

In the present case, the coadministration of a NSAID with an antibiotic was required for development of the adverse drug reaction, as the patient had tolerated the separate intake of both drugs on several occasions, either spontaneously or in challenge tests without problems, thereby complicating the diagnosis of NSAID intolerance.

The underlying mechanism of this reaction could be interference by the NSAID in the arachidonic acid metabolism (cyclo-oxygenase-1 inhibition) pattern, as the patient tolerated the intake of the implicated antibiotic together with nimesulide, a selective cyclo-oxygenase-2 inhibitor NSAID. The precise role of the antibiotic in the development of the reaction remains unknown.

The need for the association of several factors for development of intolerance or allergy has been previously reported (3–5). In these cases, the culpable associations were drug plus exercise (acetylsalicylic acid or nafylpropionic acid-dependent exercise-induced anaphylaxis) (3), food plus drug and exercise (food-dependent exercise-induced anaphylaxis in which the previous intake of an NSAID further worsened the reaction) (4), and drug plus food (anaphylaxis to walnuts and pine nuts induced by the intake of an angiotensin-converting enzyme inhibitor) (5). Nevertheless, no cases of drug-induced NSAID intolerance have been reported to date.

We believe this case report to be interesting in view of the high prevalence of NSAID intolerance, which may be underdiagnosed if a factor association is not considered.

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Accepted for publication 15 August 2000
Allergy 2001; 56:81–82
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ISSN 0105-4538

References

1. HERNANDEZ D. Severe delayed adverse reactions to non-steroidal anti-inflammatory drugs (NSAIDs). *Clin Exp Allergy* 1998;**28 Suppl 4**:47–51.
2. COROMINAS M. Mechanisms implicated in adverse reactions to non-steroidal anti-inflammatory drugs. *Clin Exp Allergy* 1998;**28 Suppl 4**:41–45.
3. GERTH VAN WIJK R, DE GROOT H, BOGAART JM. Drug-dependent exercise-induced anaphylaxis. *Allergy* 1995;**50**:992–994.
4. DOHI M, SUKO M, SUGIYAMA H, et al. Food-dependent, exercise-induced anaphylaxis: a study on 11 Japanese cases. *J Allergy Clin Immunol* 1991;**87**:34–40.
5. MONERET-VAUTRIN DA, BLAIN H, KANNY G, BLOCH Y. Anaphylaxis to walnuts and pine nuts induced by ACE. *Allergy* 1998;**53**:1233.

Oral hyposensitization with celery juice

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Key words: celery-mugwort-spice syndrome; food allergy; food hyposensitization; mastocytosis.

● IMMEDIATE-type allergy to celery is common and can cause severe anaphylaxis. Cross-reactivity to carrots, spices of the Apiaceae family, and many other foods ("celery-mugwort-spice syndrome") is common in patients with celery allergy (1). As celery is often cut into very small pieces or removed before serving, hidden allergens, as in soups, salads, or sausages, hamper avoidance.

Successful treatment of recurrent anaphylaxis.

We treated a 49-year-old woman who had suffered from seasonal rhinoconjunctivitis for 25 years. Starting about 20 years before, she had recurrent urticaria, flush, and generalized pruritus a few minutes after eating. She soon realized that celery, carrots, and several spices elicited these reactions. She tried to avoid these food items, but she later developed at least eight severe anaphylactic reactions with loss of consciousness while eating at restaurants. Each time, she later learned that the food had been prepared with celery or spices. Milder reactions developed after meals at home about three times a week. For some years, she had noticed the appearance of "freckles" on her trunk and thighs.

Skin examination revealed typical lesions of urticaria pigmentosa, and the diagnosis was substantiated histologically. An increased number of mast cells was also found in a bone-marrow biopsy. There were no indications of other systemic involvement by mastocytosis.

Skin prick tests yielded immediate-type reactions to several pollens including mugwort, celery, carrot, anise, coriander, hazelnut, peanut, and soybean. Specific IgE antibodies were demonstrable by CAP-FEIA to celery (class 1); to mugwort, carrot, apple, and peanut (class 2); to hazel pollen (class 3); and to birch pollen and hazelnut (class 4). Baseline mast-cell tryptase levels in the serum determined repeatedly by UniCAP tryptase were 15.8–18.1 µg/l (normal <13.5 µg/l).

Oral challenge with 1 g raw celery root induced within 5 min a prominent flush of the patient's face, neck, and trunk; generalized pruritus; facial edema; swelling of the hands; and shortness of breath. Ingestion of 1 g rosemary caused orbital swelling, conjunctivitis, and generalized pruritus, whereas several other spices (black pepper, red pepper, oregano, and caraway; dose each 2 g) and soybean (100 g) were well tolerated.

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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Accepted for publication 25 August 2000
Allergy 2001; 56:82–83
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ISSN 0105-4538

References

1. WÜTHRICH B, STÄGER J, JOHANSSON SGO. Celery allergy associated with birch and mugwort pollinosis. *Allergy* 1990;**45**: 566–571.
2. RUÈFF F, SCHÖPF P, FRIES C, LUDOLPH-HAUSER D, PRZYBILLA B. Constitutionally elevated serum concentration of mast cell tryptase is associated with severe anaphylactic Hymenoptera sting reactions [Abstract]. *J Allergy Clin Immunol* 1999;**103**:S45.
3. NEKAM K. Management of food allergy. *Allergy* 1998;**53 Suppl 46**:122–124.
4. OPPENHEIMER JJ, NELSON HS, BOCK SA, CHRISTENSEN F, LEUNGS DYM. Treatment of peanut allergy with rush immunotherapy. *J Allergy Clin Immunol* 1992;**90**:256–262.

5. PATRIACA G, SCHIAVINO D, NUCERA E, SCHINCO G, MILANI A, GASBARRINI GB. Food allergy in children: results of a standardized protocol for oral desensitization. *Hepatogastroenterology* 1998;**45**:52–58.
6. WÜTHRICH B, HOFER TH. Nahrungsmittelallergien. III. Therapie: Eliminationsdiät, symptomatische medikamentöse Prophylaxe und spezifische Hyposensibilisierung. *Schweiz Med Wochenschr* 1986;**41**:1401–1410.

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.