ON THE SIGNIFICANCE OF THE TRICHOPTHY Tin REACTIVITY IN ATOPIC DERMATITIS

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Abstract. Patients with pure atopic dermatitis and with and without tinea infection were investigated and compared with patients by long-lasting tinea infections and with controls, for presence of intracutaneous reactivity to trichophitin, Penicillium, Cladosporium and Alternaria antigens. RAST to Penicillium and Cladosporium was also performed. The results showed a lack of delayed reactivity, but an immediate reactivity to trichophitin in 50% of atopic patients with/without tinea infection. Non-atopics infected with tinea showed 66% immediate and 33% delayed response to trichophitin. The reactivities in atopic dermatitis (but not in the non-atopic group) went general parallel with mould reactions tested intracutaneously or by RAST. It is assumed that a positive trichophitin reaction in atopic dermatitis does not necessarily mean sensitisation to dermatophytes, but is primarily the sign of a cross sensitivity to moulds.

Key words: Trichophitin: Cross reactivity: RAST: Atopic dermatitis

Several workers have established the role of immunological factors in dermatophytic infections of atopic dermatitis patients, by means of intracutaneous tests (1, 4) or other techniques (2, 10). In earlier reports we showed that delayed reactivity to trichophitin was reduced in patients with atopic dermatitis and that a cross reactivity exists between airborne moulds and dermatophytic allergens (7, 8). Similar observations have been made on asthmatic children (4). The purpose of the present study was to evaluate the occurrence of trichophitin and mould reactions in patients with and without chronic dermatophytic infections, compared with non-atopic patients with superficial mycoses, and also with controls.

MATERIAL AND METHODS

The following patient categories were investigated: (a) AD+T-group: 10 adult patients with solely atopic dermatitis (i.e. without respiratory allergic manifestations) selected by usual criteria, also suffering from chronic dermatophytosis. Tinea infection was localised in 7 cases in the interdigital space (in 6 cases Tr. rubrum, in one Tr. mentagrophytes cultures) and in 3 cases in the groins (in 2 Tr. mentagrophytes, in one E. floccosum found). (b) AD-group: 35 patients between 10 and 45 years of age, having active atopic dermatitis. (c) T-group: 15 adult patients suffering from long-lasting tinea infection with verified microscopy/culture findings: no atopy in the family or in individual history. In 7 cases the groins were involved (in four cases Tr. rubrum, in 2 Tr. mentagrophytes and in one E. floccosum found), in 7 cases the toes were involved (in 6 of them Tr. rubrum, in one Tr. tonsurans cultures) and in 1 case of axillar tinea Tr. mentagrophytes found. (d) C-group (controls): 15 adult patients with no history or clinical lesions of either atopic dermatitis or tinea infections.

All patients were intracutaneously tested with 0.1 ml of the following extract: (a) Trichophitin 1: 30 (Hollister-Stier), (b) Trichophitin 0.5% (Bencard) and, considering that Penicillia dominate indoors and Cladosporia outdoors in our geographic area, also with (c) Penicillium notatum 0.5% (Bencard), (d) Cladosporium herbarum 0.5% (Bencard), and furthermore with (e) Alternaria tenuis 0.5% (Bencard) and control solution. The reactions were read at 20 min and wheals larger than 12 mm in diameter were evaluated as positive. Delayed reactions were evaluated as positive if after 48 h the induration was at least 8 mm in diameter. RAST values were determined to Penicillium and Cladosporium spp. by the usual Phadebas technique (Doc. K. Asa, Nyco Laboratories, Oslo). Class 2-4 reactions were evaluated as positive.

RESULTS

The more important findings are collected in Table 1 and for comparison given as percentages. The details are mentioned in the following.

Trichophitin reactions. Immediate reactivity was found in 50% of the AD+T group, in 40% of the AD-group, in 66% of the T-group and none in the C-group. Positive delayed reactions were found only in the T-group, here in 33%. The reactivity to the different commercial extracts was always parallel except in one patient of the AD-group, where an immediate reaction was found only to the second extract. A positive response to both the
Table I. Intracutaneous reactions and RAST positivity in atopic dermatitis patients with Tinea infections and controls (given as percentages)

<table>
<thead>
<tr>
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<th>Intracutaneous reactivity</th>
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<tr>
<td></td>
<td>Trichophytin</td>
<td>Penicillium</td>
<td>Cladosporium</td>
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<tr>
<td>10 Patients with AD+ Tinea</td>
<td>50 0</td>
<td>50 0</td>
<td>40 0</td>
</tr>
<tr>
<td>35 Patients with AD</td>
<td>40 0</td>
<td>40 0</td>
<td>66 0</td>
</tr>
<tr>
<td>15 Patients with LL Tinea</td>
<td>66 33</td>
<td>0 0</td>
<td>6 0</td>
</tr>
<tr>
<td>15 Controls</td>
<td>0 0</td>
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immediate and delayed reactivity was registered in 12% of the T-group.

Skin reactivity to moulds. The AD+ T-group showed immediate reactions to Penicillium not., vis-à-vis 40% to Cladosporium. In the AD-group the reactivities were identical. In the T- and C-groups, a reactivity of 6% was seen to Cladosporium, vis-à-vis none to Penicillium notatum.

Skin-RAST reactivity to moulds. The immediate skin and RAST reactivity was almost identical in most patients, though with one exception: the RAST positivity in the AD-group to Cladosporium was 50%, whereas the immediate reactivity was 40%. The negative reactivity in both tests showed good correlation, too.

Trichophytin-mould cross reactivity (immediate reactions). The immediate reactivities to trichophytin and to moulds showed close similarities in the AD and AD+ T-groups in general, although minor differences were found, mostly related to the number of Penicillium not. reactions. When both the positive and negative reactivities to trichophytin and to the entire group of moulds were considered, a discrepancy was found only in 1% of the AD-group and in 10% in the AD+ T-group. By contrast, a very clear difference was registered in cross reactivity between trichophytin and moulds in the T-group, viz. 66% versus 0-6%.

DISCUSSION

According to earlier work (3) the delayed reactivity to trichophytin is related to the resistance to dermatophytic infections and persons with immediate reactivity to trichophytin are more susceptible thereto. Present investigations have again confirmed that most patients with atopic dermatitis show a lack of delayed but a frequent occurrence of immediate reactions to trichophytin. Similarly, persons with long-lasting tinea infections showed a somewhat higher immediate reactivity and only in about one-third of the cases a delayed reactivity (and in 12% reactivity to both).

Species antigens may have the effect of eliciting immediate or delayed reactivity to trichophytin and it was shown that positive delayed reactions to Tr. mentagrophytes developed in 79%, whereas to Tr. rubrum, where immediate positive reactions are commonly seen, only in 12% (1). In the present series Tr. rubrum was found in about 60% of the groups infected with dermatophytes. Furthermore, the importance of the localisation of the dermatophytic infection correlated to the occurrence of delayed reactions was recently emphasized (6).

Only minor differences were observed between immediate or RAST reactivity of the tested mould allergens. On the other hand, there was a close correlation between reactivity to moulds and to trichophytin in the atopics, but not in the non-atopic T-group, similarly to earlier findings where it was shown that 15 atopic dermatitis patients out of 19 with Cladosporium–Alternaria–Botrytis–Rhizopus reactivity reacted to trichophytin (7).

Implications of the findings for atopic dermatitis

Although the similarity found between mould and dermatophyte allergens in atopic dermatitis is obvious, it is not clear what the primary event is. Few children (0-6 years) with atopic dermatitis, who show an immediate reactivity of about 25% to moulds, yeast or house dust, get clinical infections with Tinea (7). Furthermore, asthmatic children

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10–16 years old, without evidence of tinea infections, reacted in 39% to trichophytin (4). These findings speak in favour of mould reactivity as being the first sign in atopic dermatitis and indicate that immediate reactivity to trichophytin is not necessarily a sign of contact with dermatophytes, but of a cross-response based on mould reactivity. In some atopic dermatitis cases, however, the positive trichophytin reaction is a sign of a dermatophyte infection. In the present series the AD+ T-group showed only a slightly higher rate of immediate trichophytin sensitivity than the AD-group (50% versus 40%), whereas their mould reactivities were in general identical.

It is tempting to speculate that impaired T-cell function, considered to be a primary cause of the immunological disturbances in atopic dermatitis (9) is followed by decreased resistance and associated with high immediate reactivity to moulds and by cross reactivity to dermatophytes. This may be followed by clinical dermatophyte infection with a chronic course in a proportion of atopic dermatitis patients, especially if elicited by Tr. rubrum.

REFERENCES


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