Ind J Dermatol Venereol Leprol 1993; 59 : 127 - 8

PEMPHIGOID WITH EOSINOPHILIA : REMISSION AND REDUCTION IN EOSINOPHIL COUNT ON TREATMENT

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A woman with pemphigoid had massive eosinophilia (51% eosinophils in differential count and total eosinophil count 8850/10^6/L). She responded to oral prednisolone. After 2 months of starting the treatment the differential count showed 15% eosinophils and total eosinophil count fell to 950/10^6/L. This case supports the hypothesis that imflammatory cells like eosinophils have an important role in the pathogenesis of pemphigoid.

Introduction

Pathogenesis of blister formation in pemphigoid is not clearly understood. Some studies suggest that inflammatory cells like eosinophils may play a role in the formation of blister.[1,2] We report a case of pemphigoid with associated massive eosinophilia. on treatment with oral prednisolone the lesions healed and eosinophilia decreased considerably.

Case Report

A 55-year-old woman presented with complaints of itching and blisters all over the body for past 4 months. After an initial phase during which she had itching and urticarial lesions and which lasted for about 15 days, she developed blisters which involved almost the whole skin surface except palms, soles and face within 15-20 days. She had not taken any medicine during a month preceding the onset of illness. There was no history of diarrhoea. Past, personal and family histories were not significant.

Her general and systemic examinations were normal. Tense bullae of different sizes, upto 3 cm in diameter, were present all over the body except palms, soles and face. Bullae were present both over normal-appearing as well as over erythematous skin (Fig. 1). Bullae were dome-shaped and contained

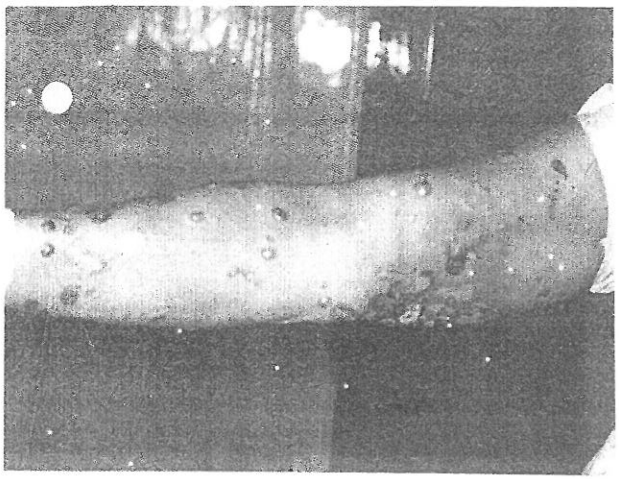

Fig. 1. Lesions of pemphigoid on upper limb

serous fluid. A few hypopigmented macules were present at the sites of previous lesions, but there was no scarring. Oral mucous membrane showed 2 bullae, each with a diameter of 1 cm. Genital mucous membrane was normal.

Differential leukocyte count (P 20, L28, M1, E51) and total eosinophil count (8850x10^6/L) showed massive peripheral blood eosinophilia. Tzank smear did not show acantholytic cells. Other investigations were normal. Skin biopsy

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was taken from a bulla over normal-appearing skin. Histology showed subepidermal bulla due to degeneration of the basement zone. Epidermis at the roof of the bulla was intact. Dermis showed sparce perivascular infiltrate consisting mainly of mononuclear cells with a few eosinophils.

The diagnosis of pemphigoid was made. The patient did not get any relief on dapsone 100 mg tid given for 7 days. She was given oral prednisolone 80 mg/day. She responded to this treatment. Prednisolone was gradually tapered after 3 weeks and 20 mg/d was continued as maintenance therapy. After 2 months of starting oral prednisolone the desease was in remission. Differential count showed 15% eosinophils and total eosinophil count had fallen to 950×10^6/L.

Comments

Binding of anti-basement membrane antibodies alone does not produce lesions in pemphigoid. Chemotactic factors for eosinophils have been identified in pemphigoid blister fluid.[1] Further, lysosomal enzymes from eosinophils have been found in basement membrane zone in the early stage of blister formation.[2] These inflammatory cells may have an important role in the formation of blister in pemphigoid. Our case with massive eosinophilia, which decreased considerably after successful treatment with steroid, supports this view. Also, to our knowledge, eosinophilia of this degree has not been reported with pemphigoid.

References

1. Baba T, Sonozaki H, Seki K, et al. An eosinophil chemotactic factor present in blister fluid of bullous pemphigoid patients. J Immunol 1981; 116 : 112-6.
2. Dubertret L, Bertaux B, Forse M, et al. Cellular events leading to blister formation in bullous pemphigoid. Br J Dermatol 1980; 103 : 615-24.