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Lettuce allergy

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Key words: Compositae; food allergy; lettuce.

● LETTUCE (*Lactuca sativa*), a plant of the Compositae family, is widely eaten. It is known to provoke allergic contact dermatitis (1), which is sometimes made worse by ingestion (2). IgE-dependent allergy is less frequent and has more recently been reported among workers handling lettuce, but also among other people sensitized to various pollens (3–5). We report the case of a man presenting with a digestive and respiratory form of food allergy to lettuce confirmed by oral challenge test and identification of specific IgE to a 40-kDa protein.

A 32-year-old man suffering from pollinosis presented with chronic urticaria and grade 1 perennial asthma. Prick tests were negative to perennial allergens, but positive to grass pollens and herbaceous

pollens (solidago, goosefoot, sorrel, alfalfa, plantain, and clover).

The prick tests were positive to Umbelliferae (fennel [4 mm], celery [4.5 mm], carrot [4.5 mm], against a 9% codeine control at 3 mm) and to Compositae (Jerusalem artichoke [5 mm], lettuce [3.5 mm], chicory [3 mm], artichoke [3 mm], and tarragon [2.5 mm]). Screening for specific IgE (Phadebas CAP RAST, Sweden) was negative. The oral challenge test to celery (16 g) provoked abdominal pain 2.3 h later. Oral challenge test to lettuce (30 g) caused labial pruritis, dry cough, and stomach pain 30 min later. A 15% decrease in peak expiratory flow was observed 2 h after ingestion.

Identification of the allergen was performed by SDS–PAGE electrophoresis followed by Western blot. The core and leaves of the lettuce were crushed separately and placed in an aqueous solution of 2% polyvinylpyrrolidone, 2 mM EDTA, for 1 h at room temperature (4). The proteins were assayed by a pyrogallol red method. Electrophoresis was performed on a PhastSystem (Pharmacia) on 8–25 polyacrylamide gel. One electrophoresis was stained silver, whereas a Western blot was performed on a nitrocellulose membrane using a second electrophoresis.

The membrane was saturated for 1 h in a 5% bovine serum albumin buffer, and then submerged for one night at –4°C in a 1:10 dilution of the patient’s serum in 1% BSA–0.1% PBS Tween buffer.

The fixed IgE was observed by chemiluminescence using anti-IgE secondary antibodies, marked with peroxidase (Dako) diluted 1:1000 in 1% BSA–0.1% PBS Tween buffer. After washing, the membrane was submerged in Luminol (Covalab) with H₂O₂.

The protein concentrations were 2.3 g/l (core of the lettuce) and 1.8 g/l (leaves).

The electrophoresis showed nine main bands of molecular mass 16, 22, 28, 37, 51, 64, 82, 86, and 94 kDa for the core, and five main bands of 22, 28, 40, 51, and 60 kDa for the leaves.

The Western blot revealed a protein band of 40 kDa in the extract of the core. No band was observed with the extracts of the leaves or with the chicory extracts.

The 40-kDa protein identified by specific IgE was present in the core of the lettuce, but was not found in the leaves. This protein is mentioned in previous studies, most often in association with other proteins (3–6). There appeared to be no major difference in the electrophoresis conditions, all the authors having worked in denaturing conditions. However, a variability in IgE response existed for several reasons:

- 1) the protein content of lettuce is very low, and the leaves do not contain certain allergens
- 2) these allergens are thermolabile
- 3) the variety of lettuce contributes to the variability of the response.

The Western blot development technique by chemiluminescence is highly adapted to screening for delicate allergens because it increases the sensitivity of detection by at least 10 times (7) compared with conventional techniques.

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Hazards in the use of diphencyprone

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Key words: diphencyprone; topical immunotherapy.

● THE topical use of diphencyprone (DCP) is frequently recommended in clinical dermatology to treat alopecia areata, the side-effects of this substance being well known among users (1, 2). We present a case of professional sensitization in a nurse who developed systemic contact dermatitis simulating a fixed drug eruption, and another of consort contact dermatitis in a housewife with acute facial eczema.

Patient 1. A 60-year-old nurse, with no personal or family history of allergy, who had received topical immunotherapy (DCP) for 3 years, complained of the appearance of

a 10×5 cm lesion in the right neck region. The lesion was erythematous, violet in color, and slightly scaly, and the outbreaks exhibited a weekly incidence and a development of 10 months. It was very pruriginous and simulated fixed drug eruption (Fig. 1). The patient had never before had dermatologic lesions, either on the hands or elsewhere. She presented a flare up of the lesion when going into the room where this topical therapy was being applied, with reactivation of her clinical manifestations not only in the previously affected area but also on her back, where epicutaneous tests had been applied.

Patient 2. A 48-year-old housewife, without previous illness, went to the emergency room for acute lesions on her face. Clinical examination revealed acute edematous erythema and itchy plaques on the cheeks and neck. She had been treated with systemic steroids in three previous episodes. The dermatitis developed 10 h after her husband underwent topical immunotherapy with DCP solution in 2% acetone, for extensive alopecia areata, for

which he was treated once a week. She could not share a bed with her husband after his use of DCP because her clinical manifestations worsened.

In both cases, patch tests with the standard contact antigen panel of the Spanish Contact Dermatitis Research Group were negative. Patch tests with DCP (dilutions 0.0001–0.1% in acetone) in the Finn Chamber, and Leukotest showed eczematous changes at 48 and 96 h.

Shah et al. (3) report the risk to medical and nursing staff. We also think (3, 4) that sensitization to DCP among pharmacists and dermatologic staff is well recognized. Notifying health-care personnel of these adverse reactions is important because the use of DCP could be questioned unless all the prophylactic measures were implemented.

Consort contact dermatitis may be due to cutaneous exposure to the allergens employed by the partner (5). Medicaments, perfumes, hair dyes, acne preparations, sunscreens, condoms and rubber diaphragms, and clothes may be allergenic



Figure 1. Clinical aspect of lesions in patient 1.