

Severe Kaposi's varicelliform eruption in mild atopic dermatitis complicated by rhabdomyolysis: A case report

Dear Editor,

Rhabdomyolysis is a clinical syndrome characterized by the disruption of striated muscle tissue, and may arise from various infections.¹ There are a few reports that herpes simplex virus (HSV) infection contributes to rhabdomyolysis.²⁻⁵ Here, we present a case of severe Kaposi's varicelliform eruption (KVE) in atopic dermatitis (AD) complicated by rhabdomyolysis.

A 19-year-old Japanese man had a history of herpes labialis and untreated mild AD. Six days before his first visit to our department, he experienced intense facial itching. On the next day, erythema, vesicles, and erosions appeared on his face and neck. He was referred to our department, and these eruptions spread widely with disturbance of consciousness. No signs of herpes keratitis were observed.

The patient showed a body temperature of 40.2°C, a Glasgow Coma Scale score of 11 (E3 V3 M5), and neck rigidity. Physical examination revealed widespread erosion, crusts, and edematous swelling from the scalp to upper trunk. Aggregated vesicles were found at margins of skin lesions. The conjunctiva became hyperemic, and the labial mucosa was covered by hemorrhagic crusts (Figure 1A). Other areas presented with atopic dry skin.

Laboratory tests showed leukocytopenia, thrombocytopenia, highly elevated C-reactive protein of 26.2 mg/dL, creatine phosphokinase (CPK) of 7109 IU/L, and lactate dehydrogenase of 1028 IU/L. Thymus and activation-regulated chemokine, creatinine and blood urea nitrogen were within the normal range. Anti-HSV IgG and IgM antibodies were positive, and vesicular fluid samples were positive for HSV type-1 (HSV-1) antigen. Urine examination revealed an increased urinary myoglobin of 56 ng/mL (normal: <10 ng/mL). Thus, severe KVE in AD complicated by rhabdomyolysis was diagnosed. As our patient did not have any other suggestive history or well-known causes of rhabdomyolysis, we assume rhabdomyolysis was caused by HSV-1 infection.

Treatment was initiated with blood transfusion according to burn therapy protocols, and with acyclovir at a dose of 1500 mg daily for 2 weeks in consideration of possibility of herpes meningitis. Mucocutaneous lesions were treated with saline lavage, topical use of dimethyl isopropylazulene ointment 0.033%, and nonadhesive dressings. Topical use of ACV ophthalmic ointment was used for the eyes.

Thereafter, his consciousness was regained rapidly, and mucocutaneous lesions and laboratory abnormalities were improved gradually. Renal disorders did not occur during the course. Finally, he recovered without any neurological complications, and the skin erosion had been epithelized (Figure 1B).

KVE typically affects children and young adults with a history of skin disease, particularly uncontrolled severe AD.⁶ It is rare, but HSV infection in severe AD patients can lead to multiorgan complications, such as rhabdomyolysis, hemophagocytic syndrome, and encephalitis.^{4,7-9} In this case, however, mild AD led to KVE and even rhab-



FIGURE 1 A, Clinical presentation on the initial visit. Widespread erosion, crusts, and swelling were seen over the scalp, face, neck, upper trunk and proximal upper arm. The conjunctiva became hyperemic, and the labial mucosa was covered by hemorrhagic crusts. Other areas of the skin presented atopic dry skin. B, Clinical presentation 2 wk after the initiation of the treatment. Erosion had epithelized, and a mild degree of erythema was seen on the face

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domyolysis. It should be kept in mind that mild AD can potentially afford serious HSV infection and associated multiorgan systemic complications at an early stage as seen in our case. Therefore, early recognition of signs for severe complications even in mild AD and prompt administration of sufficient antiviral is important in clinical practice.

DECLARATION


Informed consent: The patient's consent to publish this case was obtained.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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