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CASE REPORT

Predicting visual function after an ocular bee sting

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Abstract

Purpose To report a case of toxic optic neuropathy caused by an ocular bee sting.

Methods Case report and literature review.

Results A 44-year-old female presented with no light perception vision 2 days after a corneal bee sting in her right eye. She was found to have diffuse cornea edema with overlying epithelial defect and a pinpoint penetrating laceration at 6 o'clock. There was an intense green color to the cornea. The pupil was fixed and dilated with an afferent pupillary defect. A small hyphema was seen, and a dense white cataract had formed. A diagnosis of toxic endophthalmitis with associated toxic optic neuropathy was made. The patient underwent pars plana vitrectomy and lensectomy with anterior chamber washout. She was also placed on systemic broad-spectrum antibiotics. She had noted clinical improvement over the course of her hospitalization and was discharged with light perception vision. A corneal opacity precluded viewing of the fundus. We utilized ganzfeld electroretinography and flash visual evoked potentials (2 and 10 Hz) to assess the visual function. Both tests were normal and predicted improvement following restorative surgery. She underwent a secondary lens implantation with penetrating keratoplasty 7 months later. This was followed by an epiretinal membrane peel 1 year after the bee sting. Her best corrected visual acuity improved to 20/80.

Conclusion Toxic endophthalmitis and toxic optic neuropathy can be complications of ocular bee sting. We discuss the management of this rare occurrence and the role of electroretinographic testing and visual evoked potentials in predicting visual outcome.

Keywords Ocular bee sting · Toxic optic neuropathy · Electroretinogram · Visual evoked potential

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Introduction

Ocular bee stings are a rare event, previously described in the literature to result in a myriad of clinical symptoms and with varied visual outcomes. Honeybees (Hymenoptera order) use their barbed stinger to inject a toxin. Bees leave their stinger

complex upon penetration, dying in the process [1]. Injuries reported in the literature range from cornea opacities to lens opacities to fundus changes and optic neuritis [2].

The bee stinger contains multiple enzymes that can affect ocular structures. The stinger is composed of chitin and remains inert in the cornea after clearance of venom [3–6]. Melittin comprises the majority of the toxin and has been shown to cause cataract formation and lens subluxation [4, 7]. Apamin is a neurotoxin that affects potassium channels and may result in demyelination leading to optic neuritis [4, 8, 9]. Other components play vital roles in cellular membrane breakdown and spread of toxin.

Here we present a unique patient with corneal bee sting who developed toxic optic neuropathy and endophthalmitis. We describe our approach to guiding management based on electrodiagnostic evidence to ascertain visual potential.

Case description

A 44-year-old female was referred urgently to our emergency department with concern for corneal ulcer. She recalled a bee sting in the ocular region with significant periocular swelling and acute vision loss in her right eye. This occurred 2 days prior to presentation. She denied any previous ocular history. Medical history was significant for hepatitis C.

Initial examination showed no light perception vision in the right eye and 20/20 vision in the left eye. She had mild periorbital edema on adnexal examination. Her right pupil was mid-dilated and fixed, and a relative afferent pupillary defect was present on reverse examination. The biomicroscopical examination demonstrated ciliary flush with subconjunctival hemorrhage at 6 o'clock. The cornea showed a 4.4-mm epithelial defect centrally with edema (Fig. 1). The endothelium had keratic precipitates inferiorly. Corneal opacification with neovascularization was visualized (Fig. 2). There was a full thickness penetrating pinpoint lesion at 6 o'clock which was Seidel negative. The anterior chamber was formed with hyphema and 3 + flare. The iris demonstrated posterior synechiae, and there was a dense white cataract precluding view of the fundus. A B-scan ultrasound showed choroidal effusions with vitreous hemorrhage (Fig. 3). The examination of the left eye was normal.

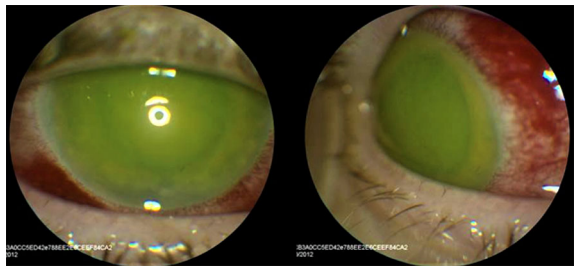


Fig. 1 Slit lamp photographs showing cornea with green discoloration, diffuse haze. There is conjunctival injection globally

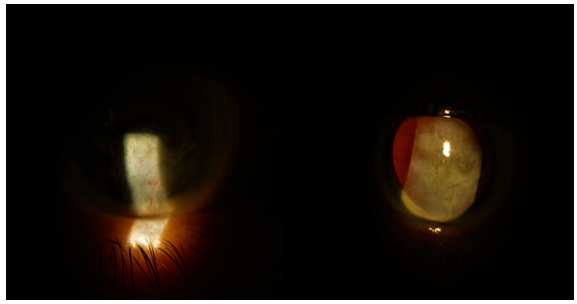


Fig. 2 Slit lamp photographs showing corneal haze with neovascularization

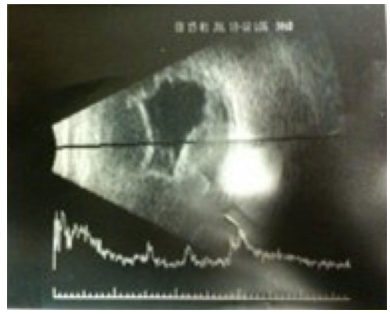


Fig. 3 B-scan ocular ultrasound showing choroidal effusion with vitreous hemorrhage

The patient was admitted to the hospital and started on 250 mg of intravenous (IV) methylprednisolone for presumed venom ophthalmia with toxic optic neuropathy. She was also started on topical erythromycin, moxifloxacin, and cyclopentolate. The patient underwent a pars plana vitrectomy and lensectomy, with anterior chamber washout. The vitreous was described as a hazy green color presumably secondary to the toxin. She received intravitreal injection of vancomycin (1 g), ceftazidime (2 g), and dexamethasone (0.4 mg). Vitreous cultures initially grew budding

yeast, and patient was given intravitreal amphotericin. Systemic therapy with IV vancomycin (1 g every 8 h), ceftazidime (2 g every 8 h), and amphotericin (340 mg per day, approximately 4.5 mg/kg per day) was also initiated. On hospital day 3, IV amphotericin was discontinued, and oral fluconazole 800 mg per day was started. On hospital day 10, the systemic antibiotic therapy was discontinued, because the follow-up of culture results showed that they were negative. Patient was discharged on oral steroid taper and followed up with her local ophthalmologist.

Six months later, she returned to our clinic to see whether there was further treatment to be offered. The right eye's vision was light perception at this time. There was a dense corneal opacity, which prevented clear imaging of the retina. Consequently, we selected standard flash visual evoked potentials (VEPs) [10] and standard electroretinograms (ERGs) [11] to assess visual function. Flash VEPs were recorded using the UTAS ganzfeld system (LKC Technologies) employing both 2- and 10-Hz stimuli (Fig. 4a, b) [10, 12, 13]. The amplitude recorded from the right eye was 16.9 μV with a peak time of 118 ms for P2. The amplitude of the left eye was 26.6 μV, and the peak time of P2 was 119.5 ms for the left eye with the 2-Hz stimuli. The 10-Hz VEP in the right eye showed an amplitude of 14.1 μV. In the left eye, we observed an amplitude of 18.2 μV. Both amplitudes were greater than the lower limit of the laboratory reference value, 5.26 μV. The right eye amplitude was lower than that of the left eye; however, the interocular amplitude ratio was 0.77, which was within normal limits.

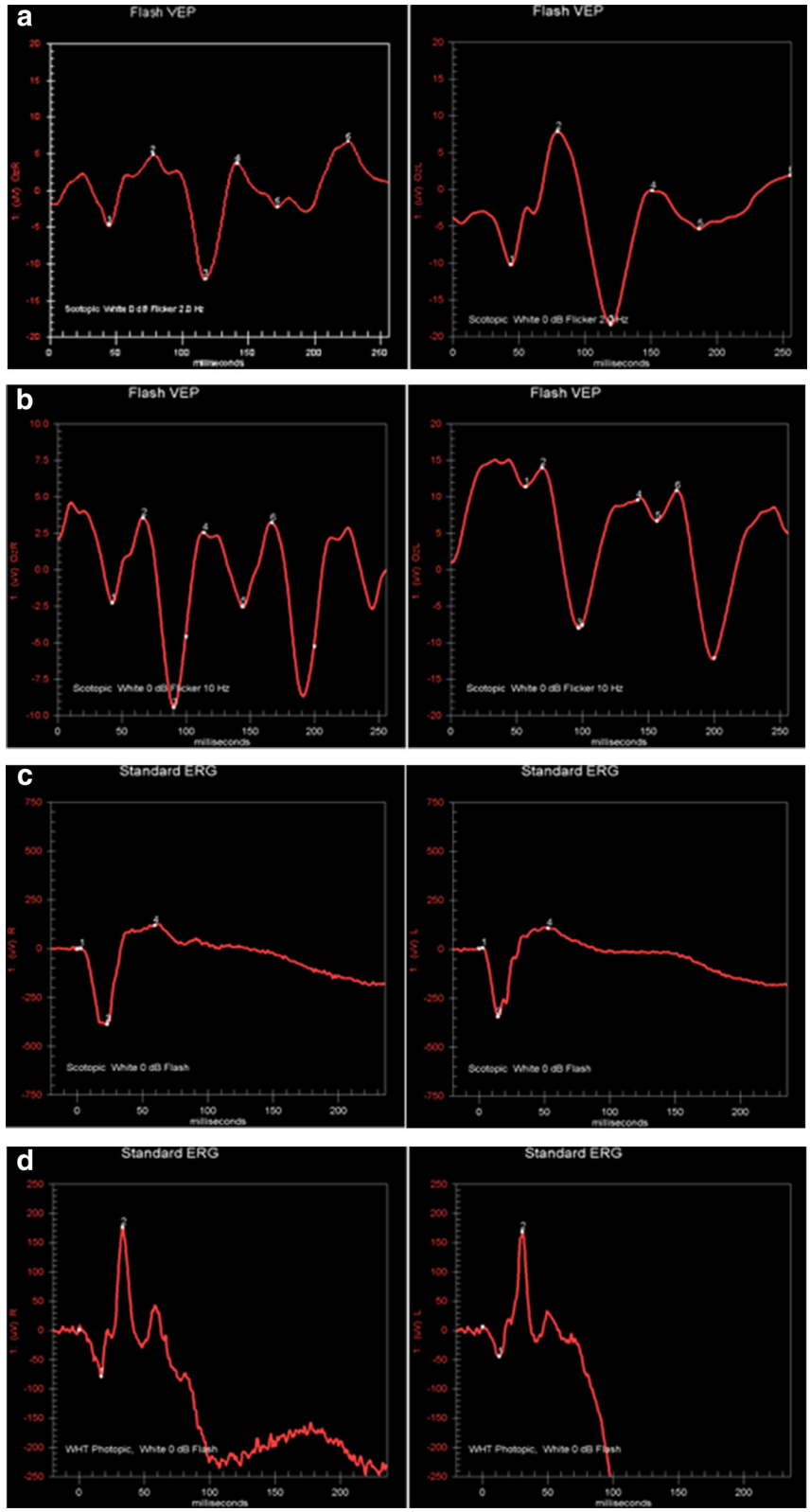
Standard ganzfeld ERGs were also performed. Figure 4c, d presents dark-adapted and light-adapted ERGs, respectively. Dark-adapted ERG b-waves elicited by 0.0 dB (0.01 cd s m^{-2}) flashes following 20 min of dark adaptation were 483.9 μV for the right eye and 452 μV for the left eye. Light-adapted ERGs b-waves elicited by 0.0 dB (0.01 cd s m^{-2}) flashes following 10 min of light adaptation were 254.9 μV for the right eye and 213.4 μV for the left eye. The amplitude parameters were all within normal limits.

Based on the results of these functional tests, it was determined that the patient possessed visual potential that was significantly greater than light perception. She underwent corneal transplant and secondary lens placement 7 months after her injury. She further had a removal of an epiretinal membrane that was visually significant 1 year after the bee sting. Her best corrected visual acuity was 20/80.

Discussion

Optic neuropathy is a previously described consequence of ocular bee sting [4, 14, 15]. These studies have also examined the role of functional testing in diagnoses and progression. Choi et al. [16] reported a case where the patient's initial visual acuity was light perception. When the visual acuity improved to 20/60, pattern-reversal VEP testing was performed and showed delayed P100 latency. However, 2 days later when the visual acuity returned to 20/20, latency time had reverted to standard levels. The seminal report of this was by Song and Wray in 1991 [17]. They showed an initial pattern-reversal VEP with prolonged P100 latency. Two years later, however, the P100 of the pattern VEP was absent. They postulated that early prolonged latency of P100 showed acute demyelination, and loss of pattern VEP 2 years later was due to irreversible damage. Since pattern ERG latency and amplitude were normal, they assumed that the retina must have been intact. In a case with a non-recordable standard and bright-flash ERGs in the affected eye and flash VEPs with decreased P2 amplitudes without significant delays in the peak times, it was hypothesized that obstructive vasculopathy might have been the culprit for optic neuropathy as opposed to demyelination [14].

Here we present our method for guiding surgical intervention based on electrodiagnostic testing. Our patient presented with no light perception vision. The patient was initially treated for optic neuropathy and presumed endophthalmitis. Whereas the symptoms of pain and inflammation improved, the vision remained light perception. Her posterior segment examination was inadequate, and the patient was determined to proceed with reconstructive surgery. Because of the dense opacity and poor visual acuity, pattern VEP or pattern ERG was not considered appropriate tests. Therefore, we opted to use flash VEPs as well as standard ganzfeld ERGs [10–13]. Prior research in our clinic has shown that 10-Hz flash VEPs can predict the likelihood of improvement in visual function following removal of dense media opacities [12, 13]. The ERG also showed mild delay in comparison with the


a
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◀**Fig. 4** Electrophysiological responses. Right eye presented on the left and left eye presented on the right, respectively. **a** Flash VEPs. Flashes were presented at 2 Hz in a darkened cupola in a dimly lit room. Flashes were 0.0 dB (0.01 cd s m^{-2}). The right eye's amplitude was 16.9 μV with a peak time of 118 ms compared to 26.6 μV and 119.5 ms for the left eye. **b** VEPs elicited by 10-Hz flicker. Flicker was presented through closed eyelids in a darkened cupola in a dimly lit room. Flashes were 0.0 dB (0.01 cd s m^{-2}). The 10-Hz VEP in the right eye showed an amplitude of 14.1 μV. In the left eye, the amplitude was 18.2 μV. **c** Standard ERG, scotopic. Dark-adapted ERG b-waves elicited by 0.0 dB (0.01 cd s m^{-2}) flashes were 483.9 μV for the right eye and 452 μV for the left eye. **d** Standard ERG, photopic, white 0 dB flash. Light-adapted ERGs elicited by 0.0 dB (0.01 cd s m^{-2}) flashes 254.9 μV for the right eye and 213.4 μV for the left eye

healthy eye, but both amplitudes and peak times were within normal limits for the patient's age, suggestive of good retinal function. Both the standard 2 and the 10-Hz flash VEPs were reduced relative to the normal eye, but they were clearly recordable, suggesting the possibility of improved visual function with surgery. Based on these results, it was recommended that the patient undergoes penetrating keratoplasty and secondary lens placement. Once a view to the fundus was available, an epiretinal membrane was seen. Vitrectomy with membrane peel was performed, and she achieved a best corrected visual acuity of 20/80.

This case highlights the importance of functional testing in guiding management in patients with ocular bee sting. Early studies have demonstrated how nerve and retinal damage can be ascertained from functional vision testing. We believe that in turn, functional testing, such as VEP and ERG, has a key role in predicting patient's visual outcome, especially in cases where opacities of the medial prevent a clear image of the retina.

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Compliance with ethical standards

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this paper.

Human and animal rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the paper.

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