Received: July 22, 2024
Accepted: September 27, 2024

Acad Med J 2024;4(3):139-147
UDC: 616-022.6:578.825]-097-053.2
https://www.doi.org/10.53582/AMJ2443139h
Case report

HERPES ZOSTER IN 5-YEAR-OLD GIRL WITH NO PREVIOUS HISTORY OF CHICKENPOX: CASE REPORT

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Abstract

Herpes zoster (shingles) is caused by VZV (varicella zoster virus) infection. VZV is an enveloped, double-stranded DNA virus belonging to the Herpesviridae family; its genome encodes approximately 70 proteins. In humans, primary infection with VZV occurs when the virus comes into contact with the mucosa of the respiratory tract or conjunctiva. From these sites, it is distributed throughout the body. After primary infection, the virus migrates along sensory nerve fibers to the satellite cells of dorsal root ganglia where it becomes dormant. Reactivation of VZV that has remained dormant within dorsal root ganglia, often for decades after the patient's initial exposure to the virus in the form of varicella (chickenpox), results in herpes zoster.

This case report describes a herpes zoster infection in an immunocompetent 5-yearold girl, whose hetero-anamnesis from the parents provides information that the girl has not had chickenpox, and that she was not vaccinated against chickenpox, the mother denies that she had chickenpox as a child, and during pregnancy.

Case report: A 5-year-old girl with the appearance of a macule, papule, vesicular rash in the area of the chest and back, in the area of the 4th, 5th, and 6th dermatome, in the form of clusters. Before the appearance of a rash accompanied by burning pain. Serological analyses in addition to Herpes Zoster infection with elevated values of ELISA VZV IgM positive + 2.8, ELFA VZV IgG-+1.38 positive.

This case of a 5-year-old girl with herpes zoster, without previous evidence of varicella infection or immunodeficiency, presents a unique and interesting clinical scenario. It highlights the complexity of VZV infections and the need for comprehensive clinical and immunological evaluations in pediatric patients with herpes zoster. Future research in the mechanisms of viral latency and reactivation in such atypical cases will be critical to enhance our knowledge and management of VZV infections in children.

Keywords: varicella; herpes zoster; pediatric; immunocompetent

Introduction

Herpes zoster (shingles) is caused by VZV infection. VZV is an enveloped, doublestranded DNA virus belonging to the Herpesviridae family; its genome encodes

approximately 70 proteins. In humans, primary infection with VZV occurs when the virus comes into contact with the mucosa of the respiratory tract or conjunctiva. From these sites, it is distributed throughout the body. After primary infection, the virus migrates along sensory nerve fibers to the satellite cells of dorsal root ganglia where it becomes dormant.
Reactivation of VZV that has remained dormant within dorsal root ganglia, often for decades after the patient's initial exposure to the virus in the form of varicella (chickenpox), results in herpes zoster[1-3] .

The incidence of herpes zoster ranges from 1.2 to 3.4 per 1000 persons per year among younger healthy individuals while incidence is 3.9 to 11.8 per 1000 persons per year among patients older than 65 years. There is no seasonal variation seen with herpes zoster. Recurrences are most common in patients who are immunosuppressed[4] .

Herpes zoster (shingles) may begin with a systemic response (e.g., fever, anorexia, and lassitude), although symptoms are frequently mild and may not be associated with the classic manifestations of the condition recognized by either a patient or a physician.

Symptoms typically include prodromal sensory phenomena along with 1 or more skin dermatomes lasting 1-10 days (average, 48 hours), which usually are noted as pain or, less commonly, itching or paresthesias[5].

This case report describes a herpes zoster infection in an immunocompetent 5-yearold girl, whose hetero-anamnesis from the parents provides information that the girl has not had chickenpox, and that she was not vaccinated against chickenpox; the mother denies that she had chickenpox as a child, and during pregnancy.

Case report Hetero-anamensis from parents

Two days before the examination, with the appearance of a rash in the area of the left side of the chest, like clusters, accompanied by pain, the girl with her parents came to the family doctor, who prescribed therapy with Blokmax pp, Hipermangan, Poxclin 3x1, but there was no improvement. The rash was characterized by severe itching and pain at the same time, which is why they came to the University Clinic for Infectious Diseases and Febrile Conditions in Skopje.They gave information that before the rash appeared, the girl felt a strong burning pain, from which she could not sleep.

Personal history

A 5-year-old child, from second pregnancy, born at term, weight 3.2 kg, APGAR score 9, with normal psychomotor development, properly vaccinated according to the immunization schedule, with malnutrition. The family denied malignancy, immunocompromisation, chronic diseases, and frequent respiratory infections.

The mother gave information that the girl has not had chickenpox, and that the she herself had also not had chickenpox as a child, and during pregnancy; only on one occasion the child had an antypical rash with fever, diagnosed as enterovirus.

All reviews of systems were negative.

Physical examination was remarkable for grouped vesicles noted on the left anterior chest and healing vesicles with eschars noted on the left upper back. The rash appeared along the fourth, fifth and sixth thoracic dermatome. The rash was painful, blanched with palpation, with induration, without discharge. Vital signs and the remainder of the physical examination were unremarkable.

Treatment with tbl. Acyclovir according to body weight was started immediately; the patient weight was 10 kg, and hence 200 mg was prescribed, five times a day for seven days[6,7]. Sol. Blokmax every 6 hours, B-complex 2x1, hygienic-dietary regimen according to

given instructions, irrigation with a mild solution of hypermanganese, and locally Poxclean 3x1, and sol. Betadine 3x1 on the excoriations.

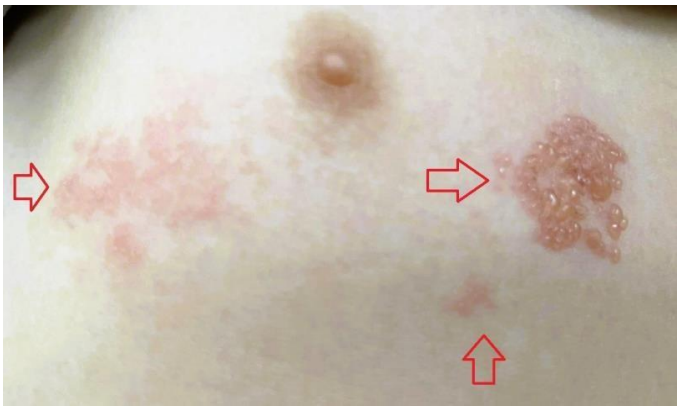

Fig. 1. Chest region, front 4th, 5th and 6th affected dermatomes. On the second day since the appearance of the rash, a maculo-papular rash appeared with rare vesicles organized in a group of clusters to the right of the areola, without clear boundaries. On the left, vesicles filled with, in a circular formation like a fetus with more defined borders. Below a small maculopapular change with central vesicles present

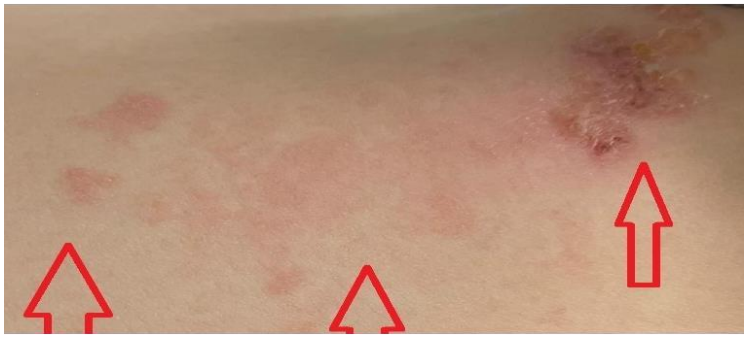

Fig. 2. Chest region, lateral left side, 4th, 5th and 6th affected dermatomes.
Second day since the appearance of the rash, on the first upper arrow, vesicles filled with clear liquid, some of them burst and ruptured with surrounding excoriation, which were caused by mechanical damage during itching, hyperemia; in the middle and lower part, maculopapular rash with individual vesicles were observed

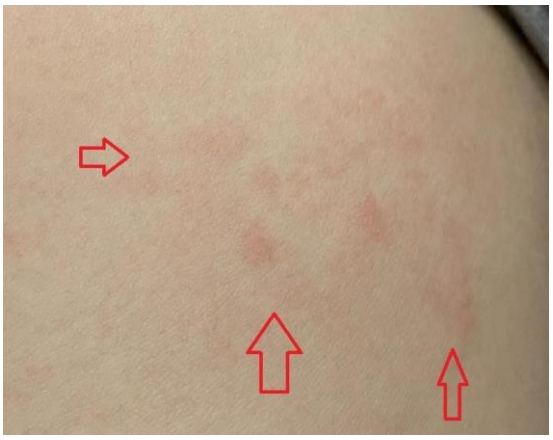

Fig. 3. Back 4th, 5th and 6th affected dermatomes.
Second day since the appearance of the rash.
Maculopapular with indistinct borders with rare vesicles

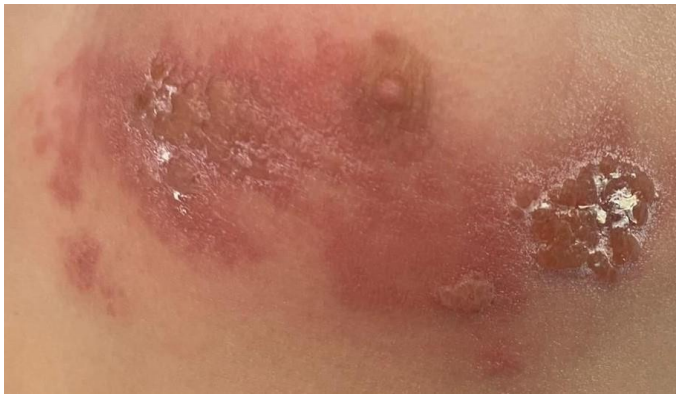

Fig. 4. Fifth day of the rash and third day of Acyclovir therapy. Maculopapular with vesicles with incipient crusts.

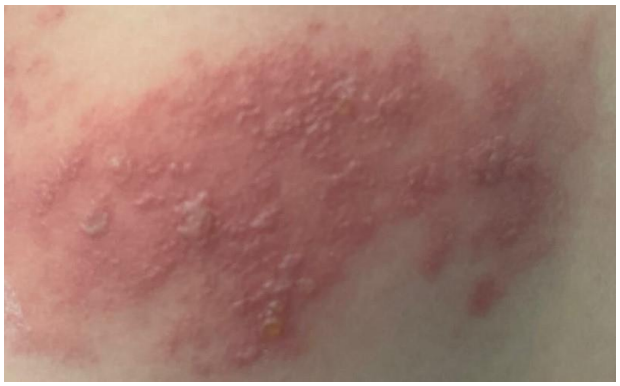

Fig. 5. Fifth day of the rash and third day of Acyclovir therapy. Maculopapular with vesicles with dried crusts.

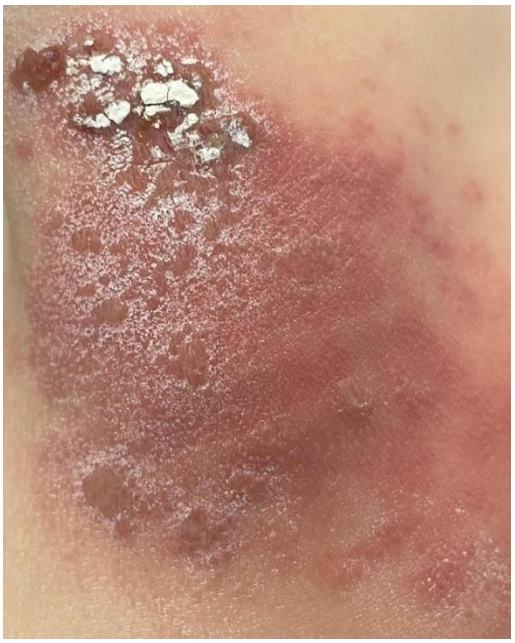

Fig. 6. Fifth day of the rash and third day of Acyclovir therapy. Maculopapular rash with dried vesicles, some in crusts, with secondary bacterial infection of the changes, with surrounding hyperemia

On the first control,the 5th day since the appearance of the rash, and the 3th day of Acyclovir therapy, a secondary bacterial infection with surrounding hyperemia was observed on the skin of the lateral side of the chest. The patient complained of severe pain and itching. The mother reported that the girl was scratching in the area of the changes, which was the most likely source of the infection. A therapy with a penicillin oral antibiotic, from the beta lactamase inhibitor group, sol. Amoxiclav 2x5 ml for 7 days was started, a probiotic, and the previously prescribed therapy was also administered. The mother did not cooperate with regard to the hygiene regime; picture 4 and 6 show that she also used liquid powder, which was not prescribed as a local skin treatment.

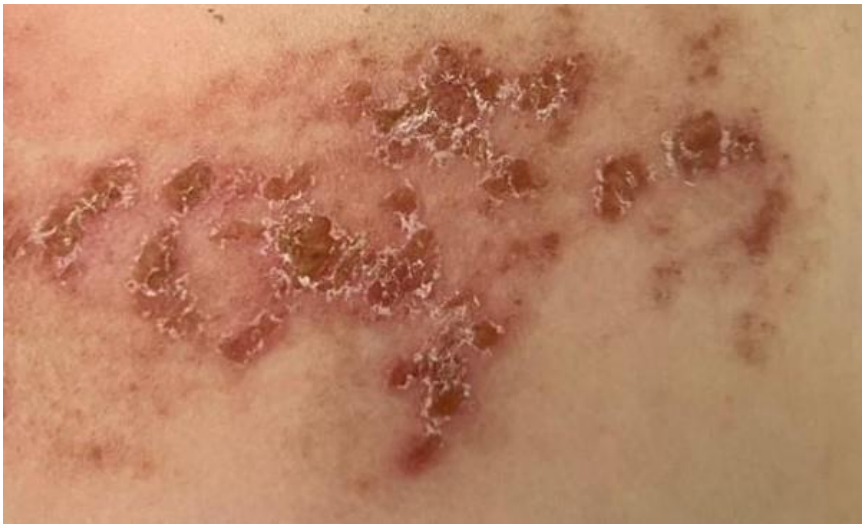

Fig. 7. Ninth day since the appearance of the rash, finished Aiclovir therapy, and 5th day of receiving Amoxiclav, back of chest

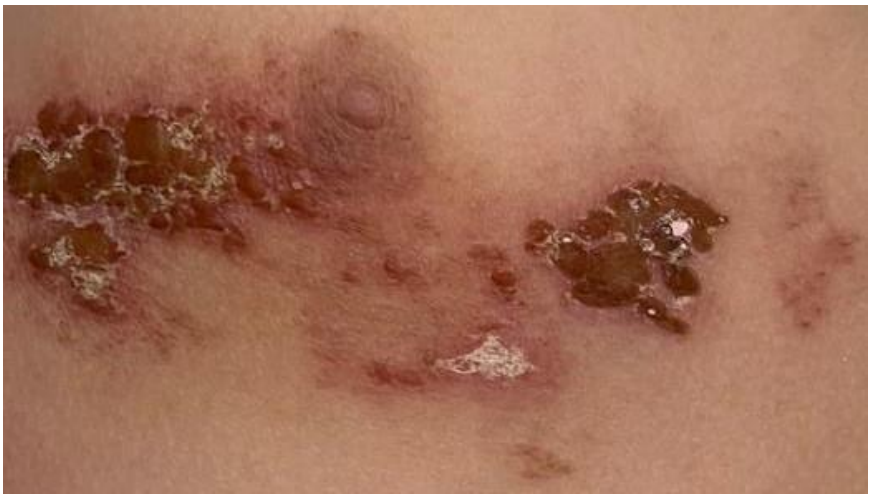

Fig. 8. Ninth day since the appearance of the rash, with finished Aiclovir therapy, and 5th day of receiving Amoxiclav, antero-lateral side of the chest

On the second control, on the 9th day since the appearance of the rash, with complete antiviral therapy, on the 5th day of the antibiotic treatment, the changes were in the crusting stage, with significantly reduced hyperemia; the girl felt better, the pain was reduced, with the itching still present, for the entire course of treatment without elevated body temperature. The

antibiotic was continued for another two days, and local treatment with Eosin 5% was started, by recommendation of the dermatovenerologist.

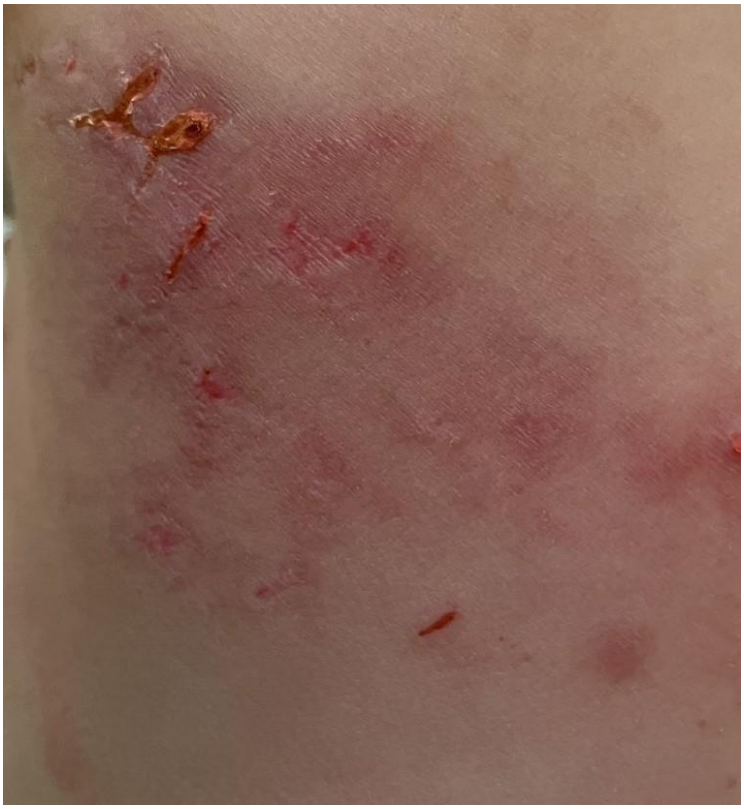

Fig. 9. Rash almost completely receding, with present excoriations-wounds that occurred during uncontrolled itching

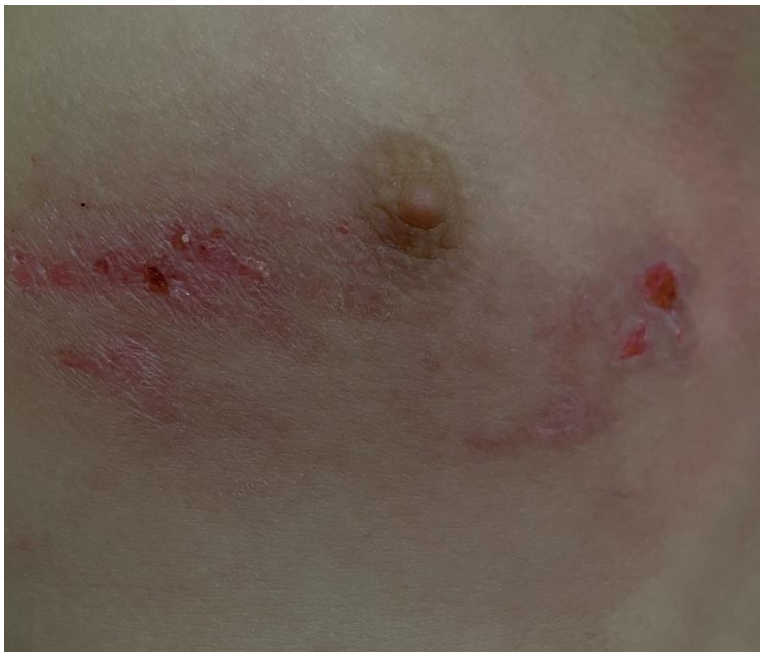

Fig. 10. Rash almost completely receding, with present excoriations-sores that occurred during uncontrolled itching

At the last check-up, the parents reported that their child felt feels much better, without an elevated temperature, that the crusts had fallen off on their own, but somewhere there were still itchy rashes. Antibiotic treatment was also completed, she was referred to a dermatovenerologist consultation regarding the wounds that were made, with a recommendation to continue dressing with Eosin, and Mupirocin 2x1 locally.

At the University Clinic for Infectious Diseases and Febrile Conditions, laboratory and serological analyses were performed, which confirmed the infection with Herpes Zoster:

- 06.05.2024 HIV-negative, ELISA VZV IgM-positive + 6,113, ELFA VZV IgGnegative
- 17.05.2024 ELISA VZV IgM-positive + 2,8, ELFA VZV IgG- + 1,38 positive

Table 1. Laboratory analyses by days

Date	**03.05.2024**	**06.05.2024**	**17.05.2024**
Hgb	138	131	127
RBC	5.37	5.12	5.08
WBC	8.6	5.7	7.6
PLT	271	263	429
HCT	0.41	0.39	0.38
MCV	76	76	76
MCH	25	25	25
MCHC	33	33	32
Granulocytes	0.59	0.50	0.45
Lymphocytes	0.23	0.29	0.42
Monocytes	0.16	0.19	0.10
Eosinophils	0.02	0.02	0.03
Glucose		4.6	3..7
AST		25	28
ALT		13	16
LDH		207	
CRP		2	1

Discussion

Herpes zoster is an infection caused by reactivation of the varicella-zoster virus (VZV), which after primary infection causes varicella (chicken pox). Herpes zoster is rare in children, especially in those without a history of previous varicella infection or known immunodeficiency. This case of a 5-year-old girl presenting with herpes zoster with no prior history of varicella infection, no history of congenital anomalies or plastic surgery, or known immunodeficiency and with serological tests showing positive IgM and elevated IgG titers presents a unique a clinical scenario that requires detailed elaboration.

Herpes zoster usually occurs in elderly or immunocompromised individuals, where the virus is reactivated due to a decline in VZV-specific immunity. In children, it is usually seen in those who have had a chickenpox infection, often in early childhood. The absence of any recorded varicella infection in this patient is significant. Possible explanations for this include:

- The child may have had a subclinical varicella infection that went unnoticed due to the mild nature of the symptoms. This may occur in a small percentage of children, leading to seroconversion without clinically apparent disease.

- Although the mother does not report varicella infection during pregnancy, asymptomatic or undiagnosed maternal VZV infection can result in transplacental transmission, leading to latent VZV infection in the child.
- If a child has received a varicella vaccine, reactivation can theoretically occur, although this is very rare and usually less severe than natural VZV reactivation.
- Positive IgM and increased IgG titers suggest active VZV infection or recent reactivation. This serological pattern is consistent with herpes zoster, where the immune response is directed against reactivated virus.
- Even in the absence of clinical evidence of immunodeficiency, subtle immune deficiencies may predispose a child to herpes zoster. A thorough immunological analysis may be required to rule out any underlying conditions that may compromise the immune response.

The presented case highlights the importance of innate immunity in controlling latent infections. It raises the question of the mechanisms of viral latency and reactivation in the absence of overt immunosuppression or prior exposure to varicella[8].

Clinical Implications

- Diagnosis and Treatment: Accurate diagnosis is based on clinical presentation and confirmatory serological tests. Early intervention with antiviral therapy, such as acyclovir, is key to managing herpes zoster and preventing complications, especially in pediatric patients.
- Public Health Considerations: This case highlights the need for careful monitoring and reporting of atypical cases of herpes zoster in children. Understanding such cases can inform vaccine policies and strategies for managing VZV infections in the pediatric population[9].
- Future Research Directions: Further studies are needed to elucidate the mechanisms behind herpes zoster in children without prior varicella infection. Investigating subclinical infections, viral latency, and immune responses in pediatric patients can provide valuable insights[10].

Conclusion

This case of a 5-year-old girl with herpes zoster, without previous evidence of varicella infection or immunodeficiency, presents a unique and interesting clinical scenario. It highlights the complexity of VZV infections and the need for comprehensive clinical and immunological evaluations in pediatric patients presenting with herpes zoster. Future research in the mechanisms of viral latency and reactivation in such atypical cases will be critical to enhance our knowledge and management of VZV infections in children.

Conflict of interest statement. The authors declare no conflict of interest.

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