Herpes zoster in infancy

Sir,

Varicella and herpes zoster are caused by the same virus, varicella zoster virus (VZV). Varicella is the primary infection, whereas herpes zoster is the result of reactivation of residual latent infection, usually of sensory neurons, infected by viremia of chicken pox.[1] The occurrence of herpes zoster is rare in early childhood and still rarer in infancy.[2,3] Varicella contracted by a pregnant woman in the last trimester of pregnancy may result in varicella in the immediate neonatal period or herpes zoster during infancy; the latter usually takes a benign course.[4] We

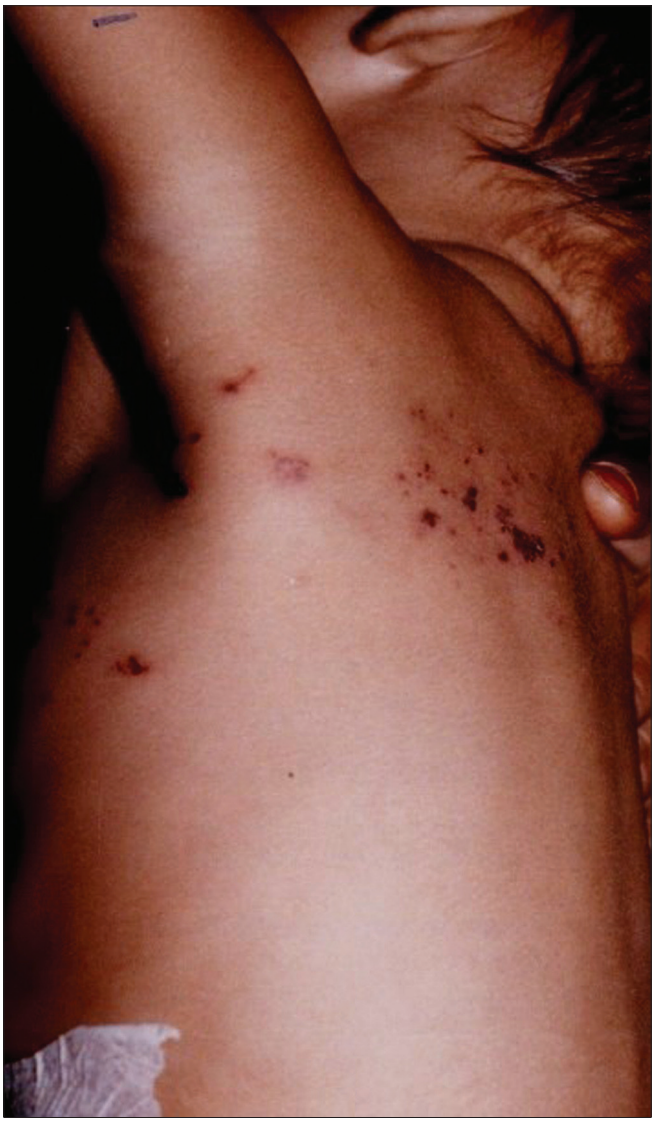

Figure 1: Typical lesions of herpes zoster affecting fourth left thoracic dermatome (T_4)

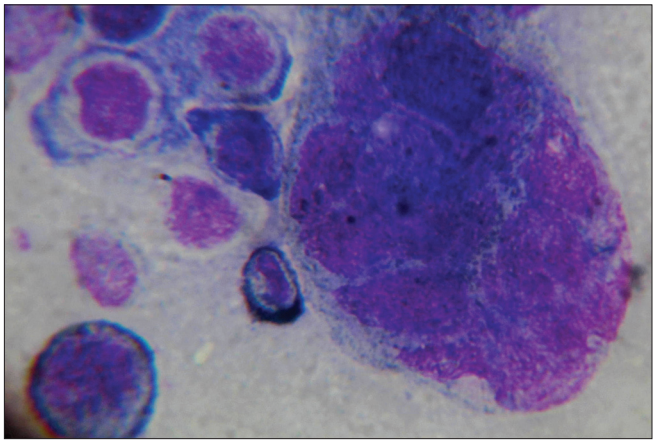

Figure 2: Tzanck smear showing typical multinucleated giant cell and a few acantholytic cells Giemsa, 1000x

report a 10-month-old infant with herpes zoster involving fourth left thoracic dermatome (T_4), whose mother had varicella during seventh month of gestation.

A10-month-old female child having intermediate type of anorectal malformation with recto vaginal fistula since birth was referred to us from the pediatric surgery unit for vesicular skin eruptions on trunk since 2 days. The examination revealed multiple, tense, grouped vesicular lesions, strictly confined to the left half of trunk in the distribution of T_4 [Figure 1]. Tzanck smear from lesions revealed multinucleated giant cells [Figure 2]. There was no past history of varicella in the infant. Her mother suffered from varicella during seventh month of gestation. The child had undergone multiple corrective surgical procedures for anorectal malformation including colostomy at birth, Pena's posterior sagittal anorectoplasty (PSARP) at 6 months, and colostomy closure at 9 months of age. Herpes zoster developed 2 weeks after the closure of colostomy. The general health of the child was unaffected. Developmental milestones were normal. There was no evidence of immunosuppression. Enzyme-linked immunosorbent assay (ELISA) for human immunodeficiency virus (HIV) 1 and 2 in child and parents was nonreactive. She was treated with topical fusidic acid cream. The lesions healed completely in 10 days with slight hypopigmentation.

Herpes zoster in infancy or early childhood generally results from reactivation of VZV infection acquired either in utero or during early infancy while partially protected by maternal antibodies.[2] The majority of cases of childhood zoster occur after the age of 5 years. In a study of 22 children, only one was an infant.[4] There have been occasional reports of infantile herpes zoster in the literature.[1,3-7] In most reported cases, there was a history of maternal varicella during gestation suggesting that the initial viral exposure occurred in utero. None of the reported cases had evidence of immunosuppression. The course of herpes zoster was benign in all with rapid recovery without any sequelae. Similar features were evident in our case too.

The progressively declining titers of protective maternal antibodies and repeated operative trauma could have possibly served as triggers in precipitating zoster at a relatively young age in our patient. Trauma has been suggested to be one of the triggering factors precipitating zoster.

The case is being reported on account of the rarity of herpes zoster during infancy.

Lalit Kumar Gupta, Ashok Kumar Khare, Asit Mittal, C. M. Kuldeep

Department of Dermatology, Venereology and Leprosy, R.N.T. Medical College, Udaipur, Rajasthan, India

Address for correspondence: Dr. Lalit Kumar Gupta,
Department of Dermatology, Venereology and Leprosy,
3-A, Sai Villa, Madhuvan, Opp. G.P.O, Udaipur,
Rajasthan - 313 001, India.
E-mail: lalitanj@yahoo.com

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