

## Bronchial Asthma and Allergic Rhinitis in Patients with Hereditary Palmoplantar Keratoderma

Sir,

More than 100 years ago it was observed that inhalation of fungi could induce bronchial asthma (1). Since then, such reactions have repeatedly been reported and shown to be correlated with positive immediate skin tests and presence of specific dermatophyte IgE antibodies (2, 3). The prevalence of bronchial asthma, reported in a northern county of Sweden, was 5–7% and that of allergic rhinitis among military service men 13–15% (4). As the prevalence of dermatophytosis in patients with hereditary palmoplantar keratoderma (1982–1987) was reported to range between 36% and 42%, it was considered of interest to study the prevalence of bronchial asthma and allergic rhinitis in 207 patients with hereditary palmoplantar keratoderma, compared to that of the population of the county (5).

Dermatophytes were isolated on Sabouraud's glucose agar from 83 of the 207 patients with hereditary palmoplantar keratoderma, and they were distributed as follows: *T. rubrum* 42%, *T. mentagrophytes* 35% and *E. floccosum* 23% (Table I). At the clinical examination a family and/or personal history of atopy was obtained in 37% of all patients. Fifty-one per cent of those had dermatophytosis. Of those patients without a family and/or personal history of atopy dermatophytes were isolated in 15%. Serum IgE levels > 100 IU/ml, determined by using the Phadebas radioimmunoassay method (Pharmacia, Sweden), was found in 17 of all patients, of whom 14 had dermatophytosis. Dermatophyte infections in patients with bronchial asthma, allergic rhinitis and urticaria together with serum IgE levels are shown in Table I.

A commercial antigen prepared on extract from *T. mentagrophytes* (Trichophyton<sup>®</sup>, Miles and Dome, UK) and a specially prepared antigen extract from *T. rubrum* (National Bacteriological Laboratory (SBL) Stockholm, Sweden) gave, however, only 2 certain positive immediate and 4 doubtfully positive reactions, when intradermally injected (I+II) (Table I).

### COMMENTS

The prevalence of bronchial asthma among 207 patients with hereditary palmoplantar keratoderma was 5.5%, corresponding well to the prevalence of the population of the county at that time. However, the prevalence of bronchial asthma among those 83 patients who had hereditary palmoplantar keratoderma and dermatophytosis was 18.0% and among those without dermatophytosis 2.3% ( $p < 0.05$ ). For the present statistical calculation the chi square test was applied. The prevalence of allergic rhinitis was also raised compared to that of the population (Table I). A family and personal history of atopy also confirms previously performed studies of increased susceptibility to dermatophyte infections in patients with atopy (6, 7).

Two possible pathways for dermatophyte antigen have been proposed, either through eczematous skin or by the airways (8). In patients with hereditary palmoplantar keratoderma, dermatophyte infections are confined to the hyperkeratosis and do not propagate to other parts of the skin, probably due to different immunological mechanisms (9). Mycotic material may pass through the airways, leading to production of specific IgE antibodies. Bronchial asthma and allergic rhinitis seem to occur more often among patients with hereditary palmoplantar keratoderma and dermatophytosis than among the population of the county and among patients without dermatophytosis; however, more extensive studies of these patients concerning the prevalence of IgE-mediated diseases should be performed.

### REFERENCES

- Blacey CH. Experimental researches in the cause and nature of catarrhus aestivus (hay fever or hay asthma). London: Dawson Publishing Co. (reprinted from Bailliere Tindall & Cox, 1873), 1959: 57–58.
- Wise F, Sulzberger MB. Urticaria and hay fever due to *Trichophyton* (*Epidermophyton interdigitale*). JAMA 1930; 95: 1504–1508.
- Ward GW, Karlsson G, Rose G, Platts-Mills TAE. *Trichophyton*

Table I. Patients with hereditary palmoplantar keratoderma with or without dermatophytosis, related to the parameters investigated *T. rubrum* (TR), *T. mentagrophytes* (TM), *E. floccosum* (EF). Figures indicate number of patients.

	All patients	No. of patients with dematophytes			
		TR	TM	EF	Total
Patients	207	35	29	19	83
Family and/or personal history of atopy	77	13	14	12	39
Patient without family and/or personal history of atopy	130	23	15	7	44
Serum IgE > 100 IU/ml	17	6	5	3	14
Serum IgE < 100 IU/ml	190	29	24	16	69
Bronchial asthma	11	3	2	4	9
Allergic rhinitis	13	1	2	2	5
Urticaria	8	0	0	0	0
Trichophyton test (I+II)	207	1	1	0	2

- asthma: sensitisation of bronchi and upper airways to dermatophyte antigen. *Lancet* 1989; 1: 859–862.
4. Lundbäck B. Asthma, chronic bronchitis and respiratory symptoms: prevalence and important determinants. The obstructive lung disease in Northern Sweden. Study I. Umeå University Medical dissertations, 1993.
  5. Gamborg Nielsen P. The prevalence of dermatophyte infections in hereditary palmoplantar keratoderma. *Acta Derm Venereol (Stockh)* 1983; 63: 439–441.
  6. Jones HE, Reinhardt JH, Rinaldi MG. Immunologic susceptibility to chronic dermatophytosis. *Arch Dermatol* 1974; 110: 213–220.
  7. Hanefin JM, Ray LF, Lobitz Jr WC. Immunological activity in dermatophytosis. *Br J Dermatol* 1974; 38: 222–250.
  8. Davies RR, Ganderton MA, Savage MA. Human nail dust and precipitating antibodies to *Trichophyton rubrum* in chiropodist. *Clin Allergy* 1983; 13: 309–310.
  9. Gamborg Nielsen P, Faergemann J. Dermatophytes and keratin in patients with hereditary palmoplantar keratoderma. A mycological study. *Acta Derm Venereol (Stockh)* 1993; 73: 416–418.

*Accepted January 9, 1995.*

P. Gamborg Nielsen  
Clinic of Dermatology and Venereology, Alegränd 1, S 31134  
Falkenberg, Sweden.