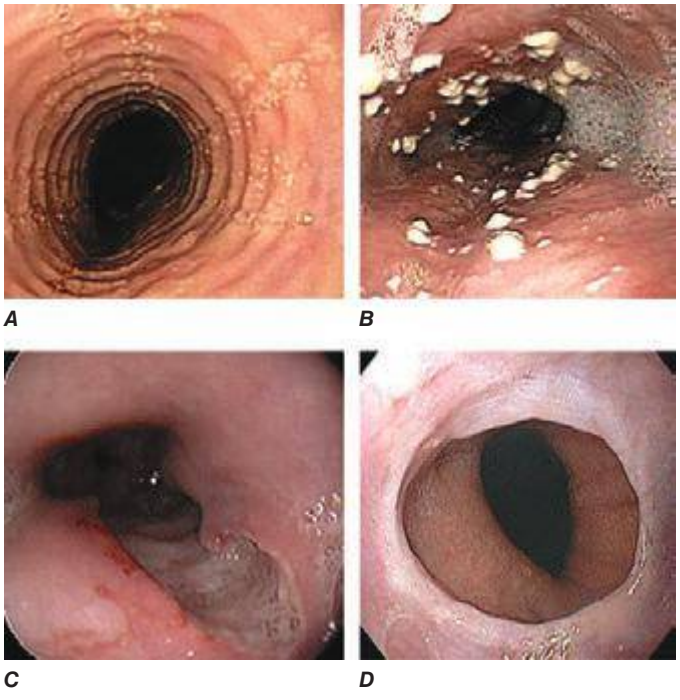

FIGURE 347-11 Endoscopic features of (*A*) eosinophilic esophagitis (EoE), (*B*) *Candida* esophagitis, (*C*) giant ulcer associated with HIV, (*D*) and a Schatzki ring.

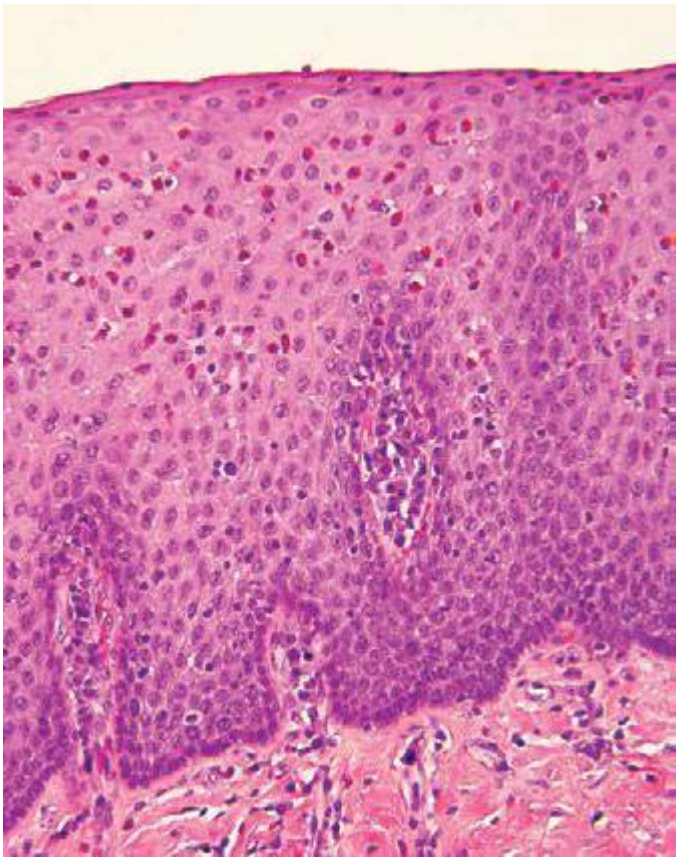

FIGURE 347-12 Histopathology of eosinophilic esophagitis (EoE) showing infiltration of the esophageal squamous epithelium with eosinophils. Additional features of basal cell hyperplasia and lamina propria fibrosis are present. Eosinophilic inflammation can also be seen with gastroesophageal reflux disease.

EoE should be strongly considered in children and adults with dysphagia and esophageal food impactions. In preadolescent children, symptom presentations of EoE include chest or abdominal pain, nausea, vomiting, and food aversion. Other symptoms in adults may include atypical chest pain and heartburn, particularly heartburn that is refractory to PPI therapy. An atopic history of food allergy, asthma, eczema, or allergic rhinitis is present in the majority of patients. Peripheral blood eosinophilia is demonstrable in up to 50% of patients, but the specificity of this finding is problematic in the setting of concomitant atopy. The characteristic endoscopic esophageal findings are loss of vascular markings (edema), multiple esophageal rings, longitudinally oriented furrows, and punctate exudate (Fig. 347-11). Histologic confirmation is made with the demonstration of esophageal mucosal eosinophilia (greatest density ±15 eosinophils per high-power field) (Fig. 347-12). Complications of EoE include esophageal stricture, narrow-caliber esophagus, food impaction, and esophageal perforation.

The goals of EoE management are symptom control and the prevention of complications. Once esophageal eosinophilia is demonstrated, patients typically undergo a trial of PPI therapy as a practical means of excluding a contribution of GERD to the esophageal mucosal inflammation. PPI-responsive esophageal eosinophilia, characterized by elimination of mucosal eosinophilia, occurs in 30–50% of cases of suspected EoE. Patients with persistent symptoms and eosinophilic inflammation following PPI therapy are subsequently considered for EoE treatments such as elimination diets or swallowed topical glucocorticoids. Elemental formula diets are a highly effective therapy that have primarily been studied in children but are limited by palatability. Notably, allergy testing by means of either serum IgE or skin prick testing has demonstrated poor sensitivity and specificity in the identification of foods that incite the esophageal inflammatory response. Allergy testing combining skin prick and atopy patch testing has been effective in children with EoE, but additional validation is needed. Empiric elimination of common food allergies (milk, wheat, egg, soy, nuts, and seafood) followed by systematic reintroduction has been an effective diet therapy in both children and adults with EoE. The intent of the elimination diet approach is the identification of a single food trigger or a small number of food triggers. Swallowed, topical glucocorticoids (fluticasone propionate or budesonide) are highly effective, but recurrence of disease is common following the cessation of therapy. Systemic glucocorticoids are reserved for severely afflicted patients refractory to less morbid treatments. Esophageal dilation is very effective at relieving dysphagia in patients with fibrostenosis. Dilation should be approached conservatively because of the risk of deep, esophageal mural laceration or perforation in the stiff-walled esophagus that is characteristic of the disease.

INFECTIOUS ESOPHAGITIS

With the increased use of immunosuppression for organ transplantation as well as chronic inflammatory diseases and chemotherapy along with the AIDS epidemic, infections with *Candida* species, herpesvirus, and cytomegalovirus (CMV) have become relatively common. Although rare, infectious esophagitis also occurs among the nonimmunocompromised, with herpes simplex and *Candida albicans* being the most common pathogens. Among AIDS patients, infectious esophagitis becomes more common as the CD4 count declines; cases are rare with a CD4 count >200 and common when <100. HIV itself may also be associated with a self-limited syndrome of acute esophageal ulceration with oral ulcers and a maculopapular skin rash at the time of seroconversion. Additionally, some patients with advanced disease have deep, persistent esophageal ulcers treated with oral glucocorticoids or thalidomide. However, with the widespread use of protease inhibitors, a reduction in these HIV complications has been noted.

Regardless of the infectious agent, odynophagia is a characteristic symptom of infectious esophagitis; dysphagia, chest pain, and hemorrhage are also common. Odynophagia is uncommon with reflux esophagitis, so its presence should always raise suspicion of an alternative etiology.