Case Report

A Case of Herpes Zoster Ophthalmicus With Oculomotor Nerve Palsy

Özlem Kayım YILDIZ, Hatice SEĞMEN, Ertuğrul BOLAYIR, Ahmet Suat TOPAKTAŞ

Cumhuriyet University Faculty of Medicine, Neurology, Sivas, Türkiye

Abstract

Herpes zoster ophthalmicus may rarely be complicated with ocular motor nerve palsies. The exact mechanism of the palsies is unclear. The present report describes a case of herpes zoster ophthalmicus complicated with an oculomotor nerve palsy. A 85-year-old woman who had suffered from right herpes zoster ophthalmicus developed ipsilateral oculomotor nerve involvement 12 days later. The patient was treated with acyclovir and prednisolone. We report the case with a brief review of the ophthalmoplegia associated with herpes zoster ophthalmicus.

Keywords: Herpes zoster ophthalmicus, oculomotor nerve, treatment

INTRODUCTION

Herpes zoster is a relatively common syndrome, its prevalence is higher among elderly and in immunocompromised people. The condition affects approximately 1 million people in the United States (US) annually\(^8,13,15\). The lifetime risk of herpes zoster is 20%, but patients older than 85 years have an incidence rate of 50%\(^7,13,15,16\). One of the nerves most commonly affected by Varicella zoster virus (VZV) is trigeminal nerve, particularly the ophthalmic branch. Herpes zoster ophthalmicus (HZO) can cause a variety of ocular and neurologic complications including paralysis of ocular motor nerves. We examined a patient exhibiting oculomotor nerve palsy associated with herpes zoster, and report this case with a review of the literature.

CASE PRESENTATION

A 85-year-old woman presented with sharp, lancinating pain and sudden vesicular cutaneous eruption in the ophthalmic division of the trigeminal nerve on the right side of her face as well as ptosis. The patient's medical history was
significant for hypertension and coronary artery disease. Family, social, and surgical history were noncontributory. Her blood pressure was 130/90 mm Hg; pulse, 86 beats per minute; respiration, 18 breaths per minute; and body temperature, 37 C.

Examination of the right eye revealed complete ptosis, congestion and chemosis of conjunctiva, corneal ulceration and melting resulting in complete loss of vision, loss of corneal sensation, absence of supraduction, infraduction and adduction movements of eye ball. The pupil could not be examined because of corneal ulceration. The left eye was normal, with 6/6 visual acuity and full extraocular motility. There were multiple grouped vesiculobullous lesions along ophthalmic division of right trigeminal nerve including side of the nose, right half of forehead and scalp (Figure). Motor functions of the trigeminal nerve were normal. The right ophthalmic and maxillary divisions of the trigeminal nerve had decreased sensation to pinprick testing. Other aspects of the neurologic and physical examinations were within normal limits.

Magnetic resonance imaging (MRI) of the brain was performed to rule out other diseases causing oculomotor nerve palsy; there were no specific findings other than old infarctions in the periventricular white matter. Blood tests were normal, including liver and renal function tests. Leucocyte count was 5800/cmm with 68% polymorphs, erythrocyte sedimentation rate was 11 mm/hr. Blood sugar was within normal limits.

Diagnosis of ophthalmic zoster with oculomotor nerve palsy was made and the patient was given oral acyclovir 4000 mg/d, prednisolone 60 mg/d, analgesics, local applications of acyclovir and tobramycin and cyclopentolate eye drops.

**DISCUSSION**

The nerves most commonly affected by VZV are the sensory nerves of the thoracic dermatomes, followed by the cranial nerves(1,7,11). HZO represents between 10% and 25% of all cases of herpes zoster and 50% of these cases involve the ocular component(7,17). Complications of HZO include scarred lid malfunction or loss; blepharoconjunctivitis, neurotrophic keratitis, scleritis, iritis, uveitis, hemorrhagic retinitis, secondary glaucoma, cataract, acute retinal necrosis, choroiditis, papillitis, retrobulbar neuritis, optic atrophy, iridoplegia, Argyll Robertson pupil, external ophthalmoplegia, sympathetic ophthalmia, and facial neuropathy(1,16,17).

The most frequently reported neuro-ophthalmologic manifestation is ophthalmoplegia. The onset of the ophthalmoplegia is usually within 2 to 4 weeks following the development of cutaneous rash but sometimes occurs more...
than 4 weeks later or simultaneously, or rarely the paralysis may precede the eruption\(^5\). The ophthalmoplegia occurs in 7–33\% of patients with HZO and the third nerve is most commonly affected and the fourth nerve the least\(^6\). Sometimes the paralysis involves several nerves simultaneously. However, complete ophthalmoplegia is reported to be a very rare complication of HZO\(^3\). The third nerve may be total or partially involved. There is always ptosis. The pupil is often dilated partially, but may be spared. Isolated paralysis of the pupil may be the only symptom\(^7\). Isolated ptosis may be seen\(^7\). In our case, there was oculomotor nerve palsy causing complete external ophthalmoplegia, however, due to corneal ulceration, we could not examine the pupil and accommodation.

The mechanism of oculomotor nerve involvement in HZO is controversial. Numerous mechanisms have been postulated including:

1. Direct contiguous inflammation of the cranial nerve from the trigeminal nerve\(^8\). Edgerton proposed that oculomotor nerve involvement in HZO results from continuous spread of inflammation from the trigeminal nerve to the oculomotor nerves as they traverse the cavernous sinus or the superior orbital fissure\(^7,18\).

2. Direct extension of the virus into surrounding neural tissue. Diffusion of viral particles from the ganglion and the branches of the trigeminal nerve to adjacent cranial nerves might be the mechanism\(^5\).

3. Secondary inflammatory occlusive vasculitis and microinfarction. The virus induces an occlusive vasculitis that causes an ischemic neuropathy\(^8\). Naumann et al. found chronic inflammatory cell infiltration in the long posterior ciliary vessels and nerves of 21 enucleated eyes affected by HZO\(^14\).

4. Immune-mediated demyelination. The delayed pattern of onset and high rate of recovery point to a demyelinating process\(^12\).

5. Compression. Chang-Godinich proposed that intraorbital inflammation induces an elevated pressure in the orbit, leading to proptosis, ophthalmoplegia and myositis, which compresses the nerves\(^3\).

6. Meningeal inflammation\(^14\).

The few cases studied at autopsy have shown monocytic infiltration, axonal degeneration, demyelination, and vasculitis of vasa nervorum\(10, 12\). It is probable that all of these mechanisms of disease may be responsible for the clinical presentation.

The compression mechanism is unlikely in our patient because of the normal MRI of the orbits, showing no signs of intraorbital inflammation or myositis. Microinfarction of the oculomotor nerve could be responsible and also the ophthalmoplegia may be the result of perineural inflammation at a specific location, due to a spread of the virus out of the fifth cranial nerve.

Treatment of HZO may reduce the incidence and severity of ocular complications\(^16\). Acyclovir, valacyclovir, and famciclovir are approved by the US Food and Drug Administration for the management of herpes zoster. Antiviral therapy is most effective if begun within the first 72 hours of rash onset, but oral acyclovir given as late as 7 days after onset has been shown to be effective\(^16\). Antiviral therapy reduces the duration of viral shedding and new lesion formation. Some authors recommend initial treatment with intravenous acyclovir for several days before starting oral forms of antiviral drugs\(^8\). Some data show that famciclovir and valacyclovir are superior in alleviating herpes zoster-associated pain than acyclovir\(^2,16\). In the case of HZO, if there are severe pain and rash, and particularly significant edema which may cause orbital apex syndrome, oral steroids may be added\(^4,16\). Because the mechanism of
herpes zoster ophthalmoplegia is poorly defined, the treatment regimes are controversial. As ophthalmoplegia tends to resolve spontaneously, the effects of specific treatment are not clear. The prognosis is good, improvement from complete ophthalmoplegia following HZO can be seen within 2 months, complete resolution generally occurs within 18 months. Rarely, patients may have severe ophthalmologic and neurologic complications of HZO, such as encephalitis, acute retinal necrosis and granulomatous angiitis of the central nervous system. Our patient has been following in our outpatient clinic and there has been no significant improvement from ophthalmoplegia.

Although rare, physicians should be aware that ophthalmoplegia may follow HZO, therefore evaluation of extraocular muscle and lid function is essential to effectively manage accompanying ocular motor palsy.

Correspondence to:
Özlem Kayım Yıldız
E-mail: ozlemkayim@yahoo.com

Received by: 01 December 2008
Revised by: 03 January 2009
Accepted: 13 January 2009

The Online Journal of Neurological Sciences (Turkish) 1984-2009
This e-journal is run by Ege University Faculty of Medicine, Dept. of Neurological Surgery, Bornova, Izmir-35100TR as part of the Ege Neurological Surgery World Wide Web service. Comments and feedback:
E-mail: editor@jns.dergisi.org
URL: http://www.jns.dergisi.org
Journal of Neurological Sciences (Turkish) Abbr: J. Neurol. Sci.[Turk]
ISSNe 1302-1664

REFERENCES