

exercise. Only oral challenge with a wheatmeal roll followed by an additional exercise test on the bicycle ergometer produced immediate-type reactions. After 20 min, itching of the skin on the neck and back occurred, followed by generalized urticaria with swelling of the upper lip. The patient received intravenous injections of 4 mg of dimetindene maleate and 250 mg of methyl-prednisolone. This relieved the symptoms within minutes.

In the literature, 163 cases of food-dependent, exercise-induced anaphylaxis are described; they have been reviewed by Steurich & Feyerabend (3). Although various foods (tomato, peanut, soybean, and seafood) have been associated with food-dependent, exercise-induced anaphylaxis, the most frequently reported cause of these reactions seems to be wheat (3). The activities required to precipitate symptoms were running, ball games, tennis, and walking.

Our patient was atopic and suffered from face dermatitis and perennial rhinitis with allergy to house-dust mites. The positive skin test and the detection of specific IgE antibodies made it possible to demonstrate an immediate-type allergy to wheat flour and gluten (gliadin). Recently, Palosuo et al. demonstrated by ELISA that the major allergen of wheat is gliadin (4). Only oral challenge with a wheatmeal roll followed by exercise produced immediate-type reactions such as generalized urticaria and swelling of the upper lip, and thus led to the diagnosis of food-dependent, exercise-induced anaphylaxis to wheat flour (gliadin).

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Successful treatment of chronic urticaria

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Key words: chronic autoimmune urticaria; leukotrienes; montelukast.

● For many years, the treatment of chronic idiopathic urticaria (CIU) has been frustrating for both patients and physicians, since the cause of the disorder could not be identified, and common antialler-

gic drugs could relieve the symptoms but seldom cure the disease. In 1986, Grattan et al. (1) reported the presence of a serologic mediator of whealing in some patients with CIU. Subsequently, it was demonstrated that the serum histamine-releasing activity in patients with CIU can be attributed to:

- 1) IgG auto-antibodies directed against the alpha chain of the high-affinity IgE receptor
- 2) IgG autoantibodies against IgE
- 3) a 30-kDa serum factor which has not yet been characterized.

An autoimmune basis can be found in 25–60% of patients with CIU (2); this observation has provided the basis for new therapeutic interventions, such as plasmapheresis, high-dose intravenous immunoglobulins, and cyclosporin. However, these treatments have been reserved for the most severe cases.

In this report, we describe a patient whose chronic autoimmune urticaria was successfully treated with the leukotriene-receptor antagonist montelukast.

A 28-year-old woman had complained of recurrent urticaria since she was 27, without any apparent relation to particular foods or drugs. Her father had suffered from CIU for 15 years as well, and he was being treated by low-dose oral prednisone and antihistamines. Physical examination of the patient was normal, and laboratory tests were normal, including erythrocyte sedimentation rate, eosinophil count, complement factors, C1 inhibitor, antinuclear, anti-DNA, and antithyroid peroxidase antibodies, viral markers for hepatitis B and C, anti-HIV antibodies, anti-*Helicobacter pylori* antibodies, urinalysis and oropharyngeal swab. The search for parasites in the stools was negative. Skin prick tests and specific IgE determination for most common food allergens were negative. Chest radiograph and abdomen ultrasound examination were normal. Before coming under our observation, the patient had been taking oral antihistamines (cetirizine or mizolastine) and short courses of oral steroids for 9 months. After temporary withdrawal of antihistamines, we performed intradermal test with autologous serum, according to the technique of Sabroe et al. (3), and we observed positive immediate and late reactions, without a clear distinction between the two phases. In fact, the patient presented a wheal and flare reaction which appeared a few minutes after the injection; subsequently, erythema and induration gradually increased, reaching a

The leukotriene antagonist montelukast seems to be effective in autoimmune urticaria.

maximum after 9–12 h, and subsided after 24–36 h. We decided to resume low-dose oral prednisone (5 mg daily) together with cetirizine (10 mg daily). This treatment controlled symptoms, but after 1 month the patient stopped taking cetirizine, and urticaria returned. In the attempt to control the patient's symptoms, we prescribed a combined therapy of low-dose prednisone, cetirizine, and the leukotriene-receptor antagonist montelukast (Singulair®) – 10 mg daily. The patient did not present any further urticarial rash, and after 1 month cetirizine and prednisone were stopped. Montelukast is now the only drug taken by the patient, and she has not presented any symptom in over 2 months.

Recently, a few reports have shown that leukotriene antagonists (zafirlukast or montelukast) (4, 5) or leukotriene-synthesis inhibitors (zileuton) (6) may be effective in controlling CIU, particularly when associated with aspirin intolerance. Moreover, chronic autoreactive urticaria serum has been demonstrated to induce leukotriene production, in addition to histamine release, by basophils (7).

Our patient's urticaria was characterized by an immediate and prolonged response to intradermal autologous serum and therefore can be considered autoreactive. The favorable clinical response to montelukast provides an indirect demonstration that cysteinyl-leukotrienes play an important role in both early and late skin responses elicited by autologous serum. Leukotriene antagonists may have a role in the treatment of some patients with chronic autoreactive urticaria.

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Anaphylaxis to starch

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Key words: basophil activation; CD63; flow cytometry; hydroxyethyl starch (Haes-Steril®); skin test.

● A 46-YEAR-OLD woman was investigated for a severe perioperative anaphylactic/anaphylactoid reaction. Induction of anesthesia with cisatracurium, propofol, and sufenta was uneventful. However, immediately after the beginning of infusion of the plasma expander Haes-Steril® (Fresenius Kabi), the patient developed hypotension (MAP <60 mmHg) associated with urticaria. The infusion was stopped, and she was successfully treated with epinephrine and dopamine. Personal history revealed an atopic background and several uneventful surgical interventions. Laboratory analyses, 4 months after the event, showed a normal blood count, normal complement profile, total IgE of 370 kU/l, and latex IgE of <0.35 kU/l (Immuno-CAP, Upjohn-Pharmacia, Belgium). Flow cytometric analysis (FACScan, Becton Dickinson) of activated basophils was performed with fluorescein isothiocyanate (FITC)-conjugated anti-CD63 and phycoerythrin (PE)-conjugated anti-IgE double labeling (Basotest®, Orpegen Pharma, Heidelberg, Germany) (Table 1). The stimulation with Haes-Steril was positive in the patient. In addition, donor basophil activation was assessed after prior incubation with serum of the patient. Upon challenge with Haes-Steril, passively sensitized basophils of the donor showed significant upregulation of CD63 expression. The patient did not allow skin testing.

A reaction to a plasma expander with basophil activation.