

recommended. This attitude raises several questions. Are we delaying the complete immunologic tolerance to egg proteins? On the contrary, can this measure favour acquisition of oral tolerance? More studies are necessary to completely answer these questions.

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## Corn-induced hypersensitivity pneumonitis

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**Key words:** corn flour; flow cytometry; hypersensitivity pneumonitis.

We studied a 50-year-old male farmer, non-smoker, who had worked harvesting corn for 17 years. For the last 4 years, he had recurrent episodes of fever, dyspnea and chest pain during the corn harvest season, 4–5 h after corn dust exposure. The

**Corn flour: a new allergen related to hypersensitivity pneumonitis.**

patient associated these symptoms with the handling of corn and showed marked relief when exposure to this material was

avoided. Several corn samples were sent to our laboratory by the patient. These samples were ground to obtain a very thin dusty material and 5 g were extracted by magnetic stirring in 50 ml of phosphate-buffered saline (PBS, pH 7.3), for 24 h, passed through filter paper, dialyzed against PBS for 24 h, and then filtered through a 0.22 µm Millipore filter for sterilization. Different samples of corn provided by the patient were studied in order to identify the presence of microorganisms: *Penicillium* sp., *Fusarium* sp., and *Rhizopus* spp. were able to be isolated. Double immunodiffusion tests for precipitin antigens were performed according to Ouchterlony's method (1). Precipitation bands were shown between our patient's serum and *Aspergillus fumigatus*, *P. notatum*, *P. nompactum*, *P. brevicompactum*, *P. roqueforti*, the corn extract and corn flour extracts. Specific IgG antibodies against *A. fumigatus*, *P. notatum*, *A. alternata*, *C. herbarum* and corn flour extracts were also found in the patient's serum using the ELISA method.

Pulmonary inhalation provocation test (PIPT) was performed with the corn extract at 1/10 p/v, following the method previously described (2). Five minutes after PIPT, a bronchoconstriction reaction was detected. Four hours afterwards, the patient suffered clinical symptoms similar to those reported in his occupational environment. The patient presented a significant decrease in PaO<sub>2</sub> from baseline blood gas and marked leukocytosis 6 h after PIPT. Spirometric

values are expressed in Fig. 1. Twenty-four hours after PIPT, bronchoalveolar lavage (BAL) studies and transbronchial biopsy were carried out. The histopathological examination of transbronchial biopsy specimens revealed interstitial alveolitis with lymphocyte-macrophage infiltrate. The lymphocytes included in the BAL were subtyped according to their expression of different surface markers. For this purpose, all cells in the BAL were washed in saline and stained with fluorochrome-conjugated monoclonal antibodies, specific for the different surface markers, prior to their analysis in a FACScan flow cytometer. By means of flow cytometry, we obtained the following results: lymphocytes, 19.5%; mean CD4/CD8, 1.3; natural killer cells, 6.6%; B lymphocytes, 2.4%; neutrophils, 29%; monocytes/macrophages, 5.5% and eosinophils, 2%. We have described a case of hypersensitivity pneumonitis (HP) caused by corn reported by our patient. His clinical history suggested a possible sub-acute form of HP. We have found precipitating antibodies in the patient's serum using diffusion techniques and ELISA IgG. Also, we performed the PIPT with the corn extract supplied by our patient. BAL fluid revealed an acute inflammatory infiltrate with prevalence of neutrophils and lymphocytes, an increase of the CD8 subpopulation and a decreased CD4/CD8 ratio compared with the normal population. There is only one case described in the medical literature of HP caused by corn contaminated with *A. flavus* (3). No pulmonary functional

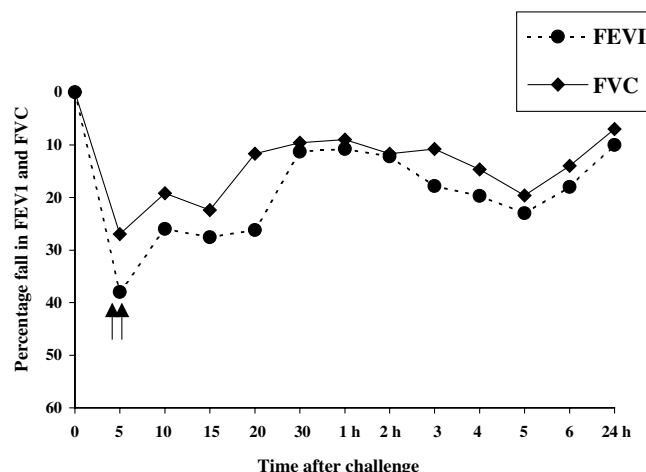


Figure 1. Five minutes after PIPT, a bronchoconstriction reaction was detected. Two puffs of salmeterol were given to the patient. Arrows indicate salbutamol inhalation (400 µg).

studies, PIPT or fiberoptic bronchoscopy had been performed. We present the first well-described case of HP in which the immunologic studies and PIPT confirm that the corn was the causal agent of this process.

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## Food allergy to alcohol

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**Key words:** alcohol; delayed hypersensitivity; ethanol; food allergy; oral challenge test.

We report an original case of food allergy to ethanol. Rare cases of contact eczema have been reported in the literature (1). Anaphylactic reactions to alcohol are exceptionally rare (only eight cases reported in the literature) (2). To our knowledge, this is the first case of food allergy reported in the literature.

**We report an original case of food allergy induced by delayed hypersensitivity to alcohol.**

At the age of 12 years, Mr X presented with skin rash 12 h after a compress containing alcohol had been applied to his calf. The skin rash lasted for 2 days. The same symptoms occurred when alcohol was used to disinfect wounds or when ointment containing alcohol was applied to his skin. He has used alcohol-free perfume and cosmetics since then, and avoids using topical products containing alcohol. At 14 years of age, he felt a sensation of skin burning and rash after a glass of champagne. The same reaction occurred after two glasses of beer, but he tolerates dishes cooked in wine very well. The skin rash occurred 4 h after ingestion and lasted for about 5 days.

Mr X has a history of seasonal rhinitis and asthma with sensitization to mite, grass, birch and ash pollens.

The patch-test was + + + positive to 10% alcohol and induced vesicular eczema.

Biopsy showed normal epidermis with a slight exoserosis. There was a selectively perivascular inflammatory infiltrate in the dermis, consisting principally of T lymphocytes, of which 10% were activated (CD45 Ra +), 80% CD4+ and 20% CD8+ mixed with rare B lymphocytes (CD20+), 3% eosinophils and rare histiocytes. There were no deposits of IgG, A, M, C1q, C3 or fibrinogen.

The oral challenge test was negative with 5 g alcohol and positive with 10 g alcohol, inducing generalized rash on the trunk and buttocks, 5 h later and lasting for 48 h. The histamine level remained stable at 0, 5, 15, 30, 60 minutes and 5 h after the oral challenge test, ranging from 2.1 and 3.2 nM. Diamine oxidase activity was normal. There was no increase in histamine metabolites: methyl histamine and methyl imidazolacetic acid (3). The oral challenge test to metabisulfites (30 mg) was negative. The lymphocyte transformation test was negative with 0.1% alcohol.

This report documents the possibility of contact allergy, later complicated by generalized skin rash caused by food allergy to alcohol and delayed hypersensitivity reaction. The hypersensitivity reaction was dose-dependant, with onset at 10 g but not occurring with 5 g. The fact that our patient can eat foods cooked in wine indicates that alcohol only is involved as it evaporates during cooking.

Alcoholic drinks contain ethanol, as do medicinal suspensions and solutions for oral ingestion. Ethanol is also applied topically for its bactericidal properties. It is found in many topical products, transdermal systems, perfumes, etc. The patient has been given a card specifying that he is allergic to medication, and that all pharmaceutical and parapharmaceutical products containing alcohol are to be avoided.

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## Prolonged cyclosporin-A treatment for severe chronic urticaria

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**Key words:** chronic urticaria; cyclosporin-A.

The beneficial effects of cyclosporin-A (CsA) in severe chronic urticaria (CU)