

asparagus. The question of whether the reaction to asparagus is species-specific, or of whether cross-reacting components exist, needs further investigation.

Key words: asparagus; IgE antibodies; Liliaceae; RAST; urticaria.

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Diclofenac-induced urticaria with aspirin tolerance

ACETYLSALICYLIC acid (ASA) and other non-steroidal anti-inflammatory drugs (NSAIDs) have been frequently involved in allergic and pseudoallergic (1) drug reactions: anaphylactic shock (2, 3), asthma, rhinitis, urticaria, angioedema, and other skin diseases such as dermatitis (4, 5), photosensitivity, toxic epidermal necrolysis, and Stevens-Johnson syndrome.

In 1984, adverse reactions to NSAIDs represented 21% of the drug adverse effects in the US Food and Drug Administration survey (6). Most of these cases were caused by intolerance of ASA and other NSAIDs (7) due to cyclooxygenase inhibition. The severity of the adverse reactions to NSAIDs depends on the potency of the cyclooxygenase inhibition by each NSAID. An important clinical question is whether patients with an allergic reaction to a particular NSAID may tolerate other NSAIDs or ASA.

Diclofenac sodium is a NSAID derivative of phenylacetic acid. We report a patient with diclofenac-induced urticaria who was able to tolerate ASA without any untoward effect.

■ A 57-year-old atopic woman with gonarthrosis was taking diclofenac (Voltaren®) per os 50 mg/day. One hour after the intake of 100 mg of diclofenac, she developed a widespread pruritic maculopapular eruption with hives, erythema, and vomiting. These symptoms subsided within 1 h after administration of dexchlorpheniramine. Seven days later and 30 min after 75 mg of diclofenac was given, she developed a similar reaction with hypotension (100/60) that resolved with dexchlorpheniramine and systemic corticosteroids. The patient reported the tolerance of aspirin after the last episode.

This study aimed to confirm the implication of diclofenac in the adverse reaction reported by our patient and thereafter assess whether or not the patient could tolerate ASA in order to establish therapeutic recommendations.

Skin prick testing with diclofenac (25 mg/ml) yielded a negative result. After that, the patient

was orally challenged with increasing doses of diclofenac at 60-min intervals. Twenty minutes after ingestion of diclofenac 6 mg orally, she developed a generalized pruritic micropapular rash and neck erythema, as well as conjunctivitis and mild dyspnea without angioedema. These symptoms subsided after treatment with dexchlorpheniramine,

Oral challenge did not indicate any cross-reactivity, but long-term follow-up studies should be performed in similar patients to determine any increased risk of sensitization.

corticosteroids, and subcutaneous epinephrine. Two months later, she was orally challenged with increasing doses of ASA up to 500 mg without ill effect. Written informed consent was obtained.

Oral provocation with diclofenac was positive, showing that this drug caused the acute urticaria in our patient.

Although skin testing with diclofenac was negative, the positive oral challenge with this drug with further tolerance of ASA suggests an immunologic mechanism. Long-term follow-up of these patients would be necessary to determine whether they are at a higher risk of developing intolerance of ASA and other NSAIDs than the general population.

Key words: acetylsalicylic acid; diclofenac; cross-reactivity; urticaria.

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Aztreonam and ceftazidime: evidence of *in vivo* cross-allergenicity

AZTREONAM is a monocyclic β -lactam antibiotic which appears to lack cross-reactivity with other classes of β -lactams. Nevertheless, *in vitro* studies have shown cross-allergenicity between aztreonam and ceftazidime (1), a third-generation cephalosporin which is the only drug with the same side-chain (Fig. 1). These facts suggest that reactions to aztreonam are caused by its side-chain.

We present a case of sensitization to aztreonam with cross-reactivity to ceftazidime, proved by skin tests and tolerance of other β -lactams. No cases of *in vivo* cross-allergenicity between aztreonam and ceftazidime have been previously reported.

■ Our patient was a 57-year-old man who had had a cutaneous reaction to clindamycin 6 years before. He had a personal history of ischemic heart disease and left pneumonectomy because of cryptogenetic lung hypoplasia with recurrent respiratory infections, and this was treated with large courses of

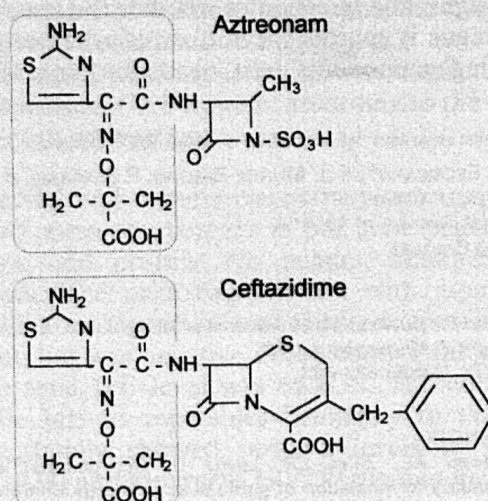


FIGURE 1 Structural formulas of aztreonam and ceftazidime.

many antibiotic agents. At the moment, pleural empyema was diagnosed, and the patient was started on amoxicillin/clavulanate (1 g IV) and aztreonam (1 g IV). Two hours after receiving the

Patients with allergic reactions to aztreonam should not be given ceftazidime.

first dose of both drugs, he developed a widespread pruriginous maculopapular eruption. Amoxicillin/clavulanate was stopped and aztreonam was continued, but the latter was with-

drawn 2 days later because of the increase of the skin reaction. Corticosteroids and antihistamines were needed to control the reaction, which had completely disappeared after 8 days.

Specific IgE was determined for penicillin and amoxicillin (Pharmacia® CAP System), and proved to be negative. Skin prick and intradermal tests were performed with penicilloyl polylysine and a minor determinant mixture (Allergopen® Merck), benzylpenicillin (10 000 UI/ml), amoxicillin (20 mg/ml), amoxicillin/clavulanate (20/5 mg/ml), cephadrine (20 mg/ml), ceftazidime (20 mg/ml), and aztreonam (2 mg/ml). The results were positive with aztreonam and ceftazidime in intradermal tests, with persistence of the reaction after 24 h. We performed patch tests with amoxicillin, amoxicillin/clavulanate, ceftazidime, and aztreonam, at concentrations of 20% in vaseline, obtaining positive results with ceftazidime and aztreonam. Negative results were obtained in 10 control subjects. We performed challenge tests with amoxicillin, amoxicillin/clavulanate, and

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