Optic Neuritis After Bee Sting

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The purpose of this report is to document an unusual case of fully recovered vision after optic neuritis caused by bee sting. A 46-year-old man presented with sudden visual loss after being stung by a bee on the left conjunctiva. He developed optic disc swelling and there was a delay in the P100 wave of the pattern visual evoked potential (VEP). The patient received acute treatment, with intravenous methylprednisolone followed by oral prednisolone. Two days later, visual acuity in the left eye was recovered to 20/20 and P100 latency in pattern VEP was also normalized. Furthermore, visual field and color vision tests revealed no remaining abnormalities. This case suggests that early corticosteroid treatment is effective in optic neuritis caused by bee sting.

Key words: optic neuritis, bee sting, steroid, visual prognosis

INTRODUCTION

Bee stings to the eye or periocular area are a well-known cause of acute inflammation in the anterior segment of the eye. Various manifestations such as corneal edema, hyphema, internal ophthalmoptlegia, partially dislocated lens, iris atrophy, cataract, and glaucoma have been reported.1,2 However, less common is the development of retrobulbar optic neuritis, papillitis, papilledema, and optic atrophy.3-7 In most patients with decreased vision, corticosteroid has been tried as the treatment of choice for visual loss secondary to the optic neuritis. However, the degree of visual acuity recovery has varied in treated optic neuritis cases and all of these patients exhibited nerve fiber bundle visual field defects.3-5 Contrary to the procedure in previous reports in which treatment was typically delayed by up to two weeks after the sting, we acutely treated a bee sting patient with optic neuritis and the patient’s condition improved with corticosteroid therapy at a faster rate.

This paper reports a case of normal visual function recovery from unilateral optic neuritis due to a bee sting in the eye, based on several visual function tests including pattern visual evoked potential (VEP). Because of the rarity of reports about visual function recovery in optic neuritis caused by bee stings, this case report may be of interest and might stimulate the implementation of immediate corticosteroid treatment in similar cases.

CASE REPORT

A 46-year-old man was stung by an unknown type of bee on the left conjunctiva while working outside at 11 A.M. on September 8th. Immediately,
the patient experienced severe ocular pain without loss of vision. He was treated with topical corticosteroid by a local ophthalmologist. Later the same day, visual acuity in the left eye decreased. About ten hours after the sting he visited the emergency department of Chungbuk National University Hospital with loss of vision in his left eye. The results of various laboratory tests, including erythrocyte and leukocyte count, sedimentation rate, chest roentgenogram, urinalysis, a serologic test for syphilis and human immunodeficiency virus were either negative or normal. The patient had no history of medical illness and did not take any medication. Dietary and toxin exposure history was denied.

The patient's visual acuity was 20/20 OD and light perception OS. Lid swelling, conjunctival injection, corneal edema, and flare in the anterior chamber were found OS. There was an afferent pupillary defect in the left pupil. Applanation tonometry was 8 mmHg in each eye. Optic disc congestion was found in the left eye. No retinal detachment or hemorrhage was observed ophthalmoscopically. Acute treatment started at 11 P.M. of the same day with intravenous methylprednisolone (Medisolu®, 500 mg and then 250 mg every 6 hours for 3 days) followed by oral prednisolone (Solondo®, 1 mg per kilogram of body weight per day for 1 week) and topically instilled cycloplegics (Homatropine®, two times per day) antibiotics (Ofloxacin®, four times per day), and corticosteroid (Fluorometholone®, six times per day). The patient's previous experiences with bee stings to the body were reported as consisting of only localized swelling without systemic reaction.

On the following day, the optic disc congestion decreased and vision improved to 20/60 in the left eye. Lid swelling, conjunctival injection and the corneal edema subsided, while flare in the anterior chamber disappeared. A VEP (EP/EMC system, Cadwell Lab., USA) was elicited using 100 Hz pattern reversal of a 50% contrast black and white checkerboard (check size: 15 minutes of arc, phase reversing: at 3 reversals/second), and the P100 latency was examined. Latency time was delayed to 127.81 msec in the left eye while latency time was 117.18 msec in the right eye (Fig. 1).

Two days after the bee sting, his visual acuity was normalized to 20/20. The anterior segment as well as the optic disc itself appeared normal. Latency time of pattern VEP was normalized to 99.06 msec in the left eye and latency time was 98.75 msec in the right eye (Fig. 2). Visual field was checked using a Humphrey field analyzer. There was no evidence of scotoma and his visual field was within the normal range of the following standard visual field indices: mean deviation, pattern standard deviation, and corrected pattern standard deviation. Color vision was normal in a Farnsworth-Munsell 100-hue color vision test.

One month later, his vision remained normal with no deterioration and the optic disc was normal in appearance. Three months after the onset of optic
neuritis the patient discontinued follow up examinations. Despite recommendations to visit hospital again, he refused because there was no visual impairment.

DISCUSSION

Bee stings to the eye cause various ocular lesions, such as chemosis, corneal edema, iridocyclitis, cataract, glaucoma, and optic neuritis. These severe reactions seem to be related to the venom injected rather than the sting itself. The bee sting can produce both toxic and allergic reactions via several biologic amines. Therefore, patients who have had anterior segment involvement have been treated with topical corticosteroids and cycloplegics. Smolin and Wong reported that there were no corneal abnormalities after 48 hours in a patient who was immediately treated for corneal edema. Tuft et al. reported that the best-corrected visual acuity at 6 months after a bee sting was 20/20. In that particular case, a patient having keratitis and anterior uveitis was treated in the evening of the day the injury was sustained. In the case reported here, abnormalities of the anterior segment were treated with topical corticosteroid. The inflammatory reaction and the consequent effectiveness of topical corticosteroid in the anterior segment are consistent with the etiology whereby this reaction is due to an allergic reaction to the insect venom.

However, the visual prognosis is still controversial, even though the corticosteroid treatment of patients with optic neuritis caused by bee sting has been suggested in previous reports. The reported effect of corticosteroid on optic neuritis caused by bee sting was not consistent with lesions of the anterior segment. In a case of treatment with an intravenous corticosteroid, Berrios and Serrano reported a patient with bilateral optic neuritis after bee sting whose final visual acuity was 20/25 in the right eye and 20/30 in the left eye. Unfortunately, treatment was started only after two weeks had elapsed. In the case reported by Song and Wray, where treatment with corticosteroid started on the third day after the sting, there was no vision recovery. Unrecordable electoretinographic responses were reported without any visual recovery in a patient who had been stung by a wasp and was treated with an intravenous corticosteroid.

Contrary to the delayed treatment in these previously reported cases, treatment for the patient with optic neuritis due to bee sting in the case reported here commenced immediately. The patient's visual acuity improved at a faster than expected rate. Before starting the treatment, it was intended to treat the patient with intravenous methylprednisolone for 3 days followed by an oral prednisolone for 11 days. However, he was treated for only 7 days because his visual acuity improved to normal by the third day.

The patient had regained normal color vision, visual field and recovery of latency time of pattern VEP on the third day of treatment. These results suggest complete recovery of optic nerve function. There were no reports about the effects on color vision in optic neuritis cases caused by bee sting. Contrary to the case reported here, previously reported patients showed nerve fiber bundle visual field defects. A few studies reported on VEP in optic neuritis caused by bee stings. Consistent with the results reported here, Song and Wray reported an initial pattern VEP recording showing a prolonged latency of P100. However, two years later, the pattern VEP showed absent potential with no recovery of vision. With this result they suggested that demyelination of the optic nerve played a major role in acute stages and that secondary degeneration of denuded axons and retinal ganglions occurred.

Recently, Maltzman et al. reported a case of bee sting optic neuritis. In their case, presenting visual acuity was 20/80 in the right eye and 20/70 in the left eye. The visual acuity had returned to 20/15 bilaterally after a 3-day course of intravenous methylprednisolone, consistent with the clinical course of the present case.

There are some reports of possible neurotoxic effects on the retina and the optic nerve. The toxic components can cause thrombocytes to release serotonin and the mast cells to release their pharmacologic mediators, including bradykinins. Bradykinin is a well-known substance that plays an important role in the production of oxygen free radicals. Another mechanism of increased production of oxygen free radicals may be related with partial ischemia and reperfusion of transiently ischemic regions. In the reported cases of papilledema and
cerebral edema, the ischemia induced by tissue edema generate oxygen free radicals is an important feature of secondary injury following these reactions. Therefore, these releases of oxygen free radicals are thought to result in peroxidation of the lipids in the cell membrane, thus damaging the neural membrane.

Demopoulos et al. reported on the effect of methylprednisolone in preventing irreversible damage to injured tissue from oxygen free radicals. These mechanisms may explain why corticosteroid may be an effective treatment in optic neuritis caused by bee stings. From a theoretical point of view it would be important to institute therapy with cortisone immediately after the onset of optic neuritis. Goldstein et al. recommended immediate therapy with cortisone after the onset of neuritis caused by insects. The case reported here of a 12 hour recovery following bee sting suggests that the immediate use of intravenous corticosteroid is imperative when the optic nerve is involved. As shown in the analysis of the previous reports, the time of treatment was one of the important factors in determining visual prognosis. As soon as possible after the patient is stung, therapeutic action must be taken to offset venom activity. From the present result, it may be that the visual outcome, in patients without light perception following optic neuritis due to bee stings, can be significantly improved by early treatment. It seems that the immediate use of corticosteroid treatment controls the inflammation and prevents later complications.

In reported literature, this is an unusual case of overall visual function recovery from optic neuritis after a bee sting. Moreover, it is noteworthy that the present case showed a complete recovery of visual function with acute corticosteroid treatment. Therefore, the initial goal in treating optic neuritis after bee sting must be early recognition of the condition and early treatment by intravenous corticosteroid.

REFERENCES