

Uncommon presentation of orbital myositis following herpes zoster ophthalmicus

¹Neena Baby, ²Sachin Ajith, ²Priyadarshini Malini, ³Rekha George, ⁴Jayasree Chandrasekharan, ¹Minu George

Departments of ¹Neurology, ²Radiology, ³Ophthalmology, ⁴Dermatology, Renai Medicity Multi Speciality Hospital, Kochi, Kerala, India

Abstract

Herpes zoster ophthalmicus (HZO) is a manifestation of the reactivation of the varicella zoster virus, characterised by vesicular rash along the ophthalmic division of the trigeminal nerve. A rare but significant complication of HZO is orbital myositis, marked by painful swelling of extraocular muscles and periorbital tissues. We present the case of a 72-year-old woman initially presenting with ophthalmological symptoms, notably conjunctival congestion and superficial punctate keratitis, preceding the typical vesicular rash of herpes zoster. The subsequent development of diplopia led to the detection of lateral rectus palsy. Neuroimaging revealed orbital myositis, with the patient showing marked improvement following treatment with oral steroids. This case emphasize the importance of recognizing orbital myositis as a complication of HZO.

Keywords: Herpes zoster ophthalmicus, orbital myositis, lateral rectus palsy, diplopia

INTRODUCTION

Herpes zoster ophthalmicus (HZO) arises due to the reactivation of the dormant varicella zoster virus within the trigeminal ganglia and accounts for a notable fraction, ranging between 10% and 25%, of all herpes zoster cases.^{1,2} Among those affected by HZO, a significant proportion, estimated to be between 20% and 70%, experience ocular and periocular manifestations.³ This condition is typically characterized by a diverse range of ocular symptoms, which may include keratitis, conjunctivitis, along with more severe complications like acute retinal necrosis and optic neuritis.⁴ In the majority of instances, the diagnosis of HZO is straightforward, mainly because the symptoms often emerge several days to weeks following the appearance of a vesicular rash.⁵ However, instances of acute orbital inflammation, manifesting as conditions like optic neuritis, cranial nerve palsies, or orbital myositis, are relatively uncommon.⁶

In this context, we present a case of HZO, distinguished by presence of orbital myositis with distinctive MRI findings. The case is noteworthy for its initial presentation with symptoms indicative of acute orbital inflammation, prior to the development of a vesicular rash.

CASE REPORT

A 72-year-old woman with a history of hypertension, dyslipidemia, and hypothyroidism presented to the ophthalmology clinic with redness in her left eye. During clinical examination, she had signs of conjunctival congestion, superficial punctate keratitis, and lymphadenopathy of left pre-auricular region. She was not exhibiting any skin lesions at that point of time. Her initial treatment included antibiotics, acyclovir eye ointment, and anti-inflammatory medications. However, two days later, she developed skin lesions on the left side of her face, specifically on her cheek and forehead. These lesions were characterised by clusters of multiple grouped vesicles on erythematous base, indicative of HZO. She was prescribed oral valacyclovir, 1 gram three times daily for seven days by the dermatologist. This treatment led to an improvement in her skin lesions.

Three weeks after her initial visit, she returned with new symptoms of headache on the left side, pain in her left eye, photophobia, blurring of vision, and double vision in her left side. A neurology consultation was sought. Upon examination, her skin lesions on the left forehead had healed. Notably, she had limited left eye

Address correspondence to: Dr. Neena Baby, Consultant Neurologist, Department of Neurology, Renai Medicity Multi Super-Speciality Hospital, Kochi, Kerala, India. Email: neenaneuro2018@gmail.com

Date of Submission: 8 March 2024; Date of Acceptance: 21 March 2024

<https://doi.org/10.54029/2024wkp>

abduction accompanied by double vision during left lateral gaze, although her other eye movements remained intact. No other cranial nerve or motor system abnormalities were observed.

Subsequently, a contrast enhanced MRI of the brain and orbits was performed. MRI showed mild thickening and increased signal intensity in the belly of the left lateral rectus (Figure 1) and inferior rectus muscle (Figure 3), along with slight increased enhancement (Figures 2, 3). The brain parenchyma and brainstem appeared normal. Based on these findings, she was prescribed oral prednisolone, starting at 40 mg daily, which was gradually tapered off over a 20-day period.

At her follow-up visit, she showed significant improvement. Her vision had improved, and she no longer experienced double vision. Examination of her eyes revealed no significant abnormalities, and there were no keratic precipitates observed. Additionally, a spectacle correction was performed, resulting in a visual acuity of 6/6 in both eyes with the aid of glasses.

DISCUSSION

HZO is a common presentation in ocular emergencies, resulting from the reactivation of the varicella zoster virus (VZV). This condition typically manifests as a vesicular rash following

the distribution of the ophthalmic division of the trigeminal nerve (V1). Clinically, it begins with ocular discomfort and progresses to more severe symptoms such as chemosis, ptosis, diplopia and visual impairment. Early identification of HZO is critical for effective treatment, aimed at preventing serious complications like postherpetic neuralgia and potential blindness.^{1,4}

Ocular myositis, a rare inflammatory disorder of the extraocular muscles, is seen in two major forms: a milder version with conjunctival injection, and a severe form characterized by pronounced exophthalmos, ptosis, diplopia and chemosis.⁵ Postherpetic neuralgia, the most frequent neurological sequel of HZO, is a chronic pain condition that significantly affects quality of life. Prompt and aggressive antiviral therapy for HZO may reduce the risk of developing postherpetic neuralgia.¹

Symptoms of orbital myositis may be confused with other conditions like infections, orbital cellulitis, orbital neoplasms, optic neuritis, thyroid-associated orbitopathy, cranial nerve palsy, sinus thrombosis, myasthenia gravis, and autoimmune diseases.⁷ Pathogenesis of ophthalmoplegia in herpes zoster can be due to the direct cytopathic effect of the virus or a reactive immunologic response to the virus.

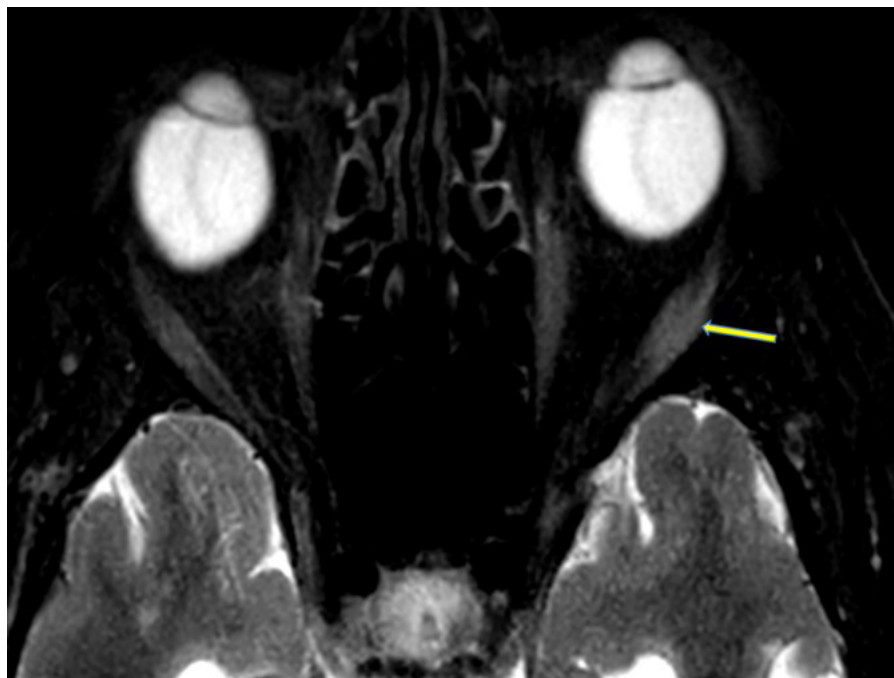


Figure 1. MRI Short Tau Inversion Recovery (STIR) axial image of both orbits showing enlargement and hyperintensity of the left lateral rectus muscle (arrow) with relative sparing of the tendon.

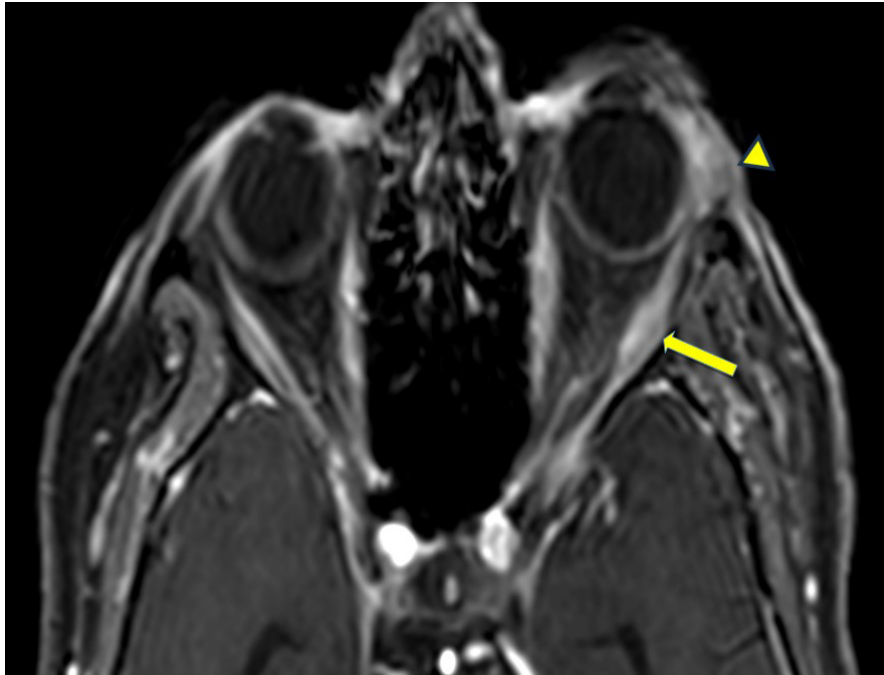


Figure 2. MRI T1 fat saturated postcontrast axial image of both orbits shows thickening and increased enhancement of the left lateral rectus muscle (arrow) along with enhancing pre-septal soft tissue (arrow head).

Other mechanisms include occlusive vasculitis induced by the virus or activation of another latent neuropathic virus by the zoster virus.⁸ In our case, orbital myositis might have occurred

as a reactive immunologic response to virus and has responded well to steroids.

Radiological findings on computed tomography (CT) or magnetic resonance imaging (MRI) in

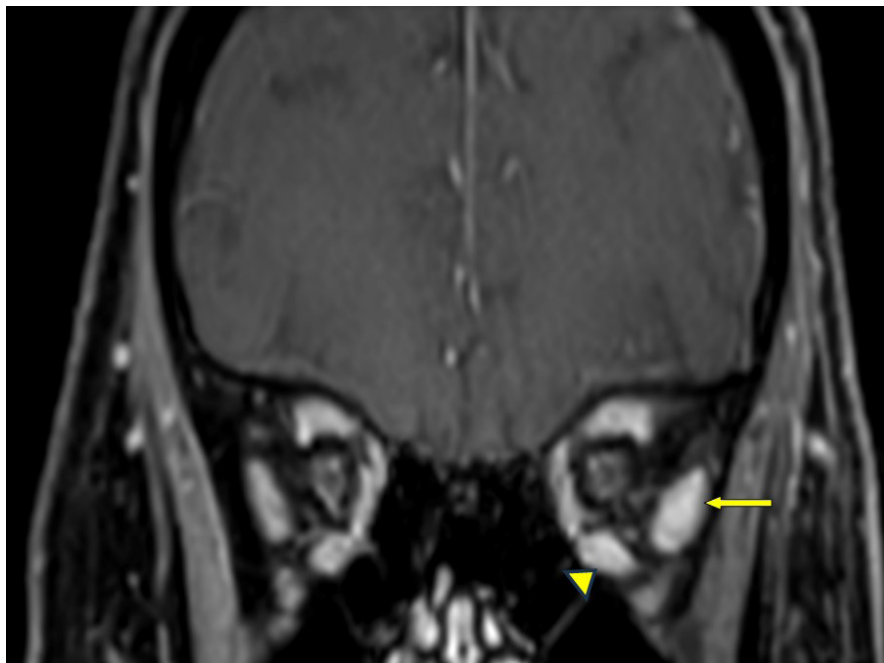


Figure 3. MRI T1 fat saturated post contrast coronal image of both orbits shows thickening and enhancement of the lateral rectus (arrow) and inferior rectus (arrow head) on left side.

herpes zoster-induced myositis include fusiform enlargement of the extra ocular muscles with relative sparing of the tendon and post-contrast enhancement of the muscles, orbital fat, and periorbital soft tissues.^{7,9} Usually, the rectus muscles are the common site of involvement. It is essential to distinguish herpes induced orbital myositis from thyroid eye disease and non-specific orbital inflammation, as tendon involvement in extraocular muscles is common in thyroid eye disease but not in non-specific orbital inflammation. Orbital masses are more frequently associated with non-specific orbital inflammation or other orbital malignancies, rather than with thyroid eye disease.⁹

Antiviral therapy along with corticosteroids usually carries good prognosis especially with normal immunity. Orbital signs improve in over several months. The duration of diplopia can vary from months to years and about 87.5% of cases with diplopia recovered within one year.¹⁰ In our case the patient showed significant improvement in symptoms and she did not have any significant findings on examination during follow-up.

In conclusion, typically, HZO manifests with a vesicular rash before other ophthalmological signs are present. In this case, the patient's condition evolved from initial ophthalmic symptoms to the later appearance of a vesicular rash and the subsequent development of diplopia and lateral rectus palsy. To distinguish HZO-related orbital myositis from other orbital disorders, radiological investigations are necessary. Treatment with antivirals and steroids are crucial in management of HZO associated orbital myositis.

DISCLOSURE

Ethics: Ethics committee approval taken from institutional ethical committee. Approval no RMIEC/RIMS/534 dated 6/11/23. Consent for publication obtained.

Availability of data: Data available on request.

Financial support: None

Conflicts of interest: None

REFERENCES

1. Womack LW, Liesegang TJ. Complications of herpes zoster ophthalmicus. *Arch Ophthalmol* 1983;101(1):42-5. DOI: 10.1001/archophth.1983.01040010044004
2. Bae DW, An JY. Herpes zoster ophthalmicus presenting as acute orbital inflammation preceding the vesicular rash. *J Neurosonol Neuroimag* 2020;12(2):91-4. DOI: <https://doi.org/10.3172/jnn.2020.00090>
3. Tseng YH. Acute orbital myositis heralding herpes zoster ophthalmicus: report of a case. *Acta Neurol Taiwanica* 2008;17(1):47-9.
4. Marsh RJ, Cooper M. Ophthalmic herpes Zoster. *Eye* 1993;7(3):350-70. DOI: 10.1038/eye.1993.74
5. Vardy SJ, Rose GE. Orbital disease in herpes zoster ophthalmicus. *Eye* 1994; 8(Pt 5):577-9. DOI: 10.1038/eye.1994.140
6. Bak E, Kim N, Khwarg SI, Choung HK. Case series: Herpes zoster ophthalmicus with acute orbital inflammation. *Optom Vis Sci* 2018; 95(4):405-10. DOI: 10.1097/OPX.0000000000001204
7. Yuen SJA, Rubin PAD. Idiopathic orbital inflammation: distribution, clinical features, and treatment outcome. *Arch Ophthalmol* 2003;121(4):491-9. DOI: 10.1001/archophth.121.4.491
8. Sanjay S, Chan EW, Gopal L, Hegde SR, Chang BC. Complete unilateral ophthalmoplegia in herpes zoster ophthalmicus. *J Neuroophthalmol* 2009; 29(4):325-37. DOI: 10.1097/WNO.0b013e3181c2d07e
9. Barrio-Barrio J, Sabater AL, Bonet-Farriol E, Velázquez-Villoria Á, Galofré JC. Graves' ophthalmopathy: VISA versus EUGOGO classification, assessment, and management. *J Ophthalmol* 2015;2015:249125. DOI: 10.1155/2015/249125
10. Arda H, Mirza E, Gumus K, Oner A, Karakucuk S, Sirakaya E. Orbital apex syndrome in herpes zoster ophthalmicus. *Case Rep Ophthalmol Med* 2012; 2012:854503. DOI: 10.1155/2012/854503