Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Pharmacy and Pharmacology

Journal homepage: https://www.ijpp.org.in/

Case Report

Herpes zoster ophthalmicus of the right eye involving the ophthalmic branch of the trigeminal nerve: A rare case report

Animesh Das[1]*, E Satheesh Kumar[1]

[1]Dept. of Pharmacy Practice, RR College of Pharmacy Chikkabanavara, Bangalore, Karnataka, India.

Abstract

This case describes the clinical features, investigations, and management of Herpes Zoster Ophthalmicus (HZO), a viral infection affecting the ophthalmic division (V1) of the trigeminal nerve. The patient presented with multiple fluid-filled lesions on the right side of the face for two days, fever for three days, pain, burning sensation on the right side of the face, headache, neck pain, giddiness for one day, and burning with blurred vision in the right eye. On examination, there was swelling of the right eyelid and conjunctival redness, suggestive of eye involvement. The patient was admitted to the dermatology department for further care. Blood investigations showed haemoglobin 14.9 g/dL, red blood cells 5.45 million/cumm, hematocrit 46.9%, platelets 2.50 lakhs/cumm, and white blood cell count 8540 cells/cumm, with neutrophils 67%, lymphocytes 21%, monocytes 11%, eosinophils 1%, and basophils 0. The patient had no history of eye discharge, hearing problems, seizures, unconsciousness, facial weakness, or other comorbid conditions. His history included recurrent headaches for two years, treated with medications. He had already taken acyclovir 800 mg before admission. The patient underwent relevant investigations, and encephalitis was ruled out by the medicine team. An ophthalmology opinion was taken to assess ocular complications. Conservative management was planned, and antiviral therapy with acyclovir was continued. Herpes Zoster Ophthalmicus occurs due to reactivation of the varicella-zoster virus (VZV), which belongs to the genus Varicellovirus. Reactivation can cause direct viral damage, immune-mediated inflammation, or occlusive vasculitis (blockage of blood vessels). Treatment mainly involves early initiation of antiviral drugs, such as acyclovir, and in some cases, corticosteroids to reduce inflammation. Prompt treatment helps prevent serious complications, including vision loss, and improves overall recovery.

Keywords: Herpes Zoster, Ophthalmology, Trigeminal nerve, Varicella-zoster virus.

Received: 03-10-2025; **Accepted:** 20-11-2025; **Available Online**: 02-02-2026

This is an Open Access (OA) journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

Herpes Zoster Ophthalmicus (HZO) is a viral infection that affects the ophthalmic division (V1) of the trigeminal nerve. This condition is caused by the varicella-zoster virus (VZV), which is a human alpha herpesvirus belonging to the genus Varicellovirus. The same virus is responsible for chickenpox (varicella) during primary infection and shingles (zoster) when it reactivates later in life. The first infection with VZV usually occurs in childhood. The virus enters the body through the respiratory tract and multiplies in the epithelial cells of the upper respiratory mucosa. From there, it spreads in the blood to the skin, causing chickenpox. After chickenpox resolves, the virus does not leave the body. Instead, it hides in a sleeping (latent) form inside the sensory ganglia of cranial and spinal nerves.[1] **(Figure 1)**

When a person's immunity becomes weak, the latent virus can become active again. In such cases, the virus travels along the sensory nerves to the skin, leading to herpes zoster (shingles). In herpes zoster ophthalmicus (HZO), reactivation occurs in the trigeminal ganglion, especially in the ophthalmic division (V1) of the trigeminal nerve. This nerve has three main branches: the lacrimal, frontal, and nasociliary nerves, which provide sensation to the eyelid, forehead, brow, and the tip of the nose. Herpes zoster typically appears as a painful, unilateral vesicular rash, meaning it affects one side of the body or face. Complications may include HZO, Ramsay Hunt syndrome, acute retinal necrosis, and post-herpetic neuralgia. In Ramsay Hunt syndrome, patients may also develop facial paralysis and ear lesions.[2,3] **(Figure 3)**

The disease develops because during the primary chickenpox infection, the virus enters the skin vesicles,

*Corresponding author: Animesh Das
Email: dranimeshdas3113@gmail.com

https://doi:10.18231/j.ijpp.13649.1764070058
© 2025 The Author(s), Published by Innovative Publications.

infects the nerve endings, and then migrates through the sensory axons to remain dormant in the neurons. After many years, when the immune system weakens, the virus reactivates. It moves back along the nerve pathways, causing acute neuritis (nerve inflammation). This leads to pain, rash, and sometimes more serious nerve damage. The dorsal root ganglia often show inflammation, bleeding, and even death of nerve cells. In HZO, the virus may also cause ophthalmoplegia, which is weakness or paralysis of the eye muscles. This can happen due to a direct damaging effect of the virus, an immune reaction, or occlusive vasculitis (inflammation and blockage of blood vessels). In some cases, another dormant virus may be triggered by VZV, worsening the nerve damage.[4,5]

Risk factors for herpes zoster include advancing age, psychological stress, immune system suppression, HIV infection, cancer, chronic diseases (like diabetes, kidney disease, and lung disease), autoimmune disorders, and physical trauma. In people with weak immunity, such as those with AIDS, herpes zoster may appear in multiple areas (multi-dermatomal) or spread widely across the body (disseminated zoster). Patients with HZO may develop complications such as burning eye pain, Hutchinson's sign (rash on the tip of the nose), vision problems, and even blindness if untreated. About 10–30% of patients with HZO develop ophthalmoplegia. Usually, this is seen on the same side as the rash (ipsilateral), while bilateral cases are extremely rare.[6,7] **(Figure 2)**

Another rare complication is SIADH (Syndrome of Inappropriate Antidiuretic Hormone Secretion), which can disturb fluid balance in the body. The exact mechanism of nerve palsy in HZO is not fully understood but is thought to be due to VZV-induced vasculopathy, immune response, or direct viral toxicity.

Treatment involves the early use of antiviral drugs such as acyclovir, along with corticosteroids in some cases, to reduce inflammation. Early treatment helps reduce complications and improve recovery.[2]

2. Case Report

A 28-year-old male, residing in Bangalore, Karnataka, was admitted to the dermatology department with chief complaints of multiple fluid-filled lesions on the right side of the face for two days, fever for three days, pain, burning sensation on the right side of the face, headache, neck pain, giddiness for one day, and burning with blurring of vision in the right eye. He had no history of eye discharge, hearing difficulty, seizures, loss of consciousness, facial weakness, or other comorbidities. History included recurrent headache for two years managed with medications, and he had taken acyclovir 800 mg before admission. He had no past surgical history, family history, or addictions. Personal history revealed a mixed diet, normal appetite, adequate sleep, and regular bowel and bladder habits.

Upon examination, the patient was moderately built, well-nourished, oriented, and cooperative, with no signs of pallor, icterus, cyanosis, lymphadenopathy, or oedema. Local cutaneous examination revealed grouped vesicles on an erythematous base over the right periocular region, nose tip, and right ala nasi, with some crusted lesions, oedema of the right eyelids, and conjunctival congestion. Systemic examination showed normal cardiovascular, respiratory, abdominal, and neurological findings. His blood pressure was 122/80 mmHg, and his pulse, oxygen level, and breathing rate were within the normal range. **(Table 1)**

Investigations performed showed: renal function within normal limits (urea 24 mg/dL, creatinine 0.7 mg/dL, sodium 138 mmol/L, potassium 4.9 mmol/L, chloride 104 mmol/L), liver function with elevated total bilirubin 2.1 mg/dL (indirect 2.0 mg/dL, direct 0.1 mg/dL), proteins normal (albumin 4.1 g/dL, globulin 2.7 g/dL, A/G ratio 1.5), AST 36 U/L, ALT 55 U/L, ALP 50 U/L. Haematology showed haemoglobin 14.9 g/dL, RBC 5.45 million/cumm, hematocrit 46.9%, platelets 2.50 lakhs/cumm, WBC 8540 cells/cumm (neutrophils 67%, lymphocytes 21%, monocytes 11%, eosinophils 1%, basophils 0). Serology was negative for HCV, HBsAg, and HIV 1 & 2. LDH was 210 U/L. Coombs tests were negative, reticulocyte count was 1.2%, and peripheral smear showed normocytic normochromic RBCs, normal WBCs, and adequate platelets with no hemoparasites.

The diagnosis was confirmed as Herpes Zoster Ophthalmicus involving the ophthalmic branch (V1) of the trigeminal nerve. The patient was managed conservatively with antiviral, analgesic, and supportive therapy. Treatment included IV fluids (Inj. NS with multivitamins), Inj. PAN 40 mg IV SOS, Inj. Emeset 4 mg IV SOS, Tab Valtoval 1 g thrice daily, Tab Nervijen NP once daily, Tab Dolo 650 mg SOS, Tab Teczine 10 mg daily, Tab Pan D once daily, Tab Zerodol SP twice daily, Tab Naxodom 500 mg SOS, Calosoft AF lotion applied locally, Acyclovir eye ointment 3% five times daily for 15 days, and Refresh eye drops four times daily for 12 days. Ophthalmology consultation ruled out encephalitis and confirmed ocular involvement.

The patient improved with treatment, was hemodynamically stable, and discharged with oral continuation of antivirals, pain relief, and topical care. He was advised to follow up in the dermatology OPD after 10 days, and in the medicine OPD with repeat LFT and abdominal ultrasound for evaluation of bilirubinemia. This case highlights the clinical features, investigative findings, and effective conservative management of Herpes Zoster Ophthalmicus to prevent serious complications, such as vision loss.

Table 1: baseline data

Date	BP (120/80mm Hg)	PR (72 bpm)	SPO2 (96-100 %)	RR (18-21 bpm)
21/06/2025	120/80	80	98	20
22/06/2025	122/80	72	97	21
23/06/2025	124/80	76	98	20
24/06/2025	122/80	76	98	20

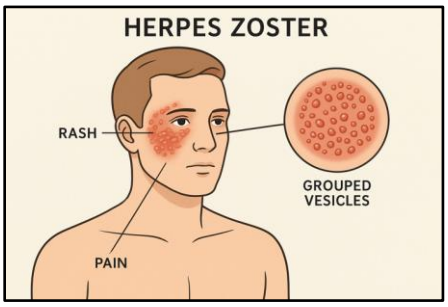

Figure 1: Clinical presentation of herpes zoster with dermatomal rash, pain, and grouped vesicular eruptions.[1]

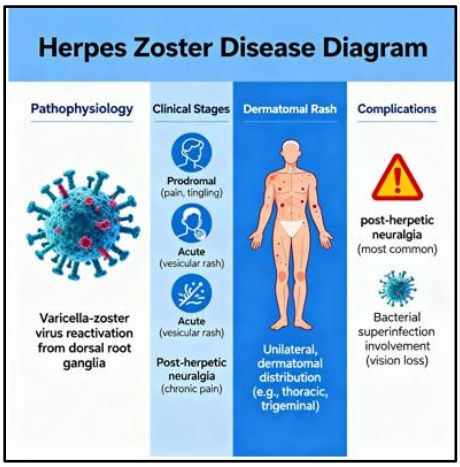

Figure 2: Pathophysiology, clinical stages, dermatomal rash, and complications of herpes zoster.[2]

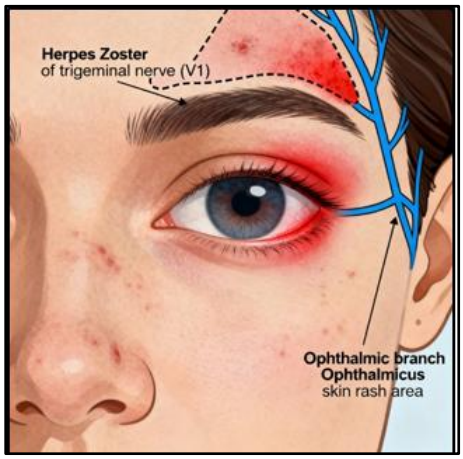

Figure 3: Herpes zoster ophthalmicus involving the ophthalmic branch (V1) of the trigeminal nerve.[5]

3. Discussion

Herpes zoster is a disease caused by the reactivation of the varicella-zoster virus, which also causes chickenpox. It is commonly seen in tertiary care hospitals. The virus stays inactive in the nerve cells after a person has chickenpox and may become active again later in life, leading to herpes zoster. The condition usually presents with fever, pain, burning sensation, and fluid-filled skin lesions along the affected nerve. When the ophthalmic branch of the trigeminal nerve is involved, it may also affect the eye, leading to redness, swelling, blurred vision, and sometimes serious complications if not treated early. The patient in this case was managed with conservative treatment using a combination of intravenous medicines, oral tablets, topical applications, and eye care. IV fluids with multivitamins were given to correct weakness and vitamin deficiency. Inj. PAN 40 mg was used to prevent acid reflux in the stomach, and Inj. Emeset 4 mg was given as an antiemetic to control nausea and vomiting. Antiviral therapy with Tab Valtoval 1 g was the main treatment, as it helps in controlling herpes zoster infection and preventing the spread of lesions. For nerve-related pain, Tab Nervijen NP was prescribed. Pain and fever were managed with Tab Dolo 650 mg (an analgesic and antipyretic) and Tab Zerodol SP (a moderate pain reliever). Tab Naxodom 500 mg was used to prevent migraine-related pain, and Tab Teczine 10 mg was given to reduce allergic symptoms like itching, swelling, and rashes. To prevent excess acid in the stomach, Tab Pan D was also included. For local care, Calosoft AF lotion was applied to soothe the skin, relieve dryness, and reduce irritation caused by the lesions. Eye involvement was managed with Acyclovir eye ointment 3%, which is effective against herpes virus infection in the eye, and Refresh eye drops, which helped reduce irritation, dryness, and burning sensation. Herpes zoster is mainly a clinical diagnosis, especially when the skin lesions are typical and follow a dermatomal pattern. In immunocompromised patients, however, the presentation can be atypical, making diagnosis more difficult.[2,8] It is important to differentiate herpes zoster from herpes simplex virus infection, as both may have similar skin lesions but require different management approaches. The main goals of treatment are to reduce the spread of lesions, control pain, and prevent complications such as postherpetic neuralgia, which is long-lasting nerve pain after the skin lesions heal.[2] Treatment should ideally begin within 72 hours of the onset of symptoms for maximum benefit. In immunocompetent patients with uncomplicated zoster, oral antivirals are sufficient. In immunocompromised patients or complicated cases, aggressive treatment is necessary to prevent severe complications. This case highlights the importance of early diagnosis, correct use of antiviral therapy, and multidisciplinary care involving dermatology and ophthalmology to ensure good recovery and prevent long-term complications.[5]

4. Conclusions

This case of herpes zoster ophthalmicus involving the ophthalmic branch of the trigeminal nerve shows the importance of early recognition and treatment of the disease. The patient presented with fever, pain, and fluid-filled skin lesions on the right side of the face, along with burning and blurred vision in the right eye. Clinical examination and laboratory investigations confirmed the diagnosis. The patient was treated conservatively with antiviral medicines, pain relievers, and eye care, which led to gradual improvement. This case highlights that herpes zoster can affect not only the skin but also the eye, leading to serious problems such as corneal damage or even vision loss if not treated on time. Early antiviral therapy and close monitoring by dermatology and ophthalmology teams are very important to prevent complications. Regular follow-up is also necessary to check for post-herpetic neuralgia, a common long-term complication. Thus, prompt management can lead to good recovery and protect vision.

5. Limitations of case study

This case report has some important limitations. It is based on a single patient, so the findings cannot represent all cases of herpes zoster ophthalmicus. Patients of different ages, health statuses, or weak immunity may show different symptoms and complications. The follow-up period was short, so we could not study long-term effects like post-herpetic neuralgia or permanent eye damage. Advanced tests, such as MRI or PCR for viral confirmation, were not done, which could have given more accurate information about nerve or brain involvement. Also, the treatment was limited to conservative management, so comparison with other treatment options like steroids or newer antiviral drugs was not possible. These factors limit the overall conclusion.

6. Source of Funding

None.

7. Conflict of Interest

None.

8. Acknowledgment

The authors are thankful to the Management and Principal of RR College of Pharmacy. The author also acknowledges the help and support provided by the Sapthagiri Institute of Medical Sciences and Research Centre Hospital. The case patient and his parents.

References

1. Patil A, Goldust M, Wollina U. Herpes zoster: A Review of Clinical Manifestations and Management. *Viruses.* 2022;14(2):192. https://doi:10. 3390/v14020192.
2. Lim D.Z.J. Herpes Zoster and Post-Herpetic Neuralgia—Diagnosis, Treatment, and Vaccination Strategies. *Pathogens.* 2024;13(7):596. https://doi:10.3390/pathogens13070596.
3. Pace E, Accardo G, Lupia T, Brizzi MF, Corcione S, De Rosa FG. Orbital Myositis after Herpes Zoster Ophthalmicus: A Case Report and a Narrative Review of the Literature. *Pathogens.* 2024;13(10):832. https;//doi:10.3390/pathogens13100832
4. Lee JH, Heo HJ, Kim KM, Lee HG, Baek SM, Jung DW. Herpes zoster in the ophthalmic branch of the trigeminal ganglia obscuring cavernous sinus thrombosis due to Streptococcus constellatus subsp. constellatus - A case report- nesth. *Pain Med (Seoul).* 2020;15(2);205–8, https://doi:10.17085/apm.2020.15.2.205.
5. T. Shima et al., "Right-sided Herpes Zoster Ophthalmicus Complicated by Bilateral Third, Fourth, and Sixth Cranial Nerve Palsies and Syndrome of Inappropriate Antidiuretic Hormone Sec. *Inte Med.* 2024;63(19);2673–7. https://doi:10.2169/internalmedicine.2878-23.
6. Afshar FMS. Khalilian M. Pourazizi MA, Najafi. Herpes zoster ophthalmicus with acute retrobulbar optic neuritis and abducens nerve palsy: a case report," *BMC Ophthalmol*. 2025:25(1):23. https://doi:10.1186/s12886-025-03870-x.
7. Mohanty P, Vivekanandh K, Mohapatra L, Dash G, Squamous cell carcinoma in long-standing chromoblastomycosis. *Wolters Kluwer Medknow Pub*. 2017;83(3):363-5. https://doi:10.4103/0378-6323.201339.
8. Hakim W, Sherman R, Rezk T, Pannu K. An Acute Case of Herpes Zoster Ophthalmicus with Ophthalmoplegia, *Case Rep Ophthalmol.* 2012;1–3. https://doi:10.1155/2012/953910.

Cite this article: Das A, Kumar ES. Herpes zoster ophthalmicus of the right eye involving the ophthalmic branch of the trigeminal nerve: A rare case report. *Indian J Pharma Pharmacol*. 2025;12(4):250-253.