

Case report

First report of anaphylactic reaction after fig (*Ficus carica*) ingestion

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We report an anaphylactic reaction which occurred very shortly after ingestion of a fresh fig. The IgE-dependent mechanism was demonstrated on the basis of positivity of the prick test performed with fresh fig (*Ficus carica*) extract. In addition, we were able to detect specific IgE to the same extract in the serum. The patient did not demonstrate sensitization to other common allergens involved in respiratory and food allergies. However, detection of specific IgE to *F. benjamina* indicated a sensitization to weeping fig. The CAP F. *benjamina* was partially inhibited by preincubation of the serum with fig extract, suggesting that these two species of *Ficus* share some common allergens. In this context, the assumption can be made that weeping fig was responsible for the initial sensitization in this patient.

Key words: anaphylaxis; cross-reactivity; *Ficus benjamina*; *Ficus carica*; fig fruit.

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Fig is the edible fruit of the fig tree (*Ficus carica*), which belongs to the Moraceae family and to the genus *Ficus*. Besides the fig tree, several species of *Ficus* are ornamental plants, and some produce rubber. Fig trees grow in Mediterranean countries; some 700 varieties of fig have so far been listed. Fresh figs are usually eaten alone, but can also be served as a vegetable with white meats or ham. The first reported symptomatology caused by the fig tree or figs involved photosensitization mechanisms. In fact, psoralens and bergaptenes are components of the bud and leaf sap (10). Phototoxic reactions have also been observed after contact with the fig itself (7). In 1985, Axelsson et al. (1) reported IgE-mediated allergy to the weeping fig (*F. benjamina*) as an occupational disease. Later they demonstrated that F.b. was also a source of aeroallergens in the domestic environment (2). Several other cases (4, 8) have been reported, confirming that F.b. is a perennial indoor allergen of increasing prevalence. This report describes, for the first time, an anaphylactic reaction after eating a fresh fig in a patient with sensitization to latex from *F. benjamina*, suggesting possible cross-reactivity between edible fig and *F. benjamina* allergens.

Case report

A 77-year-old woman presented an anaphylactic reaction after eating a fresh fig imported from Brazil. She exhibited itching, generalized urticaria, facial angioedema, asthma, and gastrointestinal complaints. The asthmatic attack occurred immediately after fig ingestion, as well as mediasternal retrosternal burning-type pains, vomiting, and diarrhea. She was rushed to the hospital emergency ward. Chest examination identified wheezing. The patient was given a 4-mg injection of betamethasone. The usual blood pressure of 20/11 dropped to 16/9.

Three days before, she had developed localized periorbital edema and wheezing dyspnea 3 h after eating the same kind of fig. Our questioning elicited a mention of family atopy (one of her three children was asthmatic). There was no personal history of atopy. This woman used to be an office worker; she is now retired and lives in an apartment with no common allergy-inducing factors present. However, she had had a weeping fig (*F. benjamina*) in her flat for 18 months but had got rid of it because it had grown too large. Eating mussels had caused immediate diarrhea and vomiting on four occasions.

Material and methods

Allergen preparation

Crude fig extract was prepared as follows: fresh figs (from France or from Italy) were minced and homogenized in a Warring blender. After centrifugation (10000 g for 30 min), the supernatant was concentrated and referred to as crude fig extract (CFE).

Skin tests

Skin prick tests (SPT) were performed with a battery of commercially available (Laboratoires Stallergènes, Paris, France) common inhalant allergens (trees, *Dermatophagoides*, Graminae, Compositae, animal danders) and food allergens (kiwi, orange, peanut, passion fruit, grape, potato, rye, rice, cow's milk, egg, mussel, tuna, beef, chicken, chocolate, *Saccharomyces cerevisiae*, and *S. minor*) as well as with latex from *Hevea brasiliensis*.

IgE antibody determinations

Total serum IgE was measured by immunonephelometry, according to the manufacturer's instructions (Behring, France).

The presence of specific IgE antibodies to fig in the serum was demonstrated by detecting IgE bound to polystyrene tubes (Startube from Nunc) which had been coated overnight with CFE. After 2-h incubation with serum, tubes were successively incubated for 1 h with rabbit antihuman IgE (Dako, Denmark), biotinylated antirabbit immunoglobulins (Vector, France), and alkaline phosphatase-labeled streptavidin (Amersham, France). Enzymatic activity was detected by incubation with paranitrophenyl phosphate solution. Results were expressed as absorbances at 405 nm (A_{405}). Specific IgE antibodies to other allergens were measured by the immunoenzymatic CAP method (Pharmacia, Sweden).

CAP inhibition

The inhibition of CAP *F. benjamina* was determined after incubating patient serum for 1 h with crude fig extract or with PBS.

Results

In the patient, the wheal diameter of the prick test with CFE was found to be of the same size as the phosphocodine control positive test. The prick test with CFE was negative in nine control allergic patients with no history of fig sensitivity. The same findings were observed by puncture of the skin through a fresh fig leaf. The skin prick tests were also

positive to wheat flour, melon, sardine, and banana. Skin tests were negative for common inhalant and food allergens as well as for latex from *H. brasiliensis*.

Total serum IgE was 98 kU/l. Specific IgE to fig extract was found in the patient's serum (A_{405} : 0.109). Under the same conditions, the mean A_{405} for nine control sera was 0.024. Specific IgE to weeping fig (F.b.: 25 kU/l) was also detected. The CAP for latex, banana, melon, mulberry, nettle, wall pellitory, mussel, peanut, wheat flour, and yeast was negative.

Preincubation of serum with 5 and 10 μ g of protein from crude fig extract inhibited CAP *Ficus* by 42 and 49%, respectively.

Discussion

To our knowledge, this observation is the first report of an anaphylactic reaction caused by fig ingestion. The IgE-dependent mechanism was supported first by immediate positive skin reaction induced either by prick test with fig extract or by puncture through a fresh fig leaf. Secondly, a significant amount of specific IgE to fig extract was present in the patient serum, as compared to sera from atopic patients not sensitized to fig. Despite a family history of atopy, total IgE was in the normal range. The patient was not sensitized to other common inhalant and food allergens. There were no sensitizations to other families related to *F. carica* such as mulberry and Urticaceae. However, since *F. carica* belongs to the same genus as *F. benjamina*, the second point of interest was to determine whether there is cross-reactivity between figs from *F. carica* and latex from *F. benjamina*. In fact, the CAP *F. benjamina* was strongly positive, demonstrating sensitization to weeping fig. Moreover, fig extract was able to inhibit CAP *Ficus*. These data suggest the existence in *F. carica* and *F. benjamina* of cross-reactive allergenic structures. This extends the observations of Axelsson et al. (3), who found extensive allergenic cross-reactivity between *F. benjamina* and seven other indoor plants of the genus *Ficus*. In addition, since the CAP latex was negative, it is likely that these structures differ from those responsible for cross-reactivity between *F. benjamina* and latex from *H. brasiliensis* (6). Finally, in reference to other respiratory and food allergy associations (5, 9), it is probable that allergy to fig fruit was a consequence of initial sensitization to weeping fig.

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