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# **Case Report**

# Bilateral abducens nerve palsy in herpes zoster: A case report

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#### ABSTRACT

Herpes zoster (shingles) is a viral infection that is caused by the reactivation of varicella-zoster virus (VZV), which remains dormant in the sensory ganglia after the preliminary infection. We document a case of ocular motor paralysis due to herpes zoster. A 17-year-old girl came to the casualty with complaints of fever, headache, rashes, and double vision. She had a few grouped vesicles involving the right side of the neck area adjacent to the midline above the suprasternal notch suggestive of herpes zoster. Extraocular movement was restrained on abduction in both eyes. Both eye anterior segment examination and fundus exam showed normal findings. The cerebrospinal fluid analysis suggested viral etiology. Magnetic resonance imaging of the brain showed normal findings. She was diagnosed with bilateral abducens nerve palsy as a result of herpes zoster virus. The skin lesions resolved and ocular movements became normal within 6 weeks.

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#### 1. Introduction

Herpes zoster ophthalmicus (HZO), is a potentially blinding disorder. It involves the 5th cranial nerve. The occurrence of HZO complicating herpes zoster ranged between 8-20%. Eye involvement can occur as a primary infection or recurrence from latent ailment and involve all ocular tissues: lid rashes, blepharitis, conjunctivitis, epithelial keratitis, stromal keratitis, endothelitis, iritis, trabeculitis and retinitis. In rare cases it can get complicated by involvement of Extraocular muscle. Here, we document an interesting case of HZO that caused bilateral abducent palsy.

## 2. Case Report

A 17-year-old- girl presented to us with complaints of fever for four days which was sudden in onset, intermittent and there has been headache associated with vomiting and neck

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ache. Three days later she developed a vesicular skin rash on the right-side face and neck with excoriation and she had double vision on primary gaze. On local examination few grouped vesicles were present over the right side of the neck adjacent to the midline above the suprasternal notch. On ocular examination, abduction was constrained in both eyes. Both eye anterior segment slit lamp examination and dilated fundus examination was normal. Raised serum C reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were noted.(Table 1) Cerebrospinal fluid (CSF) analysis confirmed raised opening pressure, elevated white blood cell count (WBC), and adenosine deaminase. CSF cytology confirmed an elevated lymphocyte count of 85%.(Table 2) CT scan and contrast-enhanced MRI brain suggested no considerable abnormality. Dermatologist consultation was sought and as the patient had multiple grouped vesicles present over the right side of face at the mandibular area and neck, the diagnosis of Herpes Zoster involving mandibular branch of 5th cranial nerve and C2 C3 dermatome with bilateral sixth nerve palsy was made. The patient was

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commenced on intravenous acyclovir 850mg three times a day for 14 days, intravenous ceftriaxone 1000mg three times a day for seven days, and dexamethasone 4mg intravenous once a day for 5 days. The skin lesions disappeared within 2 weeks and improvement in ocular motility was seen over the subsequent months. The patient sixth nerve function was completely restored on sixth week follow up. Consent was obtained from patient for publication of case report and photographs.

**Table 1:** Showing the blood investigations

S. No.	Parameter	Values
1	Haemoglobin	12.1 g/dl
2	Packed Cell Volume	36%
3	Red Blood Cell count	5millions/ $\mu$ l
4	White blood cell count	9100 cells/cu.mm
5	Neutrophil	77%
6	Lymphocyte	15%
7	Eosinophil	0%
8	Monocyte	8%
9	Platelet count	$353 \times 1000 \mu l$
10	Erythrocyte Sedimentation	1hr-30mm
	Rate	
11	C- Reactive protein	1.2 mg/dl
12	Random blood sugar	137 mg/dl
13	Serum Creatinine	0.9%

**Table 2:** Showing the cerebrospinal fluid analysis

S. No	Parameter	Values
1	Opening pressure	70cm H <sub>2</sub> 0
2	WBC count	240 cells/cu.mm
3	Protein	13 mg/dl
4	Adenosine Deaminase	15.0 IU/L
5	Lymphocyte	85%

## 3. Discussion

Infection with the varicella-zoster virus (VZV) presents in two different ways. Primary infection with VZV causes chickenpox and is characterized by vesicular lesions on the face, trunk, and extremities in different stages of development. Herpes zoster is due to the reactivation of dormant VZV infection inside the sensory ganglia. Herpes zoster virus infection results in ocular and facial lesions with capability progression to more severe complications. Mostly the third nerve is infected with herpes zoster, while abducens nerve palsy complicating herpes zoster is quite uncommon. <sup>1</sup>

We have presented a case of bilateral abducent nerve paralysis in an affected person with herpes zoster. The intracranial course of the sixth cranial nerve is long which makes it relatively at risk of inflammation and injury. Amongst all cranial nerves, sixth nerve palsy is the most common cause affecting ocular motility. Presentation is with horizontal diplopia, and face turn towards the affected side and worsening of diplopia on seeing to the affected side. <sup>1–3</sup> Our patient also presented with similar symptoms of diplopia.

The mechanism of involvement of ocular motor nerves or muscles in zoster is not very well understood. Numerous hypotheses which consist of vasculitis, muscle ischemia, contiguous intracavernous radiculomeningitis, or cranial motor neuropathy have been reported. Kreibig et al postulated that extraocular palsies are caused by perivasculitis myositis, rather than by a neural origin. Denny-Brown et al observed that inflammation of the ganglion is not the cause of motor neuritis. <sup>3</sup>

The pathophysiology may be the result of one of the following- Direct cytopathic impact of the virus on the brain tissue and the central nervous system, immunological reaction to the infection, and occlusive vasculitis.<sup>4</sup>

In 7-31% of patients, HZO may cause extraocular muscle palsies due involvement of third, fourth, and sixth cranial nerves. Extraocular muscle palsies typically develop 2-4 weeks after the rash. On presentation, our patient had both rashes and sixth nerve paralysis. Concurrent incidence of rashes and palsy has been stated previously. <sup>5</sup> HZO rarely presents with total ophthalmoplegia. <sup>6</sup>

Treatment is done with the antiviral agent (acyclovir, famciclovir, valacyclovir) that crosses the blood-brain barrier. Antiviral therapy works well if all started in the first seventy-two hours of rash onset. Treatment with antivirals induces rapid resolution of skin lesions, reduces viral shedding, and decreases the chance of corneal and uveal involvement. In case the antiviral medication is not administered, 50% of patients with HZO may have direct ocular involvement causing conjunctivitis, uveitis, episcleritis, keratitis, and acute retinal necrosis. 8 Nucleotide analogs inhibit the preliminary viral replication and severity of infection by preventing the direct cytotoxic effect of the virus, the immune response of the surrounding neural tissue, and secondary vasculitis. Systemic corticosteroids may additionally prevent occlusive vasculitis, however, there is a chance of suppressing the immune system. 5 Our patient was started on antiviral drugs on 1st day, along with steroids and the patient began showing resolution of signs and symptoms in weeks.

The prognosis of HZO is considered to be good. The reported duration of diplopia associated with ocular motor palsy is between 2 and 23 months. 9 In our case patient recovered for approximately six weeks.

## 4. Conclusion

Herpes Zoster can present with various ocular complications. Abducent nerve palsy secondary to Herpes Zoster should be kept in mind even when evaluating such cases. The pathophysiology of abducens nerve palsy in Herpes Zoster is not well known. The advantages

of antiviral medications and corticosteroids in treating oculomotor palsy remain controversial.

## 5. Source of Funding

None.

### 6. Conflict of Interest

None.

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