

Case Report

Corneal Injury by Bee Sting with Retained Stinger - A Case Report

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Objective: To report a rare case of corneal injury by bee sting with its complication and management.

Material and Method: A 3-year-old boy, who was attacked by a swarm of bees, was referred for the right eye's corneal ulcer evaluation.

Results: Two stingers were found and completely removed with jeweler forceps. Corneal epithelial defect, corneal edema, secondary bacterial keratitis, heterochromia iridis, and internal ophthalmoplegia were identified. The corneal edema markedly improved 3 days after removing the retained bee stingers and treatment by topical antibiotics and steroids. The patient was evaluated after 1 week and 1 month and was found with permanent central corneal scar, particularly at the area corresponding to the retained stinger. The patient had subsequently localized traumatic anterior subcapsular cataract corresponding to where the stinger had penetrated the lens.

Conclusion: Corneal bee sting injury is an uncommon ocular trauma, but can result in severe sight threatening complication. Even though the response of corneal edema is well inclined to topical steroid, awareness in adjusting the clinical treatment for the particular case needs a scrutinized investigation of the infection.

Keywords: Bee sting, Stinger, Corneal ulcer, Corneal injury, Cornea

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Corneal injury by bee sting is uncommon. Ophthalmologic findings after such stings are various, also unpredictable from mild tissue edema and hyperemia to permanent visual loss^(1,3,4,6,7,9-15).

To the best knowledge of the authors, while there have been some case reports worldwide, it is unprecedented in Thailand. Therefore, the authors wanted to report an interesting case of a bee sting to the cornea, with retained stingers (ovipositor), associated with secondary bacterial keratitis. This report will discuss the pathogenesis and the management of this uncommon injury.

Case Report

A 3-year-old Cambodian boy was referred for

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the right eye's corneal ulcer after being attacked by a swarm of bees while sleeping 4 days earlier. Upon examination, his vital signs were stable without dyspnea or tachypnea. There was generalized erythematous papule on his face, trunk, and extremities. His vision was unable to fix and follow in either eye. Ophthalmologic findings showed mild diffused edema of upper and lower lids of both eyes, particularly the right eye, and no limitation to eye movement. Slit lamp biomicroscopy revealed that the left eye was normal and, in his right eye there was mild conjunctival chemosis with hyperemia 2+, generalized corneal haziness with edema 3+, central corneal epithelial defect 4 mm. in diameter and mild infiltration with a brown foreign body at the center position. (Fig. 1A) The mid-dilated pupil (6 mm in diameter) with no relatively afferent pupillary defect (RAPD) was observed. The fundus examination was normal in the left eye and no view in right eye due to corneal edema.

Organism was not found on gram stain following corneal scraping. However, the cultures showed moderate growth of *Acinetobacter Iwoffii* and *Pseudomonas* spp. Both of which succumb to ampicillin, tetracyclin, bactrim, gentamicin, and amoxicillin/clavulanic acid.

He underwent the removal of the stinger under general anesthesia. This was done by extending the corneal wound and pinching them off with jeweler forceps. There were two stingers, each approximately 2 mm long (Fig. 1B) placed in the deep stroma and penetrated through the anterior chamber. Due to the depth of stingers in the anterior chamber, aqueous leakage was foreseen. Glue application with bandage contact lens usage was performed under the treatment.

He was treated with fortified cefazolin (50 mg/ml) and gentamicin (14 mg/ml) through hourly eye drops. After 48 hours of infection prevention, the authors started 1% prednisolone acetate eye drop every two hours.

A week later, his vision improved to fix and follow in the right eye. His right eye examination revealed intraocular pressure was 20 mmHg; his cornea was clearer while mild edema remained around the central area, and mid-dilated pupil. No anterior chamber reaction cell or cataract was observed. Heterochromia iridis was identified, together with the normal fundus examination (Fig. 2).

One month after treatment, ophthalmologic findings showed central corneal scar, particularly in the same area as the retained stinger. There was no anterior chamber inflammation. The anterior subcapsular cataract was localized at two areas corresponding with where the stinger pinched into the lenses, and remained heterochromia iridis. However, the pupil size became smaller to 3 mm and reactive to light. The intraocular pressure was 14 mmHg in his right eye and no toxic optic neuropathy was identified.

Discussion

Only the honey bee leaves her stinger, a modified ovipositor⁽¹⁾ that consists of paired lancets with curved barbs on the distal end (Fig. 3) and the venom sac attached at the proximal end⁽²⁾. Consequently, any attempt at removing the stinger is difficult and often a segment remains within the cornea^(3,4). Therefore, a corneal bee sting is composed of mechanical, immunologic, and toxic injuries. When the bee stings, it introduces two bodily components into the eyes: the chitinous stinger and the venom⁽¹⁾.

Bee venom is a complex toxin composed of biologic amines such as histamine, dopamine, and

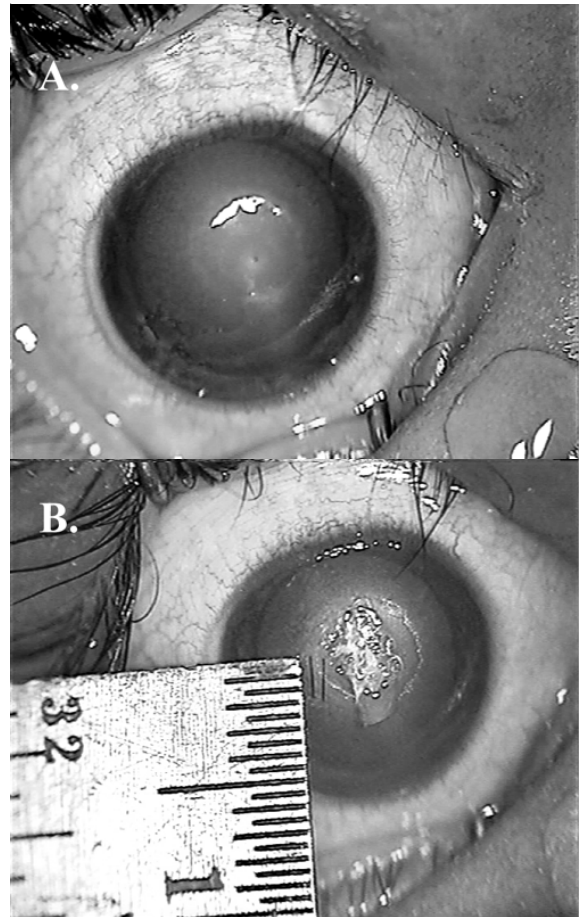


Fig. 1 A, external appearance of the right eye showing conjunctival hyperemia, corneal edema 3+, epithelial defect 4 mm in diameter with mild infiltration and brown foreign body at central area. B, showed 2 pieces of bee stingers, 2 mm long after removal

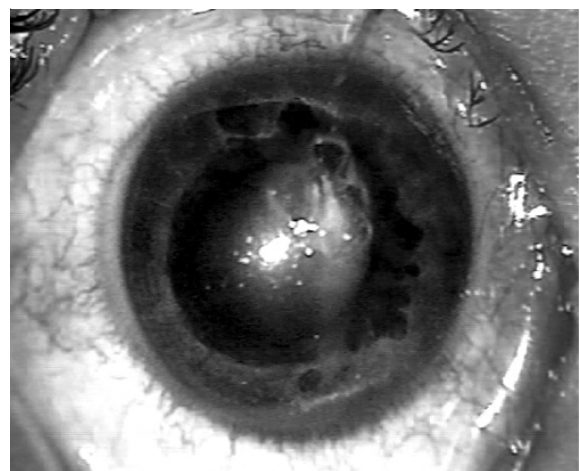


Fig. 2 One week after bee sting removal, showing clearer cornea, mid - dilated pupil and heterochromia-iridis



Fig. 3 High magnification of the bee stinger removed from the patient

nonenzymatic polypeptides toxins, such as melittin, apamin, mast-cell degranulating peptide, and minimine. It also has high molecular weight enzymes such as phospholipase A, phospholipase B, and hyaluronidase⁽⁵⁾. Sudden release of highly concentrated biogenic amines, such as histamine, causes severe eye pain, produces vasodilatation, and increase capillary permeability. This accounts for the conjunctival injection and chemosis⁽⁶⁾.

Smolin and Wong⁽⁷⁾ attributed that immunologic reaction to high molecular weight enzymes in the venom that may induce type 1 hypersensitivity reaction mediated by immunoglobulin E contributing to conjunctival injection, chemosis, and corneal edema as seen in the presented case. Mellitin is the main toxin in bee venom⁽⁵⁾. This is a basic compound with a strong surface activity. It causes membrane disruption and loss of erythrocyte cell structure resulting in direct hemolytic toxic effect and protein denature. According to previous reports, this lead to the formation of cataract and zonulysis with lens subluxation^(3,6,8). Apamin, another venom toxin, is the main neurotoxin that blocks neuromuscular junction, allowing for internal ophthalmoplegia and sector iridoplegia^(3,7,9). However, this effect could be reversible as shown in the presented case. Degeneration and lysis of chromatophores of anterior iris layers induced by hyaluronidase lead to iris depigmentation or heterochromia-iridis⁽⁹⁾. This has been reported earlier^(6,9), as was shown in the presented case. Singh⁽⁹⁾ suggested that the toxins in the venom be diluted and inactivated very early after stinging, so iris depigmentation develops more densely against the site of the sting. In the presented case, the total iris depigmentation is attributed to having two stingers in the central area.

Clinical manifestation of corneal bee sting, which still varies and is unpredictable, includes eyelids edema, conjunctival hyperemia, conjunctival chemosis, corneal epithelial defect, corneal stinger, corneal edema, corneal infiltration, fine keratic precipitate, striated keratitis, endotheliitis, endothelial cell loss, anterior uveitis, hyphema, glaucoma, lens subluxation, cataract, optic neuritis & papilledema, iris depigmentation, sector iridoplegia, and internal ophthalmoplegia^(1,3,4,6,7, 9-15). The variability of the clinical response is deemed to reflect the quantity of venom injection⁽⁴⁾, or different pathogenetic mechanisms⁽⁷⁾.

Visscher et al⁽²⁾ advised that bee stingers should be removed as quickly as possible, because delayed removal increases the dose of venom received. However, this is a controversial risk-benefit regarding the removal of a bee stinger. If a decision is made to get rid of it, it must be done neatly and completely. Contrarily, letting it remain may not affect the wound healing. Once the venom is inactivated, the chitinous stinger is believed to be inert and can be retained in situ without any untoward effect^(1,4). Sometimes, it can be completely reabsorbed⁽¹¹⁾. In the presented case, complete removal demonstrated a beneficial result. Even delayed removal of the bee sting for 5 days, corneal reaction could be markedly improved after 3 days treatment.

The adverse effect of the removal may exist, such as an infection, even if the toxins are reduced. It makes a stromal infiltration from the chemotaxis of polymorphonuclear leukocytes. This results in white corneal infiltration⁽⁷⁾.

Although coagulase-negative Staphaureus may be the most common bacteria associated with ocular foreign bodies⁽¹⁶⁾, pseudomonas infection has been reported following a corneal bee sting. This suggest that broad-spectrum agents should be used⁽¹⁷⁾. Gentamycin or fluoroquinolone groups can give an impressive result for antibiotic coverage⁽¹²⁾.

It is important for an ophthalmologist to recognize toxic optic neuritis as the onset of visual loss in one or both eyes ranges from 24 hours to 2 weeks after the sting⁽¹³⁾. However, early intervention with pulsed steroids may prevent permanent visual loss^(13,14). The exact mechanism of optic neuritis has not yet been known. In addition, under this investigation, it has been found that toxic optic neuritis may not necessarily be dose-related to toxin even if there were two stingers on the wound.

In summary, corneal bee stings with or without retained stinger, rarely occurs, but can contribute to severe visual impairments. To deal with the case, exami-

nation, diagnosis, and prompt management must be done.

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ผึ้งตอยกระจกตา

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วัตถุประสงค์: เพื่อรายงานลักษณะอาการ อาการแสดง, การดำเนินโรค, ภาวะแทรกซ้อน และการรักษาภาวะผึ้งตอยกระจกตา ซึ่งพบได้น้อย

วัสดุและวิธีการ: เด็กชายอายุ 3 ปี ถูกผึ้งตอยกระจกตา 4 วันก่อนส่งมารับการรักษาต่อ เนื่องจากภาวะติดเชื้อที่กระจกตาขวา ตรวจพบเหล็กใน 2 อันฝังอยู่บริเวณกึ่งกลางแผลทะลุเข้าช่องหน้าลูกตา และได้รับการผ่าตัดเพื่อนำเหล็กในออก

ผลการศึกษา: พบอาการแสดงดังต่อไปนี้ คือ กระจกตาบวม, แผลกระจกตาติดเชื้อแบคทีเรีย, ม่านตาขยายไม่ตอบสนองต่อแสงไฟ และมีสีอ่อนลง หลังรักษาด้วยการนำเหล็กในออก, ยาหยอดตาปฏิชีวนะ และ สเตียรอยด์ พบว่าภาวะกระจกตาบวมดีขึ้นอย่างรวดเร็วภายใน 3 วัน และตรวจติดตามต่อมา 1 เดือน พบแผลกระจกตาติดเชื้อหายดี และกลายเป็นแผลเป็นบริเวณที่ถูกตอย และมีต้อกระจกเกิดขึ้นที่เยื่อหุ้มเลนส์ตาด้านหน้าบริเวณที่ถูกเหล็กในทิ่ม

สรุป: ผึ้งตอยกระจกตาเป็นภาวะที่พบได้ไม่บ่อย แต่อาจมีภาวะแทรกซ้อนต่างๆ ตามมาได้ ตั้งแต่เล็กน้อยถึงรุนแรงมากจนทำให้ตาบอดได้ ดังนั้นการตรวจตาอย่างละเอียดรอบคอบ จะทำให้ได้รับการรักษาที่ถูกต้องและทันที่ และถึงแม้ว่าภาวะกระจกตาบวมจากการถูกผึ้งตอยนั้น จะตอบสนองดีต่อยาหยอดชนิดสเตียรอยด์ แต่ก่อนให้ยาควรพิจารณาควบคุมการติดเชื้อให้ได้ก่อน เพื่อป้องกันไม่ให้เกิดการติดเชื้อลุกลามมากขึ้น