

Mini outbreak of Kaposi's varicelliform eruption in skin ward: A study of five cases

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ABSTRACT

Background: Kaposi's varicelliform eruption (KVE) represents widespread cutaneous herpes simplex virus (HSV) infection in patients with preexisting dermatoses. Occasionally, this infection can present as a nosocomial infection in skin wards, if adequate bed-spacing and barrier nursing methods are not followed. We are reporting five cases of KVE; four cases acquired the infection in a makeshift ward after admission of the first case in May 2005, due to the renovation work of the regular skin ward. **Aim:** The purpose of this study is to create clinical awareness about this uncommon dermatologic entity and to stress upon the importance of bed-spacing and barrier nursing in skin wards. **Methods:** Five cases of KVE, three females and two males with different primary dermatoses (pemphigus foliaceus – one, pemphigus vulgaris – two, paraneoplastic pemphigus – one and toxic epidermal necrolysis – one) were included in this study. Diagnosis was made clinically and supported with Tzanck smear and HSV serology. All the cases were treated with oral acyclovir. **Results:** Four out of five cases of KVE recovered with treatment, one case of extensive pemphigus vulgaris with KVE succumbed to death. **Conclusion:** Mini outbreaks of KVE can occur in skin wards with inadequate bed-spacing and overcrowding of patients. Therefore adequate bed-spacing, barrier nursing and isolation of suspected cases are mandatory to prevent such life-threatening infections.

Key Words: Kaposi's varicelliform eruption, Nosocomial infection, Barrier nursing, Bed-spacing

INTRODUCTION

Kaposi's varicelliform eruption (KVE) is a distinct cutaneous eruption caused by *herpes simplex virus* (HSV) Type 1 and Type 2 and rarely by *coxsackie A16 virus* and *vaccinia virus* over a preexisting dermatosis.^[1] Since 1980, there has been an increase in the reporting of cases of KVE from all over the world, this is partly due to increased prevalence of HSV infections in the community.^[2] The most common predisposing condition for KVE is atopic dermatitis,^[3] but KVE has been described in various dermatoses like pemphigus foliaceus,^[3] chronic benign familial pemphigus,^[4] Darier's disease,^[5] Grover's disease,^[6] multiple myeloma,^[7] psoriasis,^[2,8] lupus

vulgaris,^[8] allergic contact dermatitis,^[9] seborrhoeic dermatitis,^[2] neurodermatitis,^[2] pityriasis rubra pilaris,^[2] ichthyosis vulgaris,^[2] rosacea^[1] following facial tacrolimus treatment for atopic dermatitis^[10] and after laser resurfacing.^[11]

Kaposi's varicelliform eruption is diagnosed mostly clinically and investigations like Tzanck smear and HSV serology are only supportive. The polymerase chain reaction of the blister fluid for HSV is confirmatory. Kaposi's varicelliform eruption is clinically characterized by sudden appearance of monomorphic, umblicated, grouped vesiculopustular lesions on the face, particularly around the eyes, neck, axillae, chest and upper extremities. The incubation period is usually

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between three to 10 days. The eruption is usually accompanied by fever and regional lymphadenopathy. The lesions heal with minimal scarring in two to six weeks. Secondary bacterial infection, viremia and multiorgan involvement are the important causes for mortality. Ocular complications like blepharitis, conjunctivitis, keratitis, uveitis and loss of vision can also occur.^[1] Before the advent of acyclovir therapy the mortality in cases of KVE was very high, now with specific acyclovir treatment, it is around 10%.

No case of KVE was reported in King George Hospital, Visakhapatnam for the last 30 years. Extensive renovation work was taken up during the study period in May 2005 and our regular skin ward with adequate bed-spacing was shifted to a small temporary makeshift ward with overcrowding of patients. During this period there was a mini outbreak of KVE in four inpatients with various dermatoses after admission of a case of KVE. We are reporting these cases and highlighting the factors responsible for the sudden development of KVE in the ward.

CASE REPORTS

Case 1

A 10-year-old female child, a known case of pemphigus foliaceus, on maintenance dose of 10 mg of prednisolone was brought to the skin OPD with widespread, grouped, monomorphic, umbilicated vesiculopustular lesions over the face, axillae and the trunk with severe constitutional symptoms [Figure 1a]. There were no active pemphigus lesions. A provisional diagnosis of pyoderma and KVE was made and the case was admitted in the makeshift ward. At the time of her admission there were three cases of pemphigus on immunosuppressive drugs and one case of toxic epidermal necrolysis which was recovering. Gram's Stain of the vesicular fluid revealed no bacteria. Tzanck smear from an umbilicated vesicle showed multinucleated giant cells. Herpes simplex virus serology revealed elevated titers of HSV-1 IgG (1.64).

Biopsy from an umbilicated vesicle showed intraepidermal vesicle and pustule formation with a ballooning degeneration at the periphery. The polymerase chain reaction of the blister fluid was not done due to nonavailability of the facility. In view of sudden onset of the lesions, underlying immunosuppression and presence of multinucleated giant cells, the diagnosis of KVE was made. Because of economic constraints, instead of intravenous acyclovir, oral acyclovir 800 mg five times daily was administered for two weeks. Prednisolone was continued at 10 mg per day and dapsone at 25 mg per day. With acyclovir treatment the patient dramatically improved and became

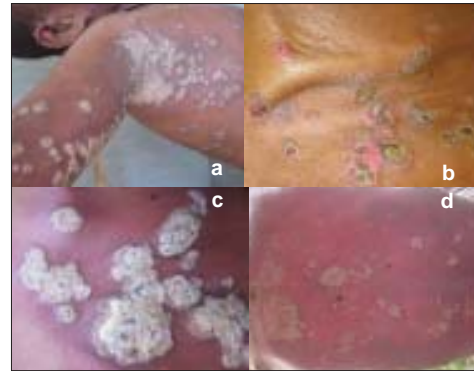


Figure 1: (a) Case 1 - Grouped umbilicated pustular lesions - KVE (b) Case 2 - Crusted lesions of pemphigus (c) Case 2 - KVE over healing lesions of pemphigus (d) Case 5 - KVE over healing lesions of TEN

afebrile. All the lesions healed within seven to 10 days.

Case 2

While our first case was recovering, the adjacent patient, a 55-year-old patient of paraneoplastic pemphigus on immunosuppressive drugs started developing multiple grouped umbilicated vesicular lesions [Figure 1 b, c] around the eyes, mouth, chest and back of the trunk, four days after the admission of Case 1. Tzanck smear revealed multinucleated giant cells. The HSV-1 serology revealed elevated HSV-1 IgM (0.98). Skin biopsy showed intraepidermal vesicle and ballooning degeneration in the periphery. The case was treated with oral acyclovir, 800 mg five times daily for two weeks. She also responded to this therapy and the lesions subsided after 10 days.

Cases 3 and 4

Simultaneously, another two cases of pemphigus vulgaris started developing similar umbilicated, grouped vesicular eruption. A 30-year-old male patient and a 45-year-old female patient who were being treated with dexamethasone cyclophosphamide pulse therapy developed this eruption. Lesions were seen around the eyes, mouth, axillae in addition to the healing pemphigus lesions. Diagnosis of KVE was made with Tzanck smear and HSV serology. HSV-1 IgM was elevated. Both the cases were treated with oral acyclovir 800 mg five times daily for two weeks. While the male patient recovered completely, the female patient succumbed to multiorgan failure.

Case 5

Our last case, a 35-year-old man recovering from toxic epidermal necrolysis, developed clusters of umbilicated vesicular lesions only on the back [Figure 1d] and no lesions elsewhere on the body, unlike in previous cases. Tzanck smear

and HSV serology supported the diagnosis of KVE. The patient recovered completely after 10 days of acyclovir treatment.

DISCUSSION

Herpes simplex virus (HSV) is an ever-present hazard to patients with skin diseases with compromised barrier function. The virus can remain viable in the environment, over the skin surface for two hours, on door handles and taps for two to six hours and for 72h over gauze pieces and swabs and also in the hospital dust for about two weeks.^[12] The pathogenesis of KVE remains to be elucidated. Impaired barrier function of the epidermis has been proposed to cause the development of infection. Hence, many diseases with defective epidermal barrier such as atopic dermatitis, Darier's disease, pemphigus, burns,^[13] facilitate the spread of HSV infection. Patients with immunodeficiency are known to be more susceptible to herpetic infection and an alteration of humoral and/or cellular immunity in patients with KVE having preexisting skin diseases have been implicated as primary factors in the dissemination of the viral infection.^[1] KVE can present as mini-outbreak in dermatology wards resulting in life-threatening nosocomial infections^[14] as in our study.

In all our five cases, there was no previous history of herpes simplex or herpes genitalis. Four out of five cases in our ward developed KVE after the admission of the first case (index case) within a period of four to 10 days. In all these cases serology was positive for HSV-1 IgM. We speculate that these patients acquired the HSV infection either by way of droplets or by fomites. In case 1, the sudden eruption of KVE might be due to reactivation of the HSV due to immunosuppression as her serology showed elevated HSV-1 IgG.

According to the normal hospital standards the floor area for 20 inpatients should be 120 square meters and inter-bed distance should be 1.2 meters. We found that our makeshift ward had floor area of 40.5 square meters with more than 20 beds and inter-bed spacing of only 0.4 meters. Hence overcrowding and close proximity of patients in this ward were the important factors responsible for this mini-outbreak of KVE. In addition low standards of personal hygiene and noncompliance regarding hand washing by the doctors and paramedical staff were also contributory factors.

Though HSV spreads as droplet infection or by direct contact, there was no mention of strict isolation of KVE cases in the literature. But our experience shows that if these cases are not

isolated, mini-outbreaks of KVE in the form of life-threatening nosocomial infection can occur, particularly in dermatology wards with inadequate infrastructure facilities. Hence to prevent such life-threatening infections basic infrastructure facilities like adequate bed-spacing, barrier nursing, hand washing of hospital staff should be strictly followed in all dermatology wards.

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