An Ambiguous Rash in an 11-Year-Old Boy

Daniel R. O'Neilla, Rachel A. Reedyb, c

Abstract

The varicella zoster virus is known for two distinct disease states, the primary varicella zoster virus and herpes zoster. Children who are vaccinated for varicella have a lower incidence of developing herpes zoster than those who have acquired the varicella virus. Regardless, vaccinated children are still at risk for developing herpes zoster and the diagnosis should be considered in patients with a clinical presentation of vesicular and erythematous lesions. An 11-year-old boy, with no significant past medical history, presented to his primary care physician for a rash on his lower back. A thorough history of possible contact exposures was ruled out and further review showed that the boy was up to date on all vaccines. The combination of his physical symptoms and appearance of the rash prompted a culture to be sent to the lab. These results returned positive for varicella zoster DNA on the corresponding polymerase chain reaction (PCR) and proper treatment was initiated. The manifestation of herpes zoster can vary in appearance and does not always present in a standard dermatomal pattern. When clinical diagnosis seems vague, pattern recognition of the distinct vesicular rash shared with a high index of suspicion due to associated symptoms should prompt the culturing of a vesicle for diagnosis of herpes zoster via DNA PCR. Early detection will help hasten appropriate treatment and education to reduce further spread of the virus.

Keywords: Herpes zoster; Varicella zoster; Virus

Introduction

Varicella zoster virus is a contagious subtype of the herpes virus with two disease states: the primary varicella zoster virus, which was once common among children as "chicken pox", and herpes zoster which is also known as "shingles". While the primary varicella zoster virus is not seen as often among children in the USA due to vaccine administration, herpes zoster is still present. Herpes zoster is commonly thought to occur

Manuscript submitted May 23, 2019, accepted June 27, 2019

^aUniversity of Florida College of Medicine, Gainesville, FL, USA ^bUniversity of Florida Health, Gainesville, FL, USA ^cCorresponding Author: Department of Pediatrics, University of Florida Health, 7046 SW Archer Rd, Gainesville, FL 32608, USA. Email: rreedy@ufl.edu

doi: https://doi.org/10.14740/ijcp334

in adults who have previously contracted the varicella zoster virus. However, it can also occur in immunocompetent children who have acquired the varicella virus or in children who have previously received the varicella vaccine. Herpes zoster in adults typically can be diagnosed clinically with a history of varicella and a presentation of the characteristic dermatomal distribution of a vesicular rash. In contrast, diagnosis of herpes zoster in a child remains challenging as not all herpes zoster presentations in children follow the typical long, dermatomal pattern and can instead masquerade as a myriad of other illnesses. If a child presents with vesicular and erythematous lesions, regardless of the patient's adherence to recommended vaccines and/or variation in the dermatological presentation of the rash, herpes zoster should be included in the differential diagnosis. A delayed or missed diagnosis can lead to unexplained persistence and exacerbation of symptoms as well as the possibility of further spreading the virus.

Case Report

An 11-year-old Caucasian male patient, with no significant past medical history, presents to his primary care provider with a rash on the middle, left side of his back for the past 3 days. He describes the rash as a patch of small, red blisters that itch. He reports constant "burning" and "pinching" pain that is exacerbated with palpation. He denies any attempt to alleviate the pain or itching. He denies any new exposures and has not been outside without a shirt on. He is fully vaccinated. He had the chicken pox at 9 months of age; however, he completed his two-dose series of varicella vaccines at 12 months and 7 years of age. All other reviews of symptoms are negative. There is no pertinent social, environmental, or family history.

His temperature is 36.9 °C. His weight is 37.3 kg. No other vitals were obtained. His physical exam reveals a group of a few, small, erythematous, fluid filled vesicles about 25 mm in diameter noted to be on the middle, left side of his back (Fig. 1). There is no tenderness, warmth, swelling, drainage, induration, or fluctuance. His observed neurological exam is within normal limits.

The blistering vesicles are cultured and sent to the lab for evaluation. The patient's culture returned positive for varicella zoster DNA on the corresponding polymerase chain reaction (PCR). With this new information, the diagnosis of herpes zoster is made.

Both the patient and the mother were counseled on the rash being secondary to herpes zoster. By the time the PCR resulted, the rash had been present for over 72 h and antiviral therapy was not indicated. In addition, oral steroids, such as prednisone, were not indicated based on the patient's well tolerated, low pain level. There was no medication given, no labs drawn, and no imaging performed. The family was reminded of supportive care instructions including good hand washing skills and weight-based dosing of ibuprofen as needed for pain control. The herpes zoster rash resolved spontaneously within 4 weeks and has not recurred since the original diagnosis made 2 years prior.

Discussion

In our patient's case, the manifestation of herpes zoster varied in appearance and did not present in a standard dermatomal pattern. Furthermore, he was up to date on all recommended vaccines. Yet, herpes zoster was included in the differential diagnosis due to the appearance of the vesicular rash and was found to be the cause of the rash via DNA PCR. Although no medication was administered at the time the diagnosis was made, the early detection helped prevent further spread of the virus.

Varicella zoster virus (or chicken pox) is a contagious alpha subtype of the herpes virus which is characterized by widespread papulovesicular eruptions with fever and malaise. The virus is known for two distinct disease states, the primary varicella zoster virus and herpes zoster which is a reactivation of the varicella zoster virus. After the initial infection, the virus will lay dormant within a sensory root ganglia and can be reactivated in the form of herpes zoster [1-5]. Herpes zoster will present with severe pain that precedes the typical rash, which is characterized as grouped vesicles or small bullae on an erythematous base with cutaneous manifestation in a dermatomal distribution [6]. The dermatomal pattern of herpes zoster will typically stay within one to two dermatomes and will not cross the midline of the body. The most frequent dermatomal distributions of herpes zoster are in the thoracic, cranial (trigeminal), lumbar, and sacral dermatomes [7]. Pain frequently precedes the rash, but it is possible that pain may never occur, may occur during the eruption, or occur after the eruption. The lesions that appear can last for several days and may coalesce to a larger bulla and become hemorrhagic. Lesions will tend to crust within 7 to 10 days [7].

Herpes zoster is more commonly thought to occur in adults who have previously contracted the varicella zoster virus. However, it can be seen not only in children who have previously acquired the virus but also in children who have been vaccinated. The incidence of herpes zoster found in children under the age of 12 who acquired varicella is 262.1 per 100,000 cases [8]. In children who were vaccinated for varicella zoster virus, there was an incidence of herpes zoster in 93.3 per 100,000 cases [8]. Children who are vaccinated have a lower incidence of herpes zoster than those who acquired varicella but are still at risk for developing herpes zoster [8, 9]. This information shows that herpes zoster in the pediatric population is low in probability but still significant. The reason it is possible for a healthy, immunocompetent child to contract herpes zoster after vaccination lies in the vaccine itself. The varicella zoster vaccine is a live, attenuated virus that induces



Figure 1. A group of a few, small, erythematous, fluid filled vesicles about 25 mm in diameter noted to be on the middle, left side of his back.

cell-mediated immunity in the body. The possibility of a varicella zoster vaccine-strain to induce herpes zoster in immunocompetent children has been noted to occur [10].

The diagnosis of herpes zoster is based on a clinical examination of typical presenting symptoms and patterns. If the diagnosis is ambiguous, further confirmatory results are needed. The most commonly used first-line ancillary diagnostic test is the real-time PCR for varicella DNA [11]. The treatment of herpes zoster is antiviral medications such as acyclovir, valacyclovir, and famciclovir which are all DNA polymerase inhibitors for the virus. If administered within 72 h of the onset of the herpes zoster rash, these antiviral agents have been shown to decrease the duration, pain, and severity of the rash [12]. Furthermore, Prednisone used in conjunction with acyclovir has been shown to further reduce pain [13].

In our case, the patient's rash was not consistent with a typical herpes zoster presentation or distribution pattern. Yet, there was enough distinguishable features in order to merit a PCR for further evaluation including: a history of varicella, grouped vesicles with an erythematous base, a rash within one dermatome that does not cross the midline of the body, and a rash being in a non-distinct location on the body which would be unlikely for an allergic or irritant contact dermatitis.

It is also important to note the year in which the patient contracts the varicella virus if a herpes zoster is included on the differential diagnosis. Existing literature shows that contracting the varicella zoster virus in the first year of life greatly increases the risk of pediatric herpes zoster [14-18]. In fact, children who contracted varicella younger than the age of 2 years have a significantly higher risk of developing childhood herpes zoster compared to a child who contracts varicella after 2 years of age [8].

Even with a reported history of varicella, our patient received the standard vaccination regiment of two doses of the varicella zoster vaccine. Due to the live attenuated nature of the varicella zoster vaccine, exogenous re-exposure to the varicella zoster virus results in enhanced T-cell proliferation. The re-exposure of the varicella zoster virus to an individual who has developed an existing natural immunity from previous varicella exposure leads to a significant increase in proliferating T cells that respond to the varicella zoster virus antigen and varicella zoster virus proteins [19]. Therefore, regardless of prior exposure to the varicella virus, the varicella zoster vaccine should still be administered as it has been seen to increase the proliferating T cells against the varicella virus.

Acknowledgments

None to declare.

Financial Disclosure

The authors have disclosed no financial relationship relevant to this article, no specific funding.

Conflict of Interest

None to declare.

Informed Consent

Not applicable.

Author Contributions

DRO drafted the initial manuscript and reviewed images; RAR critically reviewed and revised the manuscript and participated in direct care of the patient in clinic; Both authors approved the final manuscript as submitted and agreed to be accountable for all aspects of the work.

References

- 1. Na GY. Herpes zoster in three healthy children immunized with varicella vaccine (Oka/Biken); the causative virus differed from vaccine strain on PCR analysis of the IV variable region (R5) and of a PstI-site region. Br J Dermatol. 1997;137(2):255-258.
- 2. Uebe B, Sauerbrei A, Burdach S, Horneff G. Herpes zoster by reactivated vaccine varicella zoster virus in a healthy child. Eur J Pediatr. 2002;161(8):442-444.
- 3. Obieta MP, Jacinto SS. Herpes zoster after varicella vaccination in a healthy young child. Int J Dermatol. 2008;47(6):640-641.
- 4. Ota K, Kim V, Lavi S, Ford-Jones EL, Tipples G, Scolnik D, Tellier R. Vaccine-strain varicella zoster virus causing

recurrent herpes zoster in an immunocompetent 2-yearold. Pediatr Infect Dis J. 2008;27(9):847-848.

- 5. Liang MG, Heidelberg KA, Jacobson RM, McEvoy MT. Herpes zoster after varicella immunization. J Am Acad Dermatol. 1998;38(5 Pt 1):761-763.
- Steven S Agabegi, Elizabeth D. Agabegi. Step up to medicine - Third edition. Copyright © 2013, 2005, 2008 Lippincott Williams & Wilkins.
- 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.
- 8. Wen SY, Liu WL. Epidemiology of pediatric herpes zoster after varicella infection: a population-based study. Pediatrics. 2015;135(3):e565-571.
- 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.
- 10. Peterson N, et al. Herpes zoster in children. Pediatric Dermatology. 2016;98:93-95.
- Albrecht MA, Hirsch MS, Mitty J. Diagnosis of varicella-zoster infection. UpToDate website. http://www.uptodate.com/contents /diagnosis-of-varicella-zoster-virusinfection. Updated July 6, 2015. Accessed July 19, 2016.
- 12. Stankus SJ, Dlugopolski M, Packer D. Management of herpes zoster (shingles) and postherpetic neuralgia. Am Fam Physician. 2000;61(8):2437-2444, 2447-2438.
- Whitley RJ, Weiss H, Gnann JW, Jr., Tyring S, Mertz GJ, Pappas PG, Schleupner CJ, et al. Acyclovir with and without prednisone for the treatment of herpes zoster. A randomized, placebo-controlled trial. The National Institute of Allergy and Infectious Diseases Collaborative Antiviral Study Group. Ann Intern Med. 1996;125(5):376-383.
- 14. Huang JL, Sun PC, Hung IJ. Herpes zoster in infancy after intrauterine exposure to varicella zoster virus: report of two cases. J Formos Med Assoc. 1994;93(1):75-77.
- 15. Baba K, Yabuuchi H, Takahashi M, Ogra PL. Increased incidence of herpes zoster in normal children infected with varicella zoster virus during infancy: community-based follow-up study. J Pediatr. 1986;108(3):372-377.
- Terada K, Kawano S, Yoshihiro K, Miyashima H, Morita T. Characteristics of herpes zoster in otherwise normal children. Pediatr Infect Dis J. 1993;12(11):960-961.
- Feder HM, Jr., Hoss DM. Herpes zoster in otherwise healthy children. Pediatr Infect Dis J. 2004;23(5):451-457; quiz 458-460.
- Brisson M, Gay NJ, Edmunds WJ, Andrews NJ. Exposure to varicella boosts immunity to herpes-zoster: implications for mass vaccination against chickenpox. Vaccine. 2002;20(19-20):2500-2507.
- 19. Arvin AM. Cell-mediated immunity to varicella-zoster virus. J Infect Dis. 1992;166(Suppl 1):S35-41.