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**CONTRASTING CLINICAL MANIFESTATIONS OF HERPES ZOSTER IN
IMMUNOCOMPETENT CHILDREN: TWO CASE REPORTS**

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ABSTRACT

Background: Herpes zoster (HZ) is uncommon in pediatric population. Severe disseminated forms like Herpes Zoster Multiplex (HZM)—involving non-contiguous dermatomes—are rare but can occur in immunocompetent pediatric patients, despite HZ usually being benign. **Objective:** We report two contrasting clinical presentations of HZ in immunocompetent pediatric patients to highlight its clinical variability and the critical need for individualized therapeutic strategies. **Cases:** Case 1 describes a 9-year-old girl with a history of varicella presenting with classic HZ on the right lower lip. She experienced a mild course and recovered with supportive topical treatment. Case 2 describes an 8-year-old girl with no known varicella history presenting with severe HZM involving trigeminal, cervical, and thoracic dermatomes. **Case Management:** She required hospitalization for oral acyclovir, intravenous corticosteroids, and antibiotics. The condition resolved without lethal complications, resulting only in keloid sequelae. Both patients demonstrated non-anemic macrocytosis. **Conclusion:** Recognizing the clinical variability of HZ in pediatric patients is essential. Differentiating severe disseminated forms like HZM from classic HZ is critical, as HZM necessitates aggressive systemic therapy and hospitalization, compared to the outpatient supportive care often sufficient for classical presentations.

Keywords: Acyclovir; Herpes Zoster Multiplex; Immunocompetent; Oral Medicine; Trigeminal

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INTRODUCTION

Varicella-zoster virus (VZV) as one of subfamilies of Human herpes virus infects human skin and primary infection is known as chicken pox, followed dormant state within the sensory ganglia. Its later reactivation causes Herpes Zoster (HZ).^{1,2} Classical herpes zoster involves a single unilateral dermatome. Involvement of more than 2 non-contiguous dermatomes is termed herpes zoster multiplex (HZM).³

The incidence of HZ is higher among geriatric, particularly individuals in their 50s, 60s, and 70s, with rate of occurrence is 5 to 10 per 1,000 people.^{1,2} Pediatric incidence of 0.014 per 1,000 and incidence of Herpes Zoster Multiplex accounted for less than 0.1% of cases in major herpes zoster series. Severe HZ and sequelae occur even in immunocompetent pediatric patients making such rare clinical presentations critical to document and analyze.⁴

The risk factors that predispose to pediatric HZ including immune disorders particularly defects in T-cell-mediated immunity. These include congenital immunodeficiency and acquired conditions, such as malignancies, infectious diseases, diabetes, and malnutrition, as well as those associated with immunosuppressive therapy.^{5,6} In the immunocompetent pediatric population, HZ infections may still unfold with significant clinical severity and adverse sequelae.^{1,7} Typically, HZ diagnosis is confirmed predominantly on observational clinical findings.^{1,4,7}

Despite the recognized occurrence of severe HZ in healthy pediatric patients, there remains a paucity of literature regarding the extreme clinical variability of the disease within this specific demographic. The current literature largely lacks comparative discussions on risk stratification, the specific clinical thresholds which necessitate hospitalization, and the differential management required for localized versus disseminated forms in immunocompetent pediatric patients. To address this knowledge deficit, this article aims to report two contrasting cases in immunocompetent pediatric patients; one presenting with classical HZ of the mandibular branch of the

trigeminal nerve, and the second case with severe HZM. By comparing these cases, we aim to highlight the unpredictable disease severity of pediatric HZ and emphasize the critical need for individualized therapeutic strategies.

CASE MANAGEMENT

CASE REPORT 1

Patient Information

A 9-year-old girl presented with a burning sensation, edema, and crusting on her lower right lip that had persisted for the past 5 days, with blisters having appeared two days after the initial onset. She had a history of varicella (chickenpox) 3 years prior. There were no previous similar symptoms or a family history of the present illness. Before this visit, a pediatrician had empirically prescribed cefixime trihydrate syrup (150 mg/7.5 mL, b.i.d.) for five days.

Clinical Findings

The vital signs of the patient consisted of normal blood pressure (120/80 mm Hg), fever (37.8⁰ C), normal oxygen saturation (99%), healthy weight of Body Mass Index (BMI) (18.04). The result of extraoral examination indicated unilateral hematic crust lesions on the lower right lips (Fig. 1A) with no oral lesion. The clinical diagnosis was classical Herpes Zoster (HZ) in the crusting stage.

Diagnostic Assessment

Blood examination result in table 1 indicated macrocytosis, neutrophilia, eosinopenia, and lymphocytopenia. The presence of non-anemic macrocytosis (elevated MCV with normal hemoglobin) was considered an incidental finding, likely reflecting transient reactive reticulocytosis secondary to acute physiological stress from the viral infection rather than a primary hematological disorder. A serologic test was performed on day 30 of illness revealed anti-VZV IgG positive antibodies result (2.47 Immune Status Ratio).

Therapeutic Intervention

The treatment began with topical combination of Sodium Hyaluronate, Aloe Barbadensis, Dipotassium Glycyrrhizate applied on crusted lesion of lower right lips ter in die (t.i.d.) for 5 days and multivitamin and minerals syrup for 2 weeks. Systemic antiviral therapy was deferred because the patient presented well beyond the optimal 72-hour therapeutic window, the active vesicles had already progressed to the crusting stage, and the overall clinical course was remarkably mild.

Follow-up and Outcomes

Three days later showed erythematous mucosa on her lower right lip (Fig. 1B, 1C) and two weeks after initial visit, right lower lip was completely healed (Fig. 1D).

Table 1. Laboratory Findings of Patient 1 and 2

Full Blood Count	Reference Values	Patient 1	Patient 2	
		3 rd day	1 st day	1 week
Hemoglobin (g/dL)	10.8-15.6	12.7	12.3	13.2
Hematocrit (%)	33.0 – 45.0	38.1	37.5	41.5
Erythrocyte (10 ⁶ /uL)	3.8 – 5.8	4.8	4.9	5.1
Leukocyte (10 ³ /uL)	4.5 – 13.5	7.6	10.4	15.5 ^a
Thrombocyte (10 ³ /uL)	150 – 440	391	325	496 ^a
Erythrocyte index				
MCV (fL)	69 – 73	79.9 ^a	76.5 ^a	81.2 ^a
MCH (pg)	22.0 -34.0	26.6	25.1	25.8
MCHC (g/dL)	32.0 – 36.0	33.3	32.8	31.8 ^b
Differential Count				
Basophil (%)	0 – 1	0	0	0
Eosinophil (%)	2 – 4	0 ^b	0 ^b	4 %
Neutrophil rod (%)	0 – 6	2	2	2
Neutrophil segment (%)	50 – 70	76 ^a	74 ^a	68
Lymphocytes (%)	30 – 60	20 ^b	17 ^b	20 ^b
Monocytes (%)	2 – 8	6	7	6
Serology		Days 30		

anti-VZV IgG (ISR)	<=0.90 (negative) 0.91-1.09 (equivocal) >=1.10 (positive)	2.47 ^a	3.38 ^a
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Notes: ^a: high; ^b: low

Abbreviations: MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; MCHC: Mean Corpuscular Haemoglobin Concentration; VZV: Varicella Zoster Virus; ISR: Immune Status Ratio

CASE REPORT 2

Patient Information

An 8-year-old girl was referred to an Oral Medicine Specialist from Emergency Department with severe pain, burning sensations, pruritus, blisters on the right side of the jaw, malaise, and fever for 5 days prior. No history of chicken pox or similar illness.

Clinical Findings

Vital signs included tachycardia (157 bpm), blood pressure (110/60 mm Hg), fever (38.3⁰ C), normal oxygen saturation (99%), healthy weight (14.97). Extraoral examination revealed vesicular-bullous skin lesions and oedema on the right submandibular regions (Fig. 2A). Intra-oral examination demonstrated white plaque on dorsum of the tongue (Fig. 2B).

Diagnostic Assessment

Blood examination result (table 1) indicated macrocytosis, eosinopenia, lymphopenia and neutrophilia. Similar to the first case, the macrocytosis was interpreted as a reactive hematological response to the severe systemic viral infection, which did not warrant separate hematological intervention. The working diagnosis was HZ. A serologic test on day 30 of illness showed anti-VZV IgG positive antibodies result (3.38 ISR).

Therapeutic Intervention

The patient required hospitalization due to the severity of the symptoms. Systemic therapy began with oral acyclovir (400 mg q.i.d.) and Vitamin C for 11 days. Due to the extensive presentation and severe neurogenic inflammation, a 5-day course of methylprednisolone (8 mg daily) was administered to reduce severe acute pain and extensive submandibular edema. Furthermore, intravenous Ceftriaxone (2 mg), IV ranitidine (25 mg) and intravenous acetaminophen 250 mg quater in die (q.i.d.). Perioral lesions were treated with topical acyclovir cream 5 % 5 times a day for 5 days, mupirocin cream in between of acyclovir cream, chlorhexidine gluconate 0.2% oral gargle twice daily and topical benzydamine HCl 7.5 mg by gentle com-press the lesion three times a day. Ceftriaxone and topical mupirocin were incorporated empirically to prevent and manage suspected secondary bacterial superinfection over the extensive, ruptured vesiculobullous lesions.

Follow-up and Outcomes

On 3rd day, we found crusts on the right corner of the mouth and exfoliative upper la-bial mucosa (Fig. 2C). Sparse pruritic papules were spread on her abdomen (Fig. 2D) and upper extremities (Fig. 2E). Two days later, crusts had partially desquamated (Fig. 2F, 2G) and treated with glycerol cream and desoximetasone cream twice daily for 5 days.

By day seven, irregular erosion on the right dorsum of the tongue was noted (Fig. 3A, 3B). The patient was discharged after a 14-day hospitalization with normal tongue appear-ance, dyschromia on the right perioral regions (Fig. 3C, 3D). Keloid (Fig. 3E, 3F) was re-referred to dermatologist. The final diagnosis was HZ multiplex. All appropriate patients' consent from their parents were obtained.

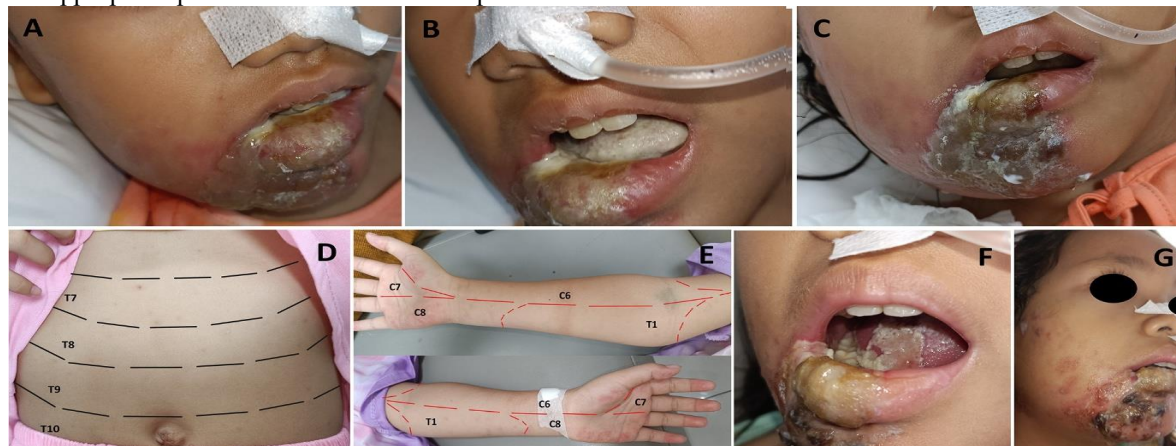


Figure 1. Extraoral, abdomen and upper extremities photographs of Severe Herpes Zoster Multiplex Presentation (Case 2).

On day 1, plaques and vesiculobullous lesions were observed along the right mandibular dermatome (A); white pseudo membrane on the dorsal tongue (B). on day 3, honey-colored crusts on the right perioral region with upper labial exfoliation (C); papules on the abdominal T7 and T10 (D), and upper extremity C6 and C8 dermatomes (E). On day 5, desquamated crusts, mucosal sloughing, and hypopigmented macules were observed across the right perioral and mental regions (F, G).



Figure 2. Extraoral photographs of Classical Herpes Zoster (Case 1). Unilateral hematic crusts on the right lower lip (A) sloughed off, revealing erythematous mucosa (B, C), and ultimately resolved with mild desquamation (D).



Figure 3. On day 7, adherent scaling was observed on the right perioral and mental regions, alongside mucosal sloughing of the right lower lip and erosions on the anterior dorsal and lateral tongue (A, B). Post-treatment, preauricular scarring and mixed dyschromia developed on the right perioral and mental regions (C), with resolving lower lip desquamation and complete healing of the dorsal tongue (D). Follow-up revealed keloid formation at 1 month (E) and 2 months (F) post-hospitalization.

DISCUSSION

Pediatric HZ is an infrequent illness that can occur in both immunocompetent and immunocompromised pediatric patients⁴ and only a few cases present with mucosal involvement.⁸ In our report, both patients were unvaccinated. Patient 1 (prior varicella) exhibited a benign, localized course consistent with the literature on mild childhood zoster which supports a diagnosis of self-limited HZ.¹ Conversely, the VZV-naïve Patient 2 with severe multi-dermatomal disease. The presence of VZV-specific IgG one month after symptom onset above the established cut-off indicates seropositivity and prior exposure, effectively distinguishing the vesicular rash from primary generalized varicella, herpes simplex virus (HSV) infection, or severe bullous contact dermatitis—which are critical differential diagnoses in pediatric vesiculobullous presentations.⁹

Three days after initiating treatment, the second patient developed papulovesicular eruptions on both upper extremities and abdomen, which parallels other case reports indicating that HZM may also progress during initial antiviral medication.⁸ The literature indicates a predilection for the cervical, trigeminal, and sacral dermatomes in pediatric patients under 10 years old with HZ.⁴ The second patient exhibited involvement of the cranial, cervical, and thoracic dermatomes which is indicative of the clinical manifestations associated with HZM; while the first patient only demonstrated isolated cranial dermatome involvement.

VZV can replicate in arteries and spread transaxonally and has been associated with other forms of vasculopathy. This was evident in the second patient, whose lesions also involved the T7 and T10 dermatomes, in addition to the C6 and C8 dermatomes of the upper extremities. The mechanism governing VZV-induced CNS vasculopathy is the trans-axonal transport of the virus from nerve fibers directly to the intracranial blood vessels.¹⁰ In immune-deficient patients suffering from either restricted or widespread HZ, the pathogen can be found in the bloodstream, infiltrating T-lymphocytes as these immune cells pass through spinal nerve nodes during viral reactivation. Although VZV characteristically induces syncytium formation in various cell types, this fusion effect has not yet been observed in T-cells, even though these white blood cells are the primary vehicles for spreading the infection systemically.¹¹ Vasculitis due to VZV infiltration, leads to intricate structural alterations within the vessel walls.¹²

The atypical clinical phenotype of HZM arises from simultaneous viral reactivation in separated ganglia. While immunosuppression is a known precipitating factor, the pathophysiology remains obscure; although a significant proportion of reported cases—including the two described in this series—occur in immunocompetent individuals, suggesting the existence of additional, undefined etiological factors. Because most reported cases of HZM, including our patients, occur in immunocompetent individuals, other unexplored underlying factors likely play a significant role in its development.¹³

The causative for reactivation in healthy immunocompetent pediatric patients remains controversial. Several possibilities related with these factors are wild-type VZV and non-anemic macrocytosis. Five cases of wild-type VZV without prior history of clinical varicella were reported, indicating subclinical primary infection plays a role in the pathogenesis of pediatric HZ.⁴ Non-anemic macrocytosis, suggested a potential vitamin B12 and folic acid deficiency may induce immune dysregulation and compromise the maintenance of VZV latency at an early age.²

An interesting incidental laboratory finding in both presented cases was the presence of non-anemic macrocytosis, characterized by an elevated mean corpuscular volume (MCV) with normal hemoglobin levels. While macrocytosis is conventionally associated with early-stage nutritional deficiencies—particularly vitamin B12 or folate deficiency which may compromise the maintenance of VZV latency²—its exact etiology in these immunocompetent pediatric patients remains undetermined.

In the context of acute viral infections such as Varicella-Zoster Virus (VZV), an elevated MCV may alternatively be attributed to reactive reticulocytosis. During acute physiological stress or systemic inflammation, the bone marrow may release larger, immature red blood cells (reticulocytes) into the circulation, transiently elevating the overall MCV. Because specific serum nutritional markers (e.g., vitamin B12 and folate levels) and reticulocyte counts were not obtained in our cases, attributing this hematological anomaly strictly to a nutritional deficiency remains speculative.

Therefore, while non-anemic macrocytosis is a noteworthy clinical observation in these patients, it should be interpreted with caution. These findings highlight the potential value of comprehensive hematological workups in future pediatric herpes zoster cases to elucidate whether such macroscopic changes are transient reactive phenomena to the viral infection or genuine underlying predisposing factors.^{2,5} Nevertheless, this incidental finding did not alter the clinical management or impede the successful recovery of either patient following tailored therapeutic interventions.

To optimize clinical outcomes, risk stratification is essential even in seemingly immunocompetent pediatric patients with HZ. While classical, localized HZ is typically managed in an outpatient setting with oral antivirals, specific clinical thresholds mandate immediate hospitalization to prevent severe morbidity.⁵ In the case of patient 2, based on current literature and clinical observations, inpatient admission is strongly indicated for patients presenting with disseminated disease or HZM (involvement of >2 non-contiguous dermatomes); systemic complications; and severe acute neurogenic pain. Furthermore, the presence of severe secondary complications—

such as extensive bacterial superinfection requiring intravenous antibiotics or any signs of central nervous system involvement—serves as an absolute criterion for inpatient management.^{14, 15}

Acyclovir and its prodrug valacyclovir are indicated for classical HZ and HZM to shorten the course of skin lesions.^{8,15} The advantage of oral valacyclovir consists of its 3- to 5-fold greater bioavailability compared to oral acyclovir.² Corticosteroids i.e. intravenous methyl prednisolone in combination with antiviral drugs remain controversial but may decrease disease severity and improve rash resolution if administered within 72-hours of eruption.^{16,17} Intravenous ceftriaxone was administered to manage such suspected severe bacterial complications.¹⁸ Conversely, patients lacking these high-risk features, as seen in patient 1, can be safely managed in an outpatient setting without aggressive interventions. Keloid management for the second patient utilized a multimodal strategy comprising pharmacotherapy, compression, surgery, laser, and psychosocial support.¹⁹

In conclusion, recognizing the clinical variability of HZ in pediatric patients is essential. Differentiating severe disseminated forms like HZM from classic HZ is critical, as HZM necessitates systemic therapy and hospitalization, compared to the outpatient supportive care often sufficient for classical presentations.

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