

Herpes Zoster Ophthalmicus and the Superior Orbital Fissure Syndrome

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ABSTRACT

Herpes Zoster Ophthalmicus (HZO) is not an uncommon condition in the elderly and the immunocompromised. The common ocular manifestations include blepharoconjunctivitis, keratitis and uveitis. Dramatic presentations like orbital apex syndrome and superior orbital fissure syndromes occur rarely in patients with herpes zoster meningo-encephalitis. We report a patient with herpes zoster meningo-encephalitis and the superior orbital fissure syndrome (SOFS).

Keywords : Herpes zoster, superior orbital fissure, meningo-encephalitis

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INTRODUCTION

Herpes zoster ophthalmicus (HZO) is a relatively common syndrome, especially in the elderly and in the immunocompromised. Common ocular manifestations include blepharoconjunctivitis, punctate keratitis and less commonly, anterior uveitis and isolated ophthalmoplegic syndromes. Rarely, however, HZO can present as a superior orbital fissure syndrome (SOFS), associated with meningo-encephalitis. We present a patient with SOFS secondary to herpes zoster meningo-encephalitis.

CASE REPORT

A 61-year-old Malay gentleman first presented to the medical department with the complaints of left hemifacial and fronto-temporal pain. He was noted to

have a mildly injected left eye on follow-up and was referred to the ophthalmology department for treatment. Visual acuity in the right eye was hand motion due to a dense cataract, while that in the left eye was 6/9. There was blepharitis noted and the left conjunctiva was injected with some mucinous discharge. The left eye was pseudophakic and the pupils were equal and reactive to light. He was treated as for left blepharoconjunctivitis with topical antibiotics and lid hygiene.

Three days after admission, he developed vesicles at the tip of his nose. His visual acuity had dropped to 6/18 in the left eye. There was no relative afferent pupillary defect (RAPD) but he was found to have decreased corneal sensation over the left eye. The cornea was, however, clear with no evidence of herpetic involvement. In view of the systemic involvement and a probable state of immunosuppression due to oral steroid therapy, he was started on oral acyclovir.

Two days later, he presented with left complete ptosis, left total ophthalmoplegia (Figs. 1 - 4) and a visual acuity in the left eye of 6/30. There was anisocoria with the left pupil being larger than the right pupil in bright light. No reverse RAPD was present. There was slight proptosis noted and fundoscopy revealed a healthy, pink optic disc. The patient was started on intravenous acyclovir.

A magnetic resonance imaging scan revealed very mild soft tissue swelling over the region of the left cavernous sinus with no obvious thrombosis. Blood investigations showed leukocytosis and a raised differential polymorph count. A lumbar puncture was also performed. The results were consistent with a

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Figs. 1-4 Looking up, down, right and left. Note left total ophthalmoplegia.

diagnosis of aseptic meningo-encephalitis. The patient completed 14 days of intravenous acyclovir therapy before being discharged.

The patient was then seen in August 2000 and his left visual acuity was 6/18. He still showed total ophthalmoplegia with a residual left ptosis that had slightly improved.

In January 2001, right extracapsular cataract extraction and intraocular lens implantation was performed on the patient for his dense right cataract. At the one month post-operative follow-up visit, the patient had a best corrected visual acuity of 6/7.5 in both eyes. His left ptosis had resolved completely and, except for minimal limitation in abduction, he had a full range of movement in his left eye.

Unfortunately, the patient died in August 2001 from respiratory failure secondary to chronic obstructive airway disease.

DISCUSSION

Our patient's clinical presentation of left total ophthalmoplegia, corneal hypoaesthesia (suggestive of involvement of the ophthalmic division of the trigeminal nerve) and the absence of optic nerve involvement was consistent with a SOFS. Only a few cases of complete ophthalmoplegia following HZO have been reported in English medical literature over the past 30 years⁽¹⁾. We report this patient with herpes zoster meningo-encephalitis and SOFS and review the possible pathogenic mechanisms that have so far been postulated.

Edgerton⁽²⁾ suggested that the oculomotor nerve involvement results from contiguous spread of inflammation from the trigeminal nerve to the cavernous sinus or the superior orbital fissure. Garg et al⁽³⁾ proposed that microinfarction of the cranial nerves was a possible etiology. In addition, Naumann et al⁽⁴⁾ found chronic inflammatory cell infiltration in the long posterior ciliary vessels and nerves of 21 enucleated eyes affected by HZO, thereby concluding that the neuropathy arose from an occlusive vasculitis. We feel

that an inflammatory vasculitis is a less likely cause for our patient's ophthalmoplegia. Simultaneous involvement of the third, fourth, fifth and sixth cranial nerves is possible but unlikely in our case. We postulate that the pattern of cranial nerve involvement is more likely to be a result of meningeal inflammation at a specific location, like the superior orbital fissure, in view of the underlying aseptic meningo-encephalitis.

Our patient was treated with intravenous acyclovir for a period of 14 days. At about two months, his vision had improved from 6/30 to 6/18, although a residual left ptosis and left total ophthalmoplegia still remained. At the eighth month follow-up visit, our patient's ptosis had resolved completely and there was no ophthalmoplegia except for mild limitation in abduction. His visual acuity had improved to 6/7.5. Chang-Godinich et al⁽¹⁾ also reported a good prognosis for recovery of the complete ophthalmoplegia following HZO. Of the 16 reported cases, nine had adequate documented follow-up and all showed significant improvement within two months and complete or near resolution within 18 months. Our patient showed near complete resolution after eight months. Treatment regimes are controversial and the effects of steroid or antiviral treatment have not been formally studied⁽¹⁾.

HZO-associated complete ophthalmoplegia tends to affect individuals over 50 years of age. The onset of the ophthalmoplegia is usually within one to two weeks following the development of cutaneous HZO. Hence, although rare, ophthalmologists should be aware that complete ophthalmoplegia may follow cutaneous HZO and has a good prognosis for recovery.

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