

Herpes Zoster in the First Year of Life Following Postnatal Exposure to Varicella-zoster Virus

Four Case Reports and a Review of Infantile Herpes Zoster

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Background: Herpes zoster, a painful vesicular dermatomal eruption, is the result of reactivation of the varicella-zoster virus (VZV) from infected sensory ganglia. Traditionally, it is considered to be a disease of adults, in contrast to primary infection with VZV, which tends to occur mainly in children.

Observations: We report 4 cases of infantile herpes zoster in healthy immunocompetent children, all of whom were exposed to primary varicella infection within the first few months of life. A review of 62 cases from the literature reveals that postnatally acquired herpes zoster is less common than intrauterine infection

(31% [n=19] vs 69% [n=43]) and that there is a 1.5:1 male predominance. All dermatomes are equally affected.

Conclusions: Although uncommon, herpes zoster can develop in immunocompetent children as young as a few weeks of age and should be considered in the differential diagnosis of vesicular eruptions in infants. Most frequently, it is the result of intrauterine VZV infection, but it can be secondary to postnatal exposure to VZV at an early age.

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HERPES ZOSTER MAY BE SEEN in immunocompetent children and is frequent in children with acquired cellular immune deficiency from chemotherapy or human immunodeficiency virus. Herpes zoster in infancy is rare but well described following intrauterine exposure to varicella-zoster virus (VZV).^{1,2} We describe 4 infants presenting with classic herpes zoster between 4 and 11 months of age following inapparent or minimally symptomatic varicella as a consequence of postnatal VZV exposure. We also review the literature on herpes zoster in infants younger than 1 year.

tous base scattered along the jawline and lower cheek, with a sharp demarcation at the midline on the front and back of the neck, corresponding to the right C3 dermatome (**Figure 1**). Some vesicles were slightly outside the primary dermatome. Findings from a Tzanck smear revealed a multinucleated giant cell. Serologic results obtained at initial presentation demonstrated a positive VZV titer. A viral culture did not grow anything. The patient was treated with 200 mg of acyclovir orally 4 times a day for 7 days, with complete resolution of zoster without sequelae.

CASE 2

A 7-month-old male infant presented with a 3-day history of grouped vesicles on an erythematous base over the sacrum, without other symptoms. The patient had a history of varicella at 3 weeks of age following household exposure at 1 week of age to an older sibling with varicella. At the time of the primary infection, the infant was treated with oral acyclovir. The infant's mother had a history of varicella at age 8 years. On physical examination, the patient had grouped vesicles on an erythematous base on the sacrum, left buttock, midportion of the left posterior thigh,

REPORT OF CASES

CASE 1

A 7-month-old male infant presented with a 1½-week history of a crop of vesicular lesions on an erythematous base on the right side of the back of his neck. This infant had no prior history of varicella, but at 5 months of age had household exposure to varicella in a sibling. His mother had a history of varicella many years before pregnancy. Initial physical examination of the infant showed grouped vesicles on an erythema-

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Figure 1. Case 1. Clusters of grouped vesicles on erythematous bases in a distribution of the right C3 dermatome in a 7-month-old boy. His sister had varicella 2 months before.

and lateral aspect of the left foot, corresponding to the left S1 dermatome (**Figure 2**). No other abnormalities were noted. Findings from a Tzanck smear were positive for multinucleated giant cells. The patient was treated with 200 mg of acyclovir orally 4 times a day for 7 days. Complete resolution occurred without sequelae.

CASE 3

A 4-month-old infant presented to the emergency department with a 2-day history of fussiness, rhinorrhea, fever, and erythematous vesicles covering the right side of his face. The rash began with a vesicle on the right cheek and progressed to involve the eyebrow, forehead, temporal area, and right upper lip. The patient had a history of exposure to varicella during the neonatal period but never developed signs or symptoms of varicella. The infant's mother had a history of varicella many years before her pregnancy. On physical examination, the patient had a temperature of 37.8°C and an erythematous vesicular eruption with a crusty exudate in the right upper quadrant of the face over the distribution of the V1 and V2 branches of the trigeminal nerve. His conjunctivae were inflamed bilaterally, and there was a yellowish discharge from the right eye. The patient's nares and upper lip were edematous. The patient was admitted for treatment of possible bacterial superinfection complicating herpes zoster. The patient began intravenous therapy, including acyclovir and nafcillin sodium. Ophthalmologic examination showed no corneal involvement. The patient showed notable improvement in the eruption and facial edema and was discharged to home after 5 days. Viral culture of the vesicle fluid grew VZV. Serologic results obtained on admission were positive for VZV. By report of his pediatrician, the infant recovered completely without sequelae.



Figure 2. Case 2. Clusters of grouped vesicles on erythematous bases over the sacrum and popliteal fossa, corresponding to the left S1 dermatome, in a 7-month-old boy who had varicella at age 3 weeks after being exposed to his infected sister.

CASE 4

An 11-month-old male infant presented to the dermatologist's office with a chief complaint of a facial rash. Four days before the visit, he developed a single papule on his left cheek, which did not respond to over-the-counter 1% hydrocortisone cream. Multiple new vesicles appeared. The child had not been eating or sleeping well, but remained afebrile. History revealed that the patient's older brother had visited him in the hospital on the day he was born. The following day, the brother developed an eruption consistent with varicella infection, and he was removed from the home to live with his grandparents until his varicella resolved. Subsequently, although the patient had some febrile illness, he never had anything that appeared to be primary varicella. On physical examination, the patient had multiple grouped vesicles on erythematous plaques involving the left side of the chin, with sharp demarcation at the midline. Vesicles extended to the left side of the mouth, the lateral aspect of the left cheek in front of the left ear, and onto the temple and about 4 cm into the frontal area of the scalp. This corresponded to the left V3 dermatome (**Figure 3**). Findings from a Tzanck smear were negative, but direct fluorescent antibody test results for VZV and a viral culture were positive. An acute-phase VZV titer was negative. No follow-up titer was obtained. The child was treated with 200 mg of acyclovir orally 4 times a day for 1 week. His vesicles rapidly crusted over, and the erythematous plaques resolved. Two months later, he was noted to have a 4 × 5-mm hypertrophic scar on his chin. When seen 2 years later, there was a 5 × 8-mm flat white scar remaining on his chin.

COMMENT

Generally, primary varicella tends to occur in childhood, whereas herpes zoster is a disease of adults, with most patients being older than 45 years.^{3,4} The age-adjusted incidence rates of herpes zoster are the lowest (0.45 per 1000 person-years) in the group 0 to 14 years of age and highest (4.2-4.5 per 1000 person-years) among people 75 years and older.⁵ In the pediatric population, the incidence is the lowest in the group 0 to 5 years of



Figure 3. Case 4. Multiple grouped vesicles on red plaques on the left cheek, corresponding to the left V3 dermatome, in an 11-month-old boy exposed to his brother who had varicella and visited him in the hospital on the first day of life.

age (20 per 100 000 person-years) compared with adolescents (63 per 100 000 person-years).⁶ There was a male predominance (1.5:1) observed in the literature, and all 4 of our cases were boys.

Antigen-specific **T cells** are believed to be the principal gatekeepers of latent VZV. Conditions in which cellular responses were lost or diminished by immunosuppression pose a risk for reactivation of VZV and recurrent disease manifestation as herpes zoster.⁷⁻⁹ Herpes zoster in older individuals is associated with loss of VZV-specific cellular immunity.¹⁰ Herpes zoster in individuals undergoing chemotherapy is due to suppression of cellular immunity, whereas herpes zoster in human immunodeficiency virus-infected individuals is due to viral destruction of T cells.

Acquisition of herpes zoster in healthy immunocompetent children in early childhood or during intrauterine exposure has been attributed to the immaturity of the immune system.^{8,11} Terada et al¹¹ conclude that immunological status before primary infection with VZV is important and affects reactivation of VZV. They observed that, 6 to 7 weeks after primary varicella, infants had a lower response of VZV-specific cellular and humoral immunity compared with children who had infection at older ages (>1 year). In another study,¹² the peak levels of IgG antibodies after primary varicella were lower in infants compared with older children. Low response in specific VZV immunity is a valid reason to consider varicella in the first year of life as a risk factor for development of herpes zoster in otherwise healthy children.^{7,8,11,13,14} Terada et al¹⁵ showed that healthy immunocompetent children who had primary VZV before 1 year of age remained positive for VZV (as determined by polymerase chain reaction) for the longest period. From these data, Terada et al¹⁵ hypothesized that a "subclinical reactivation" puts infants with a history of primary varicella at risk for herpes zoster. In 69% of infantile herpes zoster cases (ie, <12 months of age) reported in the literature, the initial event could be traced

to maternal varicella during pregnancy. Dobrev¹⁶ observed that maternal varicella during the first trimester is likely to produce congenital varicella syndrome; when women have the disease later in pregnancy, the fetus can develop asymptomatic congenital infection and subsequently present clinically with herpes zoster within the first year of life. Newborns of VZV-immune mothers can also develop subclinical varicella within the first 6 months of life. In these cases, maternal VZV antibodies passively transferred to the infant may modify the disease into a subclinical form. In general, infants with primary varicella infection are at high risk for herpes zoster within the first year of life.¹⁶

Acute varicella has been acquired postnatally less frequently (31% [19/62] of the reported cases) than as an intrauterine infection. There are some instances, however, in which the initial episode of varicella has not been documented. Although there are 43 documented cases of intrauterine exposure to varicella with subsequent herpes zoster in the first year of life, there are only 9 cases with known postnatal exposure and 10 in which the time of exposure was not well documented. Baba et al^{8,17} suggest that in infancy the presence of maternal antibody modifies primary infection and that subclinical primary infection may predispose to herpes zoster. Although the mothers of the 4 infants in our series all had varicella in childhood, we hypothesize that their antibodies were not protective enough to have prevented primary infection with VZV in their infants.

The diagnosis of herpes zoster can be made by a Tzanck smear of scrapings from the floor of the vesicles,¹⁸ direct fluorescent antibody tests on similar smears,¹⁹ presence of high or rising titers exposed to VZV,¹⁸ and, definitively, by culture findings of the VZV virus.¹⁸ A high index of suspicion should be aroused when vesicular lesions are noted to be in a dermatomal distribution. The most common differential diagnosis is impetigo, and bacterial culture can usually distinguish the 2 conditions, unless there is concurrent bacterial infection of the herpetic lesions. Viral and bacterial cultures are often both performed.

In general, children tolerate herpes zoster much better than adults, with the disease usually being mild and lasting 1 to 3 weeks.^{7,9} Clinical features include pruritus and pain, with rare postherpetic neuralgia.^{4,7,20} Systemic reactions include fever, headache, and lymphadenopathy. Secondary bacterial infections and ophthalmic herpes zoster have been reported.⁴ The most frequently involved dermatomes are cranial, cervical, and thoracic dermatomes.^{7,9,21} Among the cases reviewed in this article, the thoracic dermatome was affected in most patients. Involvement of more than 1 dermatome can occur.

Summaries of the reported cases of herpes zoster in the first year of life are presented in **Table 1** and **Table 2**. There were 43 cases (69%) of prenatal acquisition (Table 1) and 19 cases (31%) of postnatal acquisition (Table 2). Of the cases in which patient sex was reported, 29 (60%) occurred in boys and 19 (40%) in girls. In 14 cases (23%), the reports did not specify sex. Of the 51 cases in which location was specified, 14 (27%) were in cranial nerve, 10 (20%) in cervical, 17 (33%) in thoracic, and 10 (20%) in lumbosacral dermatomes. In many cases, more than 1 dermatome was affected.

Table 1. Reported Cases of Herpes Zoster in the First Year of Life Due to Intrauterine Exposure

Source	Case No./Sex/Age	Exposure	Dermatome
Vachvarichsanong, ²² 1991	1/M/5 mo	8 mo Gest	CN V1
Brunell, ²³ 1981	2/NS/7 mo	6 mo Gest	CN V2-V3
Brunell, ²³ 1981	3/NS/8 mo	6-7 mo Gest	T3
Laude and Rajkumar, ²⁴ 1980	4/M/4 mo	6 mo Gest	Truncal
Dworsky et al, ²⁵ 1980	5/M/4 mo	3 mo Gest	L1-2
Dworsky et al, ²⁵ 1980	6/M/4 mo	6 mo Gest	T3-4
David and Williams, ²⁶ 1979	7/F/7 mo	6 mo Gest	CN V1, 2, 3
David and Williams, ²⁶ 1979	8/F/5 mo	20 wk Gest	T8
David and Williams, ²⁶ 1979	9-10/F/4 mo and 10 mo	36 wk Gest	S2 and T2-3
Derrick, ²⁷ 1998	11/M/5 mo	6-7 mo Gest	T7, 10, 11
Chiang et al, ²⁸ 1995	12/F/11 mo	6 mo Gest	T3-4
Chiang et al, ²⁸ 1995	13/M/7 mo	6 mo Gest	T2-4
Jayawardene, ²⁹ 1994	14/M/23 d	4 mo Gest	L2
Handa, ³⁰ 1997	15/F/3 mo	7 mo Gest	Forehead and hairline
Huang et al, ³¹ 1994	16/F/7 mo	6 mo Gest	L1-3
Huang et al, ³¹ 1994	17/M/3 mo	6 mo Gest	C2
Shishov et al, ³² 1983	18/F/5 mo	16 wk Gest	Forehead, eyelid, and temple
Bennet et al, ³³ 1985	19/M/18 d	14 wk Gest	S1
Paryani, ³⁴ 1986	20/NS/7 mo	Second trimester	NS
Helander et al, ³⁵ 1982	21/NS/6 mo	Second trimester	C2
Dobrev, ¹⁶ 1994	22/F/6 mo	6 mo Gest	CN V3 and cervical-thoracic
Taki and Inamochi, ³⁶ 1991	23/F/8 mo	30 wk Gest	T3-5
Taki and Inamochi, ³⁶ 1991	24/M/8 mo	32 wk Gest	C2-3 and CN V2
Taki and Inamochi, ³⁶ 1991	25/M/7 mo	7 mo Gest	Chest
Taki and Inamochi, ³⁶ 1991	26/M/11 wk	14 wk Gest	Chest and back
Taki and Inamochi, ³⁶ 1991	27/F/8 mo	17 wk Gest	C6-8
Enders et al, ² 1994	28-35/NS/<1 y	14-33 wk Gest	Various
Feldman, ³⁷ 1952	36/NS/4 d	First trimester	L5-S2
Vachvarichsanong, ²² 1991	37/M/5 mo	8 mo Gest	CN V
Kakouri et al, ³⁸ 1998	38/M/2 mo	Second trimester	Left cervical
Kakouri et al, ³⁸ 1998	39/F/7 mo	Third trimester	Left thoracic
Rivas de LaLastra and Lasso Bonilla, ³⁹ 1995	40/F/3 mo	3 mo Gest	L4-S1
Rivas de LaLastra and Lasso Bonilla, ³⁹ 1995	41/F/8 mo	5 mo Gest	Trigeminus
Brunell, ⁴⁰ 1983	42/NS/3½ mo	7 mo Gest	Unknown
Brar et al, ⁴¹ 2003	43/F/16 d	Maternal exposure at 6 mo gest without clinical disease	Left CN V1

Abbreviations: CN, cranial nerve; gest, gestation; NS, not specified.

Table 2. Reported Cases of Herpes Zoster in the First Year of Life Due to Postnatal Exposure

Source	Case No./Sex/Age	Exposure Age	Exposure Source	Dermatome
Dobrev, ¹⁶ 1994	1/F/7 mo	Unknown	Unknown	CN V2
Dobrev, ¹⁶ 1994	2/F/8 mo	3 mo	Father with primary varicella infection	Left upper extremity
Dobrev, ¹⁶ 1994	3/M/1 mo	Unknown	Unknown	Right upper extremity
Kouvalainen et al, ⁴² 1972	4/M/7 mo	Unknown	Some cases in community documented	L2
Elmer and George, ⁴³ 1999	5/F/7 mo	4 mo	Primary varicella infection	Back dermatomes
Mok, ⁴⁴ 1971	6/M/5 mo	4 wk	Primary varicella infection	T5
Taki and Inamochi, ³⁶ 1991	7/M/12 d	Unknown	Unknown	CN V1-2
Taki and Inamochi, ³⁶ 1991	8/M/5 mo	Unknown	Unknown	Gluteal and right lower extremity
Taki and Inamochi, ³⁶ 1991	9/M/4 mo	Unknown	Unknown	C3-5
Taki and Inamochi, ³⁶ 1991	10/M/9 mo	Unknown	Unknown	C3-4
Taki and Inamochi, ³⁶ 1991	11/M/9 mo	Unknown	Unknown	T4-5
Madden, ⁴⁵ 1952	12/F/8 mo	Unknown	Unknown	T4-6
Lagarde et al, ⁴⁶ 2001	13/M/7 mo	4 mo	3 Siblings with primary varicella infection	Left CN V1
Kashima, ⁴⁷ 2003	14/M/9 mo	2 wk	Primary varicella infection	Left L2-S1
Takayama et al, ⁴⁸ 2000	15/M/7 mo	Unknown	Unknown	Thoracic
Present report	16/M/7 mo	5 mo	Sibling with primary varicella infection	Right C3
Present report	17/M/7 mo	3 wk	Primary varicella infection	Left S1
Present report	18/M/4 mo	Neonatal	Family member with primary varicella infection	Right CN V1 and V2
Present report	19/M/11 mo	1 d	Sibling with primary varicella infection	Left CN V3

Abbreviation: CN, cranial nerve.

Oral acyclovir is recommended by the manufacturer to be given at an oral dosage of 20 mg/kg of body weight per dose 4 times a day to children older than 2 years, and it remains the first-line therapy for VZV in children. Oral (40-60 mg/kg per day) or intravenous (30 mg/kg per day) treatment has been used for 5 to 8 days or for 2 days after new lesions stop developing.³⁸ Other authors suggest 100 mg/kg per day for 7 days⁴⁷ or 50 mg/kg per day for 5 to 7 days.^{27,31} In acute herpes zoster, it decreases the time of new vesicle formation and the number of days to crusting. Analgesics and appropriate skin care provide relief and minimize the risk of secondary infection.⁹

CONCLUSIONS

Although herpes zoster is not common in children younger than 1 year, a review of the literature reveals that it occurs. The most frequent cause in immunocompetent patients is intrauterine exposure to VZV. We have described 4 cases of herpes zoster following postnatally acquired primary varicella. Siblings infected with varicella are the most usual contact source. We hypothesize that this condition is rarely recognized because of the mild clinical manifestations in this age group and the expectation that maternal antibodies will be protective.¹⁶ It is likely that the vesicular lesions of herpes zoster in this age group are misdiagnosed as impetigo or other cutaneous disorders. With a high degree of suspicion, the dermatomal distribution of a vesicular eruption in infancy should point the clinician toward a correct diagnosis of herpes zoster. Why some children are not protected from infantile VZV infection by maternal antibodies remains a question for further investigation.

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