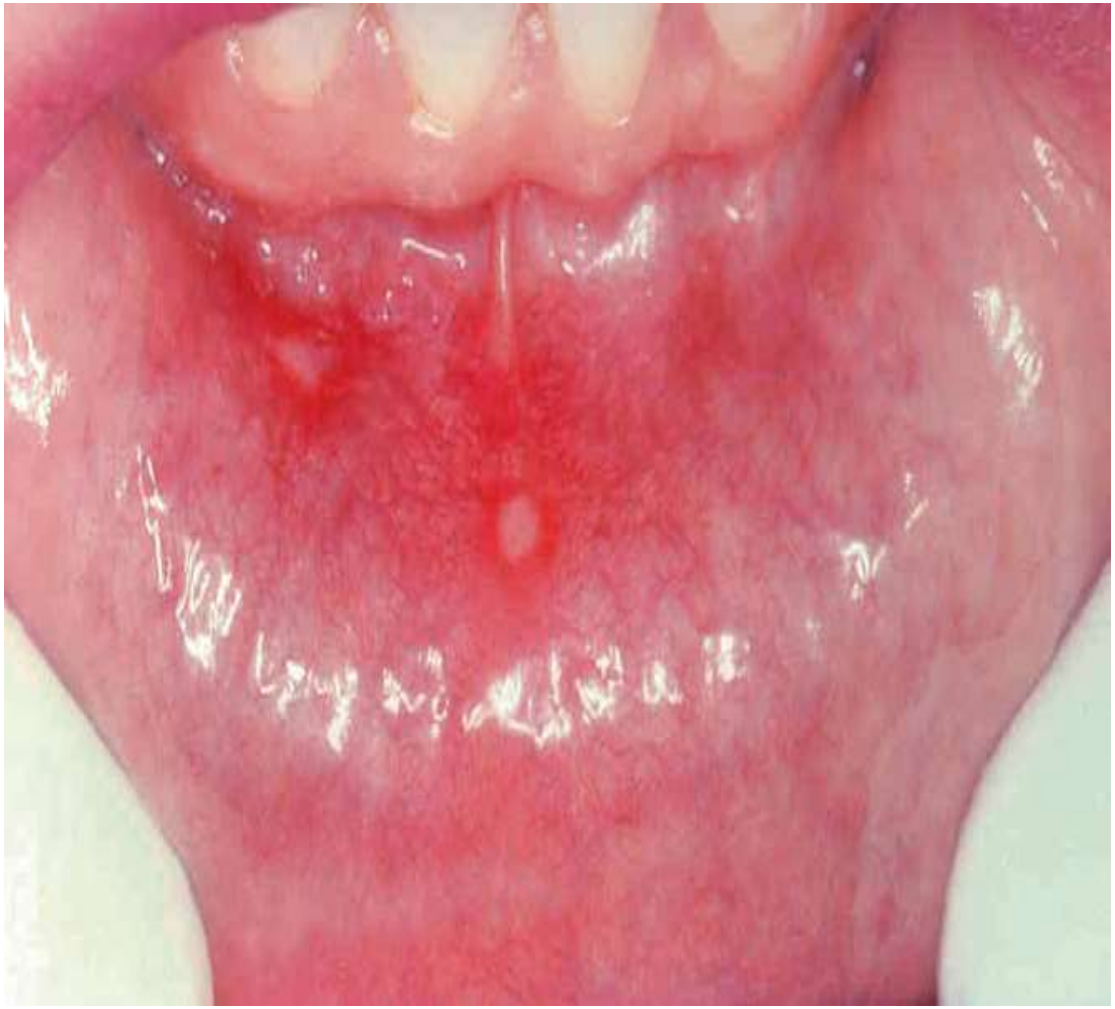
Gastrointestinal tract lab 1+2 Second year.

DR.Bushra Al-Tarawneh,MD

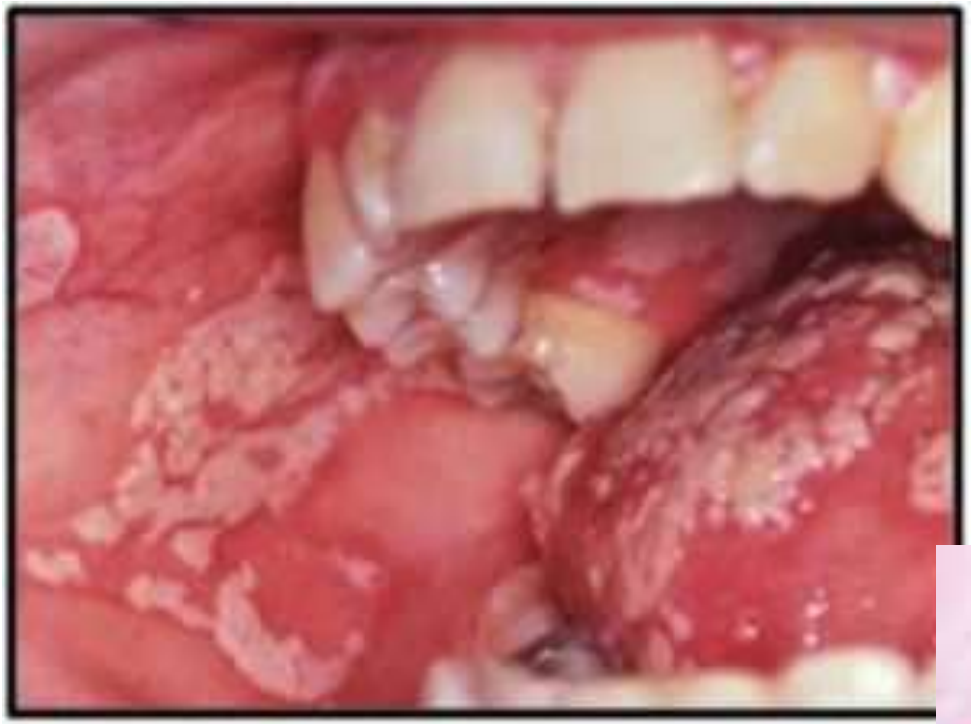
ORAL CAVITY

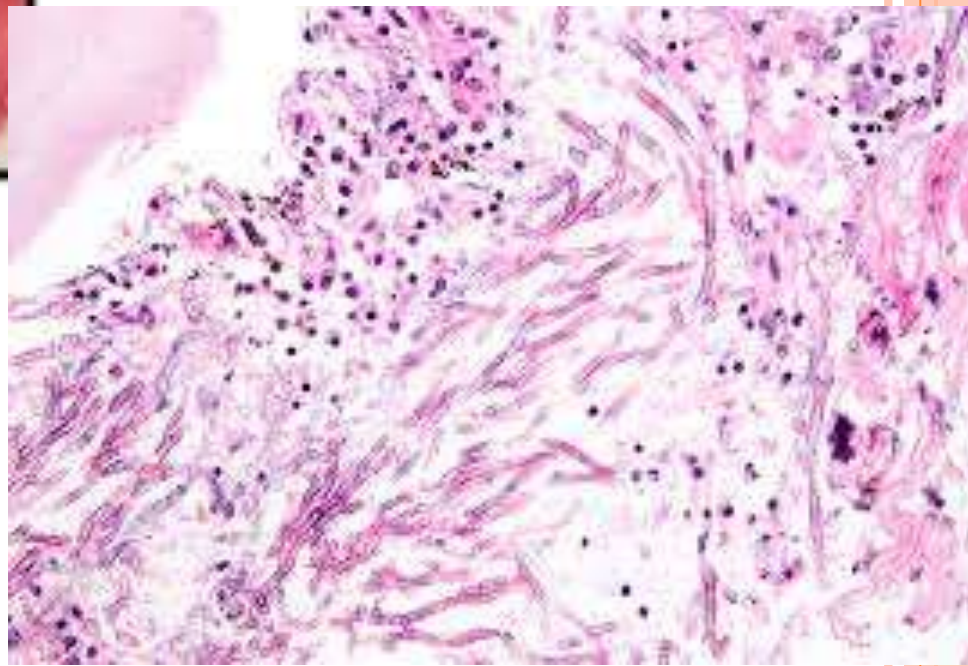
ORAL INFLAMMATORY LESIONS.

- Aphthous Ulcers (Canker Sores)

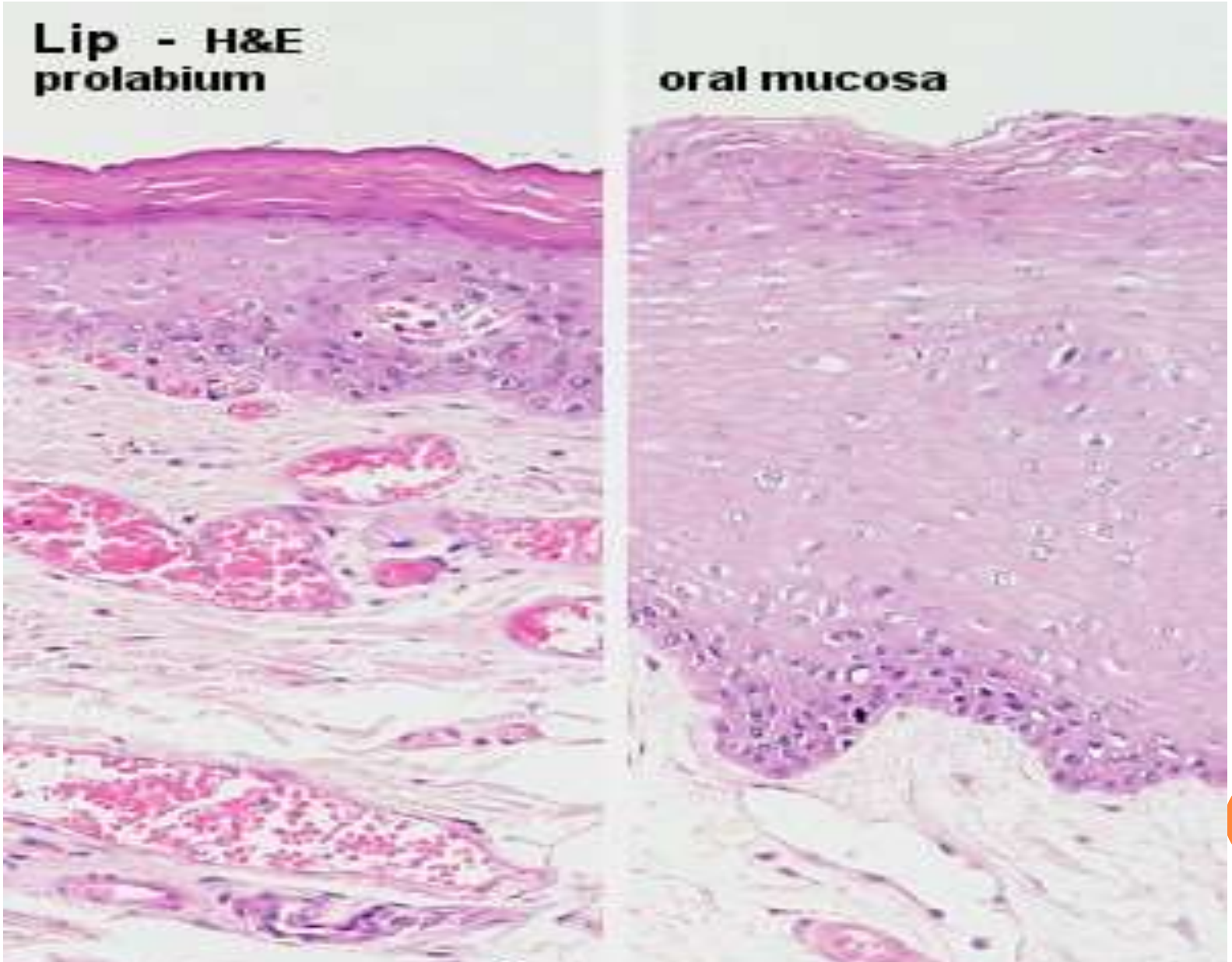


Oral Candidiasis (Thrush).

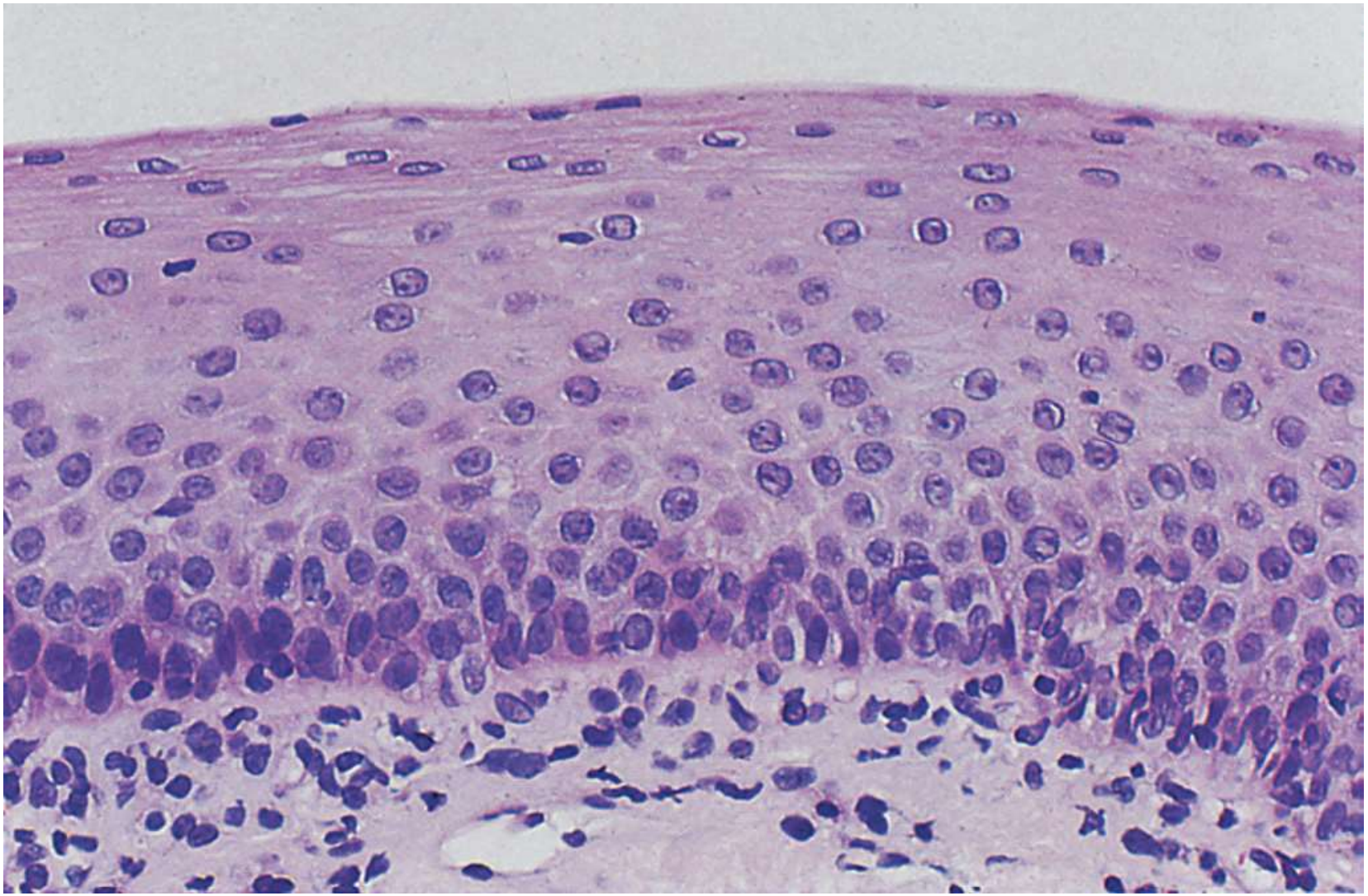




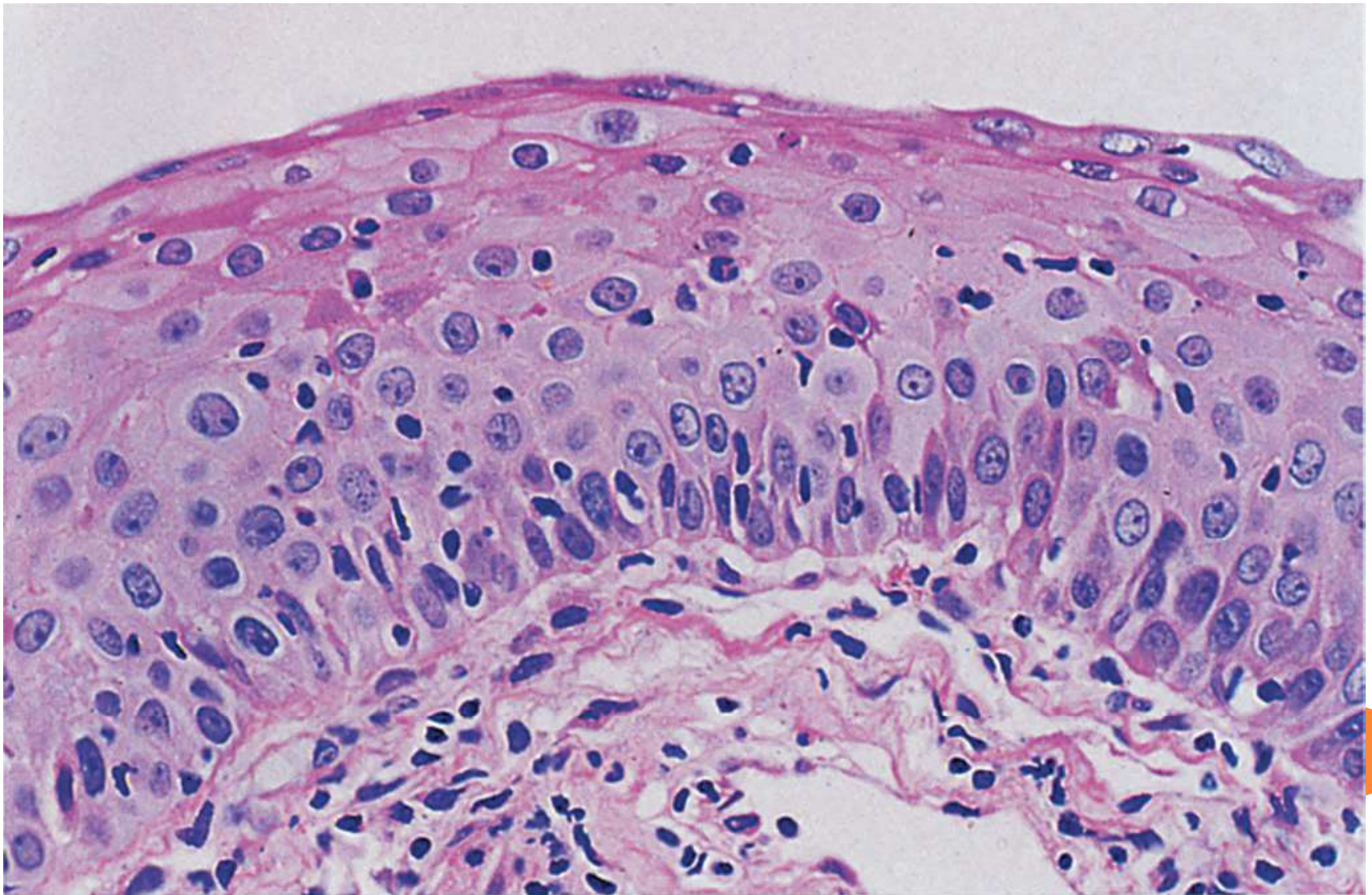
Normal histology of the oral mucosa.



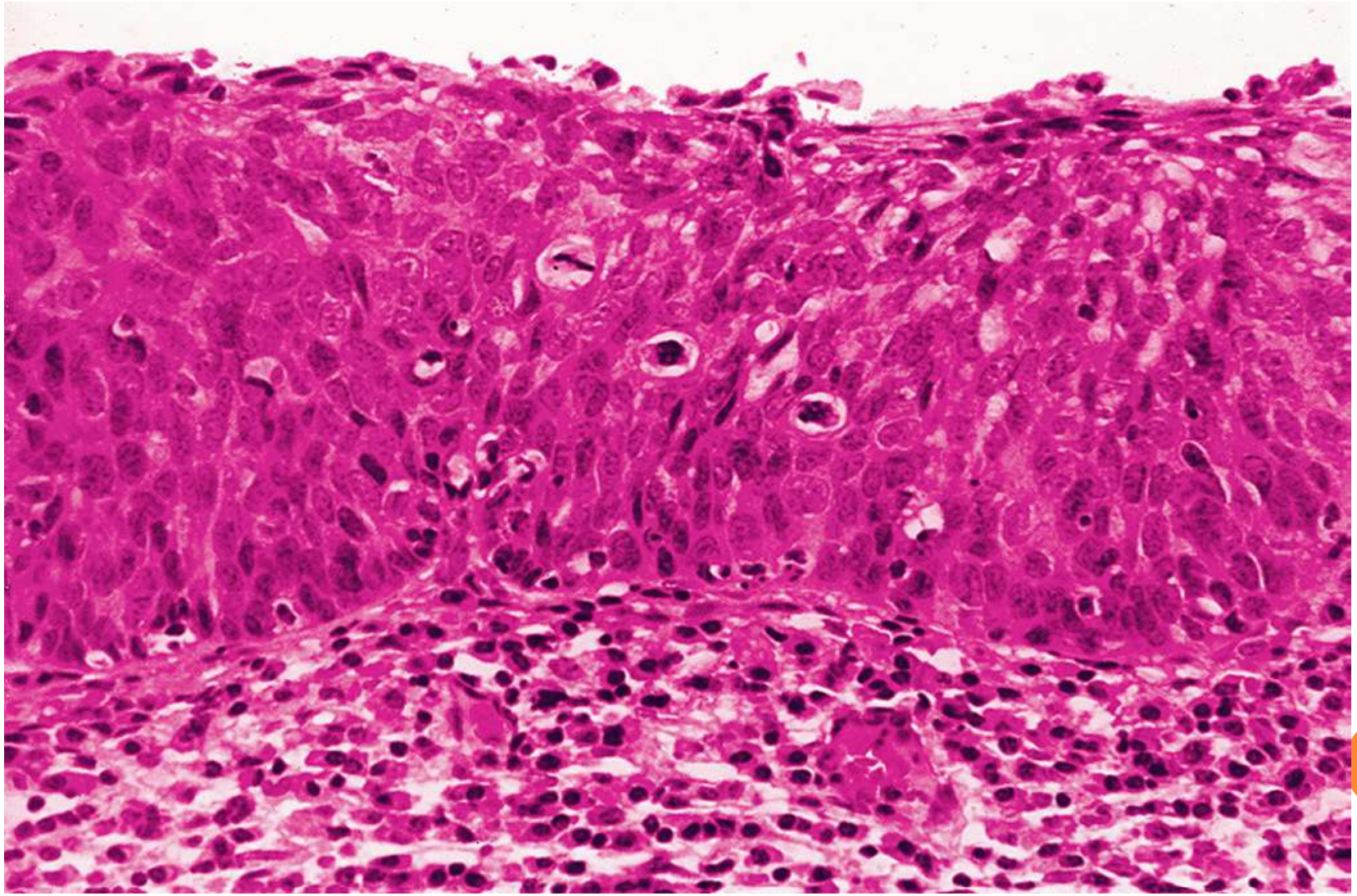
Mild dysplasia



Moderate Dysplasia.

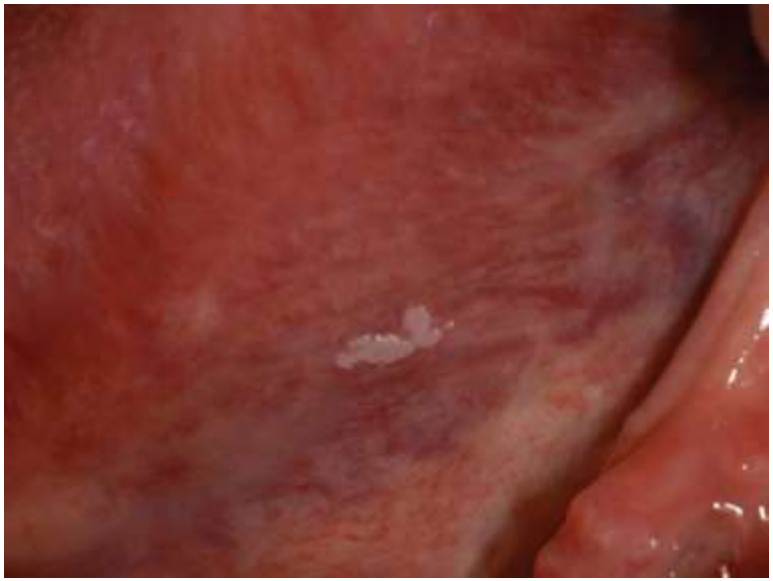


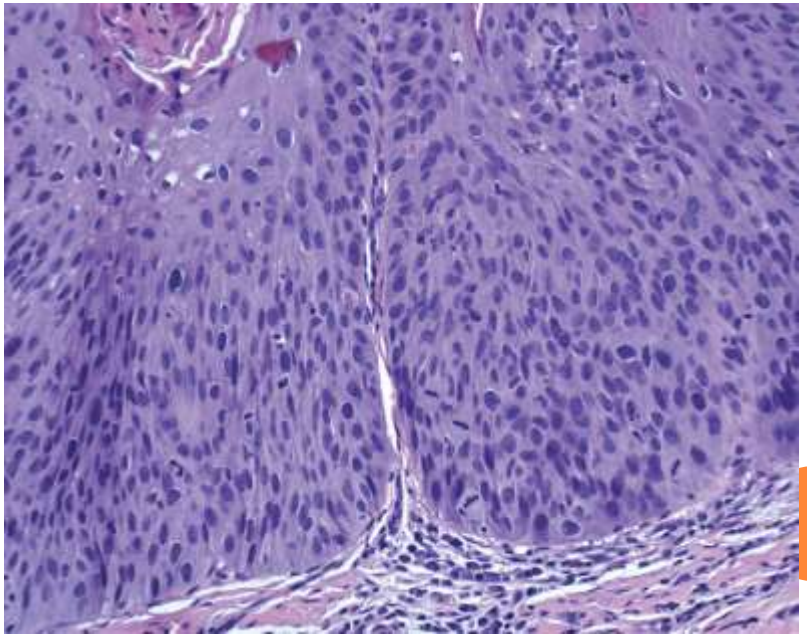
SEVERE DYSPLASIA = CIS



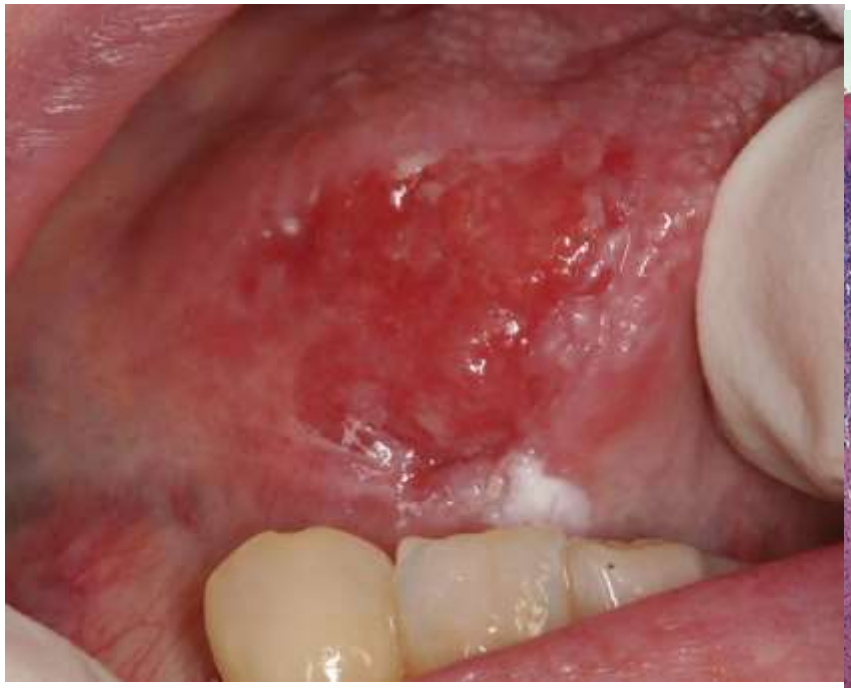
Leukoplakia

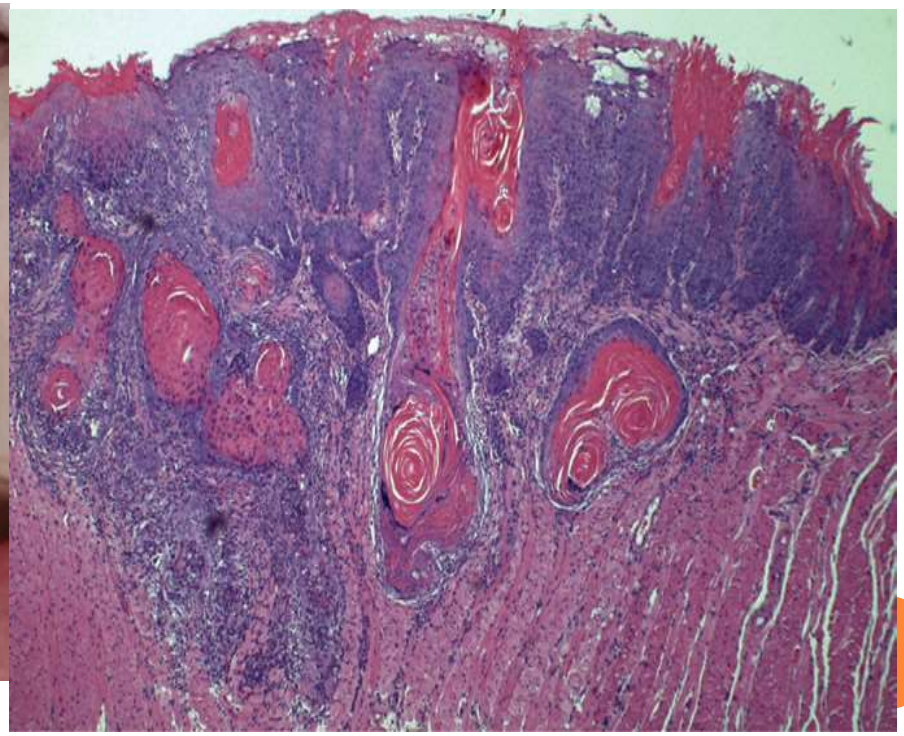
the lesion is smooth with well-demarcated borders and minimal elevation. B, Histologic appearance of leukoplakia showing dysplasia, characterized by nuclear and cellular pleomorphism and loss of normal maturation.



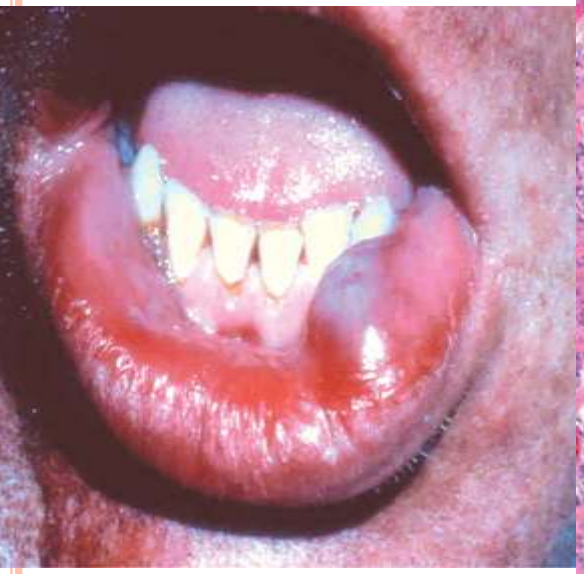


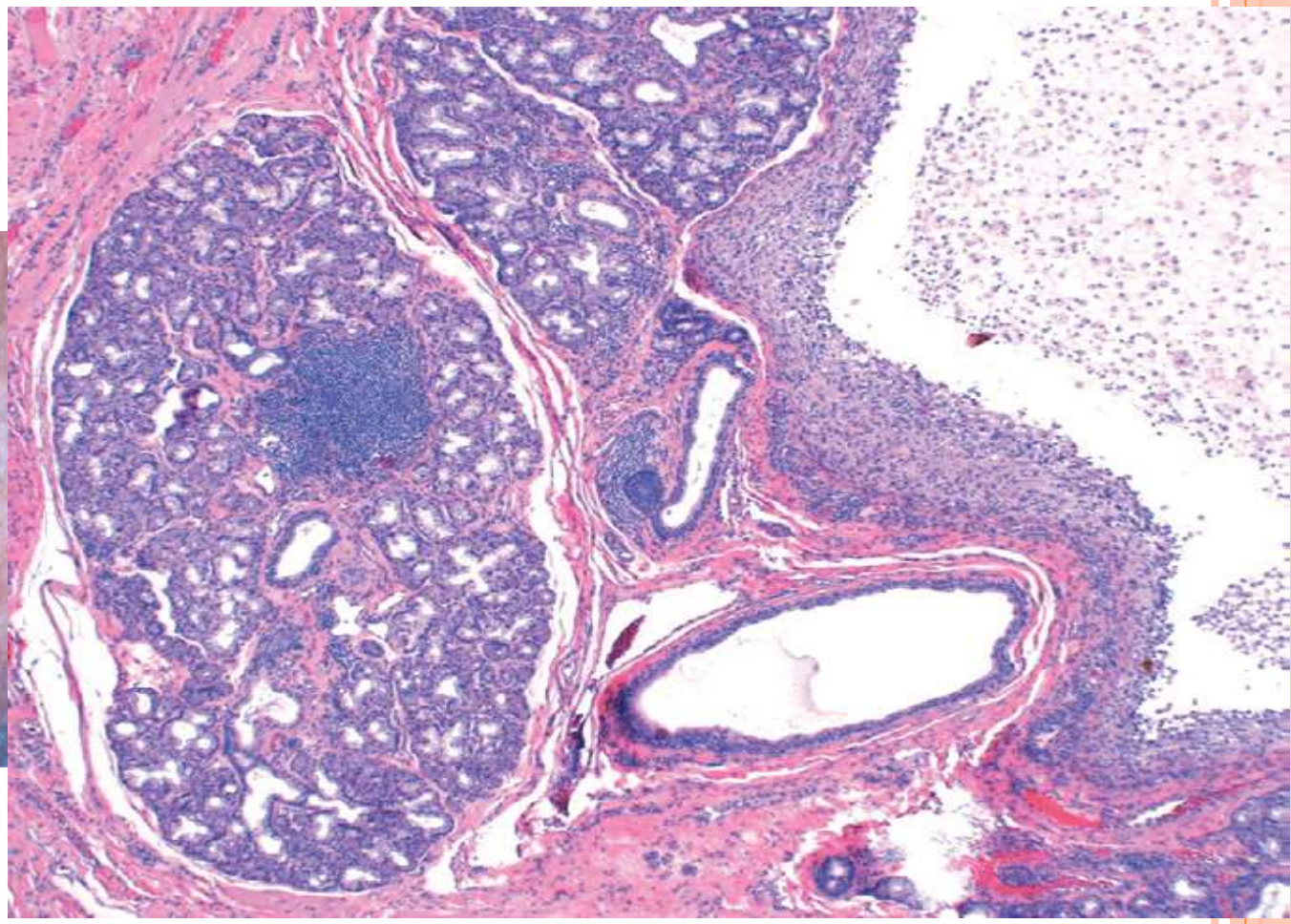
Clinical appearance demonstrating ulceration and induration of the oral mucosa. Histologic appearance demonstrating numerous nests and islands of malignant keratinocytes invading the underlying connective tissue stroma.





Mucocele





Histologic examination demonstrates a cystlike space lined by inflammatory granulation tissue or fibrous connective tissue that is filled with mucin and inflammatory cells, particularly macrophages.

Salivary gland tumors

Table 14–1 Histopathologic Classification and Prevalence of the Most Common Benign and Malignant Salivary Gland Tumors

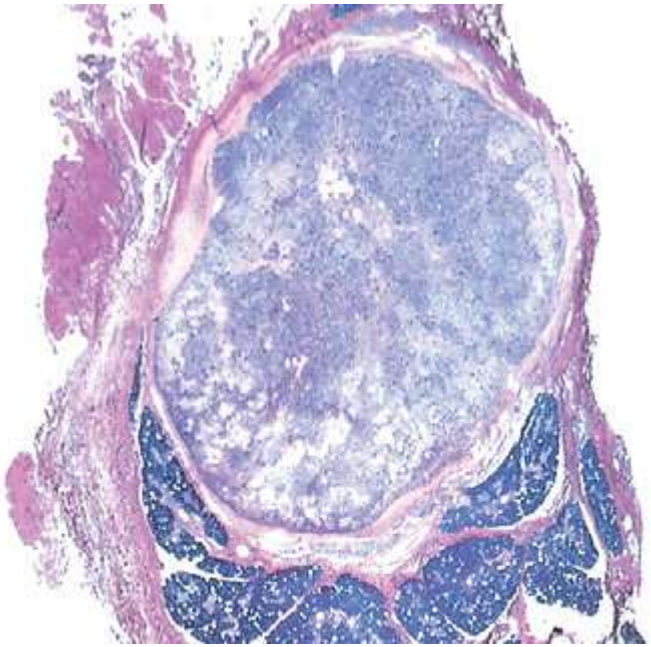
Benign	Malignant
Pleomorphic adenoma (50%)	Mucoepidermoid carcinoma (15%)
Warthin tumor (5%)	Acinic cell carcinoma (6%)
Oncocytoma (2%)	Adenocarcinoma NOS (6%)
Cystadenoma (2%)	Adenoid cystic carcinoma (4%)
Basal cell adenoma (2%)	Malignant mixed tumor (3%)

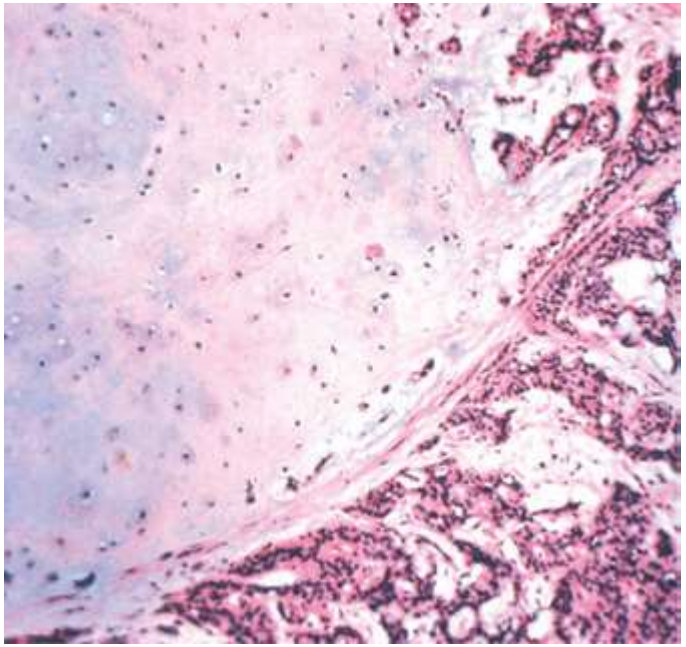
NOS, not otherwise specified.

Data from Ellis GL, Auclair PL, Gnepp DR: Surgical Pathology of the Salivary Glands, Vol 25: Major Problems in Pathology, Philadelphia, WB Saunders, 1991.

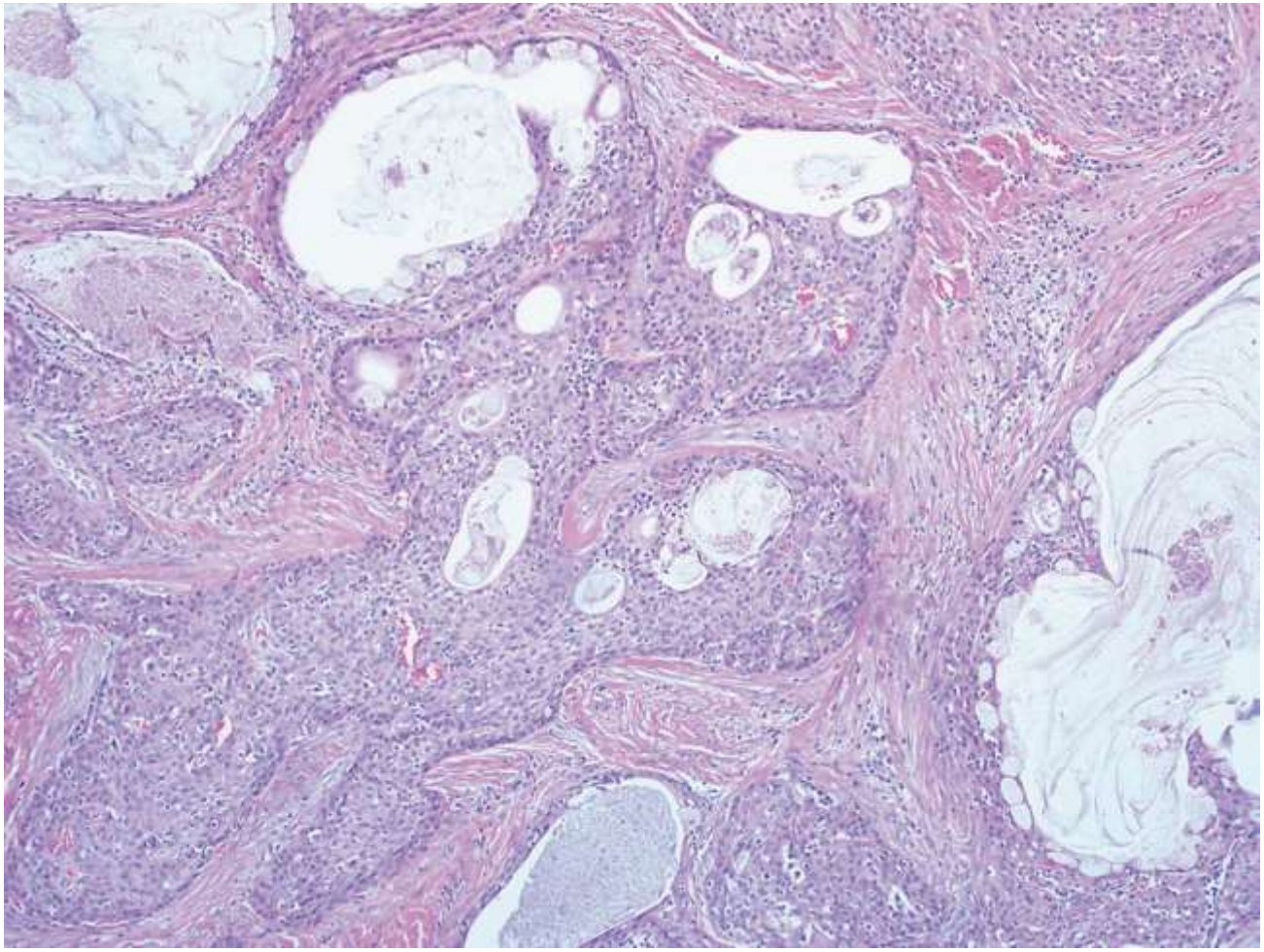
Pleomorphic Adenoma

- Low-power view showing awell-demarcated tumor with adjacent normal salivary gland parenchyma. **B,** High-power view showing epithelial cells as well as myoepithelial cells within chondroid matrix material.



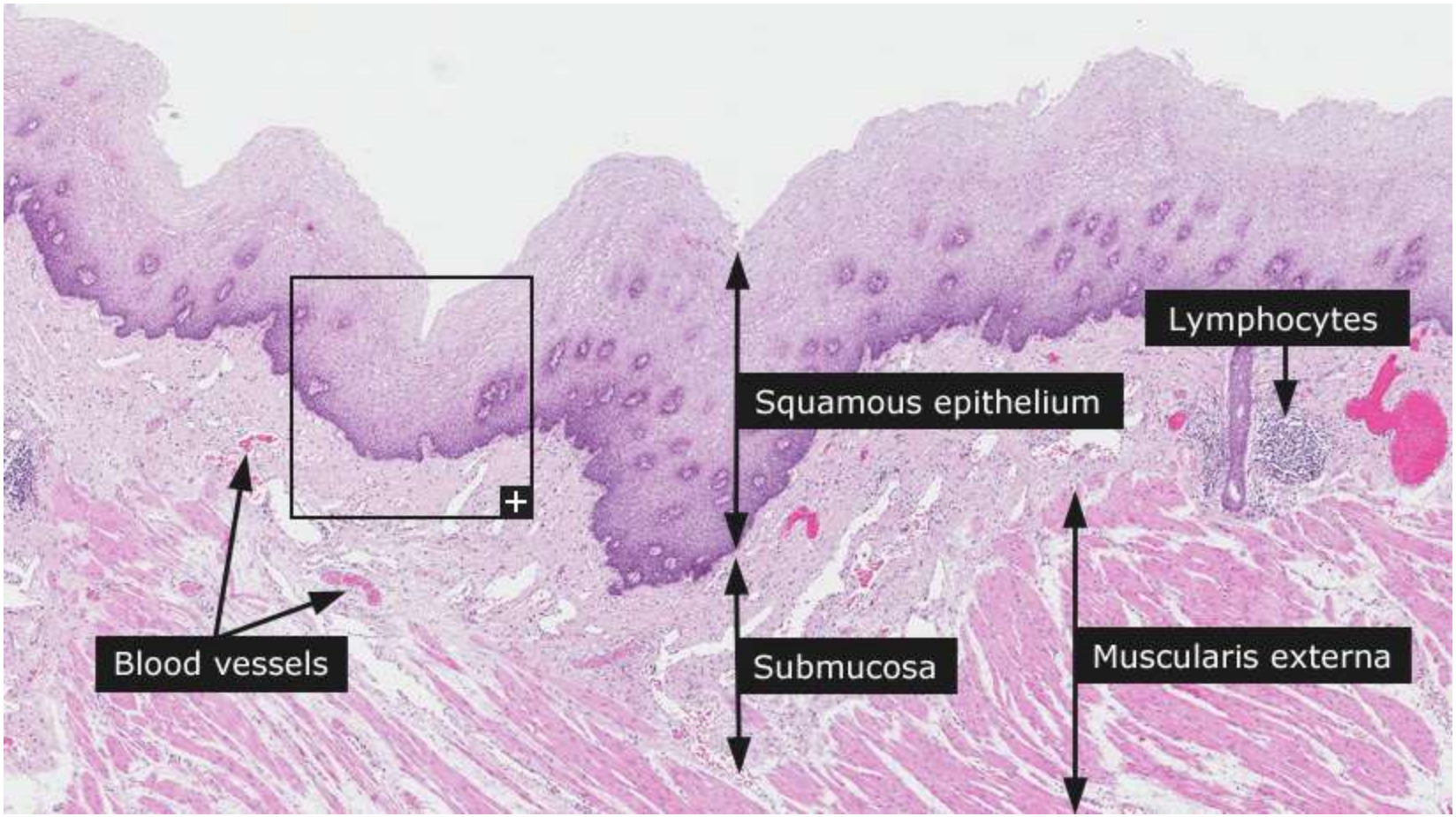


Mucoepidermoid Carcinoma

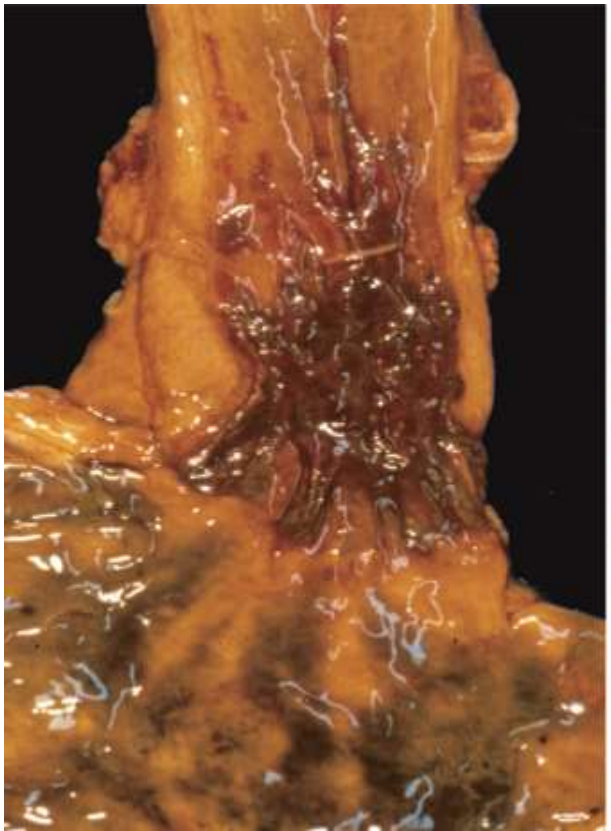


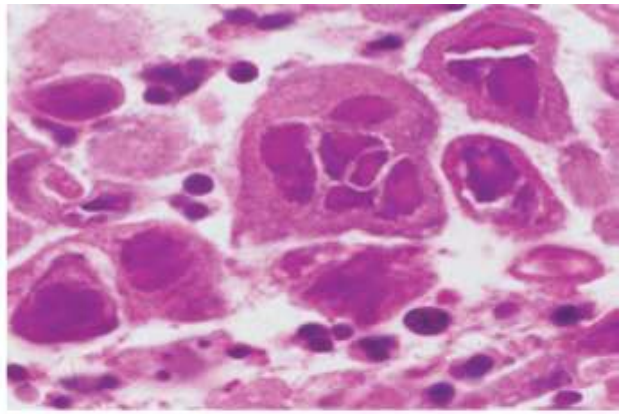
Esophagus

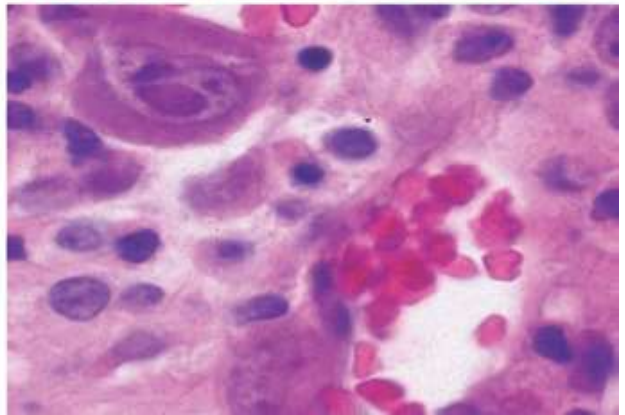
Normal histology of the oral mucosa.



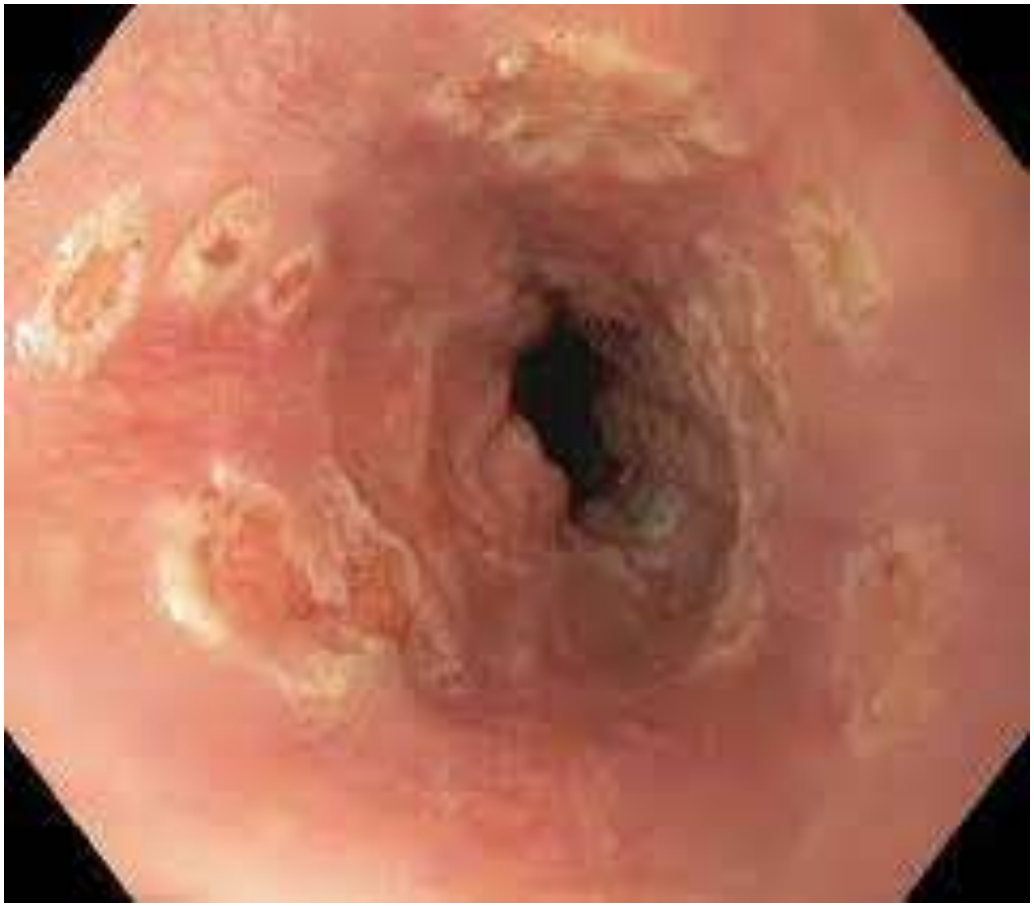
CMV causes shallower ulcerations and characteristic nuclear and cytoplasmic inclusions within capillary endothelium and stromal cells.



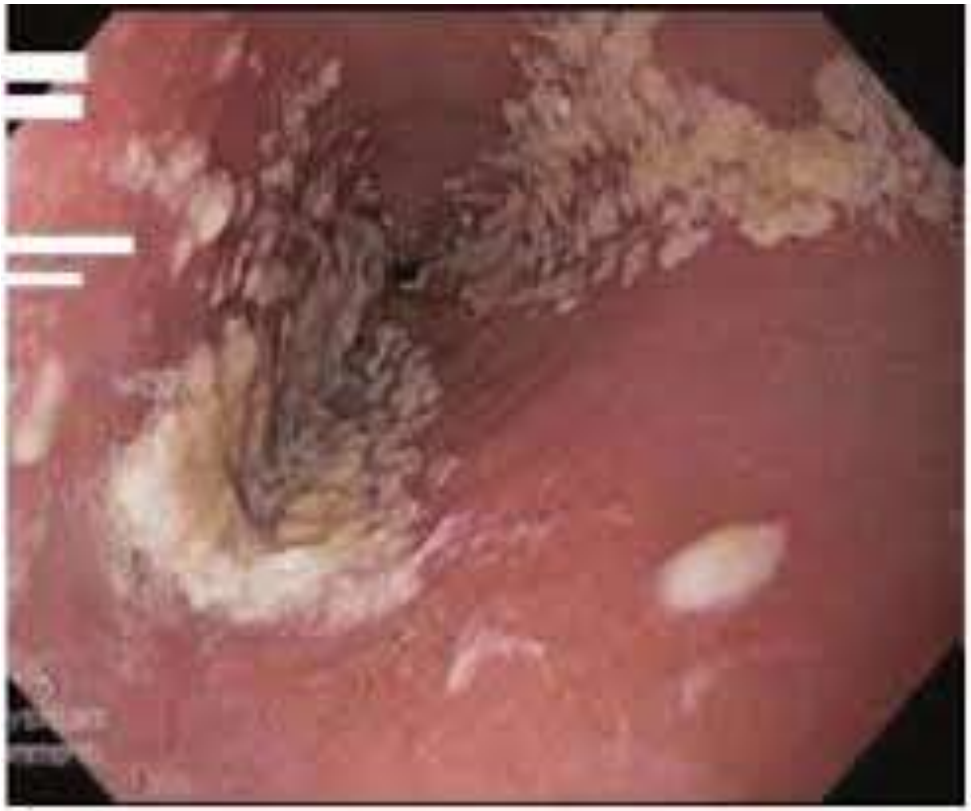




Herpesviruses esophagitis



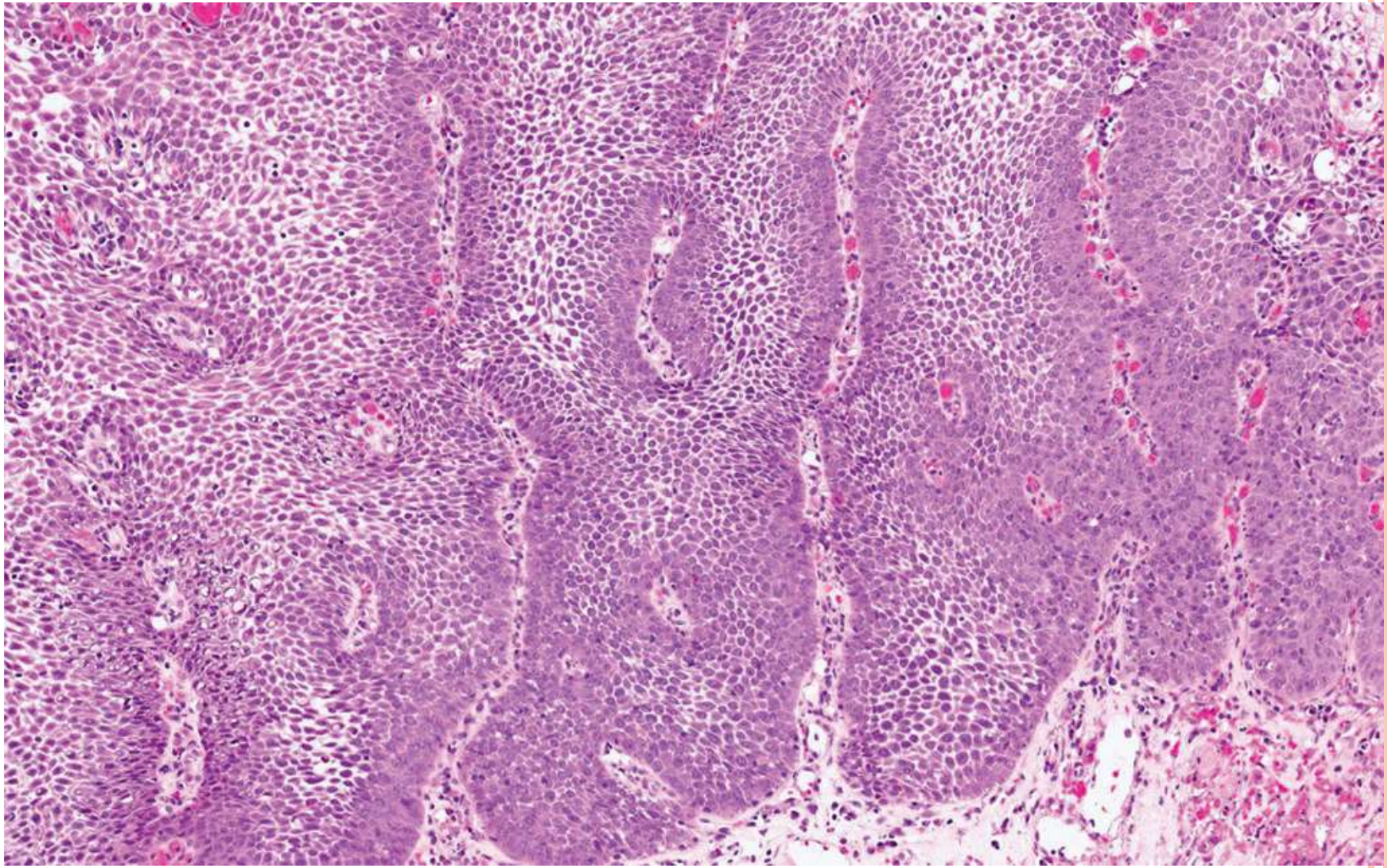
Candidiasis



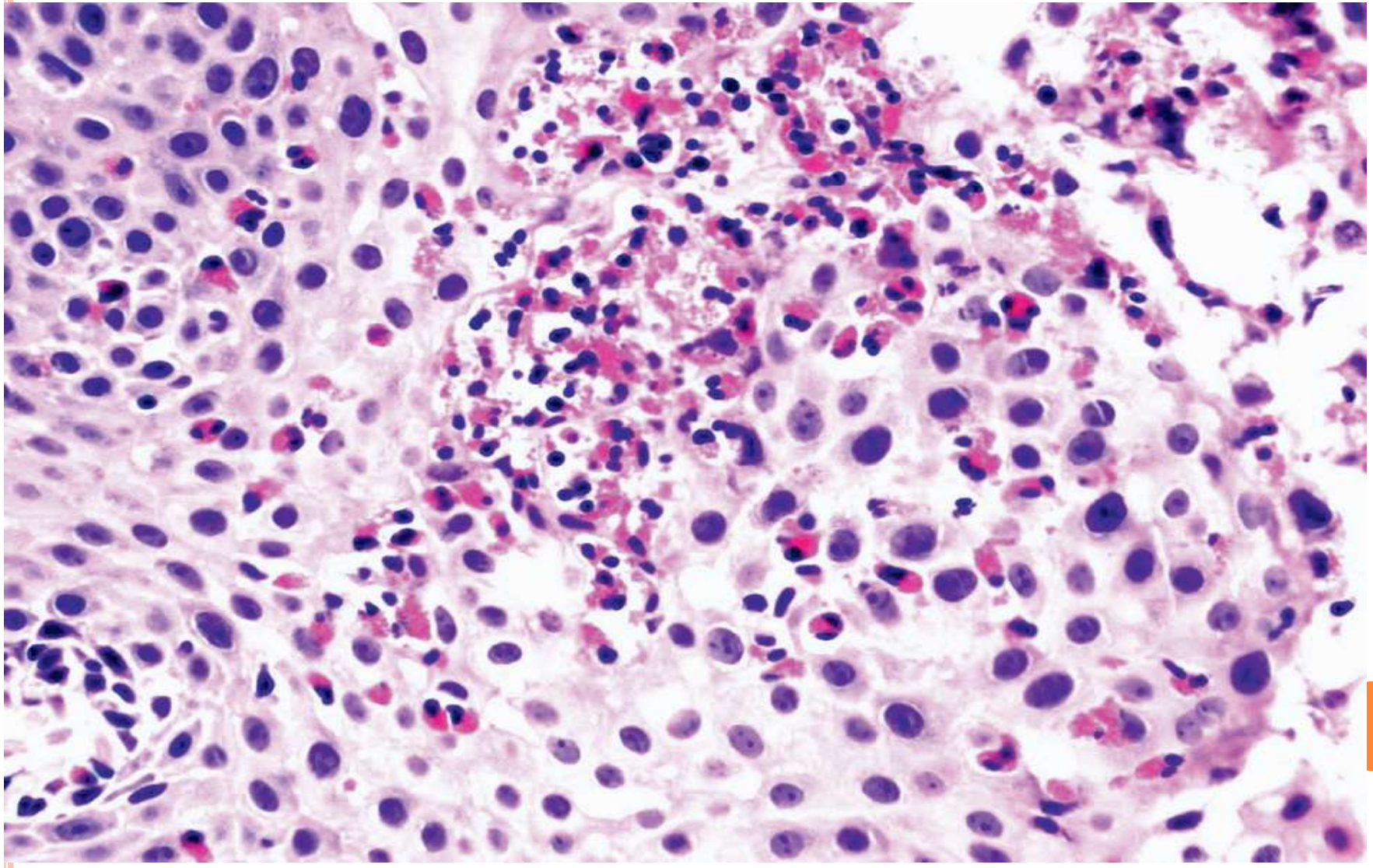
Reflux esophagitis

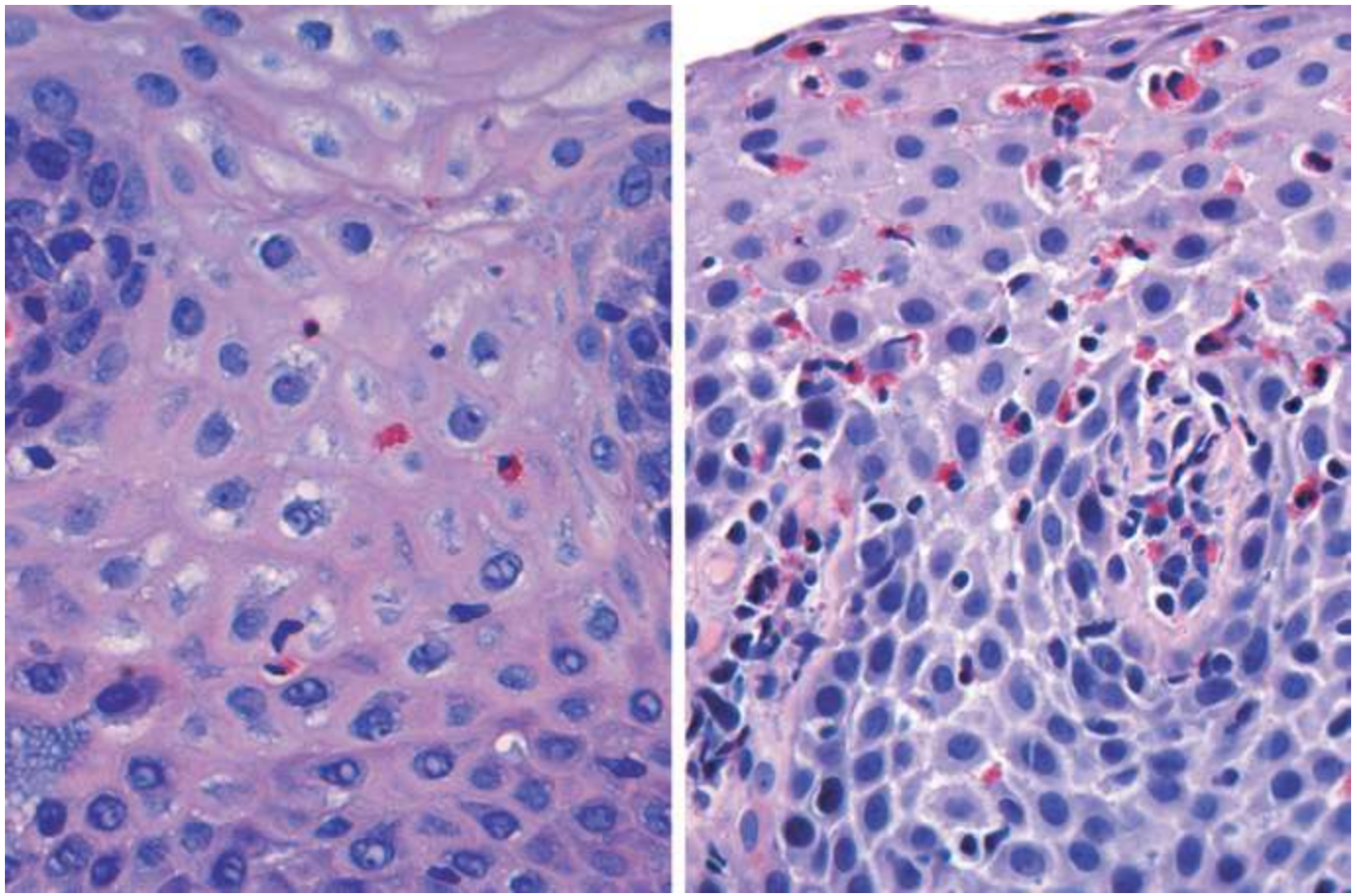
- Simple hyperemia, evident to the endoscopist as redness. **Microscopically**: Eosinophils are recruited into the squamous mucosa, followed by neutrophils.Basal zone hyperplasia elongation of lamina propria papillae.

- Treatment with proton pump inhibitors reduces gastric acidity and typically provides symptomatic relief.
- Complications include esophageal ulceration, hematemesis, melena, stricture development, and Barrett esophagus.

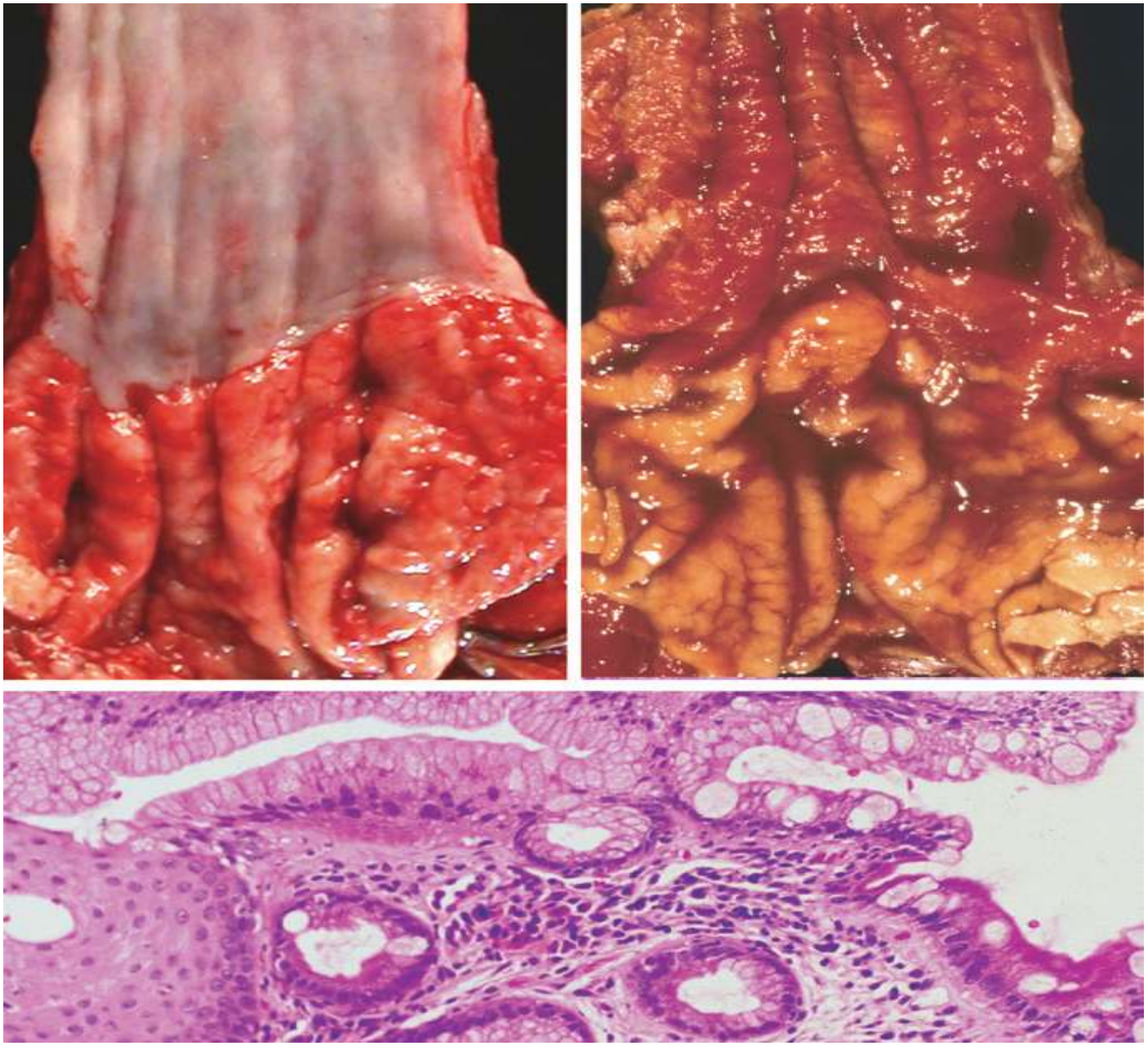


Eosinophilic Esophagitis

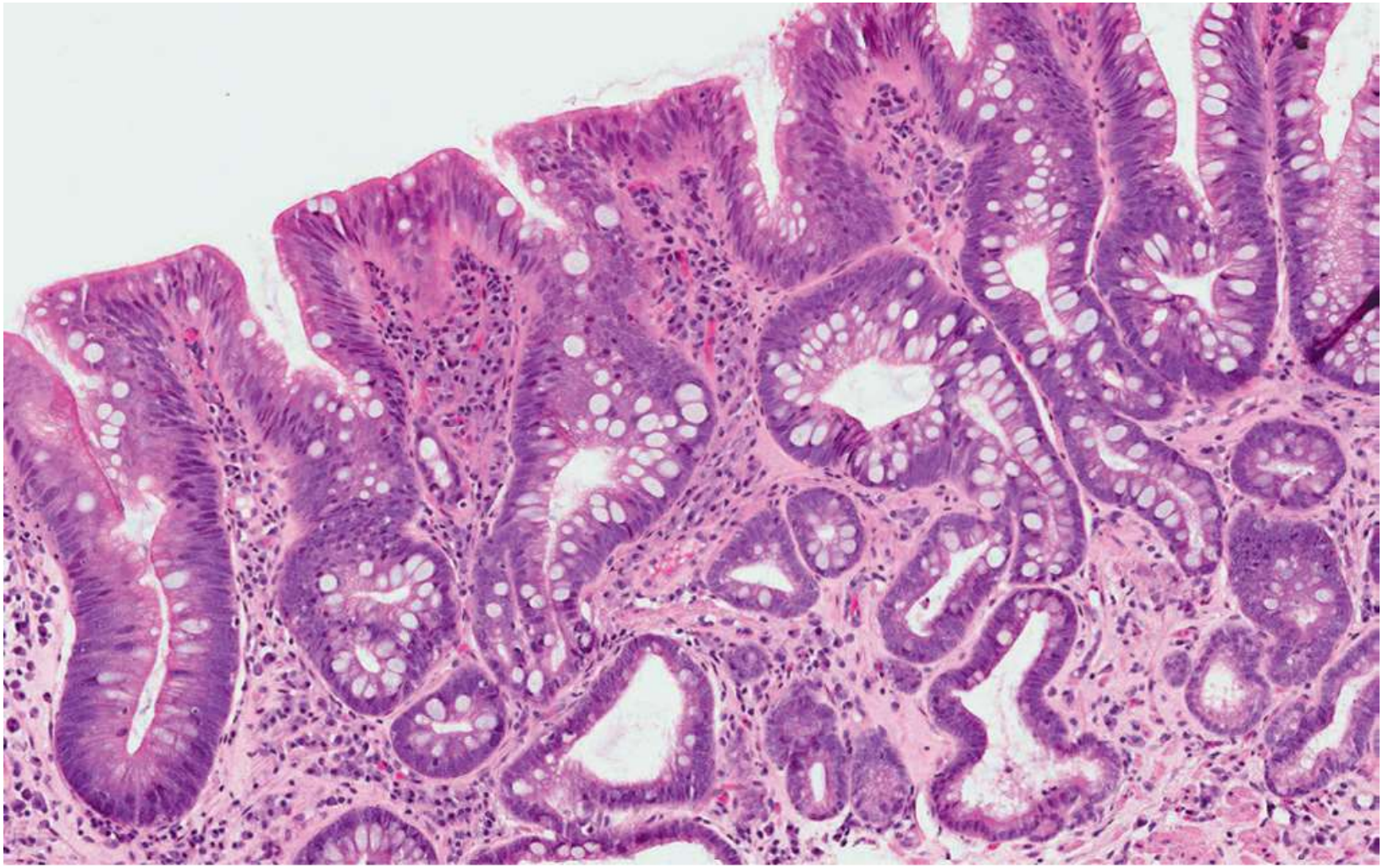




Barrett esophagus

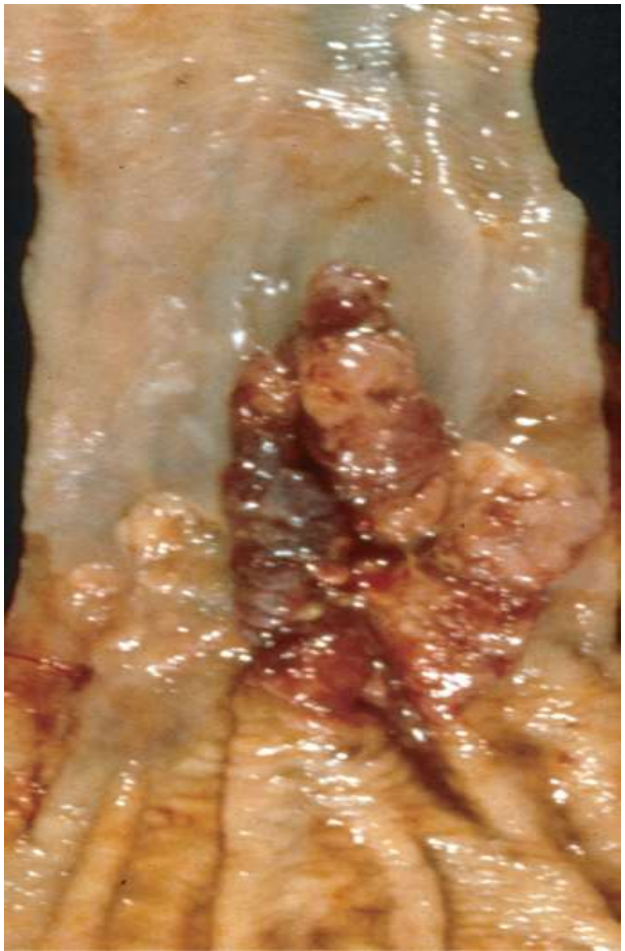


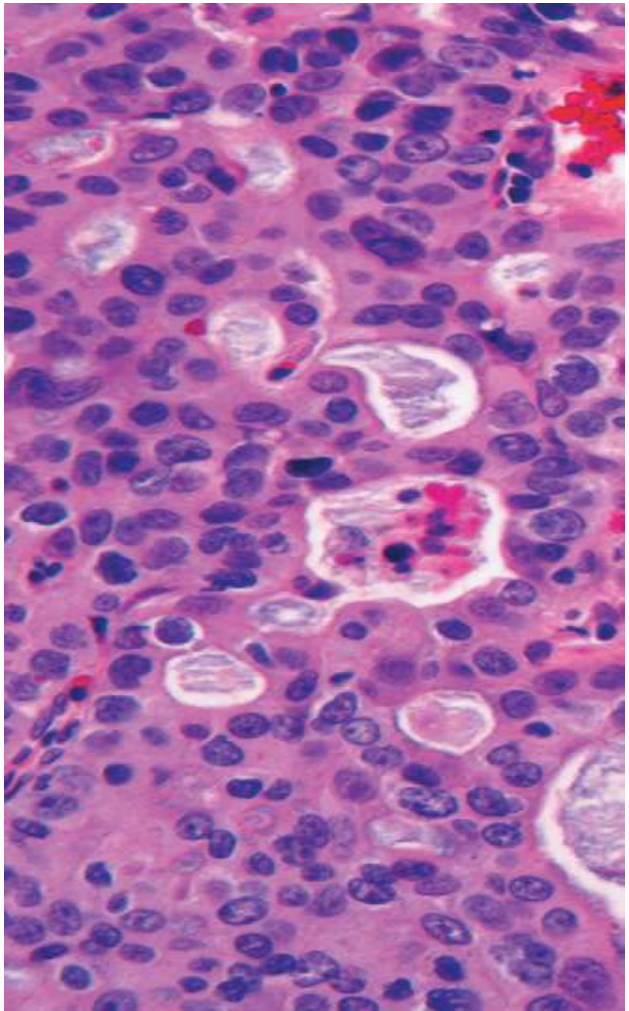
. Barrett esophagus with low-grade dysplasia, intestinal type.



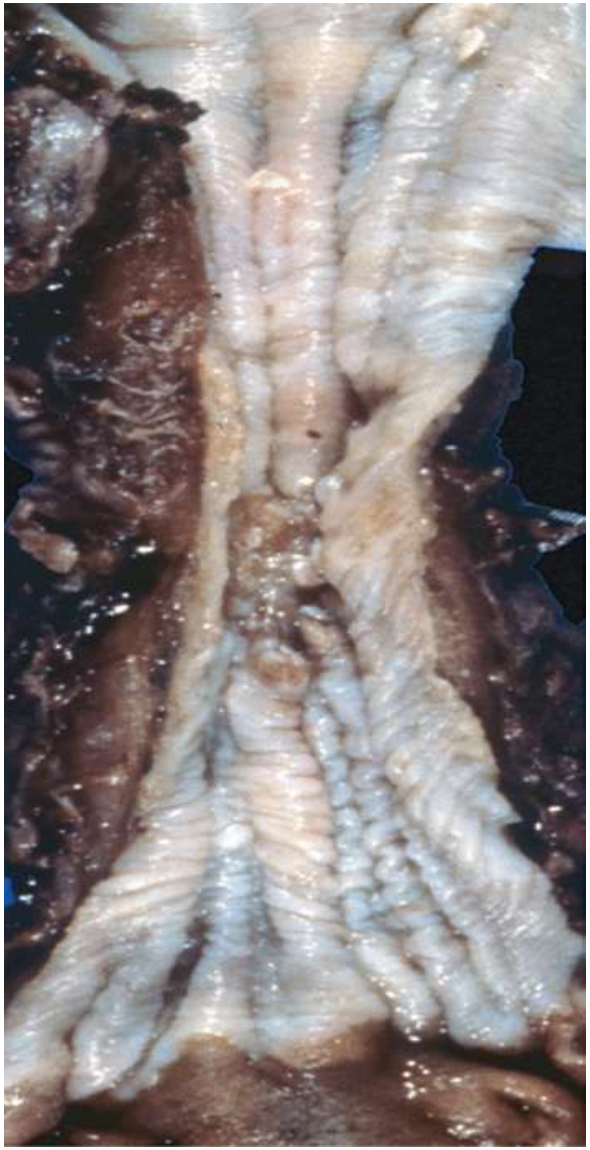
ESOPHAGEAL TUMORS

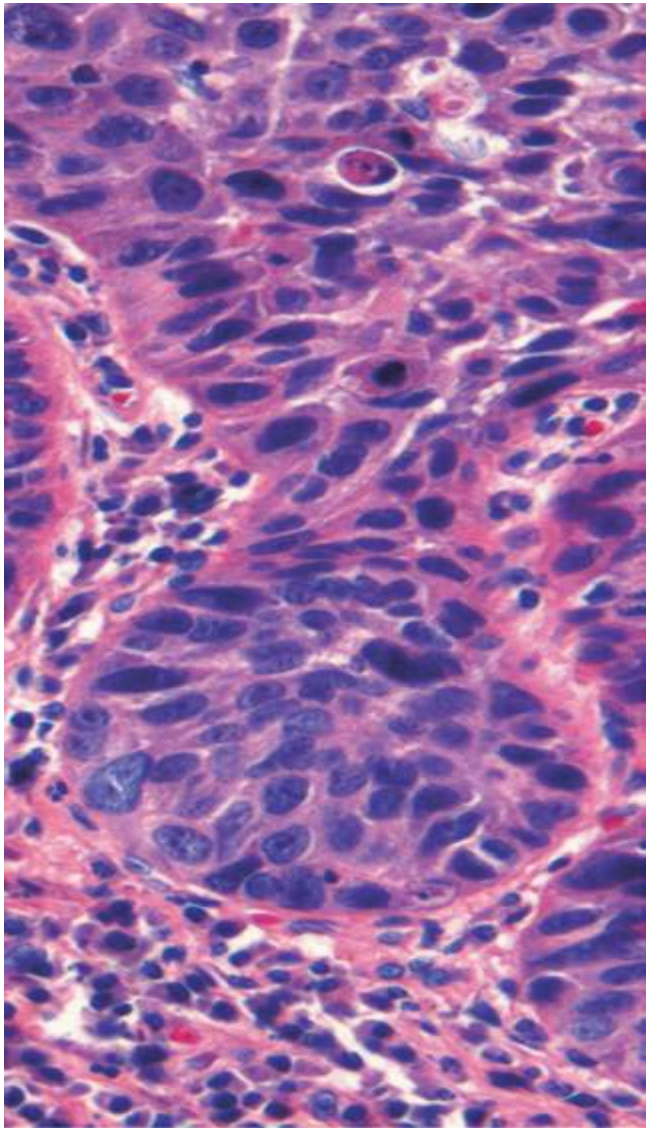
1- Esophageal adenocarcinoma.





Squamous cell carcinoma composed of nests of malignant cells that partially recapitulate the stratified organization of squamous epithelium.

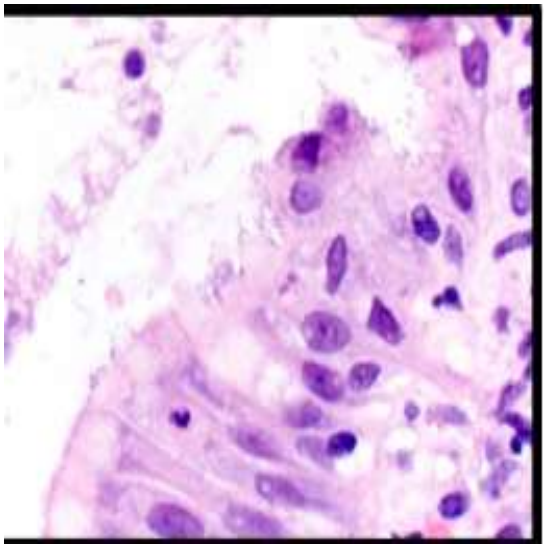


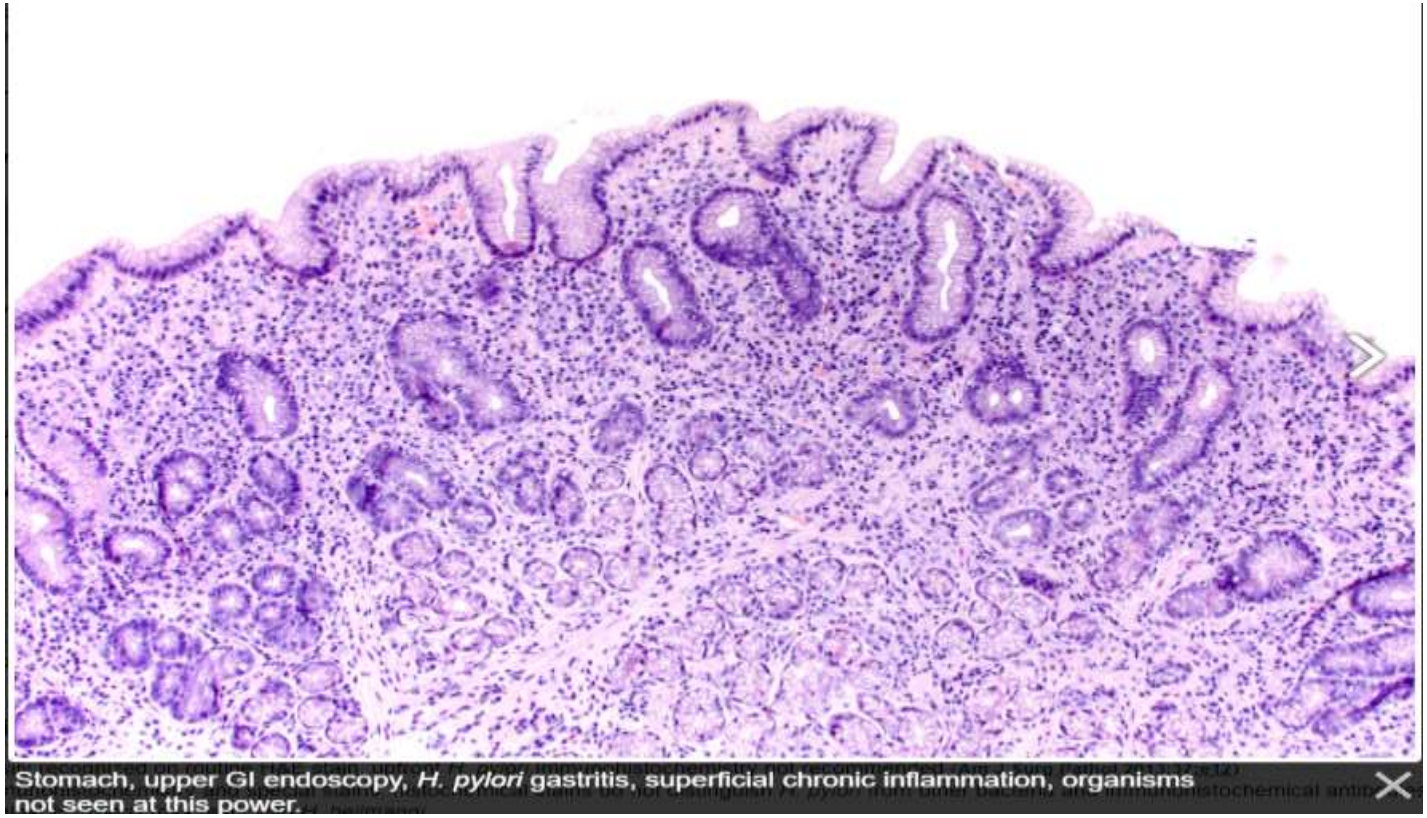


STOMACH

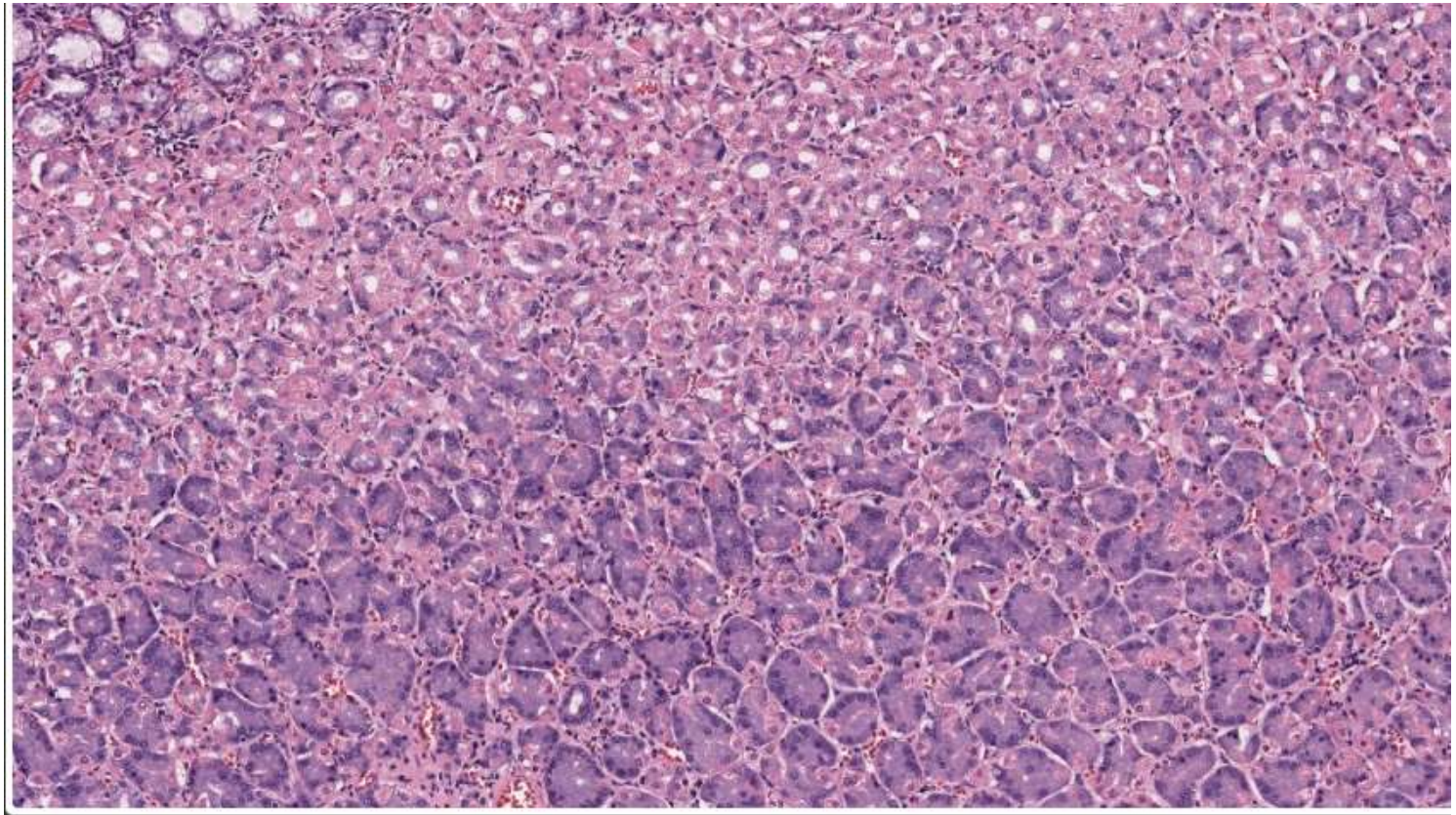
Chronic Gastritis

Helicobacter pylori *Gastritis*



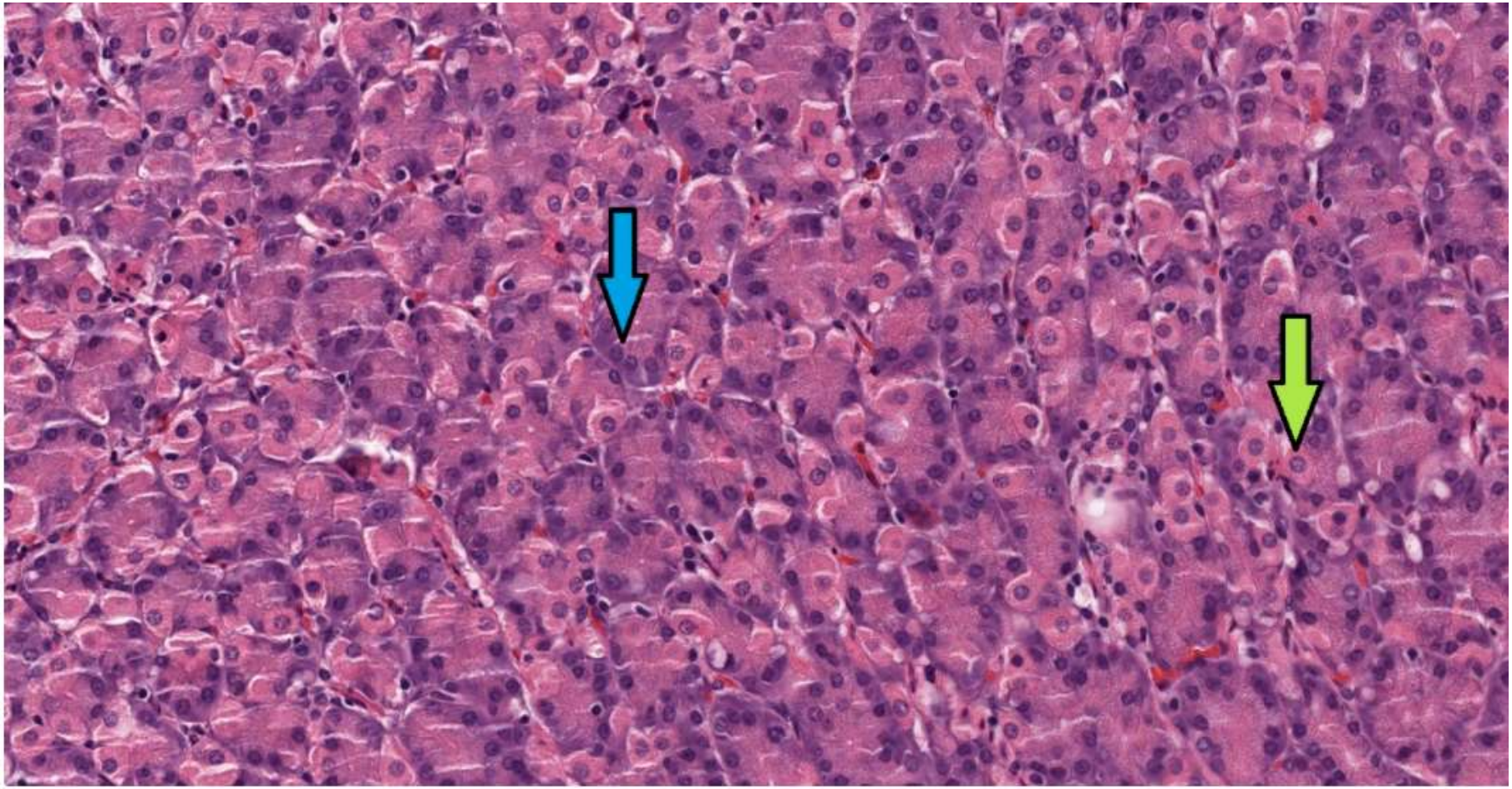


NORMAL BODY TYPE GASTRIC MUCOSA

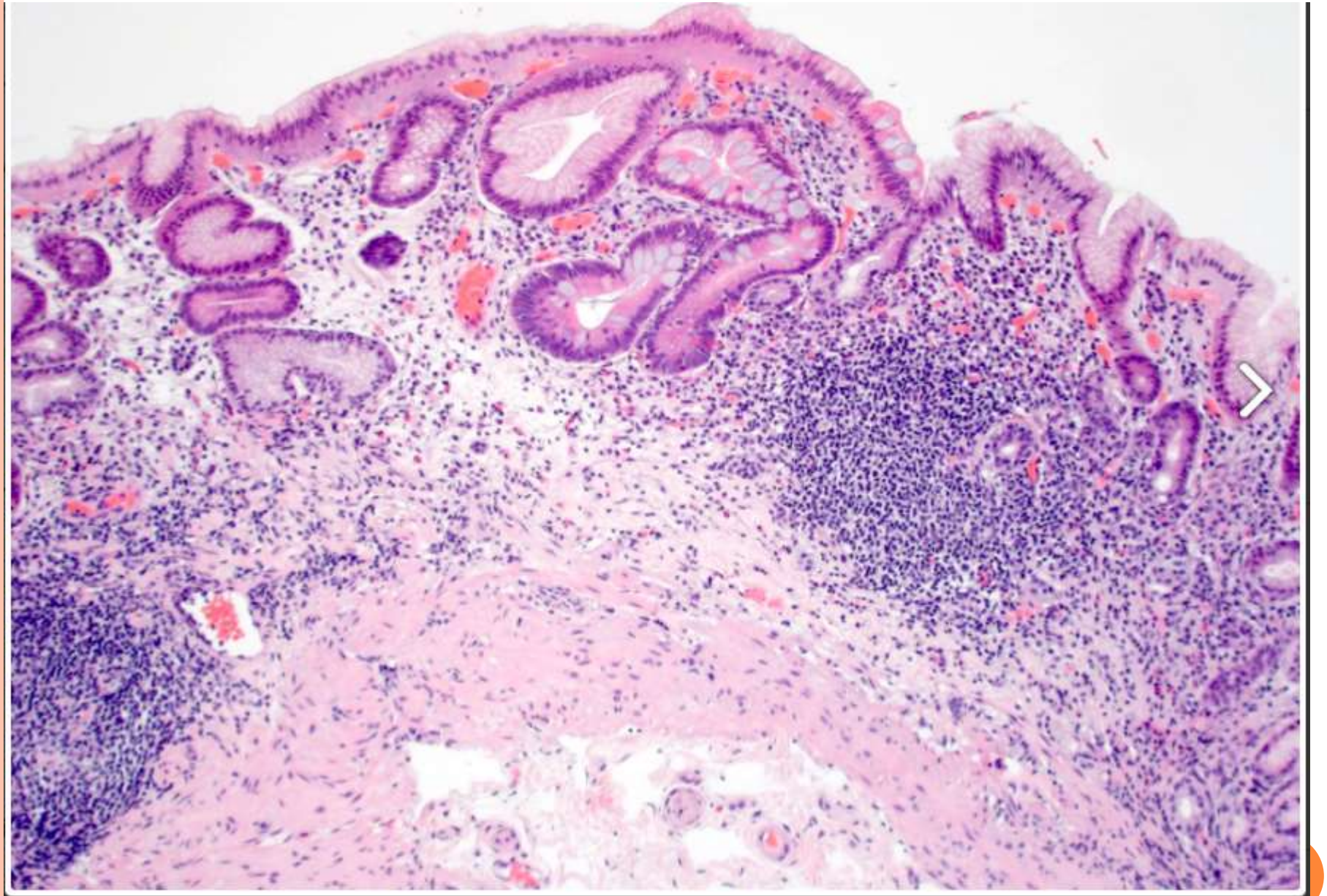


Oxyntic gastric mucosa with a tightly packed glandular component comprised of eosinophilic parietal cells and basophilic chief cells; note the predominance of parietal cells in the superficial glandular compartment and chief cells in the deep glandular compartment. The volume of the glandular mucosa far exceeds the volume of gastric pit (foveolar) mucosa.
Contributed by Kelsey E. McHugh, M.D.

NORMAL BODY TYPE GASTRIC MUCOSA



High power image of oxyntic mucosa containing relatively large parietal cells (green arrow) with abundant eosinophilic cytoplasm and centrally placed round nuclei admixed with cuboidal chief cells (blue arrow) with basophilic cytoplasm and more basally oriented round, regular nuclei



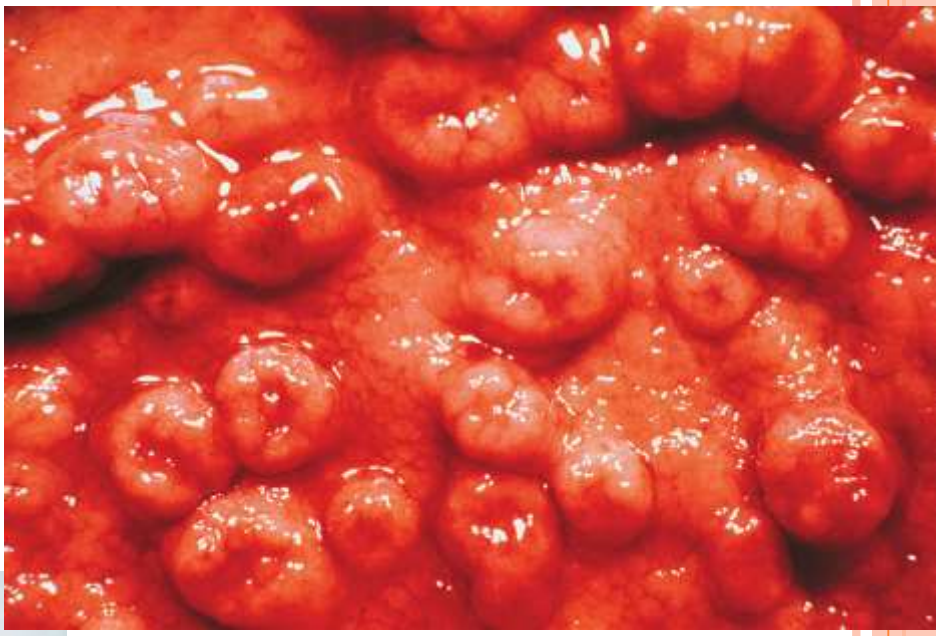
Gastric body in autoimmune gastritis. The body has become atrophic, as evidenced by antralization and intestinal metaplasia.

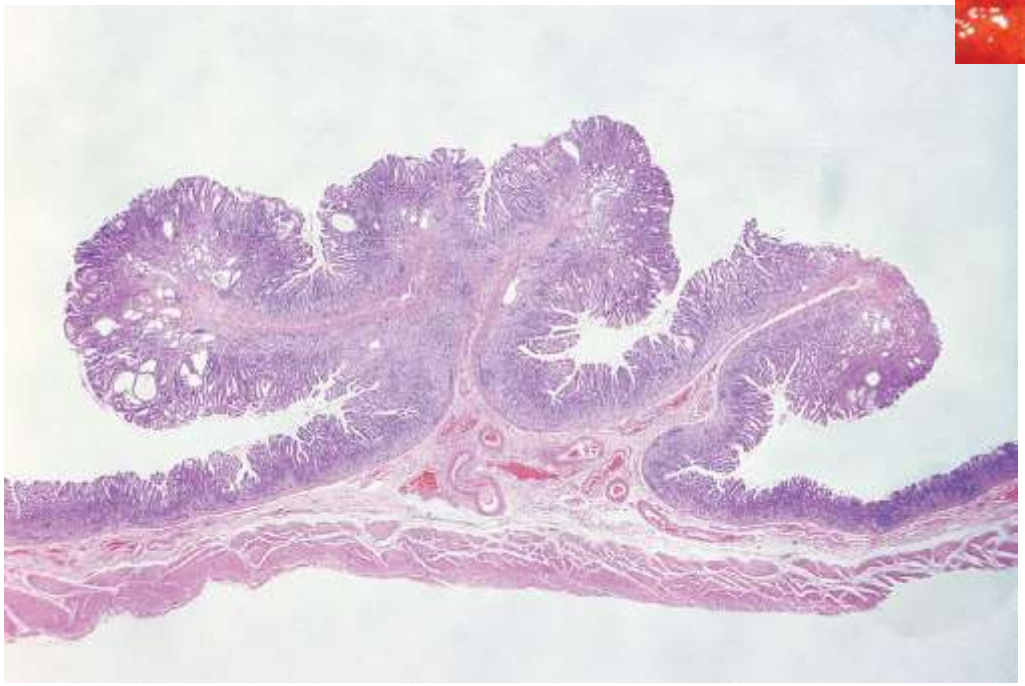
Fundic Gland Polyps.

INflammatory and Hyperplastic Polyps.

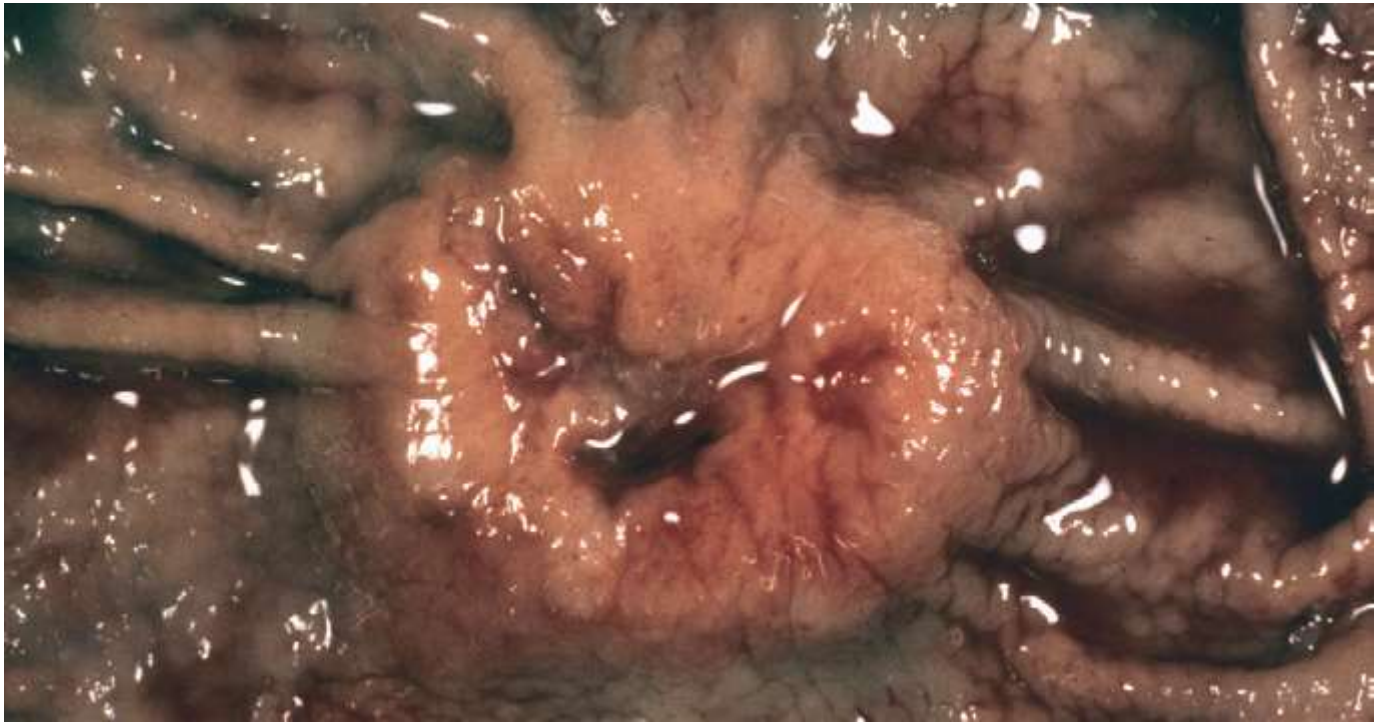
Gastric adenomas

MORPHOLOGY

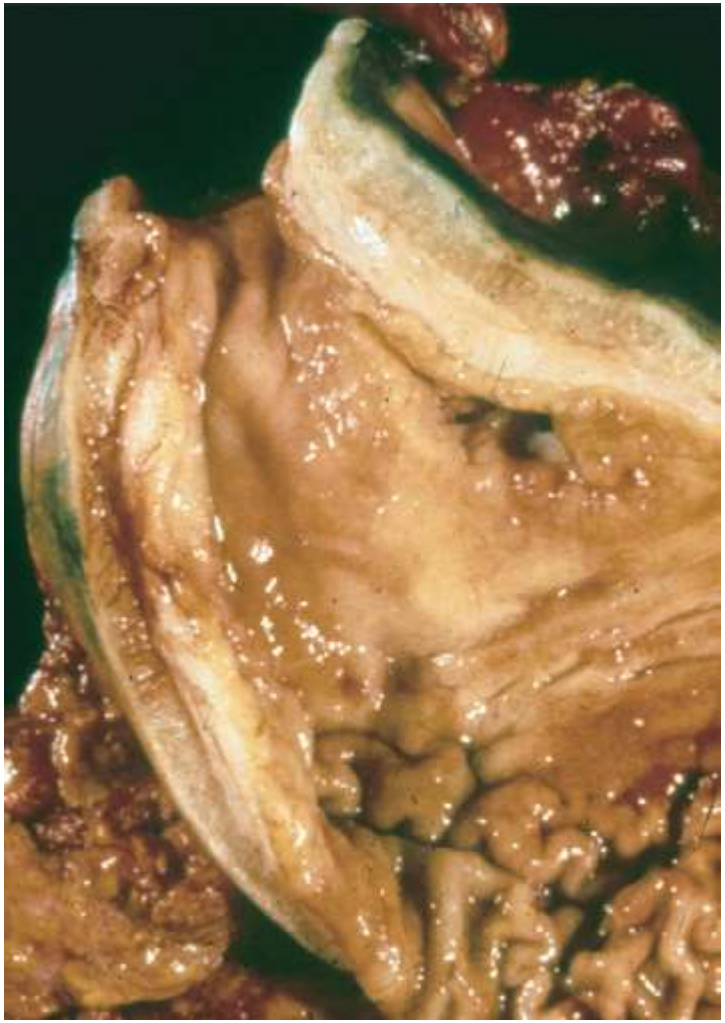


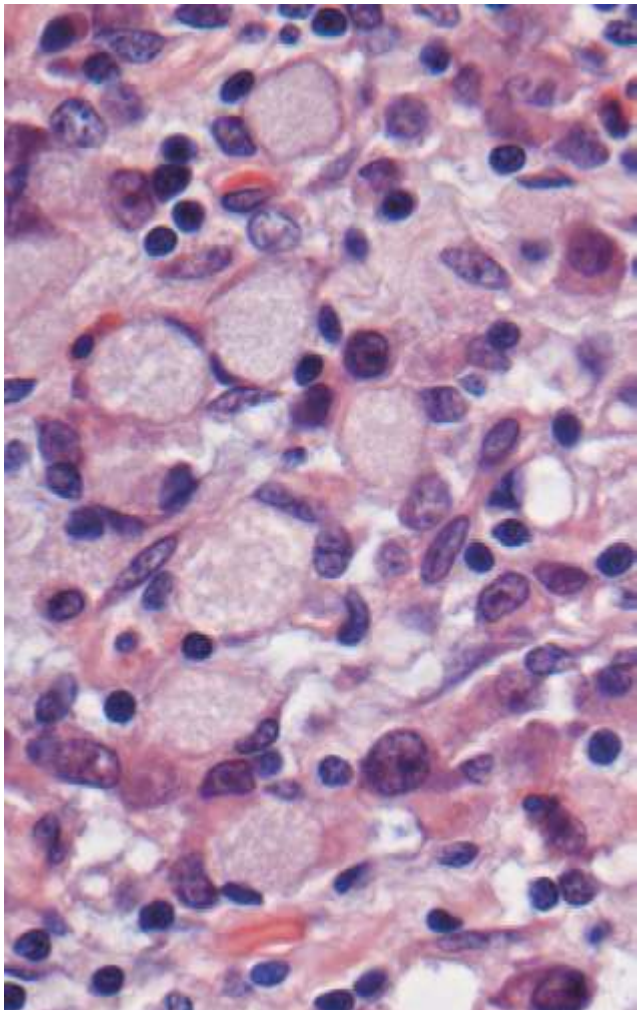


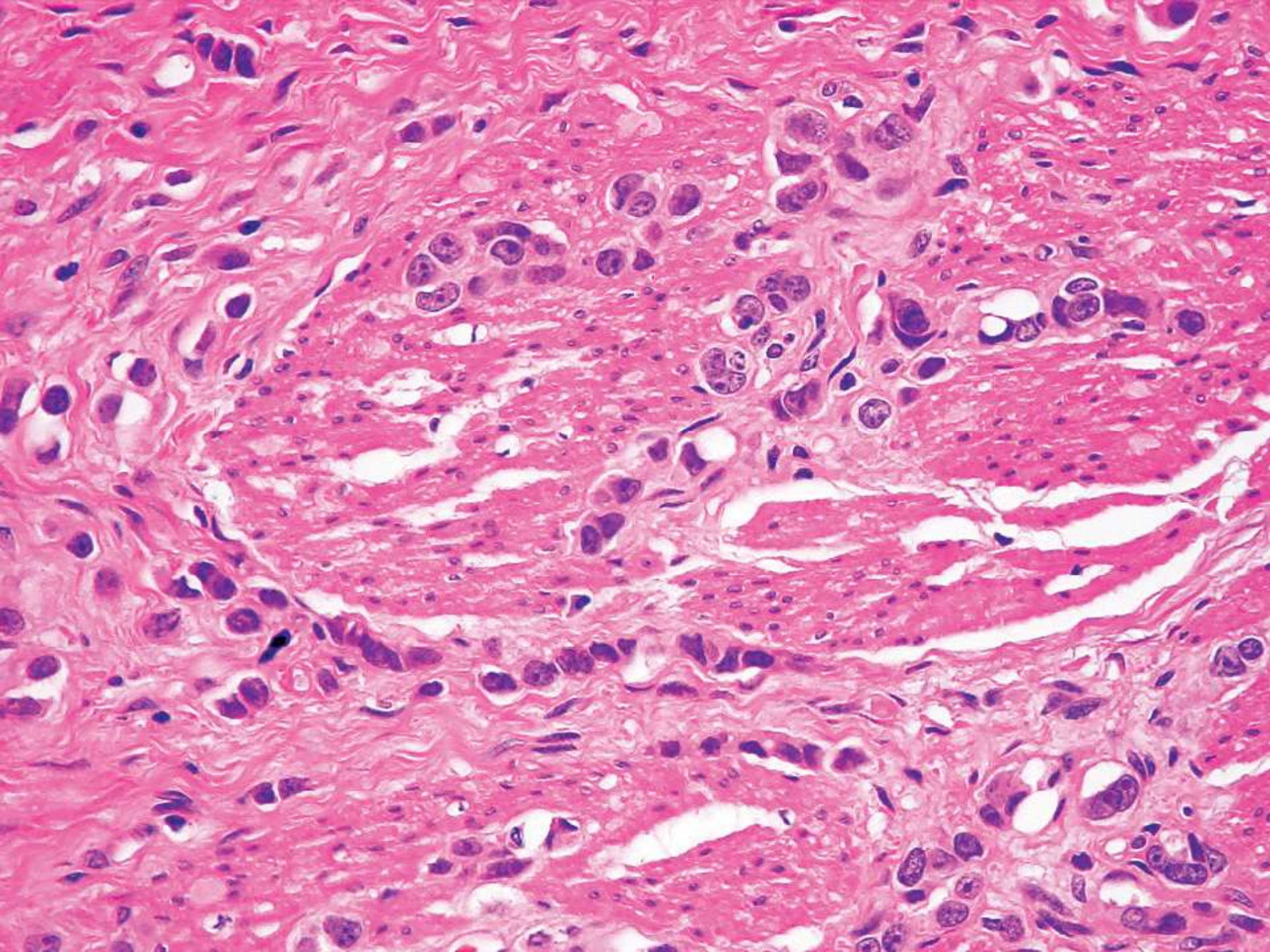
Intestinal-type adenocarcinoma consisting of an elevated mass with heaped-up borders and central ulceration



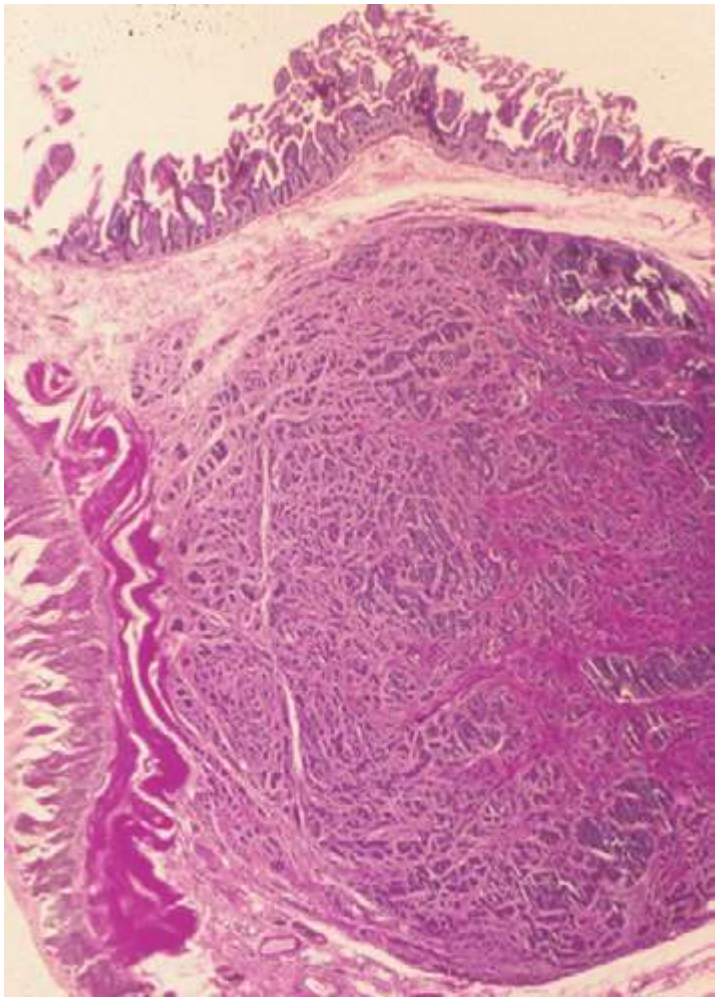
A, Linitis plastica. The gastric wall is markedly thickened, and rugal folds are partially lost. B, Signet ring cells with large cytoplasmic mucin vacuoles and peripherally displaced, crescent-shaped nuclei.

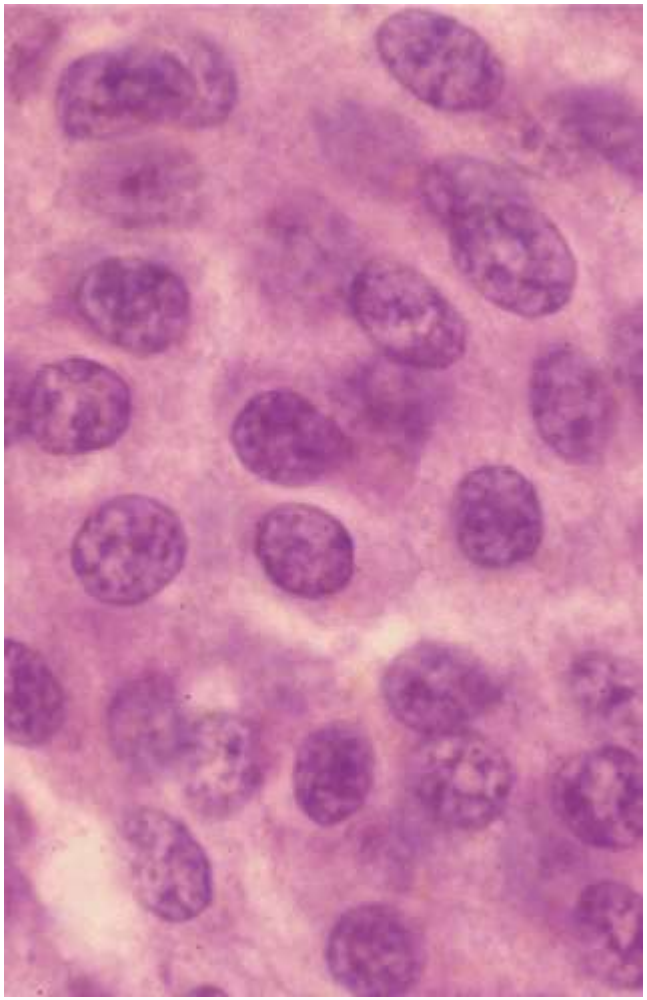






Gastrointestinal carcinoid tumor (neuroendocrine tumor). **A,** Carcinoid tumors often form a submucosal. B. Shows the bland cytology that typifies carcinoid tumors. The chromatin texture, with fine and coarse clumps, frequently assumes a **"salt and pepper" pattern**.
aggressive.



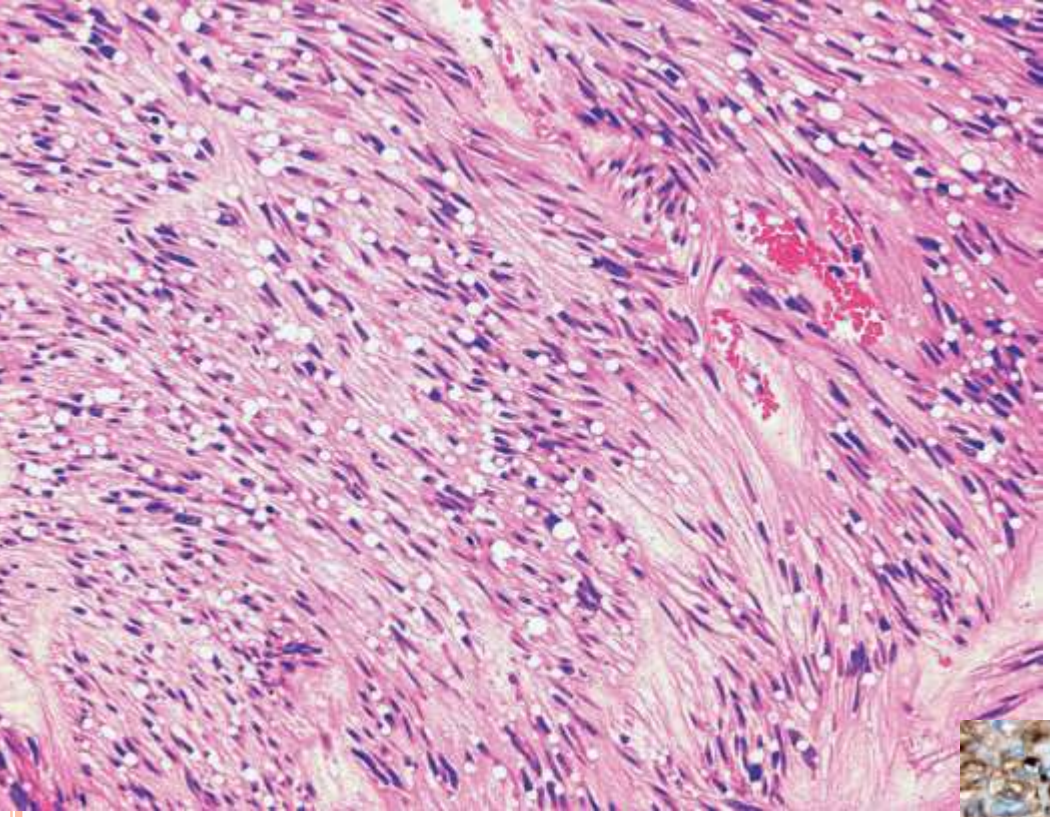


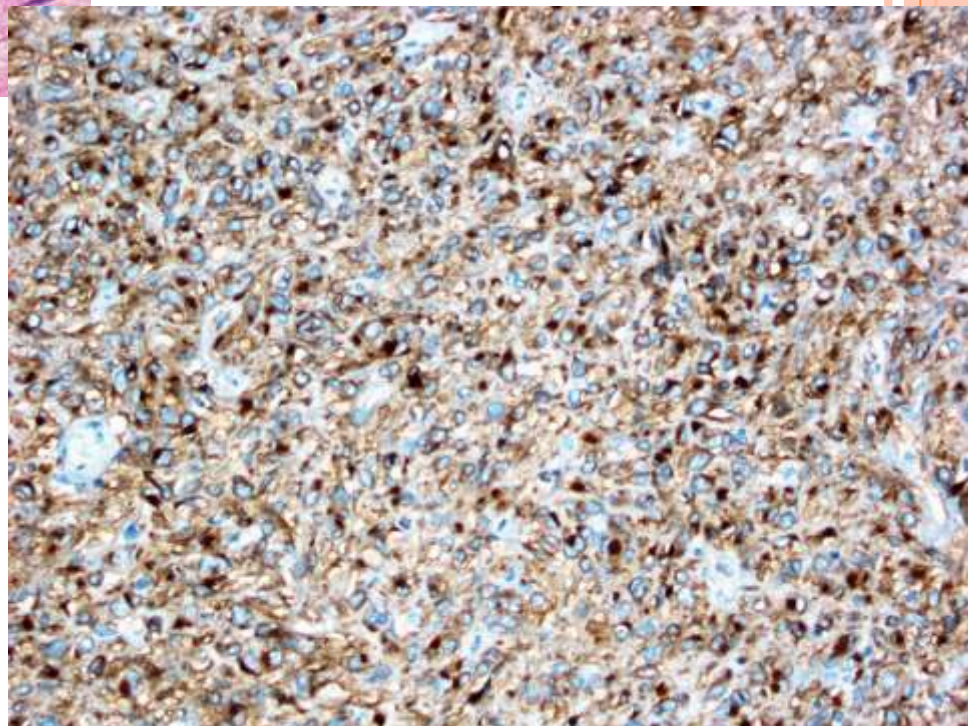
Gastrointestinal Stromal Tumor

- The most common mesenchymal tumor of in the stomach.

- Overall, GISTs are slightly more common in males. The peak incidence of gastric GIST is around 60 years of age,with less than 10% occurring in persons younger than 40 years of age.

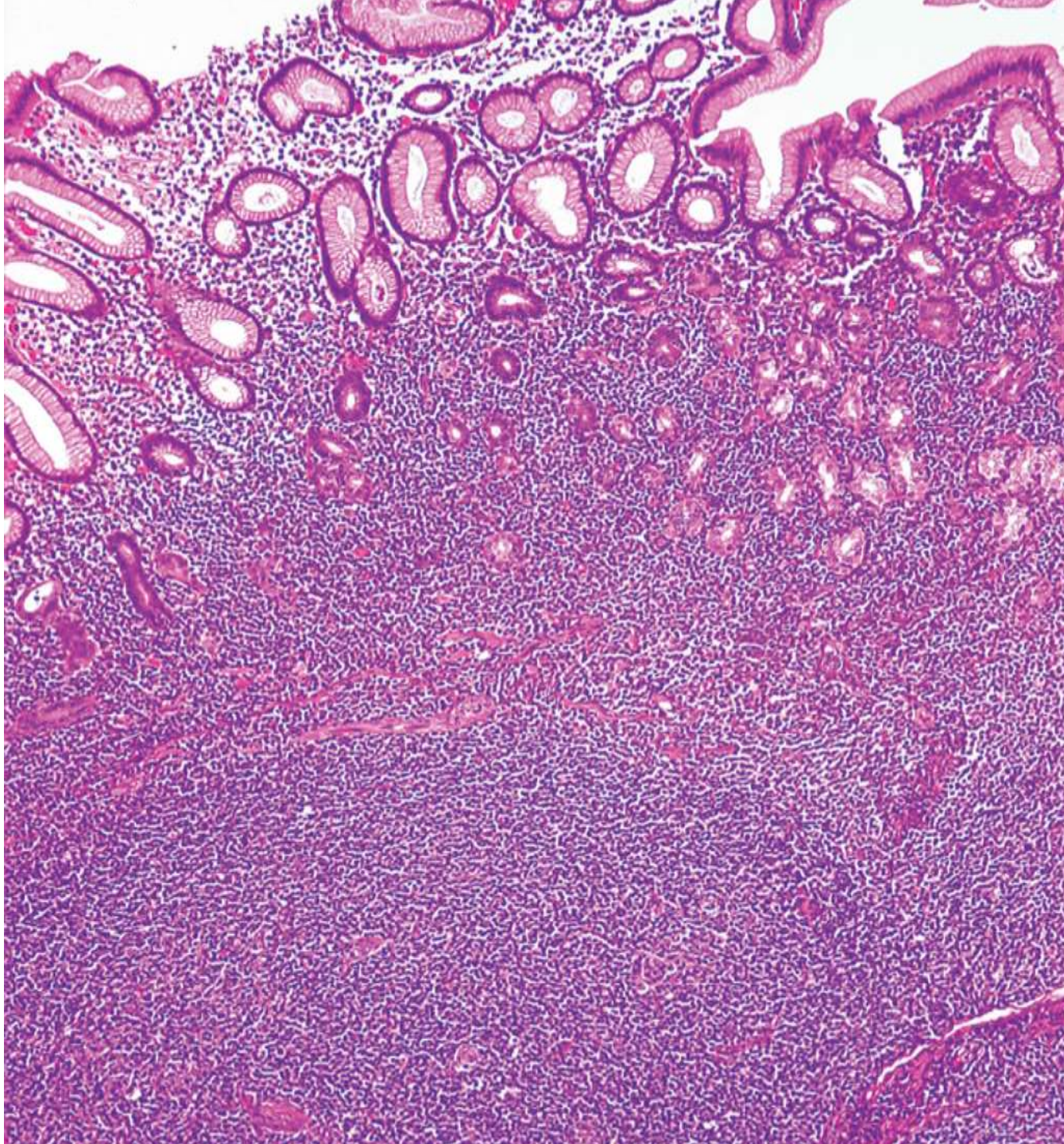
- Approximately **75% to 80% of all GISTs have oncogenic, gain-of-function mutations of the gene encoding the tyrosine kinase c-KIT,**





Diffuse CD117 (KIT) immunoreactivity

MALTOMA



GOOD LUCK IN YOUR EXAM