

Herpes zoster ophthalmicus and its varied clinical manifestations

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Abstract

Introduction: Herpes Zoster Ophthalmicus (HZO) is a viral infection caused by varicella zoster virus. The virus manifests in primary and recurrent forms. HZO involves ophthalmic division of trigeminal nerve affecting one or all other branches (lacrimal, supratrochlear, nasociliary). The diagnosis is simple with clinical examination in majority of cases. The lifetime risk of herpes zoster is estimated to be 10 to 20 percent. The complications are seen in primary and recurrent herpes zoster both in immunocompetent and immunocompromised patients.

Aims: To analyses varied clinical manifestation in herpes zoster ophthalmicus.

Materials and Methods: We analysed 40 cases of established HZO. The demographic profile, clinical signs and symptoms, and outcomes were observed and analysed. They were screened for diabetes and hypertension as a part of routine checkup. HbA1c (glycosylated Hb) was done in all diabetics. Serological testing for HIV, HbsAg, & VDRL was done in all cases. History of chicken pox in the past was documented if any.

Results: All 40 cases had conjunctival and corneal involvement. 11(27.5%) out of 40cases, had complete dermatomal involvement,17(42.5%) had intra ocular involvement. 7 patients had uncontrolled diabetes with HbA1c values between 8 to 10. 13 patients had deep seated retro ocular pain before the onset of skin lesion.

Conclusion: HZO take a longer course in patients with abnormal HbA1c values. Multiple dermatomal involvement can be observed. Deep seated retro ocular pain along with burning sensation along the distribution of nerve should arouse high index of suspicion of HZO, as a premonitory symptom. The skin lesions appear different with edematous skin in albino patient.

Introduction

Herpes Zoster Ophthalmicus (HZO) is a viral infection caused by varicella zoster virus. The virus remains dormant in ganglia before its manifestation. The virus manifests in primary and recurrent forms. HZO involves ophthalmic division of trigeminal nerve affecting one or all other branches (lacrimal, supratrochlear, nasociliary). The disease has prodromal symptoms with fever, sorethroat, malaise before the onset of rash with an incubation period of 5-7 days, even before ocular manifestations. The diagnosis is simple with clinical examination, however diagnosis can be established with elisa, tzank smear in cases with varied clinical manifestation. Here we are going to analyze some of varied clinical signs and symptoms in our series of cases. The lifetime risk of herpes zoster is estimated to be 10 to 20 percent.^{1,2} The complications are seen in primary and recurrent herpes zoster both in immunocompetent and immunocompromised patients. The complications include encephalitis, myelitis, cranial and peripheral nerve palsies, and a syndrome of delayed contralateral hemiparesis.³

Materials and Methods

The study was an observational prospective study, done at Sapthagiri Institute of Medical Sciences, Bangalore, between January 2014 to June 2017. We had 40 cases of established HZO. The demographic profile, clinical signs and symptoms, and outcomes were observed and analysed. All the patients were included irrespective of age (Fig. 1). There were 15 male and 25 female patients. All cases were diagnosed by clinical examination. A thorough ocular examination was done under slit lamp biomicroscope. Fluorescein staining was done in 100% of cases. Corneal sensation was tested in all cases. Visual acuity was recorded using Snellen's drum at first visit as well as in their last visit. A thorough dilated ophthalmoscopic examination was done in all the cases. They were screened for diabetes and hypertension as a part of routine checkup. HbA1c (glycosylated Hb) was done in all diabetics. Serological testing for HIV, HbsAg, & VDRL was done in all cases. History of chicken pox in the past was documented if any.

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Observations

The different variations in presentation of HZO is summarized in table 1. All 40 patients developed papular, vesicular, pustular skin lesions in the distribution of V1 dermatome. One female developed skin lesions in both V1 & V2 dermatomes, which is unusual and rare (Fig. 2). HZO of left sided involvement was more observed in 10 males and 21 females. 18 out of 40 patients had skin lesions on the forehead and upper lid area. 11 patients had skin lesions on the forehead, upper lid and lower lid area. 11 patients had skin lesions in the entire V1 dermatome, like on the forehead, upper lid, lower lid and lateral part of the nose involving the tip of the nose. One patient had unusual presentation with two dermatomal involvement, this patient happened to be uncontrolled diabetic with HbA1c value of 10. In another case HZO was seen in an albino with uncontrolled diabetes. All 40 patients had conjunctival involvement. Corneal sensation was totally absent in about 24 patients, while remaining 16 patients had reduced or equivocal response. The corneal involvement was seen as punctate keratitis and was confirmed with fluorescein staining in 25 cases. Out of 11 patients involving the complete dermatome, 4 patients had corneal affection alone, 5 had keratitis and iridocyclitis, one patient had iridocyclitis with secondary glaucoma and one patient had 6th cranial involvement. A total of 8 patients had keratitis with secondary glaucoma. A total of 3 patients had secondary glaucoma alone in addition to skin involvement. A total of 7 patients had diabetes with HbA1c values between 8 to 10. 13 patients had severe retro ocular pain prior to skin involvement. Skin lesions received warm saline soaks with antibiotic application till scab formation. Thirteen patients with deep seated retro ocular pain received oral acyclovir 800mg five times a day and continued for 5 days after the onset of skin lesions. Intravenous acyclovir (10mg/kg) was given in one case of neurological involvement for 10 days. Patients with keratitis and iridocyclitis, received oral steroids under cover of oral acyclovir. An another patient with iridocyclitis and secondary glaucoma was treated with oral acetazolamide, oral steroids under cover of oral acyclovir.



Fig 1: HZO in different age groups



Fig 2: Two dermatome involvement

Discussion

HZO presents with V1 dermatome involvement after a prior infection of chicken pox any time during lifetime in the past. The disease accounts for 10-20% of all herpes zoster cases.^{1,2} The patients will have flu like illness before the onset of skin lesions. All our cases had conjunctival and corneal involvement. In our series, 11(27.5%) out of 40 cases, had complete dermatomal involvement, out of which 7 cases (63.63%) showed intra ocular involvement as against 100% involvement in some other case series.^{3,4} There is 50% chance of patients with herpes zoster ophthalmicus (HZO) developing significant ocular complications like neurotrophic keratopathy, episcleritis, iritis, epithelial or stromal keratitis, etc.⁵ In our series all patients had skin, corneal and conjunctival involvement. 17(42.5%) out of 40 cases had intra ocular involvement. One patient had 6th nerve palsy. Patients with immunocompromised status, age over 60 years, uncontrolled diabetes, HIV, overabuse of steroids, anti cancer therapy, patients on terminal stages of cancer may all predispose an individual to develop herpes infection. There are very few studies comparing diabetic effects in HZO.⁶⁻⁸ In our study 7 patients had uncontrolled diabetes with HbA1c values between 8 to 10.⁹ Hence HbA1c values have more relevance in disease manifestation & management of HZO cases. There is a strong evidence to support the routine and early use of systemic antiviral therapy in all patients with HZO in an effort to reduce the risk of ocular complications.¹⁰ All patients were treated with topical acyclovir, 13 patients with deep seated retro ocular pain were treated with oral acyclovir before the onset of skin lesion, diabetics with abnormal HbA1c and albino with HZO were also treated with oral acyclovir. Patients with keratitis and uveitis, received oral acyclovir along with oral steroids. It was observed that patients who received oral therapy before the onset of skin lesion had less pain and faster recovery with average of 2 weeks.¹¹⁻¹⁴ High risk cases like HZO in AIDS, multiple dermatome involvement, immunocompromised patients, nerve palsy & anti cancer therapy patients require intravenous acyclovir therapy.^{15,16} Intravenous acyclovir (10mg/kg) was given in one case of neurological involvement for 10 days in our study. However, famciclovir and Valciclovir can also be used orally other than acyclovir.^{17,18} The role of vaccine in

preventing the reactivation was unremarkable.¹⁹ Various clinical trails have proven its efficacy. The herpes zoster vaccine was approved for use in United states in the year 2006. The administration of Varicella zoster vaccine has been in use above the age of 60 years, as it reduces the incidence of herpes zoster and post herpetic neuralgia to a large extent. Development of a heat-inactivated VZV vaccine for use in immunocompromised patients is an area of active investigation,²⁰ however I_gG immunoglobulins can be administered as a post exposure therapy in immunocompromised patients.

Table 1: HZO manifestation

S. No	Signs & Symptoms	No of cases and %
1	Skin lesions	40(100%)
2	Conjunctival lesions	40(100%)
3	Corneal involvement alone	40(100%)
4	Keratitis & iridocyclitis	5(12.5%)
5	Iridocyclitis with secondary glaucoma	1(2.5%)
6	Keratitis with secondary glaucoma	8(20%)
7	Secondary glaucoma alone	3(7.5%)
8	Burning with deep seated retro ocular pain	13(32.5%)
9	6 th cranial nerve involvement	1(2.5%)
10	Diabetes with zoster	7(17.5%)

Conclusion

HZO take a longer course in patients with abnormal HbA1c values. An HbA1c value >9, appears to have a correlation with the rapid onset of skin lesions. Multiple dermatomal involvement can be observed even though it is unusual. Deep seated retro ocular pain along with burning sensation along the distribution of nerve should arouse high index of suspicion of HZO, as a premonitory symptom. The skin lesions appear different with edematous skin in albinotic patients.

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