Varicella zoster virus (VZV) causes varicella as a primary infection. After the primary infection, the virus remains latent within the sensory dorsal root ganglia and can reactivate as herpes zoster (HZ). It is most common in adults, especially elderly and immunocompromised patients, but rarely occurs in children. Herpes zoster is most often seen in individuals previously infected with VZV, but it also has occurred in individuals without known varicella infection, possibly because these individuals had a prior subclinical VZV infection. We present a case of HZ in an otherwise healthy 18-month-old boy with no known varicella exposure who received the VZV vaccine at 12 months of age.

Keywords
Varicella Zoster, Herpes Zoster, Vaccination, Child

INTRODUCTION

Herpes Zoster (HZ) infection is caused by the reactivation of endogenous latent VZV. In general, the disease reactivation occurs in the adult population when resistance against this virus declines due to selective cell-mediated immune suppression or there is a generalized immune suppression.

CASE REPORT

An otherwise healthy 18-month-old boy presented to the dermatology clinic with a rash that began 7 days prior on the right leg. The patient’s mother denied that the child had been febrile and noted that the rash did not appear to bother him in any way. The patient was up-to-date on his vaccinations and received the first dose of the varicella series 6 months prior to presentation. He had no personal history of varicella, no exposure to sick contacts with varicella, and no known exposure to the virus. He was otherwise completely healthy with no signs or symptoms of immunocompromise. Physical examination revealed grouped vesicles on an erythematous base on the right leg (Figure). There were no other pertinent physical examination findings. Polymerase chain reaction assay for VZV and HSV from skin swabs was ordered. The patient was prescribed acyclovir 20 mg/kg every 6 hours for 5 days. Laboratory testing revealed a positive result for VZV on polymerase chain reaction and a negative result for HSV. The majority of the patient’s lesions had crusted after 2 days of treatment with acyclovir, and the rash had nearly resolved 1 week after presentation. Subsequent evaluation with a complete blood cell count with differential and basic metabolic profile was normal. Levels of IgG, IgA, IgE and IgM also were normal.

DISCUSSION

Herpes Zoster (HZ) infection, also known as “shingles,” is caused by the reactivation of endogenous latent VZV that resides in a sensory dorsal root ganglion usually after primary infection with VZV which causes chickenpox. It usually develops after several years of the primary infection of
chickenpox or vaccination with chickenpox vaccine. In general, the disease reactivation occurs in the adult population when resistance against this virus declines due to selective cell-mediated immune suppression or there is a generalized immune suppression\(^2\). Although HZ is predominantly an infection of the adult population, the chances of pediatric infection are more if the patient had chickenpox infection in the first year of life or had an in-utero exposure to the virus\(^3\). Usually the course of herpes is milder in children, and the mean duration of the disease is 1–3 weeks. Though lesional pain and itching may be present, post herpetic neuralgia has been rarely reported. The first line of therapy in childhood HZ is oral acyclovir given at a dose of 20–40 mg/kg body weight four times a day\(^4\). There has been a recent trend in the rise of HZ cases in childhood. Vaccination with live attenuated virus could be one of the reasons for this rise in the number of cases\(^5\). Children developing HZ de novo without the development of chickenpox can be possibly explained by the fact that the episode of chickenpox is mild and goes unnoticed by the parents and the treating physician. Mild episode of chickenpox is attributed to the general course of mild chickenpox in children, and the symptoms and disease duration is further ameliorated by immunizing the child. Also, the episode of chickenpox in childhood, if not associated with classical signs and symptoms, can be diagnosed as a different viral exanthema and is not documented. This rising incidence of HZ in otherwise healthy children may be due to acquiring primary varicella infection in utero or in infancy, wherein the body's immunity is not fully developed\(^6\). Herein we present a case of HZ in an otherwise healthy 18-month-old boy with no known varicella exposure who received the VZV vaccine at 12 months of age. There are very few cases of a one-year-old shingles without any underlying disease, such as our case. We present this case because we think that as varicella vaccine is a live attenuated virus, there is a possibility that a vaccine recipient can develop HZ like this case.

REFERENCES