Bullous Scabies in a patient on anticancer therapy

Sir,

Scabies is highly contagious and intensely pruritic skin infestation caused by mite *Sarcoptes scabiei var hominis*. Bullous scabies is a rare manifestation of scabies which is occasionally seen in the immunosuppressed.[1] We report a case of bullous scabies in an immunosuppressed 30 year old lady on anticancer therapy.

CASE REPORT

A 30 year old female patient, who was taking methotrexate, cyclophosphamide, and actinomycin D for gestational trophoblastic disease (choriocarcinoma) since 3 months, was referred for highly pruritic, vesiculobullous lesions which were present since 8 days. Itching was more severe during nights. On examination, excoriated papules and vesicles with clear fluid filled tense bullae (1-1.5 cm) were present over dorsa of feet, ventral surface of right forearm, finger webs, chest, abdomen and thighs (Figure 1). Closer examination revealed burrows over finger webs. Direct microscopy of scraping from the burrow revealed eggs of *S. scabiei* mite. Hemogram was normal. Bulla fluid on culture was sterile. Histopathology showed subepidermal bullae with cavity containing predominantly neutrophils and some eosinophils. The patient was treated with topical 5% permethrin. Resolution of the lesions was noted after 4 weeks.

DISCUSSION

Though the vesicular lesions are common in infants and children, bullous lesions are uncommon in scabies. Bullous scabies is seen in immunocompetent infants, young children and elderly although some reports have shown its occurrence in immunosuppressed adults.[3] In communities where scabies is not endemic, the index of suspicion is low and bullous scabies can be confused with bullous pemphigoid, insect bites, linear IgA dermatosis, epidermolysis bullosa or chronic bullous disease of childhood (CBDC). Bullous lesions over scabies prone sites, nocturnal itching and detection of mite eggs in burrow scraping finally confirmed scabies in our case. Bullous scabies has on occasions resembled bullous pemphigoid clinically, light microscopically and immunopathologically.[1,4] Previous case reports suggest that several cases of bullous scabies were misdiagnosed and treated as bullous pemphigoid, due to false positive DIF and IIF in bullous scabies.[4]

Several theories have been proposed to explain pathogenesis of bullae formation in bullous scabies. Veraldi et al[3] has given the theory of alteration of bullous pemphigoid antigen with mite secretions and the resultant production of antibasement membrane zone antibody formation. Other theories proposed to explain bullae in scabies are superinfection with *Staphylococcus aureus*,[6] toxins producing bullae and id reaction to scabies mite.[7]
Konishi et al have demonstrated induction of bullous pemphigoid by Western Blot analysis of circulating antibodies against BP 180 and BP 230 antigen which lead them to propose that some of the lesions occurring in bullous scabies are true bullous pemphigoid.

Bornhovd et al postulated persistence of parasites as cause of Th2 cells activation with increased IL5 and eosinophils secretion of proteolytic enzymes near BMZ (basement membrane zone) producing bullae formation. The exact mechanism for bullae formation is not known and further studies need to be done to explain the pathogenesis of bullous scabies. In a patient with itchy, vesiculobullous lesions, burrows should be looked for, as they are seen in majority of cases of bullous scabies.

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REFERENCES


Bullous systemic lupus erythematosus: Response to dapsone in two patients

Sir,

Bullous systemic lupus erythematosus (SLE) is a transient autoimmune bullous disease that occurs in established cases of SLE. Some authors use this term for all subepidermal autoimmune bullous conditions in SLE, whereas others use it for conditions where dermal antigens are involved.[1]

Histopathologically, this condition is characterized by subepidermal bullae with neutrophilic microabscesses, nuclear dust and fibrin at the tips of dermal papillae. Direct immunofluorescence reveals a linear deposition of IgG, IgA, IgM and C3 in the basement membrane zone. The target antigen is believed to be type VII collagen in the dermis. We came across two cases of bullous SLE at our institute.

An 18-year-old female presented with arthritis of both knee and ankle joints since the last 1 month, fever since 15 days, oral ulcerations since 10 days, and lacrimation and irritation of the left eye since 4 days. After 3 days of hospitalization, she developed asymptomatic erythematous, non-itchy, maculopapular rash over the abdomen, which remained confined to the trunk. On the 7th day of hospitalization, she developed vesicles on the left retroauricular area, left upper eyelid, right axillary area, chest and abdomen. These vesicles gradually increased in size and number.

On examination, she had pallor, bilateral cervical lymphadenopathy, multiple tense blisters over apparently normal skin, oral erosions over the hard palate, and redness of the left eye. Nikolsky’s sign was negative.

The second patient was a 20-year-old female who presented with arthritis of both ankle and wrist joints with low grade fever, swollen feet and face and bullous lesions over the face and neck. She was a known case of nephrotic syndrome since the last 1 year and gave a