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CASE REPORT

Herpes Zoster in a 9-Month-Old Infant Following Maternal Varicella Infection: A Rare Case Report

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Abstract: Herpes zoster is caused by reactivation of latent varicella-zoster virus (VZV), typically occurring in older adults or immunocompromised individuals, and is rarely seen in infants. We report a rare case of herpes zoster in a 9-month-old infant, likely due to intrauterine exposure to VZV. The patient presented with dermatomally distributed vesicular lesions on the left anterior chest and back. Treatment included intravenous acyclovir, oral antiviral therapy, semiconductor laser therapy, boric acid compresses, and traditional Chinese herbal fumigation.

Keywords: herpes zoster, infant, intrauterine infection, medicine, traditional Chinese, varicella-zoster virus

Introduction

Herpes zoster (HZ), commonly known as shingles, is caused by reactivation of latent varicella-zoster virus (VZV). VZV is an ubiquitous, double-stranded DNA virus which belongs to the subfamily of human alpha herpes virus, and, like other herpes viruses, it causes both primary and recurrent infection and remains latent in neurons present in sensory ganglia.[1] After a primary VZV infection (varicella/chickenpox), the virus establishes lifelong latency in sensory ganglia and may later reactivate as HZ. HZ typically progresses in three clinical stages. Two to three days prior to the onset of the rash, patients typically exhibit prodromal symptoms, including fatigue, headache, low-grade fever, and abnormal skin sensations such as itching burning, and prickling.[2] During the active phase, HZ manifests at the affected dermatome with the appearance of rash accompanied by pain.[3] This condition frequently results in a deterioration of the patient's quality of life. Approximately 20% of patients continue to experience pain during the chronic phase after the rash has resolved, a condition known as post herpetic neuralgia.[4] Antiviral therapy remains the cornerstone of treatment, and early initiation can reduce disease duration and the risk of chronic pain. Reactivation of latent VZV is primarily associated with waning VZV-specific cell-mediated immunity.[2] Epidemiological studies suggest that age-related immune senescence, psychological stress, mechanical trauma, and immunosuppression are possible triggers for viral reactivation.[2] However, most zoster cases occur in individuals without overt immunosuppression, and the precise determinants of VZV reactivation remain unclear. Reactivation in immunocompetent infants is exceedingly rare due to the presence of maternally transferred antibodies and immature immune system.

Infantile HZ accounts for only a very small proportion of HZ cases globally, and reported incidence rates are extremely low compared with adults.[5] In infants, reactivation is most commonly associated with intrauterine exposure to maternal varicella infection. Maternal varicella during pregnancy may result in congenital varicella syndrome or lead to fetal VZV latency without clinical symptoms at birth.[6] As maternal antibodies wane during the first months of life and infants' cell-mediated immunity is still developing, viral reactivation may occur even without immunodeficiency. Despite the clinical significance, research on infantile HZ remains limited. Existing reports focus primarily on clinical presentation and short-term outcomes, while information on treatment variation, particularly non-pharmacological adjunctive therapies, is scarce.[5] Documenting rare cases such as infantile HZ not only enhances understanding of atypical reactivation mechanisms but may also guide early diagnosis and optimize treatment decisions. We present an uncommon

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case of HZ in a 9-month-old infant with presumed intrauterine VZV exposure. This report adds to the limited literature on infantile HZ, highlights diagnostic considerations, and describes a multidisciplinary treatment approach.

Case Description

A 9-month-and-14-day-old female infant presented with a 4-day history of vesicular rash on the left chest and back. The lesions began as millet-sized vesicles on the forehead, neck, and left chest without an identifiable trigger (see Figure 1). Initial treatment at home with calamine lotion was ineffective. The rash progressed to involve the left posterior thorax, prompting a visit to a local clinic, where external traditional Chinese medicine applications and rectal suppositories were administered, with no improvement. The infant became increasingly irritable but remained afebrile with stable appetite, normal urination and defecation, and only slightly disturbed sleep.

There was no significant past medical history. The mother reported a history of varicella infection at 4.5 months of gestation. The infant had no postnatal exposure to varicella, no history of G6PD deficiency, epilepsy, encephalitis, congenital heart disease, or other chronic conditions. Immunizations were up to date per local schedule.

On admission: Temperature 36.7°C, pulse 118 bpm, respiratory rate 26 bpm, weight 8.5 kg. Cardiopulmonary and abdominal examinations were unremarkable. Dermatologic exam revealed scattered tense vesicles on the forehead and neck. The left anterior chest and back showed clusters of vesicles from millet- to mung bean-sized, distributed in a dermatomal pattern with some areas of exudation. No enlarged lymph nodes were palpated.

Differential diagnoses included: impetigo (excluded due to clear dermatomal pattern and absence of honey-colored crusts), eczema herpeticum (excluded based on unilateral distribution and maternal VZV infection history), and contact dermatitis (less likely due to vesicles following a dermatome rather than exposure distribution).

Laboratory results revealed leukocyte differentials consistent with viral infection (decreased neutrophil count and elevated lymphocyte and monocyte ratios) and mild elevations in AST and cardiac enzymes (CK-MB, LDH, α-HBDH) were noted. These findings supported the clinical suspicion of VZV reactivation.

The diagnosis of HZ was clinically established based on: unilateral dermatomal vesicular distribution, progression typical of HZ lesions, maternal varicella infection during pregnancy (suggesting intrauterine exposure) and laboratory results.

Treatment

The patient was initially started on oral acyclovir 0.1 g three times daily, along with topical acyclovir cream and fusidic acid cream. Acyclovir inhibits viral DNA polymerase and viral replication, and topical fusidic acid prevents secondary bacterial infection. In addition, semiconductor laser therapy was applied locally twice daily for 10 minutes. The laser

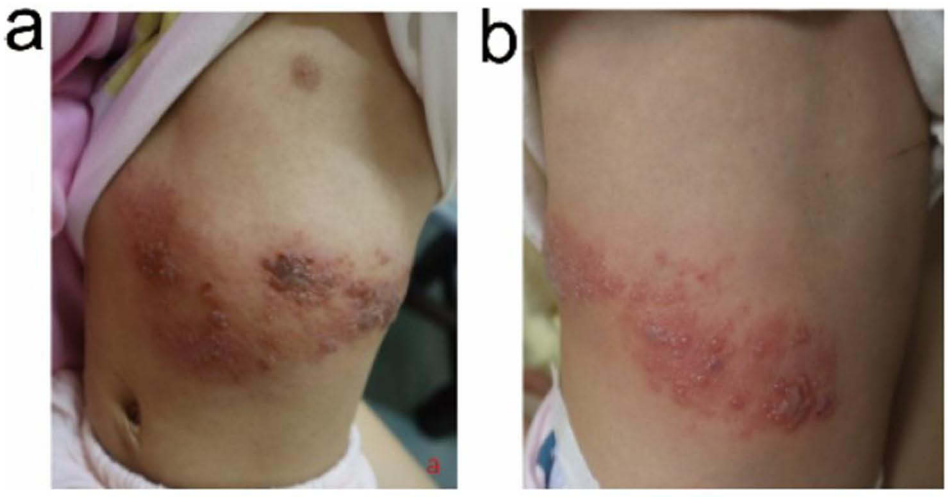

Figure 1 Clinical presentation before treatment. (**a**) millet-sized, tense vesicles with a dermatomal distribution on the left anterior chest; (**b**) similar grouped erythematous vesicles on the left posterior back in a characteristic unilateral band-like pattern.

therapy was used to reduce inflammation, promote local circulation, and alleviate discomfort at the lesion site. Wet compress therapy using 3% boric acid was applied twice daily to promote astringency and reduce erythema.

Traditional Chinese medicine fumigation therapy was also administered using a decoction containing honeysuckle (15 g), wild chrysanthemum (20 g), Senecio scandens (20 g), rhubarb (20 g), gardenia (20 g), and forsythia (20 g). The herbal mixture was decocted and applied externally once daily. These herbs are traditionally recognized for their anti-inflammatory and antimicrobial properties, and were used as an adjunct therapy to support lesion drying and healing.[7,8]

On day 2 of hospitalization, due to the appearance of new vesicular lesions and onset of fever (up to 39°C), the treatment regimen was intensified. Oral acyclovir was replaced with intravenous acyclovir 0.1 g every 8 hours. Antiviral oral solution (5 mL, twice daily) was continued. Wet compresses and laser therapy were maintained. The lesions began to dry and crust by day 5, and no new eruptions appeared (see Figure 2). The fever resolved with symptomatic treatment. The patient showed significant improvement within five days, with complete resolution over two weeks. At the two-week follow-up, the vesicles on the posterior back had completely resolved, leaving only faint residual erythema, as shown in Figure 3.

Discussion

HZ in infants is rare because most infants possess residual maternal VZV antibodies and have limited exposure to wild-type virus. The incidence of HZ in children varies considerably across countries and study methodologies. Recent epidemiologic data indicate that HZ in children typically occurs at a median age of 8–12 years, with only a small fraction of cases seen in infants.[9] Large retrospective studies reported a median onset age of 9.9 years, even after varicella vaccination became universal.[10] In contrast, infantile HZ is usually associated with intrauterine exposure, rather than postnatal infection.[11]

In this case, the infant's mother developed varicella during mid-pregnancy, supporting the hypothesis of transplacental transmission with latent fetal infection. Maternal VZV-specific antibodies typically protect the infant for the first months of life; however, as these antibodies decline around 6 months and the infant's own cellular immunity remains immature, viral reactivation may occur even in otherwise healthy infants.[12,13]

A comparison of infantile HZ cases (Table 1) reveals three main patterns of viral acquisition: intrauterine exposure (prenatal infection), neonatal varicella infection (postnatal household exposure), and reactivation of vaccine-strain VZV after vaccination. Our patient fits the first mechanism: intrauterine VZV exposure with postnatal reactivation, and vaccination was not yet due.

Clinical misdiagnosis is common because HZ in infants may initially resemble eczema herpeticum, impetigo, or contact dermatitis. The unilateral dermatomal pattern, vesicular morphology, and maternal infection history supported the diagnosis. Laboratory viral profile was consistent with a viral etiology; polymerase chain reaction (PCR) was discussed but declined by parents, which we acknowledge as a limitation.[19] Early antiviral treatment is recommended to shorten disease duration and

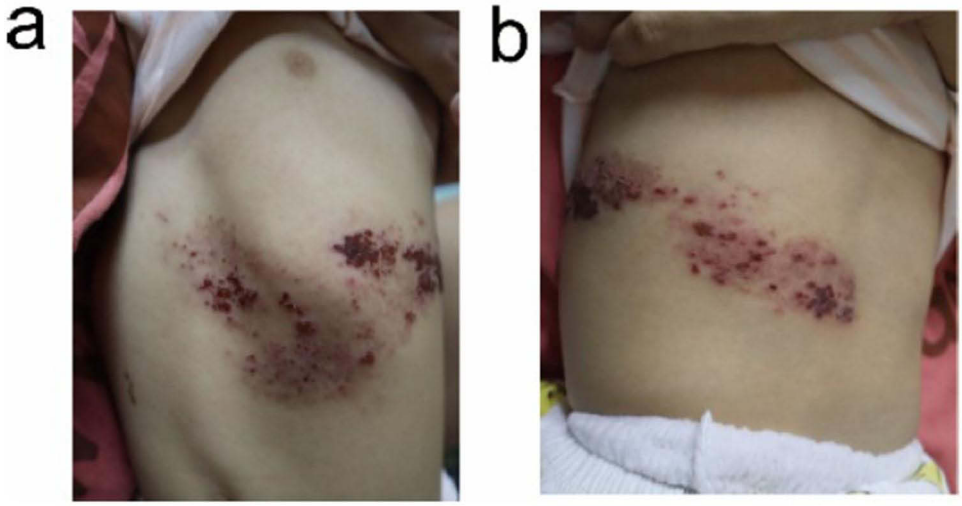

Figure 2 Clinical improvement after 5 days of treatment. (**a**) resolution of the vesicles on the anterior chest; (**b**) resolution of the vesicles on posterior back.

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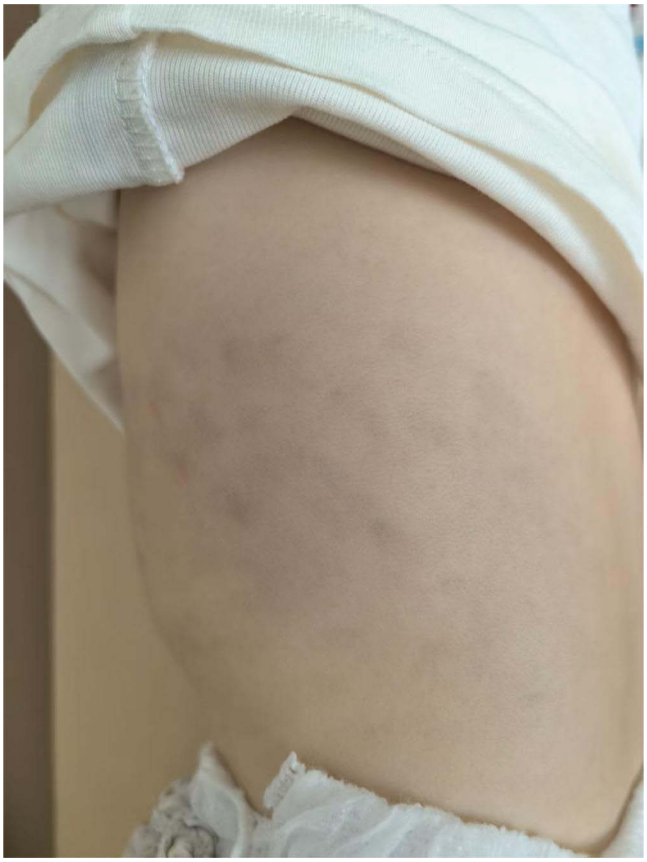

Figure 3 Clinical improvement after 14 days of treatment, showing near-complete resolution of the vesicles on the posterior back.

reduce the risk of complications.[5] In our patient, oral acyclovir was initiated and later changed to intravenous acyclovir when new lesions appeared. Adjunctive therapies included: semiconductor low-level laser therapy, which has been shown to promote wound healing, and relieve neuropathic pain in pediatric dermatologic conditions;[7,20] boric acid wet compresses, which provide local astringent and anti-exudative effects;[21] and traditional Chinese medicine fumigation, using anti-inflammatory and antimicrobial herbal extracts as complementary care.[7,22]

The infant demonstrated rapid clinical improvement, with crusting by day 5 and complete resolution within two weeks, similar to outcomes described in other pediatric HZ case series. Gupta et al reported that immunocompetent pediatric patients generally show a short disease course and favorable prognosis.[23]

Table 1 Summary of Previously Reported Infantile Herpes Zoster Cases

Author, Years	Age at HZ Onset	Varicella Vaccination Status	Maternal/Postnatal VZV Exposure	Dermatome Involved	Diagnostic Method	Treatment
Wang YB et al, 2024[14]	3 months	Fully vaccinated according to schedule; varicella vaccine not administered yet	No	Right craniofacial region	Clinical and serology	Oral acyclovir; laser therapy
Shameem Y et al, 2025[15]	25 days	Not stated	Postnatal exposure from infected sibling	Scalp, behind the ear and upper chest	PCR	IV acyclovir, IV flucloxacillin, and discharged on oral acyclovir

(*Continued*)

Table 1 (Continued).

Author, Years	Age at HZ Onset	Varicella Vaccination Status	Maternal/Postnatal VZV Exposure	Dermatome Involved	Diagnostic Method	Treatment
Koide T et al, 2022[16]	14 months	Received varicella (Oka strain) vaccine at 12 months; administered simultaneously with measles and rubella and mumps vaccines.	No	Right upper limb and right dorsal region	PCR	No antiviral treatment, acyclovir was considered but not given because lesions began resolving
Costa RG et al, 2025[17]	6 months	Fully vaccinated according to the national schedule; but not including varicella vaccine	Postnatal exposure from infected sibling	Right upper limb and posterior shoulder	Clinical diagnosis based on dermatomal pattern and history of neonatal varicella; evaluated via images by pediatric infectious disease team	Supportive care only (no acyclovir due to >72 h since onset, good clinical status)
Deguchi E et al, 2011[18]	8 weeks	Not stated	Maternal varicella at 7 weeks gestation; no postnatal varicella	Left upper extremity (fingers, palm, forearm)	Immunostaining using anti-VZV monoclonal antibody	Oral valacyclovir
This case	9 months	Fully vaccinated according to schedule; varicella vaccine not administered yet	Maternal varicella at 4.5 months gestation	Left thoracic	Clinical, dermatomal pattern, and virological profile	IV acyclovir, laser, boric acid, and traditional Chinese medicine fumigation

Abbreviations: PCR, polymerase chain reaction; IV, intravenous injection.

The infant's parents expressed relief at learning that HZ can occur even without postnatal varicella exposure and appreciated early antiviral treatment. This case underscores that intrauterine VZV exposure should be considered in infants presenting with dermatomal vesicles, even if postnatal exposure is absent.

This case has several limitations. First, virologic confirmation (PCR or viral culture) was not performed due to parental refusal of invasive sampling; diagnosis was therefore clinical. Second, the efficacy of adjunctive treatments (laser therapy and traditional medicine fumigation) cannot be generalized, as controlled evidence in infants remains limited. Lastly, as a single case, the findings cannot be extrapolated to broader populations.

Conclusion

In summary, this case reinforces that infantile HZ can develop in immunocompetent, fully vaccinated infants when intrauterine VZV exposure has occurred. Awareness of maternal infection history and careful assessment of dermatomal vesicular eruptions are key to early diagnosis. While adjunctive treatments such as semiconductor laser therapy and traditional Chinese medicine appeared beneficial in symptom relief, conclusions should be drawn cautiously, as this report represents a single case. Further research is needed to establish diagnostic guidance for infantile HZ and to evaluate the safety and efficacy of complementary therapies in pediatric populations.

Ethic Statement

The patient's legal guardians have granted permission for the images to be published together with the case report. The Fifth People's Hospital of Hainan Province approved the disclosure of the case details after review and approval by its Ethics Committee.

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Disclosure

The authors report no conflicts of interest in this work.

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