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An Acute Case of Herpes Zoster Ophthalmicus with Ophthalmoplegia

KEYWORDS: Herpes zoster ophthalmicus, complete ophthalmoplegia, shingles

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ABSTRACT *Herpes Zoster Ophthalmicus (HZO) with oculomotor nerve involvement is rare, even rarer as an acute presentation rather than sequelae of HZO. In this paper , we present a case of cutaneous HZO in which our patient's initial presentation was one of complete ophthalmoplegia.*

INTRODUCTION

- Herpes Zoster (or Shingles) refers to a typically vesicular rash caused by reactivation of latent varicella zoster virus (chicken pox) from a dorsal root ganglia neurons and cranial nerve sensory ganglion. It usually present in thoracic or cranial dermatomes. The lifetime risk of herpes zoster is estimated to be 10% to 20%; but in patients over the age of 80 years, the risk rises to 50%.

- Reactivation can occur for a number of different reasons including trauma, ageing or immune deficiency[1] and cause necrosis and inflammation of affected sensory ganglion causing corneal anesthesia that may result in neurotropic keratitis.[2]

- HZO is rare form of Shingles, reported in 15-20% of cases[3], that presents with a rash in the distribution of trigeminal nerve dermatomes mainly the ophthalmic and maxillary division. It is often reported to be associated with a variety of complications, including episcleritis and scleritis, keratitis, glaucoma, anterior uveitis, cataract, neurological complications, but there are very few reports of compete ophthalmoplegia being one of those[1, 2].

CASE PRESENTATION

- A 80- year-old lady presented to the Ophthalmology Outer Patient Department (OPD) with an inability to open her left eye. She describes a 10 day history of blister formation surrounding her left eye that extends to her left forehead and scalp. The family noticed her rye was increasingly droopy, red, painful, and swollen culminating in it being permanently shut for fourty- eight hours prior to her arrival in Ophthalmology OPD and then was send to Dermatology OPD and Medicine OPD for further evaluatin where she got admitted to the Medicine Department Ward . She never received any medication before attending the Ophthalmology OPD.

- On Examination; she had a vesicular rash covering her left scalp, forehead and upper cheek surrounding her left eye. It was erythematous, swollen and tender. She had complete ophthalmoplegia, and her pupil was fixed and dilated. Her visual acuity in left eye was reduced to perception of light (PL) and projection of rays (PR). Rest of her neurological examination was found to be normal.

Fig.1- patient presenting with complete ophthalmoplegia.

- She was commenced on oral and topical acyclovir, prednisolone and topical cycloplegic and systemic NSAID's. There was no evidence of vasculitis on slit-lamp examination. The vesicular rash resolved some two weeks later and then she was discharged. She gain some of her eye movements partially but the ptosis remained.

DISCUSSION

In the limited literature, that does report ophthalmoplegia as part of the sequelae of HZO, this has typically been described as a late complication, often up to 2 months after the initial herpetic rash [4] and is seen in only 11–29% of patients with HZO. However, in our case it has developed as part of the acute viral infection. The most commonly affected nerve is the third cranial nerve and, less commonly, the fourth nerve [5].

With a third nerve palsy it has been reported as being partial or complete but there is always ptosis. Isolated ptosis and isolated paralysis of the pupil have also been seen [6]. In our case, there was third cranial nerve (oculomotor) palsy causing complete external ophthalmoplegia, with her pupil fixed and middilated. Several hypotheses have been proposed for the mechanism behind which HZO can result in ophthalmoplegia [5–8] although it is most likely that there are many contributing factors. It is known that the reactivated virus causes inflammation of the axons that supply the dermatomes in question.

Edgerton suggested that the inflammation of the trigeminal nerve could actually spread via the cavernous sinus to affect the oculomotor nerve [6]. In addition, Naumann et al. found chronic inflammatory cells suggesting an occlusive vasculitis[7]. Carrol thought that due to the onset and rate of

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recovery, this pathology would suggest a demyelinating disease, and Lavin et al. were in agreement with this hypothesis based on autopsy reports [8, 9].

Diagnosis of this condition is essentially a clinical one based on history and examination findings. Once the diagnosis is made, it is reported that the earlier treatment is initiated, the better the prognosis is, preferably within 72 hours, although beneficial effects have been reported with treatment started as late as 7 days after onset [10]. Acyclovir, famciclovir or valacyclovir have all been used, and they act by resolving skin lesions, decreasing viral shedding and decreasing the risk of ocular involvement. Some report that famciclovir and valacyclovir are better at resolving pain associated with HZO than acyclovir [10]. In addition to the antivirals it is important to treat any further complications detected. For example, if keratitis or episcleritis has developed, then topical steroids can be used. The effectiveness of steroids and antivirals alone or in combination does not, however, appear to have been formally studied, and treatment options are limited partly due to the poorly defined mechanism of HZO.

The prognosis for full recovery after complete ophthalmoplegia following HZO is good. In one literature review, of 16 total cases, 9 were followed up, and all of those cases showed significant improvement in symptoms after 2 months and almost complete resolution by 18 months [11].

CONCLUSION

Ophthalmoplegia is a rare complication of HZO. Furthermore, the cases in the literature describe it as relatively late sequelae, unlike the acute presentation we have reported.

REFERENCE

[1] R. J.Marsh, "Herpes zoster ophthalmicus," Journal of the Royal Society of Medicine, vol. 90, no. 12, pp. 670–674, 1997. | [2] Jack J Kanski, Brad Bowling; Clinical Ophthalmology, 7th Edition, pg. 187-188, 2011. | [3] D. Pavan-Langston, "Clinical manifestation and therapy of herpes zoster ophthalmicus," Comprehensive Ophthalmology Update, vol. 3, pp. 217–225, 2002. | [4] A. Chang-Godinich, A. G. Lee, P. W. Brazis, T. J. Liesegang, and D. B. Jones, "Complete ophthalmoplegia after zoster ophthalmicus," Journal of Neuro-Ophthalmology, vol. 17, no. | 4, pp. 262–265, 1997. | [5] T. DeLengocky and C. M. Bui, "Complete ophthalmoplegia with pupillary involvement as an initial clinical presentation of herpes zoster ophthalmicus," Journal of the American | Osteopathic Association, vol. 108, no. 10, pp. 615–621, 2008. | [6] A. E. Edgerton, "Herpes Zoster ophthalmicus: report of cases and a review of the literature," Transactions of the American Ophthalmological Society, vol. 40, pp. 390–439, 1942. | [7] G. Naumann, M. Gass, and R. L. Font, "Histopathology of herpes zoster ophthalmicus," American Journal of Ophthalmology, vol. 65, no. 4, pp. 533–541, 1968. | [8] W. M. Carroll and F. L. Mastaglia, "Optic neuropathy and ophthalmoplegia in herpes zoster oticus," Neurology, vol. 29, no. 5, pp. 726–729, 1979. | [9] P. J. M. Lavin, S. G. Younkin, and S. H. Kori, "The pathology of ophthalmoplegia in herpes zoster ophthalmicus," Neuro- Ophthalmology, vol. 4, no. 2, pp. 75–80, 1984. | [10] D. Pavan-Langston, "Herpes Zoster. Antivirals and pain management," Ophthalmology, vol. 115, supplement 2, pp. S13–S20, 2008. | [11]A. Chang-Godinich, A. G. Lee, P. W. Brazis, T. J. Liesegang, and D. B. Jones, "Complete ophthalmoplegia after zoster ophthalmicus," Journal of Neuro-Ophthalmology, vol. 17, no.4, pp. 262–265, 1997.