

The association of perennial rhinitis with *Trichophyton* infection

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Summary

Trichophyton was recently reported to be the cause of respiratory allergy in patients with severe bronchial asthma. We describe eight patients with perennial rhinitis in combination with skin or toe-nail infection, in whom Type I hypersensitivity reaction to *Trichophyton* was confirmed by skin test, RAST and nasal provocation. When treated with oral fungicidal therapy, there was significant improvement in both their skin and nasal symptoms. Local therapy produced only a mild, transient improvement. We emphasize the need for thorough evaluations in patients with respiratory allergies and negative routine skin tests.

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Introduction

Dermatophyte sensitivity associated with either asthma or urticaria has been described [1–4]. Immediate hypersensitivity to dermatophyte antigens may be common in patients with severe fungal infection [5]. Recently, Ward *et al.* [6] described 12 patients with asthma, dermatophyte infection and immediate hypersensitivity to *Trichophyton*. In eight of these patients, nasal challenge to the allergen induced symptoms.

We describe an additional eight patients in whom only perennial rhinitis with immediate hypersensitivity skin response occurred. In five of the patients, nasal provocation test further confirmed the association between *Trichophyton* infection and allergic rhinitis.

Subjects and methods

Over the last three years, we included mixed *Trichophyton* extracts (Hollister-Stier) to our extended panel of prick skin tests which encompassed 50 different allergens. These consisted of the following groups: grass, weeds, trees, mould, dust, mite, cockroach, and epidermals. *Trichophyton* mixture is prepared from the filtrates of mixtures of 21-day maltose broth cultures of *Trichophyton mentagrophytes*, *rubrum* and *tonsurans*.

Patient selection was based on allergic rhinitis with runny nose and sneezing. All the patients presented with

moderate to severe perennial symptoms and were referred for allergy evaluation. There were 5 males and 3 females, with a mean age of 30 years. Ten patients, without respiratory allergies and who had *Trichophyton* skin infection served as control. Spirometry (Fukuda spirometry), performed to exclude airway disease, was normal in all patients ($FEV_1 > 80\%$ of predicted).

Nasal provocation

After careful examination of the nasal passage, nasal challenge was performed in a double blind placebo controlled study. *Trichophyton* mix (0.2 ml) or placebo were sprayed into the nostrils starting with 10^{-6} w/v increasing up to 10^{-2} , and allowing 20 min between each challenge. The number of sneezes was counted and the degree of congestion and secretion was estimated by the patients on a scale of 0–4; the final score being the total of the three symptoms.

Radioallergosorbent test

Serum IgE antibodies to *Trichophyton* were examined by radioallergosorbent test, with *Trichophyton tonsurans* extract linked to solid phase matrix and alkaline phosphatase as an enzyme (Meljia Diagnostik, Kassel, Germany). Results were expressed as RAST class (from 0 to +4). Skin culture for moulds was taken from all the patients at the site of infection (skin, nails). Analysis of variance was used to evaluate the response to nasal provocation.

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Table 1. Characteristics of eight patients with perennial allergic rhinitis and positive skin test to *Trichophyton*

No.	Age (years)	Sex	Skin response weal (mm)	RAST class	Site of fungal infection	Treatment	Response
1	20	F	10/6	+++	Nails	Griseofulvin	Moderate
2	26	M	5/7	+++	Nails	Griseofulvin	Mild
3	32	M	10/8	ND	Skin, nails	Griseofulvin	Moderate
4	27	M	7/5	ND	Skin, nails	—	
5	28	F	10/9	+++	Nails	Griseofulvin	Moderate
6	42	M	5/6	ND	Skin	—	
7	21	F	8/6	+++	Skin	—	
8	45	M	5/5	ND	Nails	—	

$\bar{X}$ 30. $\pm$ s.d. 9. ND, not done. IgE, immunoglobulin E to *Trichophyton tonsurans*.

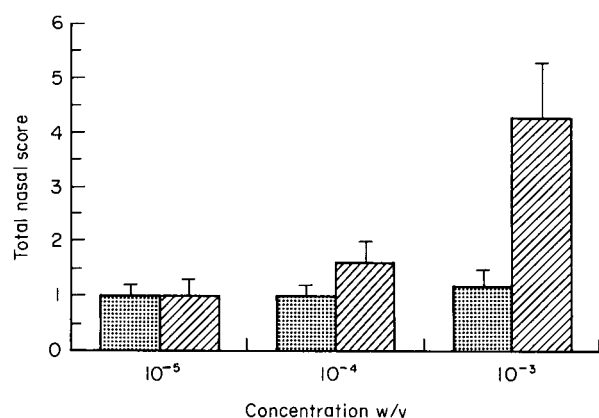


Fig. 1. Double-blind placebo control nasal challenge with *Trichophyton* in five patients. Significant difference was seen only at concentration of 10^{-3} w/v *Trichophyton* ($P < 0.01$). ■, placebo. ▨, *Trichophyton*.

Results

In Table 1, we summarized the patients' characteristics, skin test results, RAST class, and site of infection. None of the patients had clinical sinusitis or nasal polyps. Sinus X-rays performed on three patients were normal. The patients had no skin response to allergens other than *Trichophyton*. Cultures grown from the infected skin and toe nails of all the patients were *Trichophyton* positive. Four of the patients had positive RAST class (mean reading of 0.66 optical density).

Nasal response to provocation with *Trichophyton* extract was tested in five of the patients (1, 2, 4, 5, 6) and is presented in Fig. 1. There was a significant increase in symptoms after challenge with 10^{-3} w/v *Trichophyton* extract ($P < 0.01$). Four patients with widespread nail or skin infections were given griseofulvin, and significant

improvement was seen in both skin infection and nasal symptoms, 2–3 months later. One of the patients relapsed while on therapy. Two of the other patients were treated locally with miconazole and reported mild improvement in the nasal symptom score.

Skin test to *Trichophyton* was repeated in all the patients six months later, with no significant change in weal size. The results in the control subjects (10 patients) are as follows. All had positive *Trichophyton* cultures grown from either skin or nails; none had allergic respiratory symptoms, in one patient an immediate skin response to *Trichophyton* was seen (4×5 mm). In four of the control group, nasal provocation, with *Trichophyton* extracts up to a concentration of 10^{-2} w/v, did not induce symptoms. RAST test was not done in the control group.

Discussion

Trichophyton infection of skin and nails is common in adults, but frequently induces type I hypersensitivity reaction in patients with more severe fungal infection [5,7]. Over the last few years, a few articles have described patients with supposedly *Trichophyton*-induced allergic respiratory symptoms [1–4,6]. In the majority of these patients, there were concomitant allergens (mite, dog, cat and fungi) [6]. Thus far, *Trichophyton* sensitivity has been described in association with bronchial asthma (usually of late onset), as well as rhinitis [6]. These reactions represent a systemic response to the fungi. Our group of patients is unique in that perennial rhinitis was found to be related to chronic skin and nail infection which induced type I hypersensitivity. Since *Trichophyton* is present in the air in small quantities [8], one cannot exclude it as a cause for sensitization. Our group is also younger than the group of bronchial asthma patients

described by Ward *et al.* (mean of 30 years *vs.* 47 years). RAST test was positive in four patients with immediate weal response to *Trichophyton* skin test and, therefore, may be contributory in the diagnosis of Type I hypersensitivity.

In asthmatic adults treated in a hospital emergency room, 2% had IgE antibody to *Trichophyton* [6]. Data from patients with chronic rhino sinusitis is unavailable. In addition to the patients described by us with isolated sensitivity to *Trichophyton*, approximately twice as many are followed-up in our clinic for perennial rhinitis and sensitivity to multiple allergens, including *Trichophyton*.

Nasal provocation induced symptoms in five patients in the group, supporting the association between *Trichophyton* infection and nasal symptoms.

In asthma patients, fungicidal therapy was found to be helpful. Patients were referred to our clinic, since they failed to respond to antihistamine or nasal beclomethasone. Four patients in whom fungal infection had spread to the skin and nails, were treated with oral fungicidal therapy, with significant improvement in both their skin and nasal symptoms. The other two who received only local therapy, had only a mild, transient improvement. These findings support the association between fungal infection and nasal symptoms. Immunotherapy to *Trichophyton* was initiated in two other patients without benefit after 4–6 months. This could be due to the fact that the allergen remained on the skin, continuing to induce symptoms.

In summary, *Trichophyton* infection of the skin and nails can induce type I hypersensitivity reaction, and may also manifest itself as perennial rhinitis, as well as asthma

[6]. Further studies are needed in order to examine the role of fungal infection in patients with allergic respiratory diseases.

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