Atopy as a risk factor for dermatophytoses

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الخلاصة

التضاد بين المناعة الخلوية والمناعة الخلطية والذي ينتج في مرضى الحالة التأثبية قد يكون سببا في زيادة تحسس هولاء المرضى للأصابة بعدوى الفطار الجلدي. شملت الدراسة مئة وواحد مريض في الفترة الممتدة من أيلول 2002 الى أيلول 2003 للكشف عن وجود علاقة بين الحالة التأثبية للمرضى وزيادة التحسس للأصابة بعدوى الفطار الجلدي لنفس المرضى. لقد تم التوصل الى أن الحالة التأثبية للمرضى لاتعمل على زيادة التحسس للأصابة بعدوى الفطار الجلدي وبمختلف أنواعه ولكنها تزيد حساسية هؤلاء المرضى للأصابة بعدوى الفطار الجلدي المزمن عن الأصابة بعدوى الفطار الجلدي الحاد.

ABSTRACT

Antagonism between cell-mediated immunity and humoral immunity that develops in atopic patients may be correlated with susceptibility of those patients to dermatophytoses. One hundred and one patients were included in this study from September 2002 to September 2003 to discover the association between atopy and dermatophytoses. The atopic status of patients did not act as a predisposing factor for different types of dermatophytoses, but it increased the susceptibility of such infections to become chronic rather than acute.

INTRODUCTION:

It is not clear if chronic dermatophytoses stimulate IgE production or if this condition tends to develop in persons who are already atopic. However, many patients with chronic dermatophytoses neither are atopic nor manifest immediate hypersensitivity to trichophytin, indicating that other factors must also be involved in producing susceptibility to this type of infection[(1)].

Extracts of various dermatophyte species have usually been found to contain a mixture of antigens that are either species specific or broadly cross-reactive with those of other dermatophytes or those of other fungi. This phenomenon may relate to the susceptibility of atopic patients to chronic dermatophytoses because these patients may develop immediate hypersensitivity to airborne molds that cross-react with the dermatophyte antigens and this immediate hypersensitivity may later interfere with the development of delayed responses to these antigens. Subjects with immediate hypersensitivity to trichophytin have been found to be more easily infected experimentally with Trichophyton mentagrophytes than are those without this type of response. Therefore, it is possible that IgE response in some patients with chronic dermatophytoses may inhibit the development of protective cell-mediated immune responses to dermatophyte antigens[(1)].

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Neonatal exposure to the fungus or the cross-reacting antigens of molds may induce tolerance by confusing antigen recognition of self versus nonself[2].

The study tried to investigate that if the atopic status of the patient acts as predisposing factor for dermatophytoses and increases the susceptibility to the chronic type instead of the acute type of the infection.

MATERIALS & METHODS:

This study included specimens collected from 101 patients attending the Dermatology Outpatient Clinic of Al-Kadymia Teaching Hospital in Baghdad, Iraq from September 2002 to September 2003.

The collected specimens were as skin scrapings, hair clippings, and nail clippings and scrapings.

Patient's questionnaire for baseline data included: Type of infection, systemic disorder like atopy, infection history like acute or chronic infection, any pervious fungal infection.

All specimens showing fungi on direct examination by KOH preparation were cultured on Sabouraud's dextrose agar medium. Cultures were incubated at 25-30C and were examined weekly. Negative plates were kept for at least three weeks before discard. Readings were made between the second and third weeks after inoculation[3].

RESULTS:

Patients' questionnaire revealed that thirty-seven out of the 101 patients (36.63%) were atopic patients with different allergic diseases.

The atopic patients showed no significant differences with the clinical aspect of dermatophytoses, i.e.: either acute or chronic infection and in turn the susceptibility to dermatophytoses (P=0.24), (Table 1). But still the number of atopic patients with chronic dermatophytoses was more than those with acute dermatophytoses, 20 and 17 patients, respectively.

Table 1 . Correlation between atopy and acute or chronic dermatophytoses.

Atopy	Acute infection	Chronic infection	P value
Yes	17	20	0.24
No	37	27	

As is in the susceptibility to dermatophytoses the atopic patients showed no significant correlation with the incidence of any previous fungal infection (P=0.26), (Table 2).

Table 2 . Correlation between atopy and previous fungal infection (superficial mycoses).

Atopy	Yes	No	P value
Yes	25	12	0.26
No	36	28	

The atopy as a risk factor showed no significant association with the type of the causative agents isolated from the clinical specimens (P=0.17), (Table 3).

Table 3 . Correlation between atopy and isolate causative agents for fungal infections.

Atopy	Candida albicans	Trichophyton rubrum	Microsporum canis	others	P value
Yes	9	10	3	4	0.17
No	14	9	8	16	

DISCUSSION:

Personal or family history of atopy was present in approximately 50% of the chronic Trichophyton rubrum patients, which suggested chronic dermatophytoses might develop due to an immunological predisposition to develop humoral immunity[4].
Johns et al.[4], in their following table showed a schematic sequence of immunologic responses to dermatophyte infection that may lead to chronic infection :

Antigen Challenge	Primary response	Intermediate response	Late response
Dermatophyte Infection (fungal antigen)	CMI	CMI +/- Humoral Immunity (+/- IgE)	CMI Unresponsiveness +/- Humoral Immunity (+/- IgE)

From our study we demonstrated that the atopic status did not have any predisposing effect for different types of dermatophytoses. It also seemed that atopic state did not have a predisposing effect on any previous fungal infection (superficial mycoses); and the isolated fungus as a causative agent as well, i.e. atopic patient previously had fungal infection like onychomycosis was not susceptible to be infected with the same clinical picture or the same causative agent for this fungal infection. In spite of, the non-significant statistical result between the atopic status and the chronically and acutely infected patients, the number of patients with chronic infection was more than the number of patients with acute infection. This supported the previous hypothesis demonstrated by the table showed that the location of the humoral immunity that may predispose to chronic infection was within the intermediate and late response and not within the primary response that may predispose to the susceptibility to the infection.
Escalante et al., in 2000[5], supported this hypothesis by their result that trichophytosis caused by T.rubrum was not more prevalent in atopic than non-atopic subjects; atopic diseases were not more frequent in culture-positive than in culture-negative patients.

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