Expert Opinion

Herpes Zoster Ophthalmicus, Ophthalmoplegia, and Trauma

Case History Submitted by Randolph W. Evans, MD
Expert Opinion by Andrew G. Lee, MD

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Abbreviations: HZO herpes zoster ophthalmicus, HZV herpes zoster virus

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A case of herpes zoster ophthalmicus (HZO) raises interesting questions about risk factors for reactivation and neuro-ophthalmologic complications.

CLINICAL HISTORY

A 78-year-old man underwent extraction of an abscessed right mandibular molar. One week later, he developed a foreign body sensation in the right eye. After a few days, he broke out with a cutaneous vesicular eruption consistent with herpes zoster virus (HZV) infection over the right forehead and side of the nose. He reported mild right-sided headache. Within 2 days of onset of the rash, he was started on oral famciclovir 500 mg three times a day for 7 days. Examination showed minimal corneal epithelial irregularity. Five days after onset of the rash, he developed horizontal diplopia due to a mild right abducens paresis. Findings on ophthalmologic examination were otherwise normal.

Questions.—Could the molar extraction have caused reactivation of the herpes zoster? What are the potential neuro-ophthalmologic complications of HZO? What treatment would you recommend?

EXPERT COMMENTARY

Chickenpox and shingles are both caused by HZV. After the acute primary disease (chickenpox), the virus lies dormant within the sensory dorsal root ganglia, but may reactivate later producing the classic, localized, cutaneous eruption (shingles).[1] Clinical manifestations of HZV include local pain, itching, and a characteristic, cutaneous, segmental, grouped, vesicular rash. The most commonly involved nerve or nerve root distribution is thoracic (50% to 60%), followed by trigeminal (10% to 20%), cervical (10% to 20%), lumbar (5% to 10%), and sacral (<5%). Trigeminal involvement (ophthalmic division) may be associated with conjunctivitis, keratitis, increased intraocular pressure, and iritis (HZO). In addition, there are several well-recognized clinical syndromes of HZV including: (1) otic zoster, the Ramsay Hunt syndrome (internal and external ear vesicles, facial nerve palsy, hearing loss, tinnitus, vertigo, and nystagmus); (2) cervico-facial-oculomotor zoster (cervical ganglion involvement, ophthalmoplegia, facial palsy, tinnitus, and deafness); (3) glossopharyngeal-vagal zoster (paralysis of the palate, pharynx, and larynx; loss of taste of the posterior tongue; and pharyngeal/esophageal vesicles); (4) herpes zoster brachialis (brachial plexopathy); (5) Bell palsy possibly associated with HZV (facial nerve palsy); and (6) herpes zoster hemiplegia (herpes zoster cranialis).

Is Reactivation of the Herpes Zoster Virus Related to Surgery or Trauma?—In many retrospective cases, HZV infection has been associated with an inciting or predisposing event or condition.[2-5] Proposed "triggers" have included advanced age, splenectomy, emotional stress, corticosteroids, concomitant infections (eg, syphilis, sinusitis), underlying neoplasm

Address correspondence to Dr. Randolph W. Evans, Suite 1370, 1200 Binz, Houston, TX 77004 or Dr. Andrew G. Lee, Department of Ophthalmology, The University of Iowa Hospital and Clinics, 200 Hawkins Drive PFP, Iowa City, IA 52242.

(eg, Hodgkin lymphoma, leukemia), ultraviolet light or irradiation, and heavy metal (eg, lead, arsenic) or chemical exposure or toxicity.[1] These mechanisms presumably operate through suppression of the cell-mediated immune response allowing reactivation of the latent HZV. Interestingly, trauma also has been implicated as an inciting factor and may initially cause posttraumatic pain without external signs of HZV; only later evolves the classic, cutaneous, vesicular eruption.[7] Netland et al reported a 38-year-old man who developed HZO 1 day following blunt trauma to the eye as the presenting sign of human immunodeficiency virus (HIV).[2] Surgical procedures (eg, spinal surgery) also have been reported to precipitate local HZV eruptions. Wackym et al reported HZV of the larynx after intubational trauma.[3] The mechanism by which trauma might reactivate HZV is unknown, but reflex irritation and hyperemia of the ganglion have been proposed.[4] It is plausible that the dental surgery in this case could be associated with ipsilateral HZV reactivation.

What Are the Neuro-ophthalmic Manifestations of Herpes Zoster Virus?—Herpes zoster ophthalmicus may be associated with iridoplegia, external ophthalmoplegia, optic neuropathy, facial nerve palsy, and brain stem encephalitis.[6] The most frequently reported neuro-ophthalmologic manifestation is ophthalmoplegia, and the third nerve is most commonly affected; the sixth nerve is affected less commonly, and infrequently, the fourth nerve.[8-11] Complete ophthalmoplegia also has been reported. Onset of cranial neuropathy is often delayed by several days to weeks following the cutaneous eruption. The mechanism of neurologic deficit in HZV is ill defined and numerous possibilities have been suggested including: (1) direct contiguous inflammation of the cranial nerve from the trigeminal nerve, (2) direct extension of the virus into adjacent cranial nerves, (3) secondary inflammatory vasculitis and ischemia, (4) immune-mediated demyelination, (5) muscle ischemia, or (6) orbital perivasculitis/myositis. In this case, contiguous inflammation is possible; the sixth cranial nerve directly communicates with the trigeminal nerve via sympathetic fibers passing from the carotid plexus to the ophthalmic nerves within the cavernous sinus. Less likely mechanisms include orbital myositis or granulomatous vasculitis.

What Treatment Should Be Instituted?—Most reported cases of ophthalmoplegia related to HZV have improved over several weeks to months. Chang-Godinich et al reviewed 16 cases of complete ophthalmoplegia following HZO.[5] Nine cases with adequate follow-up showed complete or nearly complete resolution within 18 months. Of the 16 cases, 9 received no documented treatment. Three patients received steroid treatment alone, 3 patients received acyclovir, and 1 patient was treated with steroid and cyclophosphamide. No firm conclusion could be drawn about the efficacy of treatment in these cases. The mechanism of HZV-related neuro-ophthalmic disease is poorly defined, and what constitutes optimal treatment remains controversial. The high rate of improvement apparently unrelated to therapy argues against any one standard approach. Conceivably, however, corticosteroids might improve any inflammatory component, and antiviral therapy is not unreasonable during the acute infection. We have treated patients with evidence of acute or progressive neuro-ophthalmic deficit (eg, ophthalmoplegia or optic neuropathy) from HZV with systemic antiviral treatment and corticosteroids, but the evidence supporting this approach is limited at best.

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