Abducens Palsy Associated with Herpes Zoster Opthalmicus: Case Report

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Abstract: The occurrence of ocular palsies in the course of herpes zoster opthalmicus (HZO) is a rare but severe complication. The oculomotorius nerve is most commonly involved, whereas there are only a few published cases reporting paralysis of the abducens nerve. We describe the case of a 55-year-old woman who developed a sixth nerve palsy a few days after appearance of a painful skin lesions on the right side of her forehead. Polymerase chain reaction (PCR) was positive for VZV. The patient was treated with intravenous acyclovir. 6 months after initial manifestation, the patient still had a partial palsy of the abducens nerve.

Keywords: Abducens nerve, palsy, herpes zoster opthalmicus, ocular complications, varicella zoster virus.

INTRODUCTION

Herpes zoster opthalmicus (HZO) occurs after reactivation of varicella-zoster virus (VZV) infection of the ophthalmic first division (V1) of the trigeminal nerve. It is the second most common type of all herpes zoster (HZ) cases (10-20%) [1,2]. Without antiviral therapy, about 50% of HZO cases develop ocular complications [3,4].

Risk for developing HZ increases with depression of specific cell-mediated immunity to VZV caused by immunosuppressive conditions (chemotherapy, immunosuppressive medication, consuming infectious or neoplastic disease) or, most commonly, with old age [2,5].

The clinical characteristics of HZO include a painful, unilateral, vesicular rash consisting of blisters standing together in groups (herpetiform) on forehead, periocular area, eye, and nose in dermatomal distribution [6,11].

The most common complication of HZ is the development of postherpetic neuralgia, which is defined as a persisting pain lasting up to several months after cutaneous clearing.

Ocular involvement may be of acute or chronic nature and includes conjunctivitis, keratitis, uveitis, iritis, episcleritis, scleritis, uveitis, secondary glaucoma, cataract, HZ-associated chorioretinitis, retinal necrosis, periorbital edema, optic atrophy and ocular cranial-nerve palsies [2,5,8-10]. Greatly dreaded permanent sequelae of HZO may include chronic ocular inflammation, loss of vision by retinal necrosis, and debilitating pain, which could result in permanent damage, such as blindness [2,12].

CASE REPORT

A 55-year-old woman was referred to our department due to a blistering eruption in the right hand-sided ophthalmic area which was accompanied by severe headache and ocular pain. Physical examination showed multiple grouped vesicular-bullous lesions on tremendously swollen erythematous skin of the ipsilateral scalp, forehead, temple, and orbital region. There were also signs of a bacterial superinfection. In addition, the patient presented with conjunctival injection (Figure **1**).

Figure 1: Typical clinical manifestations of HZO at subacute stage: grouped vesicular-bullous lesions, partly crusted on erythematous and swollen skin along the sensory distribution of the first (ophthalmic) division (V1) of the trigeminal nerve.

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Due to the severe edema of the eyelids, our patient was not able to open her right eye actively. On ophthalmologic consultation, the patient was primarily diagnosed with keratitis and iritis. Laboratory examination only revealed elevated levels of C-reactive protein (3.15 mg/dL). PCR was positive for VZV. A CT scan ruled out orbital cellulitis.

From these clinical and laboratory findings, we diagnosed the patient with HZO and started parenteral treatment with acyclovir (750mg 3x/day) and clindamycin (600mg 3x/day). As topical therapy, she received acyclovir containing eye cream and gentamicin-containing eye drops.

After a few days, in the subacute stage of the disease, we noticed severe problems of eye motility. The involved eye could no longer follow the examining finger to the right (Figure **2**).

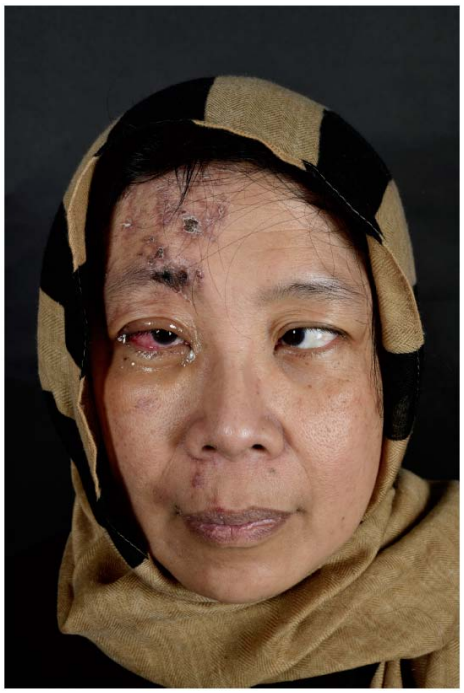

Figure 2: Impaired ocular motility: The right eye cannot follow the finger of the examiner to the right. Please, notice ipsilateral conjunctivitis.

Neurologic examination together with MRI did not show any abnormal findings such as multiple sclerosis, tumor, or stroke which could explain abducens nerve palsy other than HZO-associated.

The skin lesions as well as conjunctivitis and iritis improved, whereas abducens nerve paralysis remained unchanged even after the patient had been discharged. We therefore advised our patient to continue with valacyclovir (1000 mg 3x/day) as well as gabapentin (300mg 3x/day), metamizole (1000mg 3x/day) and a topical therapy with acyclovir (5x/day). After 6 months, the patient still had a partial palsy of the abducens nerve (Figure **3**).

Figure 3: After 6 months of follow-up, the patient still had a partial palsy of the abducens nerve.

DISCUSSION

The occurrence of ocular palsies in the course of HZO is a rare but severe complication. The oculomotorius nerve is most commonly involved, whereas there are only a few published cases reporting paralysis of the abducens nerve [1-8]. Interestingly, the occurrence of ocular palsies is neither correlated with age, sex nor with severity of HZV infection [7]. The pathogenesis of these severe ocular complications is poorly understood at present. Possible mechanisms may include a cytopathic effect of the virus on the surrounding nerve tissue, innate immune responses against parts of the central nervous system, perivasculitis-myositis, muscle ischemia, or a combination of both, orbital nerve and muscle inflammation [3,5-7]. Complete recovery of nerve palsies usually may be expected after 2 to 24 months [6,7].

ETHICAL APPROVAL

Written informed consent was obtained from the patient.

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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None.

ABBREVIATIONS

VZV = varicella zoster virus

HZ = herpes zoster

HZO = herpes zoster ophthalmicus

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