A Case of Kaposi's Varicelliform Eruption Associated with Nitrofurantoin Induced Allergic Contact Dermatitis: Case Report

Nitrofurantoin Kullanımının Sebep Olduğu Allerjik Kontakt Dermatit ile İlişkilendirilmiş Kaposi’nin Variselliform Erüpsiyonu Olgusu

ABSTRACT Kaposi’s varicelliform eruption describes the cutaneous dissemination of the viral infection superimposed on a pre-existing dermatosis such as atopic dermatitis, seborrheic dermatitis, ichthyosis, Darier’s disease, pemphigus foliaceus, mycosis fungoides and contact dermatitis. Most commonly, it is caused by a disseminated Herpes virus (HSV) infection in patients with atopic dermatitis. The incidence of Kaposi’s varicelliform eruption has increased since 1980, likely secondary to the increased incidence of HSV infections. It is a severe and potentially life-threatening condition. Mortality ranges from 1 to 9 percent, with reported rates as high as 75% before the advent of effective antiviral drugs. To date, the pathophysiology of Kaposi varicelliform eruption remains unclear. In this report, we present a case of Kaposi’s varicelliform eruption developing on allergic contact dermatitis followed by nitrofurantoin ointment. To our knowledge, this is the first reported case associated with nitrofurantoin induced allergic contact dermatitis.

Key Words: Kaposi varicelliform eruption; dermatitis, allergic contact; nitrofurantoin


Anha touring Kelimeler: Kaposi variselliform döküntü; dermatit, allerjik kontakt; nitrofurantoin


Apo’s varicelliform eruption (KVE) is the name given to a distinct cutaneous lesions caused by frequently herpes simplex virus (HSV) type 1, HSV type 2 and less frequently coxsackie virus A16 or vaccinia virus in patients with preexisting dermatosis. Since the original description of KVE by Moriz Kaposi in 1887, more than 20 skin diseases have been described in relation to it including atopic dermatitis, Darier’s disease, various bullous diseases of the skin particularly patients receiving immunosuppressive therapy, Hailey-Hailey disease, ichthyosis vulgaris and other inflammatory dermatosis. KVE has also been described in association with allergic contact dermatitis (ACD), rosacea, pemphigus foliaceus and follo-
wing dermatologic medications such as topical tacrolimus for pre-existing eczematous palmar dermatitis, after dermabrasion and vaccination with BCG.3–6

We describe here a 62 year old man of KVE, associated with nitrofurantoin induced ACD which to our knowledge is the first reported case in the literature.

CASE REPORT

A 62-year-old man, presented for evaluation of painful vesiculopustular eruption on his face and body along with malaise and fever. A month prior to presentation, he underwent mammalian cist operation and was prescribed nitrofurantoin postoperatively. After nitrofurantoin therapy significant right breast swelling, pain and generalized itchy, erythematous, honey colored crusted rash on his body have been developed. He was diagnosed as ACD due to nitrofurantoin ointment. He had been managed by withdrawing nitrofurantoin and treated with topical corticosteroids and antihistamines without any improvement.

Following couple of days, the patient was noted to have a sudden onset of marked erythema and vesicular lesions on his face and body. He had no known history of atopic dermatitis and recurrent herpesvirus infection. There was no personal or familial history of atopic diseases.

On admittance, the patient had a temperature of 39 °C, painful burning skin sensation, general malaise and itching. Dermatologic examination revealed widespread clusters of umbilicated vesiculopustules and eroded vesicles across the face and upper trunk (Figure 1, 2). Honey-colored crusting some certain areas, suggesting staphylococcal super-infection, and bilateral inguinal lymphadenopathy were also present. Ophthalmologic examination was normal. The rest of physical examination was unremarkable.

Tzanck preparation of facial vesicles showed multinucleated giant cells. Routine laboratory examinations including biochemical analysis and complete blood counts were within normal limits except for increased sedimentation rate (37 mm/h) and C-reactive protein (27.3 mg/dL).

A diagnosis of KVE on pre-existing ACD was suspected and it was confirmed positive Tzanck smear. After diagnosis of KVE, he was treated with oral acyclovir 200 mg five times a day for 10-day course. For honey-colored crusting lesions, mupirocin 2% ointment twice daily was applied. On the second day of antiviral therapy, patient’s fever was dramatically decreased. All skin lesions rapidly responded to the treatment and complete healing was observed within 10 days.

DISCUSSION

KVE is clinically characterized by sudden appearance of monomorphic, umbilicated, grouped vesiculopustular lesions on the face, particularly around the eyes, neck, axillae, chest and upper ex-

FIGURE 1: Umbilicated vesiculopustules on patient’s trunk.

FIGURE 2: Close up images of eroded vesiculopustules on patient’s trunk.
The eruption is usually accompanied by malaise, fever and regional lymphadenopathy. The incubation period is usually between 3 - 10 days. It is a potentially life-threatening skin condition that arises in preexisting dermatosis most commonly atopic dermatitis.\(^4\,5,6,7\) Frequently, a delay in diagnosis occurs because the eruption is confused with the underlying skin disease until the characteristic umbilicated vesiculopustules appear. In some cases, it may progress to fulminant, life-threatening viral infection and may cause severe sequelae including herpes keratitis, disseminated infection with visceral involvement and death. Mortality rates of up to 10 percent being reported before antiviral therapy was available and it was usually primarily caused by bacterial superinfection and bacteremia. It must be recognized and managed appropriately for avoidance of sequence.\(^6\,8\) In our patient the vesicles rapidly became pustular, later erosions formed and these erosions got secondarily infected. These secondarily infected honey colored crusted lesions were successfully managed by topical mupirocin ointment.

The pathogenesis of KVE remains to be elucidated. There is no plausible explanation for disseminated HSV infection occurring in dermatosis. Impaired barrier function of the epidermis has been proposed to explain the development of the eruption.\(^1\,4\,6,9\) Patients with immunodeficiency and atopic dermatitis are well known to be more susceptible to herpetic infection.\(^3\) Defective cytokine secretion and decreased cell-mediated immunity in skin affected by atopic dermatitis and other skin diseases also appear to play a role in the pathogenesis of KVE. However no specific abnormality in immune function has been demonstrated to date.\(^2\,4\,10\) Paradise et al. reported a case of KVE in patient with eczematous palmar dermatitis treated with tacrolimus. They suggested that suppressing the local skin immunity due to application of tacrolimus and epidermal barrier disruption caused by eczematous dermatitis may have been predisposing factors for KVE.\(^3\) Likewise, in our patient epidermal barrier impairment due to eczematous skin lesions and topical corticosteroid medications promoting local cutaneous immunosuppression and skin fragility may have contributed to the widespread dissemination of HSV infection.

The occurrence of KVE in ACD has not been fully explained. However defective skin barrier function caused by ACD might have played essential role in the pathogenesis of KVE. In conclusion, KVE should be considered in the differential diagnosis of diffuse, painful vesiculopustular, and crusted lesions accompanied by fever, in patients having pre-existing ACD.

### REFERENCES