CASE REPORT

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Oculomotor Nerve Palsy Induced by Herpes Zoster Ophthalmicus

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ABSTRACT

Herpes Zoster Ophthalmicus (HZO) is an infection involving the ophthalmic branch of the trigeminal nerve due to reactivation of latent varicella –zoster virus in the sensory ganglion of the trigeminal nerve. After suppression of immunity for any reason, the virus reactivates and causes vesicular lesions on the dermatomes innervated by the ganglion. Although HZO can be seen at any age, it is a disease often seen in the elderly or immunocompromised individuals. All ocular structures may be involved, with various ocular findings. Cranial nerve involvement is a rare complication in HZO. The most commonly involved cranial nerve is the oculomotor nerve. In this case report, a 65-year-old patient who developed oculomotor paralysis, anterior uveitis and neurotrophic keratitis after herpes zoster ophthalmicus is presented.

Keywords: Herpes Zoster Ophthalmicus, Oculomotor Paralysis, Shingles

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Introduction

Varicella Zoster Virus (VZV) is one of the human alpha herpes viruses and shows structural similarity to the herpes simplex virus. It causes chickenpox and shingles in humans. After chickenpox infection, the virus settles in the dorsal root ganglion cells and remains there for years. Herpes Zoster occurs after reactivation of the primary infection. After suppression of immunity for any reason, the virus reactivates and causes vesicular lesions on the dermatomes innervated by the ganglion. Herpes Zoster Ophthalmicus (HZO) infection occurs with activation of the latent VZV and involvement of the ophthalmic branch of the trigeminal nerve. Although ocular findings vary, they can be seen in 20-70% of HZO cases. Conjunctivitis, blepharoconjunctivitis, epithelial keratitis, stromal, nummular and dissiform keratitis, anterior uveitis, scleritis, chorioretinitis, optic neuropathy and cranial nerve involvement are HZO clinical manifestations that may occur (1,2).

CASE

A 65-year-old male patient was admitted to the ophthalmology clinic with left eyelid edema, redness and ptosis. In the ophthalmological examination, there was mild lid edema, ptosis, limitation of eye movements in upward, inward and downward gaze, and diplopia in the left eye (Picture 1). Right eye vision 0.8, left eye vision 0.4, intraocular pressure right:16mm Hg Left:17mm Hg. Biomicroscopic examination of the right eye was normal, except for grade 1 nuclear sclerosis. The left eye conjunctiva was hyperemic, grade 2 nuclear cataract was present, the cornea was transparent and the anterior chamber was calm. Fundus examination did not reveal any significant pathology. In his anamnesis, it was learned that he had received antiviral treatment due to lesions on the same side of the forehead and scalp in the dermatology outpatient clinic about 15 days ago. The patient was given unpreserved lubricant drops and topical antiviral ointment (ganciclovir). The patient was referred to the neurology outpatient clinic for consultation due to cranial nerve involvement and a cranial MRI was requested. In the control examination 3 weeks later, stromal infiltration and endothelial edema and precipitates developed. Oral antiviral (Valacyclovir 1gr 3*1), topical steroid and cycloplegic treatment were started. No significant pathology was found in the cranial MRI. Gabapentin 300mg 3*1 was started for the patient by neurology. When the intraocular pressure was found to be 24 mmHg on the left at the follow-up 1 month later, anti-glaucomatous drops were added to the current treatment. The patient did not come to follow-ups regularly and it was noticed that he used antiviral treatment intermittently, but in the follow-up 3 months later, the stromal reaction and the precipitates in the endothelium disappeared. Anti-glaucomatous therapy was discontinued because left intraocular pressure decreased. Sectoral atrophy was present in the iris of the left eye between 3 to 4 o'clock. Considering that the anterior uveitis picture had passed, the patient was given maintenance oral antiviral treatment in case of recurrence, and artificial tears were continued topically. Ptosis and limitation of eye movements were significantly improved (Picture 3). In the examination 1 month later, neurotrophic keratitis was thought to have developed due to the presence of punctate lesions in the epithelium of the cornea. A silicone hydrogel lens was applied, and topical antibiotic drops were added in addition to artificial tears. Maintenance therapy was discontinued after 4 months due to headache and pruritus. However, after 3 months, antiviral treatment was started again due to recurrent anterior uveitis. The patient did not come for control after being followed up for about 4 months.

DISCUSSION

Cranial nerve involvement is rarely seen in HZO. Of the cranial nerves, the most common oculomotor nerve is involved, followed by the sixth and fourth nerves (3). Sometimes more than one cranial nerve may be involved in the same case. Other neurological complications that can be seen in HZO are postherpetic neuralgia, partial ophthalmoplegia, orbital apex syndrome, optic neuritis, encephalitis and contralateral hemiplegia (4,5). Sometimes complete ophthalmoplegia may develop. Complete ophthalmoplegia can be seen in the first one or two weeks after skin manifestations. Its prognosis is good and it completely recovers in about 18 months (6,7).

If the pupil is involved, there may be fixed dilatation. Pupil involvement was also present in our case.

Ocular complications associated with HZO may be more severe, especially in immunosuppressed patients. One of the ocular complications seen in HZO is anterior uveitis. Anterior uveitis is often seen with stromal keratitis 2-4 weeks after the onset of infection, but it can occur years later. Keratitis precipitates in the endothelium, increased intraocular pressure, iris sectoral atrophy and posterior synechiae may accompany anterior uveitis. Herpetic anterior uveitis may recur and the patient should be given maintenance therapy to prevent any recurrence (8). As maintenance therapy, valacyclovir 1gr 2*1 or acyclovir 400mg 3*1 can be given. In our case, maintenance treatment was discontinued after 4 months due to itching and headache. Oral antiviral treatment was started again with the recurrence of uveitis.

Petrhram et al., in their study with confocal microscopy, found a decrease in cell density, an increase in cell sizes and squamous metaplasia in the corneal superficial epithelium and stroma, and it was said that HZO developed due to the involvement of the corneal nerves (9). Hypoesthesia may develop in HZO due to the involvement of the sensory nerves. Depending on the decrease in corneal sensitivity, decrease in blinking and dry eye may develop. Neurotrophic keratitis can range from punctate keratitis to corneal perforation. The presence of hypoesthesia in the patient may prevent them from realizing that the clinical picture has worsened (10).

Permanent vision loss and recurrent attacks reduce the quality of life of patients. In patients presenting with HZO, cranial nerve involvement should also be kept in mind, and examination of the extraocular muscles should be performed together with a detailed eye examination (11). Patients should remain under follow-up due to ocular complications and recurrent attacks that may develop after HZO.

* Informed consent was obtained from the patient regarding this case report.

Conflict of interest

The authors declare that there is no conflict of interest.

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