• Letter to the Editor •

Abducens-nerve palsy with ipsilateral herpes zoster ophthalmicus and skin rash: a case report

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Dear Editor,

Herpes zoster (HZ), which is characterized by a unilateral painful dermatomal rash, is caused by reactivation of the latent varicella-zoster virus (VZV) in the dorsal root ganglia following primary infection during childhood[1]. HZ tends to occur more frequently in older adults, in whom cell-mediated immunity often declines. The incidence of HZ among immunocompetent unvaccinated individuals aged >50y is 9.92/1000 person-years[2]. HZ ophthalmicus (HZO) occurs when HZ involves the first division of the trigeminal nerve, *i.e.*, the ophthalmic nerve[3]. Symptoms related to ocular involvement occur in 50% of HZO cases. Owing to their potential to affect all ocular tissues, manifestations such as conjunctivitis, uveitis, episcleritis, keratitis, and retinitis are possible.

Although cranial neuropathy is common in patients with HZO, involvement of the 3rd (oculomotor), 4th (trochlear), or 6th (abducens) cranial nerves leading to extraocular-muscle paralysis is rare. In a recent study of 330 patients with HZ with cranial-nerve paralysis, only one case of 6th cranial-nerve palsy was identified[4]. Another report indicated that among 11 patients with HZ-associated cranial polyneuropathy, seventh cranial-nerve palsy was the most common, occurring in 81.8% of the cases, whereas 6th cranial-nerve palsy was observed in only one patient[5].

Although cases of 6th cranial-nerve palsy associated with HZO have been reported, they are extremely rare[6]. Here, we present a case in which a skin rash was observed during the acute phase of HZ, with concurrent neuralgia, keratitis, and ipsilateral 6th cranial-nerve palsy along with a literature review.

Ethical Approval The Institutional Review Board (IRB) for Human Studies of Daegu Catholic University Hospital reviewed and approved the study protocol (IRB No. CR-23-173). The patient provided informed consent for publication of this case. The study was conducted in accordance with the Declaration of Helsinki. Informed consent for publication was obtained.

A 79-year-old woman presented to the emergency room with sudden-onset horizontal diplopia in both eyes that started two days earlier. She had no significant medical history, such as diabetes, hypertension, or autoimmune diseases, and no notable ophthalmic history. The patient reported worsening of diplopia on looking to the right side. Approximately one week before the onset of diplopia, the patient had experienced discomfort around her right eye, described as a stabbing sensation, and had noticed a rash on her right forehead five days prior to presentation. Brain computed tomography and magnetic resonance imaging performed to determine the cause of diplopia revealed no significant intracranial lesions. Serum VZV immunoglobulin G (IgG) was detected at three times the reference value in routine blood and biochemistry tests. Additionally, VZV IgG was detected in the cerebrospinal fluid (CSF). The patient was admitted to the neurology department because of suspected HZ. After admission, treatment was initiated with intravenous acyclovir (Acrova®, KyungDong pharm. Co., Republic of Korea) 500 mg/d and intravenous methylprednisolone sodium succinate (Methysol®, Alvogen Korea co., Republic of Korea) 1000 mg/d. Additionally, a topical acyclovir cream (Acyclovir Cream KDC, Korean Drug Co., Republic of Korea) was applied to the skin lesions four times a day. A collaborative consultation with the ophthalmology department was requested to evaluate the bilateral diplopia.

During the initial ophthalmic examination, the best-corrected visual acuity was 0.6 in the right eye and 1.0 in the left eye. Intraocular pressure and visual fields were normal. Fundus

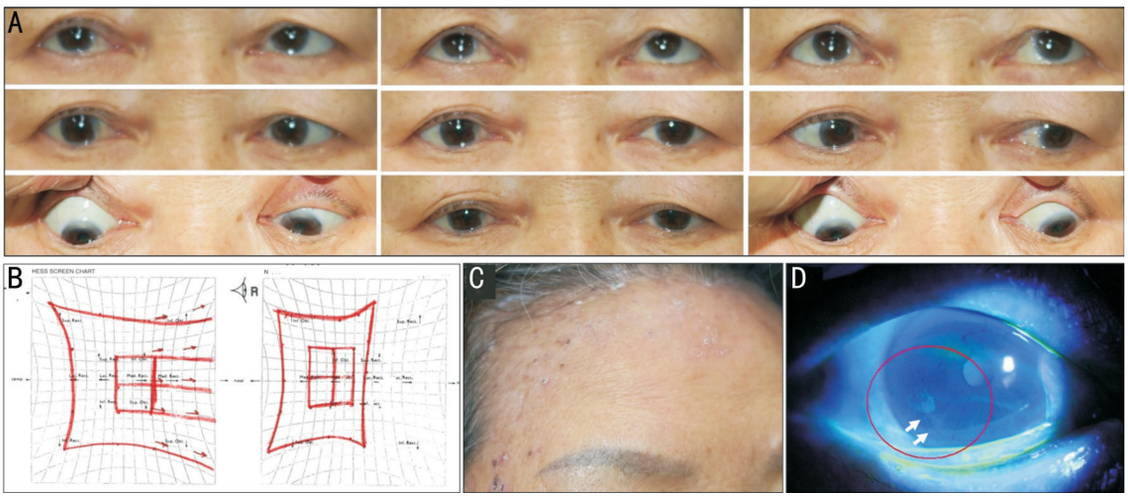

Figure 1 Ocular and external findings at the initial visit A: Nine gaze photographs show esotropia of 16 prism diopters and abduction limitation of grade -3 in the right eye; B: Hess screen test shows limitation of right gaze in the right eye; C: Multiple vesicular and scab-like lesions are observed on the right side of the forehead (photos in almost healed stage); D: Slit-lamp examination shows punctate keratitis (red circle) with linear pseudodendritic lesion (white arrows) in the right eye.

examination and optical coherence tomography revealed no abnormalities of the optic nerve or macula. Ocular motility examination revealed restricted abduction of -3.0 in the right eye (Figure 1A), and the alternate prism cover test showed esotropia of 16 and 6 prism diopters in the right eye at distance and near, respectively. The Hess screen test revealed limited right lateral rectus movement, suggesting abducens-nerve palsy (Figure 1B). External examination revealed clustered vesicles and crusts on the right forehead; however, no signs of Hutchinson syndrome were observed (Figure 1C). Slit-lamp examination revealed conjunctival swelling, rounded superficial punctate keratopathy, and pseudodendritic lesions in the right eye (Figure 1D) along with anterior-chamber inflammation, raising the suspicion of ophthalmic HZ. Therefore, application of acyclovir ophthalmic gel (Herpesid Ophthalmic Gel; Samil Pharm. Co., Republic of Korea), prednisolone (Predbell®, Chong Kun Dang Pharm. Co, Republic of Korea) and 0.3% sodium hyaluronate eye drops (Hyaluni E. D., Taejoon Pharm Co., Republic of Korea) four times daily to the right eye were initiated. Levofloxacin eye drops 1.5% (Cravit®, Santen Pharm. Co, Republic of Korea) with the same number of times was used in combination since concurrent infection with HZO and bacterial keratitis could not be excluded.

After one week of treatment, the alternate prism cover test revealed esotropia of 18 and 6 prism diopters at distance and near in the right eye, respectively. However, the patient reported a subjective decrease in the frequency of visual disturbances and no ocular discomfort. After one month of treatment, the patient reported a further reduction in the frequency of diplopia. Owing to the improvement in the degree of strabismus and abduction restriction in the right eye, the dosage of topical medications was gradually reduced. After three months of treatment, the patient's visual disturbances completely resolved. The alternate prism cover test confirmed the presence of orthophoria in all directions (Figure 2A), and the Hess screen test indicated resolution of abduction restriction (Figure 2B). Slit-lamp examination revealed completely resolution of the corneal abnormalities without scarring (Figure 2C). The patient was diagnosed with ipsilateral 6th-nerve palsy associated with HZO presenting as a skin rash and keratitis.

HZ can affect various nervous systems, such as the cranial, spinal, and autonomic nerves, and their innervated target tissues, resulting in complications such as neuropathy and encephalitis[7]. Several studies have described that cranial neuropathy caused by HZ shows a wide spectrum of clinical presentations and various degrees of recovery[2,4,7-9]. However, ophthalmoplegia due to involvement of the 3rd, 4th, and 6th cranial nerves is extremely rare, and few individual cases have been reported[5-6,10]. Pupić-Bakrač *et al*[5] reported one case of abducens-nerve palsy with permanent cranial-nerve damage. In addition, Tsau *et al*[4] reported a rare case of HZ affecting the ophthalmic branch of the trigeminal nerve that developed into complete ophthalmoplegia, affecting the oculomotor, trochlear, and abducens nerves. Because ophthalmoplegia due to HZ is extremely rare, the present case, in which shingles, HZO, and paralytic strabismus presented simultaneously due to concurrent trigeminal and abducens nerve involvement, is meaningful.

Cranial neuropathy caused by HZ infection includes neuropathic pain and dysfunction of the cranial nerves involved[7]. The specific mechanisms underlying HZ reactivation are not fully understood. Immunocompromised

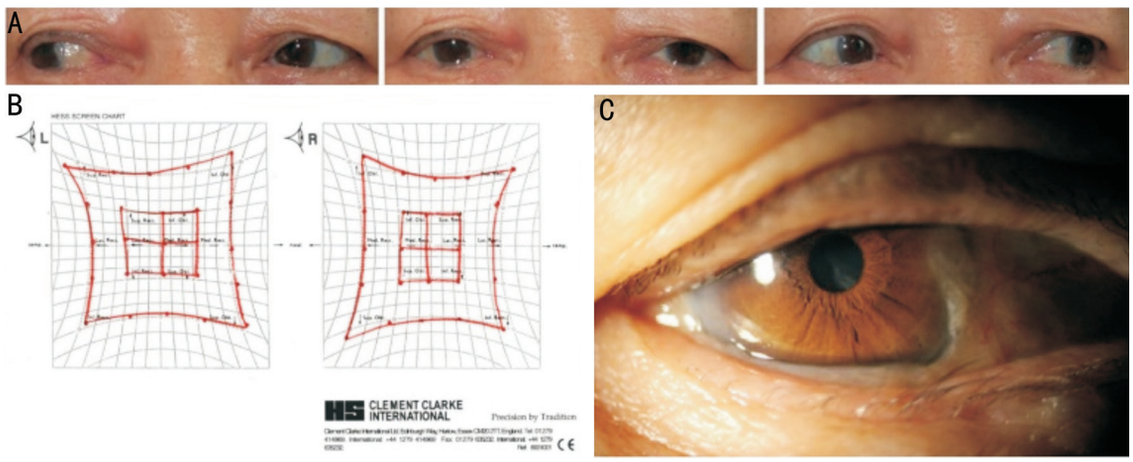

Figure 2 Ocular findings during the follow-up period A: At the 3-month follow-up, diplopia is completely. Orthotropia is observed the primary position, and right gaze limitation is not observed; B: At the 3-month follow-up, Hess screen test shows no limitation of right gaze; C: Slit-lamp examination shows healing of the corneal lesions.

status, mental stress, old age, and systemic vascular disease are the risk factors. However, HZ can also occur in immunocompetent patients[9]. Since our patient did not have hemodynamic risk factors such as diabetes and hypertension, we presumed that sensory and motor dysfunction may have been caused by VZV reactivation due to age-related temporary weakness of the immune system rather than ischemic causes. Cranial motor neuropathy in HZ may be attributed to activation of cellular immune responses in response to reactivation of latent VZV in the sensory ganglia, which causes collateral damage to the adjacent cranial motor nerves[4,11]. With respect to ocular motor-nerve involvement, unilateral ophthalmic neuralgia with eyeball involvement, ocular-muscle paresis, or both, can occur.

Rash and pain are usually observed in HZ, and in most cases, the diagnosis of HZ is based on clinical history and physical examination[7]. Regarding virological testing, polymerase chain reactions tests for detection of VZV DNA in the blood, saliva, and CSF can be helpful for the diagnosis of HZO. Mehta *et al*[12] performed polymerase chain reaction tests on saliva collected from 54 patients with HZ on the first day of skin lesions, and all 54 patients (100%) tested positive for VZV DNA. Measuring the levels of immunoglobulins against VZV is also beneficial for diagnosing HZO. Kangro *et al*[13] detected VZV immunoglobulin M (IgM) antibodies in all 261 patients with HZ using an IgM antibody-capture radioimmunoassay (MACRIA) to detect IgM antibodies. VZV IgM antibodies have been reported to be meaningful when detected within 3.5wk of symptom onset[14]. In the study by Ihara *et al*[15], VZV IgG was detected in 92 of 98 (93.9%) patients. Our patient experienced stabbing pain around the eye starting two days before the appearance of skin lesions on the right forehead. Slit-lamp examination revealed pseudodendritic lesions in the corneal epithelium. Furthermore, blood and CSF tests revealed VZV IgG, which may serve as an important clue in the diagnosis of HZO. Because IgM, an indicator of acute infection, was not detected in either serum or CSF, the diagnosis of HZO in this case was made based on a comprehensive evaluation of the characteristic clinical symptoms and blood test results.

Once cranial-nerve neuropathy is thought to be caused by VZV, systemic antiviral treatment is warranted. Antiviral therapy is substantially more effective if initiated within 72h of the onset of neuropathy[5]. Systemic steroids are often added, and Tsau *et al*[4] reported that the one-month recovery rate was higher in patients administered steroids compared to that in patients not administered steroids (88.4% *vs* 40.0%). In the present case, the administration of both drugs was initiated immediately after diagnosis and resulted in favorable outcomes without any sequelae after 3mo of treatment.

The progression of ophthalmoplegia due to HZ varies, and the recovery rate of cranial motor neuropathy reported in previous studies varies from 63.63% to 96%[4-5]. In individual case series, favorable prognoses have been reported by several authors[6,10]. In contrast, Tokoro *et al*[8] reported a case in which the skin lesions healed within a month, but abducens-nerve paralysis persisted for >1y despite aggressive antiviral treatment. Pupić-Bakrač *et al*[5] demonstrated that 63.6% of patients achieved completely recovered within an average of 2.76mo but permanent abducens-nerve damage was observed in few patients. The mean duration of significant improvement in cranial motor-nerve palsy was 26.2d, which was longer than that of sensory nerve palsy (14.6d)[4]. In the present case, the rash and vesicles resolved earlier than the abducens-nerve palsy. Our patient showed complete recovery of abducens nerve palsy 3mo after symptom onset, which may have been due to aggressive systemic antiviral treatment or her immunocompetent status without underlying disease.

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Conflicts of Interest: Kim JW, None; **Lee GW,** None; **Lee D**, None.

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