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**Case Report**

# Herpes zoster ophthalmicus: An eye and skin emergency

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**Abstract** Herpes Zoster (HZ) is an acute painful infection caused by reactivation of latent Varicella Zoster Virus (VZV) manifesting as a painful vesicular rash in a dermatomal distribution. Herpes Zoster Ophthalmicus (HZO) is implicated with high risk of ocular involvement which can even result in permanent vision loss. We report a case of an elderly female who presented with severe pain on right upper face followed by a papulovesicular rash, was found to have early ocular involvement and later developed the symptoms of encephalitis. Timely diagnosis and prompt treatment resulted in complete recovery in minimal duration.

***Key words***
Varicella Zoster Virus; Herpes Zoster; Ophthalmicus; Encephalitis.

## Introduction

The Varicella Zoster Virus (VZV) is responsible for two distinct diseases: Varicella and Herpes Zoster (HZ). After the primary infection i.e. Varicella, the virus lies inactive inside dorsal root ganglia or cranial ganglia. When reactivated, it travels to the skin via afferent nerve to cause herpes zoster in the corresponding dermatome.[1] Herpes zoster is more common in adults aged >50 years, the reason being decline in cell mediated immunity with rising age.[2] The clinical presentation of herpes zoster is variable. In the majority of cases, a prodrome of dermatomal pain or paraesthesias is followed by unilateral dermatomal rash that begins with macules and papules on an erythematous base. Subsequently, clustered vesicles appear which progress to pustules and then crust. Healing occurs in 2 to 4 weeks with or without scarring.[3,4]

Herpes zoster ophthalmicus (HZO) is a particularly severe variant that accounts for about 20% of all zoster cases. It occurs when VZV reactivation presents in the ophthalmic division of the trigeminal nerve. Reactivation manifests as pain and cutaneous rash limited to the periorbital area; however, 50 to 72% of cases develop involvement of the eye itself.[1,3] Rash involving the tip of the nose, the Hutchinson sign, is highly correlated with ocular involvement.[3,4]

Encephalitis is a rare but deadly complication of VZV infection seen in approximately 0.25% of herpes zoster cases.[5] It typically presents with altered behaviour and delirium, especially in elderly patients. In all cases of suspected CNS involvement imaging studies (CT or cMRI) and a lumbar puncture including VZV PCR should be performed immediately.[5,6]

The cornerstone of treatment for HZO is prompt initiation of intravenous acyclovir therapy (8-10mg/kg) for all patients alongwith adjuvant

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therapies such as antibiotics, topical or systemic corticosteroids as well as supportive care for symptoms control.[7,8]

**Case description**

A 60 years old housewife presented in Outpatient Department of Dermatology with severe pain on right side of forehead radiating down the right side of face and nose accompanied with a papulovesicular rash on same area for the last 3 days. Around one week ago, patient experienced a tingling sensation over right side of forehead that gradually increased in intensity and changed into severe continuous burning pain, non-responsive to paracetamol and NSAIDS. On fourth day, patient developed macular erythema over right side of forehead and periorbital area that progressed to papules followed by vesicles formation within the course of next 2 days. Patient developed marked swelling of right eye accompanied with watery discharge and photophobia. She also reported a flu like illness with low grade fever, bodyaches and malaise for last one week. She had history of chickenpox in her childhood but no history of zoster rash before and no history of zoster vaccination. Patient was on medications for hypertension for ten years with good control. Physical examination revealed grouped papules and vesicles and a few hemorrhagic blisters on an erythematous and edematous base over right side of forehead extending down to involve right periorbital area, right side of nose, right nasal ala and tip of nose (Hutchinson's sign). Both upper and lower eyelids of right eye were grossly swollen .The skin manifestation strictly respected the midline along the distribution of ophthalmic branch of trigeminal nerve **(Figure 1)**. Patient had blood pressure 140/90mmHg, temperature 100F, pulse rate 92/min, body weight was 72kg. Her blood counts were normal and biochemical profile was unremarkable. Tzanck smear taken from the base of fresh vesicles showed multinucleated giant cells. Patient was admitted in Dermatology ward with clinical diagnosis of herpes zoster ophthalmicus (HZO) and treatment was started on Acyclovir 500mg IV tid accompanied with symptomatic treatment. Detailed eye evaluation by ophthalmology department revealed visual acuity of 6/18 in both eyes and right sided conjunctival injection and chemosis. Sensations over right cornea were lost. Slit lamp examination revealed few punctate corneal erosions. Intraocular pressure was normal in both eyes. The ophthalmologist established the diagnosis of blepharoconjunctivitis, episcleritis/ scleritis and neuropathic keratitis secondary to HZO, and prescribed cold compresses, artificial tears and topical antibiotics therapy in addition to ongoing systemic antiviral therapy. On next day after admission patient started having symptoms of delirium and confused behaviour alongwith episodes of agitation and visual hallucinations. There were no clinical signs of meningeal irritation nor any focal neurological deficits. Her serum electrolyte levels, hepatic and renal profiles were checked and were normal. CT brain and CSF analysis were advised by Neurology department on suspicion of encephalitis. CT revealed mild cerebral edema. Lumbar puncture was refused by the patient and her family. Dexamethasone 4mg qd was started

**Figure 1** Papulovesicular lesions involving right side of forehead, periorbital region and tip of nose.

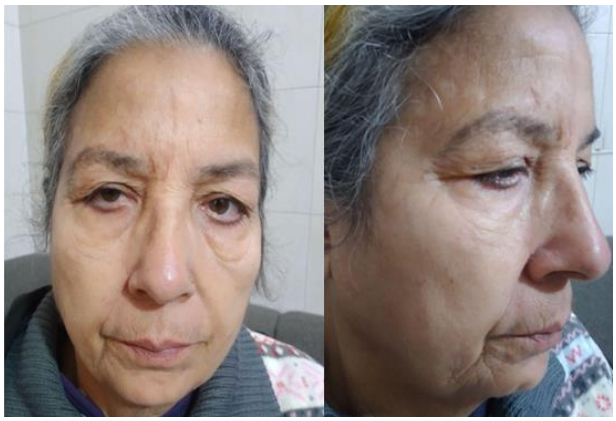

**Figure 2** After treatment: lesions healed with mild scarring.

and acyclovir dose was escalated to 750mg tid. Patient's sensorium and behaviour showed rapid improvement. Dexamethasone was stopped after 3 days. Cutaneous lesions and pain improved gradually and there was almost complete healing at 2 weeks. Antiviral was stopped after completion of 14 days and patient was discharged from hospital. She was followed two weekly for two months and then monthly for a further 6 months. There was no recurrence of pain or neurological complaints and the rash healed with minimal scarring **(Figure 2)**.

## Discussion

Herpes zoster ophthalmius has very characteristic history and clinical appearance. Additional procedures like slit lamp examination, ocular tonometry and corneal esthesiometry can be performed for a more thorough evaluation for ocular complications.[9] Studies have shown that the diagnosis of herpes zoster solely based on clinical findings has a specificity of 60–90%, depending on severity of disease and site of involvement.[8] Other diagnostic investigations such as viral cultures and polymerase chain reaction (PCR) using blister fluid, aqueous humor or ocular swabs should be reserved for atypical cases.[9]

Differential diagnoses include herpes simplex (HSV 1), cellulitis/erysipelas, insect bite reaction, contact dermatitis and drug reaction. There was no preceding history of exposure to irritants, drugs or any insect bite. Herpes simplex caused by HSV-1 mostly occurs around mouth but can present at any site and rarely can come in a dermatomal pattern (zosterifom herpes simplex). The main clinical clue for differentiation is that vesicles of herpes simplex are smaller and uniform in size within a cluster in contrast to those seen in herpes zoster which has a polymorphic rash.[10]

Our patient had typical history of prodromal illness and pain followed by a classical rash so diagnosis of HZO was straightforward. Tzanck smear showed many multinucleated giant cells that are characteristic of infections caused by herpes viruses. Extent of eye involvement was assessed by slit lamp examination and tonometry done by ophthalmologist.

Confused behaviour and delirium developing suddenly during hospital admission was another challenge in the management of our patient. Probable causes for delirium in elderly patients include organ failure leading to metabolic encephalopathies, electrolyte imbalances, cerebrovascular accidents, intracerebral hemorrhage, drugs toxicity or withdrawal, dehydration, and cardiovascular events.[11] All these causes were ruled out after thorough clinical evaluation and laboratory tests of metabolic profile and serum electrolytes. CT brain showed mild cerebral edema in both temporal lobes which is a non specific feature of brain injury or inflammation. Lumber puncture was refused by the patient. Based on clinical symptoms suggestive of encephalitis, co-occurrence of zoster rash, and absence of other causes for altered behaviour, the diagnosis of encephalitis was established in collaboration with neurology department and decision was made to start dexamethasone and to escalate the

dose of acyclovir to 750mg (10mg/kg) tid which was continued for 14 days. Steroids were stopped as soon as her clinical condition improved.

To summarise, herpes zoster ophthalmicus is an ophthalmological and skin emergency and prompt diagnosis and treatment is needed to relieve acute symptoms and prevent devastating complications.

***Declaration of patient consent*** The authors certify that they have obtained all appropriate patient consent.

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***Conflict of interest*** Authors declared no conflict of interest.

***Authors' contribution***

**ZA,AK**: Diagnosis and management of the case, manuscript writing, has given final approval of the version to be published.

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