling, difficulty in eye opening, bulbar conjunctival congestion and edema, large-area corneal epithelial detachment, central corneal edema and opacity, contusion in the corresponding iris area, turbid anterior chamber, and unobservable posterior segment.

3.1.3. Discharge status

Left eye pain reduced, visual acuity (Vos) 0.04, intraocular pressure (Tos) 42.7 mmHg. Left eyelid remained swollen with difficulty in opening, bulbar conjunctival congestion and edema, rough corneal surface with incomplete epithelial healing, reduced corneal edema but central corneal opacity still evident, contusion in the corresponding iris area, turbid anterior chamber, mild lens opacity, and unobservable posterior segment.

3.1.4. Discharge diagnosis

(1) Left eye toxic bee/wasp sting with venom poisoning reaction;
(2) Left eye blunt ocular trauma with corneal epithelial defect and iris contusion;
(3) Left eye secondary glaucoma.

3.2. Summary of medical records from an affiliated hospital

3.2.1. First admission (November 24–December 3, 2024)

The patient was admitted as an emergency due to left eye bee/wasp sting for 1 day with pain and decreased vision.

3.2.2. Initial examination

Left eye visual acuity: light perception, intraocular pressure: T + 2, mixed conjunctival congestion, significant corneal edema, large-area corneal epithelial defect, corneal endothelial folds, a pinpoint gray-white opacity at the 2 o'clock position of the cornea, fluorescein staining (FL) positive, central anterior chamber depth slit-like, hypopyon of about 2 mm in the inferior anterior chamber, pupil diameter approximately 5 mm, exudative membrane on the lens surface, lens opacity, and unobservable intraocular structures. Course during hospitalization: corneal OCT showed corneal penetration at the 2 o'clock position opacity (**Figure 1**). Anterior chamber irrigation of the left eye was performed under local anesthesia on November 30, 2024.

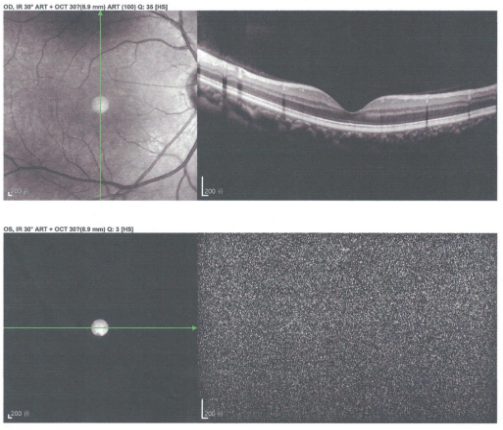

Figure 1. OCT examination of both eyes.

3.2.3. Discharge diagnosis

(1) Left eye bee/wasp sting;
(2) Left eye corneal injury;
(3) Left eye anterior uveitis;
(4) Left eye complicated cataract;
(5) Left eye blindness.

3.3. Second admission (December 18–December 20, 2024)

3.3.1. Chief complaint

Decreased left eye vision for over 20 days.

3.3.2. Specialist examination

Left eye no light perception, intraocular pressure 16 mmHg, patent lacrimal irrigation, mixed congestion, bandage contact lens in place, pigmented keratic precipitates (KP) visible, pinpoint gray-white corneal opacity at 2 o'clock position, central anterior chamber depth 3 corneal thickness (CT), no hypopyon, aqueous flare, round pupil approximately 6 mm in diameter, lens opacity, and unobservable intraocular structures.

3.3.3. Course

OCT indicated opaque refractive media with no signal captured. Phacoemulsification with intraocular lens implantation was performed under local anesthesia on December 19, 2024.

3.3.4. Discharge status

Left eye no light perception, intraocular pressure 9 mmHg, mild conjunctival congestion, corneal edema, endothelial folds, moderate anterior chamber depth, aqueous flare, round pupil approximately 5 mm in diameter, intraocular lens well-positioned, unobservable fundus.

3.3.5. Discharge diagnosis

Left eye traumatic cataract; left eye bee/wasp sting; left eye blindness.

3.4. Third admission (February 15–February 26, 2025)

Admitted due to left eye distension for over 2 months.

3.4.1. Specialist examination

Left eye no light perception, intraocular pressure 41 mmHg, hazy corneal edema, diffuse pigmented KP, central anterior chamber depth 3 CT, aqueous flare, round pupil approximately 6 mm in diameter, intraocular lens well-positioned, and unobservable intraocular structures.

3.4.2. Course

Anterior chamber irrigation, anterior chamber formation, and goniosynechialysis were performed under local anesthesia on February 24, 2025, followed by anti-infection and anti-inflammatory treatment.

3.4.3. Discharge status

No significant eye distension or pain reported.

3.4.4. Specialist examination

Left eye no light perception, intraocular pressure 16 mmHg, mild corneal edema, diffuse pigmented KP, central anterior chamber depth 3 CT, aqueous flare, round pupil approximately 6 mm in diameter, intraocular lens well-positioned, and unobservable intraocular structures.

3.4.5. Discharge diagnosis

Left eye secondary glaucoma; left eye anterior uveitis; left eye blindness; left eye status post intraocular lens implantation; left eye bee/wasp sting.

3.5. Outpatient follow-up (May 26 and June 6, 2025)

May 26, 2025: Diagnosis: 1. Bee/wasp sting; 2. (Secondary) glaucoma. June 6, 2025: Specialist findings: left eye no light perception, intraocular pressure unmeasurable, left corneal edema, central corneal patchy epithelial defect, fluorescein staining positive, anterior chamber disappeared, peripheral anterior synechiae, intraocular lens possibly adherent to corneal endothelium, fundus unobservable. Diagnosis: 1. Bee/wasp sting; 2. (Secondary) glaucoma.

4. Forensic analysis

The examinee was conscious, walked into the examination room independently, spoke clearly, responded appropriately, and cooperated during the examination. The left eye exhibited corneal edema and opacity, elevated intraocular pressure, and normal ocular motility. The right pupil diameter was 3 mm with prompt direct light reflex and absent consensual light reflex. The intraocular structures of the left eye could not be visualized (**Figure 2**). Muscle strength and tone of the limbs were normal, and no other significant abnormalities were noted on routine examination.

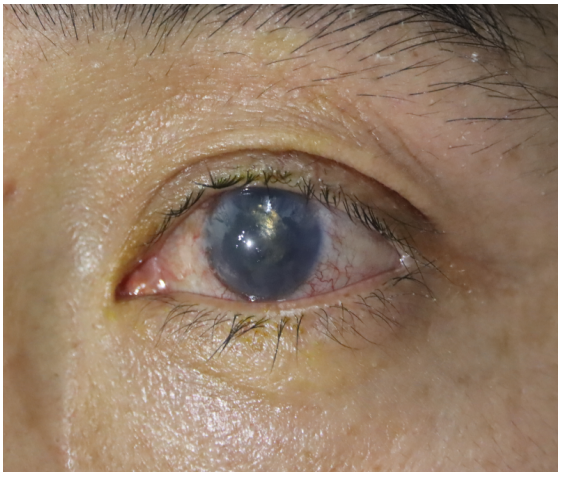

Figure 2. Intraocular structures of the left eye cannot be visualized.

Bee venom components can cause varying degrees of damage to different ocular tissues [10]. Biogenic amines (e.g., histamine, dopamine) induce vasodilation, leading to congestion and edema. Non-enzymatic polypeptides (e.g., melittin, apamin, mast cell degranulating peptide) disrupt cell membranes, causing direct hemolysis and protein denaturation, thereby damaging endothelial cells, inducing cataract, zonular laxity, and lens subluxation [11]. High-molecular-weight enzymes (e.g., phospholipase A, B, and hyaluronidase) lead to degeneration of anterior iris pigment cells, manifesting as iris heterochromia [12]. Other severe ocular complications reported in the literature include uveitis, optic neuritis, extraocular and intraocular muscle palsy, optic atrophy, and papillitis [13,14].

Upon ocular sting, direct contact of ocular surface tissues with bee venom initiates injury. Early corneal damage, especially with retained stinger, can trigger nonspecific inflammation. High-molecular-weight enzymes mediate type I hypersensitivity reactions; immune responses lead to the release of chemokines and anaphylatoxins, recruiting inflammatory cells and ultimately causing cell death. Clinically, this presents as corneal edema and sterile infiltration around the trabecular meshwork, often accompanied by decreased endothelial cell density in later stages [15]. Current reports on the treatment of ocular bee/wasp stings are limited and mostly consist of case reports [4–10,12–14]. Although various treatment strategies have been documented, severe complications such as corneal decompensation and refractory glaucoma still occur, similar to the present case [4,5].

In forensic evaluation, particular attention should be paid to the temporal sequence and cumulative nature of ocular complications following bee/wasp stings. In this case, despite early interventions including anterior chamber irrigation, cataract extraction, and antiglaucoma surgery, immune memory responses and impaired tissue repair induced by bee venom led sequentially to corneal decompensation, anterior synechiae, and angle closure, ultimately resulting in uncontrolled intraocular pressure and loss of optic nerve function. Such cases highlight that forensic assessment should not focus solely on the initial injury but must comprehensively evaluate the treatment

response, evolution of complications, and final visual functional status to ensure accuracy and fairness in disability grading.

The pathophysiological cascade following an ocular bee/wasp sting involves a complex interplay of toxic, inflammatory, and immune-mediated processes [13]. Bee venom contains a cocktail of bioactive substances, including enzymes, peptides, and amines, which collectively contribute to widespread ocular tissue damage [14]. Melittin, a major component, disrupts lipid membranes through detergent-like action, leading to rapid cell lysis and necrosis in corneal epithelium and endothelium. Phospholipase A2 and hyaluronidase degrade structural components of the extracellular matrix and cell membranes, facilitating venom spread and exacerbating tissue injury.

The initial clinical presentation corneal epithelial defects, edema, and anterior chamber inflammation reflects direct cytotoxic effects and acute phase responses. Subsequent development of uveitis and cataract underscores the involvement of persistent intraocular inflammation, likely driven by both toxic damage and immune sensitization to venom antigens. The emergence of secondary glaucoma is multifactorial: inflammatory debris and peripheral anterior synechiae obstruct aqueous outflow, while trabecular meshwork dysfunction may result from direct venom toxicity and chronic inflammation.

Despite aggressive surgical and medical management, the progression to phthisis or blindness, as observed here, is not uncommon in severe cases. This outcome emphasizes the limited regenerative capacity of corneal endothelial cells and the vulnerability of the trabecular meshwork and optic nerve to sustained inflammatory insult. Forensic evaluators must consider the natural history of such injuries, recognizing that even optimal treatment may not prevent functional loss due to the profound and multifocal nature of venom-induced damage.

In this examinee, the left eye was stung by a toxic bee/wasp, prompting immediate hospitalization. Initial findings included left eyelid swelling, difficulty in eye opening, bulbar conjunctival congestion and edema, large-area corneal epithelial detachment, central corneal edema and opacity, contusion in the corresponding iris area, turbid anterior chamber, and unobservable fundus. The clinical diagnosis of left eye toxic bee/wasp sting, ocular blunt trauma, corneal epithelial defect, iris contusion aligns with manifestations of bee venom-induced corneal injury. After treatments including left anterior chamber irrigation, left phacoemulsification with intraocular lens implantation, and left anterior chamber irrigation with anterior chamber formation and goniosynechialysis, the condition stabilized six months post-injury, meeting the timing requirement for forensic assessment. Forensic clinical examination revealed left corneal edema and opacity, elevated intraocular pressure, normal ocular motility, prompt direct light reflex and absent consensual light reflex in the right eye, and unobservable left intraocular structures. Two visual acuity examinations on May 26 and June 6, 2025, confirmed no light perception in the left eye (uncorrected). Fundus photography and OCT failed to capture valid signals, and F-VEP indicated abnormal visual pathway function in the left eye. These forensic clinical findings are consistent with the degree of visual dysfunction, visual functional test results, and the nature of the primary injury.

In summary, the injury resulted in left eye blindness (visual acuity grade 5) following a bee/wasp sting. According to the Personal Insurance Disability Assessment Standard and Code (JR/T 0083-2013) Article 4.2.2 on visual dysfunction, one eye blindness grade 5 corresponds to disability grade 7. Therefore, the injury is assessed as personal insurance disability grade 7.

5. Conclusion

Although ocular bee/wasp stings are clinically rare, they can cause severe and multifocal ocular tissue damage

through mechanisms involving direct toxicity and immune-inflammatory responses from various bioactive components in the venom. In this case, despite active surgical and pharmacological interventions following a left eye bee/wasp sting, the examinee developed serious complications including corneal injury, uveitis, cataract, and secondary glaucoma, ultimately resulting in loss of vision (blindness grade 5) in the left eye. Forensic evaluation should comprehensively analyze the injury mechanism, treatment course, and final visual outcome, integrating objective examination results to accurately apply disability assessment standards. This study underscores the need in similar cases to pay particular attention to the progression of multiple complications induced by bee/wasp stings and their long-term impact on visual function, providing a scientific basis for forensic practice.

Disclosure statement

The authors declare no conflict of interest.

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