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TUBERCULIN REACTION IN ATOPIC DERMATITIS

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Abstract. Serial examinations of tuberculin reactivity were performed in 16 patients with atopic dermatitis (AD) and in 14 patients with allergic contact dermatitis. Transient suppression of already established tuberculin reactivity was seen in the contact dermatitis patients. Tuberculin reactivity in the AD patients also fluctuated with the condition of the dermatitis. When the dermatitis was active, there was diminished tuberculin reactivity. During remission of the dermatitis, however, a significant increase in tuberculin reactivity occurred. It is suggested that, in some patients with AD, the suppressed cell-mediated immunity may be secondary to the eczematous inflammatory process.

Key words: Tuberculin reactivity; Cell-mediatied immunity

Patients with atopic dermatitis (AD) often show diminished tuberculin reactivity (2, 4). However, it still remains to be determined whether the hypo-reactivity is primary or secondary. I previously reported (5) that, in patients with AD as well as in those with allergic contact dermatitis, their tuberculin reactivity was diminished when the dermatitis was active, and that a significant increase of their tuberculin reactivity occurred while they were in remission or free from the dermatitis.

In the present study, the tuberculin reactivity in each of the patients with atopic dermatitis was examined at the time of exacerbation, during the subsequent remission, and at the re-exacerbation of the dermatitis.

MATERIALS AND METHODS

Patients. A total of 16 patients (age range 16–47 years) suffering from widespread AD, were selected. They all showed seasonal fluctuation in the severity of their dermatitis, which worsened in winter and improved in summer. They received local corticosteroids. Systemic use of corticosteroids was avoided.

Clinical controls consisted of 14 adult patients with widespread allergic contact dermatitis. They too were treated with topical application of corticosteroids.

Skin tests. Each patient was injected intradermally with 0.1 ml of a tuberculin solution (0.5 μg of PPD per 1 ml). The test site was normal-appearing skin on the flexor surface of the forearm. The reaction was read at 48 hours.

To see if a widespread eczematous dermatitis influenced already established tuberculin reactivity, each of the AD patients was tested at the time of the exacerbation of the dermatitis, while the patient was in remission for a period of 4 weeks or more, and when the re-exacerbation of the dermatitis occurred. Patients with allergic contact dermatitis were tested when the dermatitis was active and during a period of 2 to 4 weeks after the disappearance of the dermatitis.

RESULTS

Patients with allergic contact dermatitis. When they had active dermatitis, the mean value of their tuberculin reactions was 3.36 ± 3.49 mm S.D. When tested after the subsidence of the dermatitis, the mean value of their tuberculin reactivity strength was 12.57 ± 6.05 mm S.D. Thus, a widespread allergic contact dermatitis brought about a significant suppression of tuberculin reactivity ($P < 0.01$).

Patients with atopic dermatitis. When they had florid dermatitis, the mean value of their tuberculin reactions was 4.88 ± 5.03 mm S.D. When tested during a period of remission, the mean tuberculin reaction strength was 12.37 ± 10.59 mm S.D. Thus, a significant increase in tuberculin reactivity occurred during the remission of the dermatitis ($P < 0.01$). At the time of re-exacerbation of the dermatitis, the mean value of tuberculin reactions was 5.50 ± 6.12 mm S.D. This was significantly low when compared with the tuberculin reactivity strength during the preceding remission ($P < 0.01$).

DISCUSSION

Transient suppression of tuberculin reactivity was seen in patients with widespread allergic contact dermatitis. Tuberculin reactivity in patients with widespread AD also fluctuated with the condition of the dermatitis. When there was exacerbating AD, they showed diminished tuberculin reactivity. During remission of the dermatitis, however, a significant increase in tuberculin reactivity occurred.

Thus, it is apparent that a widespread eczematous inflammation of varying cause transiently suppresses already established tuberculin reactivity. This association of eczematous inflammation and anergy indicates that, in the evaluation of cell-mediated immunity in patients with atopic dermatitis, it is important to investigate them not only when the dermatitis is active but also while they are in remission.

Several investigators (1, 3) report that the lymphocyte hyporeactivity to PHA often seen in AD patients may normalize during remission of the dermatitis. Therefore, it is possible that, at least in some patients with AD, the diminished cell-mediated immunity may be secondary to the eczematous inflammatory process.

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DISCUSSION

Barnetson (Edinburgh). Q: We have done similar studies and emphatically confirm your observations. We have been studying the lymphocyte transformation responses to *S. aureus* using two different preparations and we have found that during exacerbations of the eczema the responses are greatly diminished and that when the patients go into remission they come up again into the normal range. This does seem to suggest that some sort of non-specific mechanism of anergy comes into play during exacerbations of the eczema and this presumably is something different from the T-cell deficiency which we have all heard described.

Hanifin (Portland). Q: Did you have any patients who were not treated with topical steroids?

A: My patients were treated with topical corticosteroids, but earlier I made guinea pig experiments without using corticosteroids.

Rorsman (Lund). Q: Dr Uehara, do you think there is a vascular background for the phenomenon of the reduced tubeculin reaction? Seeberg & Magnusson found 30 years ago that not only in inflammatory edema but also in cardiovascular edema there was a greatly reduced tuberculin reaction. Furthermore, they observed that if one had an immediate reaction to tuberculin—as one may have sometimes—then one got a very weak response of delayed type because of rapid absorption of the antigen, due to the immediate response.

Wahlberg (Stockholm). Q: Have you considered applying primary irritants at the same time as tuberculin?

A: In an earlier study I used croton oil and formalin as primary irritants. Delayed skin reactions to these substances were quite similar in atopic dermatitis and normal controls.