THE BLISTERING TRUTH: A SURPRISING CASE OF CHILDHOOD HERPES ZOSTER

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Summary

*The **Varicella-zoster** virus is a neurotropic human herpes virus belonging to the genus **alpha herpesviridiae.** The virus is responsible for primary infection resulting in varicella and herpes zoster representing a reactivation of latent infection. It shows a worldwide distribution. While most frequently seen in older adults, herpes zoster can also develop in otherwise healthy children and adolescents. It is widely accepted that early childhood chickenpox infection may be linked to the occurrence of herpes zoster in immunocompetent children. In this article we present a rare case of pediatric herpes zoster in a 7-year-old girl who had a confirmed history of primary varicella infection during infancy, at one year of age. Although herpes zoster is uncommon in the pediatric population, this case illustrates a characteristic clinical course in an otherwise healthy child and underscores the importance of an interdisciplinary approach involving dermatology in collaboration with family medicine, pediatrics, and infectious diseases specialists.*

Key words: *"childhood herpes zoster", "herpes zoster in immunocompetent children", "pediatric herpes zoster", "early childhood varicella".*

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Introduction

The *Varicella-zoster* virus (VZV) or human herpes virus 3 is the causative agent for both chickenpox/varicella and shingles/*Herpes zoster* (HZ). HZ represents a reactivation of VZV in the host and has gained interest because of variable clinical presentation, which is important in the differential diagnosis of diseases, especially since HZ complications may potentially be life-threatening. Treatment options and prevention by vaccination are of clinical importance. The literature related to HZ continues to evolve, especially in regard to patients with comorbidities and immunocompromised patients [1,2].

Varicella is transmitted through inhalation of respiratory droplets or direct contact with fluid from its characteristic vesicular lesions, making it one of the most highly contagious human diseases. After entering the body, the virus first replicates in the respiratory tract and then spreads to nearby lymph nodes. This is followed by viremia, which leads to the appearance of the classic vesicular skin eruptions. These skin lesions typically show multiple stages at once, ranging from newly formed vesicles to crusted lesions and, in some cases, residual scarring [3,4].

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The incubation period for varicella ranges from 10 to 21 days. Individuals are contagious starting 1 to 4 days before the appearance of the skin rash and remain so until all vesicular lesions have completely dried [5]. When varicella occurs during pregnancy, the virus can cross the placenta and infect the fetus. Such fetal infections may result in severe, potentially life-threatening disseminated disease. Maternal vaccination helps protect the fetus from these risks [6,7].

Following primary infection, VZV establishes lifelong latency in neural tissues. The virus has been identified in the dorsal root ganglia, cranial nerve ganglia, autonomic ganglia within the enteric nervous system, and in astrocytes. Cell-mediated immunity plays the central role in maintaining VZV latency, and reactivation is believed to occur when this immune surveillance declines [8].

When VZV reactivates, it begins replicating within neuronal cell bodies. The newly formed viral particles then travel along the nerve fibers to the corresponding dermatome. In this region, the virus triggers inflammation and the formation of vesicular lesions. The pain associated with HZ results from inflammation of the nerves infected by VZV. Unlike varicella, HZ does not endanger a developing fetus because maternal antibodies that cross the placenta provide protection [9].

Herpes zoster occurs globally and shows no seasonal pattern in its incidence [10]. Major risk factors include age over 50, immunosuppression, certain infections, and psychological stress. Although most frequently seen in older adults, HZ can also develop in otherwise healthy children and adolescents [10]. It is widely accepted that early childhood chickenpox infection is linked to the occurrence of HZ in immunocompetent children [10,11]. Because the immune system is still developing in early childhood, the relatively weak specific immune response – particularly cell-mediated immunity – is thought to be the primary reason HZ occurs in pediatric patients [12].

Clinical manifestations of HZ progress through three stages: pre-eruptive, acute exudative, and chronic. The pre-eruptive phase is marked by burning or pain in the affected dermatome beginning at least two days before skin lesions appear. Patients may also experience extra-cutaneous symptoms such as headache, malaise, and sensitivity to light [13].

During the acute eruptive stage, clusters of painful, umbilicated vesicles develop. These lesions often rupture, ulcerate, and eventually crust over. This phase is the most contagious. Pain is typically intense and may not respond to nonsteroidal analgesics. The acute phase lasts about 2–4 weeks, although discomfort may persist beyond this period [13].

Chronic HZ is defined by persistent pain lasting more than four weeks. Patients may experience dysesthesias and paresthesias. This pain can be debilitating and may continue for several months [14].

In most cases, diagnosis is based on clinical evaluation. However, the variable and sometimes atypical presentation of HZ can make diagnosis challenging in certain patients [14, 15, 16].

Material and Methods

We report a pediatric case of HZ in a 7-year-old girl with a documented history of varicella infection in early childhood. This case provides insight into the clinical manifestations and management considerations of HZ in the pediatric population.

Additionally, we queried the PubMed database to identify further information regarding previously published case reports of pediatric HZ, using keywords such as ‘childhood herpes zoster,’ ‘herpes zoster in immunocompetent children,’ and ‘pediatric herpes zoster.”

Case presentation

A 7-year-old girl presented with a distinctive dermatologic eruption consistent with herpes zoster, characterized by grouped vesicles on an erythematous base which formed well-defined, polycyclic plaques localized to the left L1 dermatome. The onset of symptoms was accompanied by intense pain and a burning sensation, signaling the initial phase of the condition. The prodromal phase lasted several days, marked by significant pain in the left lumbar region and upper thigh. This was soon

followed by the development of erythematous papules that rapidly progressed to vesicular lesions. The lesions were restricted to the left L1 dermatome, which is consistent with the involvement of a single spinal nerve (Figure 1). The patient had a confirmed history of primary varicella infection during infancy, at one year of age. No systemic signs of infection, such as fever or malaise, were observed. Laboratory investigations revealed mild lymphocytosis and a mild increase in the erythrocyte sedimentation rate (ESR), which are common findings in viral infections. No other significant abnormalities were noted. With respect to therapeutic management, the patient was promptly started on antiviral treatment with oral acyclovir at a dosage of 20 mg/kg per dose, administered four times daily (every six hours) for seven days. In addition to the antiviral therapy, nonsteroidal anti-inflammatory drugs (NSAIDs) were prescribed to alleviate pain and reduce the inflammation associated with the condition.

At the 7-day follow-up, the patient presented with significant improvement in the clinical appearance of the rash. The vesicular lesions had started to dry and crust, leaving behind post-inflammatory hyperpigmentation (Figure 2).

By the 14-day follow-up, the post-inflammatory hyperpigmentation had notably diminished, and the patient exhibited a favorable clinical progression towards complete resolution (Figure 3). Notably, the patient did not develop postherpetic neuralgia (PHN), a complication commonly associated with herpes zoster in older adults but only infrequently observed in the pediatric population.

Discussions

In immunocompetent children, HZ is quite uncommon. Early childhood varicella has proven to be the primary risk factor for pediatric HZ in otherwise healthy children [17]. Pediatric HZ may also be the result of varicella vaccination, as demonstrated by Plachouri KM in a case report from 2019 about an 11-year-old child who developed HZ after two doses of the VZV vaccination [18]. In a retrospective study from 2019, Gündoğdu M. et al. assessed the annual pattern and clinical aspects of HZ in immunocompetent children. The study encomprised 69 immunocompetent pediatric patients aged between six months and seventeen years old. Regarding the annual distribution of the disease,

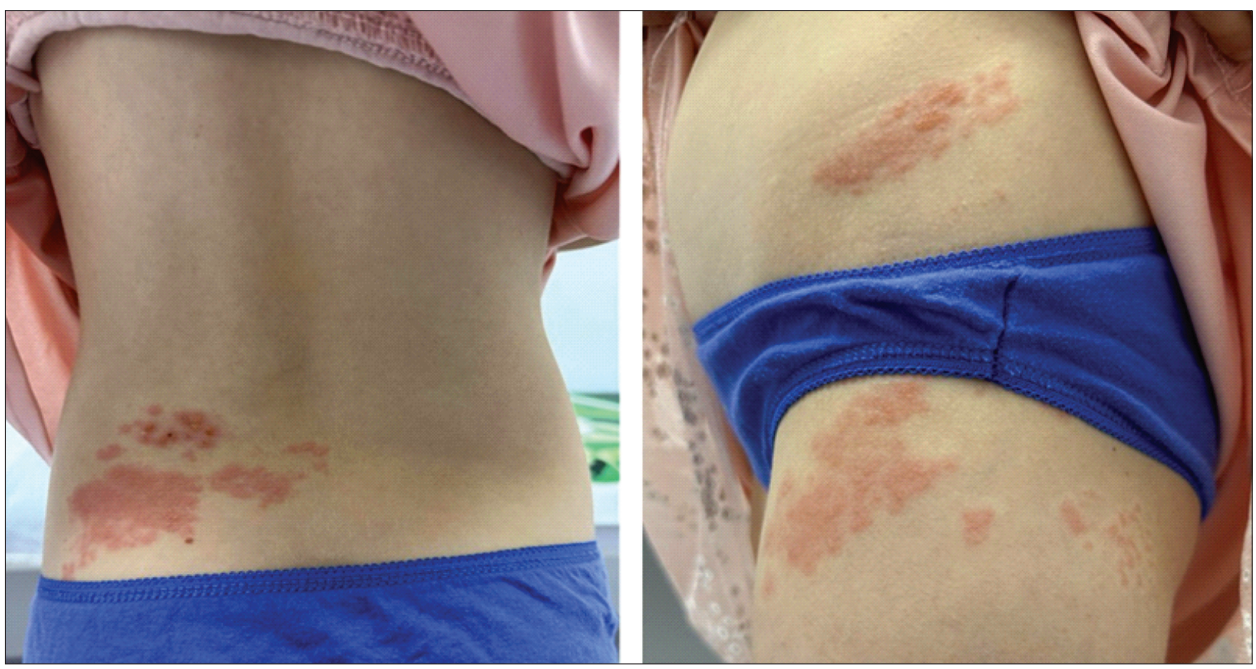

Figure 1. Well-defined, polycyclic, erythematous plaques covered by grouped vesicles localized to the left L1 dermatome.

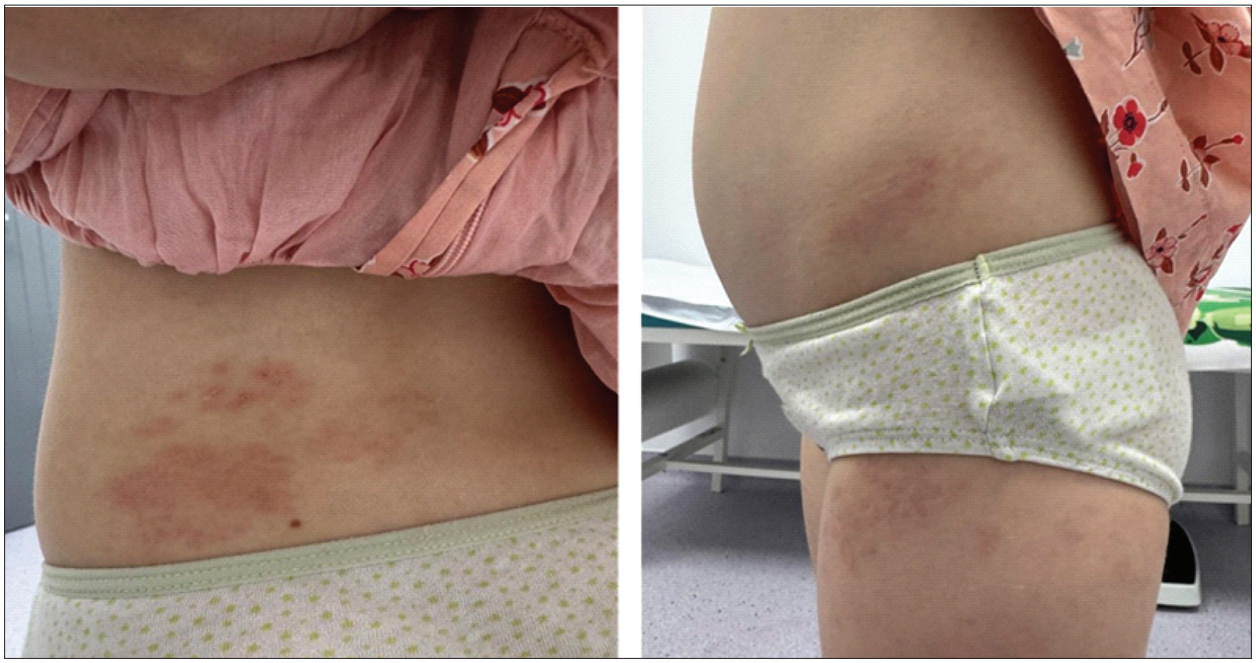

Figure 2. Significant improvement may be noted with vesicular lesions starting to dry and crust, leaving behind post-inflammatory hyperpigmentation.

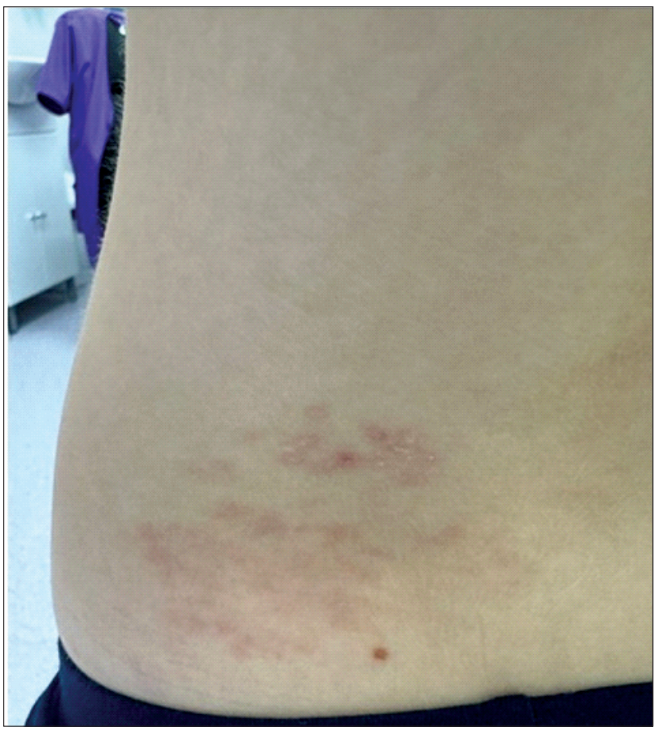

Figure 3. The post-inflammatory hyperpigmentation had notably diminished, and the patient exhibited a favorable clinical progression towards complete resolution.

two peaks of HZ were highlighted – in March and September, respectively, with no cases reported in August. Concerning the clinical aspects of pediatric HZ, the most common site of involvement was the thoracic dermatome [19].

HZ is an infrequent cause of dermatologic manifestations in the pediatric population and may raise concern for an underlying immunodeficiency. In a recent publication from 2025 by Zhang S et al, the authors studied manuscripts addressing HZ in children [20]. In most instances, the diagnosis was established based on characteristic clinical findings in conjunction with epidemiological information. HZ is known to be associated with defects in T-cell–mediated immunity, which may be secondary to infections such as HIV, tuberculosis, or other pathogens, as well as to conditions including diabetes, malnutrition, malignancy, or primary immunodeficiency, as shown in Table 1 [20].

The authors found key clinical indicators suggestive of an underlying immune disorder

Table 1. Conditions Associated With Herpes Zoster and Defects in T-Cell–Mediated Immunity

• Human Immunodeficiency Virus (HIV) infection
• Tuberculosis
• Diabetes
• Malnutrition
• Malignancy
• Primary immunodeficiency

which included recurrent HZ within a short timeframe; disseminated disease; the appearance of new lesions more than one week after the initial presentation; a prolonged course despite appropriate antiviral therapy; a history of recurrent, invasive, or unusually persistent infections caused by other pathogens; and a family history of immunodeficiency or consanguinity [20]. In children who have received the VZV vaccine, vaccine-strain HZ should be considered, as well. The authors recommend that most children with HZ should be evaluated based on their clinical history and standard laboratory tests. Nevertheless, if any worrisome clinical features or abnormal test results were to be identified, referral to an experienced specialist is recommended [20].

Although uncommon, HZ in the pediatric population may exhibit a multisegmental pattern of cutaneous involvement [21]. Multisegmental HZ, also referred to as multidermatomal HZ, is defined by the involvement of two or more dermatomes in a characteristic zosteriform distribution [22, 23, 24]. This clinical manifestation is predominantly reported in immunocompromised or partially immunocompromised patients, reflecting impaired cell-mediated immunity, and is only rarely described in the pediatric population [25]. Elsaie M et al reported the case of a 6-year-old immunocompetent girl with a medical history of early childhood varicella who developed a widespread HZ eruption on the right side of the body and on the abdomen, further highlighting the multifaceted clinical presentation of HZ in both children and adults [21].

In this case report, our patient exhibited a characteristic clinical presentation of HZ, beginning with intense, burning pain localized to the left L1 dermatome, followed by the development of a cutaneous eruption consisting of grouped vesicles on an erythematous base that coalesced into well-demarcated, polycyclic plaques. The patient had a confirmed history of primary varicella infection during infancy, at one year of age. Laboratory evaluation demonstrated mild lymphocytosis and a slight elevation in the erythrocyte sedimentation rate (ESR), findings frequently associated with viral infections. No additional significant abnormalities were identified. Following the initiation of antiviral therapy, the patient demonstrated a favorable clinical response with subsequent complete disease resolution.

Conclusions

Herpes zoster, also known as shingles, is a reactivation of the varicella-zoster virus, which remains dormant in the dorsal root ganglia after an initial varicella infection. The condition is rare in healthy pediatric populations, with most cases occurring in immunocompromised individuals.

In this case, the patient had a documented history of varicella infection during early childhood, which constitutes a significant predisposing factor for varicella-zoster virus reactivation in pediatric populations. Furthermore, the localized dermatomal distribution of the cutaneous eruption was consistent with the characteristic clinical presentation of herpes zoster. Early initiation of antiviral therapy, such as acyclovir, has been demonstrated to reduce symptom severity and duration, while also decreasing the likelihood of complications, including post-herpetic neuralgia.

This case highlights a typical clinical coursed of herpes zoster in an otherwise healthy child. The patient exhibited a favorable therapeutic response, with significant improvement observed within one week and complete resolution by the two-week follow-up. The absence of postherpetic neuralgia further emphasizes the overall favorable prognosis.

Although herpes zoster is uncommon in the pediatric population, this case illustrates a characteristic clinical course in an otherwise healthy child and underscores the importance of an interdisciplinary approach involving dermatology in collaboration with family medicine, pediatrics, and infectious diseases specialists.

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Conflict of interest
NONE DECLARED

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