Acta Dermatovenerol Croat 2006;14(2):86-89 CLINICAL ARTICLE

The Role of *Dermatophagoides pteronyssinus* in Atopic Dermatitis

Ilko Kuljanac

Department of Dermatology and Venereology, Karlovac General Hospital, Karlovac, Croatia

Corresponding author:
Ilko Kuljanac, MD, MS
Department of Dermatology and Venereology
Karlovac General Hospital
A Štampara 3
HR-47000 Karlovac
Croatia
ilko.kuljanac@ka.t-com.hr

Received: December 20, 2005
Accepted: April 20, 2006

SUMMARY The role of *Dermatophagoides (D.) pteronyssinus* in atopic dermatitis (AD) was investigated by use of skin prick test (SPT) and total and specific IgE (RAST) to *D. pteronyssinus*. The study included 43 patients (17 male and 26 female), mean age 42.3 (range 19-77) years. All study patients met the Hanifin and Rajka criteria. Patients were divided into two groups: "pure" AD (n=27; 12 male and 15 female), mean age 46.3 (range 19-77) years; and AD with respiratory symptoms (AD+RS, n=16; 5 male and 11 female), mean age 38.4 (range 17-75) years. Control group consisted of 15 healthy subjects (7 male and 8 female; mean age 49.0, range 24-64 years), with no personal or family history and signs of atopy. Both patient groups had a higher total serum IgE than control subjects ($p<0.05$). In the "pure" AD group, SPT was positive in 5/27 (18.5%) and RAST to *D. pteronyssinus* in 4/27 patients. In the AD+RS group, SPT was positive in 10/16 (62.5%) and RAST to *D. pteronyssinus* in 8/16 (50%) patients. Concordance between SPT and RAST was observed in both groups; 80% of SPT positive patients were RAST positive. *D. pteronyssinus* was found to play an important role as a trigger factor in AD patients.

KEY WORDS: *Dermatophagoides pteronyssinus*; atopic dermatitis; skin prick test; specific IgE

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease associated with cutaneous hypersensitivity to environmental triggers, and is often the first step in the march that results in asthma and allergic rhinitis (1). Atopic and allergic diseases have been on an increase for several decades (2). The lifetime prevalence of AD is 10%-20% in children and 1%-3% in adults (1).

There is increased evidence that T-cell response to environmental allergens is important in the pathogenesis of AD. In these patients, AD is most often associated with the existence of environmental allergen-specific IgE (3). Detection of IgE antibodies is essential to confirm the diagnosis of allergy, to identify specific sensitivities, and to guide the treatment (4). Skin prick testing (SPT) is a conventional way of testing the presence of allergen-specific IgE, detecting IgE bound to the surface of mast cells in the skin (5). SPT is the most common screening method for allergen evaluation (6). Hypersensitivity to house dust mite (HDM) antigens is found in 5% of all people in western nations, and in up to 90% of atopic patients suffering from allergic bronchial asthma or AD (7).

The aim of the study was to investigate the role of *Dermatophagoides (D.) pteronyssinus* in AD with and without respiratory symptoms (RS) using SPT, specific IgE (RAST) to *D. pteronyssinus* and total IgE, in comparison with healthy subjects. Also, correlation between these tests was assessed.

All subjects involved in the study gave their informed consent and the study was granted approval from the hospital Ethics Committee.

SUBJECTS AND METHODS

The study included 43 AD patients (17 male and 26 female) aged 19-77 (mean age 47.7) years, meeting the Hanifin and Rajka criteria (8). Patients were divided into two groups: "pure" AD (n=27; 12 male and 15 female; mean age 47.7 years), and AD with RS ("mixed") (n=16; 5 male and 11 female; mean age 40.4 years) (Table 1).

Control group consisted of 15 healthy subjects (7 male and 8 female; mean age 49.0, age range 24-64 years) with no personal and family history and signs of atopy.

Total IgE was measured with Imx-test (Microparticle Enzyme Immunoassay-MEIA, Abbott Laboratories, USA); normal value is <120 IU/mL). Specific IgE (RAST) was measured with CAP-RAST system (Pharmacia, Uppsala, Sweden). Levels of specific serum IgE >0.7 were considered positive. SPT with *D. pteronyssinus* was performed on the volar part of the forearm on intact non-steroid treated skin. The concentration used in SPT was 3,000 biological units/mL (BU/mL), obtained from the Institute of Immunology, Zagreb, Croatia. Positive (histamine chloride 10 mg/mL) and negative control were used. Skin reactions were read after 15 minutes and reactions <3 mm in diameter were considered negative.

Statistical analysis was performed by use of χ^2-test.

Table 1. Patients with atopic dermatitis included in the study

Sex \ Diagnosis	AD	AD + RS	Total
M	12	5	17
F	15	11	26
Total	27 (62.8%)	16 (37.2%)	43 (100%)

AD, atopic dermatitis; RS, respiratory symptoms

Table 2. Total serum IgE in atopic dermatitis patients and control group

No.	Diagnosis	n	X±SD	p<0.05
1	AD	27	284.4±264.0	S
2	AD+RS	16	531.1±563.3	S
3	Control group	15	72.5±47.9	

AD, atopic dermatitis; RS, respiratory symptoms; X, mean score; SD, standard deviation; S, significant difference

RESULTS

In both patient groups, total serum IgE level significantly exceeded that measured in the control group (p<0.05), however, the difference between the patient groups (AD vs. AD+RS) was not statistically significant (p>0.05) (Table 2). SPT was positive in 5/27 (18.5%) and RAST in 4/27 (14.8%) AD patients. One AD patient was SPT+ and RAST negative. SPT positive in 10/16 (62.5%) and RAST in 8/16 (50%) AD+RS patients. Two AD+RS patients were SPT positive and RAST negative. Concordance between SPT and RAST was observed. Considering all study patients, SPT was positive in 15/43 (34.8%) and RAST in 12/43 (27.9%) patients (Table 3). SPT and RAST to *D. pteronyssinus* were not positive in any of control subjects.

Table 3. Results of skin prick test and specific IgE in atopic patients

Test \ Disease	AD	AD+RS	All patients n	(%)	Total
SPT +	5	10	15	(34.8)	43
SPT -	22	6	28	(65.1)	
RAST +	4	8	12	(27.9)	43
RAST -	23	8	31	(72.1)	

SPT, skin prick test; RAST, specific IgE; AD, atopic dermatitis; RS, respiratory symptoms

DISCUSSION

The occurrence of sensitization in genetically predisposed individuals is probably predominantly determined by the levels of environmental allergens (9). It is generally accepted that AD may aggravate following skin exposure to aeroallergens such as house HDM, animal dander, and pollen (10). Hypersensitivity to HDM antigen is found in 5% of all people in western nations, and in up to 90% of atopic patients suffering from allergic bronchial asthma or AD (7).

Skin testing and *in vitro* testing for IgE by RAST are used to confirm the clinical finding of allergic triggers obtained by history, and not for diagnosis (11). Eighty-two AD patients were examined for specific IgE by RAST. A higher number of positive RAST reactions were found in patients with high serum IgE values and coexisting asthma and/or allergic rhinitis. Positive RAST reactions against mite and house dust were found in many of these patients and in about one-half of patients with AD only. Comparison between intradermal tests and RAST done with various allergens yielded best between-test agreement with mite extract (12).

The relation between serum levels of allergen, specific IgE (RAST) and SPT reactivity, and allergic disorders was evaluated in 137 subjects randomly selected from an adolescent population. All subjects were prick tested with six common allergens. The correlation between prick test and RAST results was better with pollen than with HDM and animal epithelia. Respiratory allergy was closely connected with both positive skin test and RAST reactivity, while AD showed lower correlation (13).

Chinoy *et al.* (2005) compared skin test reactivity and serum specific IgE antibodies to common indoor allergens in patients with respiratory allergies. In the present study of patients with respiratory allergies, the skin test and RAST results showed moderate concordance to the common indoor allergens (*D. farinae,* cockroach mix, cat epithelium and dog epidermal cells). Skin testing, particularly when percutaneous skin testing was supplemented with intradermal tests, was more sensitive than Phadebas RAST in the identification of four indoor allergens assessed in our study (6).

In our study total serum IgE was higher in both patient groups as compared with control subjects ($p<0.05$). There was no statistically significant difference between patient groups, which is consistent with literature data (14,15). The correlation between prick test and RAST results was better with pollen than with HDM. Respiratory allergy was closely connected with both positive skin test and RAST reactivity, while AD showed lower correlation (13). Mite specific IgE were detected by prick tests in 24% of AD patients and only 13% of controls subjects (16). A high, 97% concordance was observed between ID testing and RAST for *Dermatophagoides farinae*. Skin test was negative in one of 30 patients with positive RAST (17). Higher positive results of SPT and RAST were recorded in our AD patients with respiratory symptoms: SPT 62.5% and RAST 50%. Concordance between SPT and RAST was comparable in the two patient groups; 80% of SPT positive patients were RAST positive. Our results were not unexpected because the association between HDM sensitization and asthma has been repeatedly confirmed (18). In our study SPT was more sensitive than RAST in AD patients, as also reported elsewhere (6). SPT remains more sensitive and more specific than *in vitro* tests for specific IgE. In some cases RAST may be preferable to SPT, e.g., in extensive skin disease, dermographism, risk of anaphylaxis, etc. Our results as well as other literature reports (2,19) indicate a major role of *D. pteronyssinus* as a trigger factor in AD patients; thus, SPT, total and specific IgE to *D. pteronyssinus* (and other allergens) should be done in patients with AD.

CONCLUSION

D. pteronyssinus is a very important provocation factor in AD patients. SPT is the most common screening method for allergic evaluation and identification of the causative allergen; however, specific IgE and total IgE should also be done. The sensitivity and specificity as well as concordance of these test methods vary according to the causative allergen and type of allergic disease. In AD patients (especially those with respiratory symptoms) and *D. pteronyssinus* as a causative allergen, these tests produce satisfactory results.

References

1. Leung MYD, Boguniewicz M, Howell DM, Nomura I, Hamid AQ. New insights into atopic dermatitis. J Clin Invest 2004;113:1070-88.
2. Milavec-Puretić V, Lipozenčić J, Žižić V, Milavec D. Correlation among skin prick test, total and specific IgE Uni CAP tests in atopic patients from Zagreb, Croatia. Acta Dermatovenerol Croat 2004;12:257-60.
3. Werfel T, Kapp A. Environmental and other major provocation factors in atopic dermatitis. Allergy 1998;53:731-9.
4. Trombone AP, Tobias KR, Ferriani VP, Schuurman L, Aalberse RC, Smith AM, *el al*. Use of chimerie ELISA to investigate immunoglobulin E antibody response to Der p 1 and Der p 2 in mite-allergic patients with asthma, wheezing and/or rhinitis. Clin Exp Allergy 2002;32: 1323-8.
5. O'Brien MR. Skin prick testing and in vitro assays for allergic sensitivity. Austr Prescr 2002;25:91-3.

6. Chinoy B, Yee E, Bahnal S. Skin testing versus radioallergosorbent testing for indoor allergens. Clin Mol Allergy 2005;3:4-12.
7. Sporik R, Holgate ST, Platts-Mills TA, Cogswell JJ. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. N Engl J Med 1990;323:502-7.
8. Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. Acta Derm Venereol (Stockh) 1980;114:146-8.
9. Friedmann SP. The role of dust mite antigen sensitization and atopic dermatitis. Clin Exp Allergy 1999;29:869-72.
10. Bygum A, Moriz CG, Andersen KE. Atopy patch test in young adult patients with atopic dermatitis and controls: dose response relationship, objective reading reproducibility and clinical interpretation. Acta Derm Venereol 2003;83:18-23.
11. Ahuja A, Land K, Barnes JC. Atopic dermatitis. South Med J 2004;96:1068-72.
12. Takahashi I, Akiyama T, Yamura H, Sasaoka K, Anan S. Evaluation of RAST in atopic dermatitis. J Dermatol 1977;4:217-21.
13. Haahtela T, Jaakonmaki I. Relationship of allergen-specific IgE antibodies, skin prick tests and allergic disorders in unselected adolescents. Allergy 1981;36:251-6.
14. Lugović L, Lipozenčić J. Are respiratory allergic diseases related to atopic dermatitis. Coll Antropol 2000;1:335-45.
15. Czarnecka-Operacz M, Bator-Wegner M, Silny W. Atopy patch reaction to airborne allergens in the diagnosis of atopic dermatitis. Acta Dermatovenerol Croat 2005;13:3-16.
16. Seidenari S, Giusti F, Pellacani G, Bertoni L. Frequency and intensity of response to mite patch tests are lower in nonatopic subjects with respect to patients with atopic dermatitis. Allergy 2003;58:426-31.
17. Tang RB, Wu KK. Total serum IgE, allergy skin testing, and the radioallergosorbent test for the diagnosis of allergy in asthmatic children. Ann Allergy 1989;62:432-5.
18. Platts-Mills TA, de Weck AL. Dust mite allergen and asthma: a worldwide problem. J Allergy Clin Immunol 1989;83:416-20.
19. Holm L, van Hage-Hamsten M, Öhman S, Scheyns A. Sensitization to allergens of house-dust mite in adults with atopic dermatitis in a cold temperate region. Allergy 1999;54: 708-15.

Nivea cream before exposure to fresh air and sun, year 1934.
(from the collection of Mr. Zlatko Puntijar)