

Case of Disseminated Vesicles of Herpes Zoster Developing One Day before the Onset of Local Eruption in a Hospitalized Immunocompromised Patient

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Disseminated herpes zoster is not rare in immunocompromised patient. It is defined as at least 20 lesions in multiple dermatomes that occur within a week of the onset of local eruption. Herein, we report that a case of disseminated vesicles of herpes zoster (HZ) that developed one day before the onset of local eruption in an immunocompromised patient. A 44 year-old Japanese male, who had been in the hospital with acute myelocytic leukemia, developed disseminated hemorrhagic vesicles of 5 to 10 mm in diameter. The next day, grouped vesicles, including hemorrhagic vesicles erupted on the right side of the second to third cervical (C2–C3) dermatomes. At this point, the diagnosis was made as disseminated herpes zoster. The activation of varicella-zoster virus (VZV) is believed to be due to waning of VZV-specific memory T cell responses. In our case, the memory immunity to VZV which had been increased by last episode of HZ might affect on the appearance of skin eruptions.

Key words: disseminated herpes zoster, generalized herpes zoster, VZV-specific memory T cells, reinfection, recurrence

INTRODUCTION

Disseminated herpes zoster is extremely rare in immunocompetent patient, but is as high as up to 40% in immunocompromised patient [1]. It is defined as more than 20 vesicles outside the primary and immediately adjacent dermatomes [1]. Disseminated vesicles follow local eruption within a 5–8 day interval [2]. Herein, we report that a case of disseminated vesicles of herpes zoster that developed one day before the onset of local eruption in an immunocompromised patient. Although such a case is occasionally seen clinically, to our knowledge, no such case has been reported in English medical literature previously.

CASE REPORT

A 44 year-old Japanese male who had been in the hospital with acute myelocytic leukemia visited our outpatient clinic with skin lesions which had begun on the same day. We noticed disseminated hemorrhagic vesicles of 5 to 10 mm in diameter on his trunk and extremities (Fig. 1a). He had experienced varicella in his early childhood and herpes zoster (HZ) four months ago. We diagnosed him as varicella reinfection by Tzanck smear, which revealed multinucleated giant cells, and started treatment with oral valacyclovir. The next day, grouped vesicles of 5 mm in diameter with surrounding erythema developed on along the right side of the second to third cervical (C2–C3) dermatomes (Fig. 1b). At this point, the diagnosis was made as disseminated HZ. All the vesicles had become crusted within a week. A skin biopsy was performed

from the vesicle on patient's left shoulder on the first day. Histopathological examination showed intraepidermal vesicles (Fig. 2a) with multinucleated giant cells (Fig. 2b) which were positive with monoclonal antibody against varicella-zoster virus (VZV). Antibody titers for VZV at day 10 suggested previous or past infection; VZV-IgG was positive and VZV-IgM was negative. The hematologic tests revealed pancytopenia: WBC, 1,300/ μ l; RBC, $2.52 \times 106/\mu$ l; Hb, 7.2 g/dl; Plt, $3.3 \times 10^4/\mu$ l, biochemistry tests and urinalysis revealed normal except for the following: TP, 5.5 g/dl; Alb, 3.0 g/dl; CRP, 1.92 mg/dl.

DISCUSSION

Patients with lymphoma/leukemia have a 1.9-fold higher risk of HZ event than the controls [3]. In immunocompromised patients, local eruption often becomes necrotic with delayed healing and subsequent scarring. Disseminated HZ cases typically are treated with intravenous acyclovir 10 mg/kg every 8 hour for 5–7 days [4]. In our case, local eruption had been mild and all vesicles had become crusted within a week with oral valacyclovir therapy. He was relatively mild case when it is compared to typical disseminated HZ.

The activation of VZV is believed to be due to waning of VZV-specific memory T cell responses [1]. Memory immunity to VZV can be boosted by the occurrence of HZ as well as vaccination. Whereas the local eruption results from reactivation of latent virus in nerve ganglion cells, disseminated vesicles probably results from hematogenous spread of the virus

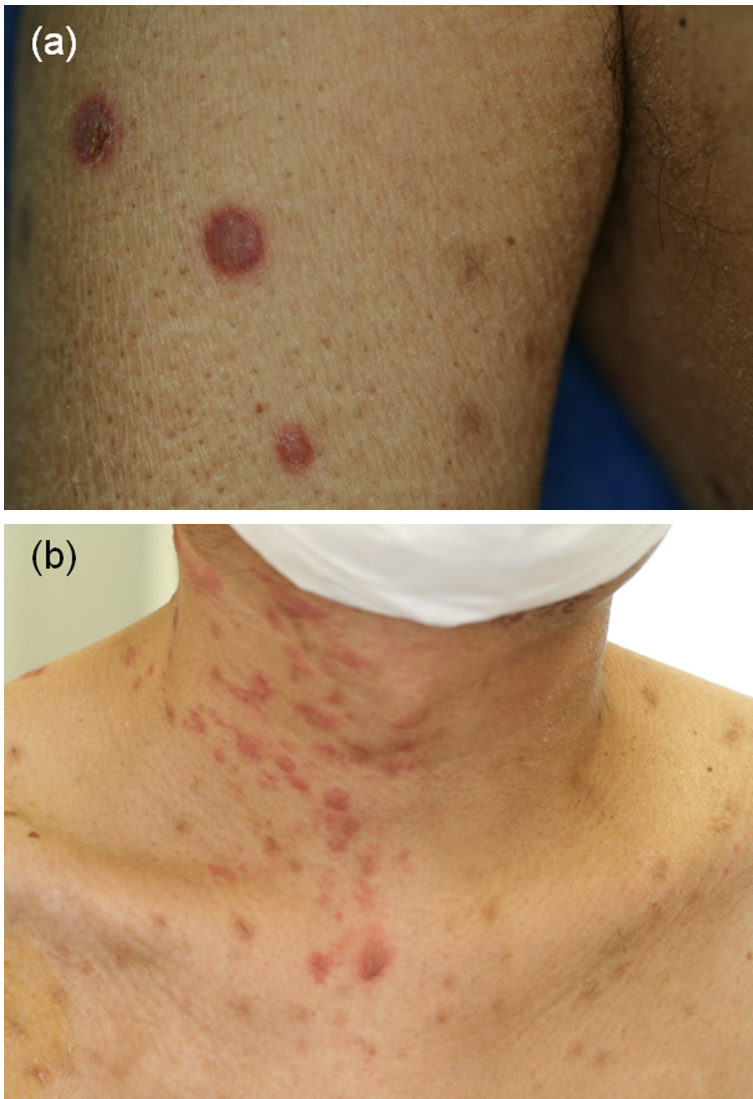


Fig. 1 (a) Disseminated hemorrhagic vesicles of 5 to 10 mm in diameter had developed on the trunk and extremities. (b) Grouped vesicles of 5 mm in diameter with erythema erupted on the right side of C2–C3 dermatomes on the second day.

[1]. Therefore typically disseminated vesicles follow local eruption within a 5–8 day interval. VZV-specific memory T cells are thought to control the latest stages of reactivation rather than replication of latent VZV genomes in the ganglia [5]. In our case, the patient had experienced HZ four months ago. Last episode of HZ may have increased the memory immunity to VZV. Although it did not prevent the recurrence of HZ, it may have reduced the severity of HZ and delayed the appearance of the local eruption. Furthermore, disseminated vesicles may result from hematogenous spread of the replicated VZV which cannot be controlled by VZV-specific memory T cells in the ganglia.

We hope our case could contribute to a better understanding of diversity of development of disseminated vesicles in HZ.

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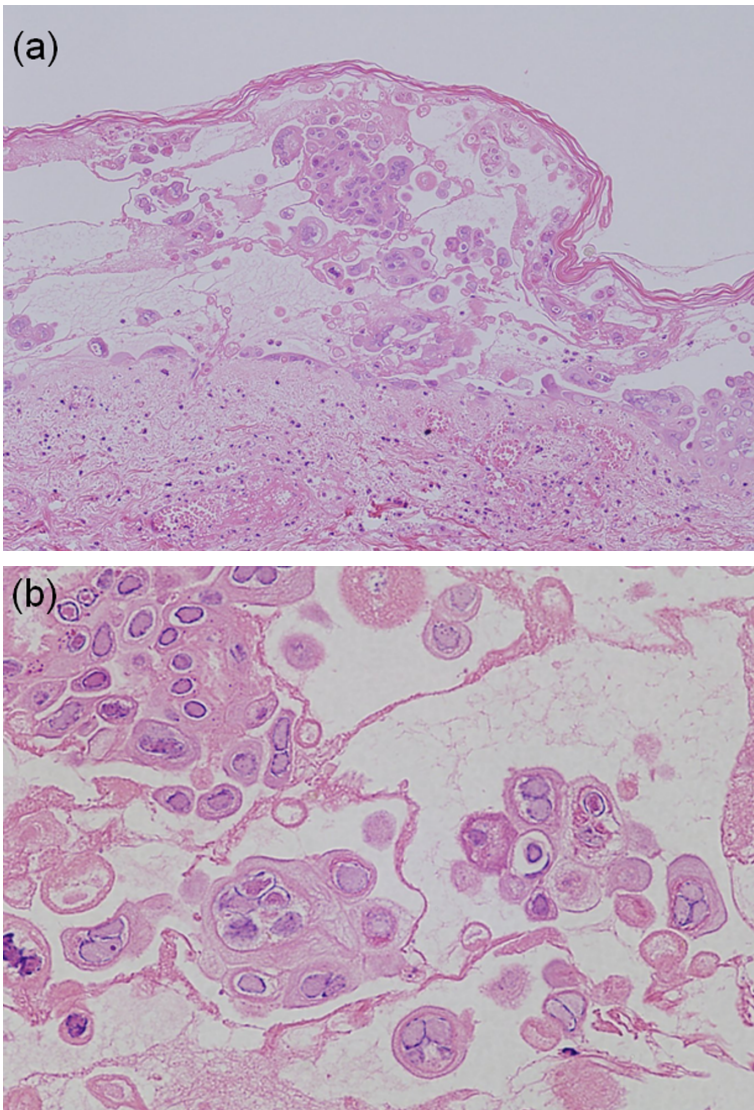


Fig. 2 Histopathological examination of skin biopsy showed intraepidermal vesicles (a) with multinucleated giant cells (b) (hematoxylin-eosin, original magnifications: [a] $\times 10$; [b] $\times 40$).