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Anaphylaxis to persimmon

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Key words: anaphylaxis; cross-reactivity; *Diospyros kaki*; persimmon fruit.

● THE persimmon is the edible fruit of the persimmon tree (*Diospyros kaki*), which belongs to the Ebenaceae family. The fruit consists of a berry, as large as an apple, orange in color, with soft, juicy pulp, sweet if ripe. In northern Italy, it becomes ripe in late autumn and is usually eaten fresh by itself.

This report describes, for the first time, an anaphylactic reaction after eating of a fresh persimmon by an atopic patient.

A 20-year-old man exhibited itching, generalized urticaria, facial angioedema, asthma, gastrointestinal complaints, and diarrhea, 10 min after eating a fresh persimmon fruit. He was rushed to the hospital emergency ward. Chest examination

identified wheezing. The usual blood pressure of 120/80 mmHg dropped to 90/60.

The patient had already been known to us for more than 10 years, initially for chronic rhinoconjunctivitis, and subsequently also for asthma. In 1986 and in 1994, the skin prick test (SPT) had been positive for dust mite (*Dermatophagoides pteronyssinus* [+++]) and *D. farinae* [+++]

and for *Alternaria alternata* (++) in 1994, total and specific serum IgE was determined by ImmunoCAP tests (IgE-FEIA and RAST-FEIA, respectively; Pharmacia CAP-System, Uppsala, Sweden). The total IgE was >1000 kU/l. Specific serum IgE was class 4 for Der p (21.5 kU/l), class 6 for Der f (>100 kU/l), and class 4 for *Alternaria* (26.8 kU/l). In 1986 and 1994, SPT was negative for pollens, animal dander, molds, and a battery of commercially available (Lofarma, Milan, Italy) food allergens.

Since the patient's symptoms were perennial without seasonal modifications and since the atmospheric dispersion of *Alternaria* spores in our region occurs only from April to September, the positivity to *Alternaria* was considered only nonclinical sensitivity.

Eating persimmon had never affected the patient before, although he always used to eat it in autumn, but he had recently experienced oral allergy syndrome (OAS) to kiwi (*Actidinia chinensis*).

SPT were performed by placing a piece of persimmon fruit and a piece of kiwi on the forearm and pricking through them (the prick-prick method). SPT with persimmon and kiwi yielded a wheal and flare reaction (persimmon wheal 15 mm in diameter with pseudopods; the kiwi wheal was 9 mm in diameter). The RAST to persimmon and kiwi was negative, despite clearly positive SPT. At a follow-up after 1 year, the patient refused further blood examination.

To our knowledge, this is the first report of an anaphylactic reaction caused by

persimmon fruit ingestion. The IgE-dependent mechanism was supported by immediate positive skin reaction. The RAST negativity was probably due to a marked increase in level of serum total IgE (total IgE was >1000 kU/l in 1994) and/or to the short time between symptoms and blood examination.

The patient was sensitized to dust mite and *Alternaria*, but not to pollens or food allergens except kiwi. Actidin (Act c 1), the main proteic component of kiwi (1), is an enzymatic protein belonging to the cysteine proteases, the structure of which is similar to Der p 1 (2).

These data suggest the intriguing possibility of cross-reactive allergenic structures in *D. kaki* and *D. pteronyssinus*.

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Azapropazone in aspirin intolerance

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Key words: aspirin intolerance; azapropazone; nonsteroidal anti-inflammatory drug (NSAID).

● PATIENTS with aspirin intolerance often react also to many nonsteroidal anti-inflammatory drugs (NSAID) such as indomethacin or diclofenac. The reactions do not depend on an allergic mechanism but are related to the pharmacologic action of the NSAID. The inhibitory effect on

cyclooxygenase activity leads to an imbalance of prostanoids. In the respiratory tract, this provokes asthma attacks. NSAID which do not have an inhibitory influence on cyclooxygenase activity (paracetamol, salicylamide) are usually well tolerated by these patients (1).

Azapropazone is a moderately potent anti-inflammatory analgesic agent in comparison with standard drugs, such as aspirin, indomethacin, and phenylbutazone (2). It is known to have only a weak anti-cyclooxygenase-1 and anti-cyclooxygenase-2 activity (3). Its safety in patients with immediate-type hypersensitivity to pyrazolones has already been demonstrated (4). We investigated the tolerance of this drug in patients with aspirin intolerance.

Thirty-four patients with documented aspirin intolerance were included in the study, whereas patients with IgE-mediated hypersensitivity to pyrazolones were excluded. The patients suffered from urticaria and/or angioedema after intake of aspirin. The diagnosis was based on an unequivocal case history and a negative prick and intradermal reaction to various analgesics including acetylsalicylic acid, pyrazolones, and azapropazone. The test concentration of each drug was 0.1%. Oral challenge tests with azapropazone were performed with a maximum single dose of 600 mg, which is the common single dose. The cumulative dose was 1060 mg (Table 1). The time interval between the dosages was 1 h. In case of doubtful reactions, the oral challenge was repeated with double-blind placebo controls.

The skin tests were completely negative in all patients. In 33 out of 34 patients, the oral administration of azapropazone in a cumulative dose of 1060 mg did not cause any adverse reaction. One patient experienced pruritus and redness of the neck 4 h after the 600-mg dosage, but this could not be confirmed in the double-blind, placebo-controlled rechallenge.

The result of this study is unequivocal, since none of the patients with aspirin intolerance showed any adverse reaction to azapropazone after placebo-controlled challenge. At first glance, this result might be surprising since azapropazone has a pyrazolidine moiety in its molecule, and it resembles phenylbutazone structurally (2). Nonetheless, it has only a weak anticyclooxygenase activity *in vitro*. Obviously, this inhibitory effect is negligible *in vivo*. Our study confirms the result of a smaller study in which the tolerance of azapropazone in 12 patients with aspirin-induced asthma was demonstrated (5).

The fact that cyclooxygenase inhibition is not a major effect of azapropazone is necessary for the good tolerance of the drug in patients with aspirin intolerance. Azapropazone has a more potent anti-inflammatory and analgesic effect than paracetamol (2). Therefore, azapropazone has a broader spectrum of indications than paracetamol. In addition, its general toxicity is relatively low.

In conclusion, azapropazone can be added to the list of NSAID that can be prescribed with little risk for patients with aspirin intolerance. From the allergologic point of view, this drug is as safe as paracetamol.

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Tannin inhibits histamine release

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Key words: histamine; mast cell; synthetic tannin.

● THE synthetic tannin tamol is a polyphenolic mixed condensation product. It is widely used for the topical treatment of pruritic dermatoses and has shown antipruritic effects in clinical studies of experimentally induced pruritus (1). Since skin mast cells play an important role as effector cells in type I allergic reactions and pruritic diseases, we have investigated the influence of the synthetic tannin tamol on IgE-dependent activation of human skin mast cells.

Table 1. Azapropazone in patients with aspirin intolerance

Number of study patients	Maximum single dose of azapropazone	Cumulative dose of azapropazone	Adverse reactions after open challenge	Adverse reactions after double-blind, placebo-controlled challenge
34	600 mg	1060 mg	1	0