

An emergency kit comprising epinephrine, an H₁-blocking antihistamine, and a corticosteroid was prescribed for the patient. As there was a high risk of further anaphylaxis, oral hyposensitization with a commercial celery extract (SDL oral, SmithKlineBeecham) was started. However, oral rechallenge with 1.2 g raw celery after 6 months of maintenance therapy evoked the same symptoms as before. Treatment with this extract was stopped, and tolerance induction with a commercial natural celery juice (Selleriesaft neuform) was considered. Skin prick testing the patient with this celery juice elicited a wheal 8 mm in diameter (no reaction in 10 controls), and oral challenge with 5 ml juice caused an anaphylactic reaction. Oral hyposensitization started with 0.1 ml of the juice given five times on day 1, and then over 4 weeks incremental doses of 5 ml were administered up to five times daily. Three months after reaching this dose, oral challenges with incremental doses of 1.0/2.0/5/10 g of raw celery were tolerated without symptoms, but another 20 g celery again provoked flush. For the last 3 years, the patient has ingested 25 ml celery juice each day. No further severe anaphylactic reactions have occurred, and she is able to consume restaurant meals without symptoms.

This patient had tried to avoid the offending foods, yet she continued to suffer from frequent reactions, which in part were severe. Probably, the increased mast-cell load of mastocytosis was an aggravating factor of her reactions (2).

Hyposensitization (immunotherapy) with food allergens is not a routine measure (3). To treat peanut allergy, subcutaneous immunotherapy was tried and found to be effective, but side-effects were considerable (4). Successful oral hyposensitization of a few patients with native food (cow's milk, hen's eggs, fish, and orange) has been reported (5, 6).

A prerequisite for the use of native food is a permanently available and quantitatively divisible allergen source. Our patient benefited from oral hyposensitization with celery juice. Although she still was not able to ingest larger amounts of celery, the previously severe reactions did not recur, and inevitable minor accidental allergen exposures were evidently tolerated without symptoms. Thus, in certain patients with a high risk of severe reactions due to unintentional allergen ingestion, oral hyposensitization with native food might be tried. The long-term effect of such oral hyposensitization remains to be assessed.

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Oral anaphylaxis to mites

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Key words: allergy; anaphylaxis; corn flour; dust mites; food allergy; storage mites.

● It is known that anaphylaxis can be induced in some mite-sensitized patients by the ingestion of foods contaminated by mites. These patients usually have current atopic disease, and most of them present acetylsalicylic acid sensitivity. House-dust mites and storage mites can precipitate these episodes. Wheat flour is the major source of mite allergens related to these cases. Here, we report a case of anaphylaxis possibly induced by the ingestion of corn flour contaminated with mites.

A severe reaction after ingestion of mite-contaminated flour.

An 18-year-old woman developed nausea, abdominal cramps, wheezing, cough, dyspnea, and urticaria/angioedema a few minutes after eating polenta, a dish prepared with cooked corn flour. In hospital, she received subcutaneous epinephrine, intravenous hydrocortisone, and intramuscular antihistamine. The symptoms gradually subsided and she was discharged 6 h later.

Table 1. Allergic evaluation

IgE determinations

Total serum IgE: 334 IU/ml

D. pteronyssinus IgE: 11.9 IU/ml

B. tropicalis IgE: >17.5 IU/ml

Skin test results (mean wheal and flare in mm)

D. pteronyssinus: 4×15 mm

D. farinae: 5×15 mm

B. tropicalis: 7.5×25 mm

T. putrescentiae: 4×10 mm

Histamine: 3×12 mm

Corn flour: negative

She was interviewed 5 days after the anaphylactic episode. She denied taking any drugs or other foods in the 6 h before the episode. She had a history of mild symptoms of perennial rhinitis, and in the past she had suffered an episode of urticaria/angioedema induced by acetylsalicylic acid. It is noteworthy that 2 years before, she had experienced a milder episode of anaphylaxis of unknown cause. She now remembered that the symptoms had also begun minutes after she ate food prepared with corn flour, which seems to have been the trigger in both episodes.

Allergic sensitization was assessed by skin prick tests with foods and common inhalant allergens. *Dermatophagoides pteronyssinus* and *Blomia tropicalis* serum specific IgE were determined. Skin tests gave negative results to common foods, including corn flour, but the patient showed strong reactions to mite allergens (Table 1). In the absence of any possible trigger, we suspected anaphylaxis induced by mite-contaminated food. We examined a sample of the corn flour used in her home and found a total of 36 mites/g of corn flour, identified as *Tyrophagus putrescentiae* (11/36), *D. pteronyssinus* (8/36), *D. farinae* (5/36), and others (12/36). Analysis of marketed flour of the same brand gave negative results. Levels of the *Dermatophagoides* mite allergens Der p 1, Der f 1, and group 2, and of *B. tropicalis* allergen Blo t 5, assayed by mAb-based ELISA (1), were undetectable in extracts made from samples of corn flour.

Erben et al. (2), Matsumoto et al. (3), Blanco et al. (4), and Sanchez-Borges et al.

(5) have all reported on anaphylaxis caused by mite-contaminated food. All the patients were atopics sensitized to mite allergens, and anaphylaxis was precipitated briefly after the ingestion of contaminated foods.

Concomitant ASA intolerance was also reported among these patients. These findings indicate that mite allergenicity persists despite the cooking process. In most of these reports, the major source of mite allergens was wheat flour. Our case supports the hypothesis that anaphylaxis may be induced by corn flour contaminated by mites. We were unable to identify any other possible causes of the reaction. In addition, our patient was strongly sensitized to mite allergens, and the corn flour containing mites was the only food ingested within the 6 h preceding the episode. Our findings also indicate that this association must be considered in idiopathic or unexplained anaphylaxis in atopic patients.

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Selective immediate hypersensitivity to ceftazidime

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Key words: ceftazidime; immediate hypersensitivity; skin tests.

● CEFTAZIDIME is a third-generation cephalosporin with a frequency of hypersensitivity reactions (mostly rashes) of about 0.5% (1) and very few reports of causing IgE-mediated reactions (2).

We studied a 16-year-old girl who suffered from allergic oculorhinitis (grasses and olive tree) and had experienced anaphylactic shock (urticaria, rhinitis, wheezing, dyspnea, and hypotension) within 10 min after intravenous administration of the first dose (1 g) of ceftazidime for bronchopneumonia. Her symptoms subsided 6 h after subcutaneous epinephrine, betamethasone (8 mg intravenous), and chlorphenamine (10 mg intravenous).

Anaphylaxis with positive intradermal skin test.