**ESOPHAGITIS**

**Etiology**

1. Chemical
2. Infectious
3. Eosinophilic
4. Reflux

**Chemical esophagitis**

The stratified squamous mucosa of the esophagus may be damaged by a variety of irritants:

1. Alcohol
2. corrosive acids or alkalis
3. excessively hot fluids
4. heavy smoking
5. Pill-induced (e.g. doxycycline and bisphosphonates)
6. Iatrogenic ( chemotherapy and radiation)

Clinically, Esophagitis due to chemical injury generally causes only self-limited pain, particularly odynophagia (pain with swallowing). Hemorrhage, stricture, or perforation may occur in severe cases.

The morphologic changes are nonspecific, consisting of ulceration and acute inflammation.

**Infectious esophagitis**

It is most frequent in those who are debilitated or immunosuppressed.

Causes:

1. Viral : HSV, CMV
2. Fungal: candidiasis , mucormycosis and aspergillosis
3. Bacterial :

Candidiasis is characterized by adherent, gray-white pseudomembranes composed of densely matted fungal hyphae and inflammatory cells covering the esophageal mucosa.

Herpes viruses typically cause punched-out ulcers, and histopathologic analysis demonstrates nuclear viral inclusions within degenerating epithelial cells.

CMV causes shallower ulcerations. Biopsy of these lesions shows the characteristic nuclear and cytoplasmic inclusions within capillary endothelium and stromal cells.

**Eosinophilic esophagitis**

Eosinophilic esophagitis is a chronic immunologically mediated disorder. Symptoms include food impaction and dysphagia in adults and feeding intolerance or GERD-like symptoms in children.Most patients are atopic, and many have atopic dermatitis, allergic rhinitis, asthma, or modest peripheral eosinophilia.

The cardinal histologic feature is epithelial infiltration by large numbers of eosinophils, particularly superficially and at sites far from the gastroesophageal junction. Their abundance can help to differentiate eosinophilic esophagitis from GERD and other causes of esophagitis. Endoscopically evident rings in the upper and mid portions of the esophagus may also help to distinguish eosinophilic esophagitis from GERD.

**Reflux Esophagitis**

Reflux of gastric contents into the lower esophagus is the most frequent cause of esophagitis. The associated clinical condition is termed *gastroesophageal reflux disease (GERD).*

**Pathogenesis**

Reflux of gastric juices is central to the development of mucosal injury in GERD. High lower esophageal sphincter tone protects against reflux of acidic gastric contents, which are under positive pressure. Conditions that decrease lower esophageal sphincter tone or increase abdominal pressure contribute to GERD; they include alcohol and tobacco use, obesity, central nervous system depressants, pregnancy, hiatal hernia, delayed gastric emptying, and increased gastric volume. In many cases, no definitive cause is identified.

**Morphology**

In mild GERD, the mucosal histology is often unremarkable. With more significant disease, eosinophils are recruited into the squamous mucosa, followed by neutrophils, which usually are associated with more severe injury. Basal zone hyperplasia exceeding 20% of the total epithelial thickness and elongation of lamina propria papillae, such that they extend into the upper third of the epithelium, also may be present.

**Clinical features**

GERD is most common in those over 40 years of age but also occurs in infants and children. The most frequent symptoms are heartburn, dysphagia, and, less often, noticeable regurgitation of sour-tasting gastric contents. Rarely, chronic GERD is punctuated by attacks of severe chest pain that may be mistaken for heart disease.

Complications of reflux esophagitis include esophageal ulceration, hematemesis, melena, stricture development, and Barrett esophagus (a precursor lesion to esophageal carcinoma).

**Barrett esophagus**

Barrett esophagus is a complication of chronic GERD that is characterized by intestinal metaplasia within the esophageal squamous mucosa. It is estimated to occur in as many as 10% of individuals with symptomatic GERD. Epithelial *dysplasia* develops in 0.2% to 1% of individuals with Barrett esophagus each year.

The greatest concern in Barrett esophagus is that it confers an increased risk for development of esophageal adenocarcinoma. However, most individuals with Barrett esophagus do not develop esophageal cancer.

**Morphology**

Barrett esophagus is recognized endoscopically as tongues or patches of red, velvety mucosa extending upward from the gastroesophageal junction. This metaplastic mucosa alternates with residual smooth, pale squamous (esophageal) mucosa. The defining microscopic feature of intestinal metaplasia is the presence of goblet cells, which have distinct mucous vacuoles that stain pale blue by H&E. Dysplasia, if present, is classified as low-grade or high-grade on the basis of morphologic criteria.

***Most experts require both endoscopic evidence of abnormal mucosa above the gastroesophageal junction and histologically documented intestinal metaplasia for diagnosis of Barrett esophagus.***

**Clinical features**

White males are affected most often and typically present between 40 and 60 years of age. Diagnosis of Barrett esophagus is usually prompted by GERD symptoms and requires endoscopy and biopsy. Most clinicians recommend periodic surveillance endoscopy with biopsy to screen for dysplasia.