Acute Herpes Zoster with Hutchinson’s Sign: A Case Report and Overview

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ABSTRACT

The hallmark of herpes zoster ophthalmicus (HZO) is a vesicular rash that involves the first (ophthalmic) division of the fifth cranial nerve that presents in a dermatomal distribution in the midline. Patients with HZO present with a periorbital vesicular rash and/or edema according to the affected dermatome. Sequelae of HZO infection may include chronic ocular inflammation, loss of vision, and debilitating pain. Antiviral medications, such as acyclovir, valacyclovir, and famciclovir remain the mainstay of therapy and are most effective in preventing ocular involvement when begun within 72 hours after the onset of the rash. Diagnosis and management of ophthalmic nerve involvement in early stage are important to limit visual morbidity.

Keywords: Herpes zoster ophthalmicus, Hutchinson’s sign, Varicella zoster virus.

INTRODUCTION

Herpes zoster is a common infection caused by the same virus that causes varicella (i.e., chickenpox). Reactivation of latent varicella zoster virus (VZV) dormant in cranial nerve or dorsal root ganglia produces the characteristic manifestations of herpes zoster, commonly known as shingles. The key risk factor for the development of herpes zoster is waning of the cell-mediated immune system associated with the normal aging process. The lifetime risk of herpes zoster is currently estimated to be 10 to 20%. The other well-defined risk factor for herpes zoster is acquired by inhibiting cell-mediated immune response, immunosuppressive drugs, or infection with human immunodeficiency virus. Thus, normal aging, poor nutrition, and immunocompromised status correlate with outbreaks of herpes zoster, whereas physical or emotional stress and fatigue may precipitate an episode.1-3

Herpes zoster ophthalmicus occurs when reactivation of the latent virus in the trigeminal ganglia involves the ophthalmic division of the nerve. Eye and surrounding structures of perineural and intraneural inflammation of sensory nerves are damaged by the virus; only 10 to 20% of herpes zoster shows ophthalmic involvement.4,5

Extraocular Manifestations of Herpes Zoster Ophthalmicus

The skin manifestations of HZO strictly obey the midline with involvement of one or more branches of the ophthalmic division of the trigeminal nerve, namely the supraorbital, lacrimal, and nasociliary branches. The HZO cases typically present with a characteristic unilateral, localized, vesicular eruption accompanied by pain in the affected dermatome with complaint of insomnia, anxiety, and general malaise. The vesicular eruption is often preceded by localized prodromal pain, sometimes accompanied by headache, malaise, and disrupted sleep. Vesicles contain clear serous fluid, which develops into pustules.6 These lesions rupture and typically crust over, requiring several weeks (approx. 2–3 weeks) to heal completely, but postherpetic neuralgia can persist for months or years thereafter. Immunocompromised persons have a higher risk of developing HZO than the normal population, with generalized vesicular rash in 1 to 2 weeks and which develop into more serious visual sequelae. Viral transmission from patients with HZO is less frequent than transmission from patients with chickenpox, through direct contact with secretions from vesicles and secretion-contaminated articles.7

Ocular Manifestations of Herpes Zoster Ophthalmicus

In HZO, the first (ophthalmic) branch of the trigeminal nerve is affected. The blistering rash associated with
Shingles is followed by numbness, pain, or tingling around the eye. The eye itself is affected in approximately 50% of cases of ophthalmic shingles.8-10 If the nose tip shows the rash, then this signifies involvement of the nasociliary branch of the trigeminal nerve. This branch supplies the globe and makes it highly probable that the eye will be affected. Classically, involvement of the tip of the nose (Hutchinson’s sign) has been thought to be a clinical predictor of ocular involvement followed by chronic ocular inflammation, vision loss, and debilitating pain. Symptoms of HZO include eye pain, conjunctivitis, tearing of mucosal membrane, decreased vision, and eyelid rash. The upper eyelid is commonly involved with edema, inflammation, and resultant ptosis. Other ocular complications include scleritis and iritis, keratitis and corneal anesthesia, and glaucoma.11

CASE REPORT

A 50-year-old male patient came with chief complaints of fever, malaise, and fatigue since 1 week, and also complained of unilateral rash with pain over the right side of eye and forehead region since 2 days. On local examination, he showed peri orbital edema which was restriction to open the right eye, multiple small and large fluid-filled vesicles on the right side of forehead, supraorbital, and lateral aspects of nose and its tip along with low-grade fever. Patient gave history of chickenpox in his childhood, at around age of 10 to 11 years; there was no history of diabetes, no history of any immunosuppressive drugs, and human immunodeficiency virus test was negative. Hutchinson's sign is a clinical predictor of ocular involvement (Fig. 1); periorbital edema is present, but vision was not affected. As patient reported early period of onset of lesion, he was diagnosed as acute herpes zoster with Hutchinson’s sign. Treatment was started with oral acyclovir (800 mg, 5 times daily) for 7 days, and lesion of patient responded well. Follow-up of patient was done for next 6 months and no complaint of postherpetic neuralgia was reported.

DISCUSSION

The vesicular eruption of herpes zoster generally occurs unilaterally in the distribution of a sensory nerve, which involves the trunk or the fifth cranial nerve. Two to 4 days prior to the eruption, there may be pain and paresthesia in the involved area. By definition, HZO is reactivation of VZV in the opthalmic division of the trigeminal nerve (V1), and accounts for 10 to 25% of all herpes zoster cases. While HZO does not necessarily affect the structures of the eye, many of the acute and long-term complications associated with the disease are due to inflammatory response in the eye. It is thought that approximately 50% of those diagnosed with HZO will develop complications. Many of these poor outcomes can be prevented or ameliorated with early recognition, treatment, and referral.9 Classically, HZO begins with flu-like symptoms including fever, myalgia, and malaise for approximately 1 week followed by painful unilateral dermatomal rash in the distribution of one or more branches of V1 nerve: supraorbital, lacrimal, and nasociliary; the skin manifestations usually begin as an erythematous macular rash, progressing over several days into papules, vesicles, and then pustules. These eventually rupture and form scab, but in immunocompetent individuals, this will resolve over the course of 2 to 3 weeks. In about 60% of cases, patients will complain of a painful dermatomal prodrome prior to the development of any rash and ocular involvement is not invariable, but patients with nasociliary nerve involvement (Hutchinson’s sign) have a 100% chance to develop eye pathology. One-third of those without nasociliary involvement will eventually develop eye manifestations.11,12

Diagnostic tests like Tzanck smear or Wright stain are rarely indicated, as diagnosis can almost always be made by a combination of history and physical, but viral culture, direct immunofluorescence assay or polymerase chain reaction can be used to confirm the diagnosis. The main treatment consists of local wound care, pain management, antiviral medication, and antibiotics to prevent secondary infection.5,6 Acyclovir and other similar antivirals have been shown to significantly decrease adverse outcomes related to HZO if started within 72 hours of window period. This leads to reduced pain during the outbreak, reduced likelihood of postherpetic neuralgia, increased rate of skin healing, decreased duration of viral shedding, and decreased incidence of corneal involvement.13,14 It is not proven that the patient accrues these benefits if the medications are started after the 72-hour window. Steroids (topical and systemic) with full antiviral

Fig. 1: Acute herpes zoster with hutchinson’s sign
coverage may also play a role in the treatment of HZO for initial management of pain due to uveitis or scleritis. Oral opiate and nonsteroidal anti-inflammatory medications are frequently indicated for pain and may be augmented by the use of cycloplegics in patients who display features of iritis.

In otherwise healthy individuals with minimal eye involvement (as in our case), most sources suggest 7 to 10 days of 800 mg of acyclovir five times a day improves patient compliance. In high-risk cases, intravenous acyclovir is indicated. Admission is recommended for those with known immunodeficiency and patients with immunosuppressive medications to prevent involvement of multiple dermatomes (which may indicate immunosuppression), retinal involvement, corneal ulceration, or serious bacterial superinfection. All patients with the possible diagnosis of HZO require ophthalmologic consultation prior to discharge from the hospital in order to ensure full evaluation for any serious complications; ophthalmologic follow-up is mandatory. Zaal et al did a study to determine the prognostic value of nasociliary skin lesions (Hutchinson's sign) for ocular inflammation and corneal sensory denervation in acute HZO and suggested that Hutchinson's sign was a powerful predictor of ocular inflammation and corneal denervation in HZO [relative risk: 3.35 (95% confidence interval, CI: 1.82–6.15) and 4.02 (95% CI: 1.55–10.42) respectively]. The manifestation of herpes zoster skin lesions at the dermatomes of both nasociliary branches was invariably associated with the development of ocular inflammation. Clinicians should be alert for early skin lesions within the complete nasociliary dermatome, because they are a reliable prognostic sign of sight-threatening ocular complications in acute HZO.

CONCLUSION

Herpes zoster is diagnosed mostly on the basis of history of patient, characteristic pain, and appearance of the dermatomal rashes. Most often than not, laboratory tests are unnecessary. Empirical treatment should be given without waiting for results of diagnostic tests because delay in treatment could result in less response to treatment and postherpetic neuralgia. Acyclovir and other similar antivirals have been shown to significantly decrease adverse outcomes related to HZO, if started within 72 hours of initial symptoms. So the early diagnosis and treatment are a must to prevent further complications.

REFERENCES