

IgE antibodies to mites in Saudi Arabia

THE CITY OF TABUK is situated in the far northwest corner of the quadrangular Kingdom of Saudi Arabia. It is about 2500 m above sea level and located in a flat, savannah-like region with a Mediterranean climate. The town is dry, with air-conditioned houses, upholstered with soft, padded furniture, creating a habitat favorable to mites. The inhabitants have come to associate living in this area with complaints of "allergy", locally called "hasaseyah".

Allergic diseases, such as bronchial asthma and atopic eczema, take a heavy toll of the health and economic status of any country. The loss of working or school hours and disability imposed on allergic patients may lead to economic and intellectual damage to that country. Because of their high morbidity and mortality rate (in the case of bronchial asthma), efforts are needed to identify the population at risk and to monitor the severity of these ailments, which also include rhinitis and conjunctivitis.

The clinical severity and progression of bronchial asthma and eczematous diseases are often modulated by various environmental agents including mites (1). Constant exposure of allergic patients to airborne allergens may generate specific antibodies which may be of prognostic importance (2, 3).

Therefore, a study was carried out among asthmatic and eczematous Tabuk patients to identify those with abnormal SMEA. This paper is a report of such an experiment. To our knowledge, this is the first publication on mite allergology from this northwest corner of Saudi Arabia.

■ This prospective pilot study was carried out at the pioneering allergy clinic of the North West Armed Forces Hospital, Tabuk, Saudi Arabia, a 350-bed medical facility managed in collaboration with the Conjoint Boards of the Royal Colleges of Surgeons and Physicians of Ireland.

Thirteen randomly selected patients attending the allergy and asthma clinics were registered for the

study: four having atopic eczema, four bronchial asthma, and five controls (12 male, one female) (aged 2–35 years, mean 18.5 years).

The control subjects were blood donors who had no family or personal history of allergy or parasitic diseases. The atopic score of each patient was determined from the age of the appearance of the disease, and personal and family history of atopy (4): atopic score 0 = no family or personal history of allergy, 1 = positive family history of allergy; 2 = positive personal history of allergy; 3 = over 2-year history of frequent attacks of bronchial asthma or atopic eczema; 4 = 4-year positive personal or family history of allergy, and taking oral prednisolone. Only parasite-free, nonsmoking participants were admitted to the study.

SMEA levels were quantified with the specific IgE Alastat-Allergen Kit (Diagnostic Products Corporation, Los Angeles, CA, USA). This kit is based on the liquid-phase immunoenzymometric assay specifically designed for the measurement of allergen-specific IgE in plasma or serum (5).

■ The antigen used was derived from *Dermatophagoides pteronyssinus* and *D. farinae* domestic mites.

Each patient's serum was initially incubated with the antigen, and subsequently with the supplied multivalent antibody. A final incubation with horseradish peroxidase-labeled monoclonal anti-IgE antibody identified SMEA, which was calculated from the optical densities of the resultant mixture.

■ *Control subjects.* Table 1 shows that the participants were males except for one female (for local logistic reasons). A healthy-looking employee, C5, who was believed to have family history of allergy, was disqualified from the study.

■ *Atopic eczema patients.* The mean SMEA of the participants was 35 IU/ml. Although this was high, these patients had the highest atopic scores.

■ *Bronchial asthma patients.* The mean SMEA of this class was 22.5 IU/l. Fifty percent of all the patients had a very high atopic score.

■ This study reveals one of the practical problems in choosing a control population in this environment. Table 1 shows patient C5, who was disqualified because of a consanguineous allergy background. Hence, the choice of control population in this area must be scrupulously screened for family background.

This experiment shows that antibodies generated serologically against a particular allergen to which

Table 1. Serum mite antibodies among allergic patients in Tabuk, Saudi Arabia

Patient no.	Age (years)	Sex	Diagnosis blood donor	SMEA IU/ml	Atopic score
C1	22	M	Blood donor	0	0
C2	24	M	Blood donor	0	0
C3	27	M	Blood donor	0	0
C4	30	M	Blood donor	0	0
C5	22	M	Blood donor	4.4	0
A1	2	M	Atopic eczema	20	2
A2	4	M	Atopic eczema	60	3
A3	19	F	Atopic eczema	0	2
A4	5	M	Atopic eczema	60	4
B1	7	M	Bronchial asthma plus atopic eczema plus atopic areata	29.5	3
B2	6	M	Bronchial asthma	0	3
B3	35	M	Bronchial asthma	80	2
B4	10	M	Bronchial asthma	0.5	2

a patient is sensitized may form the basis of laboratory diagnosis of hypersensitivity.

Table 1 also reveals that the SMEA levels were high both in atopic eczema and atopic scoring. It may be assumed that the atopic score was directly related to, or was a clinical marker of, serologic SMEA levels. Patients A2 and A4 had very severe atopic eczema, while the illness of the latter had to be controlled with intensive prednisolone therapy.

It is interesting to observe that the asthmatic patient B2, who had a family and personal history of atopy, had no SMEA. Seventy-five percent of the asthmatic group demonstrated circulating antibodies against the mite antigen, while 25% did not reveal any, even though this subgroup had personal or family history of allergy.

Despite the low number of participants in this study, certain preliminary conclusions can still be drawn. The result suggests that mites are involved in the pathogenesis of bronchial asthma and atopic eczema. Moreover, antigenicity in dust-allergic patients is universal, irrespective of the geographic source of either the acarologic agents or the patients (6). But Tabuk mite fauna may be different. Even though the SMEA may offer the advantage of a diagnostic and prognostic use in clinical allergy, a larger population study to validate this conclusion is still needed.

This principle of SMEA measurement may be used to screen relatives of house-dust-mite-sensitive patients with bronchial asthma or atopic eczema for evidence of serologic SMEA. This may develop into a predictive allergy procedure; e.g., using cord blood (in neonates) and finger-prick blood testing in the mass screening of a population at risk of mite allergy. However, strict control of mite exposure is most important in the prophylaxis of these diseases.

Key words: bronchial asthma; eczema; mite antibodies; Saudi Arabia.

S. N. AL-DUSARI, A. O. SOMORIN*, A. U. ZAMAN

*Department of Medicine
North West Armed Forces Hospital
PO Box 100
Tabuk
Saudi Arabia
Tel. 966 4-423-3988
Fax: 966 4-422-2324

Accepted for publication 6 November 1996

Copyright © Munksgaard 1997

ALLERGY 1997;52:234-235

ACKNOWLEDGMENTS

Research approval no. MR 054 by the North West Armed Forces Hospital, Tabuk, Kingdom of Saudi Arabia. The abstract of this paper was presented at the American Academy of Allergy, Asthma, and Immunology Conference, New Orleans, LA, USA, 19 March 1996.

REFERENCES

- Platts-Mills TAE, de Weck AL. Dust mite allergens and asthma. A worldwide problem. *J Allergy Clin Immunol* 1989;82:416.
- Wide L, Bennich H, Johansson SGO. Diagnosis of allergy by an *in vitro* test for allergen antibodies. *Lancet* 1967;2:1105-7.
- Somorin AO, Heiner DC. California house dust IgE antibodies in the sera of allergic Nigerian patients. *Cent Afr J Med* 1980;26:59-63.
- Abdullah AK, Zaman A. Serum IgE levels in adults with asthma. *J Asthma* 1987;24:207-13.
- El Shami AS, Alaba O. Liquid-phase *in vitro* allergen-specific IgE assay with *in situ* immobilization. *Adv Biosci* 1989;74:191-201.
- Miyamoto T, Ishizak T, Oshima S, Sano S. Allergen identity between the common floor mite (*D. farinae* Hughes 1961) and house dust as a causative agent in bronchial asthma. *J Allergy* 1968;42:14-28.

Oral allergy syndrome to apple after a lover's kiss

IT IS WELL documented that patients suffering from birch pollinosis frequently show immediate-type allergic symptoms such as itching and swelling of the lips, mouth, tongue, palate, and throat after

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.