Sequential Presentation of Sixth Nerve Palsy in Herpes Zoster ophthalmicus

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Abstract

Herpes zoster ophthalmicus (HZO) is a reactivation of varicella zoster virus involving the ophthalmic division of trigeminal nerve. The acute course is usually benign; however, serious ocular complications like keratitis, iritis, muscle palsies and optic neuritis can occur in approximately 50% of the cases. The extraocular muscle palsies associated with HZO are mostly transient and self-limited, seen in 7 to 31% of patients. We report a case HZO with sequential presentation with dendritic corneal lesion, followed by maculo-papular vesicular skin eruptions followed by sixth nerve palsy within a week of onset of rash. The patient was treated with oral and topical antivirals along with topical steroids. The skin lesions healed over 6 weeks' time with improvement in extraocular movements. Radioimaging and blood investigations are important in these cases to rule out any co-existing possible life threatening conditions. Once these conditions are ruled out then one could be sure that the nerve palsy was secondary to HZO, having a favorable outcome.

Introduction

Herpes zoster affects 20% to 30% of the population at some point in their lifetime, and approximately 10% to 20% of these have Herpes zoster ophthalmicus (HZO), which is the reactivation of varicella zoster virus involving the ophthalmic division of trigeminal nerve. Severity is related to age of the patient with older patients (≥60 years) being affected much more severely than younger patients [1]. The acute course of HZO is usually benign; however, serious ocular complications like keratitis, iritis, muscle palsies and optic neuritis have been documented in approximately 50% of the cases [2,3]. The extraocular muscle palsies associated with HZO is a transient, self-limited condition seen in 7 to 31% of patients; usually appearing 2-4 weeks after the rash, but sometimes occurring simultaneously with rash or more than 4 weeks later [4,5]. Herein, we report a case HZO with sequential presentation with dendritic corneal lesion, followed by zosteriform skin rashes followed by sixth cranial nerve (CN VI) palsy within a week of onset of rash.

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was revised to herpes zoster ophthalmicus (HZO) with keratouveitis L/E and the patient was started on oral valacyclovir, topical steroids, cyclopentolate and timolol maleate. R/E examination was unremarkable.

Four days later, on seventh day of presentation, patient again came with complaints of doubling of vision for one day which was more pronounced in left gaze. On examination, esotropia of 10 PD was seen with restricted abduction in left eye (Figure 3). Diplopia charting was consistent with sixth nerve palsy (CN VI) in left eye. The general physical examination and rest of the neurological examination was unremarkable.

MRI brain and orbit shows age related changes in the form of diffuse cerebral atrophy with foci of chronic ischemic infarcts and perivascular gliosis with no obvious mass lesion (Figure 4). The negative neurological imaging and all other blood investigations ruled out acquired causes of the paresis of the nerve, such as vascular, cerebral accident, brain tumor and encephalitis.

Oral valacyclovir was continued for 10 days. Antiglaucoma medication was stopped after intraocular pressure control had been achieved. Dermatological consultation was sought for skin lesions which resolved in 2-3 weeks. Diplopia improved in primary gaze within 2 weeks and at 6 weeks there was recovery in abduction in left eye (Figure 5).

Figure 1. a) Peripheral small dendrites at 6 o'clock position b) Fluorescence staining dendrites

Figure 2. a) Vesicular eruptions over left eye and tip of the nose involved. b) Scalp lesions respecting the midline.
Figure 3. Extraocular movements showing right esotropia and limited abduction of Left eye

Figure 4. a) FLAIR axial images of the brain shows diffuse cerebral atrophy with foci of chronic ischemic infarcts and perivascular gliosis. b) T2 weighted axial image of brain with orbit shows no obvious mass lesion
Discussion

HZ typically affects individuals in 6th to 8th decades that are exposed to varicella zoster virus previously in their childhood. Our patient presented at 81 years with isolated CN VI palsy induced by HZO without any memorable incident of symptomatic zoster infection previously. The total incidence of extraocular muscle palsy following HZO was reported by Edgerton to be 13% [6]. The oculomotor nerve (CN III) is most commonly involved (47%), followed by the abducens nerve (CN VI) (23%), and then the trochlear nerve (CN IV) (10%). Complete ophthalmoplegia (CN III, CN IV, and CN VI palsies occurring concurrently) following an outbreak of HZO, is estimated to occur 20% of the time [2, 4].

On an average, HZO-induced ophthalmoplegia occurs 9.5 days after the onset of HZO rash in 75% of cases with a range of 2–42 days [7]. Our patient presented with diplopia on forth day after the appearance of first zosteriform rash and seven days after the appearance of corneal epithelial keratitis. Kreibig et al. postulated that the extraocular palsies were due to perivasculitis-myositis, rather than to a neural origin. Muscle ischemia remains a strong possibility, as does a combination of cortical nerve and muscle inflammation. Therefore, isolated abducens nerve palsy might be caused by circumscribed orbital myositis or a lymphocytic cranial motor neuropathy [4]. Typically, the ophthalmoplegia is self-limiting and results in complete or near-complete resolution on its own in 4.4 months on an average in 65–76.5% of afflicted individuals. However, duration varies and can range from 2 weeks to 1.5 years [7]. Our case showed resolution of diplopia in primary gaze after 2 weeks and recovery of abduction in 12 weeks after the onset.

The treatment of HZO-related CN palsy is controversial. Systemic antivirals have been reported to reduce the risk of viral dissemination. Some authors have recommended systemic corticosteroids to treat the possible vasculitic component and/or to prevent postherpetic neuralgia [8]. However, steroids should not be given alone (without antiviral therapy) due to concerns about promotion of viral replication. Additionally, they may increase the risk of secondary skin infection [9]. In our case, the patient responded quite well to valacyclovir treatment and had no side effects. Since the patient was already hypertensive, systemic steroids were avoided.

Conclusion

HZO can present with a wide range of acute and chronic complications. One should keep in mind, especially in the elderly, that CN palsy may result due to some other co-morbidity. Thus, timely referral, imaging and blood investigations are important to rule out possible life threatening conditions. Once these conditions are ruled out then one could be sure that the nerve palsy was secondary to HZO, having a favorable outcome.

References


