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Herpes Zoster in Otherwise Healthy Children

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Abstract: In normal infants and children, zoster can occur at any time after varicella or varicella vaccination. It is usually diagnosed clinically: a unilateral vesicular eruption following a dermatome or dermatomes. The incidence of zoster increases with age, although children who have had varicella during the first year of life (or *in utero*) are at increased risk of developing zoster. The incidence of zoster is less after varicella vaccination than after natural infection. Zoster in children is frequently mild, postzoster neuralgia rarely if ever occurs, and antiviral therapy is usually not needed. In a previously normal child with zoster, if the history and physical examination are normal, a laboratory search for occult immunodeficiency or malignancy is not needed. We present five cases of zoster in healthy children and review zoster in the pediatric age group.

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LEARNING OBJECTIVES

1. Describe the epidemiology of zoster in immunocompetent children.
2. Describe the clinical presentation of zoster in otherwise healthy children.
3. State the practical laboratory methods for confirming a suspected diagnosis of zoster.
4. Recall the clinical management of a child with zoster.

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Primary infection with varicella-zoster virus (VZV) causes varicella (chickenpox). VZV then establishes latency in the dorsal (spinal) sensory ganglia or the cranial nerve ganglia. Reactivation of VZV from the ganglia results in a sensory dermatomal distribution of vesicles called zoster (from the Greek meaning belt or girdle) or shingles (from the Latin cingulus[r] meaning an encircling structure). Zoster can occur any time after varicella but the incidence of zoster increases with age (Table 1).[1–4] A pediatric practice responsible for 1000 patients may see about 1 case of zoster every 2 years. The following are 5 brief case reports of zoster in previously normal infants and children. These cases are presented to emphasize the pediatric risk factors for zoster and the diagnostic and treatment options.

CASE REPORTS

Case 1

A previously well 6-year-old girl developed erythema on her right cheek. During the ensuing 72 h she developed painful, pruritic vesicles on the right cheek, neck and ear. She was examined by her pediatrician who found more than 100 vesicles in the distribution of the right V-2 and V-3 dermatomes. Suspecting zoster, the pediatrician sent her to the pediatric infectious disease clinic. Her medical history was relevant for having had a mild case of chickenpox at 3 months of age.

On our examination the patient was afebrile, and the only abnormal finding was the rash (Fig. 1). Zoster was confirmed by a positive VZV direct immunofluorescence (DFA) test performed on scrapings from the bases of vesicles. Antiviral therapy was not given but she was prescribed

TABLE 1. Incidence of Zoster

Age (yr)	Cases/1000 Person-yr	Reference
0–14	0.47	2
15–24	1.06	2
25–34	1.93	2
35–44	2.28	2
45–54	3.13	2
55–64	5.68	2
65–74	9.99	2
>75	13.74	2
1–12*	4.10	3
1–19†	0.14	4

*Patients with a history of chickenpox during the first year of life.
†Patients who received the varicella vaccine after age 1 year (incidence based on passive surveillance after immunization).

hydroxyzine for pruritus. No further testing was performed. The family was reassured that zoster is not uncommon in infancy/childhood if varicella occurs in the first year of life. Since the diagnosis of zoster, the patient has been well for 1 year.

Case 2

A previously well 3-year-old girl had 12 h of fever and irritability preceding a vesicular eruption of the left lumbar area. She had had varicella at age 6 months manifested by ~10 vesicles on her chest and abdomen. When seen by her physician on the first day of rash, the physical examination was normal with the exception of fever and rash. Her physician suspected zoster and referred her to infectious disease for possible therapy.

On our examination her temperature was 38.4°C (101°F) rectally. The physical examination was normal except for confluent vesicles involving the left L-1 and L-2 dermatomes (Fig. 2). Vesicles were present around her urethra. She was able to void but had dysuria. A vesicle was aspirated and placed in tissue culture within minutes of collection. The base of the unroofed vesicle was scraped and placed on a slide for Tzanck smear which was positive for multinucleated giant cells (consistent with VZV infection). Later the culture was also positive for VZV virus.

Because of the severity of her zoster, she was treated with oral acyclovir (20 mg/kg every 6 h) and acetominophen with codeine. Within 72 h fever, pain and dysuria resolved. The skin lesions were completely crusted by Day 7 after onset, and the acyclovir was stopped after 7 days of therapy. Other than zoster she had no history or physical findings suggestive of an underlying immunodeficiency or other serious problem. Therefore an immunologic work-up was not undertaken. The patient has remained well for 5 years since the diagnosis of zoster.

Case 3

A previously healthy 8-year-old girl developed a painful, pruritic vesicular eruption on her right thorax. She had no fever or systemic symptoms. On Day 3 of rash she was seen by an infectious disease specialist. She was afebrile, and the physical examination was normal except for 20 to 30 vesicles involving the right T-6 dermatome. Zoster was confirmed with a positive VZV DFA. She had had varicella a year before and was treated at that time with oral acyclovir. The family was reassured that zoster can occur any time after varicella and that there is one case in the medical literature[5] of a child who developed zoster after acyclovir treatment of varicella. Our patient's zoster healed within 7 days without treatment, and she has remained well during the last 8 years.

Case 4

A previously well 3-year-old boy presented to his doctor with a 3-day history of left eye pain and photophobia. There was no rash. He was evaluated by his pediatrician who referred him to an ophthalmologist because of conjunctival injection with severe eye pain. Slit lamp examination revealed slight corneal staining. He was treated for a corneal abrasion with patching and erythromycin eye ointment. During the next 2 days, the eye pain persisted, and he developed fever and a rash. He was referred to infectious disease.

On examination his temperature was 101°F (38.4°C). He had a papular vesicular eruption in the area of the left ophthalmic branch of the trigeminal nerve (Fig. 3). He was photophobic, the bulbar conjunctiva was injected and there was a clear conjunctival discharge. The remainder of the physical examination was normal. Scrapings from the base of a vesicle on the tip of his nose was VZV DFA-positive. He was examined by an ophthalmologist who found small mucous plaques involving the cornea. The child was hospitalized and treated with intravenous acyclovir (10 mg/kg every 8 h) and pain medication. He improved greatly during the next 3 days and was discharged to receive oral acyclovir (20 mg/kg every 6 hours) therapy for 7 days.

Eye pain recurred the week after stopping the acyclovir. He was evaluated by his ophthalmologist who found faint corneal stromal abnormalities and white blood cells in the anterior chamber (diffuse uveitis). The facial rash had resolved. He was treated with atropine and prednisone eye drops for 7 days during which pain resolved. He had received the varicella vaccine at 1 year of age. The child had been adopted from Central America when he was 9 months of age and, in retrospect, he had a history of a vesicular eruption (probable varicella) at 6 months of age when he was in Central America. He has now been followed for 2 years and has remained well.

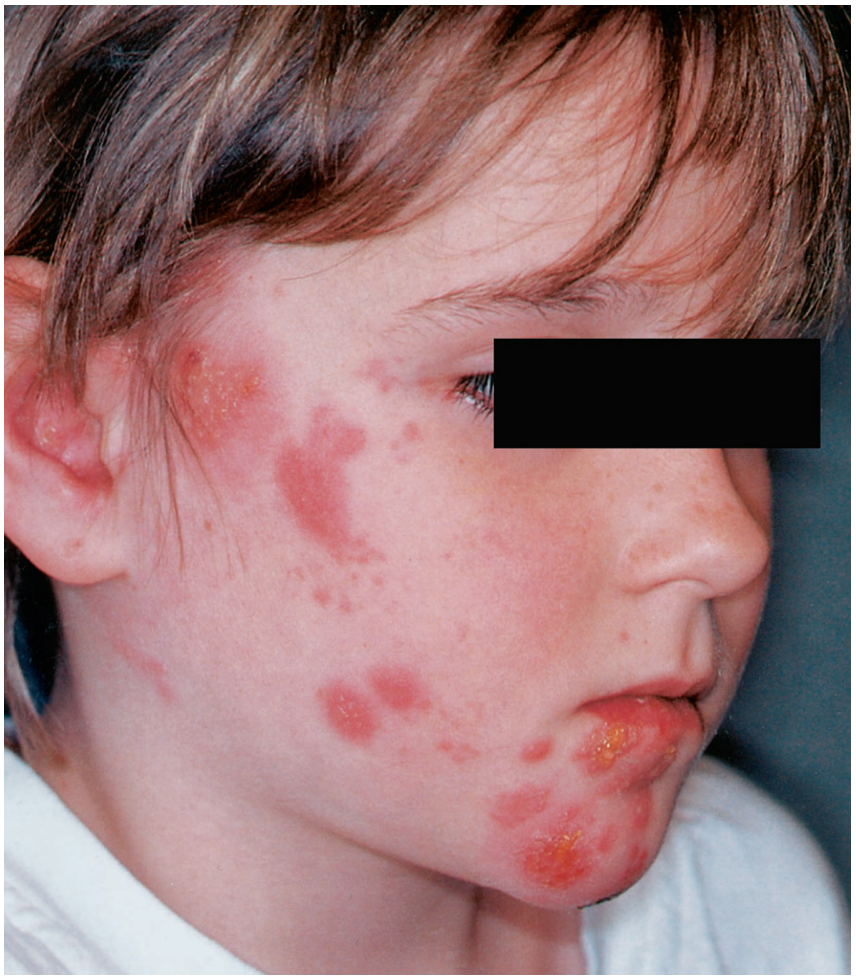

FIGURE 1. Zoster involving the V-2 and V-3 dermatomes.

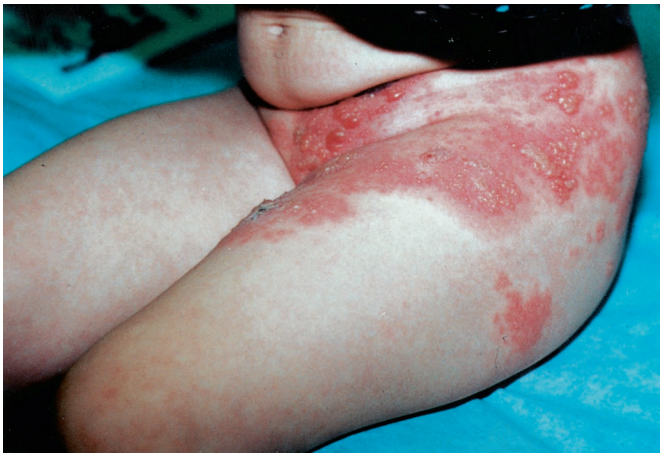

FIGURE 2. Severe zoster involving L-1 and L-2 dermatomes.

Case 5

A previously well 5-year-old girl presented to her pediatrician with a 10-day history of painful pruritic blisters which began on her left buttocks and spread to her left lateral thigh. She had received the varicella vaccine at age 15 months. Her pediatrician considered herpes simplex, zoster and contact dermatitis and referred her to infectious disease. When we examined her that same day, she was afebrile and had a vesicular rash involving the L-2 dermatome (Fig. 4). The base of a vesicle was scraped and was VZV DFA-positive.

Because this patient had zoster after varicella vaccine, some crusts of the lesion were removed and sent to Merck (West Point, PA) for DNA typing to differentiate the wild type VZV strain from vaccine (Oka/Merck) strain. No other studies were done, and the patient was reassured. The pain, pruritus and rash resolved within the week. The VZV isolate was typed (by Merck) as wild type VZV. It is possible that preceding varicella vaccine she had had a mild case of varicella that was not diagnosed or that after the varicella vaccine she was exposed to wild type VZV and was asymptomatically infected. The wild type VZV established latency and reactivated as zoster. After vari-

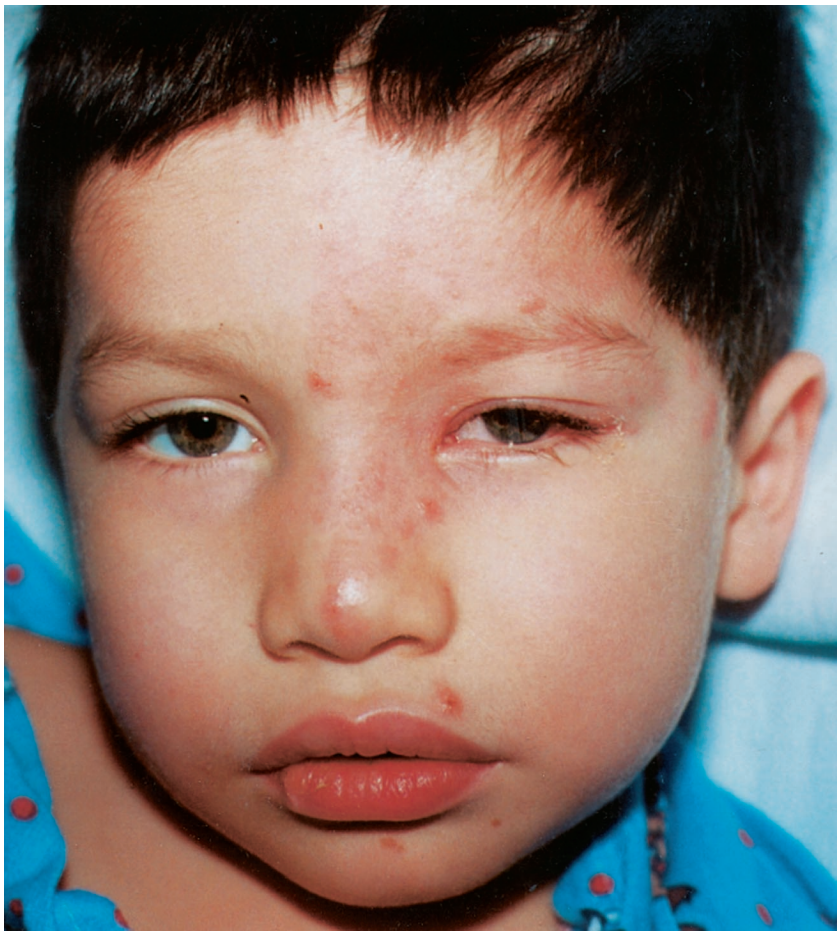

FIGURE 3. Zoster involving the V-1 dermatome including the tip of the nose (Hutchinson's sign) and corneal involvement manifested by a painful red eye.

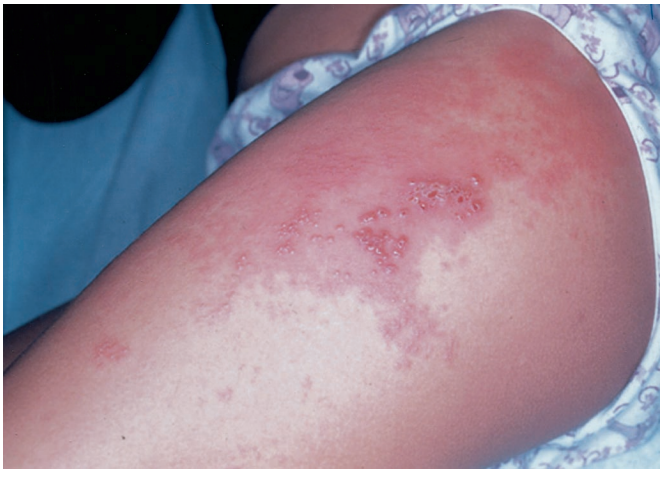

FIGURE 4. Zoster involving the L-2 dermatome.

cella vaccination patients can develop vaccine strain or wild strain zoster. The patient has remained well for 2 years.

DISCUSSION

History

In 1943 Garland[6] reported that a child could develop varicella after exposure to zoster. Ten years later Weller[7] was able to recover by tissue culture what he thought were different viral isolates from patients with varicella and patients with zoster. Then in 1958 Weller et al.[8] demonstrated that these viral isolates were the same, thus, the name VZV. In 1971 VZV antigens were identified by immunofluorescence and electron microscopy in the trigeminal ganglion in a patient who had ophthalmic zoster and died.[9] In 1974 VZV was cultured from the thoracic dorsal root ganglion of another patient with zoster.[10] VZV has not been cultured from cranial or dorsal root ganglia from autopsy tissue of adult patients without clinical zoster; however, using PCR small amounts of VZV DNA have been identified during latency in neuronal fractions of cranial and dorsal root ganglia.[11] During latency only a few of the more than 70 VZV genes are expressed, which is how VZV evades the immune system.[12]

Clinical and Laboratory Diagnosis

Zoster begins with pain and an erythematous maculopapular, then vesicular, rash in a dermatomal distribution. Adjoining dermatomes can be involved, and a few crops of vesicles are sometimes scattered outside the primary eruption. During the course of 1 week, the rash becomes pustular and then ulcerates and crusts. Healing occurs within weeks and sometimes results in scarring.

VZV can be cultured from the blister fluid if plating on tissue culture is done immediately. VZV may not survive in transport media. The most practical way to confirm VZV is to scrape the base of a new vesicle and apply the scrapings on a glass slide for VZV DFA testing, which is sensitive and specific. PCR can also be used to identify VZV in vesicular fluid.[12,13] Occasionally, other rashes mimic zoster including herpes simplex skin infection (usually does not follow a dermatomal pattern), contact dermatitis (not dermatomal and more pruritic than painful), staphylococcal impetigo (purulent vesicular fluid with bacteria on Gram stain) and ECHO virus infection (not locally painful).[14]

In adults postzoster neuralgia, defined as incapacitating pain present >30 days after onset of the rash, occurs commonly among patients older than age 60 years. It is more likely to occur after zoster that is initially severely painful and extensive.[15,16] Postzoster neuralgia occurs rarely, if ever, in children.[17]

Another important complication of zoster is visual impairment. Zoster affects the cranial nerves in 5% of children *vs.* 13% of adults (Table 2). When the ophthalmic division (V-1) of the trigeminal nerve is involved with VZV (herpes zoster ophthalmicus), ~50% of these patients have eye involvement. The ophthalmic division of the trigeminal nerve divides into the frontal, lacrimal and nasociliary nerves. Eye involvement is most common when accompanied by vesicles on the side or tip of the nose, meaning involvement by the nasocilliary branch of V-1 (Hutchinson's sign). Eye involvement presents clinically with a painful red eye most commonly caused by corneal keratitis and/or uveitis. VZV eye involvement can cause chronic inflammation and scarring.[18,19]

TABLE 2. Zoster Site in Children *vs.* Adults[1]

	Children (%)	Adults (%)
Thoracic	65	57
Cervical	13	11
Lumbar	11	13
Cranial	5	13
Sacral	4	4
Disseminated	2	2

On rare occasion zoster can involve the sensory part of the genticulate ganglion (8th cranial nerve) and the facial nerve. The zoster rash is confined to the tympanic membrane and ear canal, and there may be loss of taste in the anterior two-thirds of the tongue on the affected side. Complications include tinnitus, loss of hearing and paralysis of facial muscles. This condition is called Ramsay Hunt syndrome.[20]

Zoster in Pediatric Patients

Several studies of pediatric patients with zoster[1,3,21,22] reported that the most common risk factor for developing zoster was having had varicella before 1 year of age. This can be explained by a decreased development of cellular and humoral immunity to VZV, when varicella occurs during the first year of life.[23] Children who have had varicella during the first year of life develop zoster at an increased incidence, 4.1 cases/1000 patient-years *vs.* 0.45 case/1000 person-years for pediatric patients who have varicella after the first year of life.[3] The interval between varicella and childhood zoster averages 3.8 years if varicella occurs during the first year of life *vs.* 6.2 years if varicella occurs after the age of 1 year.[22] Studies of zoster in pediatric patients[1,3,21,22] did not find that childhood zoster was the harbinger of an underlying immunodeficiency, malignancy or HIV infection.

If a woman develops varicella during pregnancy, her fetus may become infected *in utero.* The child may then develop zoster, which occasionally disseminates, during infancy.[21,24–28] In 12 reported cases of infants who developed zoster after intrauterine varicella exposure, the average age at zoster presentation was 12 months (range, 2 to 41 months).[21,24–28]

Zoster in children causes minimal pain and fever, and most frequently involves the thoracic dermatomes (Table 2).[1] Pediatric zoster is rarely associated with postzoster neuralgia and usually resolves spontaneously in fewer than 8 days.[17,21,22]

The pediatric malignancies most commonly associated with zoster are leukemia and Hodgkin's lymphoma. Zoster does not precede the clinical manifestations of these malignancies but instead is associated with chemotherapy or relapse.[29] In the United States zoster has not been reported as the initial manifestation of HIV infection in children[30]; however, in central Africa where HIV is highly endemic, zoster may be the initial manifestation of HIV infection.[31]

The initial double blind controlled studies of oral acyclovir therapy for varicella in children showed no effect on the development of humoral and cellular immunity to VZV.[32–35] There is, however, one anecdotal case report of mild zoster in a 5-year-old who had been treated with oral acyclovir for varicella 1 year earlier.[5] That case is similar to our third case. Long term studies of children with varicella who were treated with acyclovir have not been done. The

significance of these two cases of zoster after acyclovir-treated varicella is unknown.

Zoster After Varicella Vaccination

Zoster can occur with either wild strain VZV or vaccine strain VZV in vaccinated children. The vaccine strain can establish latency after vaccination. Alternatively exposure to wild strain before or after vaccination may result in asymptomatic infection after which the wild strain can establish latency. The incidence of zoster appears to be less in pediatric patients given the varicella vaccine than in pediatric patients who develop varicella infection (Table 1). Among 9454 infant varicella vaccinees who were followed by passive surveillance for 44,994 patient-years, 8 developed zoster (0.18 case/1000 patient-years). One of the 8 patients had zoster with wild strain VZV (by PCR), and the other 7 virus isolates were not tested. The zoster occurred 1 to 6 years after vaccination.[36,37] There have been at least 6 case reports[38–40] of zoster in children after varicella vaccination, including our case report. Four cases were caused by wild VZV.[38,40] One case caused by vaccine strain VZV[28] was a 19-month-old who developed zoster on the right arm and shoulder 4 months after receiving the varicella vaccine in her right shoulder. All cases of postvaccination zoster have been mild and uncomplicated.

Zoster Contagiousness

Chickenpox is highly contagious. After home exposure to varicella, a nonimmune family member has a >90% chance of developing varicella.[41] Varicella is spread via the air and contact from the respiratory tract and skin lesions. Zoster is much less contagious and is spread directly from skin lesions. Patients are not considered contagious if the zoster is covered by a bandage or clothing.[42] Varicella can develop in a nonimmune individual after exposure to cases of varicella or zoster. Exposure of an immune individual to varicella or zoster should not result in zoster. In fact it boosts cellular immunity to varicella and may prevent zoster.[43] There are, inexplicably, reports of outbreaks of zoster[44–47] in immune individuals after common exposure to varicella or zoster. These zoster outbreaks can be explained by recognition artifact, i.e. after one case is diagnosed, it becomes more common to diagnose other cases.

Treatment

Three antivirals (acyclovir, famciclovir, and valacyclovir) are approved for treatment of zoster in adults.[48] Famciclovir and valacyclovir (available only in tablets) are better absorbed than acyclovir (available in liquid and tablets and much less expensive than famciclovir and valacyclovir). Famciclovir and valacyclovir are not approved for use in pediatric patients. In treatment studies of zoster in adults, famciclovir and valacyclovir were slightly more effective than acyclovir.[49] Treating zoster with antivirals (with or without prednisone) has little effect on the acute course but is important for reducing postzoster neuralgias.[49–51] For example in a study of adults that compared famciclovir with placebo for the treatment of zoster, the median time to resolution of vesicle formation was reduced from 6 days to 5 days.[50] The median duration of postzoster neuralgia was reduced from 160 days to 62 days. The incidence of postzoster neuralgia is 0% in patients 0 to 29 years old, 4% in patients 30 to 49 years old, 21% in patients 60 to 69 years old, 29% in patients 70 to 79 years old and 34% in patients >80 years old.[17] Antiviral treatment of zoster in immunocompetent children should be limited to treating ophthalmic zoster and zoster that at onset causes a moderate to severe rash and pain.[48] Acute pain management in children with zoster can usually be accomplished with ibuprofen; however, for severe acute pain, codeine may be needed.

The incidence of zoster in children is higher after natural varicella infection than after varicella vaccination. As the childhood population becomes increasingly immunized, zoster in children should become a medical curiosity.

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