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CASE REPORTS

CONCOMITANT PRESENTATION OF VARICELLA AND HERPES ZOSTER – IS IT POSSIBLE?

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ABSTRACT

A five-years-old boy was referred to the emergency department due to a 12-hour evolution of skin lesions similar to generalized varicella and concomitant herpes zoster in the C6 dermatome. Without history of prior disease or vaccination against varicella-zoster virus. There was an outbreak of varicella in nursery school. He was treated with oral acyclovir, with complete resolution. To our knowledge, it is the first published case of concomitant varicella and herpes zoster due to wild virus. There are only four similar cases described in the international literature, all related to vaccination against varicella-zoster, which is not present in this case. The mechanism responsible for the simultaneous manifestation of varicella and herpes zoster is not known, however a theory under discussion is that the initial viremia during primary infection may cause immediate viral replication in the sensory ganglia, causing cutaneous manifestations and pain in the surrounding dermatomes.

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Introduction

The varicella-zoster virus (VZV), which belongs to the herpes virus family, is highly contagious and has a worldwide distribution. It can cause 2 clinically distinct forms of disease: primary infection that causes widespread self-limited vesicular lesions, which characterizes varicella (chickenpox) and the endogenous reactivation of the latent VZV, which results in localized and painful skin lesion that characterizes the herpes zoster (shingles). Although varicella primary infection confers lifetime immunity to varicella, in most cases VZV can remain latent in the sensory nerve ganglia and reactivate later in life leading to herpes zoster. (1) We report a case of a clinical diagnosis of concomitant varicella and herpes zoster, in a child without prior history suggestive or apparent of immunodeficiency or immunosuppression. To our knowledge, this is the first published case of concomitant varicella and herpes zoster due to wild virus. There are only four similar cases described in the international literature, all related to vaccination against VZV. (2-4).

Case Report

A 5-years-old male child presented with 12-hour evolution of vesicular lesions and pain in the left forearm, with posterior generalization of the vesicles to the rest of the integument and mucous membranes. He had intense pruritus and a single fever episode. He had been immunized with measles, mumps and rubella (MMR) vaccine 4 days earlier and there was slight redness at the vaccination site. He had not received varicella vaccine. There was no history of frequent or recurrent infections, complications of vaccination or previous clinical diagnosis of varicella. There was an outbreak of varicella in the child's preschool. The child had a personal history of febrile convulsions and atopic eczema. Relevant family history of Crohn's disease in the mother was obtained.

On examination, he had generalized maculo-papulo-vesicular lesions (more than 25), including in the scalp and perineum (Fig. 1A) and maculo-papulo-vesicular lesions on the left C6 dermatome (forearm) with increased pain sensitivity to touch (Fig. 1B). Clinically the lesions were compatible with generalized varicella and concomitant left C6 dermatome herpes zoster. He was discharged with oral acyclovir (80 mg/kg/day, 4 times a day) for 5 days and advised general skin care to avoid infection. On the eighth day of illness all the lesions crusted.

The analytical study, performed on that same day, did not reveal a primary or secondary immunodeficiency (HIV I/II ELISA was negative). It presented slight lymphopenia of all the subpopulations, without inversion of the CD4/CD8 ratio [CD3 - 1024 cell/uL (67.5%), CD4 - 518 cell/uL (34.8%), CD8 - 366 cell/uL (24.6%), CD56 - 110 cell/uL (7.1%) and CD19 - 381 cell/uL (24.6%)]. There was also a slight hyper IgE (626 KU/L, normal values 0-70 KU/L) and eosinophilia (9.6%, normal values 0-2.4%), though serum IgG (1280 mg/dL), IgA (165 mg/dL), IgM (167 mg/dL) were normal without criteria for hyper IgE syndrome and in the absence of other stigmata of this syndrome. C3 (144 mg/dL) and C4 (28 mg/dL) were normal. Serum IgM (6.5 mg/dL) and IgG (6.7 mg/dl) anti-VZV antibodies were positive. The patient had slight hyperesthesia up to 2 weeks followed by complete resolution without sequelae, scars or post-herpetic neuralgia. Lymphocyte subsets repeated after 6 months were normal.

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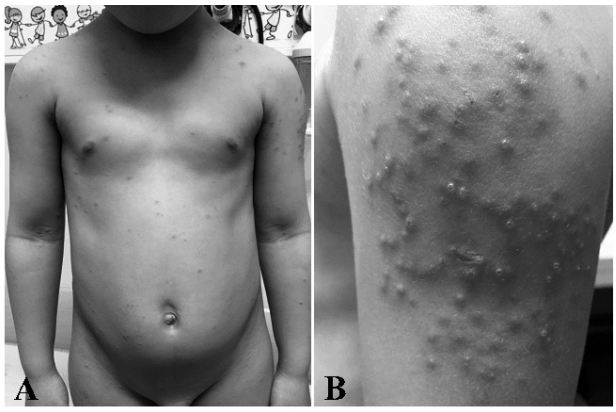
Figure 1. Maculo-papulo-vesicular lesions generalized in the trunk, upper limbs, thighs, perineum (A) and on the left C6 dermatome, left forearm (B).



Discussion

Although varicella is a common disease in countries without high coverage of vaccination against VZV, herpes zoster is rare in children and adolescents without immunocompromised state, with an incidence of 42 per 100,000 person/years. The occurrence of varicella in the first year of life is the most frequent risk factor in the pediatric population, especially if maternal varicella occurred during pregnancy. (5) Herpes Zoster may also occur in healthy children who have been vaccinated against VZV, although this risk appears to be equal to that of unvaccinated children who acquire immunity from infection with wild-type virus. (6) Some studies have shown that a higher incidence of herpes zoster may be associated with low seroconversion after primary infection with VZV. (7)

The pathogenesis of the herpes zoster is still poorly understood, however reactivation is thought to occur associated with transient viremia by hematogenous spread of ganglion virus, resulting from the decline of cellular immunity against VZV. (8) Cases in children with widespread dissemination of VZV, from a small area of herpes zoster or an affected ganglion, are described in the absence of an obvious rash, which can be easily mistaken for varicella. (1,8) These can be classified into: (a) Herpes zoster with aberrant vesicles - in which transient viremia may allow some virus to reach the skin prior to its control by the host immune system, resulting in disseminated vesicles (up to 25) of the affected dermatomes; or (b) Disseminated herpes zoster - viremia is persistent, usually due to some underlying immunosuppression, resulting in disseminated vesicles (> 25). (5,8)

Serological techniques may be used to determine susceptibility to VZV infection and document the increase in antibody titers after primary infection. Acute infection can be confirmed by the positivity of serum specific IgM anti-VZV antibodies titers and serum IgG becomes detectable several days after varicella onset, reaching peak values after 2 to 3 weeks. (9) Although our patient does not present a history of previous varicella, we cannot exclude whether he had an earlier infection with subclinical or mild manifestations that went unnoticed. If so, this could be a case of a widespread herpes zoster. The increase in IgM antibodies in this hypothesis would be secondary to disseminated herpes zoster viremia, which may stimulate an immune response and increase these titers. However, this hypothesis seems unlikely, since we did not identify an immunodeficiency or immunosuppression that may explain a widespread herpes zoster.

The mechanism responsible for the simultaneous manifestation of varicella and herpes zoster is not known, however a theory under discussion is that the initial viremia during primary infection may affect the sensory ganglia, with immediate viral replication inside them, causing cutaneous manifestations and pain in the surrounding dermatomes. (4)

We cannot ignore the fact that MMR vaccination occurred only 4 days before the onset of the described condition and that there was varicella outbreak in the child's preschool, raising the question whether this concomitant manifestation of varicella and herpes zoster could have been caused by cell-mediated immune depression resulting from MMR vaccination. The current recommendations warn against the administration of varicella vaccine within 28 days after MMR vaccine. This recommendation is based on the observed reduction in responsiveness to varicella vaccine after MMR vaccine and the reported increased risk of breakthrough disease (defined as the occurrence of varicella disease with the wild-type VZV occurring more than 42 days after varicella vaccination). The cell-mediated immune depression, similar to that observed after natural measles infection, might explain this finding. (10-12) It is also known that the analysis of lymphocyte populations can be altered by the VZV, which is in favor of the changes observed. (7) Lymphocyte subsets were repeated 6 months after the initial infection, with normalization of the results, conforming the absence of immunodeficiency.

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Compliance with Ethical Standards

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Conflict of Interest : None

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