

CAP-RAST technique. Patients with food allergy to wheat still had specific IgE to wheat isolates, showing the absence of allergen denaturation during the industrial process of gliadin solubilization.

The absence of ELISA inhibition of wheat isolates by means of gliadin fractions could argue for a neoallergenicity. Deamidation of the gluten fraction by means of technologic processes (chemical or enzymatic) could have induced the exposure of cryptic allergenic epitopes or the formation of new allergenic epitopes. Because digestion has been shown to reveal masked epitopes of proteins and the patient is not allergic to wheat flour, the first hypothesis is not retained. Thus we can conclude that the treatments used for gluten deamidation generate new allergenic epitopes. Furthermore, recent works have shown that deamidation of gliadins or gliadin peptides by tissue transglutaminase has a major role in celiac disease.^{11,12} A case of contact urticaria has been recently attributed to hydrolyzed wheat in cosmetics combined with a generalized urticaria induced with the ingestion of sausages with lentils and a French cassoulet.¹³ This patient could also eat cereal-based products without any problem.

Wheat isolates are currently labeled as wheat proteins, achieving patient protection affected with celiac disease. This label might not be precise enough for patients tolerating the native wheat food but specifically allergic to the deamidated forms. Because wheat isolates are new food ingredients, the prevalence of their allergenicity is not known, but wheat isolates should be tested when a food allergy to finished foods is suspected.

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The marble test for gastric outlet obstruction in chronic granulomatous disease

To the Editor:

Chronic granulomatous disease (CGD) is a genetically heterogeneous disorder of the nicotinamide dinucleotide phosphate oxidase complex that results in impaired generation of reactive oxygen species and defective phagocyte antimicrobial activity against catalase-positive organisms. Most patients with CGD have chronic inflammatory complications, including potentially obstructive granuloma of the genitourinary or gastrointestinal tracts.¹ We describe a child with CGD in whom gastric outlet obstruction was suspected without radiographic evidence of delayed gastric emptying, because of an unusual finding.

A 20-month-old boy with CGD was evaluated in the immunology clinic for 2 weeks of nausea and nonbilious, nonbloody vomiting. He was afebrile, and his daily activity, appetite, stools, and sleep patterns were unaffected. Before this clinic visit, his regular medications, trimethoprim-sulfamethoxazole and IFN- γ , had been stopped for 4 days, but the vomiting had persisted. His primary pediatrician had prescribed ranitidine for anti-reflux prophylaxis.

The patient was diagnosed with CGD at 17 months of age, when he had a culture-negative cervical abscess that required surgical drainage, despite appropriate oral and intravenous antibiotics. The diagnosis was confirmed by nicotinamide dinucleotide phosphate oxidase immunoblotting and DNA analysis. After diagnosis, he remained healthy, other than several episodes of folliculitis that responded well to oral cephalixin.

Physical examination revealed a thriving toddler in no distress. The abdomen was nondistended, had good bowel sounds, and was nontender, with no palpable masses and no hepatosplenomegaly. No enlarged lymph nodes were palpable. The remainder of the physical examination was normal.

The differential diagnosis of this persistent vomiting included gastritis, gastric outlet obstruction, chronic reflux, and medication-induced vomiting from IFN- γ . An upper gastrointestinal series revealed normal gastric emptying, but a small marble that the child had reportedly swallowed several weeks earlier was visible in the stomach (Fig 1).

The child was started on 0.1 mg/kg metoclopramide 3 times daily in addition to the ranitidine. Two weeks later, he returned to the clinic with continued vomiting and had

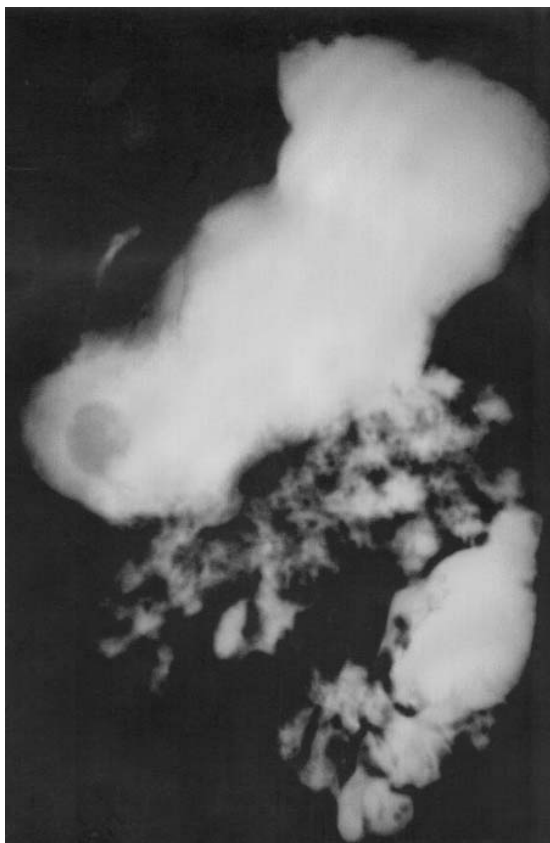


FIG 1. A 20-month-old child with CGD had persistent nausea and vomiting. His upper gastrointestinal series revealed a normal pattern of gastric emptying. However, the retention of a small marble provided evidence of functional gastric outlet obstruction.

not yet passed the marble in his stool. He continued to appear well, despite the persistent vomiting, and therefore he was started on a therapeutic trial of $2 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$ oral prednisolone. His vomiting resolved by the next day, and he passed the marble in his stool within 3 days.

The predisposition to chronic granulomatous inflammation seen in patients with CGD suggests that reactive oxygen species are required for the degradation of some inflammatory mediators.¹ As a result, gastrointestinal obstruction is a common problem in patients with CGD, especially before 3 years of age.² Symptoms may precede the diagnosis of CGD^{2,3} and may occur anywhere from the mouth to the anus.⁴ Nausea and vomiting are most commonly caused by gastric outlet obstruction, although Crohn's disease of the stomach, eosinophilic gastritis, eosinophilic granuloma, gastric abscesses, gastric ulcers, and infectious gastroenteritis may also produce these symptoms.^{1,3,4} In more severe cases of gastric outlet obstruction, abdominal pain, fever, and weight loss may also be seen. Granuloma formation, submucosal lymphoid infiltration, and mucosal edema in the gastric antrum can all contribute to gastric outlet obstruction.^{1,5}

Gastric outlet obstruction is usually diagnosed by ultrasonography or upper gastrointestinal series, both of which are subjective assessments of gastric emptying.

Upper gastrointestinal series may reveal delayed gastric emptying, antral narrowing, pyloric obstruction, and thickened or swollen mucosal folds.⁴ Although this child's upper gastrointestinal series was normal, retention of a small marble within his stomach suggested functional gastroduodenal obstruction, because a marble of less than 2 cm in diameter should pass easily out of the stomach of a 20-month-old child.⁶

Inflammatory obstruction in CGD usually responds well to systemic corticosteroids, and their use is often justified, despite the risk of increased susceptibility to infection.^{1,5,7} In this case, treatment with oral corticosteroids rapidly relieved the clinical symptoms and helped the marble to pass. In patients with fever or other concerning features on history or examination, additional diagnostic tests, such as endoscopy, should be strongly considered to confirm the presence of granulomatous inflammation before starting corticosteroids.

In summary, we describe a child with X-linked CGD who had probable gastric outlet obstruction that resolved promptly after he was treated with prednisolone. A unique feature of this case is the evidence of functional obstruction provided by failure to pass a small marble from the stomach, without evidence of gastric outlet obstruction or mucosal thickening on upper gastrointestinal series. Although we do not recommend marble ingestion as a diagnostic test, this case demonstrates the need for a high index of suspicion for gastric outlet obstruction in patients with CGD, despite normal radiographic studies. A closely monitored therapeutic trial of oral corticosteroids may be of benefit to some children with CGD and recurrent vomiting, even when there is no radio-graphic evidence of obstruction.

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Allergy to canned tuna

To the Editor:

Clinical allergy to fish is quite common.¹ Although these reactions can be severe and even life-threatening, it has been reported that patients with allergic reactions to fresh fish "appear to be able to safely eat canned tuna" because of a loss of allergenicity in processing.² This phenomenon is believed to be so consistent that canned tuna has been used as a placebo in double-blind, placebo-controlled food challenges to fish.¹ Reported here is a patient allergic to canned tuna.

A 19-year-old man reported that for several years, he had episodes of total body flushing and itching with facial and throat swelling. On some occasions, the throat swelling resulted in shortness of breath. He denied light-headedness or loss of consciousness. The symptoms resolved with treatment with diphenhydramine. The episodes occurred exclusively after and within 15 to 30 minutes of eating certain foods, namely beans, peas, chicken, turkey, and fish, including canned tuna.

Skin prick tests were performed with a commercial extract of cod (Hollister-Stier, Spokane, Wash) and with crude extracts made by pulverizing small amounts of canned tuna and fresh tuna in normal saline. The test results were positive with all extracts (Table I). Skin test results were also positive to the other foods reported by the patient to cause symptoms (data not shown). An extract of canned tuna was analyzed by means of gel electrophoresis followed by protein staining and immunoblotting with patient sera by using methods previously described.³ Sera from a nonatopic subject and another subject with fish allergy were used as control subjects. The immunoblot revealed IgE in the patient's serum that bound to high-molecular-weight protein bands. No such binding was seen with the 2 control sera (Fig 1).

Most patients with fish allergy show no reactivity on IgE immunoblotting to canned tuna and can safely consume canned tuna.² One previous study evaluating "tuna fish-positive sera" found that one of 8 samples assayed showed binding on IgE immunoblotting to a canned tuna extract,⁴ although no clinical information was available on the subject who provided that serum. Another report describes a patient who reacted to the ingestion of canned tuna but not to fresh tuna or other fish.⁵ His reactions were believed to be due to an undeclared additive in the canned tuna.

The patient described here seems clearly allergic to canned tuna. The history of his reaction to the ingestion of canned tuna is consistent in terms of nature and timing with an IgE-mediated event. His skin test results with canned tuna extract were positive, and his serum demonstrated clear IgE binding to proteins from a canned tuna extract not recognized by sera from a nonatopic subject and another subject with fish allergy. Thus although most patients with fish allergy can be advised that they can safely consume canned tuna, this cannot be a universal recommendation.

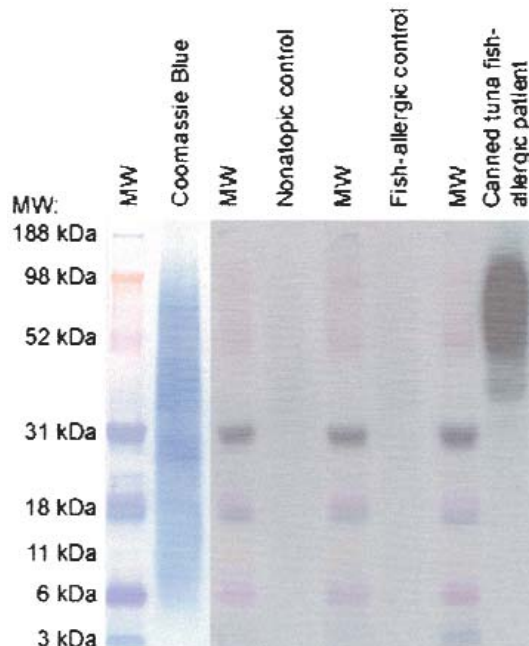


FIG 1. Canned tuna extract: protein Coomassie blue protein stain, immunoblotting for IgE antibody with control sera from a nonatopic subject and a subject with fish allergy and patient serum.

TABLE I. Skin prick test results (mm wheal/mm erythema)

(-) 50% glycerin	0
(+) Histamine	4/30
Commercial extract: cod	6/30
Canned tuna*	6/50
Fresh tuna*	5/23

*Extract prepared by pulverizing in normal saline.

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