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A rare presentation of shingles (herpes zoster) in an infant

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ABSTRACT

Herpes zoster (HZ) in children is very rare. We report a case of HZ in an infant which responded well to the treatment. HZ should also be considered in an infant with vesiculo-bullous lesion in a dermatomal distribution.

Key words: *Dermatome, Herpes zoster, Immunity, Infant*

Primary varicella tends to occur in childhood, whereas herpes zoster (HZ) is a disease of adults, with most of the patients being older than 45 years [1,2]. It is very rare in healthy children <5 years with the exception of infants who are infected in utero. In literature, very few cases of shingles have been reported in infants. We report a case of an infant who presented to us with shingles.

CASE REPORT

A 1-year-old boy presented with fever and grouped vesiculo-bullous lesions over left axilla, left side of upper chest, and back of 5 days duration. Mother had varicella infection during the 4th month of gestation treated with oral acyclovir and resolved completely. All antenatal ultrasonograms were normal. Baby was born by full term lower segment cesarean section delivery (indication: Failed induction) with birth weight of 2.7 kg. No anomalies were present at birth. Postnatal period was uneventful with no history of varicella or contact with varicella. The child had normal development history.

Physical examination revealed grouped vesiculo-bullous lesions with an erythematous base and serous discharge in the left side of chest, axilla, and back in the dermatomal distribution of T4, associated with itching (Fig. 1). Systemic examination was within normal limits. Blood examination revealed leukocytosis. Tzanck smear showed multinucleated giant cells. Varicella zoster virus (VZV) IgG report awaited. The child was treated with oral acyclovir 80 mg/kg day in 5 divided doses for 7 days, and other supportive measures and child improved (Figs. 2 and 3a and b).

DISCUSSION

VZV causes primary infection, manifested as varicella (chickenpox), and can reactivate after establishing latency in the dorsal (spinal) sensory ganglia or cranial nerve ganglia to cause reactivated infection, manifested as HZ [3,4]. The development of HZ is associated with a decline in cell-mediated immunity due to aging, an immunosuppressive illness or treatment, or an immature cell-mediated immune system [4,5]. HZ is more common in older adults; however, it can occur in healthy infants, children, and young adults, in whom the disease is usually milder [6].

HZ is uncommon among children. In classic studies, its incidence in the pediatric age ranged from 0.2 to 0.74 cases/1000 person-years [7,8]. A large population-based study from Iceland in the early 90s reported a higher incidence rate up to 1.6 cases/1000 person-years [9].

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 acquisition of HZ in healthy immune-competent children in early childhood or during intrauterine exposure has been attributed to the immaturity of the immune system [10].

Terada et al. [10] observed that immunological status before primary infection with VZV is important and affects the reactivation of VZV. They noticed that, 6-7 weeks after primary varicella, infants had a lower response of VZV-specific cellular and humoral immunity compared with children who had an infection at older ages (>1 year). In another study [11], 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 1st year of life as a risk factor for the development of HZ in otherwise healthy children.

Terada et al. 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 [12]. From these data, Terada et al. [12]

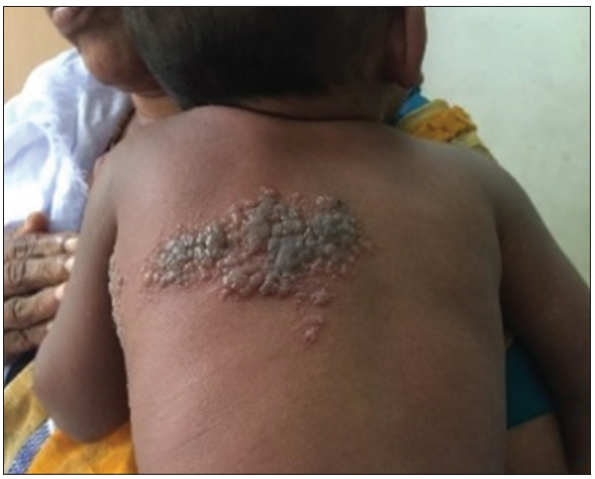

Figure 1: Vesiculo-bullous lesions of herpes zoster in the T4 dermatomal distribution

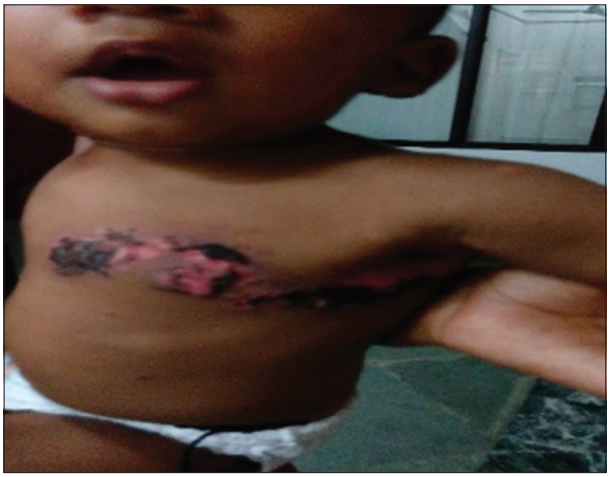

Figure 2: Crusting lesions

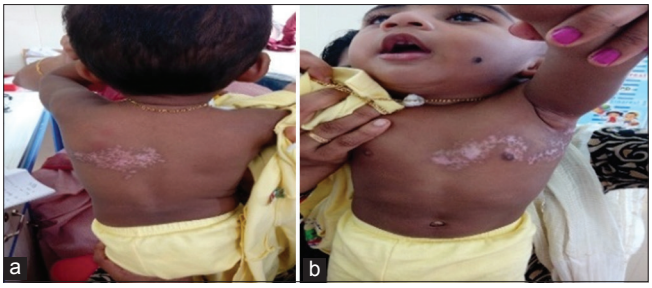

Figure 3: (a and b) Healed lesions after acyclovir treatment

hypothesized that a "subclinical reactivation" puts infants with a history of primary varicella at risk for HZ. In 69% of infantile HZ cases (i.e., <12 months of age) reported in the literature, the initial event could be traced to maternal varicella during pregnancy. Dobrev [13] 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 HZ within the 1st 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 HZ within the 1st year of life [13].

In infant, however, HZ typically demonstrates the skin lesions only, without acute pain or postherpetic neuralgia [14,15]. In both, the skin lesions typically develop from central to peripheral and do not cross the midline [16]. During this vesicular stage, the condition is contagious, as virus is found at the lesion sites [16] and vesicles and erosions are susceptible to bacterial super-infection. After several days, the vesicles will erode and crust, heralding resolution [16].

HZ is usually diagnosed clinically by the characteristic presentation of the disease, and laboratory testing is not usually necessary [15]. However, it has been shown that herpes simplex infections may have a zosteriform presentation. Two situations in which laboratory confirmation can provide helpful information are to confirm the diagnosis of varicella before initiation of antiviral therapy in a patient who presents with unusual symptoms and to confirm susceptibility or immunity in exposed pregnant women. A common confirmatory laboratory test, Tzanck smear, can be done to support the clinical diagnosis, demonstrating a viral cause. However, to differentiate a possible HZV infection from that of herpes simplex, cultures or other antigen-based testing must be done [4]. In general, the treatment of HZ is indicated to limit the spread, duration, and bacterial super-infection of the skin lesions, to reduce the acute symptoms of pain and malaise, and to prevent the development of postherpetic neuralgia and ophthalmological complications in HZ ophthalmicus whenever these complications exist or are likely to develop.

Infants do not suffer postherpetic neuralgia, so oral antiviral use has been questioned. Some authors have recommended that oral antiviral use in immunocompetent children with HZ be reserved for the more severe cases, as with ophthalmic involvement [14]. Other authors have used oral antivirals as a general approach [15]. In the uncomplicated infant case, whether treated with antivirals or not, resolution of the condition is quick and free of sequelae [14,15].

CONCLUSION

The incidence of infantile HZ is very rare. However, the possibility of HZ also should be considered in an infant with vesiculo-bullous lesion in a dermatomal distribution, even if history is not available.

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