Key words: amoxicillin; asthma; occupational disease.

I. Jiménez*, E. Antón, I. Picáns, I. Sánchez, M. D. Quiñones, J. Jerez
*Sección de Alergia
Hospital Marqués de Valdecilla
Santander
Spain
Tel. 34 42 202543
Fax: 34 42 202543

Accepted for publication 13 August 1997
Copyright © Munksgaard 1998
ALLERGY *1998:53:104–105*

REFERENCES
1. Pepys J, Hutchcroft BJ. Bronchial provocation tests in etiologic diagnosis and analysis of asthma. Am Rev Respir Dis 1975;112:829-59.
2. Davies RJ, Hendrick DJ, Pepys J. Asthma due to inhaled chemical agents: ampicillin, benzylpenicillin, 6-amino penicillanic acid and related substances. Clin Allergy 1974; 4:227-47.
3. Coutts I, Dally B, Newman-Taylor J, et al. Asthma in workers manufacturing cephalosporins. BMJ 1981;283:950.

Anaphylaxis from linseed

A case of IgE-mediated anaphylaxis caused by linseed.

In the past 6 years, a 40-year-old man with no personal or family history of atopy had had five or six episodes of intestinal/abdominal pain, vomiting, diarrhea, generalized urticaria, acute dyspnea without bronchospasm, hydrorrhea, successive sneezing, nasal obstruction, pruritis, and intense general malaise which required emergency treatment. All episodes had occurred within 2–3 min of his consuming multigrain bread. The patient was currently asymptomatic as he had been avoiding contact with or consumption of products that contain linseed.

We prepared an extract of linseed with a concentration of 1:10 w/v, as described previously (1). Skin prick tests (SPT) were performed with the linseed extract and were strongly positive (wheal diameter of 14 mm). Besides a battery of the most common inhalant allergen sources in our area, we performed SPT with cereals, milk, egg, peanuts, and dried fruits (Abelló SA, Madrid, Spain), all of which were negative. SPT with the linseed extract were negative in 10 atopic and 10 nonatopic patients.

Specific IgE for linseed was determined by coating the allergen onto cellulose disks activated by CNBr. A Pharmacia-Trazer kit (Kabi Pharmacia) was used during the development process. Specific IgE to linseed was positive with a value of 38 Phadebas RAST units/ml. Specific IgE to linseed was negative in six control subjects.

SDS–PAGE by the technique of Laemmli and SDS–PAGE immunoblotting with the patient's serum were performed, in reducing (the reductor agent breaks the disulfide bonds between polypeptides) and nonreducing conditions, with the linseed extract. Immunoblots of linseed under nonreducing conditions showed intense IgE-binding bands with an apparent high mol. wt. (over 150–175 kDa), as well as discrete bands of about 90–100 kDa. When linseed extract was carried out under reducing conditions, these bands disappeared, and very discrete bands of a broad range (35–100 kDa) were detected. This different reactivity is in agreement with the bands detected in SDS–PAGE (Fig. 1). We also performed immunoblotting with the serum of six patients having total IgE <2 UI/ml that showed no bands.

For ethical reasons, we did not perform the oral provocation with linseed.

Hypersensitivity to linseed was described in 1930 (2, 3). However, cases of linseed hypersensitivity are extremely rare. Currently, due to the use of linseed as a laxative and the rise in consumption of bakery products containing linseed, new cases may appear. Our patient presented with various episodes of anaphylaxis as a result of having consumed linseed. Type I hypersensitivity was strongly suggested by the positivity of SPT and by the demonstration of specific IgE antibodies to linseed in the patient's serum. This case demonstrates the importance of finding the specific cause of an allergic disorder since the patient experienced five or six anaphylactic episodes until linseed was

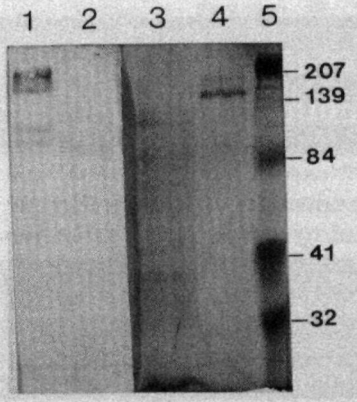

FIGURE 1 Lane 1) Immunoblotting in nonreducing conditions; 2) immunoblotting in reducing conditions; 3) SDS–PAGE in reducing conditions; 4) SDS–PAGE in nonreducing conditions; 5) mol.-wt. standards.

detected as the provoking agent. Afterwards, he had been asymptomatic, as he avoided consumption of or contact with linseed-containing products.

Linseed contains many potential allergens. In a recent case study of linseed hypersensitivity (1), five allergens with mol. wt. of 38, 35, 30, 22, and 20 kDa were found by SDS–PAGE immunoblotting. In contrast, by SDS–PAGE immunoblotting under nonreducing conditions, we have found that allergens with a higher mol. wt. (150–175 kDa) intensely bound with IgE, and other more discrete bindings were noted with lower mol.-wt. allergens (90–100 kDa). With SDS–PAGE immunoblotting under reducing conditions, the high-mol.-wt. bands disappeared and discrete bands of lower mol. wt. (35–100 kDa) were detected. The results of SDS–PAGE are also in agreement with these findings. This may suggest that allergens of high mol. wt. that bind intensely with IgE separate into various subunits under reducing conditions, and that, upon separating, they lose the major part of their binding capacity.

Key words: anaphylaxis; food; linseed; specific IgE.

A. Lezaun*, J. Fraj, C. Colás, F. Duce, M. A. Domínguez, M. Cuevas, P. Eiras
*Avda San Juan Bosco 15
Department of Allergology
Hospital Clínico Universitario
50009 Zaragoza
Spain

Accepted for publication 10 September 1997
Copyright © Munksgaard 1998
ALLERGY *1998:53:105–106*

REFERENCES
1. Alonso L, Marcos ML, Blanco JG, et al. Anaphylaxis caused by linseed. J Allergy Clin Immunol 1996;98:469-70.
2. Black WC. Flax hypersensitiveness. JAMA 1930;94:1064.
3. Grant LR. A report of six cases of flaxseed sensitization with review of the literature. J Allergy 1931;3:469-77.

Cutaneous allergy to human (recombinant DNA) insulin

THE introduction of insulin therapy was soon followed by the report of cutaneous reactions. Nowadays, the biosynthesis of human insulin (HI) by recombinant DNA technology (rDNA) has greatly reduced the incidence of adverse reactions (1). We report here an unusual case of persistent local immediate-type allergy to rDNA HI at initial insulin therapy.

■ A 32-year-old woman was diagnosed as having insulin-dependent diabetes in November 1995. She began treatment with rDNA HI (Actrapid® Novolet® and Mixtard® 20 Novolet®) and had no problems for 45 days. She then developed local reactions at the injection sites with pruritus and wheal and flare 5 min after every injection of insulin. This reaction reached a diameter of 8–10 cm within 1 h, resolved within 1–2 h, and was followed by an indurated lesion that lasted for 48 h. Treatment with cetirizine (10 mg/24 h) provided considerable relief, but could not check the disorder for the moment. The patient had no previous history of atopy.

■ The patient was admitted to our department for evaluation in February 1996. Her informed consent was obtained before the study. Prick tests with different insulin preparations proved positive (Table 1), and negative with latex (ALK-Abelló, Madrid, Spain), protamine sulfate (Rovi SA, Madrid, Spain), and other additives included in insulin preparations. Intradermal tests with protamine from 0.1 μg/ml to 0.1 mg/ml were negative. Ten normal subjects were used as a control group. Total serum IgE (ALA-STAT, DPC, Los Angeles, CA, USA) was normal (45 UI/ml). Specific IgE to bovine, porcine, and human insulins (ImmunoCAP, Pharmacia, Sweden) were elevated to 7.78, 6.78, 8.11 kU/l, respectively. Specific IgE against latex and protamine were negative (ImmunoCAP, Pharmacia, Sweden). Hematologic, biochemical, and urine tests; immunoglobulins; and C3, C4, CH50, and circulating immune complexes were normal, except for fasting blood glucose (260 mg/dl). The HLA phenotype was A24,26/ B38,39/ Bw4,6/ Cw7,X/ DR4,8/ DRw53,X/ DQw4,8. Skin biopsy showed a perivascular infiltrate consisting of lymphocytes, monocytes, and especially eosinophils, mild edema affecting the superficial dermis; and slight edema of endothelial cells.

An unusual case of newly diagnosed insulin-dependent diabetes mellitus with persistent local immediate-type allergy to rDNA human insulin at initial insulin therapy.

■ Cases of local allergy against HI have been rarely reported (2–6). Some authors describe a cell-mediated hypersensitivity reaction (2); others an IgE-mediated mechanism (3–6). On the basis of the results of cutaneous and *in vitro* tests, we

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.