

Kaposi's Varicelliform Eruption Complicating Lichen Simplex Chronicus

Hee Jung Kim, M.D.¹, Jeong Hyun Yun, M.D.¹, Ji Yeoun Lee, M.D.¹, Mi Kyeong Kim, M.D.²,
Tae Young Yoon, M.D.¹

*Departments of Dermatology¹ and Internal Medicine², School of Medicine & Medical Research
Institute, Chungbuk National University, Cheongju, Korea*

Kaposi's varicelliform eruption results from a widespread infection of a virus to skin damaged by predisposing dermatoses. It is usually a manifestation of a primary herpes simplex virus type 1 (HSV-1) infection in a child with atopic dermatitis, but other skin conditions can be complicated. Here, we report an unusual case of KVE arising from lichen simplex chronicus on the male genital area.

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Key Words: Kaposi's varicelliform eruption, Lichen simplex chronicus

INTRODUCTION

Kaposi's varicelliform eruption (KVE) refers to a widespread cutaneous infection with a virus which normally causes localized or mild vesicular eruptions, occurring in a patient with predisposing skin disease¹. KVE is usually caused by primary herpes simplex virus type 1 (HSV-1), and on rare occasions by HSV-2, vaccinia virus (eczema vaccinatum), and Coxsackie A16 virus. KVE is commonly observed in individuals with atopic dermatitis. Other reported predisposing conditions include Darier's disease, mycosis fungoides, bullous diseases, contact dermatitis, burns, and so on¹⁻²⁵. Recently KVE complicating tinea cruris⁵ and rosacea⁶ have been reported. However to the best of our knowledge, KVE complicating lichen simplex chronicus (LSC) has not been reported in the English literature.

CASE REPORT

A 38-year-old healthy male was admitted to the emergency room with painful skin lesions on the genital area, which developed rapidly within a few days. Physical examination revealed superficial scattered erosions and crusts on the pubis and scrotum, and numerous monomorphic eruptions of umbilicated, dome-shaped vesicles, crusts, and purulent exudates on the groin, perineum, and perianal area (Fig. 1). The lesion was developed on the brownish lichenified patch. Underlying skin showed thickened appearance with accentuation of the surface markings. Regional lymph nodes were palpated. He complained of a mild febrile sensation, but there was no fever. He had a history of long-standing pruritic eczematous lesions on the groin and the scrotum for more than ten years, but he had never received any treatment. According to the patient, pruritus was aggravated recently. He denied a recent sexual contact. There was no personal or family history of atopic dermatitis. But he gave a history of previous episodes of recurrent herpes simplex labialis. Routine laboratory work-up was normal. A potassium hydroxide (KOH) microscopic examination revealed no fungal elements. A Gram stain, bacterial culture and fungus culture of the lesion were performed, but no pathogen was revealed. Histopathologic examination showed

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Reprint request to: Tae Young Yoon, M.D., Department of Dermatology, School of Medicine & Medical Research Institute, Chungbuk National University, 410 Sung-bong-ro, Heungdeok-gu, Cheongju 361-763, Korea. Tel: 82-43-269-6369, Fax: 82-43-266-1698, E-mail: tyoon@chungbuk.ac.kr

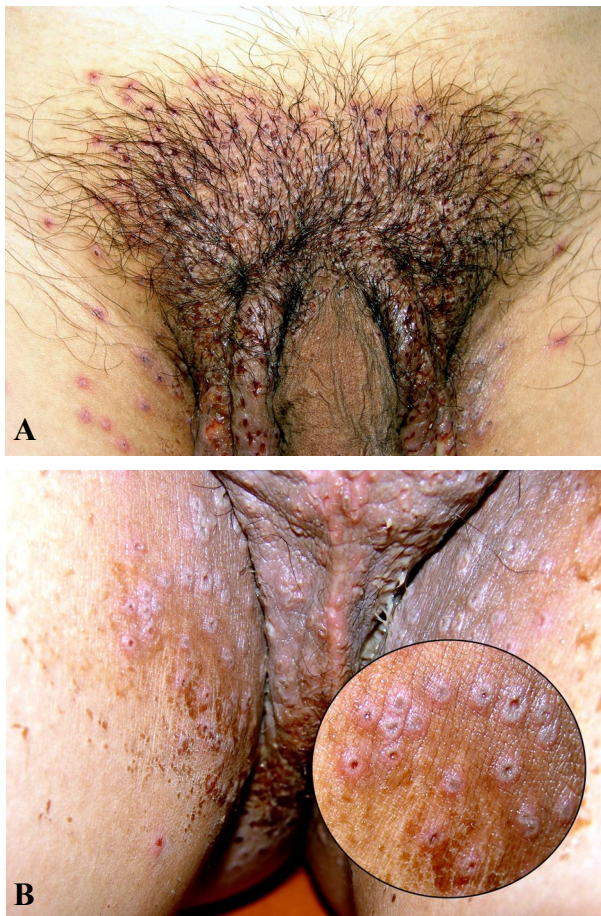


Fig. 1. (A) Scattered erythematous papules with central erosions and crusts are seen on the brownish lichenified patches of the pubis and scrotum. (B) characteristic monomorphic, grouped, umbilicated vesicles and extensive purulent exudates are seen on the groin, perineum, and perianal area.

central necrosis, ballooning degeneration, and multinucleated giant cells in the epidermis. In the dermis, a dense mixed inflammatory infiltrate was observed. There was no evidence of vascular damage (Fig. 2).

Although we did not perform PCR or culture for confirming herpes virus, we made a diagnosis of KVE based upon typical clinical and histological findings. We started intravenous acyclovir (750 mg/day) treatment for 5 days, and oral cephadrine (2000 mg/day) treatment for 7 days to prevent a secondary infection. About two weeks later, KVE was completely cleared. A KOH examination was repeated, and again no fungal elements were revealed.

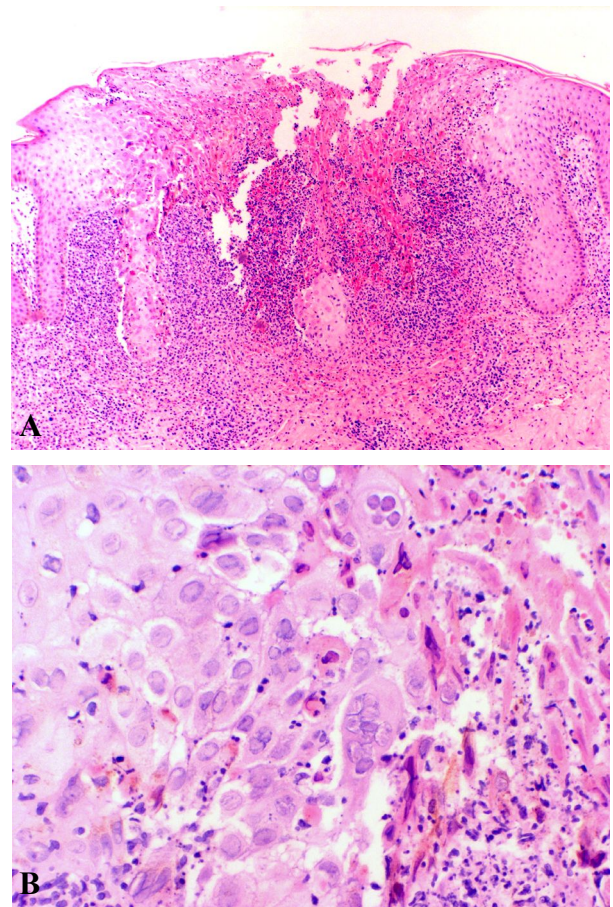


Fig. 2. (A) Biopsy specimen exhibits central necrosis, ballooning degeneration, and multinucleated giant cells in the epidermis. A dense inflammatory infiltrate is observed in the dermis (H & E, $\times 40$). (B) note multinucleated giant cells and balloon cells (H & E, $\times 200$).

DISCUSSION

KVE refers to a widespread cutaneous infection with a virus that normally causes localized or mild vesicular eruptions and, occurs in a patient with a predisposing skin disease. Various dermatologic diseases have been reported as a preexisting skin disease of KVE (Table 1)¹⁻²⁵. Patients with KVE frequently present with a diagnostic discrete, distinctly monomorphic eruption of closely grouped, painful, dome-shaped vesicles, accompanied by fever, malaise, and regional lymphadenopathy¹. The head, neck, and upper part of the body are most frequently affected²⁶. In milder cases, the lesions often are restricted to the upper part of the body. However,

Table 1. Reported various diseases complicated by Kaposi's varicelliform eruption

Atopic dermatitis ¹
Wiskott-Aldrich syndrome ²
Seborrheic dermatitis ²
Irritant contact dermatitis ³
Allergic contact dermatitis; Parthenium dermatitis ⁴
Tinea cruris ⁵
Rosacea ⁶
Pityriasis rubra pilaris ⁷
Senile erythroderma ⁸
Congenital ichthyosiform erythroderma ⁹
Ichthyosis vulgaris ¹⁰
Pemphigus vulgaris ¹¹
Pemphigus foliaceus ¹²
Bullous pemphigoid ¹³
Familial benign chronic pemphigus ¹⁴
Darier's disease ¹⁵
Grover's disease ¹⁶
Burn (second-degree) ¹⁷
Autografted skin ¹⁸
Laser resurfacing ¹⁹
Peribuccal dermabrasion ²⁰
Cutaneous T-cell lymphoma ²¹
Mycosis fungoides ²²
Sézary syndrome ²³
Multiple myeloma ²⁴
Without active eczema ²⁵

the lower half is involved most in generalized cases²⁷. But in our patient, KVE lesion was unusually localized to the genital area.

Lichenification is a pattern of cutaneous response to repeated rubbing or scratching. The underlying stimulus for the development of LSC is pruritus. Common triggers of the pruritus include psychological distress, and local environmental problems such as heat, sweating, and excess dryness. LSC may also develop on underlying, pre-existing pruritic dermatoses. The usual sites are the nape of the neck, scalp, upper thighs, vulva, pubis or scrotum, and extensor surface of the forearms.

The case of KVE complicating tinea cruris was reported recently⁵. We initially suspected tinea cruris as a preexisting skin disease, but the KOH examination revealed no hyphae. In addition, tinea cruris generally spares the scrotum, but our patient had the lesion on the scrotum. It was thought that

hyphae could be missed in KOH examination because of the superimposed KVE. Therefore, when the KVE lesion cleared completely, we took KOH examination again but still couldn't find any hyphae. So we concluded LSC as his predisposing dermatitis. To the best of our knowledge, this is the first report of KVE complicating LSC in the English literature.

In differential diagnosis, we considered herpes progenitalis. Herpes progenitalis is usually caused by herpes simplex virus type 2 (HSV-2) with sexual contact. Herpes progenitalis manifests by grouped, 1-to-2 mm sized vesicles and shallow erosions, usually located over the glans and shaft of the penis²⁸. But in our case, the lesion involved an extensive area that also spared the glans and shaft of the penis. The lesions were umbilicated papules and vesicles rather than tiny vesicles.

The exact pathogenesis of KVE remains unknown, but impaired barrier functions of the epidermis and decreased cellular or humoral immunity have been proposed as important factors in pathogenesis²⁹. Here, we report a patient who developed KVE on the LSC area. In our case, the patient was healthy but his chronic scratching that resulted in epidermal defects could contribute to induction of KVE. It is not clear whether he had a reactivation of herpes simplex infection or a newly acquired one from an exogenous source. The history of recurrent herpes simplex seems to favor the former possibility.

During the last few years the incidence of KVE has increased²⁷. Despite the proven availability of antiviral therapies, KVE still remains a dermatological emergency. Because systemic viremia can lead to multiple organ involvement and a secondary bacterial infection with subsequent septicemia, this condition may be potentially life threatening. It must be recognized that any skin condition that results in epidermal barrier function loss-even LSC-can cause KVE.

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