



HOLY C.O.W.!

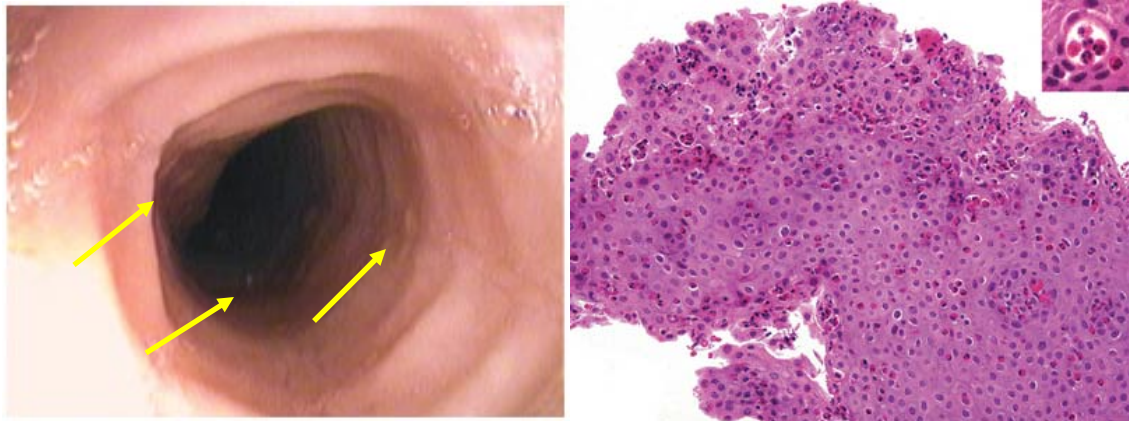
IT'S...

Clinical Question of the Week #22
November 24th, 2008 through December
1st, 2008



Welcome to the Thanksgiving edition of Holy C.O.W.! Please e-mail your answers to Kuo, Tim, Wendy, and Kevin (klian@mednet.ucla.edu; tprovias@mednet.ucla.edu; wsimon@mednet.ucla.edu; kbreger@mednet.ucla.edu) by 0800 on Monday, December 1st, 2008. The resident or intern with the most correct answers at the end of each month will receive a prize!

Case: A 62-year-old man presents to the Emergency Department for evaluation of acute retrosternal fullness and pain for the past hour, associated with some increased secretions. The patient is currently visiting his nephew, an intrepid IM resident at UCLA, and was enjoying an early holiday dinner of Turducken™, green beans, yams, and mashed potatoes. His past medical history is notable for well-controlled hypertension, GERD, and allergic rhinitis. He is allergic to PCN (rash) and peanuts; medications include HCTZ, pepcid, and occasional benadryl. In the ED, the patient was taken urgently to upper endoscopy, which revealed acute food bolus impaction. After removal of the bolus, biopsy of the esophagus was also performed. Post-procedure, the patient felt much improved and was discharged back with his family for the holiday.



The patient's upper endoscopy (food bolus removed); biopsy from esophagus with H&E stain.

Questions:

1. What is the diagnosis?

Eosinophilic esophagitis (also known as allergic esophagitis), is characterized by eosinophilic infiltration of the esophagus presumed due to allergic or idiopathic causes and affects both children and adults. The incidence of the disease is becoming more frequent over the past few decades with approximately 100 cases per 100,000, compared

with an incidence of 1:100,000 between the years of 1976-1985 in one study. The disease is seen more frequently in men/boys and among those who have had a long history of GERD. There is also an association with the condition eosinophilic gastroenteritis, consisting of malabsorption, dysmotility, and ascites.

Pathogenesis is not completely known, however an association is common suggesting a relationship between environmental allergens and esophageal eosinophil recruitment. This process is regulated by exotoxin and IL-5 which mediate eosinophil recruitment. In addition to food allergens and eosinophilic esophagitis, eosinophil recruitment is also implicated in GERD and IBD.

Diagnosis is based on clinical features dysphagia and GERD with a history of atopy. Serum IgE may be mildly elevated. Barium swallow and esophageal pH study may yield information on anatomy and reflux, respectively. Finally, esophageal biopsy yields large numbers of eosinophils. (0.5)

2. Describe the gross and pathologic findings depicted in the images above.

A variety of morphologic changes in the esophagus are noted, including strictures (most common), mucosal rings (often multiple, shown above), linear furrowing/corrugation, ulceration, polyps, and multiple whitish papules or granular exudates representing eosinophilic microabscesses (which may be confused with Candida, also shown above with arrows).

Pathologically, a large number of eosinophils (≥ 15 per HPF) despite acid suppression for two months, associated with normal gastric and duodenal biopsies. Supportive histologic features include eosinophilic microabscesses, surface layering of eosinophils, eosinophilic sheeting, basal layer hyperplasia, papillary lengthening, degranulating eosinophils, and lamina propria fibrosis/inflammation. (1)

3. Name two associated symptoms or conditions.

Clinical manifestations of eosinophilic esophagitis include predominantly dysphagia, with or without morphologic abnormalities in the esophagus. Food bolus impaction is a common symptom. GERD is usually present for years prior to diagnosis; vomiting and abdominal pain are also reported. Perforation is a rare complication.

Overlap conditions include GERD and eosinophilic gastroenteritis. Other atopic conditions are also noted, including asthma, allergic rhinitis, urticaria, atopic dermatitis, food allergy, medicine allergy, peripheral eosinophilia and positive RAST testing. (1)

4. What is the treatment?

Treatment is not clearly defined. Acid suppression may or may not improve GERD symptoms, but antisecretory therapy may be protective of further damage to the esophagus. Elimination diets and avoidance of allergens is advocated, although long-term efficacy is not clear and adherence may be difficult.

Systemic corticosteroids are effective, however relapse occurs after cessation and they are not useful for long term use. Topical corticosteroids has been shown to be effective (usually fluticasone MDI, puffed into the mouth without a spacer, swallowed and rinsed down with water); oral budesonide suspensions have also been used. Some studies also suggest that montelukast may be effective.

Esophageal rings may require dilation, and esophagoscopy should be performed in those who have changing or new symptoms. (0.5)