The Journal of dermatology (2017.10) 44(10):1180-1181.

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(Letter to the Editor) **JDE-2016-0739.R1**

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Word count: 478/500 words

Tables and figures: 1 figure

Running head: KVE with extensive skin erosion and sepsis (36/50 characters)

Key words: Kaposi's varicelliform eruption, atopic dermatitis, skin erosion, steroidphobia, sepsis, creatinin kinase

Dear Editor,

Atopic dermatitis (AD) can be accompanied by various viral/bacterial infections, such as herpes simplex virus (HSV), poxvirus and staphylococcal infection, because of the impaired barrier/immunological function (1). Among them, HSV-induced Kaposi's varicelliform eruption (KVE) usually shows diffusely scattered papules, vesicles and pustules on a particular body site including face, and extensively erosive and ulcerated skin lesions are relatively rare. Here, we report an AD-based severe KVE case presenting with diffusely spread erosive and ulcerative lesions and sepsis.

A 41-year old Japanese woman presented with a seven days-history of febrile painful skin lesions consisted of crusted erosive vesicular papules and extensively eroded diffuse erythema on the face, trunk and upper arms accompanied by high grade fever (39°C) (Fig a-c). She had been suffered from AD without proper topical treatment because of her steroidphobia. Blood test revealed leukocytopenia (960/μl), elevated C-reactive protein (16.53mg/dl), positive procalcitonin, elevated aspartate transaminase/alanin transaminase ratio (126/52 IU/L), lactate dehydrogenase (758 IU/L), serum creatinine (0.53 mg/dl) and creatin kinase (CK) (1099 IU/L), suggesting septic and possible rhabdomyolytic condition. Serum anti-HSV IgM antibody level was increased from 0.33 (day 1) to 1.04 (index) (day 24), but anti- HSV IgG antibody showed high titer at day 1

(334 index), suggesting re-activation of HSV. *Pseudomonas aeruginosa* was dominantly detected from the erosive skin lesion. Computed tomography denied any cause of the fever including internal malignancy. These clinical and laboratory findings led to the diagnosis of KVE developed on insufficiently controlled AD accompanied by secondary infection of *Pseudomonas aeruginosa*. The febrile condition and the eruptions were recovered by acyclovir (5mg/kg three times per day for 5 days), cefozopran hydrochloride (3g per day for 8 days) and topical petrolatum ointment in two weeks.

KVE is mainly caused by HSV and characterized by disseminated papulovesicular eruption. It frequently occurs on pre-existing skin disease with impaired barrier function, such as AD (1). A detailed retrospective analysis of AD accompanied by KVE suggests that an earlier age at onset of AD, a higher level of serum IgE, and insufficient control of AD can be risk factors of AD accompanied by KVE (2). This case was infantile onset and had not been sufficiently controlled because of her steroidphobia. Diffusely disseminated KVE is relatively rare, and can more frequently be observed in Darier's disease or pemphigus (3, 4) than in AD. In this case, inadequate treatment against her AD was considered as the main cause of severe KVE resulted in serious secondary bacterial infection. In addition, elevated CK was transiently detected in this case, because rhabdomyolysis was also induced by severe infection such as bacterial sepsis (5).

KVE, one of the most common complications of AD, can be successfully treated with anti-viral agents in most cases. However, as KVE can induce more serious systemic conditions, dermatologists should properly educate every AD patient on the risk of insufficient skin care.

Conflict of interest None declared

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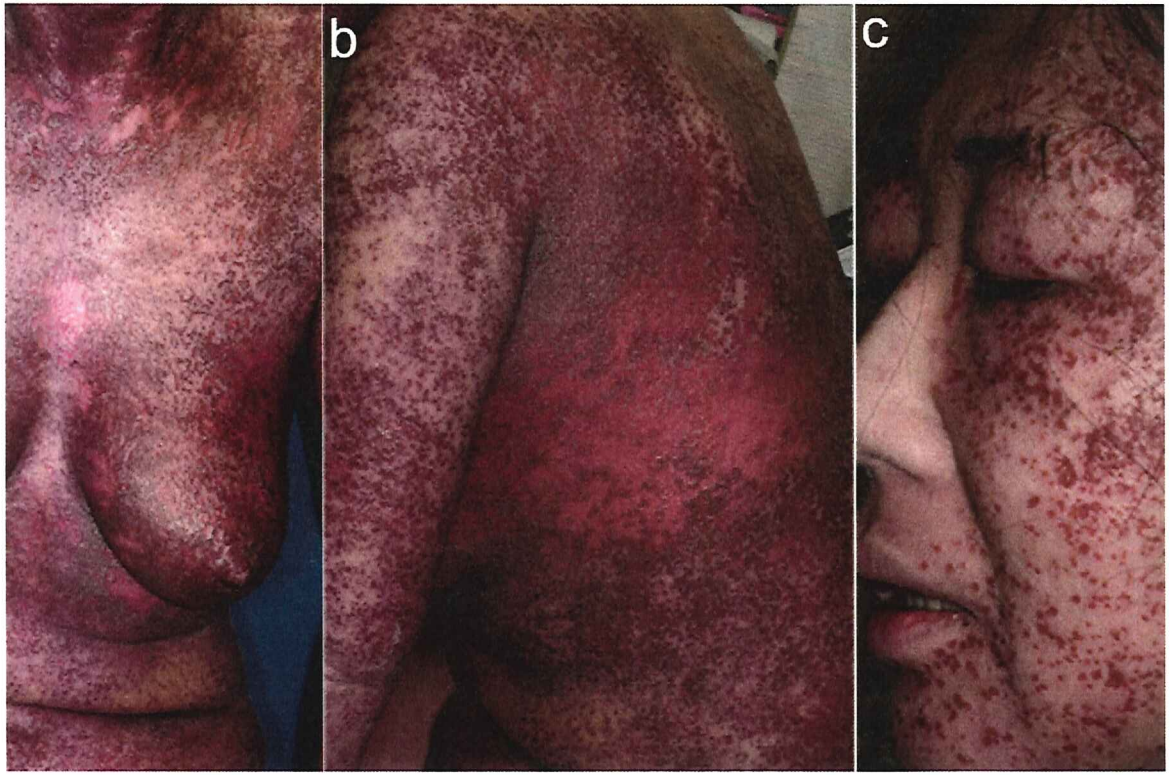
Figure legends

Figue 1

Clinical manifestations of the case (a-c). Extensive eroded/ulcerated lesions on the trunk, proximal aspect of the extremities (a and b), and the face (c).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