

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)

are well described. In Fradin et al.'s seminal work, a 6-mg/kg dose was administered for a period of 3–9 weeks to three patients suffering from CU. Despite its beneficial effects, CsA was discontinued in all the three patients because of its undesirable side-effects (1). Barlow et al. reported the beneficial effect of 3 mg/kg CsA for a period of 1 month to 10 patients (2). This group described a modified protocol administration of CsA over a 3-month period, with the initial dose at 3 mg/kg which was gradually reduced (3). One-third of the CU patients were cured, with another third experiencing reduced symptom severity. Notably, in one-third of the patients in remission, urticaria relapsed 3–6 months after CsA discontinuation (4). Furthermore, those who relapsed developed CsA unresponsiveness (escape of therapy) following a second course of CsA, even when higher doses of CsA were administered (E. Toubi, personal observation).

In the present study, the administration of CsA for prolonged periods to patients who experienced a severe urticarial relapse following the tapering of CsA has been studied.

Case I. A 54-year-old male patient in whom severe CU could be controlled only with repeated courses of prednisolone administered over 2 years, was studied. CsA, at 3 mg/kg/day induced a full remission, yet each tapering attempt below 2.0 mg/kg/day was followed by a severe relapse, necessitating the return to the starting dose. This regimen was continued for 8 months, without any side-effects, and ultimately resulted in full cure during a 1-year follow-up.

Case II. The patient was a 51-year-old steroid-dependent female with severe CU lasting 14 months. Gradual tapering of CsA dosage after 3 months resulted in a severe relapse. Despite diarrhea and mild peripheral neuropathy, she insisted on continuing a dosage of 2 mg/kg/day. After 6 months the CsA dose was gradually tapered and patient remained asymptomatic during a 5-month follow-up.

More prolonged therapy may reduce the relapse often accompanying the typically short cyclosporin-A (CsA) treatment regimens for chronic urticaria.

Case III. The patient was a 51-year-old female with severe CU lasting 3 years that was successfully treated with CsA. As previously described in the aforementioned cases, we were unable to taper the dose below 2 mg/kg/day because of subsequent relapse. Moreover, corticosteroids were contraindicated because of the patient's comorbidity to diabetes mellitus. Six months after the initiation of treatment (with six more months of treatment planned), the patient continues to be asymptomatic.

As most reports on CsA treatment suggest this regimen for a limited period of time, and urticaria relapses follow CsA discontinuation in many, caregivers are urged to consider a more prolonged therapy with CsA. The notion of allowing an extended treatment period is especially attractive in those patients in whom 'escape of therapy' may ensue with the discontinuation of CsA.

We propose that, as is customary in the management of autoimmune diseases, CsA should be considered as a possible treatment option early in the course of severe CU. When prolonged treatment with prednisolone is inadvisable, the use of CsA may have added value. Further experience with prolonged CsA treatment is needed in order to determine the extent to which this regimen might reduce the incidence of relapse upon CsA discontinuation and thus provide enhanced treatment efficacy.

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Systemic allergic reaction by a human insulin analog

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Key words: anaphylaxis; desensitization; human insulin; lispro; local reactions.

Human insulin varies from bovine and porcine insulin in three and one amino acids, respectively. Insulin lispro is a new rapid-acting analog of human insulin identical to the human species except for the location of two amino acids B28 (proline) and B29 (lysine). It is considered less immunogenic than human insulin itself.

A 62-year-old diabetic man with stable ischaemic heart, peripheral vascular disease, extrinsic asthma, and urticaria by pirazolones, had been treated for 10 years with oral hypoglycemic agents. Treatment with human insulin was required. He received daily doses of insulin lispro with protamine with good tolerance for the first 4 months. During the following 2 months, he occasionally developed self-limited local 10 mm immediate reactions. Due to a persistent bad metabolic control, insulin was increased. One week later, minutes after the subcutaneous injection of lispro (34 IU) he suffered a generalized urticaria with severe bronchospasm that required treatment with adrenalin. Insulin was discontinued.

Skin prick test (SPT) with regular and lispro insulin 100 IU/ml were positive. SPT (10 mg/ml) and intradermal tests (0.1 and 1 mcg/ml) to protamine were negative.

Specific IgE (s-IgE) to human insulin (CAP-Pharmacia, Uppsala, Sweden)TM measured 10.4 kU/l. S-IgE by enzyme-

Local reactions after a successful desensitization in an anaphylaxis by insulin lispro.