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Case Report

When skin barriers fail: the emergence of Kaposi varicelliform eruption following disulfiram-induced erythroderma

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ABSTRACT

A 35-year-old male farmer developed drug-induced erythroderma following disulfiram therapy, leading to generalized skin dryness and scaling. Treated with systemic and topical corticosteroids, he later presented with facial vesiculopustular lesions, which progressed to erosions due to manipulation. A Tzanck smear revealed multinucleated giant cells, confirming herpes simplex virus (HSV) infection and diagnosing Kaposi varicelliform eruption (KVE). He was treated with oral acyclovir (800 mg, five times daily for seven days), showing marked improvement. KVE, commonly caused by HSV-1 or HSV-2, may also result from vaccinia virus or Coxsackievirus A16. It is vital to differentiate KVE from similar conditions like eczema coxsackium and eczema vaccinatum. In this case, erythroderma compromised the skin barrier, while corticosteroid-induced immunosuppression likely facilitated viral spread. KVE can also occur with conditions like HHD, Darier's disease, burns, and psoriasis. Early diagnosis and antiviral therapy are crucial to prevent complications such as bacterial superinfections or systemic viremia.

Keywords: Kaposi varicelliform eruption, Disulfiram, Erythroderma, Herpes simplex virus, Acyclovir

INTRODUCTION

Kaposi varicelliform eruption (KVE) is a severe viral infection that predominantly occurs in individuals with underlying dermatological conditions that compromise the skin barrier. While most commonly associated with HSV-1 and HSV-2, other viral pathogens such as vaccinia virus and Coxsackievirus A16 can also be responsible.[1]

CASE REPORT

A male farmer (age 35) with a history of generalized skin dryness and scaling persisting for four months, along with the development of fluid-filled lesions on his face over the past 15 days, presented to the hospital. The patient had no prior dermatological conditions and was in good health until four months earlier when he was admitted to a de-addiction centre for alcohol dependence. He was prescribed disulfiram, which he took for 20 days. Approximately 20 days after initiating therapy, he developed erythema on his upper limbs and trunk, which later spread across his body, accompanied by itching and scaling.

Following discharge from the de-addiction centre, he was admitted to a private hospital and was diagnosed with drug-induced erythroderma secondary to disulfiram. His initial management involved systemic corticosteroids (specific details unavailable), later transitioning to topical steroids and moisturizers. After one month of hospitalization, he was discharged, although persistent generalized scaling remained.

Fifteen days before his current hospital visit, the patient developed vesicular facial lesions, which he manipulated,

leading to widespread erosions. Clinical examination revealed multiple polycyclic, punched-out erosions on the trunk, neck, and face, with a few vesicular lesions on the upper back. Routine blood investigations were within normal limits, and the systemic examination was unremarkable. A Tzanck smear from an intact vesicle on the upper back confirmed a viral etiology through the identification of multinucleated giant cells.

Based on clinical and laboratory findings, a diagnosis of KVE was established. Other baseline investigations were normal and serologies were negative. The patient was immediately initiated on oral acyclovir 800 mg, which was administered five times daily for seven days, resulting in significant clinical improvement.

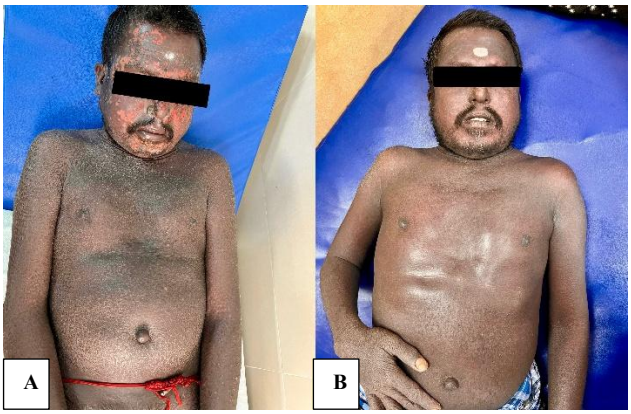

Figure 1 (A and B): Left: At the time of admission - multiple polycyclic punched-out erosions on the face, neck and trunk. Right: After treatment showing complete healing of the lesions.

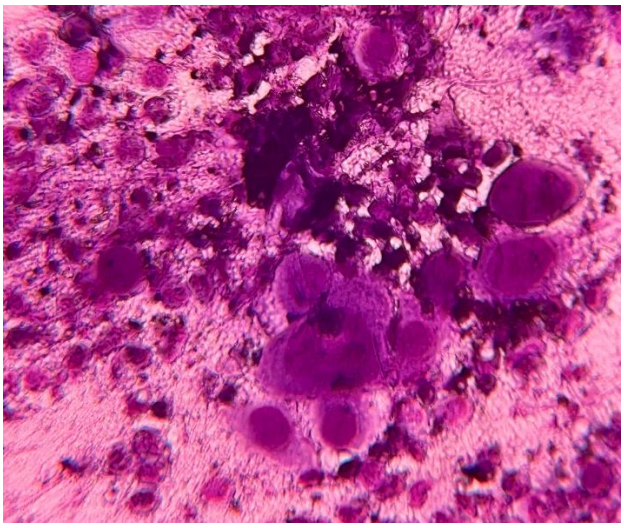

Figure 2: Presence of multinucleated giant cells on Tzanck smear.

DISCUSSION

KVE is a severe viral infection that predominantly occurs in individuals with underlying dermatological conditions that compromise the skin barrier. While most commonly associated with HSV-1 and HSV-2, other viral pathogens such as vaccinia virus and Coxsackievirus A16 can also be responsible. These infections exploit skin barrier defects, leading to extensive vesiculopustular eruptions and potential systemic involvement.[2]

Distinguishing KVE from other viral infections

HSV-related KVE

Characterized by painful, monomorphic vesicles with central umbilication. Tzanck smear reveals multinucleated giant cells. Fever, malaise, and lymphadenopathy are common, and lesions tend to coalesce and spread rapidly.[1]

Coxsackievirus A16-induced KVE (Eczema coxsackium)

Presents with monomorphic vesicles and bullae, often localized to sites of atopic dermatitis, hands, feet, and mouth. Unlike HSV, multinucleated giant cells are absent on Tzanck smear.[1]

Vaccinia virus-induced KVE (Eczema vaccinatum)

Occurs post smallpox vaccination, characterized by pustular lesions with severe systemic involvement, often requiring intensive management.[3]

Eczema monkeypoxicum

Recently reported in China, this emerging condition suggests a potential expansion of viral etiologies in patients with compromised skin barriers.[6]

Accurate diagnosis through HSV PCR, viral cultures, or immunohistochemistry is crucial for optimal management. In this case, the patient's erythroderma resulted from a disulfiram-induced hypersensitivity reaction, leading to significant barrier dysfunction and increased susceptibility to HSV superinfection. The use of systemic corticosteroids likely further compromised immune function, exacerbating viral dissemination.

Timely recognition and intervention in KVE are crucial, as untreated cases may lead to complications such as secondary bacterial infections, keratoconjunctivitis, and systemic viremia. Although KVE is frequently observed in atopic dermatitis, it may also occur in patients with burns, ichthyosis vulgaris, pemphigus foliaceus, psoriasis, Darier's disease, Grover's disease, Hailey-Hailey disease, congenital ichthyosiform erythroderma, Sézary syndrome, cutaneous T-cell lymphoma, irritant contact dermatitis, pityriasis rubra pilaris, rosacea, drug-induced eruptions, and *Staphylococcal* scalded skin syndrome.[5]

Diagnosis relies on clinical evaluation, supported by Tzanck smear findings of multinucleated giant cells and

confirmatory HSV PCR or culture. The primary treatment for KVE is antiviral therapy, with acyclovir being the drug of choice. In severe cases, hospitalization and intravenous antiviral administration may be required. Supportive care, including the use of moisturizers, antiseptic measures, and corticosteroid avoidance, plays a crucial role in preventing complications.[4]

CONCLUSION

This investigation underscores the significance of recognizing KVE as a potential complication of drug-induced erythroderma, particularly in patients with compromised skin barriers. Early diagnosis and prompt antiviral therapy initiation are essential to prevent systemic complications and ensure a favorable prognosis. Clinicians should be cautious when prescribing systemic corticosteroids in such cases, as they may increase the risk of viral superinfections. A multidisciplinary approach involving dermatologists and infectious disease specialists is crucial for optimal patient management.

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