'Persistent GERD' May Actually Be Food Allergy

Allergic eosinophilic esophagitis, first reported in 1995, can begin anytime from infancy to adolescence.

BY SHERRY BOSCHERT
San Francisco Bureau

STANFORD, CALIF. — Adults or children whose symptoms of gastroesophageal reflux disease continue despite treatment may have allergic eosinophilic esophagitis, John A. Kerner Jr., M.D., said at a conference on perinatal and pediatric nutrition.

An esophageal biopsy will show major eosinophilic infiltration of the mucosa and submucosa in a patient with allergic eosinophilic esophagitis. The proteins that are implicated in this disorder come from the "usual suspects" in the diet—cow's milk, wheat, soy, peanut, or egg, said Dr. Kerner, professor of pediatrics and director of nutrition at Stanford (Calif.) University Medical Center.

Allergic eosinophilic esophagitis can begin anytime from infancy to adolescence. "More and more of the adult literature is pointing out that patients have been missed with this disorder," Dr. Kerner said.

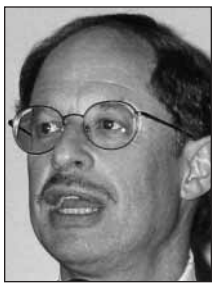
Treatment consists of avoiding the antigens, if they can be identified, switching infants to an elemental formula, and possibly using steroids. Often multiple antigens are involved, with poor correlation with skin tests for allergy, he added.

First identified in a landmark 1995 study of 10 children, allergic eosinophilic esophagitis produces symptoms that look like chronic gastroesophageal reflux disease (GERD). The child may refuse food, fail to thrive, vomit, have abdominal pain, be irritable, and have difficulty sleeping. Symptoms return despite treatment with histamine$_2$-receptor blockers or even fundoplication. Serum IgE levels are normal or slightly elevated, and peripheral eosinophils are uncommon in allergic eosinophilic esophagitis.

Older children and adults who have had allergic eosinophilic esophagitis for some time commonly turn up in emergency departments or clinics with esophageal stricture. Biopsies will show "sheets" of eosinophils in these patients. Seeing more than 20 eosinophils per high-power field in a biopsy is a "classic count" for diagnosing allergic eosinophilic esophagitis, although there is some debate about the exact number needed for diagnosis, Dr. Kerner said at the meeting, jointly sponsored by Symposia Medicus and Stanford University.

Endoscopy will show little circular rings that can be "fairly dramatic" and white plaques composed of eosinophilic complexes.

Restricting consumption of cow's milk will resolve symptoms in about 80% of cases. In infants with allergic eosinophilic esophagitis, 80% will improve after switching to a hydrolyzed protein formula such as Alimentum or Nutramigen. Those infants who do not respond usually do well when switched to an L-amino acid formula. Breast-fed infants with eczema and allergic eosinophilic esophagitis usually need an L-amino acid formula, Dr. Kerner said.



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DR. KERNER

An inhaled steroid will alleviate acute symptoms, but they recur when the inhaled treatment is stopped. Dr. Kerner said he prefers prescribing both inhaled and topical forms. Oral steroids for a systemic effect also are an option, he said.

The first published study of allergic eosinophilic esophagitis described 10 children diagnosed with GERD whose symptoms persisted despite separate treatments with five antireflux therapies, including Nissen fundoplication in six patients.

After 6 weeks on an L-amino acid–based formula (Neocate or Neocate One), eight patients had no symptoms, and symptoms improved in the other two patients. Esophageal biopsies before and after the 6 weeks of treatment showed that intraepithelial eosinophil counts decreased significantly, from a median of 41 per high-power field to less than 1 per high-power field (Gastroenterology 1995;109:1503-12).

Symptoms returned in all patients, however, after open food challenges. "This is a real disorder," Dr. Kerner said.

The study showed that chronic GI symptoms and histologic changes of the esophagus that were unresponsive to standard GERD treatments could be improved by using an elemental formula. "This was a breakthrough," he said. ■

H. pylori Eradication Reduces Dyspepsia Prescriptions, Costs

BY KATHLEEN LOUDEN
Contributing Writer

CHICAGO — A community screening and treatment program for *Helicobacter pylori* infection significantly reduced the use of dyspepsia-related health resources, Alexander Ford, M.B., reported at the annual Digestive Disease Week.

Antimicrobial eradication therapy for *H. pylori* yielded a 10-year mean savings in total dyspepsia-related costs of $117 per treated individual, said Dr. Ford, of Leeds (England) General Infirmary.

The savings, mainly due to reduced prescriptions for dyspepsia, were greater than the cost of the screening, according to Dr. Ford. The reductions in total dyspepsia-related health care costs and dyspepsia-related prescribing costs were both statistically significant.

The reductions in total dyspepsia-related health care costs and dyspepsia-related prescribing costs due to *H. pylori* eradication were both statistically significant.

"This could be the first screening study to pay for itself," he commented.

Study subjects were *H. pylori*–positive individuals who had participated in a community screening program (conducted by the Leeds HELP study group, Lancet 2000;355:1665-9) and who returned a recent dyspepsia questionnaire and consented to have their medical records examined. Of these 914 participants, 474 had originally been randomized to eradication therapy consisting of omeprazole, clarithromycin, and tinidazole for 7 days; 440 had been randomized to receive placebo.

The findings lend support to eradication therapy for *H. pylori*–positive individuals who have nonulcer dyspepsia, an approach for which Dr. Ford said there has been conflicting evidence.

In the United Kingdom, physicians give *H. pylori* eradication therapy in dyspeptic patients, he said in an interview. However, in the United States, the National Institutes of Health does not recommend eradication therapy for persons who have nonulcer dyspepsia or are asymptomatic, according to a 1994 consensus statement.

In the current study, eradication therapy was associated with a reduction in dyspepsia symptoms that was not statistically significant. Of the subjects who had dyspepsia symptoms at baseline, 59% of the treated group remained symptomatic at 10 years vs. 66% who received placebo, Dr. Ford said in an interview.

There was a possible selection bias in locating original participants of the screening program.

"We were more likely to contact people of higher socioeconomic status, and they were more likely to respond," Dr. Ford said. ■

β-Blockers Cut Risk of First Bleed From Esophageal Varices by 50%

BY MICHELE G. SULLIVAN
Mid-Atlantic Bureau

CAMBRIDGE, MD. — β-Blockers remain the best choice for primary prevention of bleeding from esophageal varices in patients with end-stage liver disease.

Variceal banding, while at least as effective as β-blockers in preventing a first bleed, should be reserved for those who don't respond to or can't tolerate β-blockers, or who are noncompliant with drug therapy, Sergey Kantsevoy, M.D., said at a hepatobiliary update sponsored by Johns Hopkins University.

Esophageal varices develop in up to 60% of patients with cirrhosis. If varices rupture, they carry a significant mortality risk of 20%-40%, depending on the severity of the liver disease. Therefore, all patients with end-stage liver disease should undergo upper endoscopy to screen for varices, said Dr. Kantsevoy of Johns Hopkins University, Baltimore.

Unselected patients don't benefit from primary prevention strategies for esophageal varices, but there is great benefit for high-risk patients, he said. However, despite the mortality risk of bleeds and the proven benefit of treatment, only 46% of those referred for liver transplantation had been screened for esophageal varices (Am. J. Gastroenterol. 2001;96:833-7).

If the initial endoscopy does not identify varices, the patient should have a repeat endoscopy every 2 years. If the varices are small, a repeat endoscopy every 1-2 years is indicated, depending on the severity of liver disease.

Patients with large varices should be offered prophylactic therapy. β-Blockers are the medical therapy of choice. They reduce portal pressure by reducing cardiac output and increasing resistance in collateral veins. The drugs have been shown to reduce the risk of a first variceal bleed by half and to reduce mortality by up to 45%, compared with placebo.

Unfortunately, Dr. Kantsevoy said, β-blockers are contraindicated in up to 20% of end-stage liver disease patients. In addition, "despite adequate β-blockage, at least 30% will not achieve reduction in portal pressure sufficient to prevent bleeding, and about 30% will have side effects including heart failure, hypotension, bronchoconstriction, fatigue, and impotence."

Endoscopic variceal banding may be considered for these patients. Band ligation has been shown to be as effective as β-blockage at reducing the incidence of bleeding, but the procedure carries no significant mortality advantage over medical therapy.

Endoscopic sclerotherapy has been investigated in these patients, but it is not recommended for primary prevention because it is associated with a high rate of adverse events.

Postsclerotherapy complications occur in up to 20% of patients and include ulceration, stricture formation, and esophageal perforation, Dr. Kantsevoy said. ■

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