Herpes Zoster in Children

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PRACTICE **POINTS**

- Herpes zoster (HZ) should be included in the differential diagnosis for children presenting with vesicular lesions in a dermatomal distribution and a history of varicella exposure.
- Clinical diagnosis of HZ and herpes simplex virus can be aided by the use of viral polymerase chain reaction testing.
- Children with HZ should be monitored for the same possible complications as adults.

Herpes zoster (HZ) in immunocompetent children is quite uncommon. Initial exposure to the varicella-zoster virus (VZV) may be from a wild-type or vaccine-related strain. Either strain may cause a latent infection and subsequent eruption of HZ. We present a case of HZ in a 15-month-old boy after receiving the varicella vaccination at 12 months of age. A review of the literature regarding the incidence, clinical characteristics, and diagnosis of HZ in children also is provided.

Cutis. 2016;98:93-95.

Herpes zoster (HZ) is commonly seen in immunocompromised patients but is quite uncommon in immunocompetent children. Pediatric cases have been attributed to 1 of 3 primary exposures: intrauterine exposure to the varicella-zoster virus (VZV), postuterine exposure to wild-type VZV, or exposure due to vaccination with the live-attenuated strain of the virus.[1]

We report a case of HZ in an immunocompetent pediatric patient soon after routine VZV vaccination. We also review the literature on the incidence, clinical characteristics, and diagnostic aids for pediatric cases of HZ.

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The authors report no conflict of interest.

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Case Report

A 15-month-old boy who was previously healthy presented with a red vesicular rash on the right upper cheek of 3 days' duration. The patient was otherwise asymptomatic and had no constitutional symptoms. The patient's mother reported an uncomplicated pregnancy and delivery with no history of maternal VZV infection. There was no known exposure to other individuals with VZV or a history of a similar rash. The patient was up-to-date on his immunizations, which included the VZV vaccine at 12 months of age.

Physical examination revealed vesicles and pustules with an erythematous base on the right zygoma extending to the right lateral canthus and upper eyelid in a dermatomal distribution (Figure). No lesions were present on any other area of the body. One group of vesicles was ruptured with a polyester-tipped applicator and submitted for polymerase chain reaction (PCR) analysis for suspected VZV infection. An ophthalmology evaluation revealed no ocular involvement.

A clinical diagnosis of HZ was made and the patient was started on acyclovir 200 mg 4 times daily for 1 week. At 1-week follow-up, the lesions had cleared and the patient was asymptomatic. The PCR analysis confirmed the presence of VZV.

Although no complications were noted on the ophthalmologist's initial examination or at the follow-up visit, 1 month later the patient's father noted a "cloudy change" to the right eye. The patient had several subsequent evaluations with ophthalmologists and was treated for HZ ophthalmicus

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with acyclovir over the following 10 months. The patient's mother reported eventual clearance of the eye findings without permanent visual sequelae. She stated that the PCR results documenting VZV positivity were extremely helpful for the ophthalmologist in establishing a diagnosis and treatment plan.

Comment

Varicella-zoster virus is 1 of 8 viruses in the Herpesviridae family known to infect humans. It is known to cause 2 distinct disease states: varicella (chickenpox) due to a primary infection from the virus, and HZ (shingles) caused by a reactivation of the latent virus in the dorsal root ganglion, which then travels the neural pathway and manifests cutaneously along 1 to 2 dermatomes.[1] This recurrence is possible in infants, children, and adults via 1 of 3 routes of exposure.

The overall incidence of HZ is lower in children compared to adults, and the risk dramatically increases in individuals older than 50 years. Evidence also shows that exposure to VZV before 1 year of age increases the lifetime risk for HZ.[2,3] Children aged 1 to 18 years who were evaluated for HZ demonstrated a decreased incidence among those who were vaccinated versus those who were not.[4,5] Interestingly, there was an increased incidence of HZ among children aged 1 to 2 years who had been vaccinated. Based on PCR analysis, it was noted that HZ was attributed to the vaccine-related strain of VZV in 92% of 1- to 2-year-old patients.[4]

There is some concern that the varicella vaccination program implemented in 1995 has led to increased rates of HZ. The literature presents mixed reports. Some studies showed an overall increased incidence of HZ,[6,7] and 2 other studies showed no increase in the incidence of HZ.[4,8] More recent studies have demonstrated that vaccination may have a protective effect against HZ.[4-6,9] In a 2013 study in which HZ samples were tested by PCR analysis to determine the strains of VZV that were responsible for an HZ outbreak in children aged 1 to 18 years, the HZ incidence was 48 per 100,000 person-years in patients who were vaccinated versus 230 per 100,000 person-years in patients who were not vaccinated. Among the subset of patients who were vaccinated (n=118), 52% of the HZ lesions were from the wild-type strain.[4]

Clinical Characteristics—The typical presentation of HZ includes grouped vesicles or small bullae on an erythematous base that occur unilaterally within the distribution of a cranial or spinal sensory nerve, occasionally with overflow into the dermatomes above and below, typically without crossing the midline.[8] The most frequent distributions in descending order are thoracic, cranial (mostly trigeminal), lumbar, and sacral. Pain in the dermatome may never occur, may precede, may occur during, or may even occur after the eruption. The initial presentation involves papules and plaques that develop blisters within hours of their development. Lesions continue to appear for several days and may coalesce. The lesions may become hemorrhagic, necrotic, or bullous, with or without adenopathy. Rarely, there can be pain without the associated skin eruption (zoster sine herpete). Lesions tend to crust by days 7 to 10.[8]

Herpes zoster typically affects children to a lesser extent than adults. The disease state often is milder in children with a decreased likelihood of postherpetic neuralgia.[10] However, there are documented cases of severe sequelae secondary to zoster infection in pediatric patients, including but not limited to disseminated HZ,[8] HZ ophthalmicus,[11,12] Ramsay Hunt syndrome,[8] and chronic encephalitis.[8] In the adult population, ocular involvement will present in 33% to 50% of cases that involve the ophthalmic branch of the trigeminal nerve without clinical involvement of the nasociliary branch of the ophthalmic nerve. Involvement of the nasociliary branch will lead to ocular pathology in an estimated 76% to 100% of adult cases.[13,14] It is unknown if this rate is the same in the pediatric population, but it highlights the importance of educating patients and/or guardians about possible complications. It also demonstrates the importance of including HZ in the differential diagnosis for pediatric patients presenting with papular or vesicular skin eruptions, particularly in the area of the ophthalmic branch of the trigeminal nerve.

Diagnosis—Herpes zoster usually is diagnosed based on its clinical presentation. Human herpesvirus 1 or 2 also may present with similar lesions and should be included in the differential diagnosis. To confirm a clinical diagnosis, additional testing may be done. A Tzanck smear historically has been the least expensive and most rapid test. Scrapings can be taken from the base of a vesicle, stained, and examined for multinucleated giant cells; however, a Tzanck smear cannot help in distinguishing herpes simplex virus from VZV. Direct fluorescent antibody testing and viral culture are less rapid but are standard tests that may help with the diagnosis. Direct fluorescent antibody testing can have a high false-negative rate, and viral cultures typically take 2 weeks for completion. These tests have largely been replaced by PCR analysis. Polymerase chain reaction has been the most sensitive test developed yet. With recent advances, real-time PCR, which can be performed within 1 hour in small hospital laboratories,[15] has become more readily available and much more rapid than standard PCR. Further PCR testing can differentiate between the 2 possible infective strains (wild-type vs vaccine related).[16] Real-time PCR is now commonly used as the first-line ancillary diagnostic test after physical examination.[17]

Conclusion

Although uncommon, HZ does occur in immunocompetent children and should be included in the differential diagnosis in children with vesicular lesions. Herpes zoster is a reactivation of VZV and initial exposure may be from the wild-type or vaccine-related strains. Clinicians must be vigilant in their evaluation of vesicular lesions in children even without known varicella exposure. Polymerase chain reaction testing can be helpful to distinguish between herpes simplex lesions and VZV. Polymerase chain reaction testing also may be of benefit to determine the strain of VZV infection.

REFERENCES

1. Myers MG, Seward JF, LaRussa PS. Varicella-zoster virus. In: Kliegman RM, Behrman RE, Jenson HB, et al, eds. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia, PA: Saunders; 2011:1104-1105.
2. Terada K, Kawano S, Yoshihiro K, et al. Varicella-zoster virus (VZV) reactivation is related to the low response of VZV-specific immunity after chickenpox in infancy. *J Infect Dis*. 1994;169:650-652.
3. Takayama N, Yamada H, Kaku H, et al. Herpes zoster in immunocompetent and immunocompromised Japanese children. *Pediatr Int*. 2000;42:275-279.
4. Weinmann S, Chun C, Schmid DS, et al. Incidence and clinical characteristics of herpes zoster among children in the varicella vaccine era, 2005-2009. *J Infect Dis*. 2013;208:1859-1868.
5. Civen R, Lopez AS, Zhang J, et al. Varicella outbreak epidemiology in an active surveillance site, 1995–2005. *J Infect Dis*. 2008;197(suppl 2):S114-S119.
6. Russell ML, Dover DC, Simmonds KA, et al. Shingles in Alberta: before and after publicly funded varicella vaccination. *Vaccine*. 2014;32:6319-6324.
7. Goldman GS, King PG. Review of the United States universal varicella vaccination program: herpes zoster incidence rates, cost-effectiveness, and vaccine efficacy based primarily on the Antelope Valley Varicella Active Surveillance Project data. *Vaccine*. 2013;31:1680-1694.
8. Arikawa J, Asahi T, Au WY, et al. Zoster (shingles, herpes zoster). In: James WD, Berger TG, Elston DM, eds. *Andrews' Diseases of the Skin*. 11th ed. Philadelphia, PA: Saunders/Elsevier; 2011:372-376.
9. Guris D, Jumaan AO, Mascola L, et al. Changing varicella epidemiology in active surveillance sites–United States, 1995-2005. *J Infect Dis*. 2008;197(suppl 2):S71-S75.
10. Petursson G, Helgason S, Gudmundsson S, et al. Herpes zoster in children and adolescents. *Pediatr Infect Dis J*. 1998;17:905-908.
11. Oladokun RE, Olomukoro CN, Owa AB. Disseminated herpes zoster ophthalmicus in an immunocompetent 8-year-old boy. *Clin Pract*. 2013;3:e16.
12. Lewkonia IK, Jackson AA. Infantile herpes zoster after intrauterine exposure to varicella. *Br Med J*. 1973;3:149.
13. Zaal MJ, Völker-Dieben HJ, D'Amaro J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. *Graefes Arch Clin Exp Ophthalmol*. 2003;241:187-191.
14. Harding SP, Lipton JR, Wells JC. Natural history of herpes zoster ophthalmicus: predictors of postherpetic neuralgia and ocular involvement. *Br J Ophthalmol*. 1987;71:353-358.
15. Higashimoto Y, Ihira M, Ohta A, et al. Discriminating between varicella-zoster virus vaccine and wild-type strains by loop-mediated isothermal amplification. *J Clin Microbiol*. 2008;46:2665-2670.
16. Harbecke R, Oxman MN, Arnold, et al. A real-time PCR assay to identify and discriminate among wild-type and vaccine strains of varicella-zoster virus and herpes simplex virus in clinical specimens, and comparison with the clinical diagnoses. *J Med Virol*. 2009;81:1310-1322.
17. Albrecht MA. Diagnosis of varicella-zoster infection. UpToDate website. http://www.uptodate.com/contents/diagnosis-of-varicella-zoster-virus-infection. Updated July 6, 2015. Accessed July 19, 2016.