

persistence of specific IgE to cefaclor. We can exclude that the persistence of IgE to this drug was due to other drugs cross-reacting with cefaclor because during this period of time the child did not take any drugs. However, an uncontrolled or hidden contact, as with β -lactams, can occur in subjects with hypersensitivity to these drugs (7). Therefore, in our patient, an uncontrolled or hidden contact with cefaclor could have maintained the production of specific IgE to cefaclor. Other possible explanations for the persistence of IgE to cefaclor are

- 1) the ingestion of foods that might have allergenic peptides in common with cefaclor
 - 2) the presence of genetic factors which might be responsible for the continuous production of specific IgE to cefaclor without the presence of a specific stimulus
 - 3) the intrinsic peculiarity of cefaclor to induce a persistent IgE immune response.
- All these are only hypotheses because there are no studies on this matter.

In conclusion, this case shows that IgE to cefaclor may persist for many years after the last administration. It is thus important to advise the cefaclor-allergic patient strictly to avoid the administration of this drug in later years. Further studies are needed to evaluate the persistence of specific IgE to cefaclor in subjects with cefaclor allergy.

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MBP deposition in eosinophilic gastroenteritis

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Key words: eosinophil; eosinophilic gastroenteritis; major basic protein.

● A 6-YEAR-OLD boy, who had been suffering from bronchial asthma attacks several times a year, developed vomiting, loss of appetite, and generalized edema for 9 days before admission.

Laboratory studies revealed mild eosinophilia (9.0% of total white blood

cells), hypoproteinemia (3.6 g/dl), and hypoalbuminemia (1.6 g/dl). The serum IgE level was 580 IU/ml.

The serum specific IgE antibodies for milk, egg, house dust, and house-dust mite were positive.

Upper gastrointestinal endoscopy revealed broad geographic erosion in the gastric body and antrum (Fig. 1a). The patient was treated with an H_2 -blocker, and all clinical and laboratory parameters normalized within a month.

Microscopic examination of hematoxylin-eosin stained sections of biopsy specimens revealed heavy eosinophil infiltration in the gastric and duodenal mucosae. Immunostaining with monoclonal antibody against eosinophil granule major basic protein (MBP) (Monosan, Uden, the Netherlands) and eosinophil peroxidase (EPO) (Oncogene, Cambridge, UK) was performed. Heavy MBP-positive eosinophil infiltration and massive extracellular deposition of MBP were observed in the gastric and duodenal mucosae (Fig. 1b). No deposition of extracellular MBP was observed in the control specimens. No extracellular deposition of EPO was observed in either the patient or the control specimens.

A few studies of adult cases indicate that in eosinophilic gastroenteritis (EOG), eosinophils become activated and release cytotoxic proteins (1). This is the first report of EOG in a young child in which massive extracellular deposition of eosinophil granule MBP was demonstrated. MBP may have played an important role in damaging the gastrointestinal mucosa.

MBP is known to stimulate histamine release from mast cells (2). In this case, histamine release stimulated by deposited MBP may have increased the secretion of gastric acid, which could have increased the

A young boy with major basic protein deposits in the mucosae.

gastrointestinal mucosal damage. This could explain why the H₂-blocker was effective.

EOG is classified into the mixed IgE- and non-IgE-mediated disorders (3). Up to 50% of patients with EOG are atopic, but food allergen-induced reactions have been implicated in a minority of patients. Although we did not exclude the food allergens, our patient's disorder had a favorable course. It is unclear whether or not the gastrointestinal mucosal damage in our patient was related to allergic reactions to food allergens.

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Hypersensitivity to formaldehyde

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Key words: formaldehyde; inhalation; oral exposure; patch test.

● Mrs Q, born in 1949, had been working as a nurse in an intensive care unit since 1988. Four months later, she began complaining of sore throat when exposed to aldehydes. In May 1998, she was inadvertently exposed to high concentrations of Pulvispray[®] (formol plus glutaraldehyde), and complained for 2 weeks of rhinitis, dry cough, chest tightness, and tracheal burning. She attended the allergy outpatient clinic on 2 June 1998 while she was out of work. Examination revealed her throat to be erythematous, but her lungs were clear. Spirometric examination demonstrated normal FEV₁ (sRaw was also normal). Methacholine challenge test up to 1800 µg was completely

Patch-test-positive patient with oral symptoms.

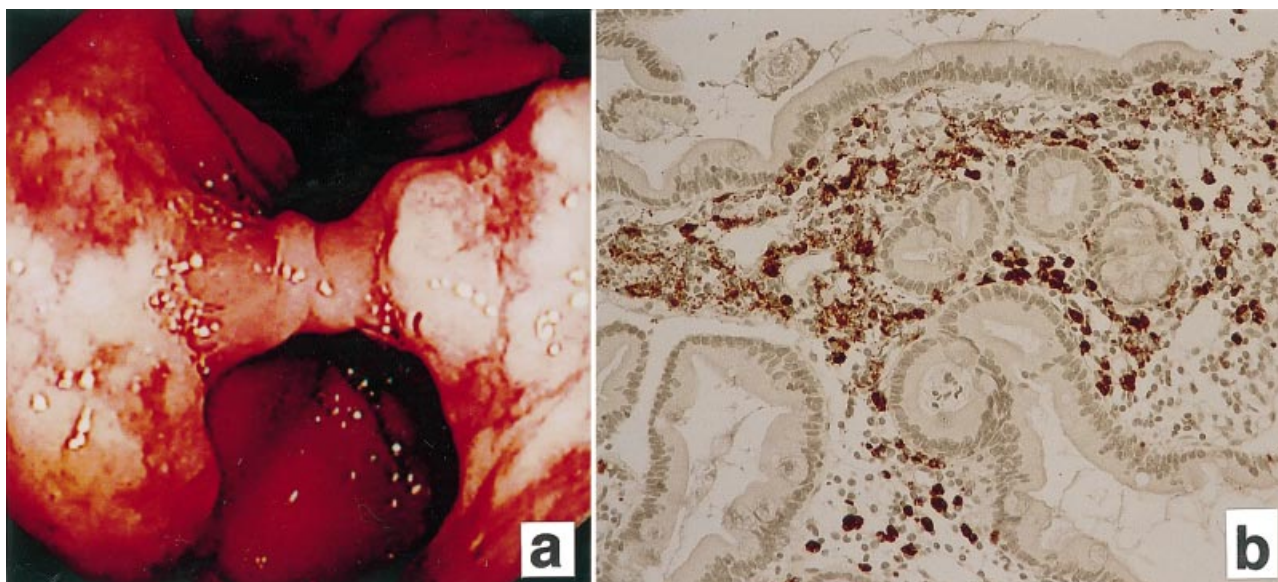


Figure 1. a) Upper gastrointestinal endoscopy revealed broad erosion in gastric body and antrum. b) In immunohistochemistry, heavy eosinophil infiltration and massive extracellular deposition of MBP were observed in gastric mucosa.