

Table 1. Allergic evaluation

IgE determinations
Total serum IgE: 334 IU/ml
<i>D. pteronyssinus</i> IgE: 11.9 IU/ml
<i>B. tropicalis</i> IgE: >17.5 IU/ml
Skin test results (mean wheal and flare in mm)
<i>D. pteronyssinus</i> : 4×15 mm
<i>D. farinae</i> : 5×15 mm
<i>B. tropicalis</i> : 7.5×25 mm
<i>T. putrescentiae</i> : 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.

Ten months earlier, she had tolerated intramuscular ceftazidime.

Two years later, the patient underwent skin tests with penicilloylpolylysine (Allergopen, Reinbek, Germany), minor determinant mixture (Allergopen), benzylpenicillin, ampicillin, and amoxicillin, as well as ceftazidime, cefotaxime, ceftriaxone, cefuroxime, cefamandole, cephalothin, cephalexin, cefaclor, and cefixime, as previously described (3). Cephalosporins were used at a concentration of 2 mg/ml in 0.9% NaCl. Intradermal testing with ceftazidime produced a wheal of 6×8 mm. Prick and intradermal histamine tests produced wheals with maximum diameters of 7 and 10 mm, respectively. Intradermal tests with the above cephalosporins were negative in 40 control subjects, seven of whom had previously tolerated ceftazidime.

Assays (UniCAP[®] specific IgE; Pharmacia & Upjohn, Uppsala, Sweden) for specific IgE to penicilloyl G, penicilloyl V, ampicilloyl, amoxicilloyl, cefaclor, and ceftazidime were negative (<0.35 kU/l). The ceftazidime assay was an experimental prototype.

We performed single-blind, placebo-controlled oral challenges with penicillin V (therapeutic dose: 1 000 000 IU), ampicillin (1 g), cephalexin (500 mg), cefaclor (500 mg), cefuroxime axetil (500 mg), and cefixime (400 mg) on different days. Increasing amounts (0.01, 0.1, and 1.0) of the therapeutic doses, indicated in parentheses, were administered at 1-h intervals, with negative results.

Clinical findings and the positive intradermal test with ceftazidime suggested a type-I allergic reaction.

The fact that we used an experimental prototype and that we performed our assays 2 years after the reaction may explain the RAST negativity. In the meantime, the IgE titer may have fallen below the cutoff value, as noted with penicilloyl IgE antibodies (4).

We previously diagnosed two cases of immediate hypersensitivity to ceftazidime on the basis of skin-test and RAST positivity (2). One patient – who displayed positive results only to ceftazidime – was not challenged with other β -lactams. The other had reacted to ceftazidime and ceftriaxone in separate episodes and showed skin-test and RAST positivity to both cephalosporins.

The present subject displayed skin-test, RAST, and challenge negativity with other β -lactams (including first-, second-, and third-generation cephalosporins). Thus, the patient's sensitization appeared to involve the entire ceftazidime molecule, as previously demonstrated with cefaclor (5). To our knowledge, this is the first case in which selective immediate hypersensitivity to ceftazidime has been demonstrated.

For evaluating immediate reactions to cephalosporins, it is crucial to test the responsible drug in addition to penicillin determinants.

In assessing the selectivity of the response, negative results in skin testing with cephalosporins other than the culprit one appear to be a reliable indicator of tolerability. However, the routine prescription of alternative cephalosporins on the basis of skin-test negativity should be made the subject of further studies. Meanwhile, such prescriptions should be given only in selected cases after a careful allergologic examination including the relevant challenge.

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Allergenicity of rHev b 10 (manganese-superoxide dismutase)

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Key words: Hev b 10; latex allergy; MnSOD; recombinant allergen.

● MANGANESE-superoxide dismutase (MnSOD) of *Hevea brasiliensis* has been identified as an IgE-binding protein in natural rubber latex (1) and was designated as latex allergen Hev b 10 (2). Since its allergenicity has not been studied so far, we produced a recombinant maltose-binding protein (MBP), latex-MnSOD (rHev b 10) fusion protein, to investigate the IgE-binding reactivity of Hev b 10 with sera from latex-allergic patients.

cDNA (2 ng) from *H. brasiliensis* leaves (3) was amplified in the presence of 25 pmol modified Hev b 10-specific primers (5'-Hev b

MnSOD is of minor significance in latex allergy.