Localized psoriasis herpeticum: Case report and review of literature

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ABSTRACT

Kaposi's varicelliform eruption (KVE) is a widespread cutaneous eruption caused by viruses, especially herpes simplex virus in patients with pre-existing dermatoses. ''Psoriasis herpeticum'' refers to the rare occurrence of KVE in patients with psoriasis. We report a case of KVE localized to the face in a patient with exfoliative dermatitis secondary to psoriasis. This case is being reported to make the treating clinician aware of the possibility of KVE in patients with psoriatic erythroderma.

Key words: Acyclovir, Kaposi's varicelliform eruption, psoriasis herpeticum, Tzanck smear

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INTRODUCTION

Kaposi's varicelliform eruption (KVE) was first described in 1887 by Moritz Kaposi.[1] It is a widespread cutaneous infection with a virus which normally causes localized or mild vesicular eruptions in patients with pre-existing skin diseases.[2] A great majority is caused by infection with herpes simplex virus (HSV) type I and is usually called eczema herpeticum (EH).[2] Localized forms are not uncommon and if complicated by secondary bacterial infection, may cause diagnostic difficulty. A rare subtype is ''psoriasis herpeticum''[3] which is the occurrence of KVE in psoriasis. Till date only four cases of psoriasis herpeticum have been reported.

CASE REPORT

A 15-year old girl who was undergoing treatment for psoriatic erythroderma developed relatively asymptomatic vesicular lesions over the forehead which rapidly became pustular and spread to involve the whole face. She gave past history of recurrent episodes of erythroderma of five years duration and skin biopsy had proved it to be secondary to psoriasis. She also gave past history of recurrent episodes of painful vesicles over the lips that healed spontaneously within a week. The present episode of erythroderma was being treated conservatively with oral antihistamines and topical emollients at the time of eruption. Constitutional symptoms were absent. Physical examination revealed an eruption of numerous vesicles and pustules, localized to the face [Figure 1]. Few lesions were umbilicated. Tzanck smear from the intact vesicle showed multinucleated giant cells [Figure 2]. Hemogram revealed leucocytosis. Bacterial culture from the pustular lesions grew staphylococci sensitive to cloxacillin. Serum IgG and IgM anti-HSV-1 antibody were positive. Other biochemical parameters were within normal limits. Ophthalmologic examination was unremarkable. Diagnosis of psoriasis herpeticum was made based on classic clinical features along with Tzanck smear finding and positive HSV serology. Patient improved with oral acyclovir 400 mg thrice daily along with oral cloxacillin 500 mg four times daily and skin lesions cleared completely by the tenth day [Figure 3].

DISCUSSION

Kaposi's varicelliform eruption is a distinct cutaneous eruption caused by HSV type 1 and 2 and rarely vaccinia virus or coxsackie A16 virus, superimposed on a pre-existing dermatosis.[1] Numerous nomenclatures have been used to describe the types of KVE depending on either the causative virus (eczema herpeticum, eczema vaccinatum, etc.) or the pre-existing dermatosis (psoriasis herpeticum). Though KVE is commonly a widespread eruption, localized forms may be seen when pre-existing dermatosis is confined to limited areas.[2] The most common predisposing

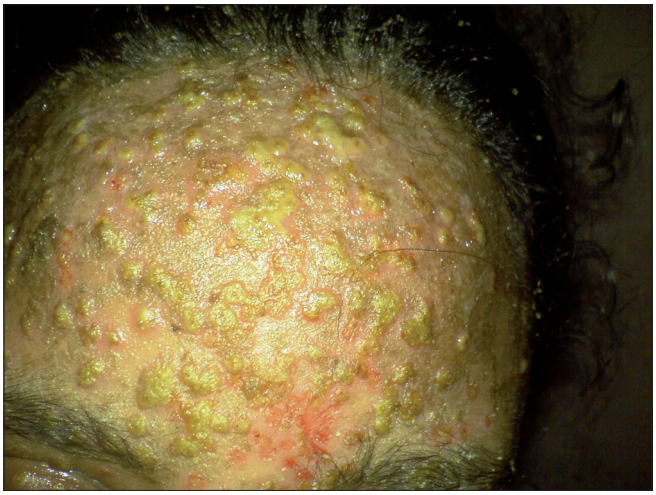

Figure 1: Vesiculopustular lesions localized to the face

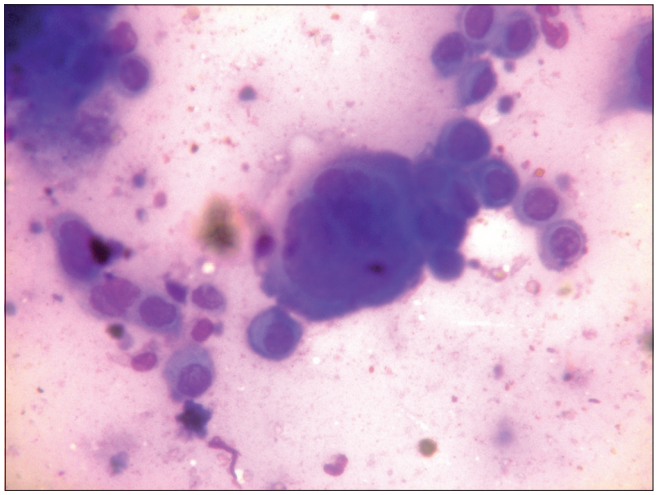

Figure 2: Multinucleated giant cells (Leishman stain, ×100)

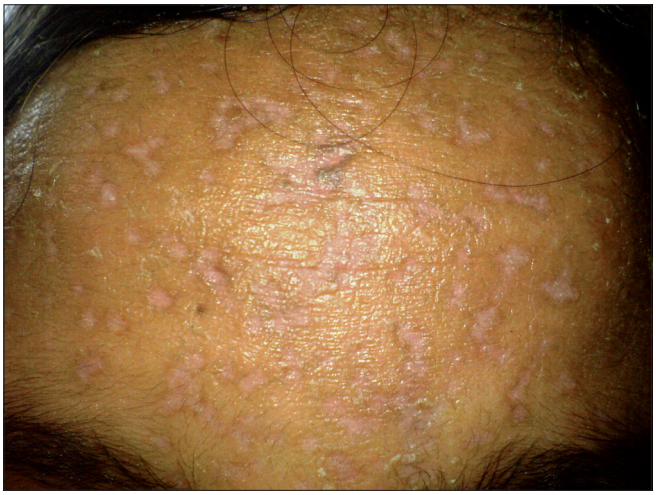

Figure 3: Healed lesions after antiviral therapy

condition for KVE is atopic dermatitis but it has been described in various other diseases[2] like pemphigus foliaceus, congenital ichthyosiform erythroderma, Darier's disease, Grover's disease, familial benign chronic pemphigus, allergic contact dermatitis, irritant contact dermatitis, pityriasis rubra pilaris, rosacea, ichthyosis vulgaris, staphylococcal scalded skin syndrome, drug eruptions, cutaneous T-cell lymphoma, and lupus vulgaris.[4] It may also follow second-degree burns,[2] topical application of tacrolimus for atopic dermatitis,[5] laser therapy,[2] dermabrasion,[2] and vaccination with BCG.[6]

KVE most commonly occurs in second and third decades of life. After an incubation period of about 10 days (range 5–19 days) vesicles erupt in crops and rapidly become pustular. These lesions may turn hemorrhagic. New crops of lesions may continue to appear for 5–7 days. Constitutional symptoms develop 23 days after the onset of eruption, with associated regional lymphadenopathy.[2]

Till date only four cases of KVE developing on pre-existing psoriatic erythroderma have been reported. The first report was by Saraswat *et al,*[4] in 2002 in a patient with erythrodermic psoriasis, while on treatment with methotrexate. Santmyire–Rosenberger *et al,*[3] in 2005 reported three cases of KVE in psoriasis and coined the term psoriasis herpeticum. Two of these patients were on oral methotrexate and the third was on acitretin, Goeckerman regimen and whirlpool therapy.

The exact pathogenesis of KVE is yet to be ascertained. It is speculated that an impaired barrier function of the epidermis and defective host immune response are factors responsible for in increased susceptibility to KVE. One recent study by Howell *et al,*[7] concluded that cathelicidin peptide LL -37 might be deficient in patients with atopic dermatitis, explaining the increased susceptibility to KVE. Cathelicidin family of inducible antimicrobial peptides is an integral component of innate immune response that has activity against bacteria, fungal, and viral pathogens. Wollenberg *et al,*[8] reported reduced levels of plasmocytoid dendritic cells, which play a role in host viral defense, in skin samples of patients with atopic dermatitis when compared to psoriasis. Even though previously psoriatic skin was thought to be immune to KVE, a study by Goodyear *et al,*[9] proved it otherwise. They found that HSV replicated quickly to a higher titre in explants of both atopic dermatitis and psoriatic skin when compared to normal skin.

Eczema herpeticum (EH) is a self-limiting disorder but carries a minor risk for severe and bilateral herpetic ocular disease. Systemic viremia with multiple organ involvement is the major cause of morbidity and mortality. EH can be successfully treated with oral acyclovir in a dose of 200 mg five times a day or 400 mg three times a day. In children with EH, 40–80 mg/kg/day is given in 3–4 divided doses (maximum of 1 g/day). Severe cases should receive intravenous acyclovir 10–15 mg/kg three times a day.[10]

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