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Childhood Herpes Zoster

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Herpes zoster (HZ) in childhood is rather unusual. This reactivation of chickenpox, the primary varicella-zoster virus (VZV) infection that lies dormant within sensory ganglia, is seen with increased frequency in otherwise healthy children who acquire chickenpox either in utero or within the first year of life. Our patient is a good example of this; he was exposed to chickenpox at the age of 3 months (by his 2 siblings) and developed HZ at 6 years of age.

Herpes zoster (HZ) is rare in the pediatric population and is most commonly seen in the immunosuppressed. HZ is caused by reactivation of the varicella-zoster virus (VZV), which lies latent in sensory ganglia after primary varicella exposure (chickenpox).[1-14] Children who have been infected with VZV in utero or before 1 year of age have an increased risk of developing childhood HZ.[15] Although complications may occur, HZ in healthy children is usually a self-limiting disease. Dermatomal pain is not as prevalent in childhood HZ as it is in adults with the disease, and postherpetic neuralgia does not occur in children. We describe an otherwise healthy $6^1/_2$-year-old boy who developed HZ after initial exposure to VZV at age 3 months.

Case Report

A $6^1/_2$-year-old boy presented with a right-sided vesicular exanthema involving an extensive area of the flank, back, and abdomen that had appeared 5 days earlier. The patient was otherwise in good health and not immunocompromised. The patient had no significant medical history, and his physical and mental development had been normal since birth. The boy's mother did not have a history of VZV infection during pregnancy. The patient's brother and sister both had primary varicella when the patient was 3 months old. The patient did not demonstrate fever, rash, or vesicles at that time, and he had not been vaccinated for VZV. The patient's father and mother have atopic dermatitis; his mother and brother also have allergic rhinitis.

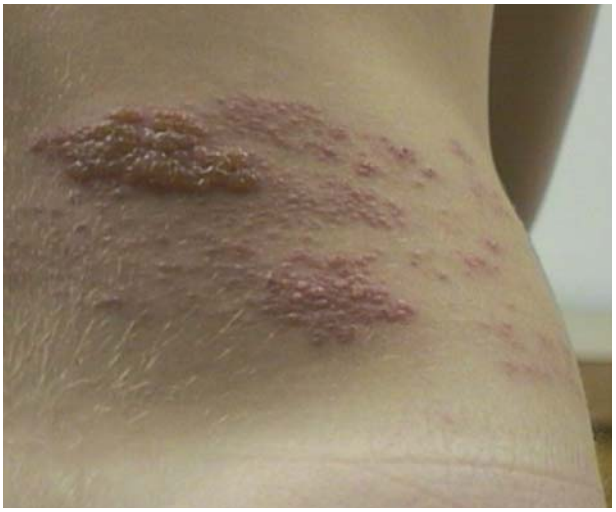

Figure 1. Grouped vesicles on an erythematous base on the right flank of a $6^1/_2$-year-old boy. The eruption is in a dermatomal distribution and does not cross the midline (features characteristic of HZ).

Findings of a complete physical examination were remarkable for the general skin appearance of fineness and dryness and grouped vesicles on an erythematous base coalescing into large vesicular patches on the right back, flank, and abdomen localized between the T11 and L2-3 dermatomes. These grouped vesicles were not painful and did not cross the midline (Figure 1). A prodrome of pain in the affected dermatomes prior to the appearance of vesicles was not noted. No lymphadenopathy, hepatosplenomegaly, or fever was noted at the initial examination. Results of a complete blood count were normal, and Tzank test results showed multinucleated giant cells with eosinophilic intranuclear inclusion bodies (Figure 2). Therapy was started with acyclovir, 200 mg, 4 times

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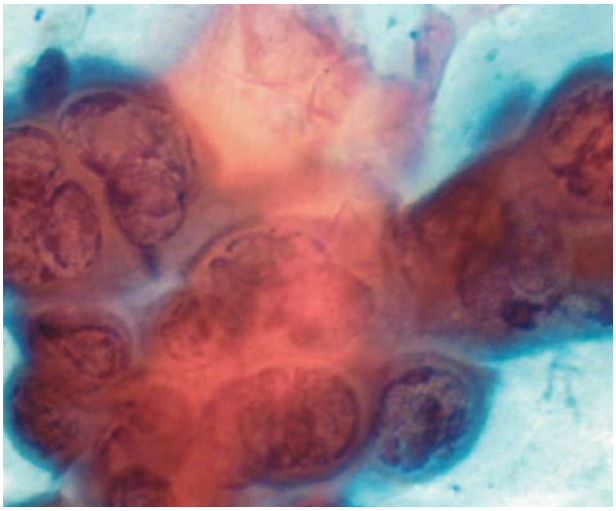

Figure 2. A Tzank test demonstrating clusters of cells with multiple ground-glass nuclei. Intranuclear inclusion bodies also are seen.

daily. The flank vesicles dried up first, and the entire area was healed between the 10th and 14th day of therapy. Slight hyperpigmentation, but no scarring, was evident after healing. Postherpetic neuralgia did not develop.

Comment

HZ is caused by reactivation of latent VZV, a double-stranded DNA virus in the Alphaherpesvirinae family that is also the causative virus of primary varicella.[1] After initial infection with primary varicella, VZV remains latent in sensory nerve ganglia, both cranial and somatic.[2] VZV persists in sensory ganglion for the life of the individual, although the exact mechanism of reactivation after primary varicella has not been fully elucidated.[3-5] The dermatomal distribution of HZ is almost pathognomonic, and the V1 (first division of the trigeminal) and T1-L2 dermatomes are the most commonly affected.[6]

HZ is rare in healthy children, but its incidence increases in older adults. The annual incidence of HZ in healthy children is 0.74 cases per 1000 in children younger than 9 years.[7] The annual incidence of HZ in adults aged 20 to 50 years has been shown to be 2.5 per 1000.[7] This number rises to 10 cases per 1000 in patients who are in the eighth decade of life.[7] This is most reasonably explained by a reduction in the cell-mediated immune-response mechanism to VZV in older adult patients, which correlates with the higher incidence of HZ in this group.[8,9] Humoral responses in the older adult population remain, for the most part, intact.[8,9]

Immunosuppressed individuals have a higher occurrence of HZ and a more severe form of the disease than their healthy counterparts.[10,11] Human immunodeficiency virus (HIV), malignancy, immunosuppressive drug therapies, and chronic drug treatments have been shown to precipitate HZ.[10-14] Immunosuppressed individuals also have a higher incidence of generalized HZ (extensive cutaneous involvement not confined to a dermatomal distribution) and visceral dissemination.[10,11,14]

Childhood HZ most commonly occurs in immunocompromised children, and it may precede the diagnosis of hematologic malignancy or acquired immunodeficiency syndrome (AIDS).[2,10,12] Healthy children who have been exposed to VZV in utero or who were primarily infected with VZV in the first year of life also show a higher incidence of disease.[10,15-17] In a study of 21 pediatric patients with HZ, ranging in age from a few months to 14 years, 13 (62%) were immunocompromised, most with hematologic malignancies such as acute lymphoblastic leukemia and acute myeloid leukemia.[17] Of the 8 healthy children who developed HZ, 2 (25%) patients were exposed to VZV in utero, and 6 (75%) were infected with VZV before their fourth year.

These findings support previous studies that reveal an increased incidence of HZ in healthy children exposed to VZV in utero or who have been infected with the virus in the first year of life.[15,16] An increased incidence of VZV in immunocompetent children infected with the virus in the first year of life also was confirmed.[10] Decreased cell-mediated response mechanisms caused by the immaturity of the immune system have been shown to be the etiology for the increased incidence of HZ in these populations.[15,18-20]

Our patient most likely contracted subclinical varicella at age 3 months after contact with his siblings. The appearance of HZ more than 6 years later supports the finding that healthy children infected with VZV before age 1 year have an increased chance of developing childhood HZ. Our patient is otherwise healthy, with no evidence of immunosuppression or malignancy.

The exanthema of childhood HZ is clinically similar to that of HZ in older adults, with grouped vesicles developing on an erythematous base in a dermatomal distribution.[2,10,17,21] In contrast to childhood HZ, adult HZ is almost invariably associated with a prodrome of pain in an involved dermatome a few days before the vesicular eruption.[11,14] The pain described is variable, ranging from deep chronic pain to episodes of severe lancinating pain, burning pain, or simply pruritic pain. *Zoster sine herpte* is a form of adult HZ in which dermatomal pain is the only clinical indication of the disease; the characteristic eruption of grouped vesicles does not develop.[22] Pain in the affected dermatome also persists after presentation of the vesicular eruption. Postherpetic neuralgia (PHN) is the

persistence of dermatomal pain after the HZ exanthema has healed.[11,14] The risk for development of PHN is estimated to be between 8% and 15% in the adult population, although the risk increases with increasing age.[23-25]

In pediatric HZ, pain is rarely associated with the vesicular eruption, and PHN does not occur.[2,10,16,17] No PHN was noted in 21 children (13 immunosuppressed and 8 immunocompetent) with zoster, and only 3 patients experienced mild pain and pruritus prior to and during the vesicular exanthema.[17] In another study, no PHN in 92 pediatric patients with HZ (47 immunosuppressed and 45 immunocompetent) was evident.[10] Similarly, our patient did not develop PHN and had only minimal pruritus from the vesicular exanthema. Prodromal pain did not occur.

Early acyclovir therapy is indicated in immunocompromised children with HZ and has been shown to decrease the incidence of morbidity and death in this population.[17] The development of HZ in healthy children is believed to be relatively benign and self-limiting. However, complications may occur in this population. In a study of 45 immunocompetent pediatric patients, there were 11 (24%) cases of complications from HZ.[10] Three children developed aseptic meningitis, 5 had extensive cutaneous involvement (generalized HZ), and 3 patients experienced facial palsy (Ramsay Hunt syndrome).[26] Early administration of acyclovir to lessen morbidity and quicken healing may thus be indicated in healthy pediatric patients who develop HZ.[2,17]

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