



## Complete Summary

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### GUIDELINE TITLE

Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment.

### BIBLIOGRAPHIC SOURCE(S)

Furuta GT, Liacouras CA, Collins MH, Gupta SK, Justinich C, Putnam PE, Bonis P, Hassall E, Straumann A, Rothenberg ME, First International Gastrointestinal Eosinophil Research Symposium (FIGERS). Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment. *Gastroenterology* 2007 Oct;133(4):1342-63. [121 references] [PubMed](#)

### GUIDELINE STATUS

This is the current release of the guideline.

According to the guideline developer, the Clinical Practice Committee meets three times a year to review all American Gastroenterological Association Institute (AGA Institute) guidelines. This review includes new literature searches of electronic databases followed by expert committee review of new evidence that has emerged since the original publication date.

## COMPLETE SUMMARY CONTENT

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## SCOPE

### DISEASE/CONDITION(S)

Eosinophilic esophagitis

### GUIDELINE CATEGORY

Diagnosis  
Evaluation  
Management  
Treatment

## **CLINICAL SPECIALTY**

Allergy and Immunology  
Family Practice  
Gastroenterology  
Internal Medicine  
Nutrition  
Pathology  
Pediatrics

## **INTENDED USERS**

Dietitians  
Physicians

## **GUIDELINE OBJECTIVE(S)**

- To document the current state of knowledge in eosinophilic esophagitis
- To determine diagnostic criteria and make recommendations for evaluation and treatment of children and adults with suspected eosinophilic esophagitis
- To determine how to advance the field by expanding knowledge and defining priorities and strategies for future research

## **TARGET POPULATION**

Adults and children with eosinophilic esophagitis

## **INTERVENTIONS AND PRACTICES CONSIDERED**

### **Diagnosis/Evaluation/Monitoring**

1. Endoscopy
2. Biopsy
3. Intraesophageal pH testing
4. Esophageal manometry (not recommended)
5. Endoscopic ultrasound
6. Radiography (upper gastrointestinal contrast study)
7. Histopathology
  - Quantitation of eosinophils
  - Observation of eosinophil morphology
  - Evaluation of other inflammatory cell types (lymphocytes, polymorphonuclear leukocytes, mast cells)
8. Monitoring via regular clinic visits

### **Management**

## **Allergic Evaluation**

1. History, physical examination, and testing for other atopic diatheses
2. Evaluation of peripheral blood eosinophil count and granule proteins
3. Evaluation of total circulating immunoglobulin E (IgE), aeroallergen-specific IgE, and food-specific IgE
4. Evaluation of peripheral cytokines and gene expression
5. Skin prick testing
6. Atopy patch testing (not yet recommended)

## **Treatment**

1. Acid suppression using proton pump inhibitors
2. Esophageal dilatation
3. Corticosteroids (systemic and topical)
4. Cromolyn sodium (not recommended)
5. Leukotriene receptor antagonists (not recommended)
6. Dietary treatment
7. Biologics (anti-interleukin 5) (not recommended)

## **MAJOR OUTCOMES CONSIDERED**

- Morbidity and comorbidity
- Relapse rate
- Quality of life
- Incidence of treatment side effects
- Sensitivity, specificity, and positive and negative predictive values of laboratory tests
- Correlation of laboratory signs with symptoms

## **METHODOLOGY**

### **METHODS USED TO COLLECT/SELECT EVIDENCE**

Searches of Electronic Databases

### **DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE**

A systematic review of the English language medical literature through September 2006 was performed using electronic databases (MEDLINE, PubMed, and Ovid), with the key words "eosinophilic esophagitis," "allergic esophagitis," and "eosinophilic oesophagitis." Review articles, letters to the editor, most case reports of <3 patients, and abstracts were excluded.

A total of 80 studies met the inclusion criteria and served as the basis of this report. They include a total of 754 children (age range, 4 months to 20 years) and 323 adults (age range, 22 to 89 years). The sample sizes varied from 7 to 381 patients (mean, 37 years). The studies were published between 1977 and September 2006. Most were conducted in academic centers in the United States, Canada, Europe, and Australia.

## **NUMBER OF SOURCE DOCUMENTS**

A total of 80 studies met the inclusion criteria and served as the basis of this report.

## **METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE**

Weighting According to a Rating Scheme (Scheme Given)

## **RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE**

The quality of evidence supporting the recommendations contained in this review was assessed as follows:

**Grade A:** Homogeneous evidence from multiple, well-designed, randomized (therapeutic) or cohort (descriptive) controlled trials, each involving a number of participants to be of sufficient statistical power

**Grade B:** Evidence from at least 1, large, well-designed clinical trial with or without randomization from cohort or case-control analytic studies or well-designed meta-analysis

**Grade C:** Evidence based on clinical experience, descriptive studies, or reports of expert committees

## **METHODS USED TO ANALYZE THE EVIDENCE**

Review of Published Meta-Analyses  
Systematic Review

## **DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE**

Relevant data were discussed among committee members in conference calls. Critical evaluations included study design, numbers of patients, definitions used, outcomes reported, and potential biases. The chair of each committee synthesized the data, and inconsistencies were resolved by discussion until consensus was achieved.

## **METHODS USED TO FORMULATE THE RECOMMENDATIONS**

Expert Consensus

## **DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS**

A task force of 31 physicians who participated in the First International Gastrointestinal Eosinophil Research Symposium (FIGERS) performed the review. The reviewers were divided into subcommittees along the lines of their recognized expertise in clinical evaluation, endoscopy, histopathology, allergy, and treatment.

Relevant data were discussed among committee members in conference calls. Critical evaluations included study design, numbers of patients, definitions used, outcomes reported, and potential biases. The chair of each committee synthesized the data, and inconsistencies were resolved by discussion until consensus was achieved.

## **RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS**

Not applicable

## **COST ANALYSIS**

A formal cost analysis was not performed and published cost analyses were not reviewed.

## **METHOD OF GUIDELINE VALIDATION**

Peer Review

## **DESCRIPTION OF METHOD OF GUIDELINE VALIDATION**

Not stated

# **RECOMMENDATIONS**

## **MAJOR RECOMMENDATIONS**

The level of evidence supporting the recommendations (A–C) is defined at the end of the "Major Recommendations" field.

### **Clinical Manifestations**

Eosinophilic esophagitis (EE) should be considered in young children with gastroesophageal reflux disease (GERD)-like symptoms, including feeding problems, and in older children and adults with GERD-like symptoms, especially in those with dysphagia or esophageal food impaction. When the primary diagnosis is EE, symptoms are unresponsive or only partially responsive to acid blockade (**Grade B**).

### **Natural History**

EE tends to be a chronic disease with persistent or relapsing symptoms. To date, esophageal strictures and small caliber esophagus, often resulting in food impaction, have been the major complications identified. When these findings are encountered, either radiologically or at endoscopy, a high index of suspicion should be raised for EE, and mucosal biopsy specimens should be obtained (**Grade B**). Although esophageal metaplasia (Barrett's esophagus or cardia-type metaplasia) has not been described as an associated finding in patients with EE, careful long-term follow-up is advised. Other chronic problems include failure to thrive and feeding intolerance in children. At present, it is unclear whether

persistent esophageal eosinophilia is always accompanied by symptoms. See "Monitoring" section below.

### **Diagnostic Testing**

#### **Biopsy Procurement and Evaluation**

The rationale for histological assessment of EE is provided in the original guideline document. Endoscopic appearances should be documented and photographed. Mucosal pinch biopsy specimens should be obtained from all patients in whom EE is in the differential diagnosis. Biopsy specimens should be obtained *regardless of the gross appearance of the mucosa*, and multiple biopsy specimens should be obtained from different esophageal locations along the length of the esophagus. Biopsy specimens should also be obtained from stomach and duodenum to rule out other diseases such as eosinophilic gastroenteritis and, when appropriate, inflammatory bowel disease. Optimal fixation is accomplished by using fixative such as formalin or paraformaldehyde (**Grade C**). The cost-effectiveness of these recommendations has not been evaluated but deserves further study.

#### **Endoscopic Ultrasound**

When the diagnosis of GERD vs EE is not apparent despite endoscopy and biopsy, intraesophageal pH monitoring may be of use in excluding pathologic reflux as either the primary or a concomitant cause for esophageal eosinophilia (**Grade B**). Alternatively, an upper endoscopy after 6 to 8 weeks of high-dose proton pump inhibitor (PPI) treatment can help determine the etiology of esophageal eosinophilia (see Treatment section). Esophageal manometry does not provide diagnostic value in patients with EE.

#### **Radiography**

In patients with dysphagia, an upper gastrointestinal (GI) contrast study may identify the presence of a stricture, as well as its caliber and length. A contrast study may be beneficial for children who present with vomiting to rule out anatomic etiologies such as malrotation (**Grade C**). This information is also potentially helpful for a subsequent upper endoscopy because it may alert the endoscopist to use a smaller caliber endoscope or to proceed particularly cautiously with passage of the instrument so as to lessen the likelihood of a mucosal tear. In addition, it prepares the endoscopist for the possible need for a dilatation. An upper GI contrast study is generally not useful in patients presenting with symptoms typical of GERD, e.g., heartburn.

#### **Histopathology**

EE is a clinicopathologic disease defined by esophageal symptoms associated with a severe isolated esophageal eosinophilia and absence of pathologic GERD as evidenced by normal pH monitoring of the distal esophagus or lack of response to high-dose PPI treatment. Intraepithelial eosinophils should be counted in the most intensely inflamed high-power field (HPF) of the biopsy (x400) to generate a peak count. Setting a fixed number of eosinophils as the sole cut-off criterion to distinguish EE from GERD is contentious, possibly misleading, and probably

unwise based on current knowledge. On the basis of this literature review and collective clinical experience, the guideline authors conclude that a peak count of at least 15 intraepithelial eos/HPF is an absolute minimum number to make the diagnosis of EE *in the proper clinical context* (**Grade B**). If all HPFs are counted, the mean eosinophil number may be less than 15 because of focal inflammation in the biopsy specimens, but at least 1 HPF must contain at least 15 intraepithelial eosinophils. For research purposes, defining EE with a higher threshold of peak eosinophils may be advisable to increase the specificity of the diagnosis.

Additional features that are not pathognomonic but may be helpful to the pathologist in recognizing EE include eosinophil microabscesses (correlate of endoscopic mucosal with specks and plaques), surface layering of eosinophils, basal layer hyperplasia, papillary lengthening, degranulating eosinophils, and lamina propria fibrosis and inflammation. These features should be assessed in all biopsy specimens and included in pathology reports in addition to the number of eosinophils. The diagnostic criteria for adults are the same as for children. Gastroenterologists treating adults and children with symptoms of esophageal dysfunction and numerous intraepithelial eosinophils in esophageal biopsy specimens should ensure that the disease cannot be attributed solely to GERD before making a diagnosis of EE.

### **Allergic Evaluation**

#### **History, Physical Examination, and Testing for Other Atopic Diatheses**

Because of the high rate of allergic rhinitis, asthma, and/or eczema in EE patients, a complete evaluation by a well-informed allergist for other atopic diatheses is recommended (**Grade C**).

#### **Assessment of Atopy by Analysis of Blood Samples**

##### *Peripheral Eosinophil Count and Eosinophil Granule Proteins*

Evaluation of peripheral blood eosinophils may provide supportive evidence for the presence of EE and the degree of tissue involvement but are not diagnostic, and correlation with disease activity is unknown (**Grade C**). In future studies, if eosinophil levels are to be followed, it is important that (1) blood eosinophil levels be drawn at diagnosis and again at each evaluation for response to treatment (dietary or medical) and (2) notation is made regarding the control of concurrent atopic diatheses and the extent of aeroallergen exposure at each time when eosinophil count is evaluated. Absolute eosinophil counts and defining criteria for "blood eosinophilia" should be reported in publications that document peripheral eosinophilia. Further studies are needed to evaluate whether eosinophils constitute an adequate surrogate disease marker either alone or in combination with other surrogate disease markers such as eosinophil-derived neurotoxin (EDN).

##### *Total Immunoglobulin E (IgE)*

No published studies document whether or not total IgE can serve as a surrogate marker for disease progression or resolution. If total IgE levels are to be followed,

it is imperative that (1) an evaluation is done regarding whether or not the patient has adequate aeroallergen avoidance and the pollen season at each time when the total IgE level is evaluated and (2) an evaluation is done regarding whether or not concurrent atopic diatheses are adequately controlled at the time that the total IgE is evaluated. If IgE levels are followed, it is recommended that levels be checked at diagnosis and at each endoscopic evaluation of disease response to intervention (**Grade C**). It is important that total IgE levels be interpreted within the context of age-defined normal values and that the total IgE level that is considered "normal" be clearly stated in any publication.

#### *Aeroallergen-Specific IgE*

Given the high rate of other allergic diatheses (50% to 80%) in EE patients and the potential of aeroallergens to have a role in the instigation of EE, it may be important to evaluate EE patients for aeroallergen sensitivity (**Grade C**). Allergy testing may predict the response to pharmacotherapy or dietary avoidance in EE patients and thus warrants evaluation.

#### *Food-Specific IgE*

There are no positive or negative predictive values for food-specific IgE level testing in EE. In vitro food allergy testing is not supported in the evaluation of EE patients at this time, and empiric food testing should utilize skin prick tests (see below; **Grade B**).

#### *Peripheral Cytokines*

Eotaxin-3 expression and its genetic variation are promising markers of distinguishing EE from other causes of esophagitis (**Grade B**). Future research concerning the reversibility of eotaxin-3 levels with therapy and their prognostic significance deserve further investigation.

#### *Gene Expression*

Although the results of eotaxin-3 expression in EE vs non-EE patients are highly promising, assessment of eotaxin-3 remains a research tool, and correlations with disease severity and activity remain to be evaluated (**Grade B**). The identified EE transcriptome may indeed have promising value for disease diagnosis, assessment of therapeutic responsiveness, and prognosis.

#### *Skin Prick Testing for Antigen Sensitization*

Skin prick testing for foods and environmental allergens should be considered so that potential allergens and the atopic status of EE patients are identified (**Grade C**).

#### **Application of Atopy Patch Testing (APT) in EE**

The combination of prick skin tests and APT has been successful in one center and is being examined at other centers to verify these results. In addition, APT has shown promise in atopic dermatitis with good predictive values, high specificity,

and low sensitivity, and APT has shown highly promising results with regard to food elimination diet and food reintroduction in patients with EE. However, its use should be reserved until additional data from multiple research teams emerge that clearly establish its value for diagnosing and/or managing EE (**Grade B**). In addition, further data regarding the types of cells and immune response that is occurring at the site of patch testing are needed (e.g., skin biopsy studies).

## **Treatment of EE**

### **Acid Suppression**

Acid suppression is useful as a part of fulfilling the diagnostic criteria for EE. In addition, it may be used in lieu of esophageal pH monitoring for patients with established EE who have symptoms secondary to concomitant GERD. PPI therapy should not be considered as a primary treatment for patients with EE. Rather, it may be considered as co-therapy because it sometimes alleviates symptoms in part (**Grade C**). It is interesting to speculate that the esophagus of EE patients may have enhanced sensitivity to acid, even in the absence of pathologic reflux.

### **Esophageal Dilatation**

Esophageal dilatation is useful for symptomatic patients who present with symptomatic esophageal narrowing secondary to fixed strictures causing food impaction (**Grade C**). However, because of the risk of mucosal tearing and perforation, whenever possible, a diagnostic endoscopy with biopsy followed by medical or dietary therapy for EE should be attempted prior to performing esophageal dilatation. Inspection of the esophageal mucosa (radiographic or very gentle endoscopic examination) should be considered following esophageal dilation to assess for laceration injury prior to the performance of sequential, larger caliber dilation.

### **Corticosteroids**

Systemic and topical corticosteroids effectively resolve acute clinicopathologic features of EE; however, when discontinued, the disease generally recurs. Systemic corticosteroids may be utilized in emergent cases such as dysphagia requiring hospitalization, dehydration because of swallowing difficulties, and weight loss. However, because of the potential for significant toxicity, their long-term use is not recommended (**Grade B**). For many patients, topical corticosteroids are also effective in inducing EE remission. Although the incidence of adverse effects with this form of administration has not been formally studied, several studies have documented its safety, except for local fungal infections.

The use of topical corticosteroids for maintenance treatment has not been studied. Age adjusted doses and administration frequency of topical corticosteroids, i.e., fluticasone, budesonide, for children and adults with EE have not been established and these formulations were not designed for esophageal administration. One study extrapolated doses from those utilized in the treatment of asthma. Since then, others have utilized higher doses without significant complications. On the basis of expert opinion and the current literature, suggested starting doses range from 440 to 880 micrograms per day for children and 880 to 1760 per day for adolescents/adults. Drug has been administered by mouth and can be split into

twice or 4 times daily doses. Equally important is the method of administration; patients should be instructed to administer the metered dose inhaler (MDI) without the use of a spacer. The MDI should be inserted into the mouth, sprayed with lips sealed around the device. The powder should then be swallowed and not rinsed. Patients should not eat or drink for at least 30 minutes. This regimen is continued for 6 to 8 weeks and then patients followed as described in Monitoring section (**Grade B**). More studies are needed to clarify specifics of topical steroid treatment plans. Also see Update section in the original guideline document for information on alternative method of administration.

### **Leukotriene Receptor Antagonists and Mast Cell Stabilizers**

Although cromolyn sodium has no significant adverse effects, it has no apparent therapeutic effect for patients with EE. Leukotriene receptor antagonists have been shown to induce symptomatic relief at high dosages; however, its use has not been shown to have any effect on esophageal eosinophilia. Measurements of mucosal leukotriene levels do not suggest potential for a therapeutic benefit. The use of these drugs for the treatment of EE is not supported by the current literature (**Grade C**).

### **Dietary Treatment**

Dietary therapy (the specific antigens removal or elemental formula) should be considered as an effective therapy in all children diagnosed with EE (**Grade B**). When deciding on the use of a specific dietary therapy, the patient's lifestyle and family resources also need to be considered. Consultation with a registered dietitian is strongly encouraged to ensure that proper calories, vitamins, and micronutrients are maintained. The use of dietary therapy in adults requires further study.

### **Biologics**

Novel biologic agents present a unique opportunity for certain patients with EE. These molecules await clinical trials and cannot be recommended for routine use at the present time (**Grade C**).

### **Monitoring**

In children and adults with EE, the guideline authors suggest regular clinic visits during which the patient and parents should be questioned about symptoms, compliance with therapy, and adverse effects (**Grade C**). This suggestion is based on improving the recognition of long-term complications associated with chronic esophageal eosinophilia; presently, the incidence of complications is unknown.

In children, options for endoscopic and radiographic monitoring should be discussed considering the issues above. One approach might be to perform repeated upper endoscopies until settling on a treatment regimen that has controlled symptoms and ideally resolved esophageal eosinophilia. Repeat examinations can be based on change in symptoms or compliance with therapy. If repeat endoscopy with biopsy is planned, it should be performed no sooner than 4 weeks after the last therapeutic intervention. These suggestions are based on

improving the recognition of long-term complications associated with chronic esophageal eosinophilia; currently, data are not available to determine the optimal method to follow patients.

In asymptomatic patients with persistent esophageal eosinophilia, a repeat upper endoscopy can be performed following institution of additional treatment. For those in whom additional treatment is deferred, a repeat upper endoscopy and/or barium swallow can be obtained every 2 to 3 years to evaluate for progressive disease, but the risks of this approach outside of a clinical research protocol need to be weighed against the unknown benefits; this is especially important because the accuracy of histologic and radiologic predictors of disease progression is unclear.

The approach to adults with EE should consider similar principles as described above for children. However, clinical experience suggests that adults may be inclined to guide treatment based mainly on symptoms. Thus, the need for surveillance should also consider willingness to accept more aggressive treatment based on the results.

### **Definitions:**

#### **Levels of Evidence**

**Grade A:** Homogeneous evidence from multiple, well-designed, randomized (therapeutic) or cohort (descriptive) controlled trials, each involving a number of participants to be of sufficient statistical power

**Grade B:** Evidence from at least 1, large, well-designed clinical trial with or without randomization from cohort or case-control analytic studies or well-designed meta-analysis

**Grade C:** Evidence based on clinical experience, descriptive studies, or reports of expert committees

#### **CLINICAL ALGORITHM(S)**

None provided

## **EVIDENCE SUPPORTING THE RECOMMENDATIONS**

### **TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS**

The type of supporting evidence is graded and identified for each recommendation (see "Major Recommendations").

The quality of evidence fell primarily into the grade C category (evidence based on clinical experience, descriptive studies, or reports of expert committees).

## BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

### POTENTIAL BENEFITS

Accurate diagnosis and appropriate treatment of eosinophilic esophagitis

### POTENTIAL HARMS

- Esophageal dilation carries the risk of mucosal tearing and perforation.
- Systemic corticosteroids are associated with significant adverse effects.
- Drug treatments are less restrictive [than dietary treatments], placing no compromises on the patient's diet, but carry potential adverse effects and unknown duration of treatment.
- Dietary treatments give the prospect of prolonged remission but entail significant lifestyle modification.
- Corticosteroids, even if given topically, have been associated with esophageal candidiasis, and the long-term safety of strategies involving such treatment is unknown.

## IMPLEMENTATION OF THE GUIDELINE

### DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

## INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

### IOM CARE NEED

Getting Better  
Living with Illness

### IOM DOMAIN

Effectiveness  
Patient-centeredness

## IDENTIFYING INFORMATION AND AVAILABILITY

### BIBLIOGRAPHIC SOURCE(S)

Furuta GT, Liacouras CA, Collins MH, Gupta SK, Justinich C, Putnam PE, Bonis P, Hassall E, Straumann A, Rothenberg ME, First International Gastrointestinal Eosinophil Research Symposium (FIGERS). Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment. *Gastroenterology* 2007 Oct;133(4):1342-63. [121 references] [PubMed](#)

### ADAPTATION

Not applicable: The guideline was not adapted from another source.

**DATE RELEASED**

2007 Aug

**GUIDELINE DEVELOPER(S)**

American Gastroenterological Association Institute - Medical Specialty Society  
North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition  
- Professional Association

**SOURCE(S) OF FUNDING**

American Gastroenterological Association Institute

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## **FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST**

Conflict of interest disclosures: Glenn T. Furuta, consultant, Ception Therapeutics; speaker's bureau, TAP; Chris A. Liacouras, consultant, SHS, Nutricia, Ception, Ross; grant/research support, Wyeth; speaker's bureau, TAP, Merck; Margaret H. Collins, consultant, GlaxoSmithKline, Ception Therapeutics; Sandeep K. Gupta, consultant, GlaxoSmithKline, TAP, AstraZeneca, Salix; speaker's bureau, Ross Products, TAP, AstraZeneca; educational grant, Ross Products; Christopher Justinich, no disclosures; Phil E. Putnam, no disclosures; Peter A Bonis, no disclosures; Eric Hassall, consultant, TAP Pharmaceuticals, Abbott Canada, Altana Pharma; clinical research grant, AstraZeneca; Alex Straumann, no disclosures; Marc E. Rothenberg, consultant, Merck, Ception Therapeutics, GlaxoSmithKline, MedaCorp; speaker's bureau, Merck; Samuel Nurko, grant/research support, Wyeth pharmaceuticals, TAP, Sucampo; Nirmala Gonsalves, consultant, Medacorp, Ception Therapeutics; Jonathan Markowitz, consultant, Ception Therapeutics; Don Antonioli, no disclosures; Eduardo Ruchelli, no disclosures; Hector Melin-Aldana, no disclosures; Margret Magid, no disclosures; Ikuo Hirano, no disclosures; David Katzka, no disclosures; Susan R. Orenstein, consultant, Ception Therapeutics, TAP, Braintree, AstraZeneca, Wyeth, Bristol Myers Squibb, McNeil; grant/research support, Braintree; Jonathan M. Spergel, consultant, Novartis, GlaxoSmithKline; grant/research support, Novartis, Nutricia; speaker's bureau, AstraZeneca, GlaxoSmith-Kline; Amal Assa'ad, no disclosures; Seema Aceves, no disclosures; Barry K. Wershil, consultant, AP Pharmaceuticals, AstraZeneca; speaker's bureau, Shire; educational grant, TAP Pharmaceuticals; Thomas Platts-Mills, no disclosures; Tusar Desai, no disclosures; Seema Khan, no disclosures; B Li, no disclosures; Amir F. Kagalwalla, no disclosures

## **GUIDELINE STATUS**

This is the current release of the guideline.

According to the guideline developer, the Clinical Practice Committee meets three times a year to review all American Gastroenterological Association Institute (AGA Institute) guidelines. This review includes new literature searches of electronic databases followed by expert committee review of new evidence that has emerged since the original publication date.

## **GUIDELINE AVAILABILITY**

Electronic copies: Available from the American Gastroenterological Association Institute (AGA Institute) [Gastroenterology journal Web site](#).

Print copies: Available from the American Gastroenterological Association Institute, 4930 Del Ray Avenue, Bethesda, MD 20814.

#### **AVAILABILITY OF COMPANION DOCUMENTS**

None available

#### **PATIENT RESOURCES**

None available

#### **NGC STATUS**

This summary was completed by ECRI Institute on December 14, 2007.

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