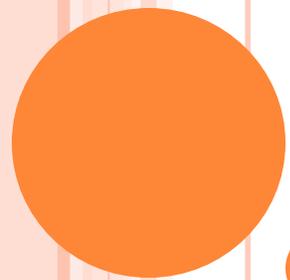




**GASTROINTESTINAL TRACT LAB 1+2
SECOND YEAR.**

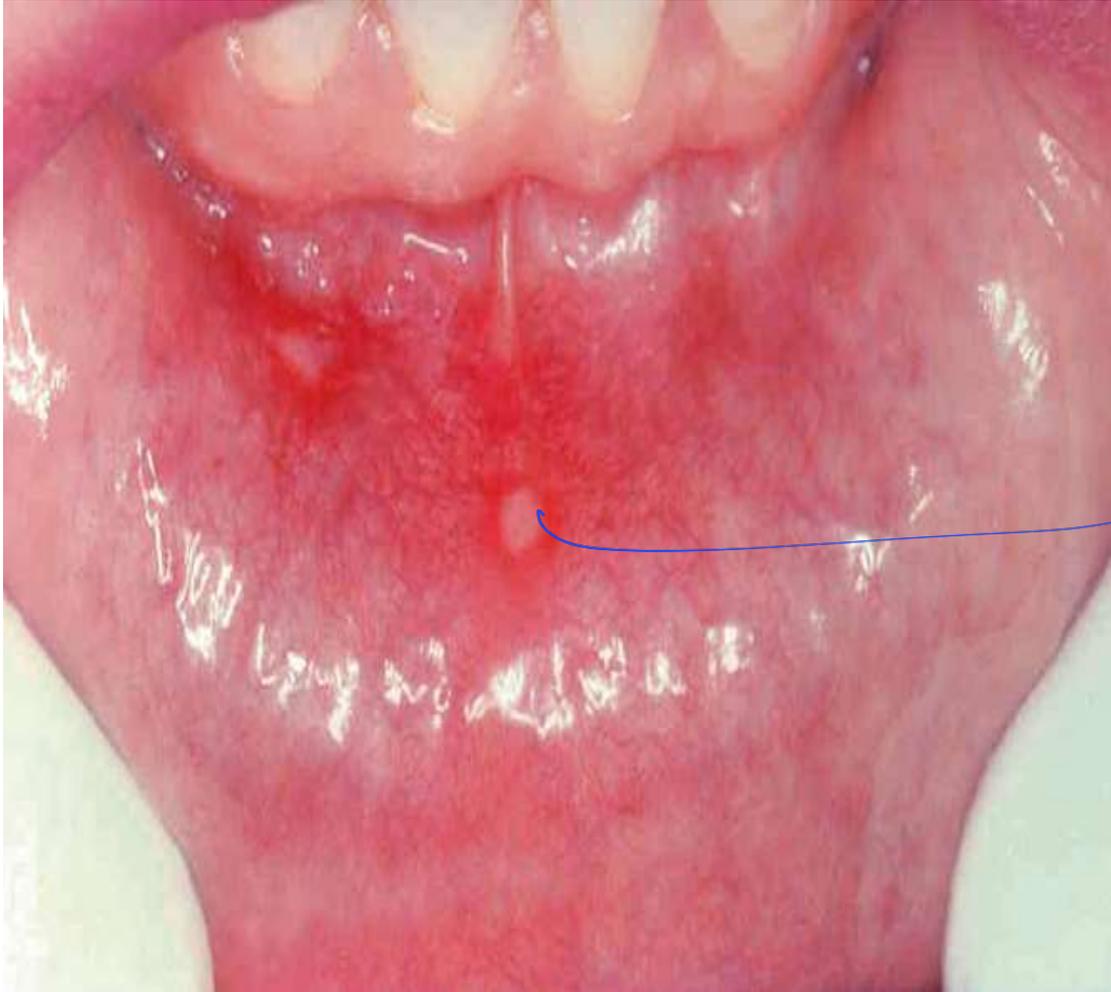
DR. Bushra Al-Tarawneh, MD



ORAL CAVITY

ORAL INFLAMMATORY LESIONS.

- Aphthous Ulcers (Canker Sores) - Common disease
 - associated w/ IBD + Behcet disease
 - disappear in 7-10 days



slightly elevated
+
Erythema.



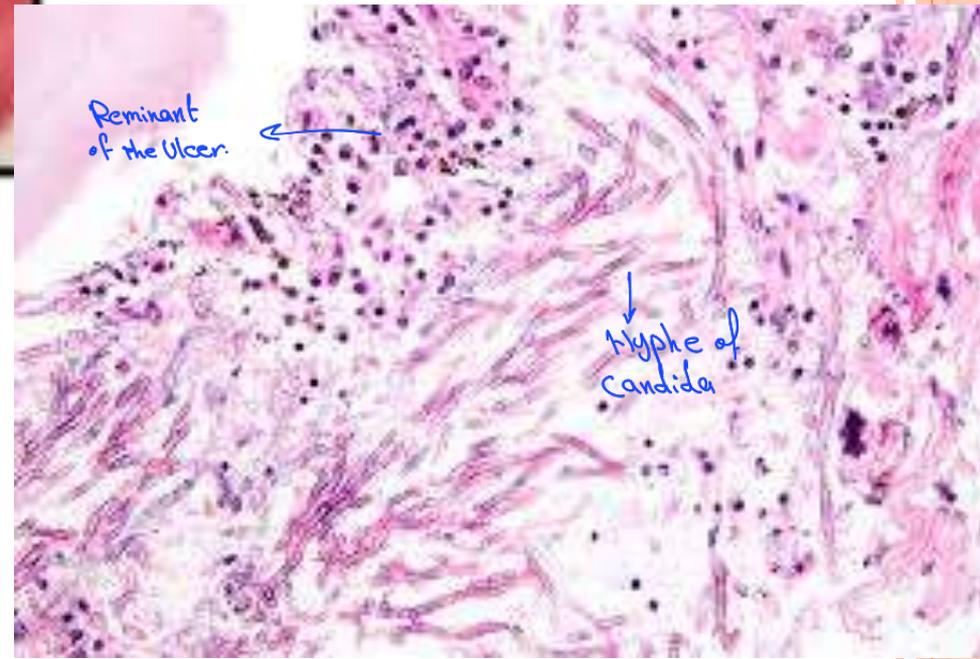
ORAL CANDIDIASIS (THRUSH) .. Fungal infection.

- Depend on the Immunity of the Host.
- can affect any part of the GI.

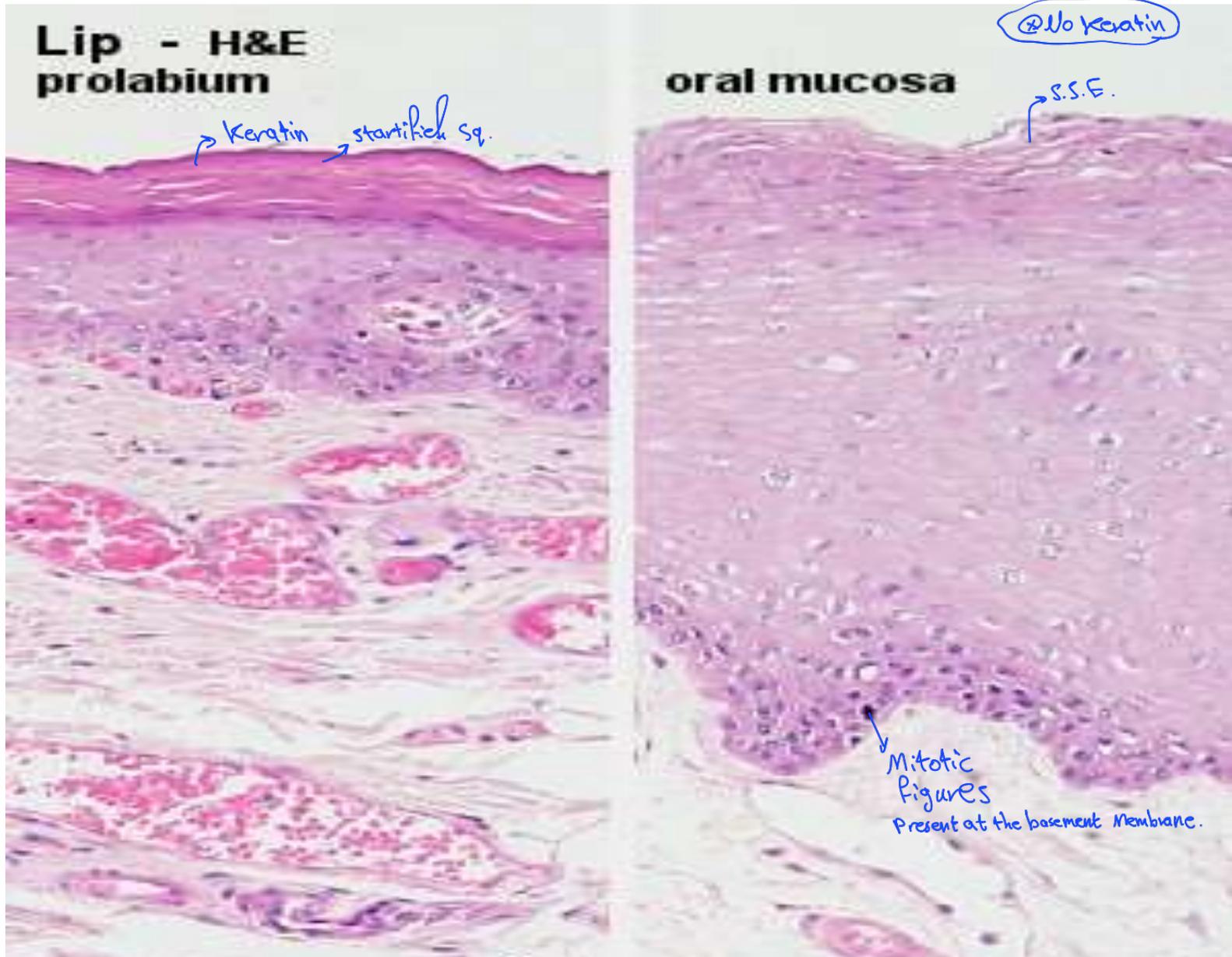


White patches
filled w/ fibrin
exudate.

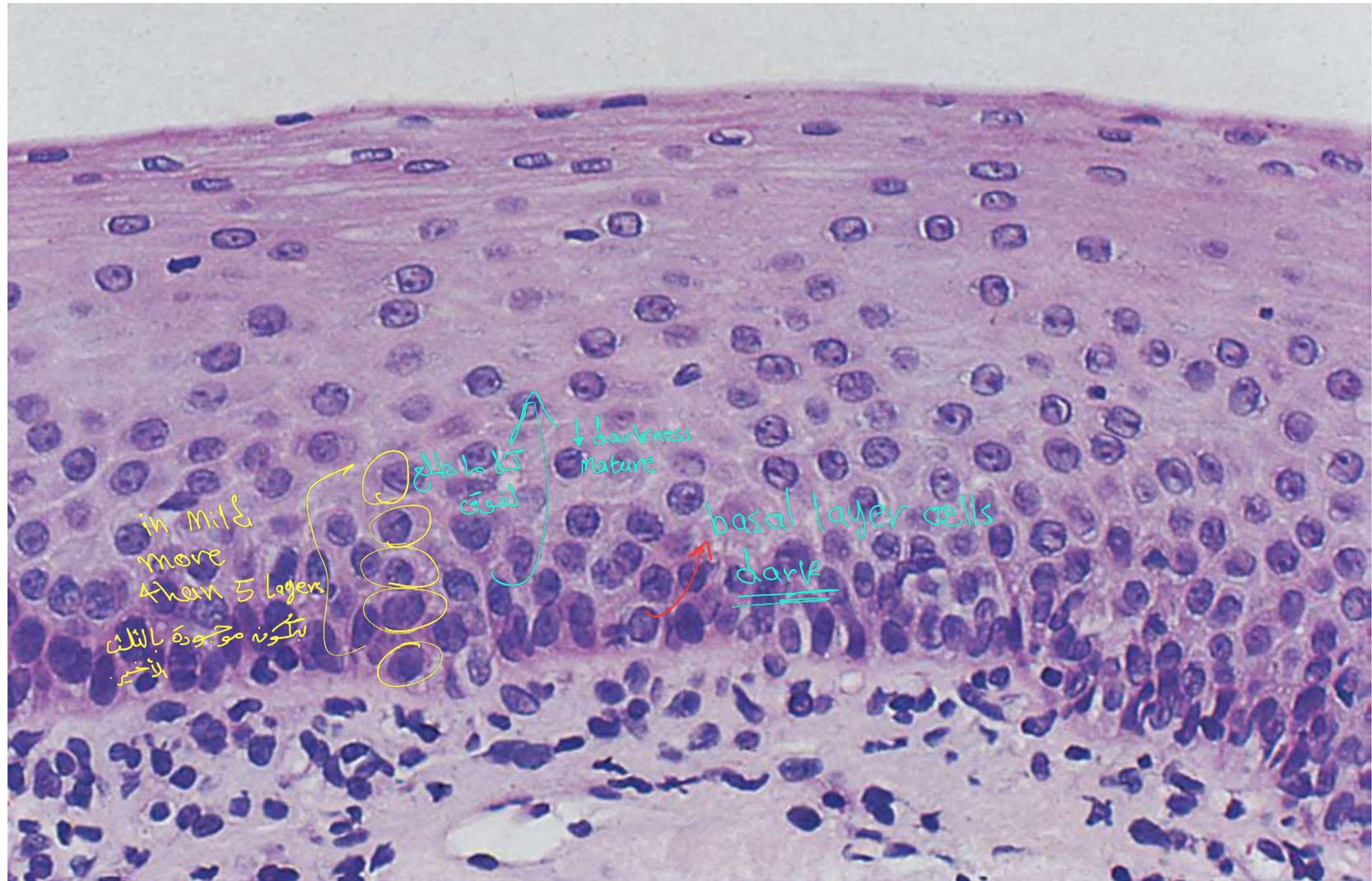
"HE stain"



Normal histology of the oral mucosa.



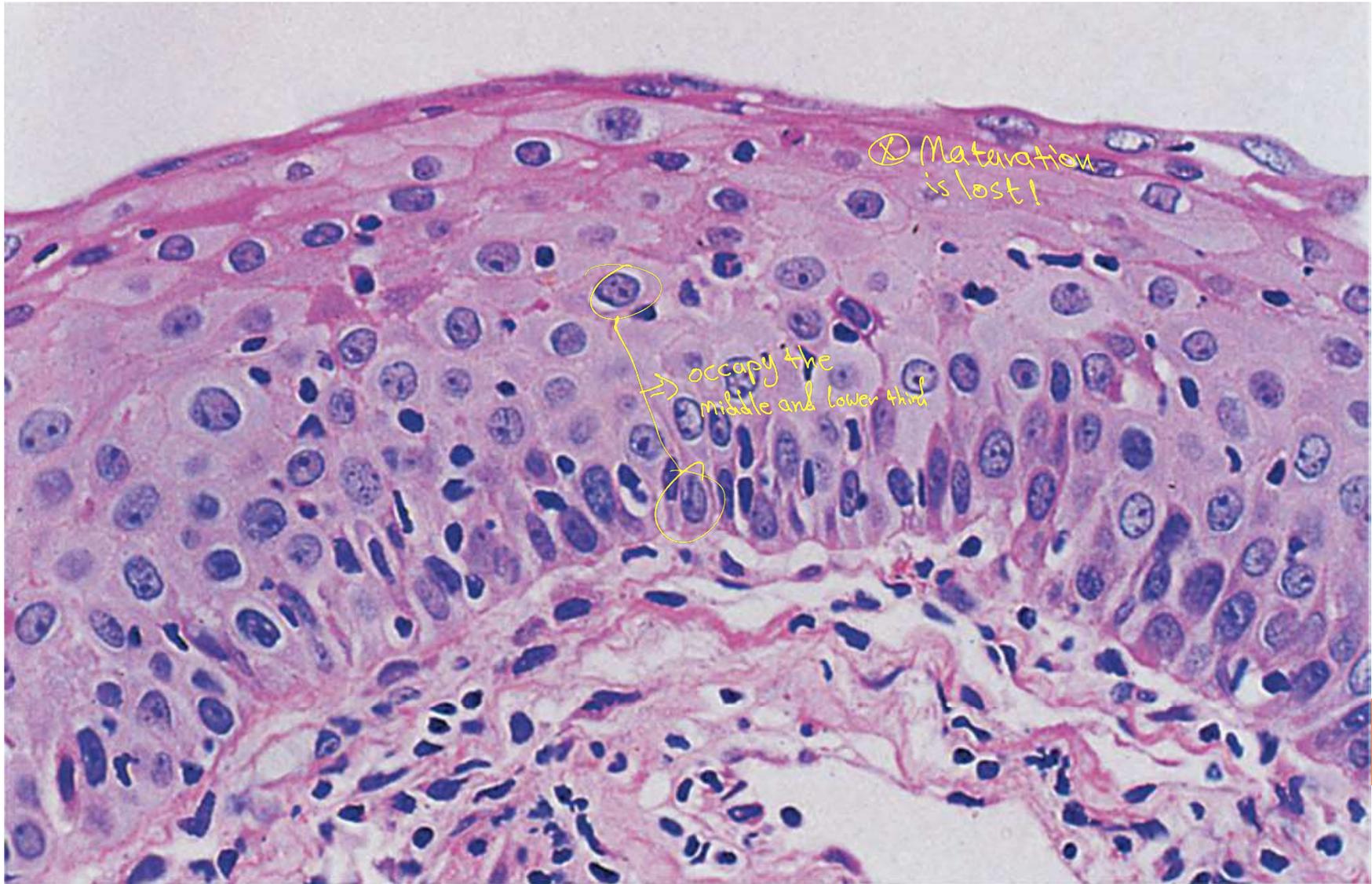
MILD DYSPLASIA



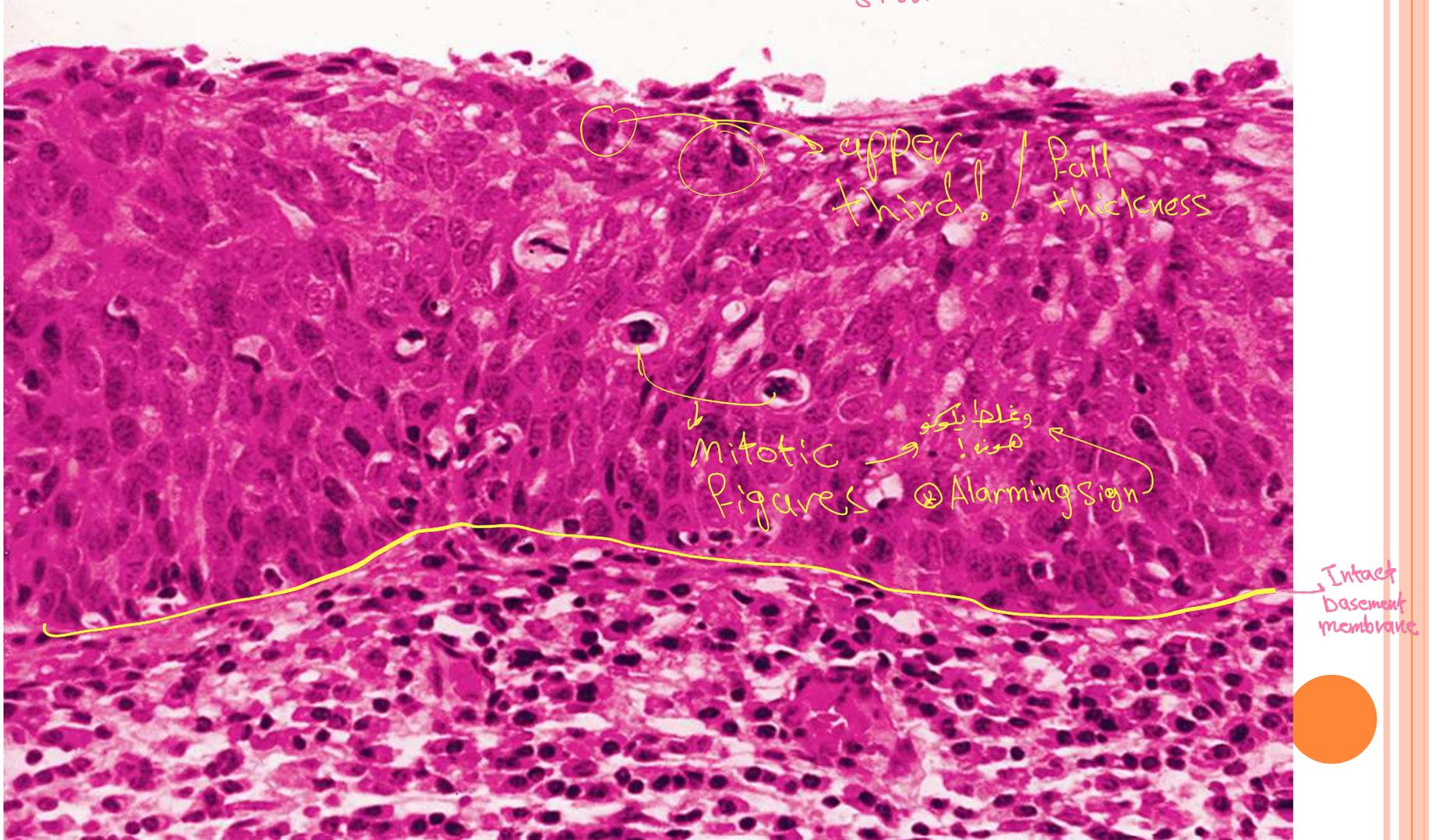
Basement membrane of Epidermis



MODERATE DYSPLASIA.



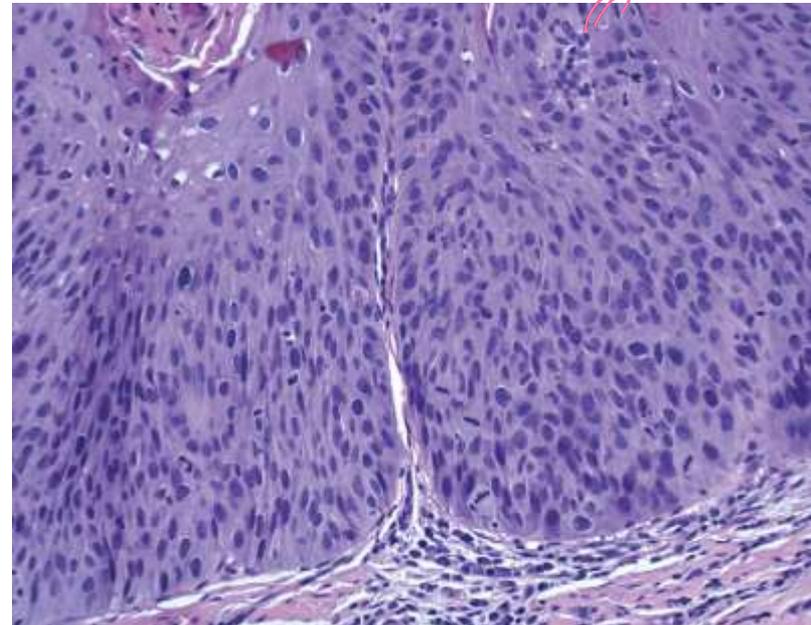
SEVERE DYSPLASIA = CIS \Rightarrow Carcinoma in situ



LEUKOPLAKIA

⊗ Can't be associated w/ other diseases!

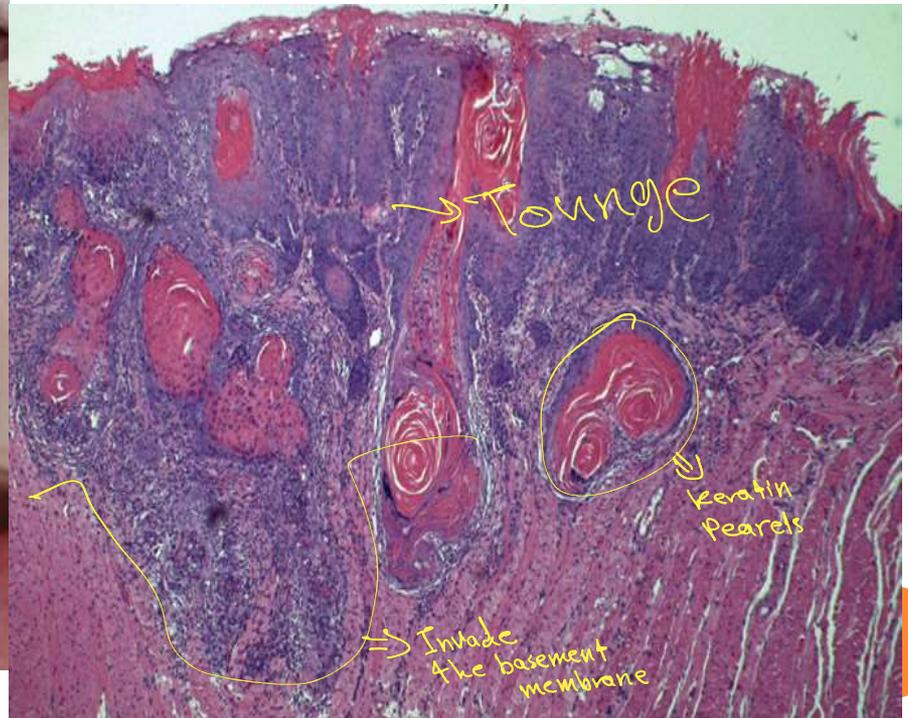
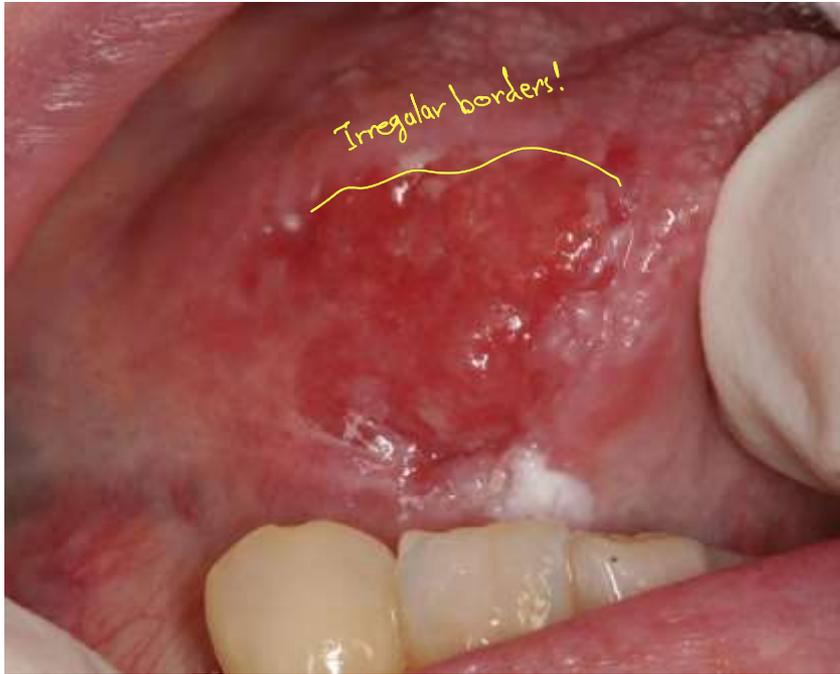
the lesion is smooth with well-demarcated borders and minimal elevation. B, Histologic appearance of leukoplakia showing dysplasia, characterized by nuclear and cellular pleomorphism and loss of normal maturation.



→ Mitotic Figures!

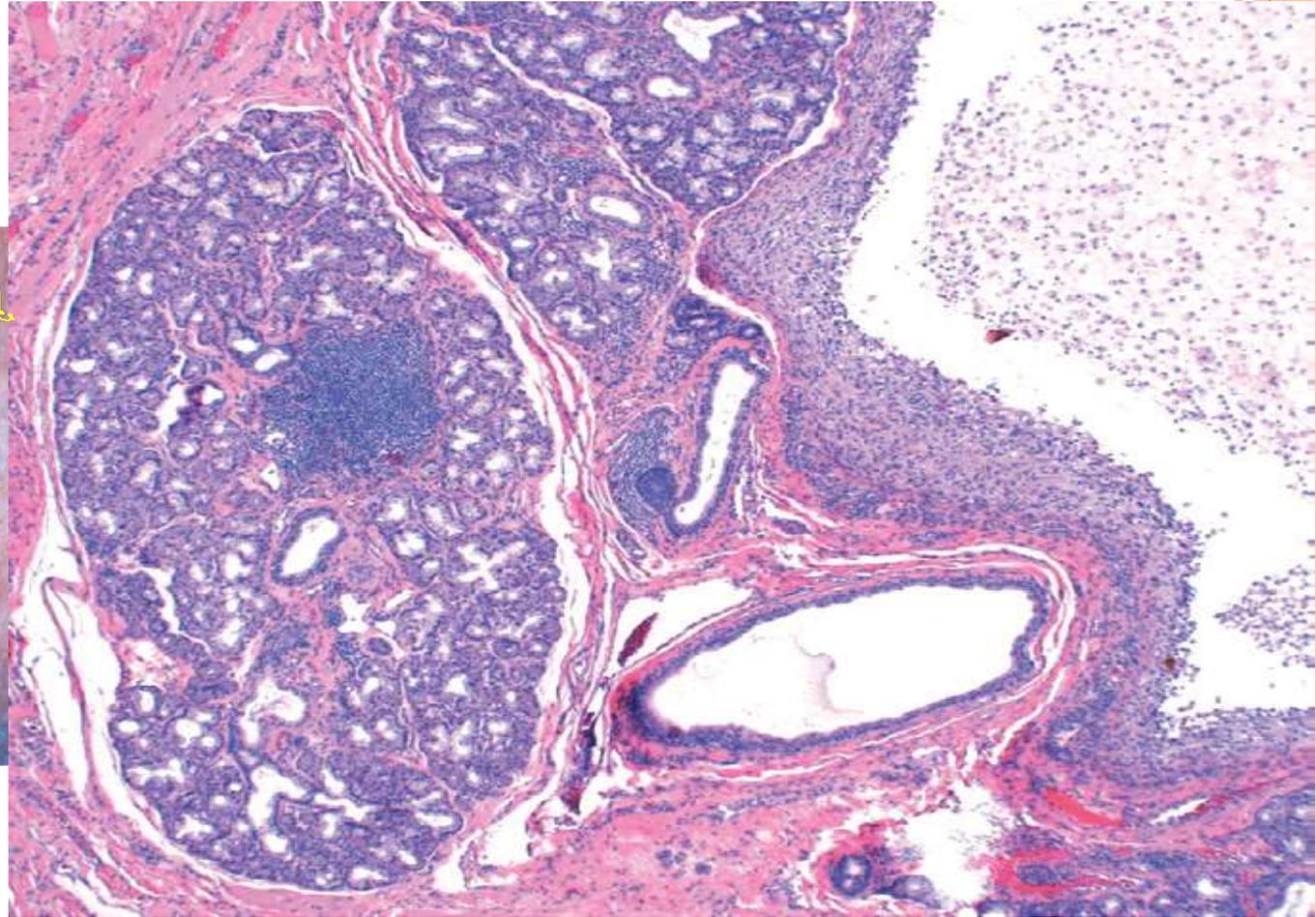
Moderate to Severe dysplasia!

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

Most common
benign condition of
lower lip!



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%)

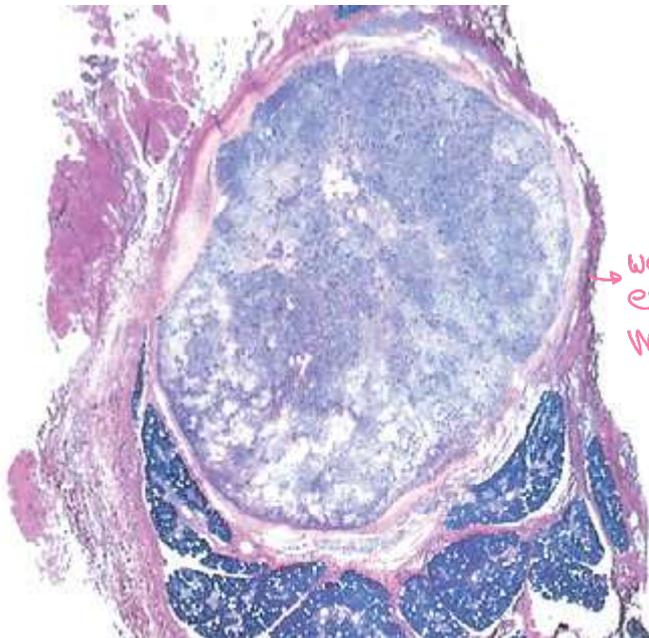
NOS, not otherwise specified.

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

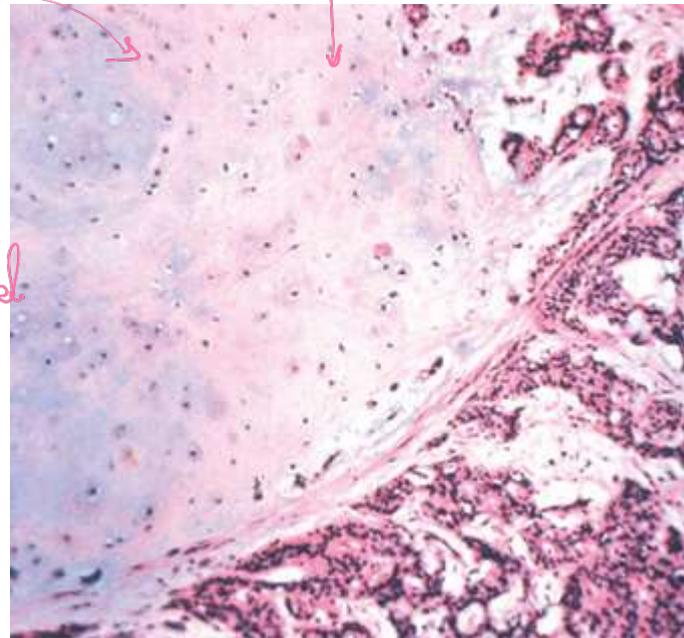


PLEOMORPHIC ADENOMA *"slowly growing" "mixed tumor"*

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

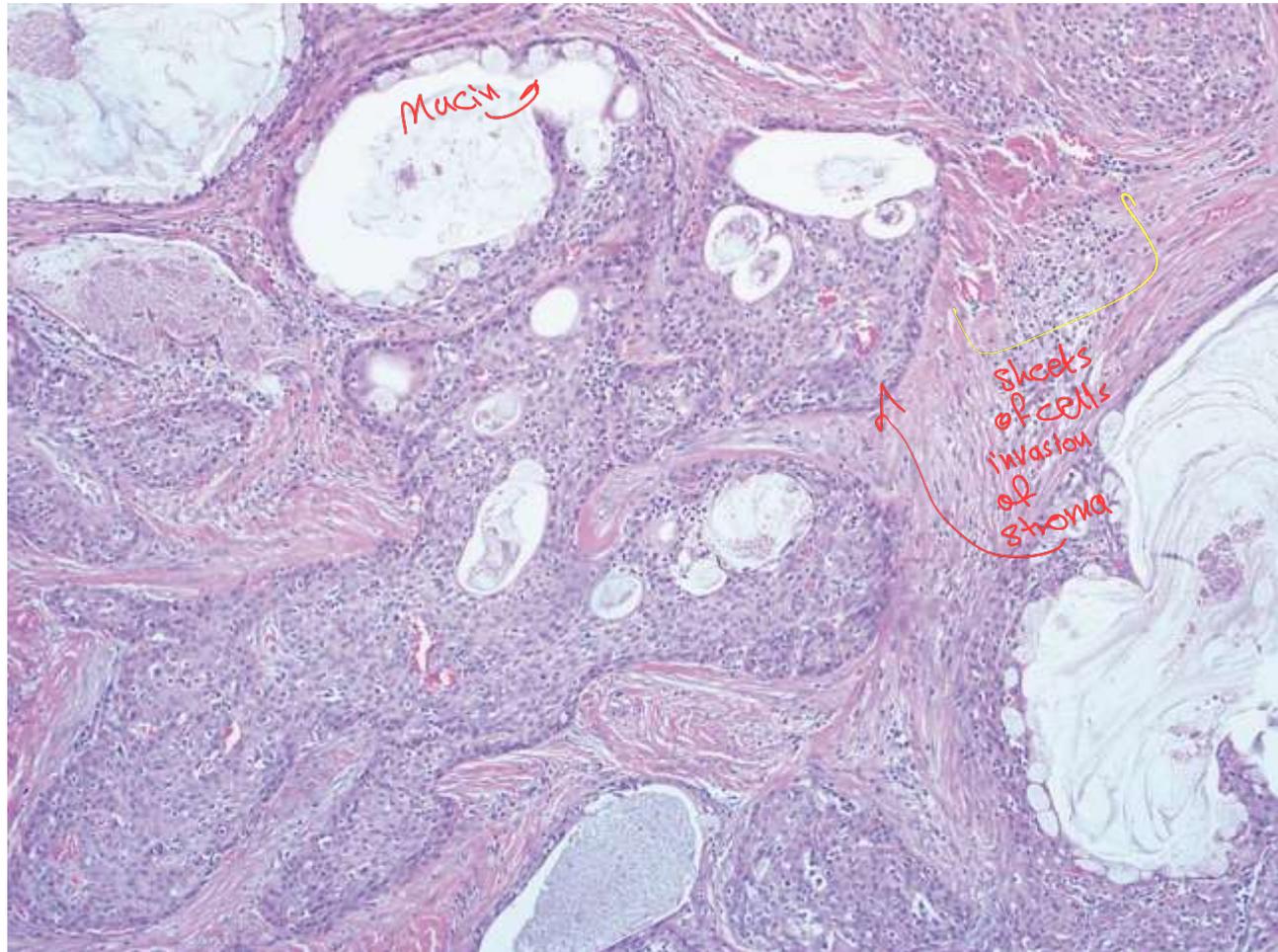


*well
encapsulated
mass*



→ Mucin cells → Squamous cells → Intermediate cells

MUCOEPIDERMOID CARCINOMA

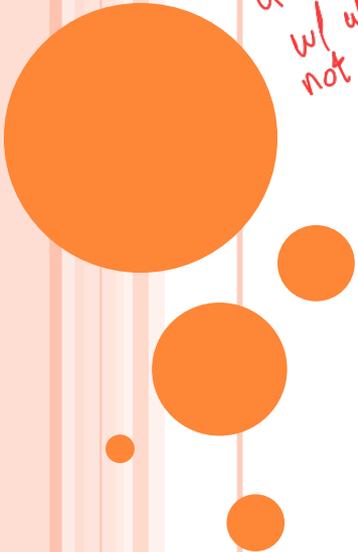


Q)

patient has taken immunocompromized therapy presented with whitish plaques that cover fibrin exudate, what is the possible underlying causative agent?

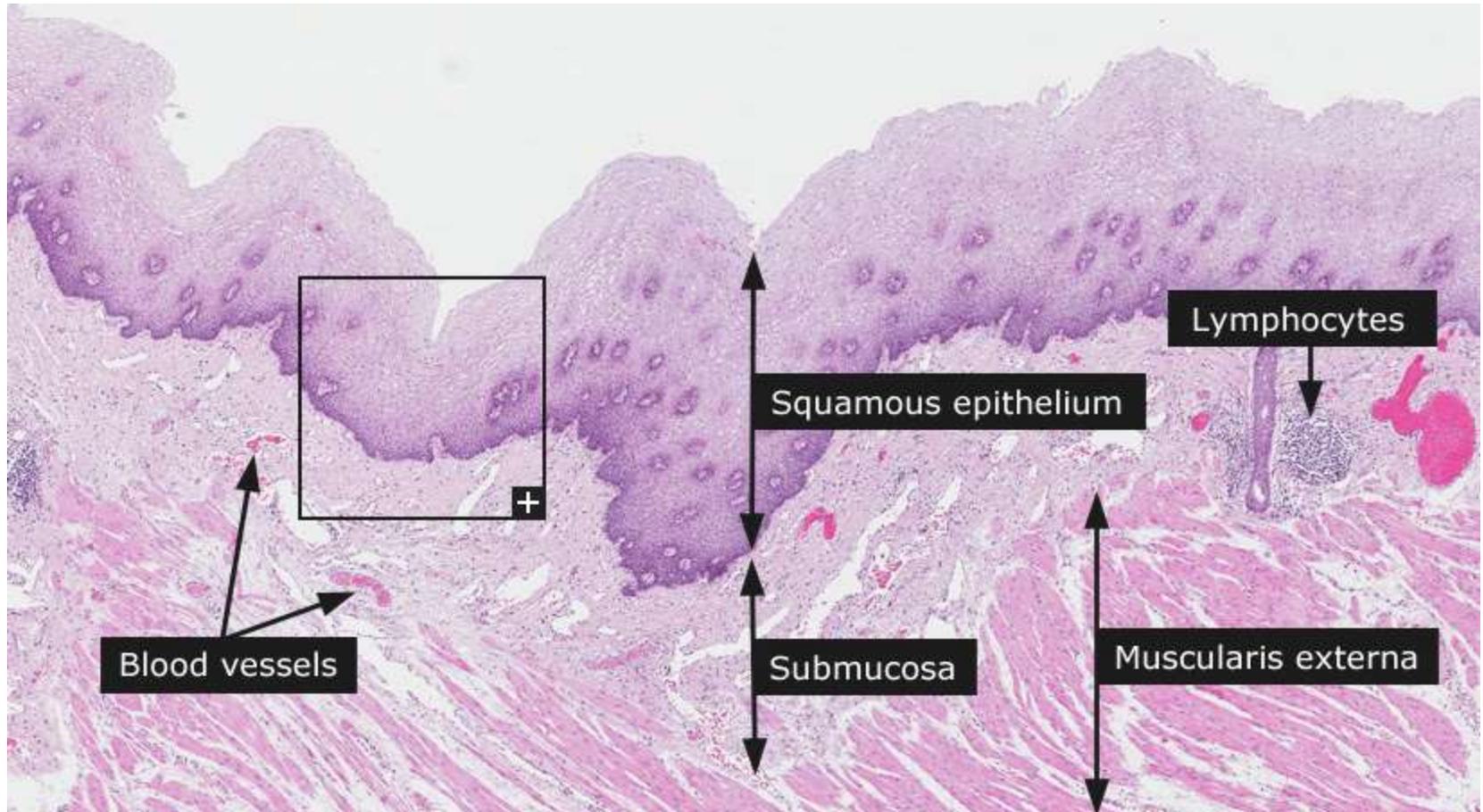
A) CMV B) HSV C) Candidiasis.

*associated
w/ ulcers
not white patches*



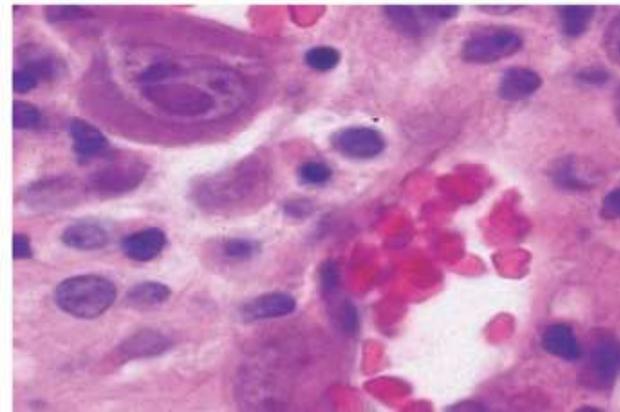
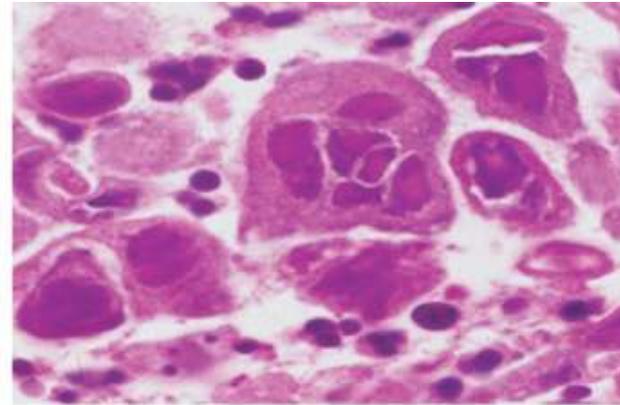
ESOPHAGUS

NORMAL HISTOLOGY OF THE ORAL MUCOSA.



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

glassy eosinophilic form.



HERPESVIRUSES ESOPHAGITIS

"Punched out ulcer"

→ biopsy? viral inclusions.



CANDIDIASIS



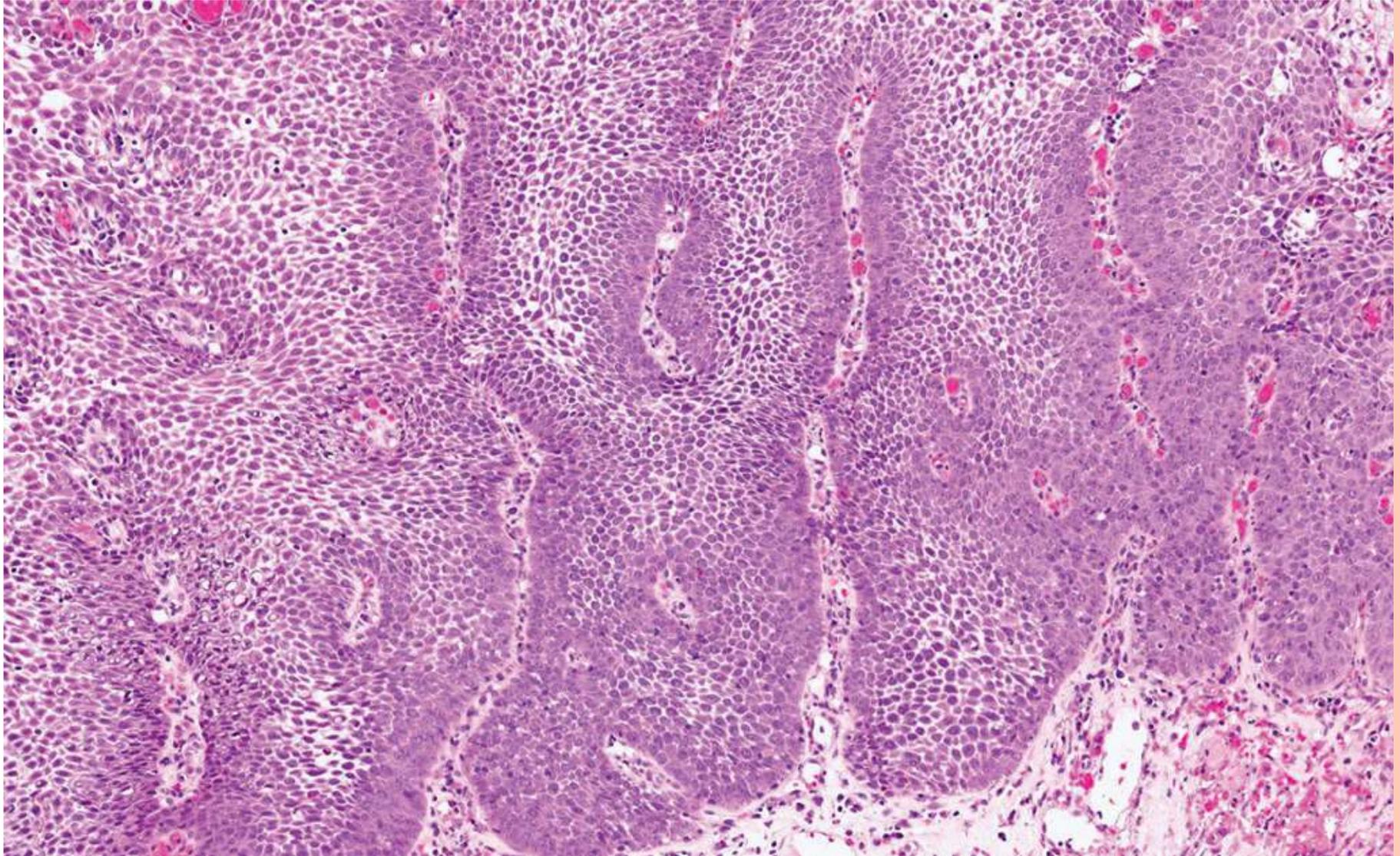
Reflux esophagitis

- 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.



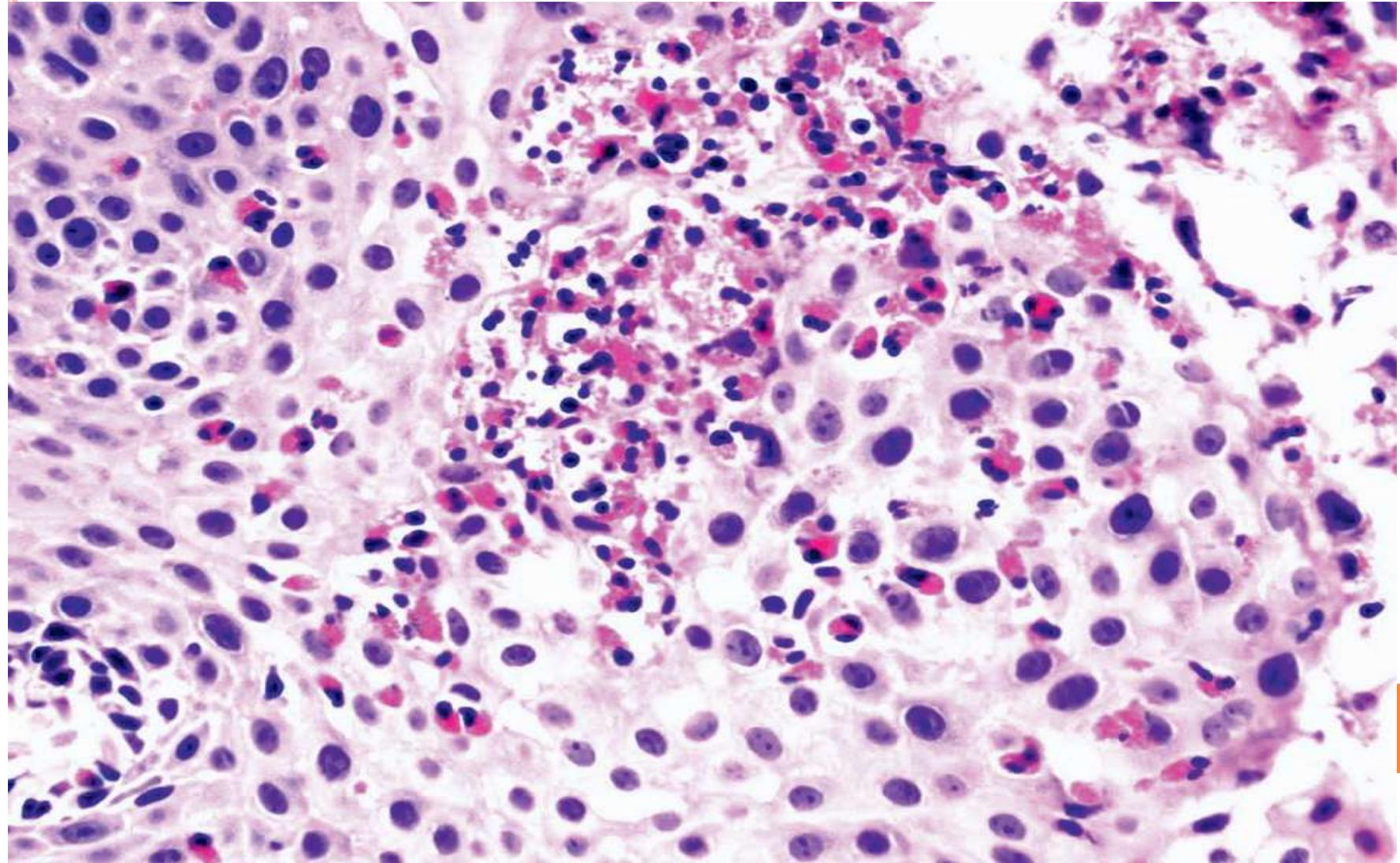
⊛ Elongation of papillae of lamina propria

⊛ basal cells go into hyperplasia and lymphocyte infiltration.

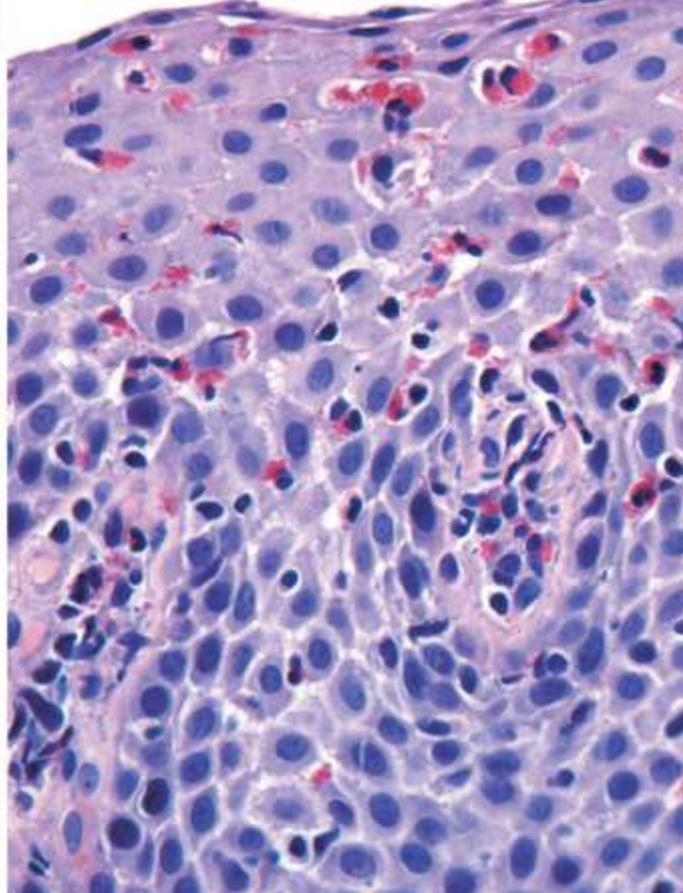
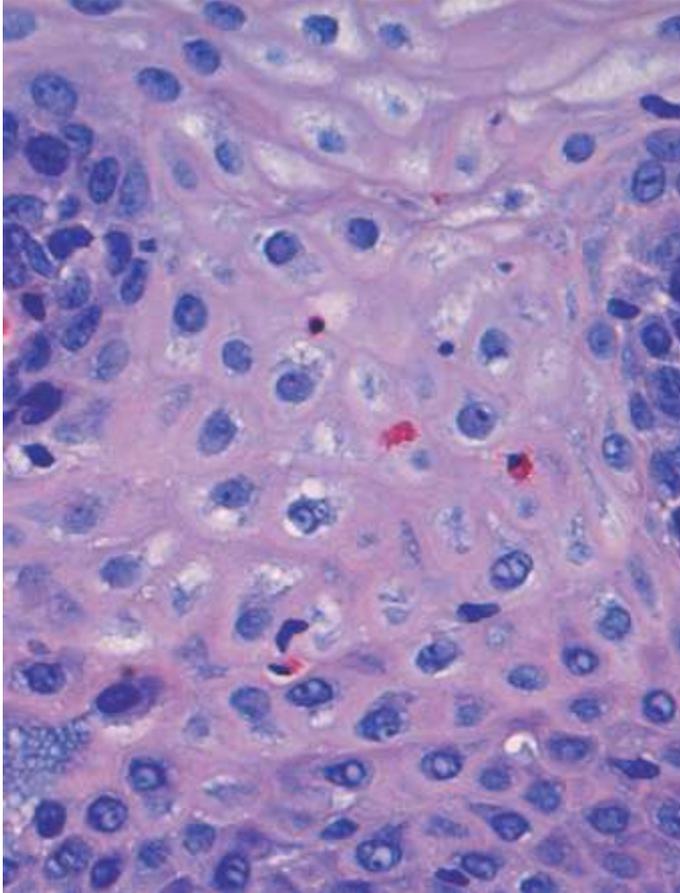


EOSINOPHILIC ESOPHAGITIS

- ⊕ Numerous eosinophils, more than (5).
- ⊕ Associated w/ lymphocytes.

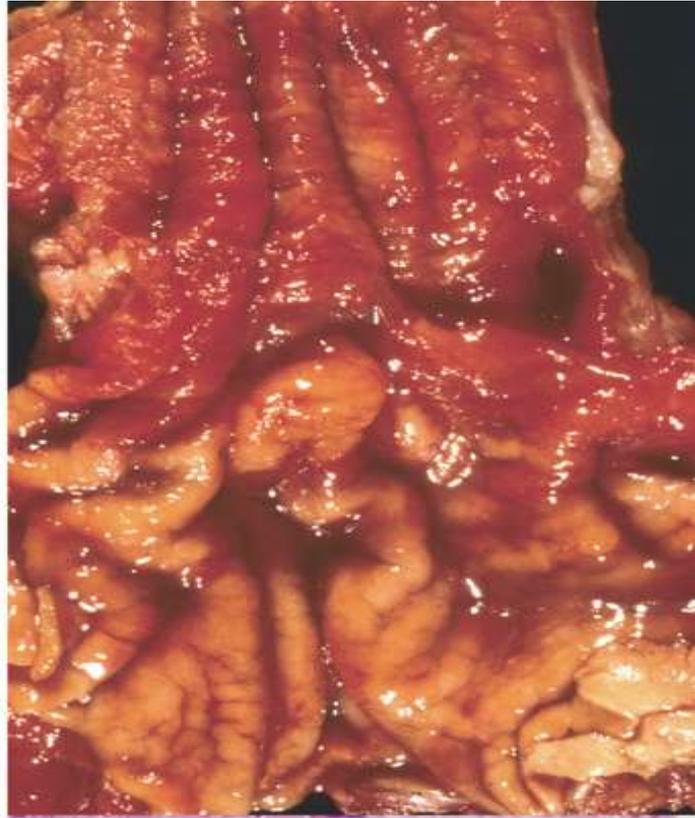
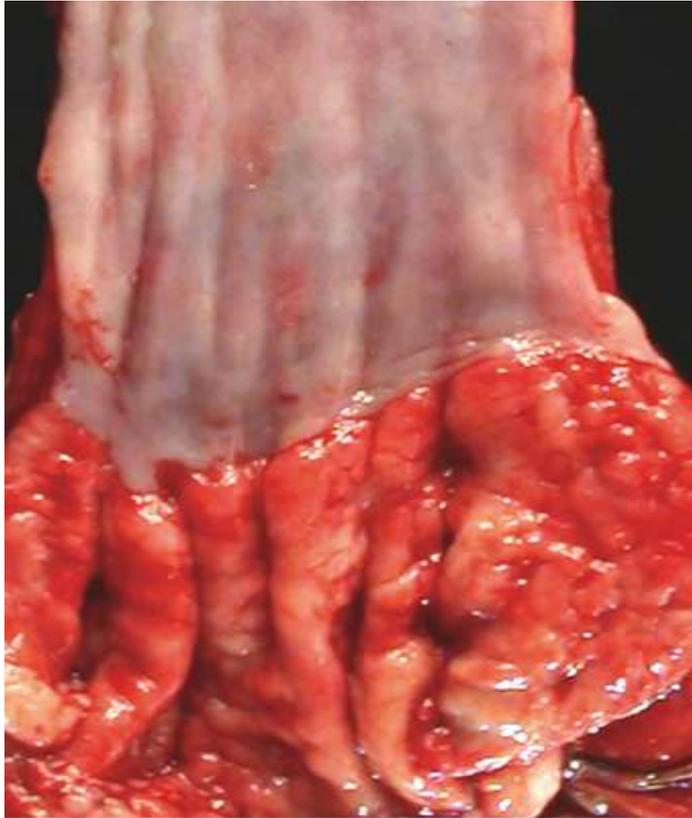


⊕ Reflux esophagitis

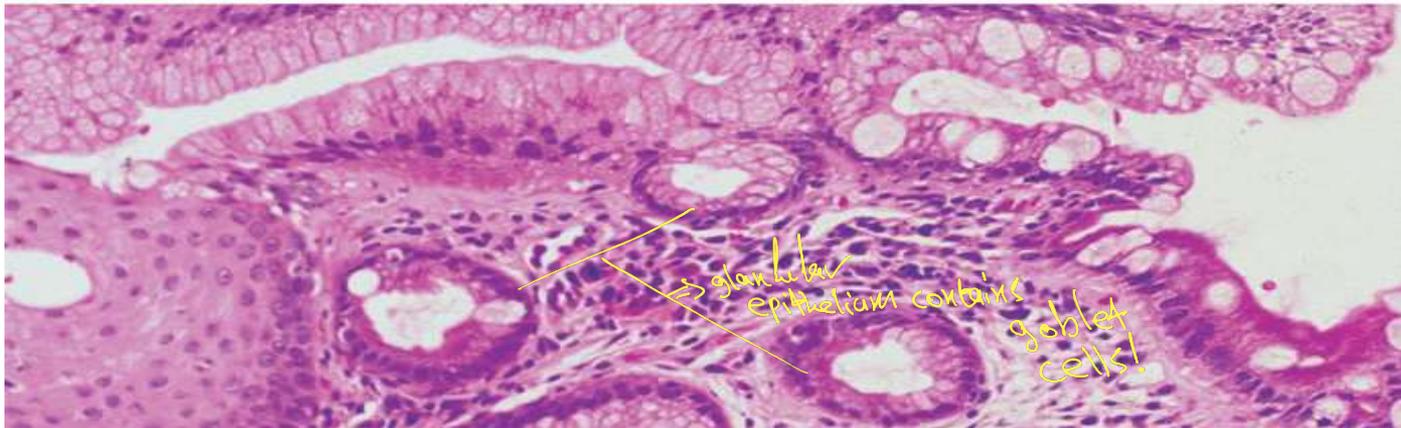


BARRETT ESOPHAGUS

* 10% of patients presented w/ Barrett are GERD patients.



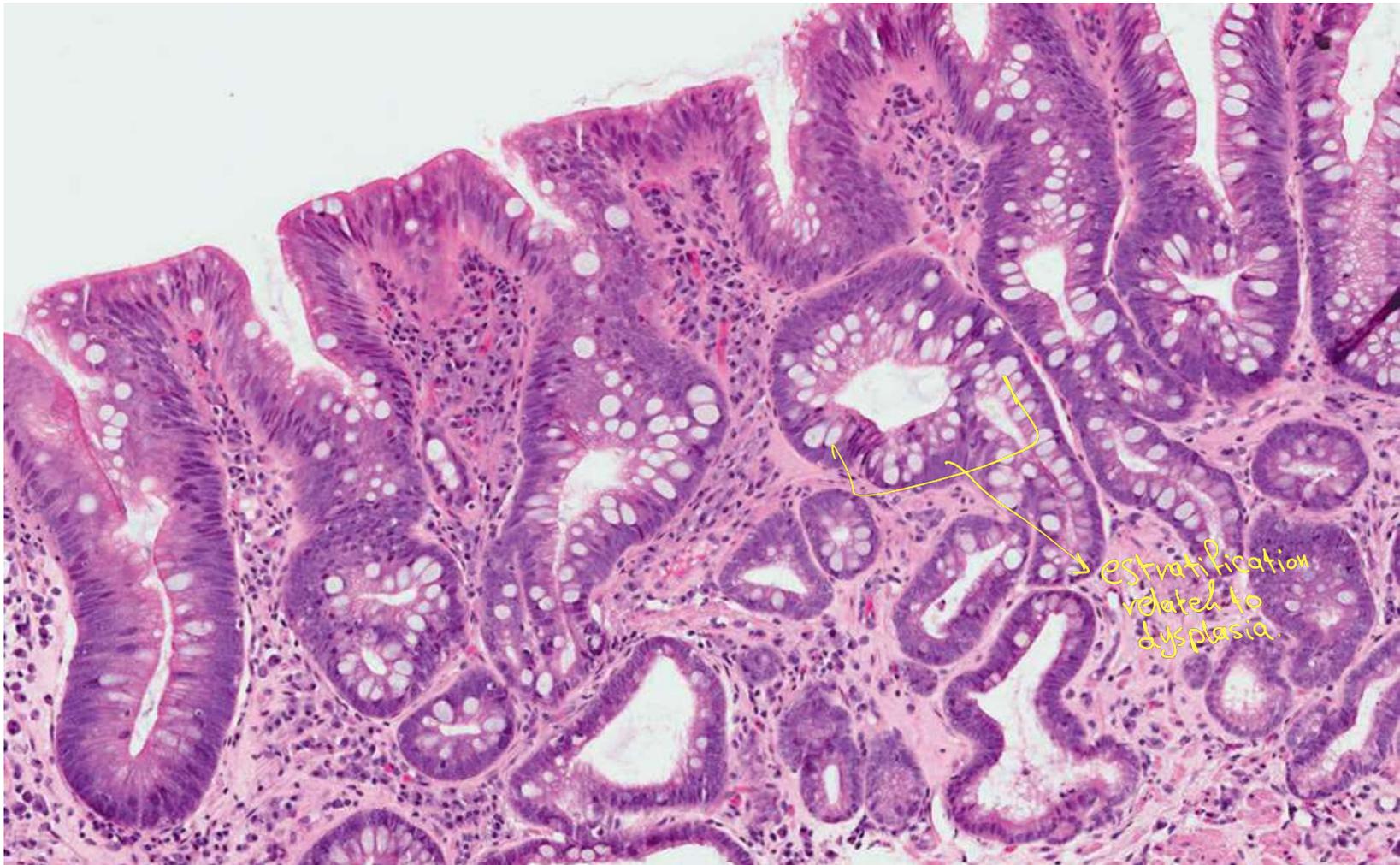
* Red
Velvety
above the
Gastroesophageal
junction.



glandular
epithelium contains
goblet
cells

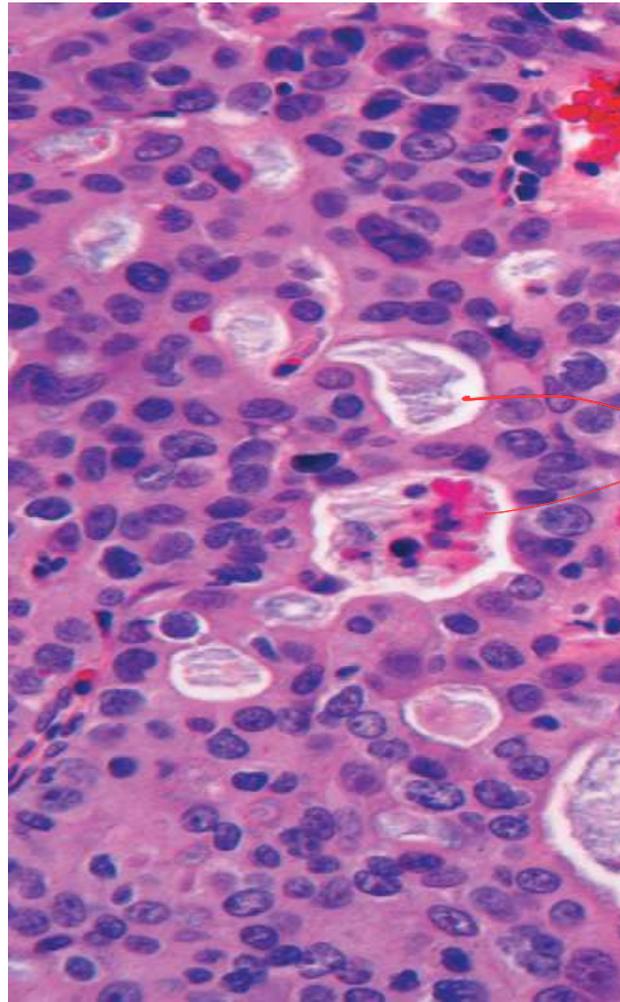


. BARRETT ESOPHAGUS WITH LOW-GRADE DYSPLASIA, INTESTINAL TYPE.



ESOPHAGEAL TUMORS

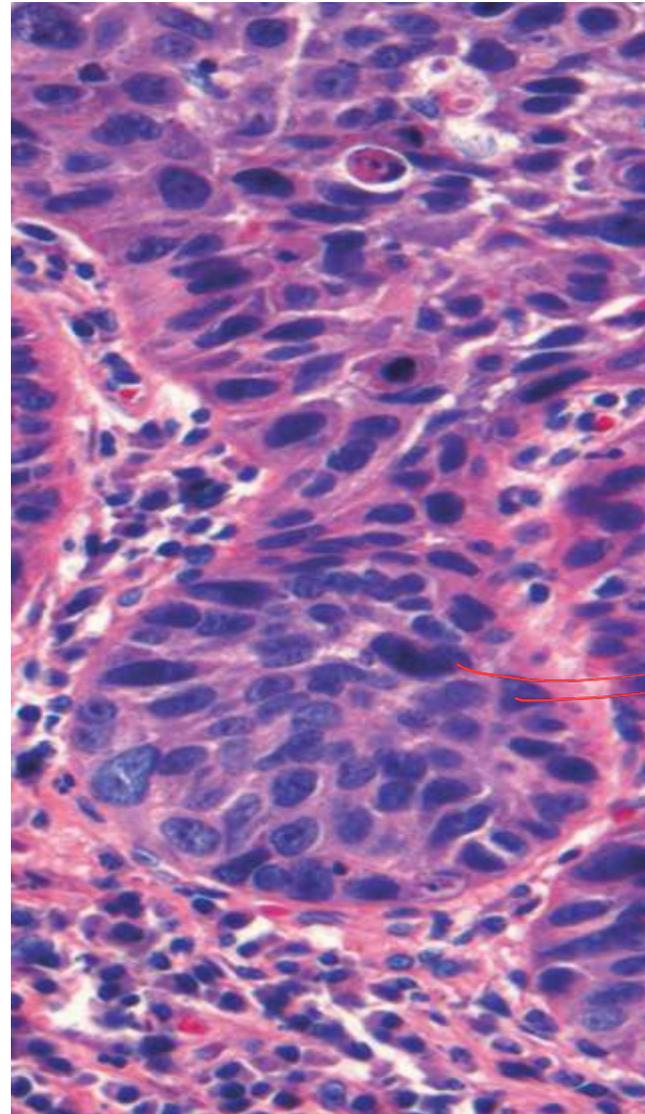
1- Esophageal adenocarcinoma. \Rightarrow in the lower third!



Neoplastic
glands
atypical glands!
w/ mucin in it!



SQUAMOUS CELL CARCINOMA COMPOSED OF NESTS OF MALIGNANT CELLS THAT PARTIALLY RECAPITULATE THE STRATIFIED ORGANIZATION OF SQUAMOUS EPITHELIUM. ⊕ Middle third!



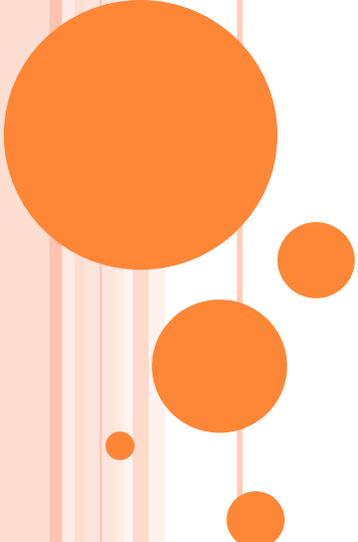
dysplastic squamous cells. w/ pleomorphism!



patient presented with adenofascia with weight loss

tumor
↓
decreased
food
intake

+
Hint ↓
complaint
or
image



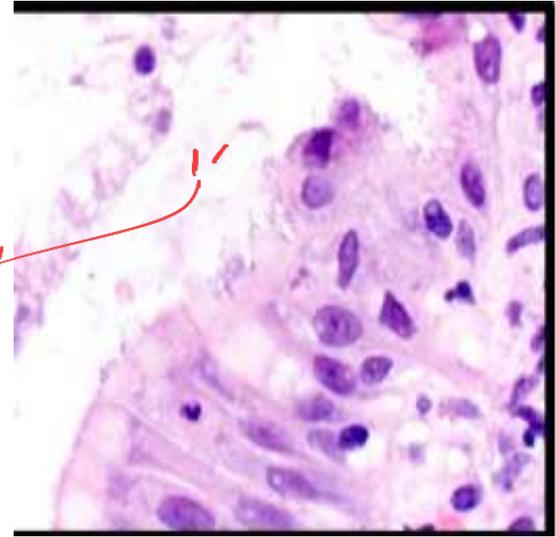
STOMACH

CHRONIC GASTRITIS

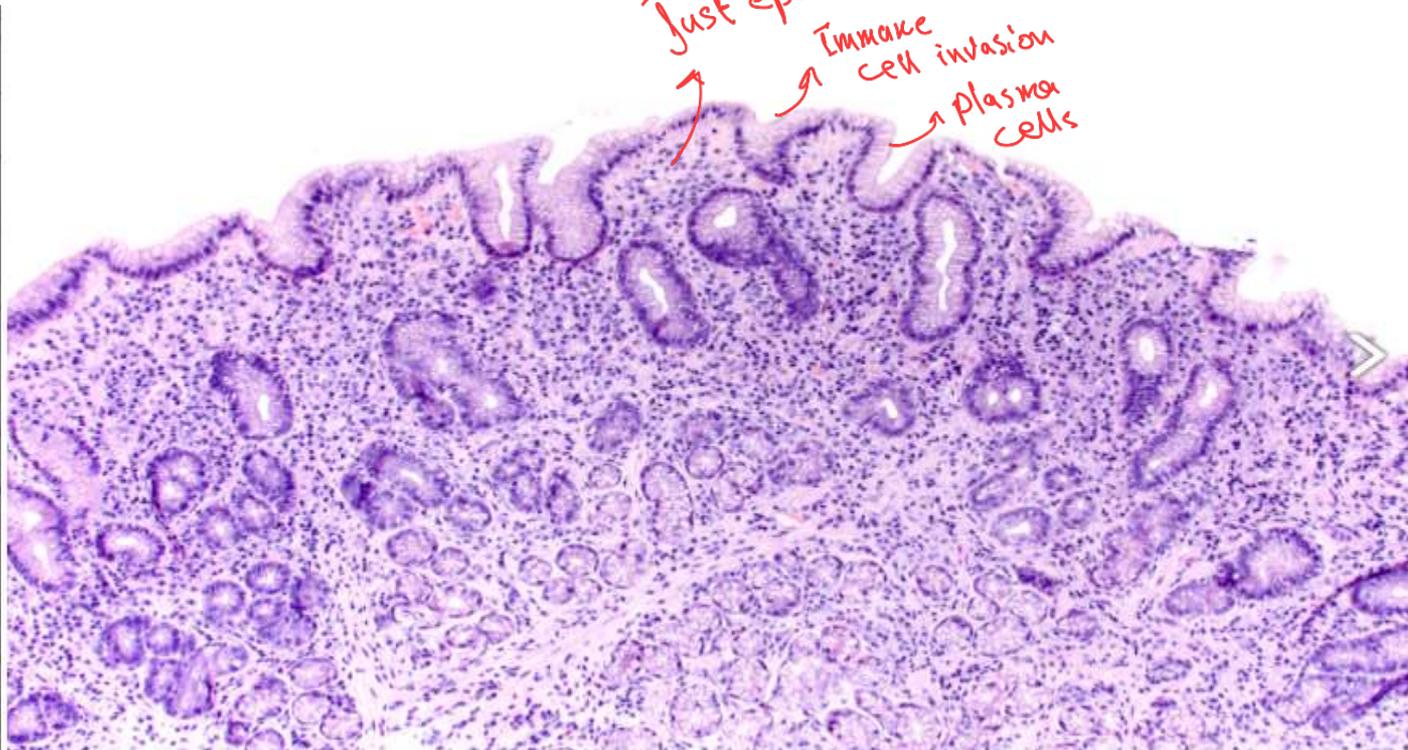
Helicobacter pylori Gastritis

↳ Most common location? Antrum

bacilli
↳ diagnostic key.



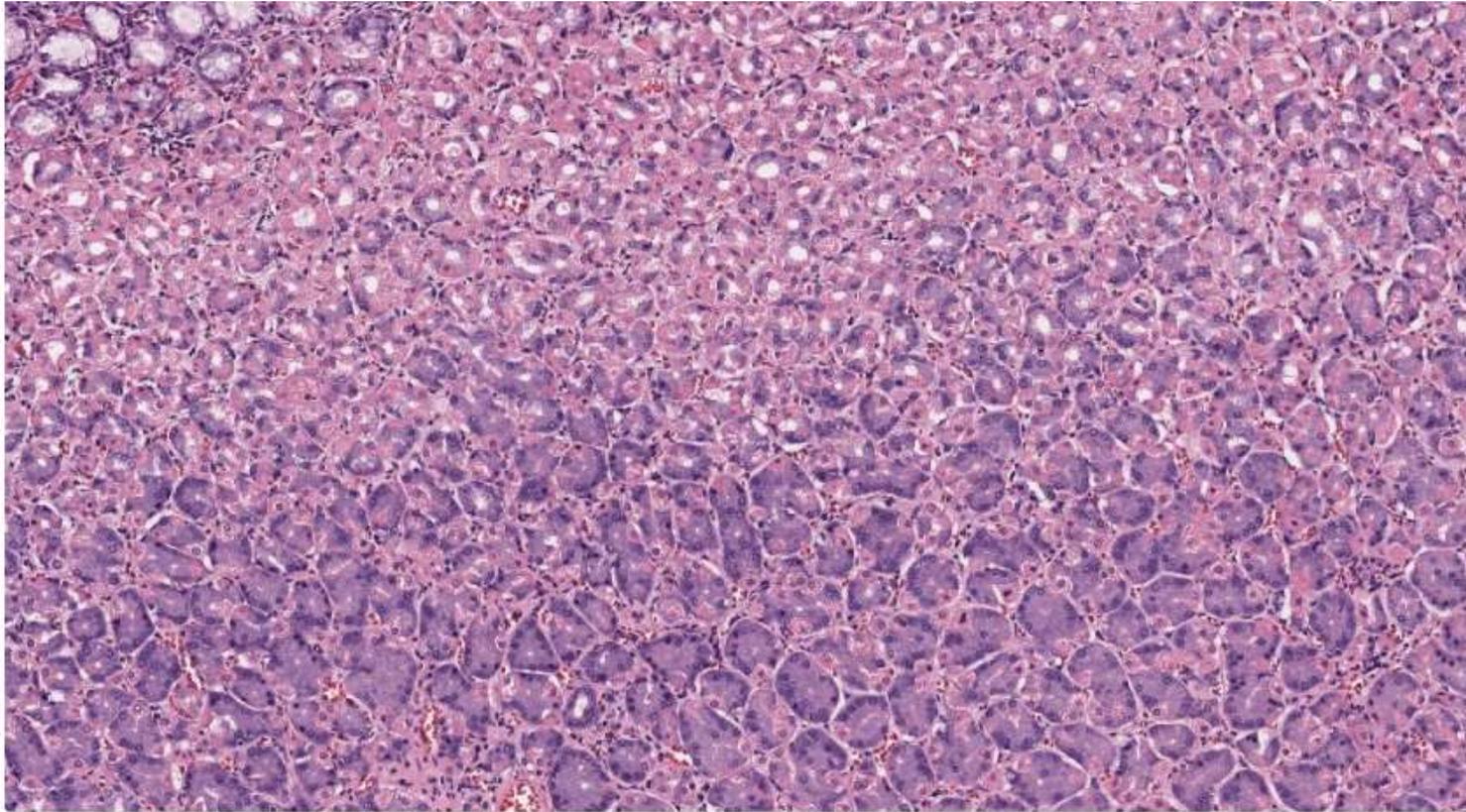
Just epithelium
↳ Immature cell invasion
↳ plasma cells



Stomach, upper GI endoscopy, *H. pylori* gastritis, superficial chronic inflammation, organisms not seen at this power.  

NORMAL BODY TYPE GASTRIC MUCOSA

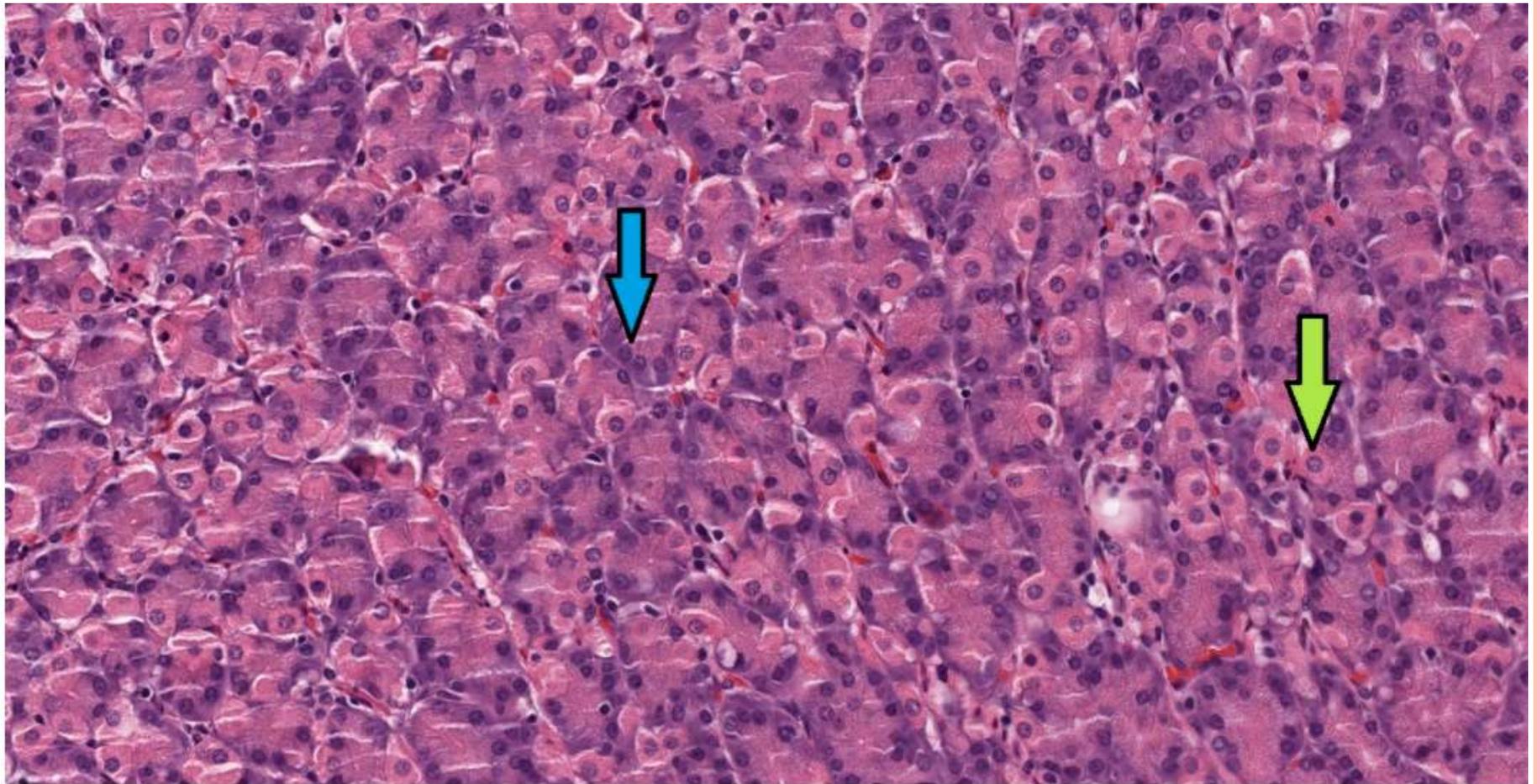
⊕ Numerous glands → eosinophilic parietal cells
↳ basophilic chief cells



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

⊗ glands are lost!

⊕ Patient presented w/ gastric pain along w/ fatigue (fatigue related w/ neuropathy) B12 deficiency associated then after endoscopy, this pic. location of biopsy → body Antrum
what is the diagnosis?



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



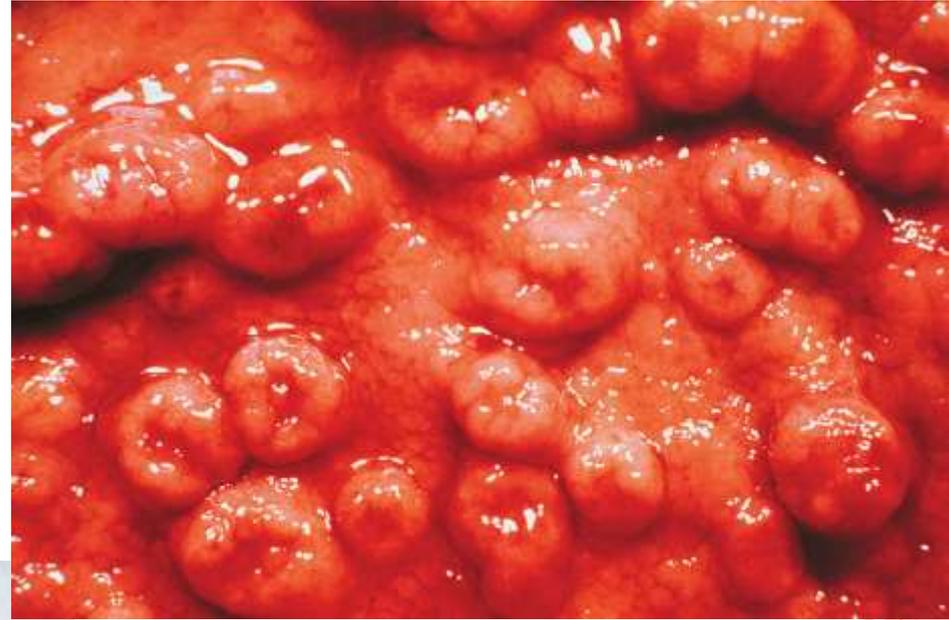
FUNDIC GLAND POLYPS.

INFLAMMATORY AND HYPERPLASTIC
POLYPS.

GASTRIC ADENOMAS



MORPHOLOGY

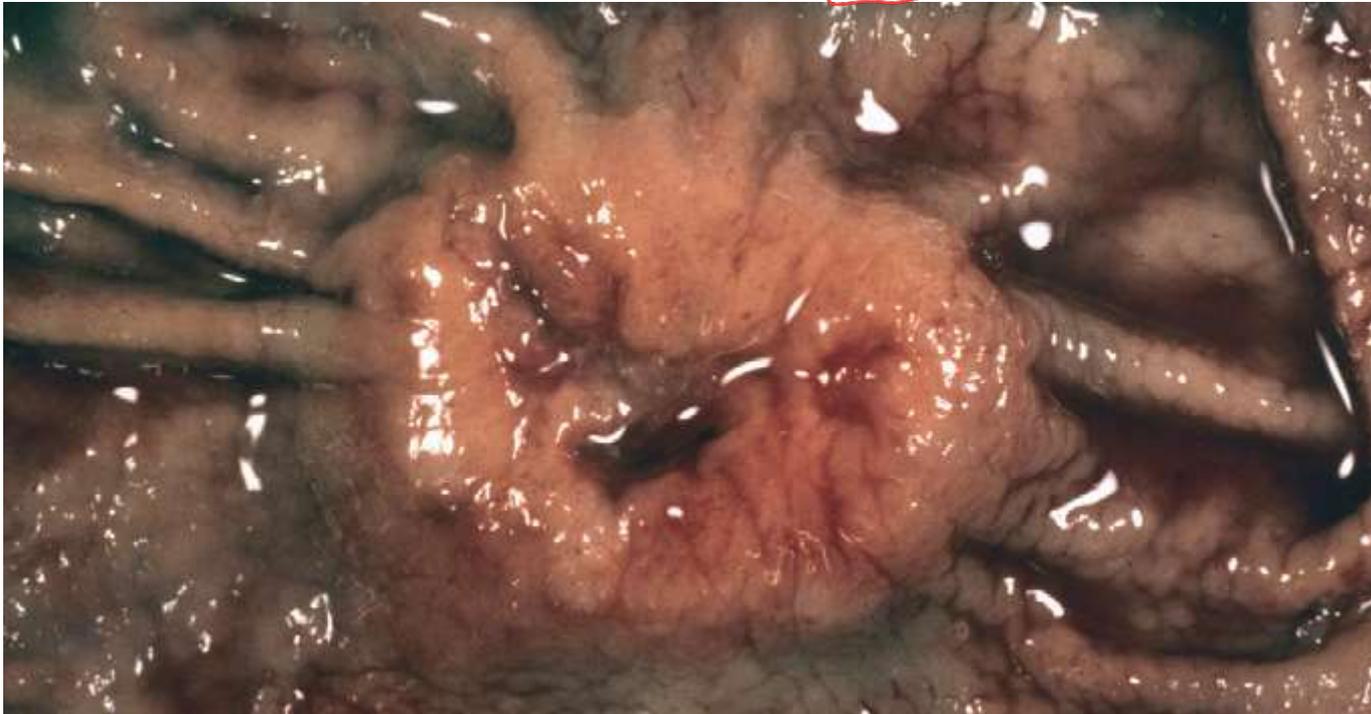


* Not associated
w/ dysplastic cells!
but Adenoma does!



INTESTINAL-TYPE ADENOCARCINOMA CONSISTING
OF AN ELEVATED MASS WITH HEAPED-UP
BORDERS AND
CENTRAL ULCERATION

⊗ one mass
might be
ulcerated!



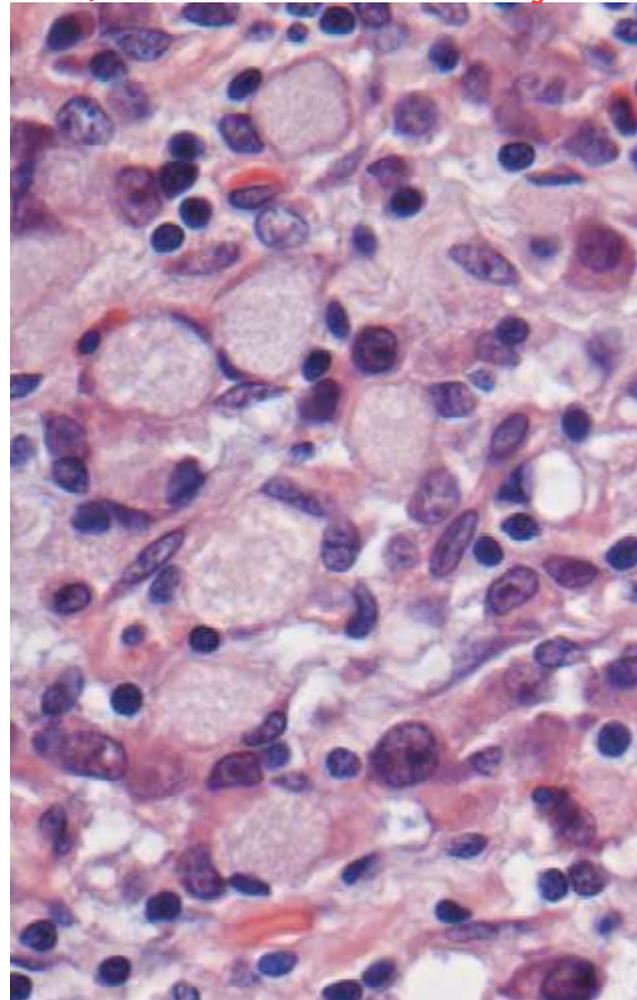
A, LINITIS PLASTICA. THE GASTRIC WALL IS MARKEDLY THICKENED, AND RUGAL FOLDS ARE PARTIALLY LOST. B, SIGNET RING CELLS WITH LARGE CYTOPLASMIC MUCIN VACUOLES AND PERIPHERALLY DISPLACED, CRESCENT-SHAPED NUCLEI.



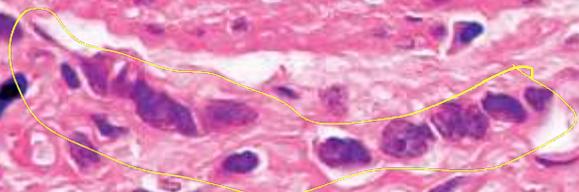
⊗ Age ⇒ 55

⊗ Poor prognosis than the intestinal type!

→ No diffuse here
↳ that's why
it's better
prognosis!

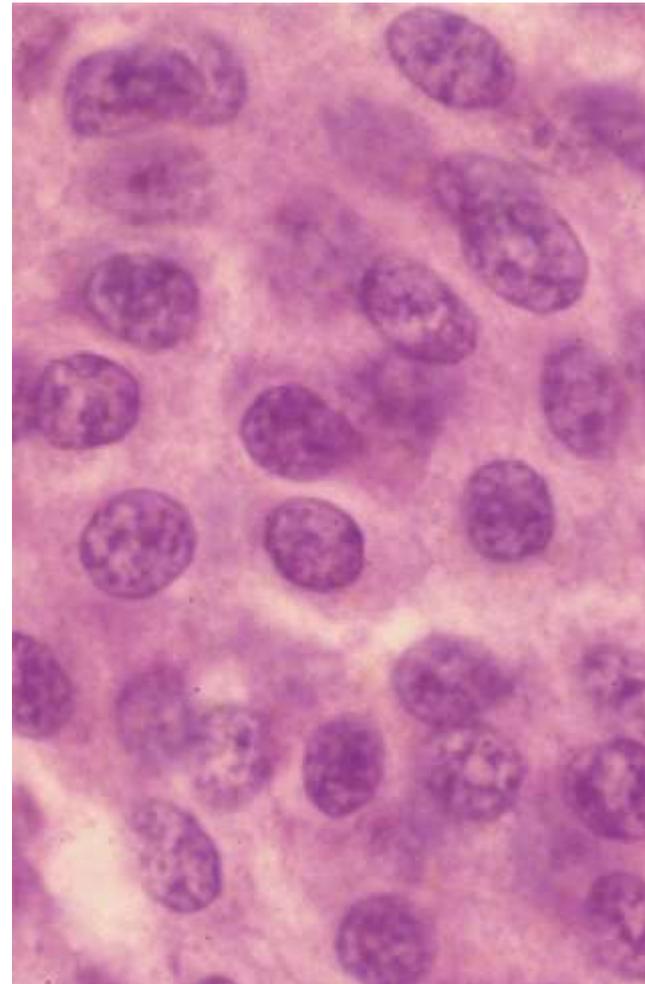
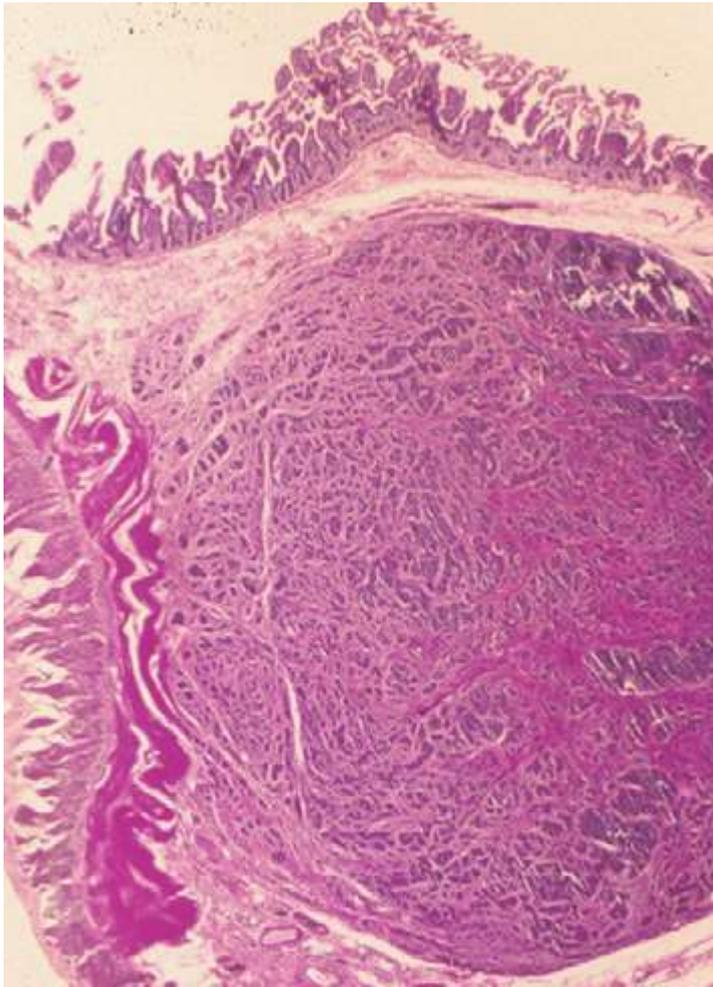


|| diffuse type ||



indium piling
بجھورا پھین!

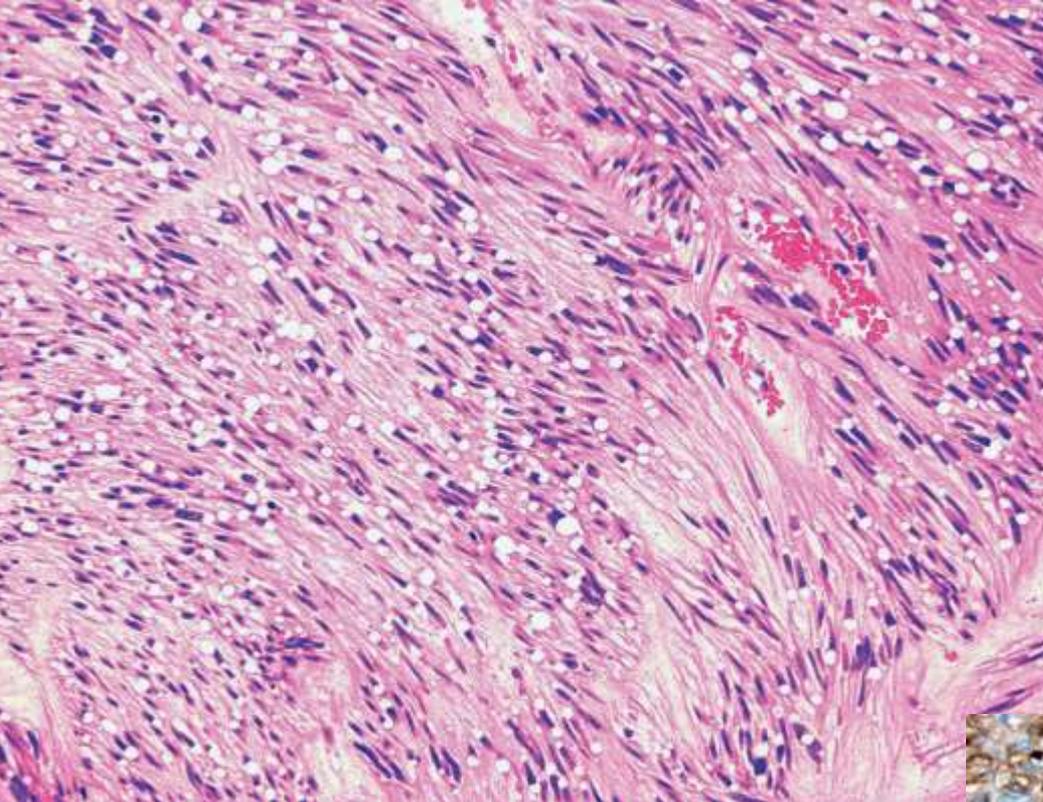
GASTROINTESTINAL CARCINOID TUMOR (NEUROENDOCRINE TUMOR). A, CARCINOID TUMORS OFTEN FORM A SUBMUCOSAL. B. SHOWS THE BLAND CYTOLOGY THAT TYPIFIES CARCINOID TUMORS. THE CHROMATIN TEXTURE, WITH FINE AND COARSE CLUMPS, FREQUENTLY ASSUMES A "SALT AND PEPPER" PATTERN. *of chromatin* AGGRESSIVE.



GASTROINTESTINAL STROMAL TUMOR

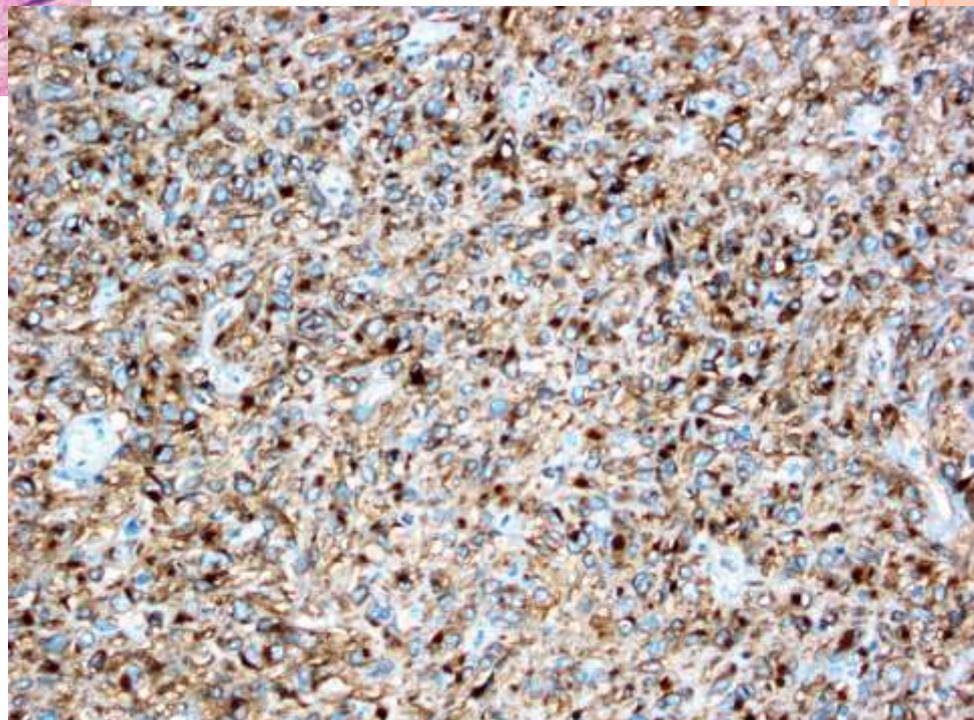
- The most common mesenchymal tumor of in the stomach.
- Overall, GISTs are slightly more common in males. The peak incidence of gastric GIST is around 60 years of age, with less than 10% occurring in persons younger than 40 years of age.
- Approximately **75% to 80% of all GISTs have oncogenic, gain-of-function mutations of the gene encoding the tyrosine kinase c-KIT,**





⇒ Spindle cells.

Diffuse CD117 (KIT)
immunoreactivity

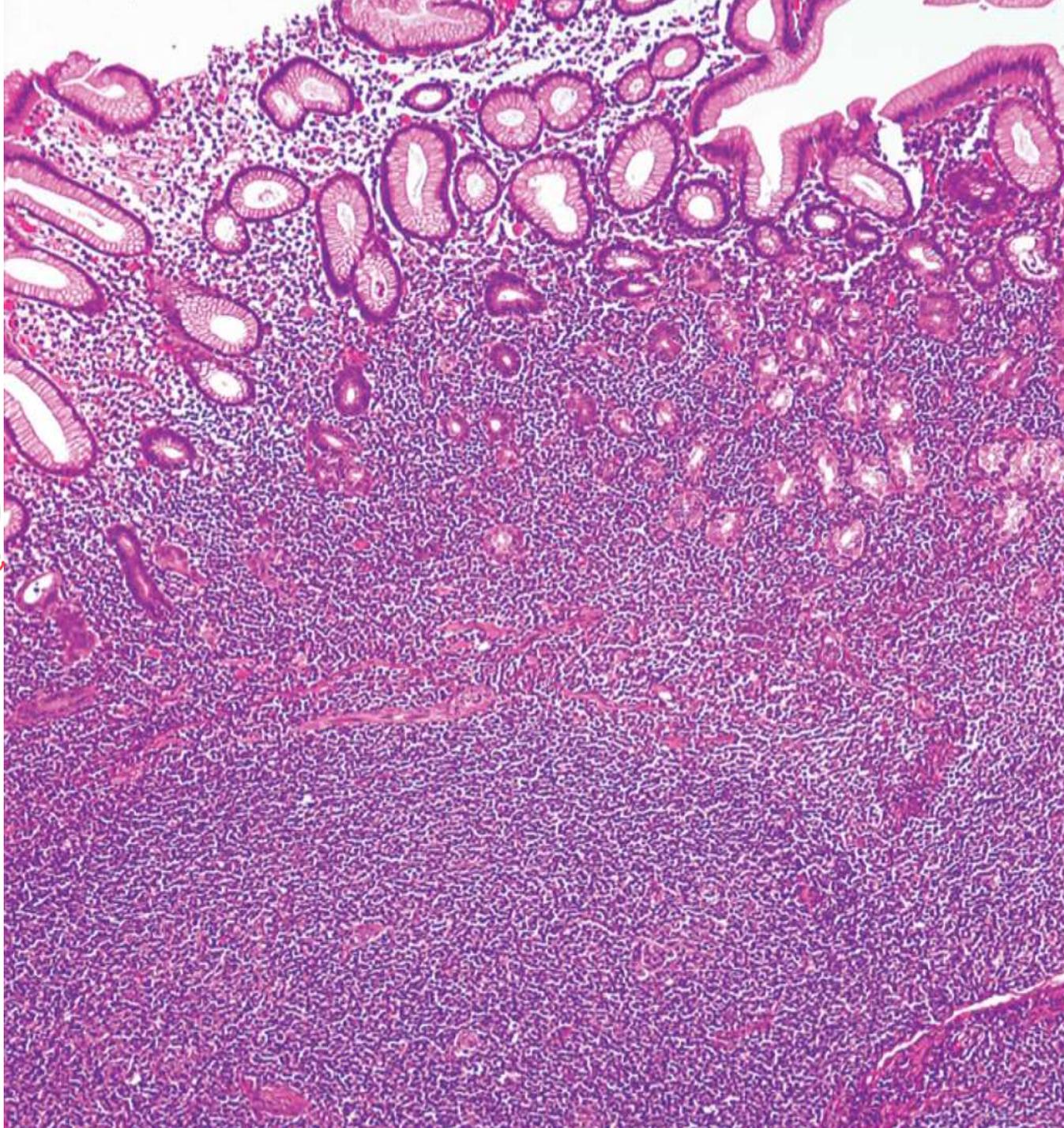


MALTOma

Large lymphoid
aggregate!

* *H. pylori* in progressed
stages may have
aggregation of
lymphocytes!
which is then known
as MALTOma

* 1st line of treatment?
Antibiotics



GOOD LUCK IN YOUR
EXAM

