

Kirkuk Journal of Medical Sciences

CASE REPORT

Simultaneous Onset of Herpes Zoster Ophthalmicus and Chickenpox in the Context of Maternal Varicella; A Rare Case Report

Ali Zedan Alkarawi ©1

¹Department of Dermatology, Azadi Teaching Hospital, Kiruk Health Directorate, Kirkuk, IRAQ. Corresponding author email: azkfderma3@yahoo.com

Received: 20 Novmber 2024 Accepted: 17 March 2025

First published online: 01 April 2024



DOI:10.32894/kjms.2025.155296.1135.

ABSTRACT

Varicella-zoster virus (VZV) is transmitted through airborne respiratory secretions, initially causing chickenpox. After the primary infection, the virus establishes latency within the dorsal sensory ganglia and may later reactivate, resulting in a vesicular dermatomal eruption known as herpes zoster (HZ). The simultaneous occurrence of herpes zoster first and chickenpox later in susceptible individuals is an exceedingly rare phenomenon and is considered an unprecedented event that necessitates further exploration. This report presents a case of an 11-year-old girl diagnosed with herpes zoster ophthalmicus (HZO), affecting the ophthalmic division of the right trigeminal nerve. Three days later, she developed chickenpox. The patient's mother had a documented history of varicella infection during her 32 weeks of gestation. An intriguing aspect of this case is the simultaneous infection of the patient's younger sister, who resided in the same household and also contracted chickenpox at the same time. Early initiation of antiviral and anticonvulsant therapy effectively mitigated the severity of HZ symptoms, while the varicella manifestations in both siblings remained mild, necessitating only symptomatic management with antipruritic lotion. The zoster lesions required approximately six weeks for complete resolution, leaving behind substantial scarring on the forehead, eyelid edema, and scarring alopecia at the affected site.

Key words: Herpes zoster ophthalmicus; Chickenpox; Varicella during pregnancy



licensed under Creative Commons Attribution 4.0 International (CC BY 4.0)

INTRODUCTION

erpes zoster ophthalmicus (HZO), is a reactivation of the varicella-zoster virus (VZV), which lies dormant in sensory ganglia after primary varicella (chickenpox) infection. It's a painful, unilateral, dermatomal vesicular eruption, accompanied by neuropathic pain. (HZO)primarily affects elderly and immunocompromised patients while chickenpox which is a highly contagious disease affects predominantly pediatric age group [1].

Clinical diagnosis of chickenpox is usually straightforward which is a sequential eruption that begins, as small pink macules progress to vesicles and then pustules within 24 hours. Secondary bacterial infection is the most common complication. Transmission occurs through respiratory droplets or direct contact with vesicular fluid. While HZ can act as a source of varicella infection in susceptible contacts, reverse transmission is quite rare. The incubation period of VZV typically ranges from 12 to 20 days [2].

Diagnosis is primarily clinical; however, laboratory confirmation via vesicular fluid culture or direct immunofluorescence may be necessary in unclear cases. Cutaneous complications of varicella include secondary bacterial infections and scarring, while systemic complications are infrequent. Primary varicella infection generally provides lifelong immunity; however, isolated reports of symptomatic reinfection have been documented. Importantly, maternal varicella infection during pregnancy can lead to in utero viral transmission, resulting in latent fetal VZV infection within the sensory ganglia. Such children may later develop HZ without any prior clinical history of varicella [3].

Herpes Zoster ophthalmicus, a severe form of HZ, poses a risk of ocular complications, potentially leading to corneal scarring and vision impairment. Postherpetic neuralgia (PHN) is uncommon in pediatric cases, while it shows increased incidence and severity in older and immunocompromised individuals. PHN is marked by persistent neuropathic pain, with allodynia reported in up to 90% of affected patients [4].

CASE PRESENTATION

In the summer of 2024, an 11-year-old girl presented to a dermatology clinic in Kirkuk, Iraq, with multiple hemorrhagic vesicles on an erythematous base distributed along the ophthalmic division of the right trigeminal nerve. The patient exhibited severe lancinating pain, intermittent jerking episodes, and recurrent syncope. Notably, the right eye was swollen,

and the aperture of the eye was barely open due to significant periorbital edema and erythema. Over the next few days, successive crops of vesicular lesions appeared on the face, neck, trunk, and proximal extremities as shown in Figure 1 A.

The condition was further complicated by pruritus, highgrade fever, generalized myalgia, and malaise, causing considerable distress to the patient's family.

Several golden-yellow crusted lesions were observed on the nearby scalp. The mother remembered contracting chicken-pox late in her pregnancy with her daughter, who now has HZO. The situation worsened as the younger sister also developed chickenpox as shown in Figure 1 B. The family had no history of tuberculosis, diabetes, or HIV.

On examination, the patient exhibited signs of severe pain. revealed a pulse rate of 120 bpm, a temperature of 39.3°C, and blood pressure of 100/65 mmHg. There is erosion induced by ALLodynia Figure 1 C. No Hutchinson's sign (vesicles on the tip or side of the nose). Anemia and lymphadenopathy were absent.

On follow-up, the patient's recovery was protracted, spanning six weeks, and culminating in residual scarring on the forehead and multiple linear patches of scarring alopecia at the affected sites as shown in Figure 1 D. PHN persisted.

On Investigation, the diagnosis was primarily clinical, which was very clear, which made us dispense with performing the PCR test.

The Laboratory Findings are as follows:

· Hemoglobin: 12 g/dL

• White blood cell count: 5000/mm³ (within normal limits)

• Platelet count: 200,000/mm³ (within normal limits)

· Random blood glucose: 6 mmol/L

· Viral screening: Negative for hepatitis and HIV

· Liver function tests:

– ALT: 18 U/L

- AST: 12 U/L

- ALP: 90 U/L (within normal limits)

· Renal function:

- BUN: 20 mg/dL

- Creatinine: 19 mg/dL

· Urinalysis and chest radiography: Unremarkable.

The Management, including patient education and reassurance, were integral to case management.

The therapeutic regimen included:

• Oral acyclovir (800 mg, five times daily for seven days)



Figure 1. (A) Multiple truncal vesicular lesions 8 days after initial HZO presentation. (B) Varicella lesions in the patient's younger sister. (The patient (right) exhibits multiple vesicular and pustular lesions on the face, whereas her younger sister (left) presents with similar varicella lesions on the forehead and arms) (C) Erosion induced by allodynia (PHN). (D) Three weeks after VZV infection (HZO and Chickenpox).

- · Paracetamol (500 mg, as needed)
- · Carbamazepine (100 mg, orally every 12 hours)
- · Oral Augmentin (625 mg, every 12 hours)
- · Topical fusidic acid ointment for bacterial superinfection.6

An ophthalmologic evaluation done by an expert ophthalmologist confirmed the absence of corneal ulceration or other ocular complications. Accordingly, topical ophthalmic drops and ointments were prescribed.

DISCUSSION

The coexistence of varicella and herpes zoster (HZ) has intrigued clinicians and researchers for over a century, with the first documentation by Bokay in 1888 [5]. Since then, there have been reports, such as those by Memarian et al. [6], describing pediatric cases of concurrent HZ and varicella. However, this case stands out as the first known instance where herpes zoster ophthalmicus (HZO) preceded the onset of varicella, making it a unique and noteworthy presentation.

The sequence of events in this case is hypothesized to stem from a primary maternal varicella infection during pregnancy, specifically at 32 weeks of gestation. This likely led to transplacental viral transmission, resulting in the establishment of latent varicella-zoster virus (VZV) within the fetal dorsal root ganglia. Over time, this latent virus reactivated, initially manifesting as HZO. Subsequently, extensive viral replication caused secondary chickenpox [7]. While VZV latency and reactivation are well-documented processes, the sequential presentation observed here is highly unusual and raises intriguing questions about the underlying mechanisms. It suggests that unique host immune responses or viral factors may have played a role, warranting further investigation into this atypical progression.

One of the significant clinical observations in this case was the absence of Hutchinson's sign, a well-recognized predictor of ocular complications in HZO. This finding reduced the likelihood of severe ocular morbidity [8], which is often a major concern in such cases. However, despite being an immuno-

competent child, the patient experienced persistent neuropathic pain and delayed lesion healing. PHN is rare in children but underscores the potential for substantial morbidity even in pediatric populations [9].

When compared to existing literature, this case diverges from previously reported instances of concurrent VZV infections. For example, simultaneous VZV reactivation alongside other infections or conditions—such as scrub typhus or autoimmune disorders—has been documented but typically involves adults or immunocompromised individuals [10]. In contrast, this case involved a child with no known immunodeficiency, emphasizing the importance of recognizing atypical presentations even in seemingly low-risk populations.

From a pathophysiological perspective, VZV reactivation is generally believed to result from diminished cell-mediated immunity that allows viral replication and spread from the sensory ganglia [11]. In this case, transplacental transmission during fetal development may have established a unique latency pattern that predisposed the patient to sequential reactivation. This aligns with existing evidence suggesting that VZV can remain dormant within the sensory ganglia for extended periods before reactivating under specific triggers, such as stress or immune suppression [12, 13].

Given the atypical nature of this presentation and the persistence of symptoms like neuropathic pain and delayed healing, meticulous follow-up is crucial for managing potential complications. Early initiation of antiviral therapy may help mitigate issues such as PHN and improve overall outcomes [14]. Furthermore, this case highlights the need for further research into the mechanisms underlying sequential VZV manifestations and potential predictors for such occurrences.

In conclusion, this case not only adds to our understanding of VZV reactivation but also underscores the importance of recognizing unusual presentations to ensure timely diagnosis and management. It serves as a reminder that even in pediatric patients with no apparent risk factors, VZV can present in unexpected ways that challenge conventional assumptions about its behavior.

CONCLUSION

There may be a vertical transmission of the Varicella Zoster virus, leading to sequential Herpes Zoster Ophthalmicus and varicella. The findings underscore the need for increased clinical vigilance in the diagnosis and management of these cases to prevent long-term sequelae.

ETHICAL DECLARATIONS

· Acknowledgements

None.

· Ethics Approval and Consent to Participate

Ethical approval for this case study was approved by Azadi Teaching Hospital - Kirkuk Directotrate.

· Consent for Publication

The parent of the patient provided written consent for the publication of this case report and any accompanying images.

· Competing Interests

The author declares that there is no conflict of interest.

Funding

Self funded.

· Authors' Contributions

Ali Zedan Alkarawi was responsible for the literature review, and writing the manuscript. The author read and approved the final version of the manuscript.

REFERENCES

- [1] Kennedy PG, Gershon AA. Clinical features of varicellazoster virus infection. Viruses 2018;10(11):609. https: //doi.org/10.3390/v10110609.
- [2] Hakim FE, Riaz K, Farooq A. Pediatric herpes zoster ophthalmicus: a systematic review. Graefe's Archive for Clinical and Experimental Ophthalmology 2023;261(8):2169–2179. https://doi.org/10.1007/s00417-023-06033-0.
- [3] De Paschale M, Clerici P. Microbiology laboratory and the management of mother-child varicella-zoster virus infection. World Journal of Virology 2016;5(3):97. https://doi.org/10.5501/wjv.v5.i3.97.
- [4] Koshy E, Mengting L, Kumar H, Jianbo W. Epidemiology, treatment and prevention of herpes zoster: A comprehensive review. Indian Journal of Dermatology, Venereology and Leprology 2018;84:251. https://doi.org/10.4103/ijdvl.IJDVL_1021_16.
- [5] FETTER F, SCHNABEL TG. CONCURRENT HERPES

- ZOSTER AND CHICKENPOX: Report of a Case. Archives of Internal Medicine 1949;83(5):502-504. https://doi. org/10.1001/ARCHINTE.1949.00220340027002.
- [6] Memarian S, Khodabandeh M, Gharib B. The Very Rare Concurrency of Herpes Zoster and Varicella in a 4-Year-Old Boy. Journal of Pediatric Perspectives 2020;8(6):11425-11428. https://doi.org/10.22038/ ijp.2020.46220.3765.
- [7] Mandelbrot L. Fetal varicella-diagnosis, management, and outcome. Prenatal diagnosis 2012;32(6):511-518. https://doi.org/10.1002/pd.3843.
- [8] Shukla I, Tiwari S, Billore O. Herpes zoster ophthalmicus followed by varicella in a young adult. Indian Journal of Ophthalmology 1973;21(3):131-133.
- [9] Forbes HJ, Bhaskaran K, Thomas SL, Smeeth L, Clayton T, Mansfield K, et al. Quantification of risk factors for postherpetic neuralgia in herpes zoster patients: a cohort study. Neurology 2016;87(1):94-102. https: //doi.org/10.1212/WNL.0000000000002808.
- [10] Kakoullis L, Parperis K, Psarelis S, Papachristodoulou E, Panos G. AB0647 CATASTROPHIC ANTIPHOSPHO-LIPID SYNDROME TRIGGERED BY CONCURRENT VZV

- AND HSV INFECTION. A CASE REPORT. Annals of the Rheumatic Diseases 2021;80:1356-1357. https://doi. org/10.1136/ANNRHEUMDIS-2021-EULAR.470.
- [11] Gilden D, Nagel MA, Cohrs RJ, Mahalingam R. The variegate neurological manifestations of varicella zoster virus infection. Current neurology and neuroscience reports 2013;13:1-10. https://doi.org/10.1007/ s11910-013-0374-z.
- [12] Kennedy PG, Cohrs RJ. Varicella-zoster virus human ganglionic latency: a current summary. Journal of neurovirology 2010;16(6):411-418. https://doi.org/ 10.1007/BF03210846.
- [13] Sorel O, Messaoudi I. Varicella virus-host interactions during latency and reactivation: lessons from simian varicella virus. Frontiers in microbiology 2018;9:3170. https://doi.org/10.3389/fmicb.2018.03170.
- [14] Pica F, Gatti A, Divizia M, Lazzari M, Ciotti M, Sabato One-year follow-up of patients with long-lasting post-herpetic neuralgia. BMC Infectious Diseases 2014;14:1-9. https://doi.org/10.1186/ s12879-014-0556-6.