

Lolium sp., *Olea* sp., *Plantago* sp.,
Artemisia sp., *Platanus* sp., *Chenopodium*
sp., and *Parietaria* sp.

The conjunctival provocation test (1) was positive in our patient at 0.5 mg/ml. The nonspecific bronchial reactivity test with methacholine (2) was negative. A specific bronchial provocation test (3) was performed with extract of linden pollen and found to be negative. Specific IgE (Pharmacia CAP System) to *T. cordata* pollen was positive (27.7 kUA/l) and also to pollens of *Lolium perenne* (13.8 kUA/l), *Artemisia vulgaris* (10.8 kUA/l), *Plantago ovata* (9.6 kUA/l), and *Platanus acerifolia* (15.5 kUA/l).

By means of RAST inhibition with paper disks sensitized to *T. cordata* and the patient's serum (4), no inhibition of RAST was detected with *Lolium*, *Olea*, and *Plantago* extracts, but a partial inhibition (~30–40%) was detected with *Artemisia* and *Platanus* pollen extracts. IgE immunodetection of *T. cordata* extract after SDS–PAGE indicated that the patient's serum had IgE against several bands, mainly at ~50 kDa and also at ~23 and ~10 kDa (Fig. 1). Similar bands were detected in the *Platanus* extract, but the ~50 kDa band was much weaker. Inhibition of *Tilia*-IgE immunodetection with *Platanus* extract showed the existence of cross-reactivity of the smaller bands, but not of the 50-kDa band (Fig. 1).

In conclusion, exposure to linden pollen can induce IgE-mediated rhinoconjunctivitis and cough, as demonstrated by SPT, conjunctival provocation, and IgE *in vitro* tests. A specific 50-kDa linden allergen has been detected. Linden pollen must be taken into account when the patient's symptoms correlate with linden pollination, as other relevant pollens in the area (olive and grass) could hide it (5).

*C/San José, 11, 4^oA
13500 Puertollano
Spain
E-mail: pmj01b@nacom.es

Accepted for publication 29 January 2001
Allergy 2001; 56:457–458
Copyright © Munksgaard 2001
ISSN 0105-4538

References

1. JIMENEZ A, MORENO C, MARTINEZ J, et al. Sensitization to sunflower pollen: only an occupational allergy? *Int Arch Allergy Immunol* 1994;**105**:297–307.
2. CHATHMAN M, BLEECKER ER, NORMAN P, SMITH PL, MASON P. A screening test for airways reactivity. An abbreviated methacholine inhalation challenge. *Chest* 1982;**83**:15–18.
3. CHAI H, FARR RS, FROELICH LA, et al. Standardization of bronchial inhalation challenge procedures. *J Allergy Clin Immunol* 1975;**56**:323–327.
4. CESKA M, ERIKSSON R, VARGA JM. Radioimmunosorbent assay of allergens. *J Allergy Clin Immunol* 1972;**49**:1–9.
5. FEO F, MARTINEZ A, PALACIOS R, et al. Rhinoconjunctivitis and asthma caused by vine pollen: a case report. *J Allergy Clin Immunol* 1999;**103**:262–266.

Ulcerative colitis possibly due to hypersensitivity to wheat and egg

D. A. Moneret Vautrin*, J. Sainte-Laudy, G. Kanny

Key words: food allergy; positive CAST; recovery; ulcerative colitis.

• A 44-YEAR-OLD woman was treated for ulcerative colitis. Abdominal pain and diarrhea had appeared in 1980. The diagnosis was confirmed by colonoscopy and histology of biopsies. Since 1987, she had

been regularly treated with hydrocortison rectal foam and mesalazine. She observed a very strict diet, poor in fiber, without roughage or green

Healed by diet, antihistamines, and cromoglycate.

vegetables. Nonetheless, the abdominal pain appeared several times per week, and she presented regularly one soft stool per day. Several episodes of glairy and bloody stools were also noted.

As food allergy to egg was diagnosed in her two children by skin tests, specific IgI determination, and standardized oral provocation tests, she underwent tests in January 2000 to detect masked food allergy.

Prick tests were negative to the usual aeroallergens and to 25 foods, particularly wheat flour and egg, which she usually consumed. Total IgE was within the normal range (138 kU/l), and egg- and wheat flour-specific IgEs were negative (Pharmacia CAP). The lymphocyte activation test (TAL) by flow cytometry (1/2) showed 8% spontaneous activation (normal <2%), normal nonspecific activation in the presence of phytohemagglutinin A (44%), and nonsignificant activation (<2%) in the presence of wheat flour, egg, and gliadin extracts (Allerbio Laboratory, France). The basophil activation test (TAB) by flow cytometry (2, 3) showed normal anti-IgE induced activation (44%) and nonsignificant activation (<2%) in the presence of wheat flour and egg. On the contrary, the LTC₄ release test was positive for the two food extracts, respectively, wheat flour and egg, and for the three concentrations tested (1/25, 1/125, and 1/625): 210, 168, and 45 pg/ml, and 316, 237, and 36 pg/ml, respectively (LTC₄ release is positive over 100 pg/ml) (3).

Cytokine determinations in serum showed normal results for IL-10 and interferon-gamma, a slight increase of IL-6 (18 pg/ml; normal values <9 pg/ml) and TNF- α (18 pg/ml; normal values <10 pg/ml). The ESR and CRP were normal. A slight increase of the platelet count was noted ($407 \times 10^9/l$). Stools were soft. Mycologic examination showed the presence of numerous colonies of *Geotrichum candidum*. There was a slight ascorbic acid deficiency (2.7 mg/l, normal values: 4–14 mg/l).

Due to her acceptable clinical status, no strict diet was recommended except in the case of an outbreak. A simple diet of well-cooked bread, pasta, and egg was recommended. Continuous treatment with cetirizine (10 mg/day) and disodium cromoglycate (600 mg/day) was started, as well as treatment with nystatine (10 days) and vitamin C (1 month).

This patient was seen again at the end of 9 months of treatment. From the first month, she was able to suspend mesalazine and local hydrocortisone treatments. According to her, she was completely cured: normal stool, no abdominal pain, and disappearance of asthenia. She felt herself "restored to life" for the first time in 20 years. She appreciably broadened her diet to include all the green vegetables.

There is no doubt that the intestinal mucosa can be involved in food allergy. However, food allergy-induced ulcerative colitis has been questioned (4). Specific IgEs to foods are more frequent in patients with inflammatory bowel disease (IBD) than in healthy subjects, but this is probably due to a greater absorption of antigens. Indeed, atopy is not significantly raised in IBD (5). However, other mechanisms have to be suspected, and there is a need for

blinded oral challenges, and for a strict evaluation of the results of well-conducted exclusion diets.

The use of the colic intramucosal provocation test (COLAP) shows immediate reactions and histologic modifications compatible with the diagnosis. In 40% of the cases of suspected digestive allergy, based on positive COLAP tests, specific IgE was undetectable. Of 70 patients, marked digestive manifestations (diarrhea and abdominal pain) were observed in 17 cases, and the first proposed diagnosis was variable: ulcerative colitis (3), Crohn's disease (5), eosinophil enteritis (4), colitis (2), and celiac syndrome (3). Patients with a positive COLAP test have been cured by exclusion of the offending food, together with antihistamines and disodium cromoglycate. Moreover, the specific treatment of ulcerative colitis was suspended (6).

In the case of our patient, the primary diagnosis of ulcerative colitis was established by endoscopy and biopsy, and we have no reason to doubt it. The healing obtained by simple restriction of diet, antihistamines, and disodium cromoglycate, where mesalazine and hydrocortisone have failed, suggests causation by food allergy. The clear positivity of the leukotriene release test may indicate a non-IgE-dependent hypersensitivity phenomenon (7) and could suggest in such cases a trial with leukotriene antagonists.

This observation leads finally to this question: are a small percentage of cases of ulcerative colitis caused by a food allergy? Or should we conclude that the digestive manifestations of food allergy, in the absence of adequate discriminating criteria, may resemble ulcerative colitis?

*Department of Internal Medicine
Clinical Immunology and Allergology
University Hospital
54035 Nancy Cedex
France

Accepted for publication 29 January
2001 Allergy 2001; 56:458–459
Copyright © Munksgaard 2001
ISSN 0105-4538

References

1. MAINO VC, SUNI MA, RUITENBERG JJ. Rapid flow cytometric method for measuring lymphocyte subset activation. *Cytometry* 1995;**20**:127–133.
2. SABBAH A, SAINTE-LAUDY J. Flow cytometry applied to the analysis of lymphocyte and basophil activation. *ACI Int* 1996;**8**:116–119.
3. MONERET-VAUTRIN DA, SAINTE LAUDY J, KANNY G, FREMONT S. Human basophil activation measured by CD63 expression and LTC4 release in IgE-mediated food allergy. *Ann Allergy Asthma Immunol* 1999;**82**:33–40.
4. BISCHOFF SC, MAYER JH, MANNS MP. Allergy and the gut. *Int Arch Allergy Immunol* 2000;**121**:270–283.
5. TRONCONE R, MERRET TG, FERGUSON A. Prevalence of atopy is unrelated to presence of inflammatory bowel disease. *Clin Allergy* 1988;**18**:111–117.
6. BISCHOFF SC. Colonoscopy allergen provocation (COLAP) – technique and results. *ACI Int* 1997;**9**:101–109.
7. GOETZL EJ, BURRALL BA, BAUD L, SCRIVEN KH, LEVINE JD, KOO CH. Generation and recognition of leukotriene mediators of hypersensitivity and inflammation. *Dig Dis Sci* 1988;**33**:36S–40S.