

Heterogeneous Distribution of Mast Cells in Lichenified Lesions of Atopic Dermatitis

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The aim of the present study was to determine whether the number of mast cells in lichenified lesions of atopic dermatitis (AD) have a relationship to familial background of atopic respiratory disease (ARD). We obtained biopsy specimens of lichenified lesions from 59 consecutive patients with AD. They included 35 AD patients who had a personal history of ARD, 9 AD patients without a personal history of ARD but with a family history of ARD, and 15 "pure" AD patients without a personal or a family history of ARD. From each biopsy specimen, 4 μ m-thick paraffin-embedded sections and/or 1 μ m-thick Epon-embedded sections were prepared, and stained with Giemsa's reagent. With both methods of preparation, the sections from AD patients with a personal history of ARD showed significantly greater mast cell number in lichenified lesions than the "pure" AD patients. It is suggested that the increase of mast cells often seen in AD lesions may be characteristic of a subgroup of AD patients who have a predisposition for ARD.

Several authors (1-3) have reported that mast cell numbers are increased in lichenified lesions of atopic dermatitis (AD). But others (4, 5) state that skin lesions of AD often show a normal number of mast cells. Thus, at present it is unclear whether the increase of mast cells in skin lesion is a common feature of AD or whether the increase of tissue mast cells occurs only in a subgroup of AD patients.

Mast cells are known to play a part in the asthmatic reaction (6, 7). A recent report (8) showed that the number of mast cells or basophil progenitors in the circulation are increased in atopic patients. In the present study, therefore, we investigate whether the presence of a personal or family history of atopic respiratory disease (ARD) is associated with an increase of mast cells in lichenified lesions of AD.

MATERIALS AND METHODS

Patients. A total of 59 patients with AD not being treated with topical corticosteroids for at least one month prior to the

examination, were included in the study. The diagnosis of AD was based on the morphology and distribution of skin lesions, the chronic course, and a family history of AD or atopic respiratory disease (ARD). All patients had lichenified lesions in the antecubital and popliteal fossae, neck, and other predisposed areas. They ranged in age from 13 to 64 years, with a mean age of 23. They were classified into three subgroups: 1) those who had a personal history of ARD (35 cases), 2) "pure" AD patients without personal or family history of ARD (15 cases), and 3) those without a personal history of ARD but with a family history of ARD (9 cases).

Biopsies. In each of the 59 patients, one or two biopsy specimens were taken from a lichenified plaque.

A) *4 μ m-thick paraffin-embedded sections.* Forty-nine biopsy specimens were fixed in 10% neutral buffered formalin and embedded in paraffin. From each specimen 20 to 40 serial sections (4 μ m) were prepared and stained with Giemsa's reagent.

B) *1 μ m-thick Epon-embedded sections.* Thirty-two biopsy specimens were fixed with 3% glutaraldehyde in 0.1 M PBS at pH 7.4. Post-fixation in 1% Osmium tetroxide was followed by dehydration and embedding in Epon. Following the methods of Mihm et al. (2), 20 to 40 serial 1 μ m-thick sections were prepared, and stained with Giemsa's reagent.

Counting of mast cells. All biopsy specimens had 1) perivascular infiltrates of mononuclear cells at interfollicular areas of upper dermis where the infiltrates were distributed in a horizontal direction, and 2) vertical distributed inflammatory cells around hair follicles. We counted the mast cells in a total of 1 000 cells at the interfollicular areas alone.

RESULTS

A) *4 μ m-thick paraffin-embedded sections.* 1) In AD patients with a personal ARD history (31 cases) many mast cells were present in the lichenified lesions (Fig. 1). The mean number of mast cells was 58.5. 2) In "pure" AD patients without a personal or family history of ARD (12 cases), the number of mast cells were not increased (Fig. 2). The mean number of mast cells was 27.5. 3) In AD patients without a personal history of ARD but with a family history of ARD (6 cases), the number of mast cells in lichenified lesions varied widely from patient to patient. The mean number of mast cells was 54.9.

B) *1 μ m-thick Epon-embedded sections.* 1) In AD

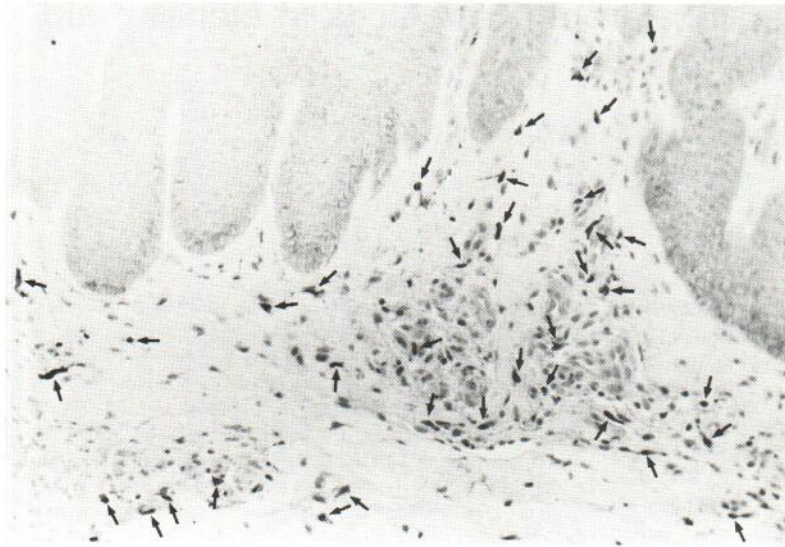


Fig. 1. 4 μ m-thick section. Giemsa's staining. Lichenified lesions of AD patient with a personal history of ARD. Many mast cells are seen in the dermal infiltrate.

patients with a personal history of ARD (17 cases), mast cells were abundant in the lichenified lesions (Fig. 3). The mean number of mast cells was 60.2. 2) In "pure" AD patients (9 cases), the number of mast cells was not increased (Fig. 4). The mean number of mast cells was 24.8. 3) In AD patients without a personal history of ARD but with a family history of ARD (6 cases), the distribution of mast cells in the dermal infiltrate was similar to that in the "pure" AD group. The mean number of mast cells was 31.2.

Fig. 5 shows the distribution of mast cells in lichenified lesions in all the AD patients examined in the present study.

Thus, from the data of both paraffin-embedded sections and Epon-embedded sections, it was evident that there was a significant difference ($p < 0.01$) in mast cell number in lichenified lesions between AD patients with a personal history of ARD and "pure" AD patients without a personal or a family history of ARD.

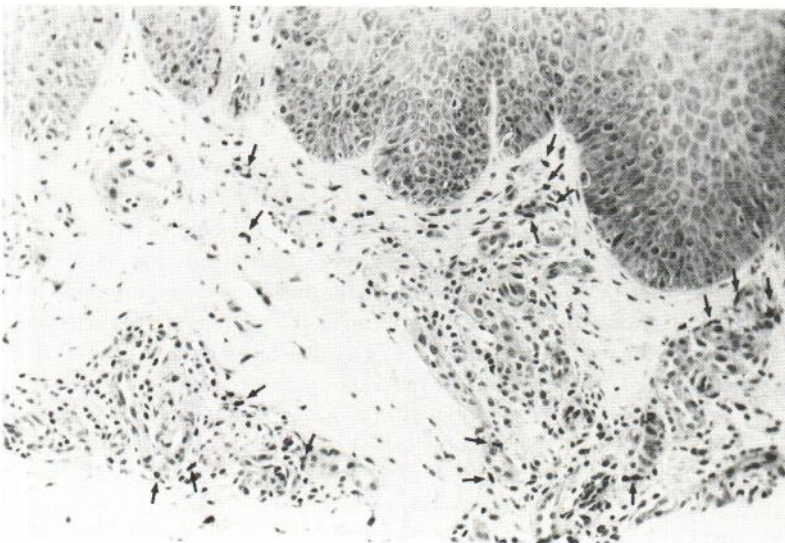


Fig. 2. 4 μ m-thick section. Giemsa's staining. Lichenified lesion in "pure" AD patient. Only a small number of mast cells are present in the dermal infiltrate.

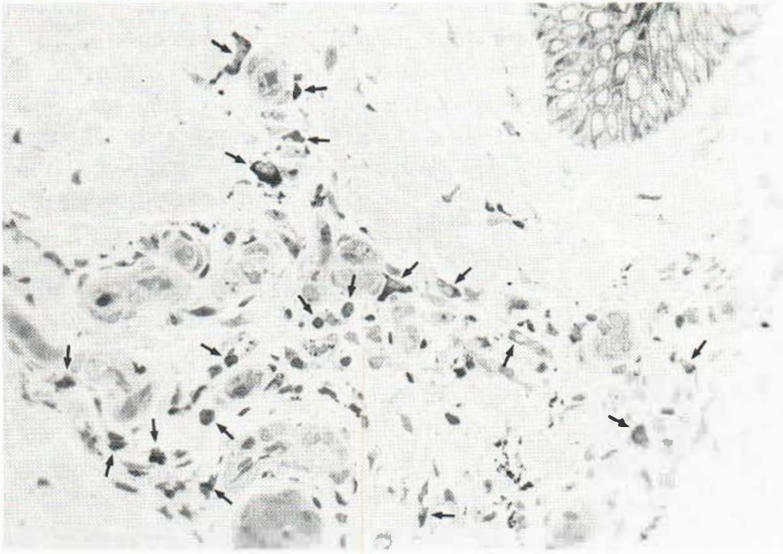


Fig. 3. 1 μ m-thick section. Giemsa's staining. Lichenified lesion in AD patient with a personal history of ARD, showing many mast cells in the infiltrate.

DISCUSSION

The present study demonstrates that the number of mast cells in lichenified lesions of AD vary widely from patient to patient. The mast cell number was significantly higher in the group of AD patients with a personal history of ARD than in the "pure" AD patients without a personal or family history of ARD. Thus, it is likely that co-existent ARD is an important factor which leads to increased mast cell numbers in skin lesions of AD. A very high number of mast cells

in lichenified lesions was observed in some patients of the AD group with only a family history of ARD. This may imply that the mast cell number in skin lesions are increased in AD patients who have a subclinical ARD, or predisposition to ARD.

Braun-Falco et al. (1) reported that an increase in mast cells was seen in lichenified lesions of AD. Mihm et al. (2), using 1 μ m-thick Epon-embedded sections, also investigated the distribution of tissue mast cells in skin lesions of AD, and they concluded

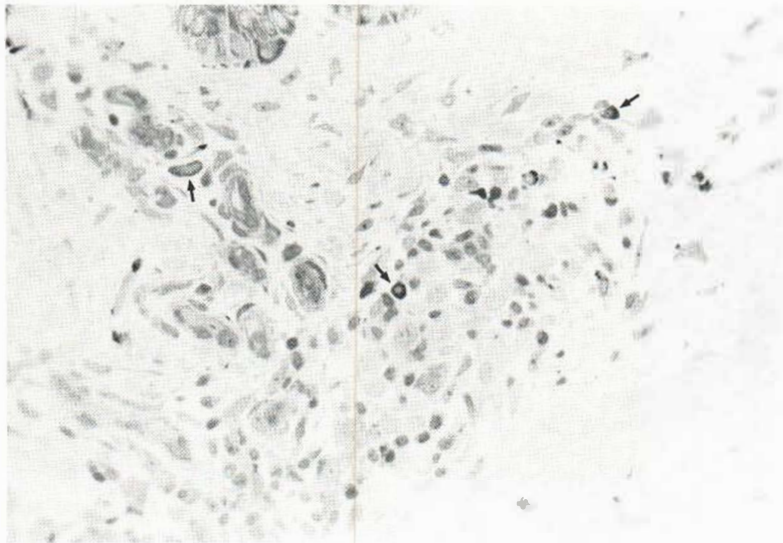


Fig. 4. 1 μ m-thick section. Giemsa's staining. Lichenified lesion in "pure" AD patient. Only three mast cells are observed in the field.

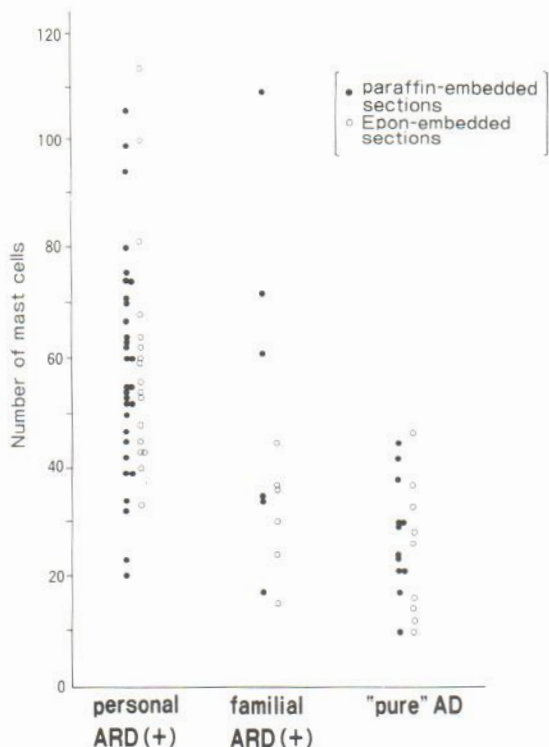


Fig. 5. Distribution of mast cells in lichenified lesions in the three subgroups of the AD patients.

that the number of mast cells in lichenified lesions of AD was strikingly increased. But these reports did not comment whether their patients had a personal or a family history of ARD.

On the other hand, Montgomery (4) concluded that the increase of mast cells was not always seen in lichenified lesions of AD patients. Braathen et al. (5) found no pathologically increased numbers of mast cells in the lesions of AD. Unfortunately, both these studies lacked an accurate description of familial background with regard to ARD in their patients.

In summary, from the results of the present study and previous reports (1, 2, 4, 5), we may conclude that the increase of mast cells often seen in lichenified lesions of AD is a feature of a subgroup of AD patients who have a predisposition for ARD.

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