ATYPICAL PRESENTATION OF RECURRENT VARICELLA ZOSTER VIRUS INFECTION: A CASE REPORT AND REVIEW OF THE LITERATURE

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Abstract. Recurrent varicella infection is rare but has been reported in immuno-compromised patients. We present a patient with atypical recurrent varicella infection who had disseminated central crusting papular lesions without dermatomal distribution. Serology showed previous varicella zoster virus (VZV) infection and the lesions were positive for VZV DNA, consistent with recurrent VZV infection. Atypical recurrent varicella infection is probably an under-recognized condition. VZV infection should be considered in the differential diagnosis of ecthyma-like lesions in an immunocompromised host.

Keywords: recurrent varicella, atypical presentation, immunocompromised host, ecthyma-like lesions, central necrosis papules

INTRODUCTION

Classical varicella infection usually occurs once in a lifetime, most often in childhood with multiple stages of macules, papules, vesicles and crusts (Mc-Crary et al, 1999). The rash usually has cephalocaudal progression (McCrary et al, 1999). Recurrent varicella may present with atypical cutaneous lesions of the same stage with central necrosis lesions (Nikkels et al, 2003). Prolonged healing time and absence of a cephalocaudal progression are found, especially in immunocompromised patients (Nikkels et al, 2003). Recurrent varicella can cause serious complications, such as pneumonia (Fraisse et al, 1998).

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CASE REPORT

A 52-year-old woman presented to Siriraj Hospital, Bangkok, Thailand, with fever and diarrhea for 3 weeks. She was admitted to the hospital and diagnosed with having disseminated tuberculosis and pneumonia. While in the hospital she developed respiratory failure and catheter related septicemia. She had no HIV infection. During hospitalization she developed ervthematous papules with central necrosis all over her body, but predominantly on the extremities and in no dermatomal distribution (Figs 1 and 2). The lesions were similar to eachother and had their onset while she was still febrile. She had a history of varicella infection at a young age and had no contacts with varicella infected patients during the weeks prior to developing the skin lesions. Her past medical history was significant for non-Hodgkin's lymphoma for 11 years previously, for which she had been treated with chemotherapy and she was in remission.



Fig 1–Erythematous papules with central necrosis predominant on the extremities (A. Right thigh; B. left thigh; C. close up of the lesion).



Fig 2–Large vesicles seen after central necrosis lesions developed.

A Tzanck smear of the vesicles was performed, which showed multinucleated giant cells. A polymerase chain reaction (PCR) of the vesicle fluid was positive for varicella zoster virus (VZV) DNA. Her serum VZV immunoglobulin (Ig) M antibody test performed on the first day that lesions appeared was negative, but a serum specific VZV IgG antibody test

was positive. The presence of disseminated lesions without a dermatomal distribution along with the laboratory findings lead to the diagnosis of atypical recurrent varicella. The patient was treated with intravenous acyclovir (500 mg every 8 hours for 7 days). The lesions gradually healed over several days. Unfortunately, the patient died due to sepsis.

DISCUSSION

VZV infection usually occurs once during a life time (McCrary *et al*, 1999). After primary VZV infection, the virus becomes latent in the sensory ganglion, but may reactivate due to a decrease in immunity resulting in herpes zoster (Kinchington *et al*, 2012). On reactivation, the virus migrates from the dorsal root ganglia to the skin, where it produces a cytolytic infection in the keratinocytes resulting in herpes zoster (McCrary *et al*, 1999).

Recurrent VZV infection is rare but has been reported in immunocompromised patients, such as children with HIV infection, renal transplant recipients, critically ill patients and elderly

patients with malignant hemopathies (Von seidlein, 1996; Rothwell *et al*, 1999; Nikkels *et al*, 2003; Hagiya *et al*, 2013). Recurrent varicella differs from primary infection by presentation and clinical course. Cutaneous lesions may have no typical vesicles, are larger in diameter, frequently have central necrosis like ecthyma lesions and are usually in the same stage (Nikkels *et al*,

2003). A long duration between primary varicella and its recurrence, a prolonged healing time of the lesions and the absence of cephalocaudal progression, may be seen in immunocompromised patients (Nikkels *et al*, 2003). Recurrent varricella can be associated with serious complications, such as pneumonia, especially in immunocompromised patients (Fraisse *et al*, 1998).

Recent VZV infection is associated with an elevated anti-VZV IgM first and then later an elevated anti-VZV IgG. A ngative anti-VZV IgM and a positive anti-VZV IgG indicated a previous VZV infection (Nikkels *et al*, 2003). Our patient had anti-VZV IgG, which points to a previous VZV infection. The finding of VZV DNA from the vesicular lesion demonstrates a current infection. Since the serology was tested early in the course of infection, the anti-VZV IgM was still negative.

Patients with VZV infection should be isolated, because they are highly contagious. Transmission occurs via direct contact with lesions or respiratory droplets (McCrary et al, 1999). Treatment of recurrent VZV infection is the same as treatment of primary infection in immunocompromised patients: high-dose intravenous acyclovir (10 mg/kg daily) (Nikkels et al, 2003; Hagiya et al, 2013). The duration of acyclovir therapy in recurrent VZV infection is unclear, so we recommend treating patients for 7-10 days, similarly to the duration of treatment in immunocompromised patients. To prevent recurrences of VZV infection among severely immunocompromised patients who had a life threatening VZV infection, secondary prophylaxis should be considered (Fraisse et al, 1998).

Our patient presented with atypical recurrent VZV infection with disseminated papular lesions and central crusting without the dermatomal distribution seen in herpes zoster. The patients developed

vesicular lesions later, suggesting VZV infection. The VZV serology showed a previous VZV infection and the lesions were positive for VZV DNA. These findings suggest recurrent VZV infection. Atypical recurrent varicella is probably an underrecognized condition. VZV infection needs to be considered in the differential diagnosis of ecthyma-like lesions in immunocompromised patients. This case highlights the need to be aware of atypical varicella infection with unusual skin signs, especially in immunocompromised patients.

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