Kaposi varicelliform eruption

Sir,

A 26-year-old man who was being regularly treated for pemphigus vulgaris since two years presented with complaints of continuous high-grade fever since two days and multiple fluid-filled lesions that appeared initially over the face, and later over the chest, back, and limbs. Cutaneous examination showed multiple umbilicated vesicular lesions(a few of them coalesced) over the face with severe facial edema [Figure 1], chest [Figure 2], trunk, and all four limbs along with multiple punched out erosive lesions and a few crusted lesions. Old crusted lesions of pemphigus were present over the scalp with patchy hair loss. The total leucocyte count was 23,000/mm^3, and liver enzymes were marginally raised. Other investigations were unremarkable. Kaposi varicelliform eruption (KVE) was suspected considering its acute onset, vesicular lesions, fever, and facial edema in a known case of pemphigus on immunosuppressants since two years. Other differentials were flare of underlying pemphigus (as many a times KVE is confused with the underlying disease), and contact irritant dermatitis which was excluded by history taking. Blood culture was negative and pus culture from one of the discharging erosive lesions grew *Staphylococcus aureus* after 48 h, which was sensitive to the antibiotics we had started. Tzanck smear showed acantholytic cells. Histopathologic examination revealed a subcorneal blister [Figure 3] with hemorrhage, vessel damage, and a mixed inflammatory infiltrate with multinucleated giant cells in the underlying dermis [Figure 4] suggestive of herpes infection. Immunofluorescence was not done due to lack of the facility.

Considering high total counts and clinical condition of the patient, he Was given intravenous acyclovir (10 mg/kg every 8 hourly), piperacillin-tazobactam (4.5 g 6 hourly) and linezolid (600 mg 12 hourly). and continued for 10 days. The patient continued to receive oral prednisolone 10 mg once daily and azathioprine 50 mg twice daily for pemphigus as before. Post treatment, his skin lesions healed with varicelliform scars [Figure 5].

KVE is a potentially life-threatening viral infection caused by herpes simplex virus (HSV) type 1, HSV-2, coxsackievirus A16, or vaccinia virus that occurs over a pre-existing dermatosis.

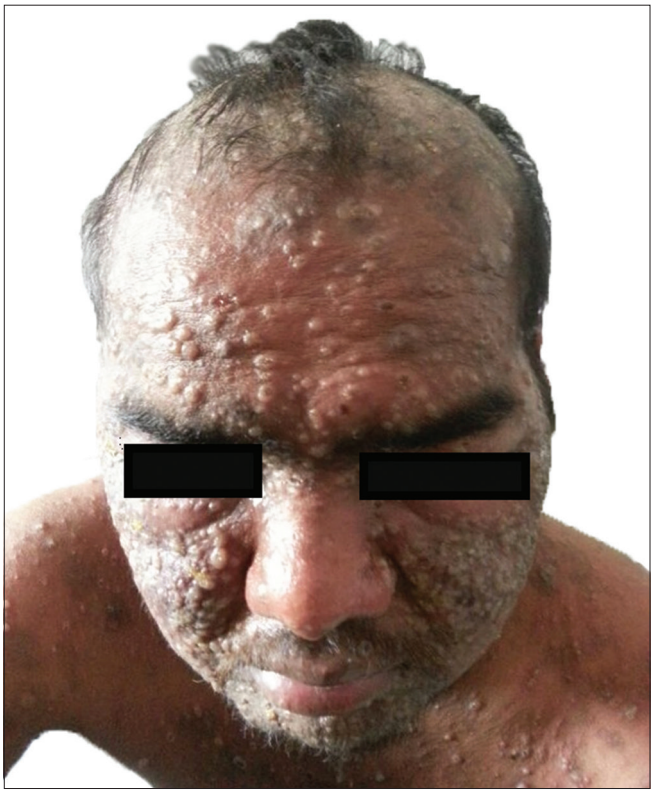

Figure 1: Multiple fluid-filled vesicular lesions over face along with multiple punched out erosive lesions and few crusted lesions with severe facial edema

Multiple skin disorders have been associated with KVE, including pemphigus foliaceus, Darier's disease, pemphigus vulgaris, pityriasis rubra pilaris, Hailey–Hailey disease, irritant contact dermatitis, cutaneous T-cell lymphoma, seborrheic dermatitis, psoriasis, Wiskott–Aldrich syndrome, congenital icthyosiform erythroderma, and Sezary syndrome.[1-3]

KVE usually begins as a sudden eruption of painful, edematous clusters of umbilicated vesiculopustules on the skin affected by a pre-existing dermatoses and may be accompanied by a flu-like syndrome. The vesiculopustules progress to painful hemorrhagic, crusted, punched-out erosions that coalesce to form denuded areas that are prone to secondary bacterial colonization.[4] The upper body is the most common site of infection, with a predilection for the head and neck.[1] In some

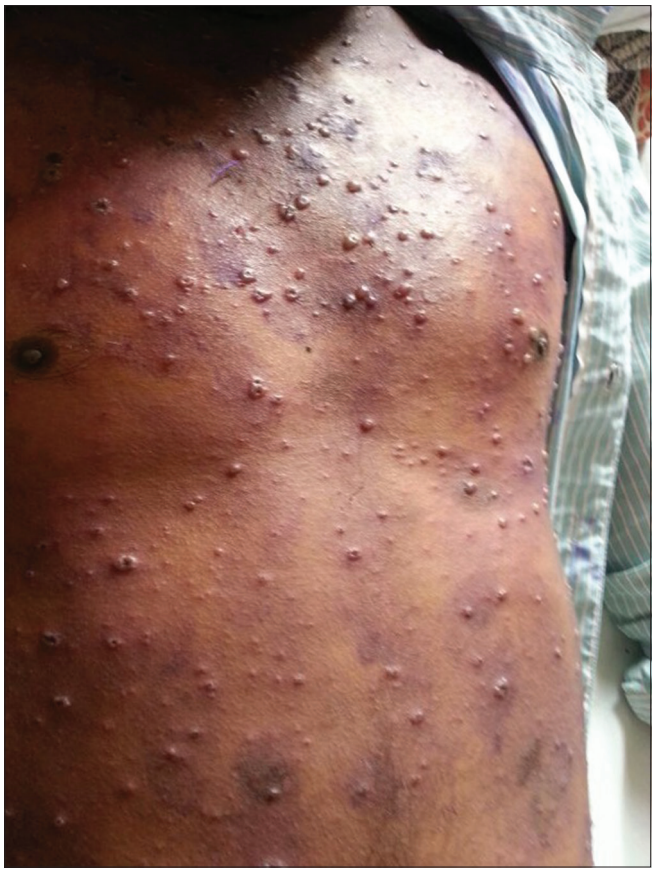

Figure 2: Clusters of umbilicated vesiculopustules over chest

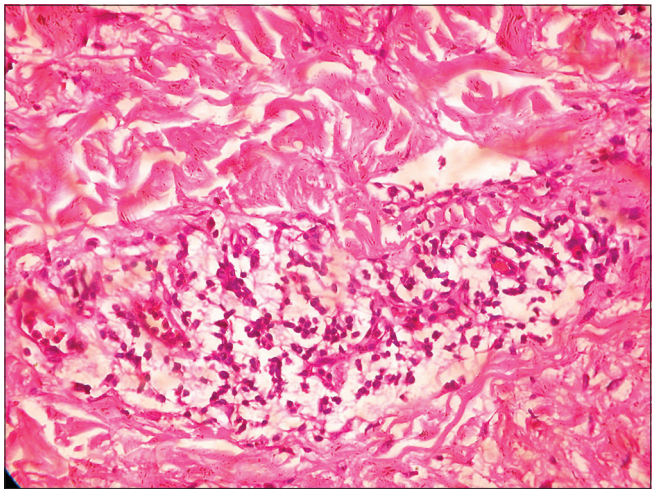

Figure 4: Dermis showed hemorrhage with vessel damage along with mixedinflammatory infiltrate: H and E staining and x40 magnification

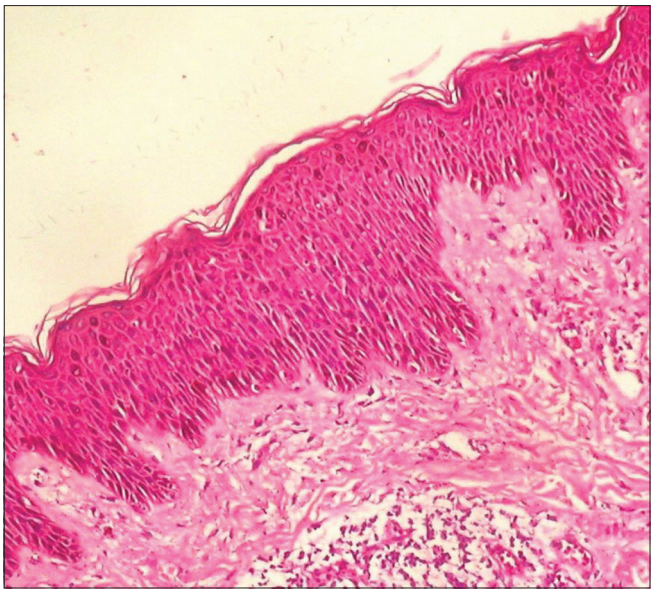

Figure 3: Subcorneal blister formation: H and E staining and x40 magnification

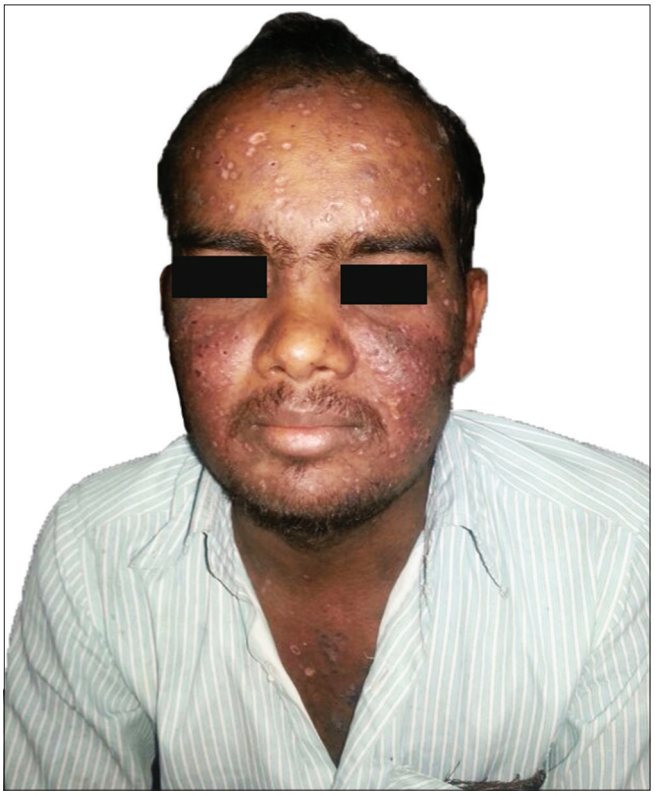

Figure 5: Post-treatment scarring

cases, it may progress to fulminating, life-threatening infection and can have severe sequelae, including herpes keratitis, disseminated infection with visceral involvement, and death.[1,4] A delay in diagnosis often occurs because the eruption is confused with the underlying disease. HSV is an ever-present hazard to patients having skin disease with a compromised barrier function. The virus can remain viable in the environment over the skin surface for 2 h, on door handles and taps for 2–6 h, over gauze pieces and swabs for 72 h, and over hospital dust for about 2 weeks.[5] The most clearly delineated risk factor for KVE is disruption of the epidermal barrier.[1,4] Hence it is necessary to create awareness about this uncommon dermatologic entity and to stress on the importance of bed-spacing and barrier nursing so as to prevent secondary bacterial infection. In a retrospective review of 100 KVE patients, Wollenberg *et al*.

found that a high serum IgE level and early onset of atopic dermatitis were both risk factors.[3] Defective cytokine secretion and decreased cell-mediated immunity in skin affected by atopic dermatitis and other diseases also appear to play a role in the pathogenesis of KVE. Early use of both antiviral drugs and antibiotics is extremely important; their use should not be delayed pending laboratory tests. When a bacterial infection is not present, patients should be given a topical antibiotic cream.[6] Patients with recurrent HSV infections and chronic skin disease predisposing to KVE should be offered prophylaxis. KVE can be life threatening. This case illustrates the need for both timely treatment and the use of appropriate laboratory tests to confirm the diagnosis. Physicians should recognize the early presentation. Effective treatment is available in the form of antiviral drugs and should not be delayed.

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