

Allergy to azufaifa fruit and latex

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Key words: anaphylaxis; azufaifa; chitinases; cross-reactivity; fruit allergy; hypersensitivity; immunoblotting; latex.

● AZUFAIFA is the edible fruit of the azufaifo tree belonging to the Rhamnaceae family. Only one case of urticaria and angiedema due to azufaifa has been previously published as an abstract (1).

The patient was a 38-year-old nurse with a previous history of latex allergy (contact urticaria, rhinoconjunctivitis and asthma) and food allergy (banana, chestnut, kiwi and avocado). In September of 2000, she suffered from urticaria, angiedema, rhinoconjunctivitis, dyspnea, wheezing, abdominal pain, diarrhea and sickness, immediately after eating some azufaifas (that she previously tolerated). She progressively improved after treatment with subcutaneous epinephrine and intravenous corticosteroids and antihistamines, and her symptoms subsided 24 h later.

Skin prick tests to common allergens (foods, pollens, molds and epithelia) were negative. Prick test with a commercial latex extract (ALK-Abelló) was positive. Prick-by-prick tests proved positive with latex gloves and crude extracts of azufaifa, avocado, chestnut, kiwi and banana. Total serum IgE was 33 UI/ml (Alastat, DPC). IgE antibodies specific to kiwi, banana, chestnut and avocado were < 0,35 kU/l; latex 2,07 kU/l and azufaifa 0,37 kU/l (CAP System, Pharmacia, Uppsala, Sweden).

A SDS-PAGE was performed with an extract of azufaifa prepared by us following previous descriptions (2). Lyophilized crude extracts of avocado, banana, chestnut, latex and grass pollen and a commercial extract of olive (Inmunotek) and a recombinant chestnut class I chitinase were used for the inhibition assays.

The SDS-PAGE showed several bands from 15 to 60 kDa. IgE-immunoblotting

with the patient's serum revealed a single band of around 30 kDa. The recognition of this band was inhibited by a latex crude extract, but not by avocado, banana, chestnut, or pollens extracts. The recombinant chestnut class I chitinase extract weakly inhibited this band. Immunoblotting with the serum of the patient revealed a strong band of around 30 kDa in the chestnut chitinase recombinant.

Immunoblotting with 19 control sera (5 non atopic and 14 atopic subjects) was performed. Only two of them (both sensitized to grass pollen) showed a strong detection of two bands of around 55 and 60 kDa. The serum which presented the strongest detection was used in cross-reactivity assays (Fig. 1). This serum showed inhibition by latex and avocado, but a greater inhibition was obtained with the grass pollen extract. It should be noted that this patient had not known sensitivity to azufaifa, avocado or latex.

Several latex allergens have been cloned and sequenced. Hevein (Hev b 6.02) contributes to the increased prevalence of fruit allergies in individuals allergic to latex. It is a polypeptide of 43 amino acid residues, which shares high-sequence similarities with pathogenesis-related proteins (PR-3 and PR-4 3–5). Class I chitinases (PR-3 family) are monomers of 25–35 kDa, and contain an N-terminal region called hevein domain which shows cross-reactivity with latex hevein (4).

Our patient recognized a single band of around 30 kDa, a molecular weight similar to class I chitinases. It would be possible that the catalytic domain of chitinases could be involved in cross-reactions. Therefore, it could explain the cross-

reactivity between latex and azufaifa but not with chestnut, banana and avocado (cross-reactivity among this foods are mainly due to hevein domain). All these findings suggest that the sensitization in our case is different to that described for other fruits in the latex–fruit syndrome. As we have not sequenced the protein we cannot affirm that it is a chitinase and, actually, it would be possible that the sensitization could be due to another allergen.

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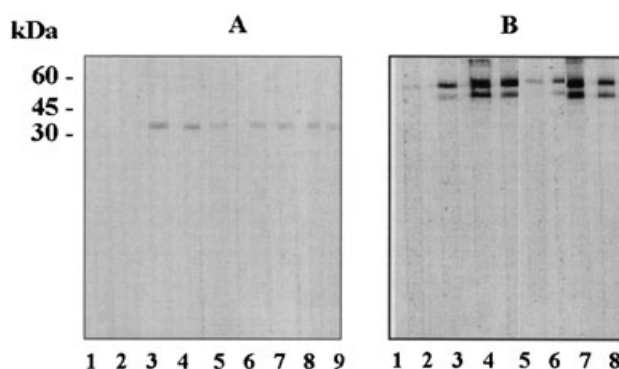


Figure 1. Immunoblotting inhibition assays with azufaifa extract as solid phase antigen and with azufaifa (lane 1), latex (lane 2), PBS (lane 3), olive (lane 4), grass pollen (lane 5), avocado (lane 6), banana (lane 7) and chestnut (lane 8) as soluble inhibitors. Serum from a pollen allergic subject.

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Occupational asthma due to mitoxantrone

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Key words: bronchial challenge; mitoxantrone; occupational asthma.

● REPORTS ON occupational allergy due to cytostatics are scarce and mainly concern allergic contact dermatitis and allergic contact urticaria (1,2). To our knowledge, there is no published case report on mitoxantrone-induced allergic asthma.

We present the case of a 41-year-old nurse who had worked for 13 years in an oncology ward with exposure to antineoplastic drugs. For three years she had suffered from rhinorrhea, dyspnea and cough attacks, occurring 1–2 h after

beginning work. For the first two years the patient presented only work-related symptoms; in the third year she developed dyspnea also away from work.

Clinical examinations, as well as routine laboratory parameters, remained normal. The total IgE (CAP System, Pharmacia, Sweden) level was low (4.89 k μ I). No specific IgE antibodies against latex, disinfectants and β -lactams were detected. Also skin prick tests to common allergens, latex (Allergopharma, Germany) chloramine and chlorhexidine (0.001, 0.01 and 0.1% solutions) were negative. The histamine provocation test showed unremarkable bronchial hyperreactivity (PC₂₀ = 0.093 mg/ml). The patient was subjected to a number of single-blind bronchial challenge tests with subsequent cytostatics monitored by spirometry and peak expiratory flow (PEFR) measurements. The provocation with mitoxantrone induced a 15% fall in forced expiratory volume (FEV₁) at 1 h, with a 20% decrease 4 h later. One week later the challenge with mitoxantrone was repeated. Bronchoalveolar lavage fluid (BAL) was taken before and at 6 and 18 h after the provocation. Significant increase in lymphocytes and neutrophil proportion was observed, especially at 18 h after the challenge (Fig. 1). We also noted eosinophil influx, up to 4%, and more than a two-fold increase (from 8.0% before to 18.2%) in the permeability index. The same provocation test was performed in an asthmatic volunteer not exposed to antineoplastic drugs. There were no significant changes in cell proportions

apart from a slight increase in neutrophils (from 1% to 6% at 18 h).

The reason for recognition of mitoxantrone-induced occupational allergic asthma was the clinical response during specific challenge test accompanied by changes in BAL (increase in the eosinophil percentage and permeability index). It seems possible that some cases of asthma induced by cytostatics are not recognized to be occupational because insufficiently sensitive diagnostic methods are used or because the allergogenic properties of antineoplastic drugs are ignored.

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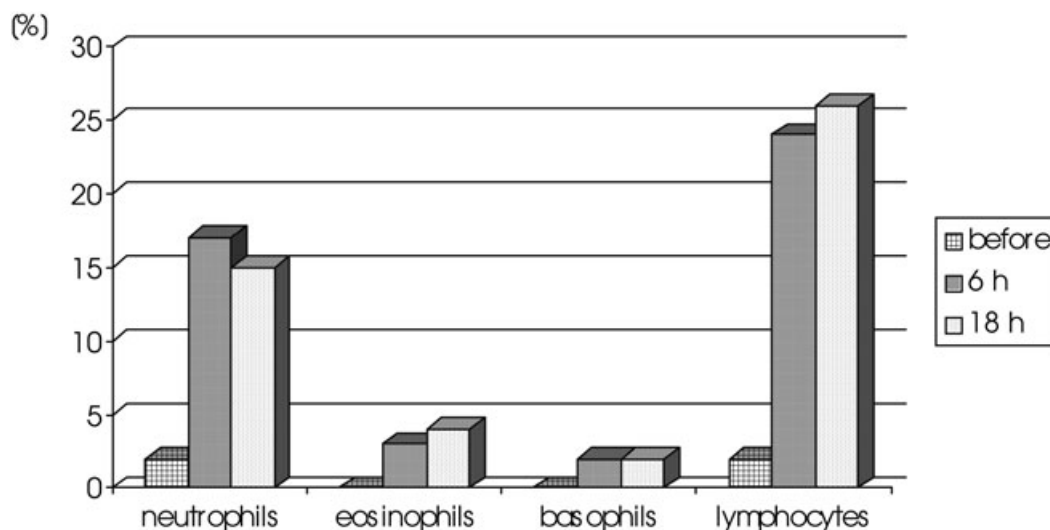


Figure 1. The proportion of neutrophils, eosinophils, basophils, and lymphocytes in BAL before and after bronchial challenge with mitoxantrone.