INDONESIAN HEALTH JOURNAL
https://ihj.rivierapublishing.id/index.php/rp
Volume 4 Number 2 June 2025
P-ISSN: 2963-3044
E-ISSN: 2963-3036

Disseminated Herpes Zoster Subsequent to Ocular Shingles in an Immunocompetent Young Man Following Facial Trauma: A Case Report

Grady[1*], Dewa Ayu Putu Mitha Paramitha Rahayu[1], Putu Dyah Ayu Saraswati[2]
Medical Doctor Intern at Wangaya General Hospital Denpasar, Bali, Indonesia[1]
Departement of Dermatology and Venerology Diseases at Wangaya General Hospital Denpasar, Bali, Indonesia[2]
Mithaparamitha185@gmail.com

Received: June 2025 Accepted: June 2025 Published: June 2025

ABSTACK

Herpes zoster (HZ) results from the reactivation of latent varicella-zoster virus (VZV), typically triggered by aging or immunosuppression. Herpes zoster ophthalmicus (HZO) affects the ophthalmic branch of the trigeminal nerve (V1), posing a risk of ocular complications. Disseminated herpes zoster (DHZ), a rare form of HZ, is defined by ≥20 vesicular lesions beyond the primary and adjacent dermatomes, potentially leading to systemic complications and increased morbidity. Case Presentation: A 36-year-old immunocompetent male presented with disseminated vesicular eruptions across the body, sparing the knees and lower legs, four days after the initial appearance of vesicles along the left ophthalmic (V1) branch of the trigeminal nerve. Notably, the patient had a history of minor trauma at the same site one week prior. A comprehensive evaluation and multidisciplinary management were undertaken, involving ophthalmologists, dermatologists, and neurologists. By the third day of treatment, his pain had significantly subsided, no new lesions had developed, and was discharged. Conclusion: DHZ can occur in immunocompetent individuals at any age, but is more common in older patients. A meticulous history and physical examination are essential, as dermatomal herpes zoster may progress to dissemination. Additionally, the potential link between recent trauma and zoster reactivation may play a contributory role, highlighting the importance of early recognition for prompt intervention.
Keywords: Herpes zoster, Disseminated herpes zoster, Immunocompetent, Herpes zoster ophthalmicus, Trauma

Coresponden: Author*
Email: email@gmail.com

INTRODUCTION

The varicella-zoster virus (VZV) is a human-specific, double-stranded DNA virus belonging to the Herpesviridae family. It primarily causes varicella (chickenpox) and is predominantly transmitted through respiratory secretions. Following initial replication in lymphoid tissues, the virus disseminates via viremia to various organs, including the liver, spleen, and epidermis, ultimately leading to the characteristic vesicular skin eruptions. After resolving within one to two weeks, the virus remains latent in sensory ganglia, maintained by cell-mediated immunity. Herpes zoster (HZ), or shingles, is caused by the reactivation of latent VZV, presenting as a painful localized, vesicular rash in dermatomal distribution, mainly due to relative immunosuppression and advanced age (Rudinsky & Jordan, 2017). A specific subtype, herpes zoster ophthalmicus (HZO), occurs when VZV reactivates in the ophthalmic division (V1) of the trigeminal nerve, potentially leading to severe ocular complications (Litt et al., 2024). Disseminated herpes zoster (DHZ) is an atypical and rare manifestation, characterized by the presence of more than 20 vesicular lesions beyond the primary dermatome, often affecting multiple noncontiguous dermatomes.

The incidence of HZ varies between 5.2 and 10.9 cases per 1000 person-years, with a significant age-related increase. The incidence rate of HZO in the general population was reported as 0.31–0.35 per 1000 person-years. The incidence rate of DHZ in the general

population was reported as 0.04 per 1000 person-years with DHZ predominantly affecting the trigeminal nerve (Giannelos et al., 2024). At Sanglah Hospital Denpasar (2019–2021), 75 HZ cases were recorded, including three DHZ cases with HIV (Stephanie et al., 2022). The diagnosis of HZ is primarily clinical, based on characteristic dermatological findings and patient history. Laboratory confirmation is typically reserved for atypical or complicated cases (Albrecht et al., 2020). The general goals of treatment for HZ are to limit the intensity and duration of HZ-associated symptoms, promote the healing of lesions, decrease the duration and severity of acute neuritis, and improve health-related quality of life.

This case report discusses a rare presentation of DHZ subsequent HZO in an immunocompetent 36-year-old male, following facial trauma, highlighting its clinical course, diagnostic challenges, and potential triggers despite an intact immune system.

Case Report

A 36-year-old Javanese male, was consulted from the Emergency Department to the Dermatology and Venereology Department at Wangaya General Hospital, Denpasar, with widespread vesicular eruptions distributed symmetrically across the body, sparing the knees and lower legs. Upon history-taking, the patient reported that the vesicular lesions first appeared on the left forehead and above the left eyelid four days prior, preceded by a severe headache and intermittent sharp pain around the left eye, which he described as a stabbing sensation, without associated numbness or tingling. He also recalled a minor wound at the same site, which had been treated with topical antibiotics, following a pinch from his child one week prior. As of today, the lesions subsequently spread to the left frontal scalp, chest, abdomen, back, both arms and thigh as per today. Additionally, he experienced left eye redness with a foreign body sensation, itchiness, and swelling on the left side of the face, especially around the left eye, that makes it hard to open his eyes and fever for the past three days. Yesterday, the patient had visited the emergency department with similar complaints and was diagnosed with blepharoconjunctivitis secondary to HZO by an ophthalmologist. He was prescribed a couple eye drops, eye ointment, painkillers, vitamin C, and Acyclovir, after which he was discharged from the hospital.

On physical examination, the patient was conscious, appearing moderately ill, and fully alert, with a Glasgow Coma Scale (GCS) score of E4V5M6. Vital signs indicated elevated blood pressure (144/96 mmHg), fever (40.2°C), with a Visual Analog Scale (VAS) pain score of 7. General assessment revealed left periorbital edema and conjunctival and pericorneal vascular injection. Cranial nerve paralysis was negative. Cardiovascular, respiratory, and abdominal evaluation was unremarkable. The extremities were warm, with no edema or lymphadenopathy. Dermatological examination identified clusters of vesicles on an erythematous base distributed along the V1 branch of the left trigeminal nerve, affecting the left forehead, the area above the left eyebrow, and the left frontal scalp. The lesions measured approximately 0.5 × 0.5 cm in diameter, with some vesicles having ruptured, resulting in yellowish crust formation, accompanied by swelling on the left side of the face. Hutchinson signs were negative. Additionally, multiple vesicles were symmetrically scattered across the chest, abdomen, back, arms, and thighs. Laboratory reports showed a normal erythrocyte, hematocrit, platelets, and leukocytes count with slightly lower neutrophils 44.4% (50–70), and slightly higher lymphocytes 41.7% (20–40), monocytes 10.9% (2–8). The Provider-Initiated Testing and Counseling (PITC) results were negative.

Based on clinical presentation and laboratory findings, the patient was diagnosed with blepharoconjunctivitis secondary to HZO and DHZ. The primary attending physician during the patient's hospitalization was a neurologist, who prescribed oral paracetamol 1000 mg twice

daily, intravenous ketorolac 30 mg every 12 hours and gabapentin 100 mg once daily, along with intravenous methylprednisolone 125 mg twice daily as the patient experienced severe, intolerable pain, particularly on the forehead and around the eyes. Pain management was adjusted according to the patient's pain level. Moreover, the patient received intravenous omeprazole 40 mg once daily and vitamin B complex twice daily as supportive therapy. This case was then consulted to dermatologist and neurologist. The dermatology team recommended continuing oral acyclovir 400 mg, two tablets, five times daily, as previously prescribed upon the patient's discharge from the emergency department. This regimen was to be maintained for a total of ten days. A thin layer of acyclovir ointment was also advised to be applied over the vesicular lesions twice daily. Ophthalmologist prescribed artificial eye drops (Cendo Lyteers®), to be administered one drop every hour, along with corticosteroids eye drops (P-Pred ED®) and antibiotic eye drops (LFX ED®), each given one drop every three hours, and Timolol 0.5% eye drop twice daily. Gentamicin ointment was advised for application three times a day around swollen eye. The treatment plan also included an eye cleansing procedure using 0.9% NaCl solution twice daily and intravenous fluid maintenance.

After three days of hospitalization, fever had declined, nearly all vesicular lesions had ruptured, dried, and formed brownish crusts, with no new lesion development. The periorbital swelling had subsided, allowing the patient to open his eyes more comfortably, and ocular discharge had significantly decreased. Patient also reported tolerable pain levels on the left side of his head. Given these improvements, the patient was deemed clinically stable and was therefore discharged from the hospital. The prescribed ophthalmic treatment, including three types of eyes drops and an eye ointment, was continued at home. Antiviral therapy was maintained at the same dosage and duration as initially planned, along with the continued application of acyclovir ointment to the affected areas. For pain management, the patient was provided with ibuprofen 400 mg twice daily and gabapentin 300 mg once daily, along with omeprazole 20 mg twice daily as adjunctive therapy. The patient was scheduled for a follow-up at the ophthalmology clinic in three days for further assessment.

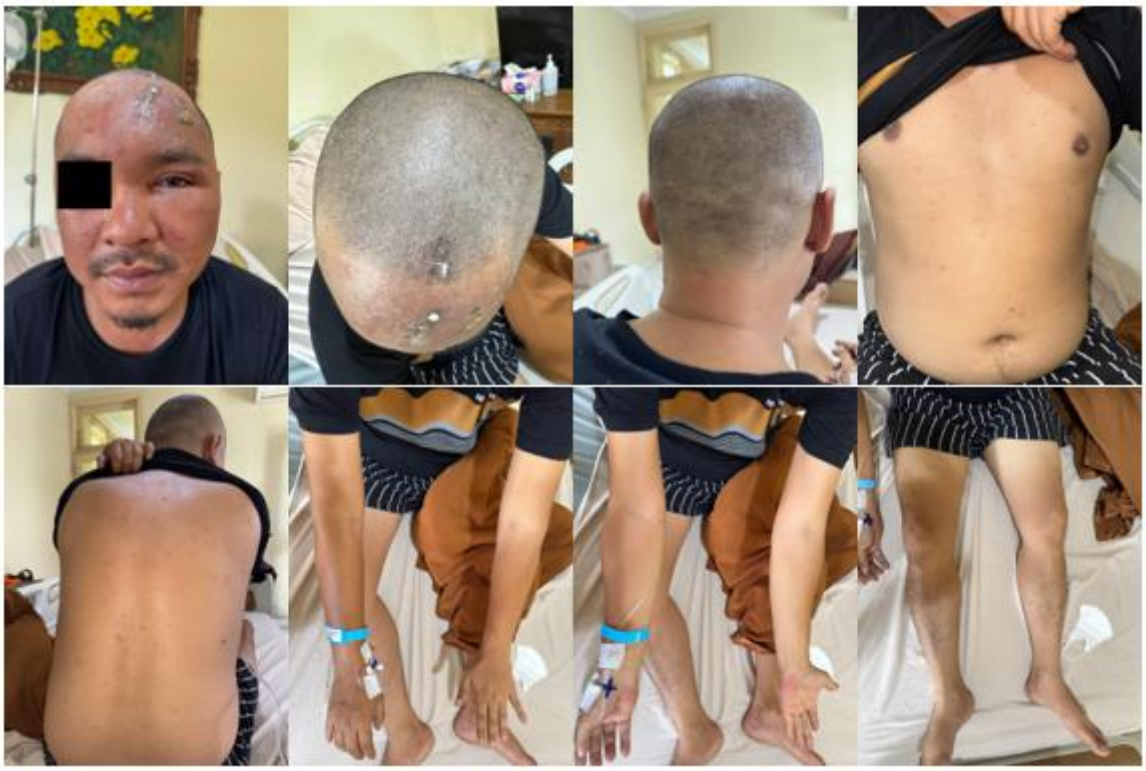

Figure 1. A cluster of vesicles on an erythematous base, partially covered by yellowish crusts, was observed on the left forehead and frontal scalp

METHOD

This study employed a qualitative case report design to explore the rare occurrence of disseminated herpes zoster (DHZ) subsequent to ocular shingles in an immunocompetent young man following facial trauma. The research focused on a single case, allowing for an in-depth analysis of clinical presentation, diagnostic challenges, and therapeutic outcomes. The data population consisted of medical records, laboratory results, and clinical observations from the patient's hospitalization at Wangaya General Hospital, Denpasar. The data sample was purposively selected, centering on the 36-year-old male patient who met the diagnostic criteria for DHZ and HZO, with no underlying immunodeficiency.

The research instruments included standardized clinical assessment tools such as the Visual Analog Scale (VAS) for pain, Glasgow Coma Scale (GCS), and dermatological examination protocols. Laboratory tests, including complete blood counts and HIV screening (PITC), were used to rule out immunosuppression. Validity was ensured through multidisciplinary consensus among dermatologists, neurologists, and ophthalmologists, while reliability was maintained by adhering to established diagnostic guidelines for HZ and DHZ. Data collection techniques involved retrospective review of patient records, clinical photographs, and real-time monitoring during hospitalization. The procedure included history-taking, physical examination, diagnostic confirmation, and documentation of treatment responses over a three-day hospitalization period.

Data analysis was conducted using descriptive and thematic techniques to identify patterns in clinical progression, treatment efficacy, and potential triggers (e.g., trauma). No specialized software was required due to the qualitative nature of the study, but findings were cross-referenced with literature to contextualize the case. The analysis highlighted the temporal association between trauma and VZV

reactivation, the role of early antiviral therapy, and the patient's response to multidisciplinary management. This approach provided a comprehensive framework for understanding atypical DHZ presentations in immunocompetent individuals, contributing to clinical awareness and diagnostic protocols.

RESULTS AND DISCUSSION

Reactivation of latent VZV leads to HZ when host immunity against VZV-specific antigens declines, commonly associated with aging, physical or psychological stress, systemic disease, malignancy, transplantation, drugs, HIV, or in some cases, trauma (Alhayaza et al., 2022). The acute eruptive stage of HZ is characterized by painful vesicles that rupture, ulcerate, and form crusts. This phase is typically preceded by the pre-eruptive stage, presenting as burning pain in the affected dermatome, often accompanied by headache, malaise, and photophobia before the rash appears. DHZ is uncommon in immunocompetent individuals, with an incidence of only 2%, whereas it occurs in approximately 15–30% of immunocompromised patients. Notably, DHZ tends to manifest at a younger age in immunocompromised individuals compared to those who are immunocompetent (Patil et al., 2022).

VZV viremia appears to occur in all individuals with HZ, regardless of T-cell immune status or clinical signs of dissemination. Kronenberg et al. successfully detected VZV DNA in serum samples from all tested immunocompetent patients with localized HZ. These findings suggest that the potential for disease dissemination exists in all HZ patients, with those experiencing greater deficiencies in cell-mediated immunity being at a higher risk of developing DHZ (Bollea-Garlatti et al., 2017).

In the study by Bollea et al., 95% of reported DHZ cases initially presented with involvement of a specific dermatome before extending to adjacent dermatomes or progressing to a disseminated form. Similarly, Gomez et al. reported that in 89% of reviewed cases, patients first exhibited localized HZ affecting one or multiple dermatomes, followed by the development of a secondary diffuse papulovesicular rash. Notably, the initial involvement of cranial nerves was most frequently observed, suggesting a potential predisposition of these patients to cutaneous dissemination. The initial rash most frequently involved cranial nerve dermatomes (42%), followed by thoracic (27%), lumbosacral (19%), and cervical (15%) regions, with cutaneous dissemination occurring within 5 days (range: 1–12 days).

This case is particularly noteworthy as it demonstrates DHZ in a relatively young immunocompetent male. Consistent with the aforementioned literature, our patient also experienced a classic pre-eruptive syndrome before developing painful vesicles in the ophthalmic branch (V1) of the trigeminal nerve, which subsequently disseminated across the body after four days. Additionally, a prior history of trauma was also an influencing factor in this case, as the patient had experienced minor trauma—a pinch from his child—at the site where the initial lesion developed. Trauma as a potential trigger for varicella-zoster virus (VZV) reactivation has been documented in previous studies, with research indicating that zoster patients were 3.4 times more likely than controls to have experienced trauma in the week before rash onset. While trauma is a recognized risk factor for dermatomal zoster, its role in disseminated disease remains uncertain (Häfelfinger et al., 2020).

Available diagnostic methods for HZ include the Tzanck smear, polymerase chain reaction (PCR), direct fluorescent antibody (DFA) testing, and viral culture. However, laboratory confirmation is generally unnecessary, as many tests are time-consuming, have limited specificity, or are not widely accessible outside of research settings. The definite diagnosis in this case was made primarily on clinical grounds, without additional confirmatory tests. Despite an otherwise unremarkable physical examination, the extensive cutaneous involvement necessitated inpatient evaluation and screening for

potential immunodeficiency. In this case, an HIV screening test (PITC) was conducted to assess any underlying immune compromise.

Oral acyclovir has long been the cornerstone of HZ treatment; however, its poor bioavailability and frequent dosing requirement led to the development of second-generation antiviral agents, such as valacyclovir and famciclovir. Antiviral therapy is most effective when started within 72 hours of rash onset and is typically given for seven days. In high-risk immunocompromised patients, treatment may still be beneficial beyond this window. However, early antiviral use does not significantly shorten pain duration, which can persist for weeks to years, affecting quality of life (Uchida et al., 2017). For DHZ, intravenous acyclovir (10 mg/kg every eight hours) is recommended, followed by oral antivirals once lesions improve. Treatment usually lasts 10–14 days, with hospitalization and infection control measures necessary in severe cases. Studies in immunocompromised patients have shown that the primary risk factor for VZV dissemination is the delay between symptom onset and the initiation of treatment, with a longer delay correlating with a higher risk of dissemination (Drone & Ganti, 2019). To the best of our knowledge, no similar studies have been conducted in immunocompetent patients. While antiviral therapy helps alleviate pain in acute neuritis, HZ-related pain can still be severe, requiring additional interventions. Pain management should be individualized, incorporating standardized pain assessments, and regular monitoring to optimize efficacy and minimize side effects. NSAIDs and acetaminophen are commonly used for mild to moderate pain, while severe cases may require opioids, corticosteroids, or gabapentin, considering the patient's comorbidities and pain severity (Albrecht et al., 2014).

In this case, the patient was prescribed oral acyclovir within 72 hours of dermatomal vesicular onset. Despite progression DHZ, intravenous acyclovir was not administered due to its limited availability in many hospitals across Indonesia. During hospitalization, the patient received a combination of oral and intravenous analgesics prescribed by a neurologist, with dosage adjustments based on pain severity. Meanwhile, all ocular symptoms were managed by an ophthalmologist. The mortality and morbidity risks in immunocompetent DHZ patients remain low (Chakraborty et al., 2020).

CONCLUSION

DHZ can occur in immunocompetent individuals of any age, although it is more commonly observed in older patients. The statistically significant age difference between immunocompetent and immunocompromised patients with DHZ suggests that age itself may be a risk factor for disease dissemination. This case underscores the importance of thorough history-taking and physical examination in patients with HZ, as its clinical course may evolve, as seen in this patient, where HZO progressed to DHZ. In patients with DHZ, underlying immunosuppression must always be considered and ruled out. The association between zoster reactivation and trauma may confound the diagnosis, underscoring the need for early recognition to ensure timely treatment. While trauma has been identified as a risk factor for dermatomal zoster, its role in predisposing individuals to disseminated disease remains unclear.

REFERENCES

Albrecht, M. A., Hirsch, M. S., & Thorner, A. R. (2014). Treatment of herpes zoster in the immunocompetent host. *Up-to-Date, Basow, DS, Editor, Waltham, MA*.

Albrecht, M. A., Levin, M. J., Hirsch, M., & Mitty, J. (2020). Epidemiology, clinical manifestations, and diagnosis of herpes zoster. *UpToDate. Waltham, MA: UpToDate*.

Alhayaza, G., Al-Omair, A., & Almohanna, H. M. (2022). Varicella-zoster virus encephalitis in an immunocompetent adult with disseminated cutaneous herpes zoster after testosterone booster supplements: case report. *Case Reports in Dermatology*, *14*(2), 164–168.

Bollea-Garlatti, M. L., Bollea-Garlatti, L. A., Vacas, A. S., Torre, A. C., Kowalczuk, A. M., Galimberti, R. L., & Ferreyro, B. L. (2017). Clinical characteristics and outcomes in a population with disseminated herpes zoster: a retrospective cohort study. *Actas Dermo-Sifiliográficas (English Edition)*, *108*(2), 145–152.

Chakraborty, U., Chandra, A., Sil, A., & Biswas, S. K. (2020). Elderly immunocompetent man presenting with disseminated cutaneous herpes zoster. *BMJ Case Reports*, *13*(8), e237480.

Drone, E., & Ganti, L. (2019). A case of disseminated zoster in an immunocompetent patient. *Cureus*, *11*(12).

Giannelos, N., Curran, D., Nguyen, C., Kagia, C., Vroom, N., & Vroling, H. (2024). The incidence of herpes zoster complications: A systematic literature review. *Infectious Diseases and Therapy*, *13*(7), 1461–1486.

Häfelfinger, R., Burgener, A.-V., Osthoff, M., & Potlukova, E. (2020). Simultaneous VZV and HSV-1 reactivation after minor head injury. *European Journal of Case Reports in Internal Medicine*, *7*(12), 1746.

Litt, J., Cunningham, A. L., Arnalich-Montiel, F., & Parikh, R. (2024). Herpes zoster ophthalmicus: presentation, complications, treatment, and prevention. *Infectious Diseases and Therapy*, *13*(7), 1439–1459.

Patil, A., Goldust, M., & Wollina, U. (2022). Herpes zoster: a review of clinical manifestations and management. *Viruses*, *14*(2), 192.

Rudinsky, D. M., & Jordan, K. (2017). Disseminated herpes zoster causing acute respiratory distress syndrome in an immunocompetent patient. *Case Reports*, *2017*, bcr-2017.

Stephanie, A., Indira, I. E., Laksmi, H., & Pratama, N. (2022). Disseminated Herpes Zoster in Elderly with Human Immunodeficiency Virus (HIV) Infection: A Case Report. *Bioscientia Medicina: Journal of Biomedicine and Translational Research*, *6*(11), 2373–2376.

Uchida, K., Teske, N., & Christensen, J. (2017). Disseminated zoster after trauma in an immunocompetent patient. *The American Journal of Medicine*, *130*(12), e539–e540.