DISEASE OF THE ORAL CAVITY AND ESOPHAGUS

Dr. Omar Hamdan

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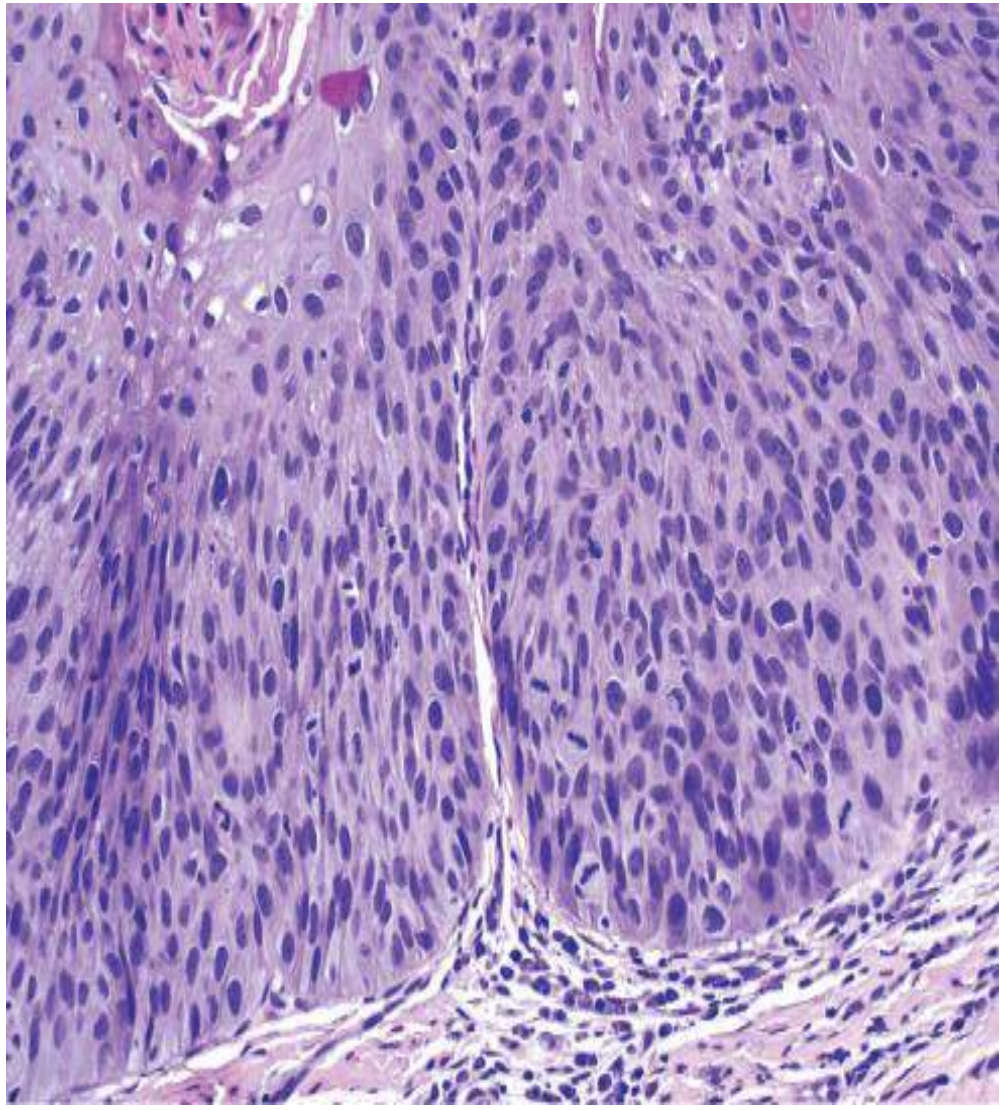
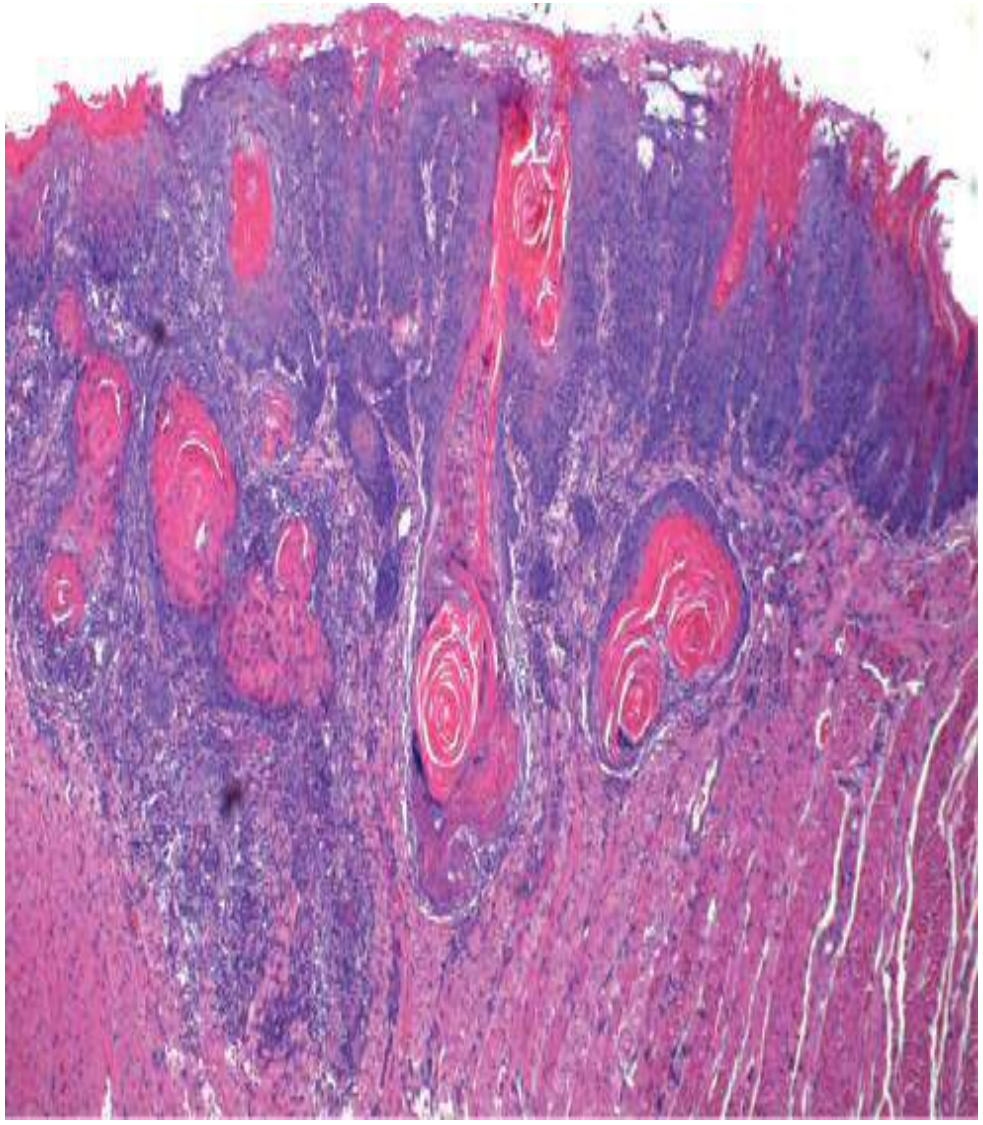
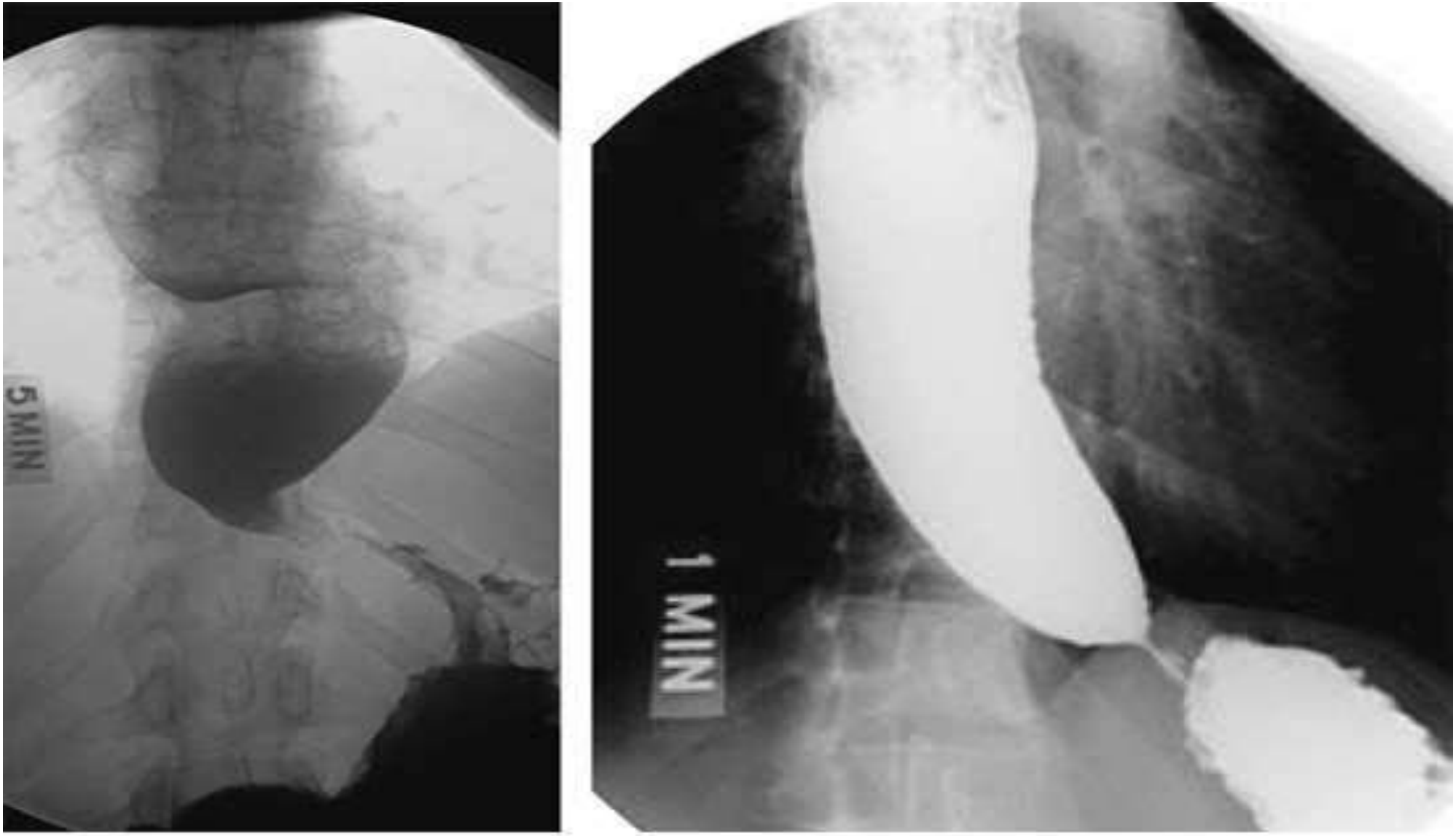
Undergraduate Lectures 2024

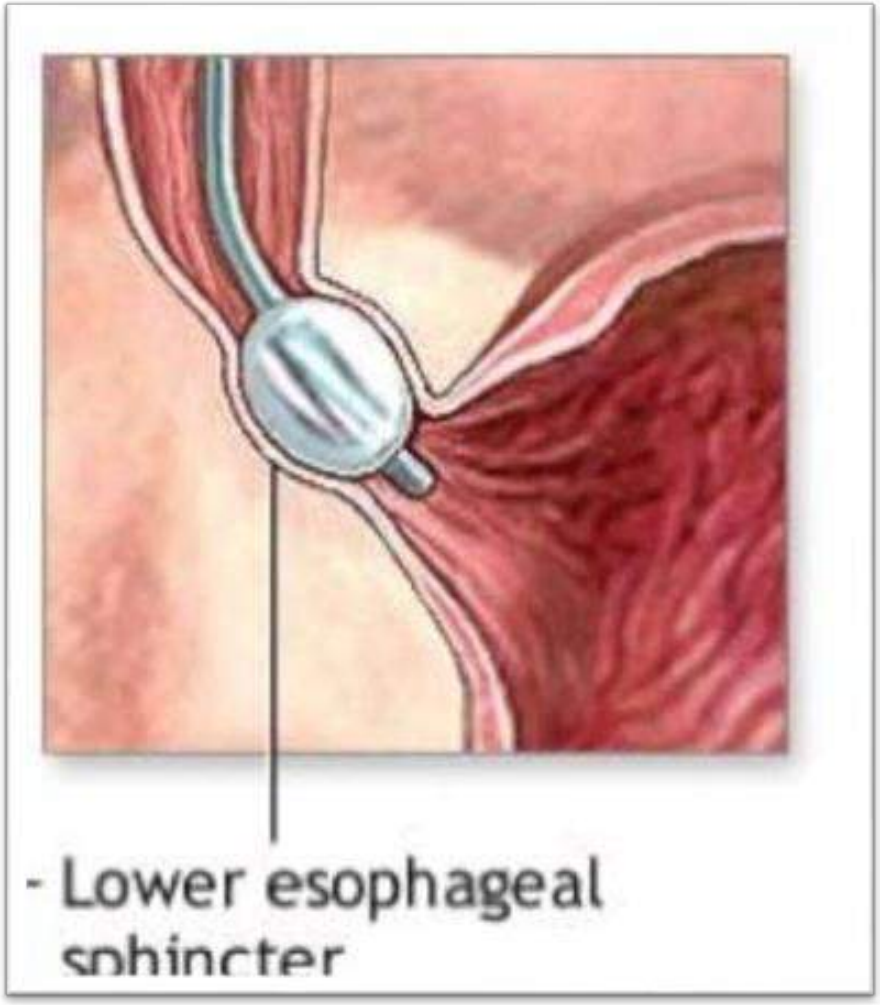






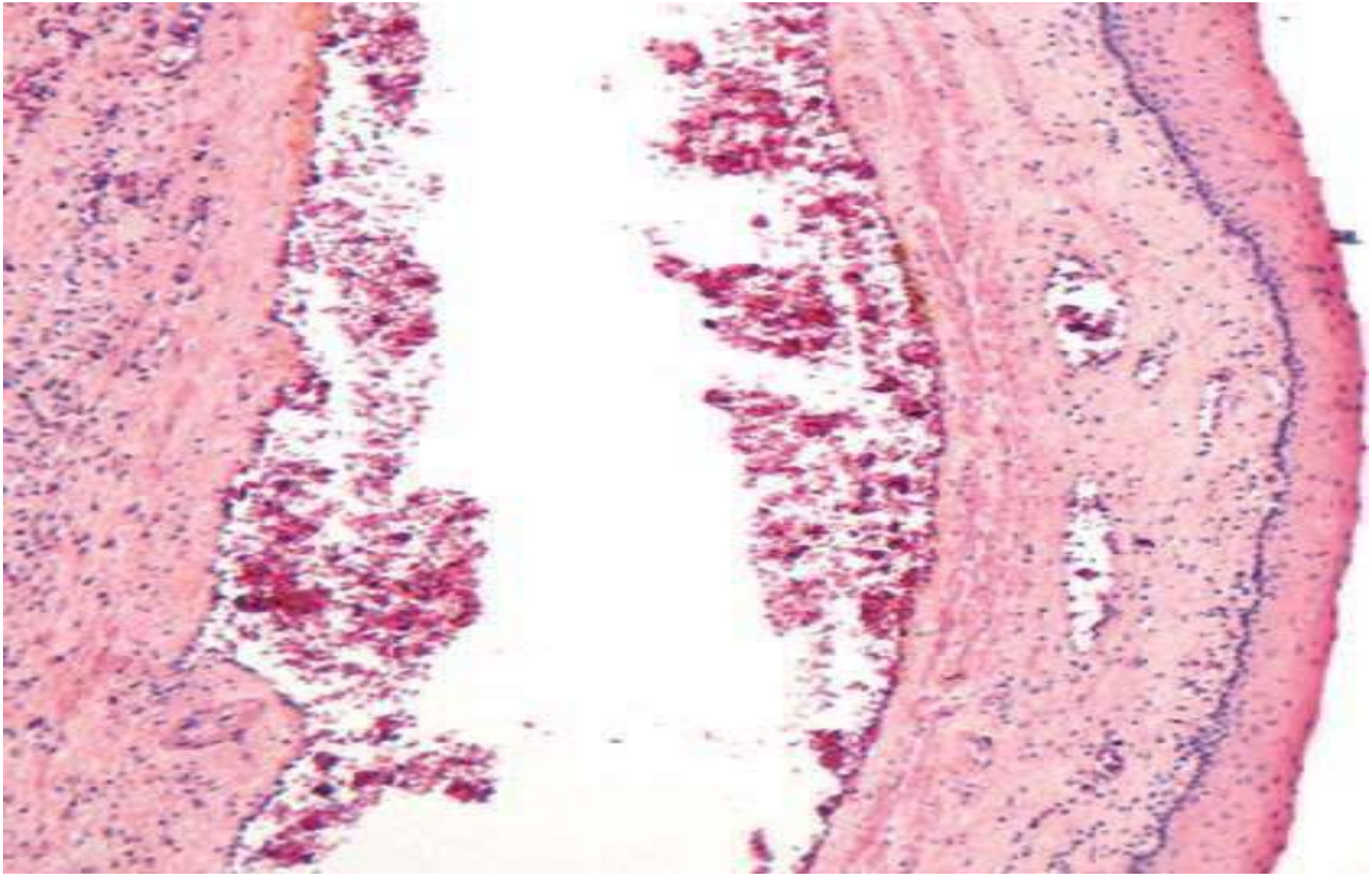
Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

Pneumatic balloon dilatation of the LES



Medpics - UCSD School of Medicine

Dilated varices beneath intact squamous mucosa



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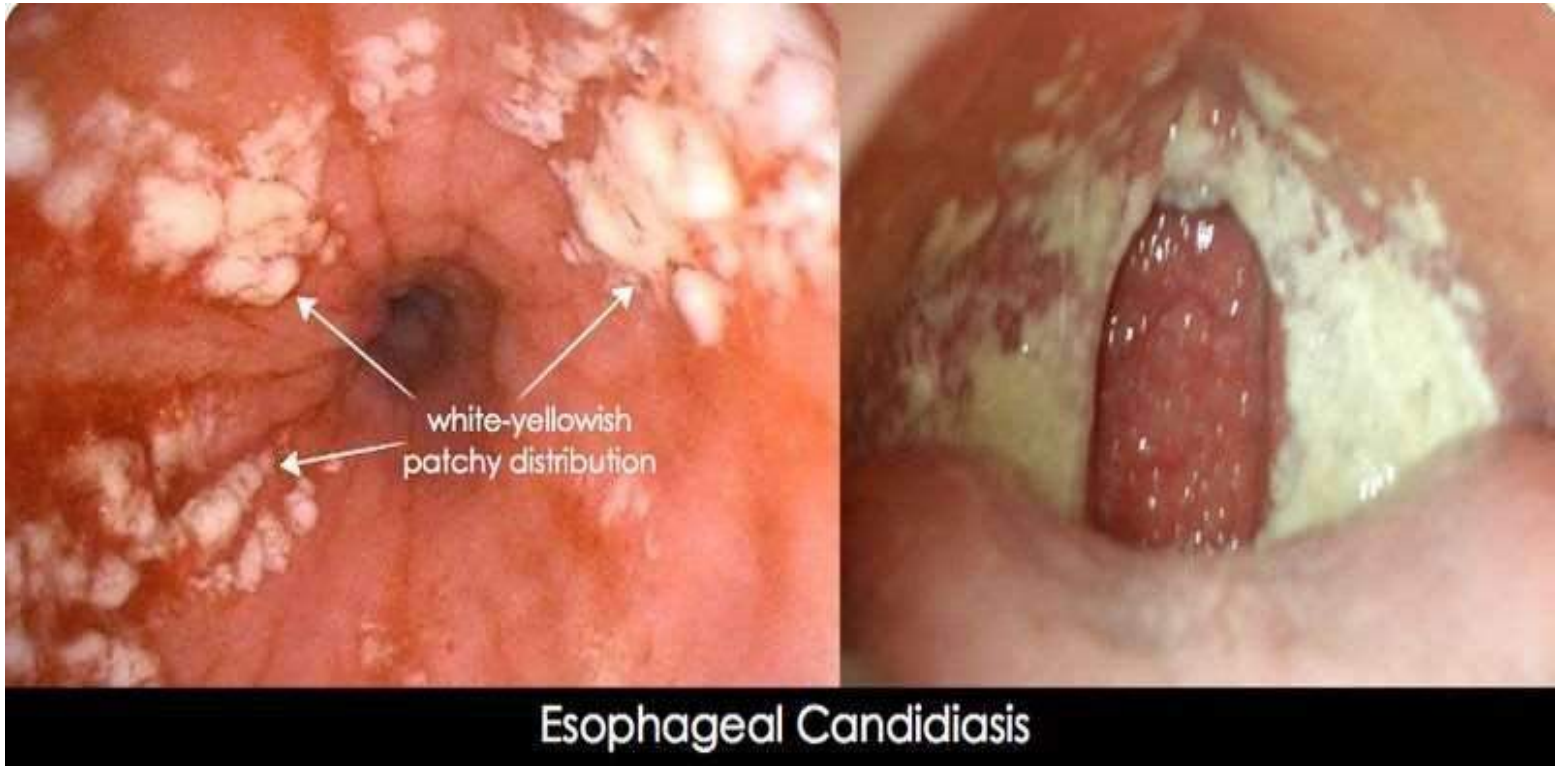
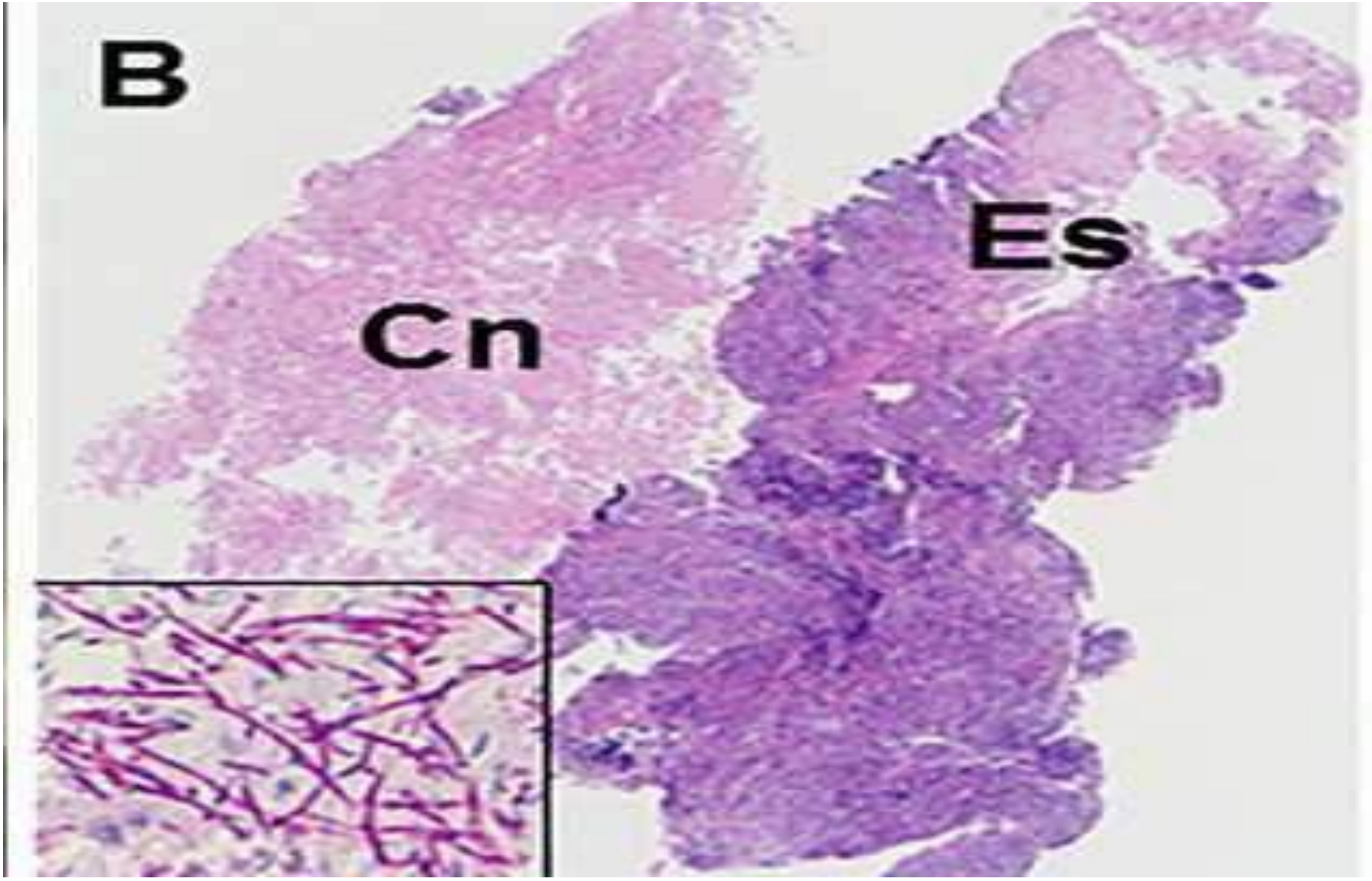
- Candidiasis :

- Adherent.
- Gray-white pseudomembranes
- Composed of matted fungal hyphae and inflammatory cells

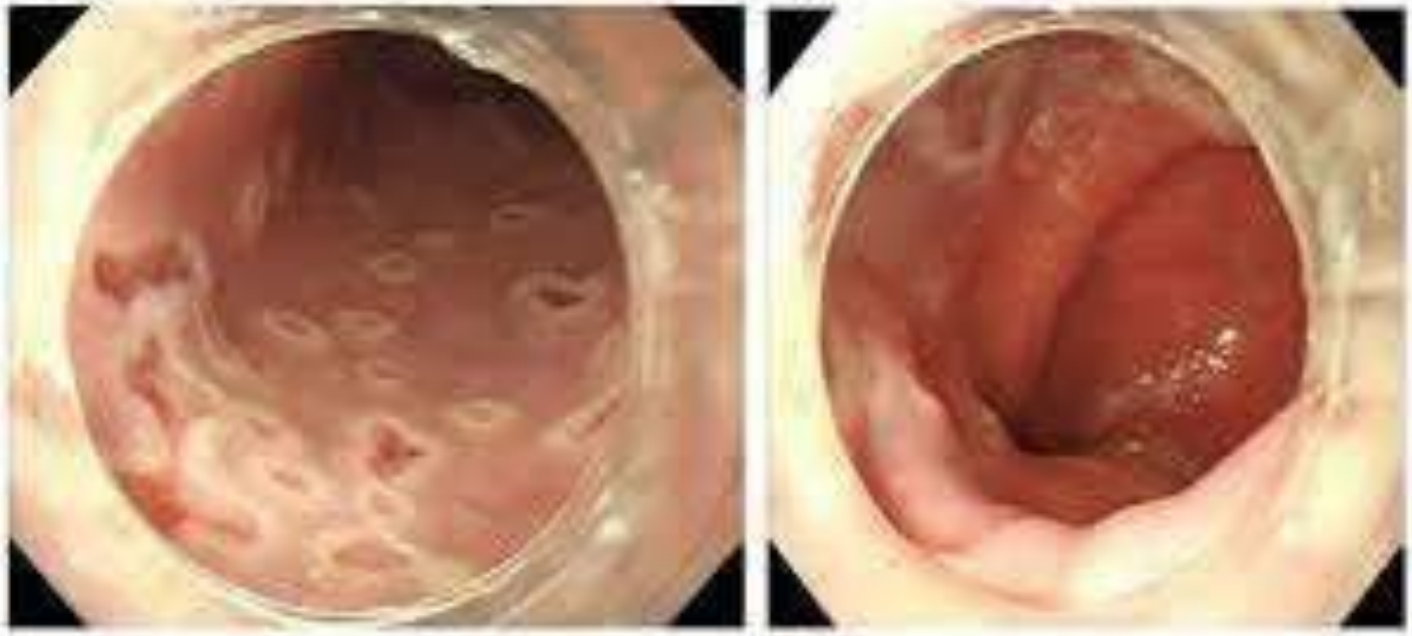


https://www.pinterest.com/pin/374291419013418659/

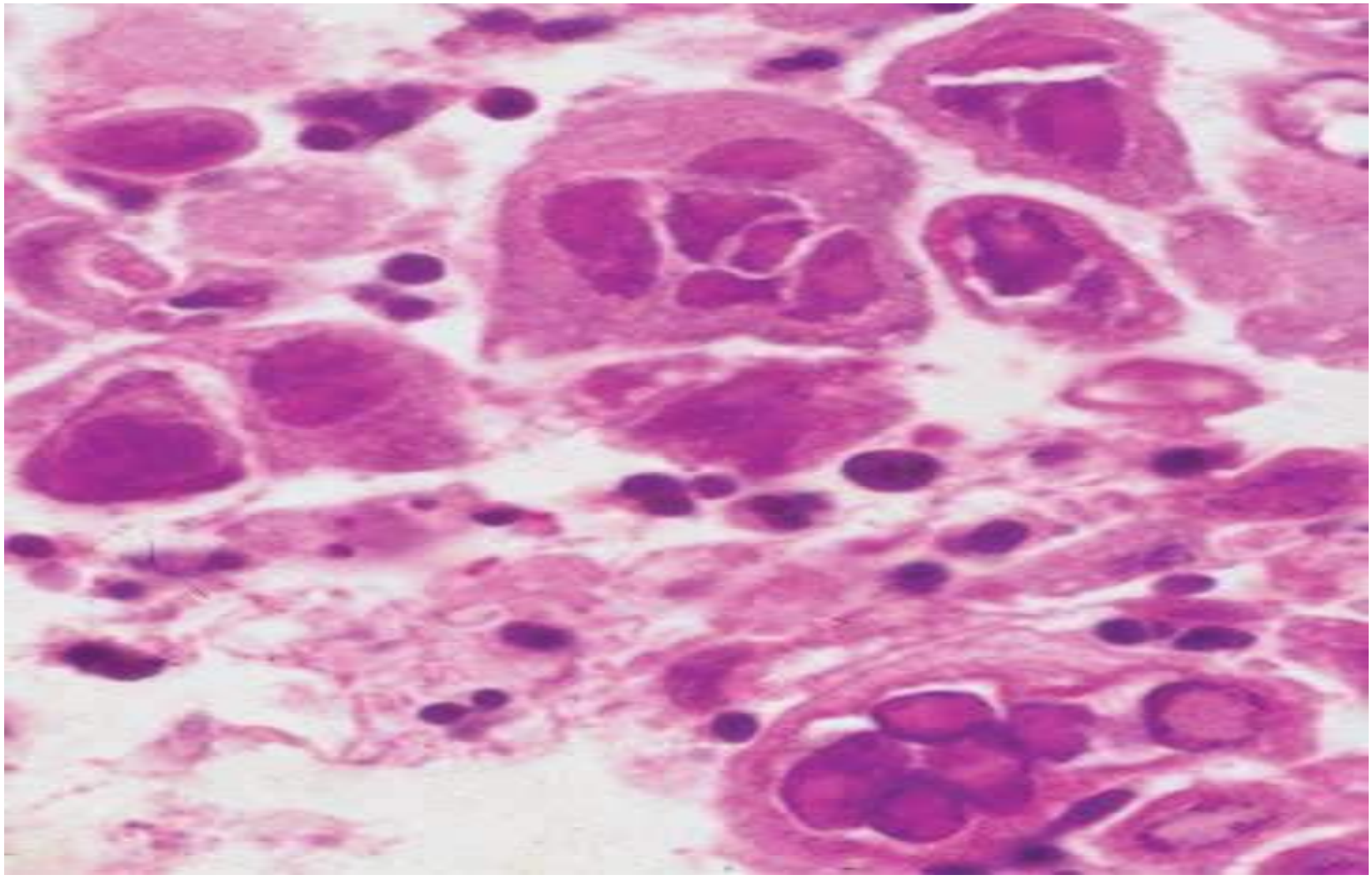


www.researchgate.net/publication/285369734_Esophageal_Candidiasis_as_the_Initial_Manifestation_of_Acute_Myeloid_Leukemia

- Herpes viruses
- Punched-out ulcers
- Histopathologic:
- Nuclear viral inclusions
- Degenerating epithelial cells ulcer edge
- Multinucleated epithelial cells.



Semantic Scholar



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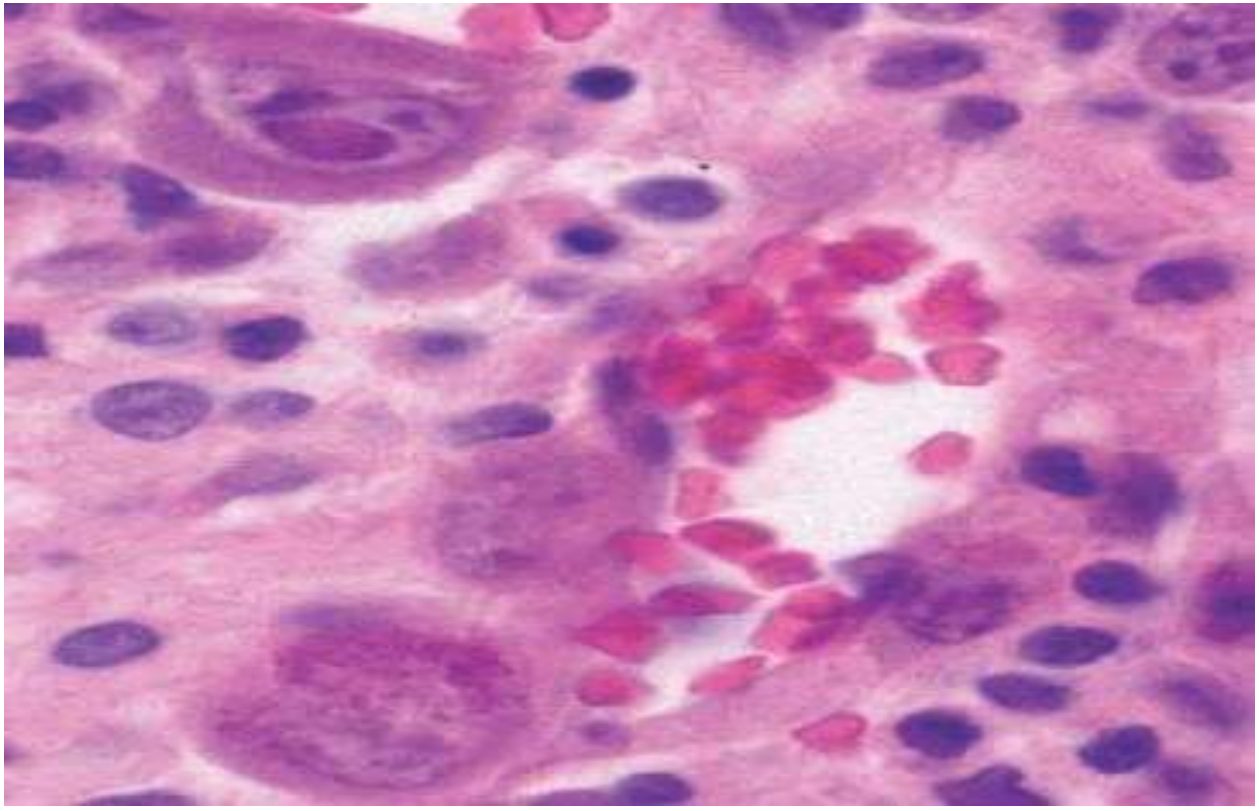
- CMV :
- Shallower ulcerations.
- Biopsy: nuclear and cytoplasmic inclusions in capillary endothelium and stromal cells



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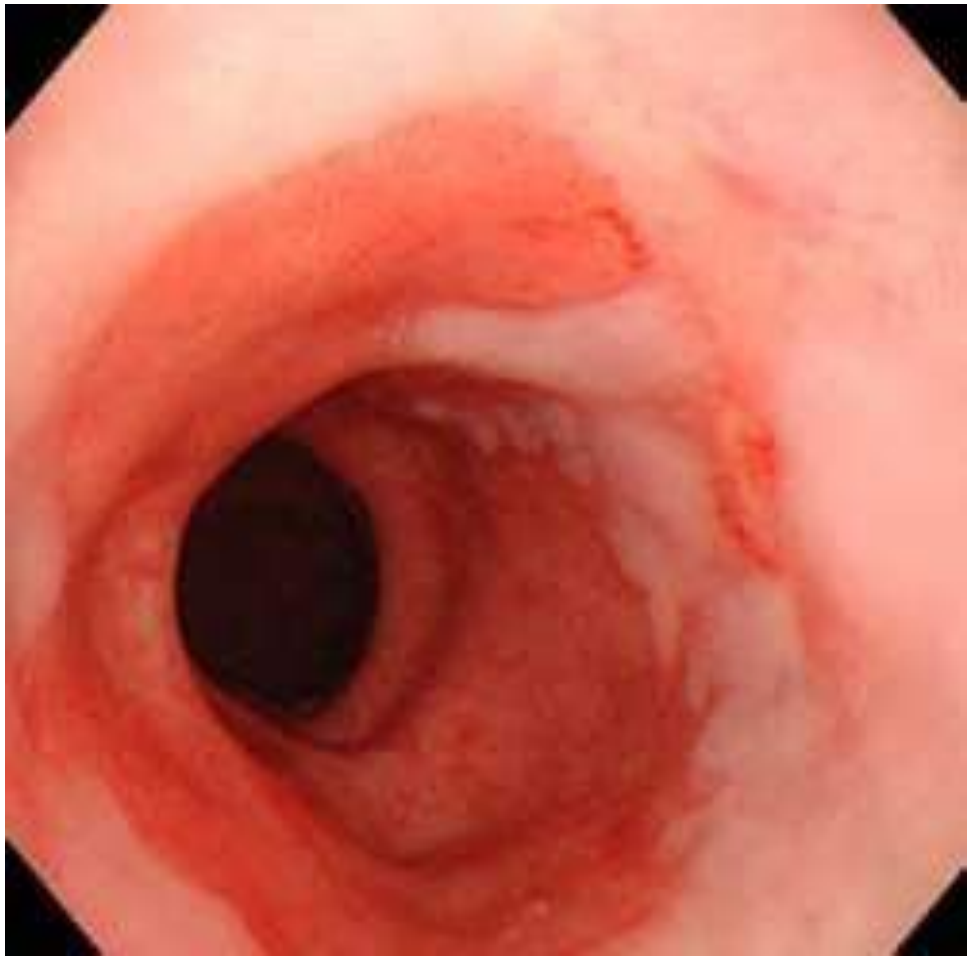

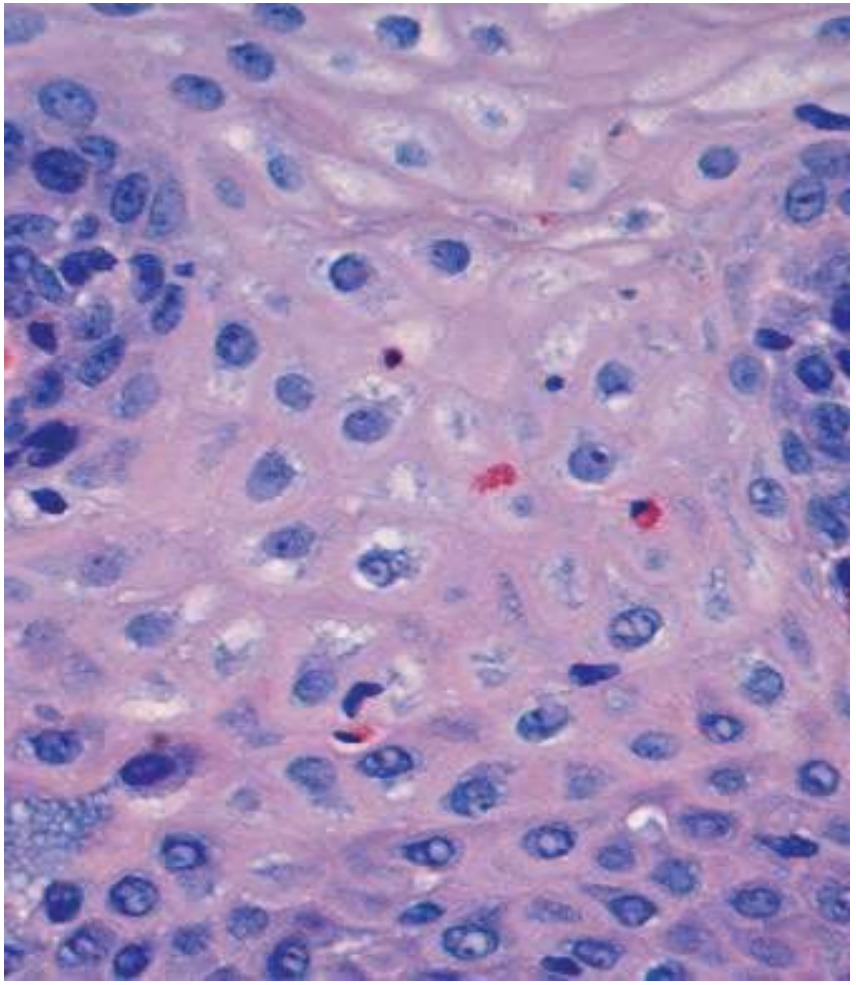

nature.com

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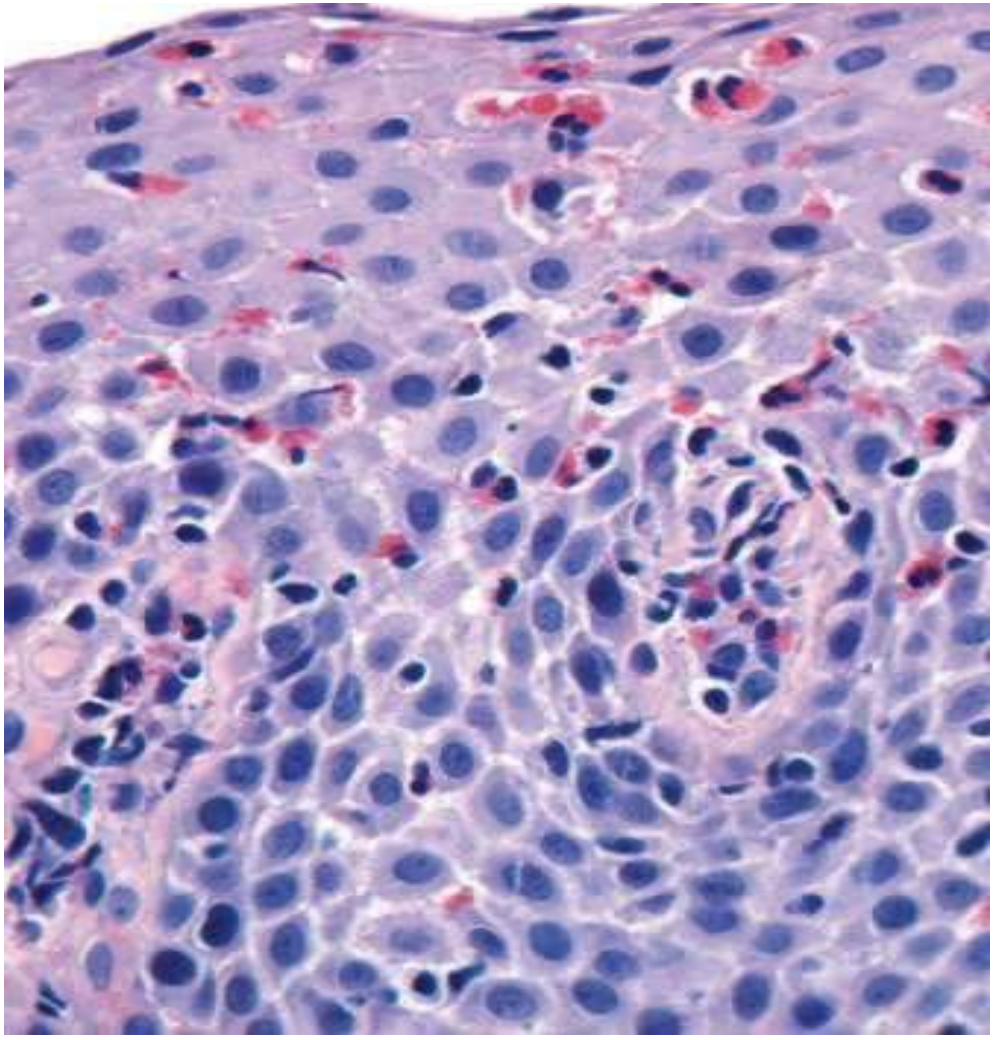

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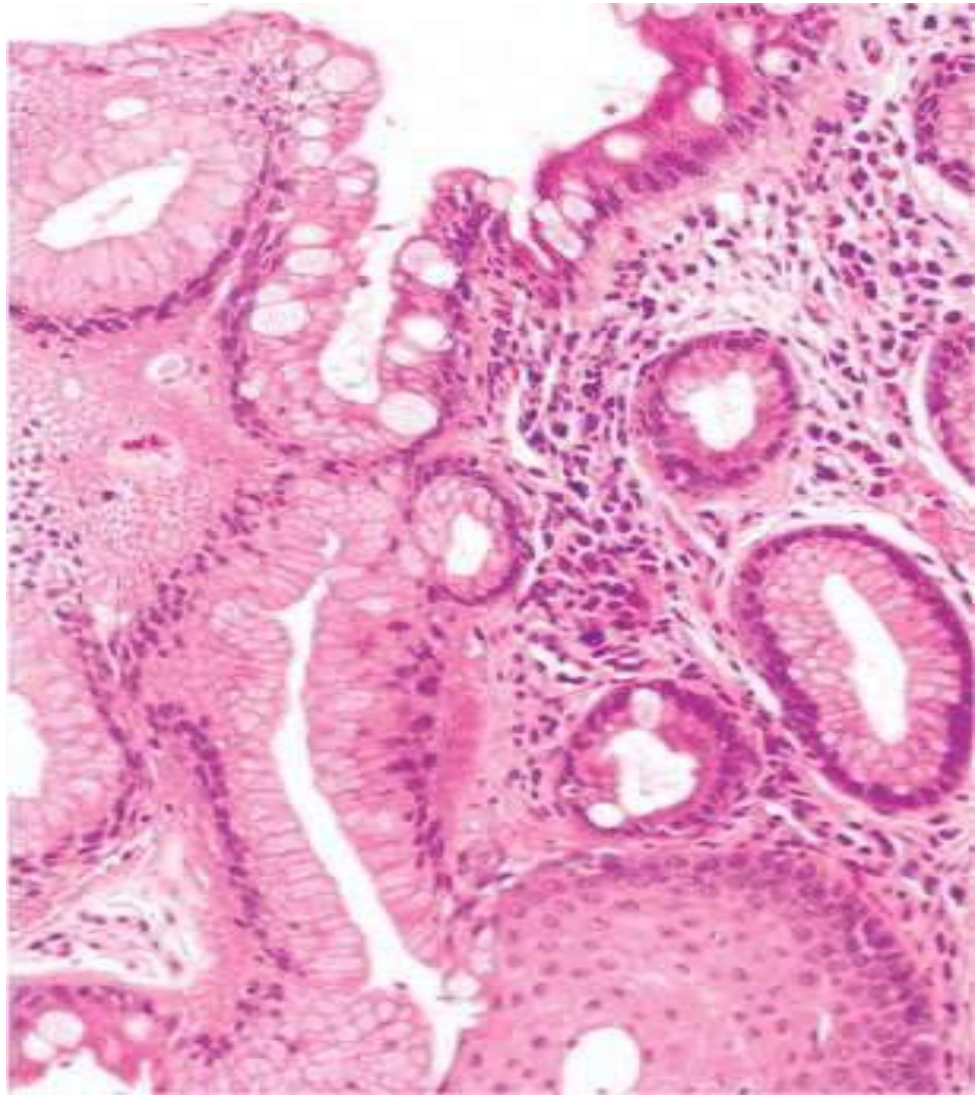
Gastroenterology Consultants of San Antonio



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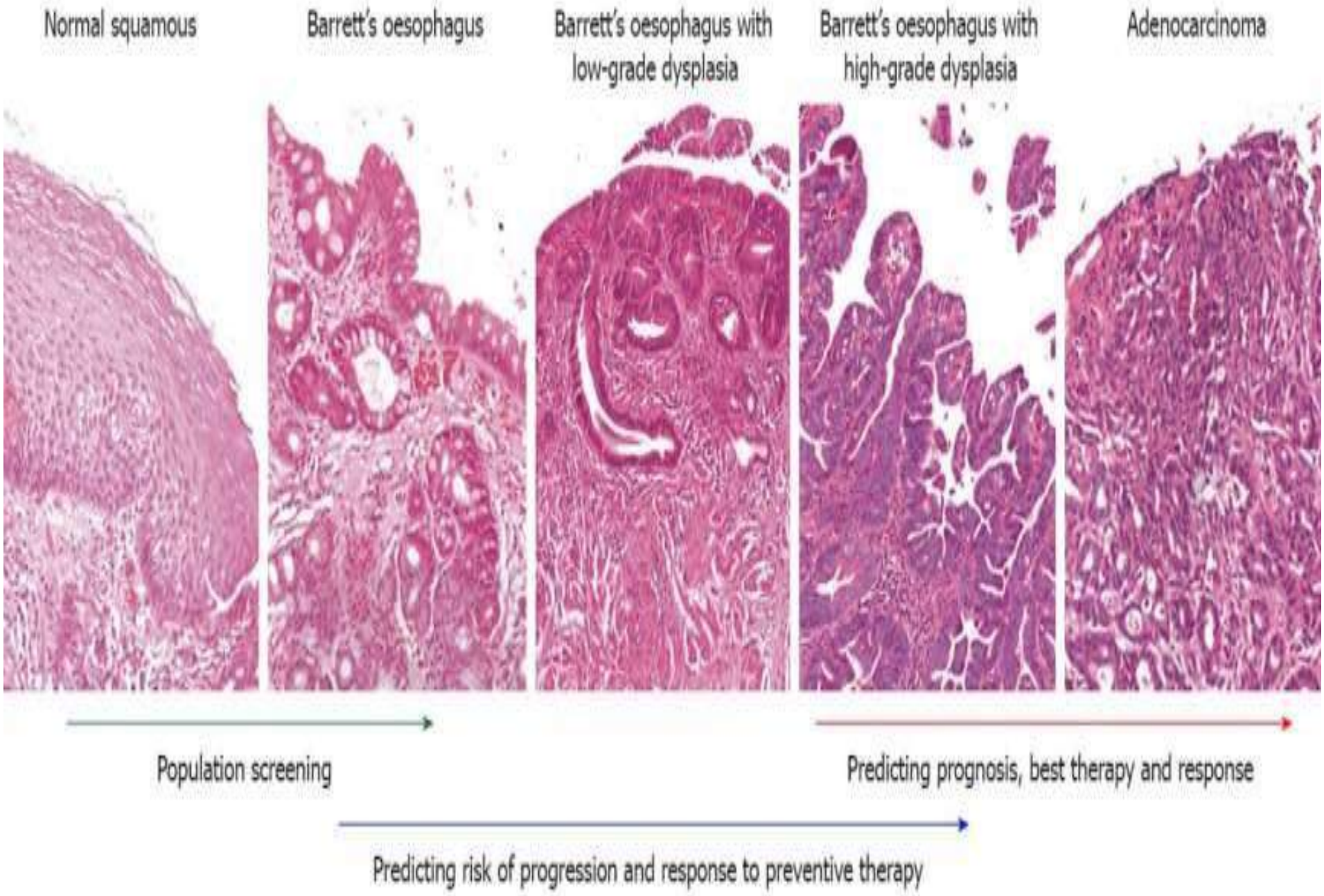
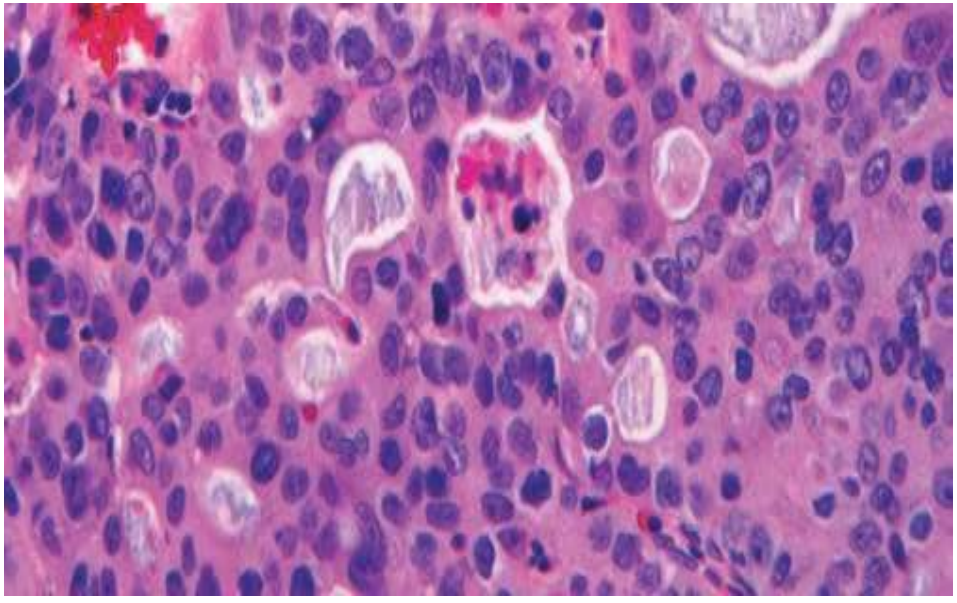


Morphology

- Distal third.
- Early: flat or raised patches
- Later: exophytic infiltrative masses
- Microscopy: Forms glands and mucin.



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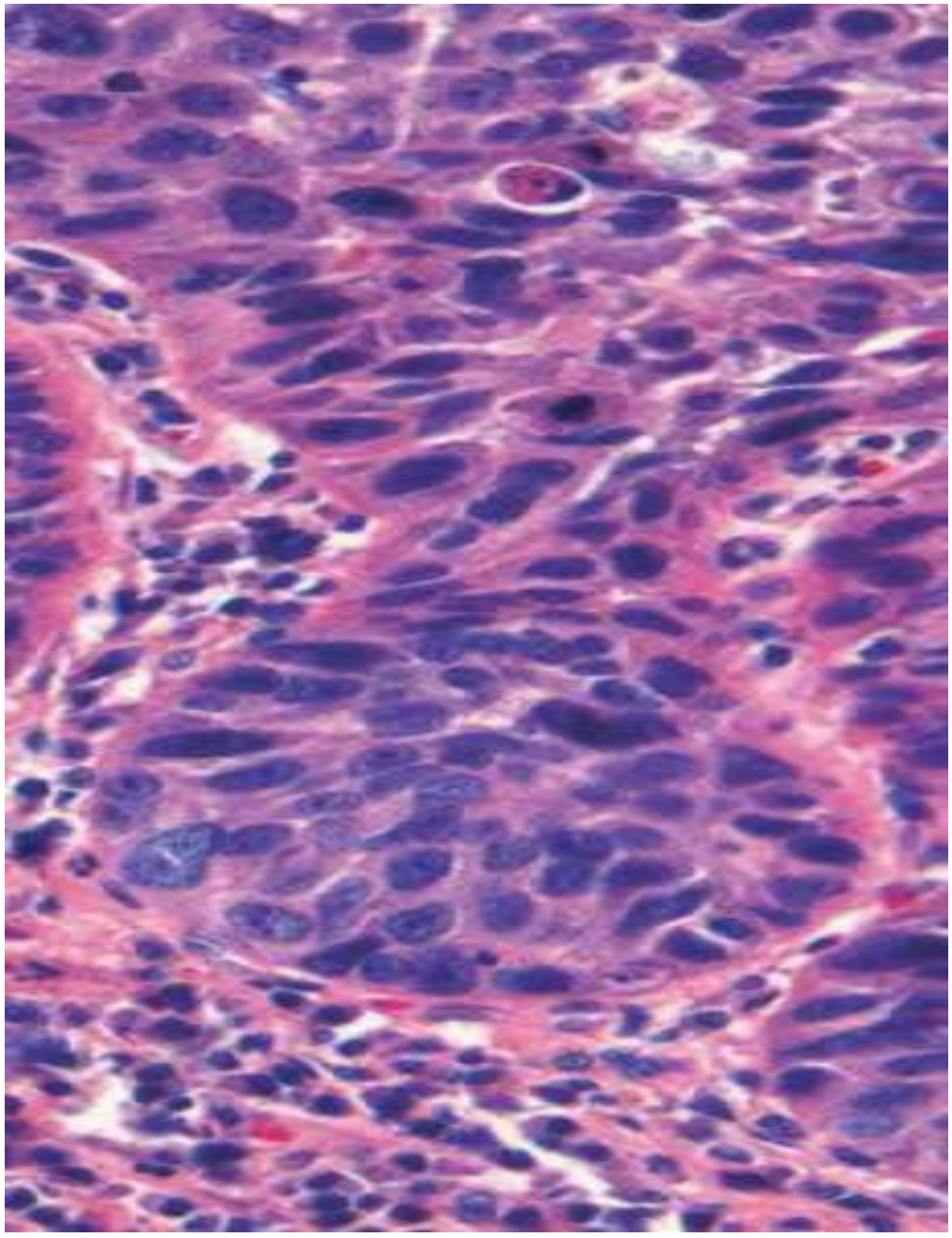

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Pathology of the stomach

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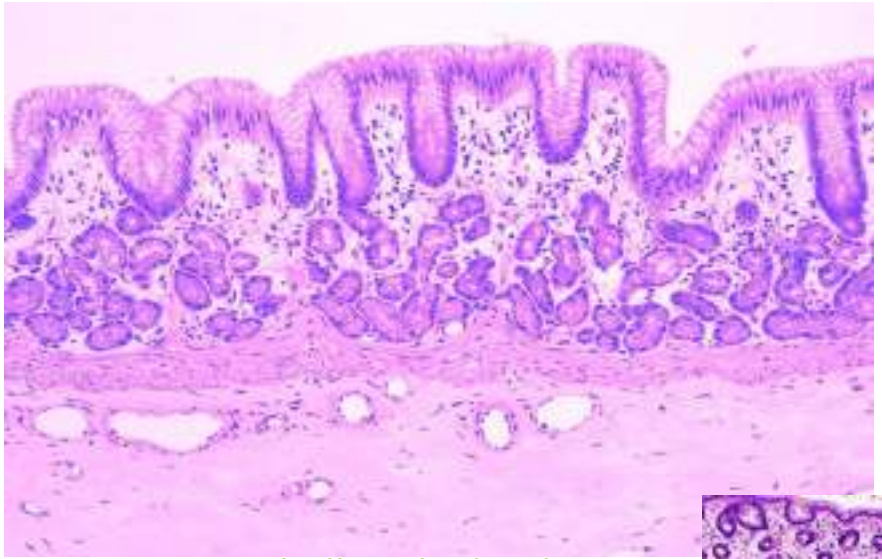
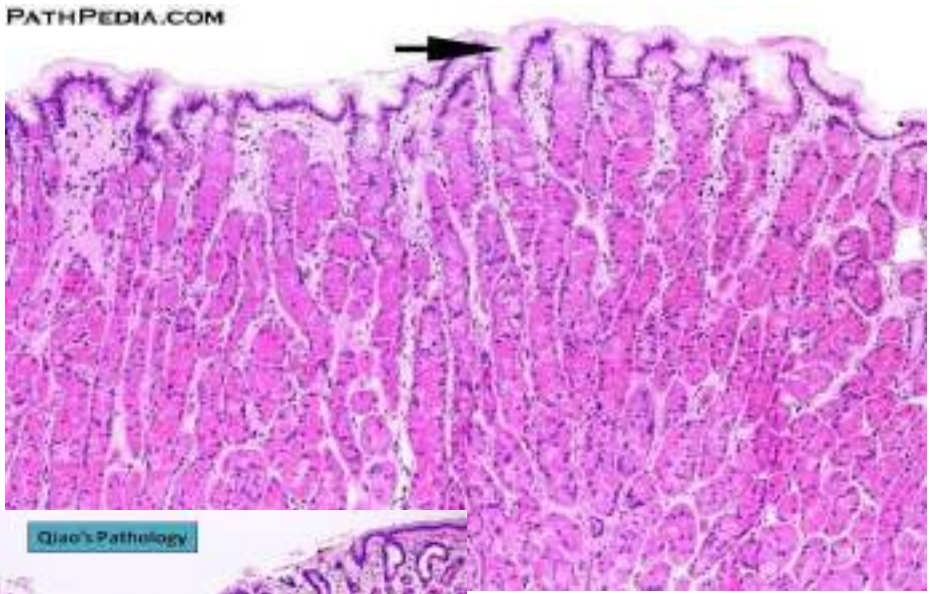
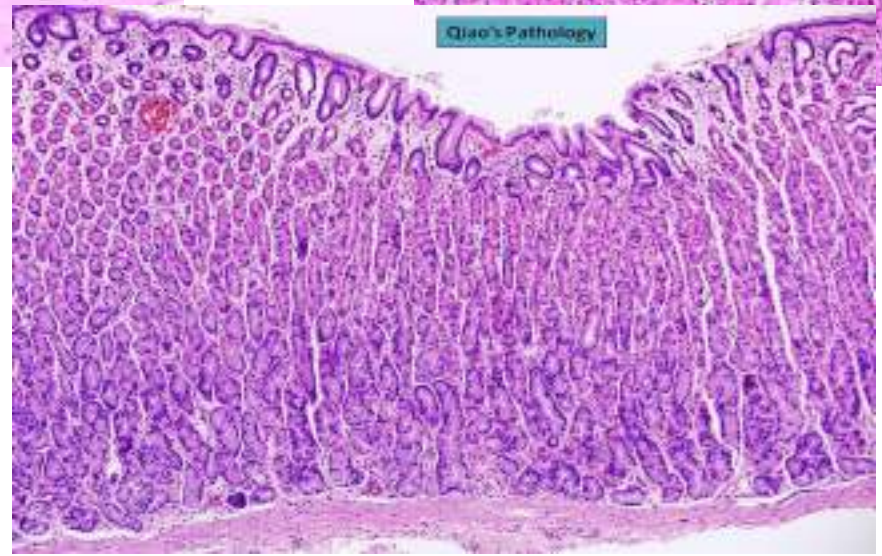
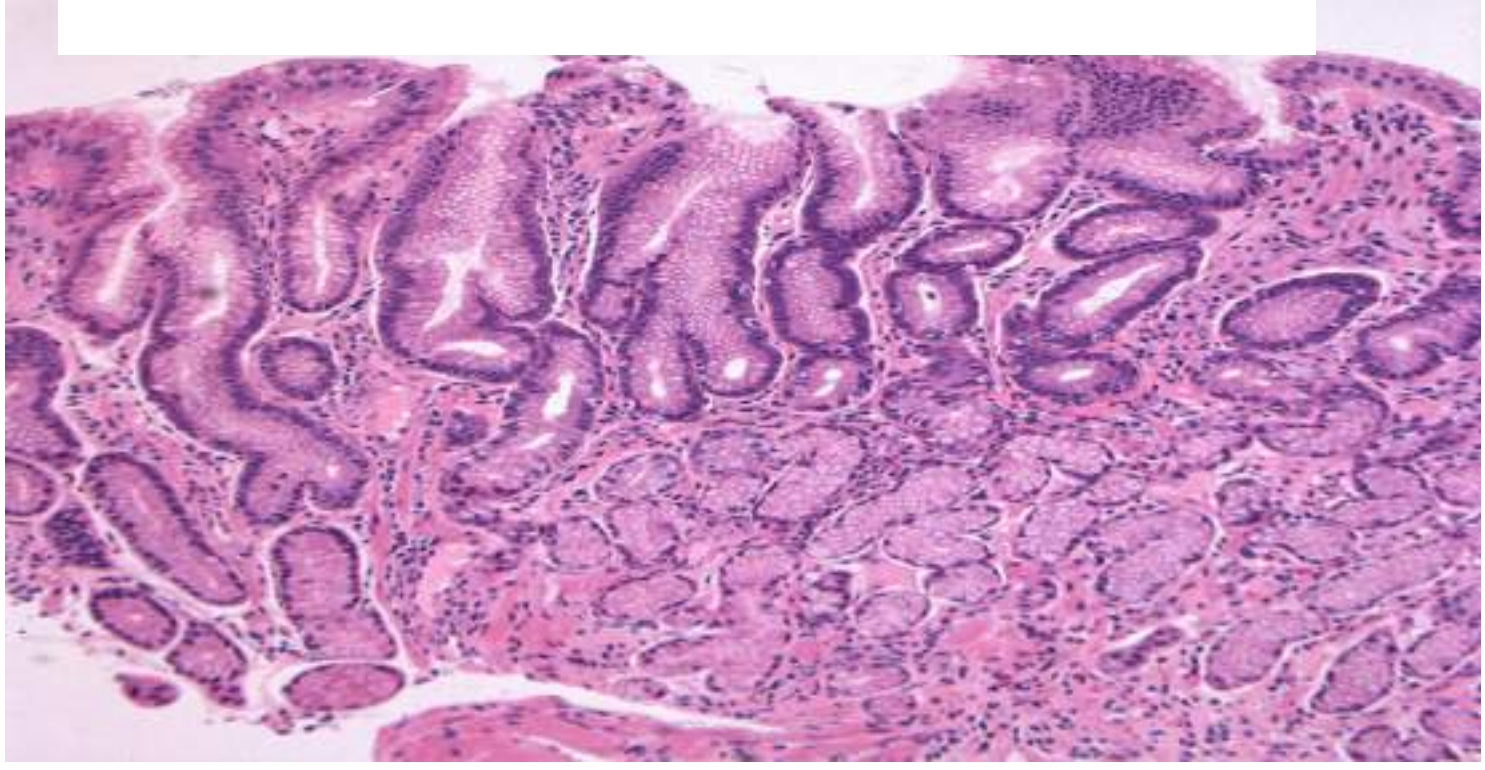


medcell.med.yale.edu





Antrum



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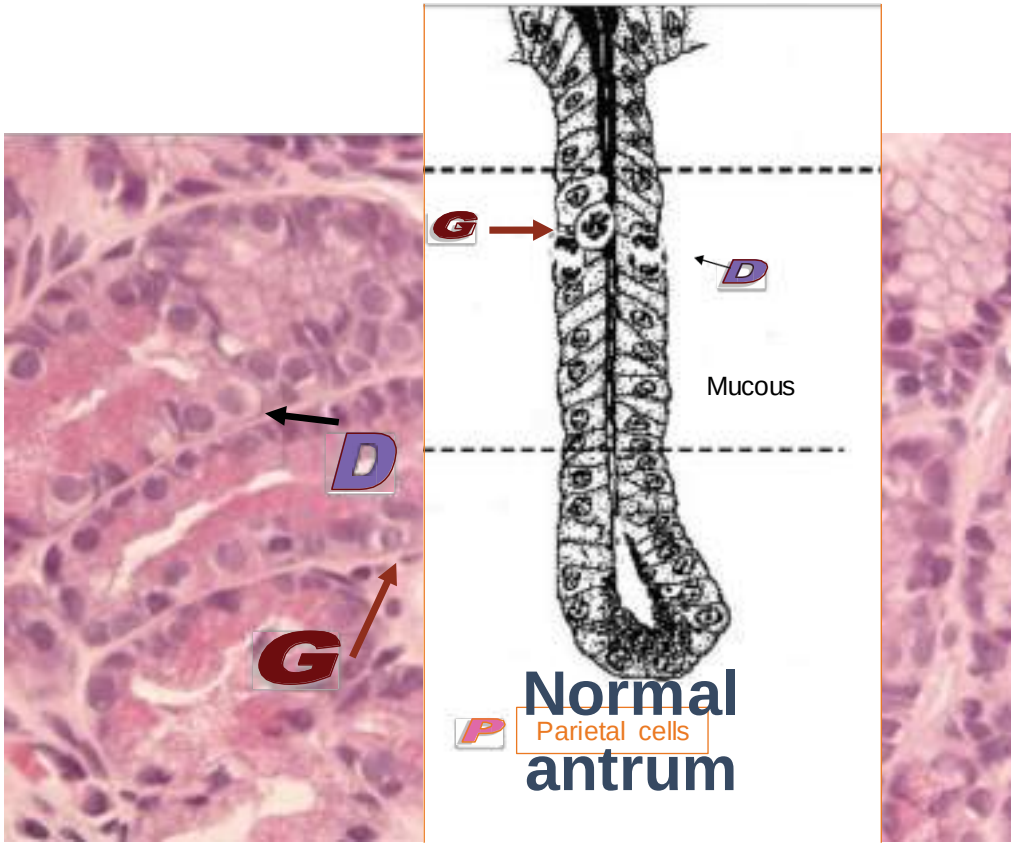
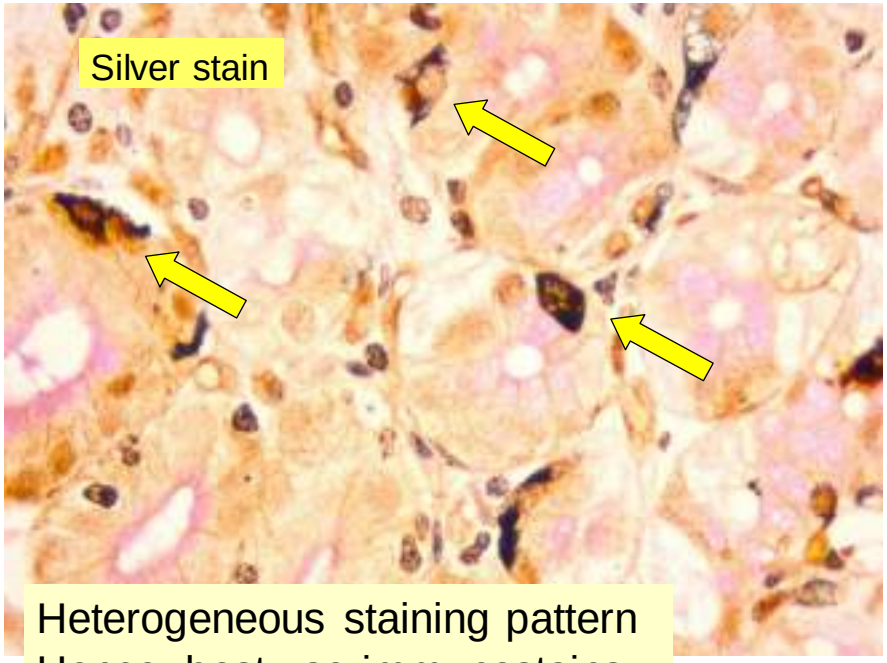
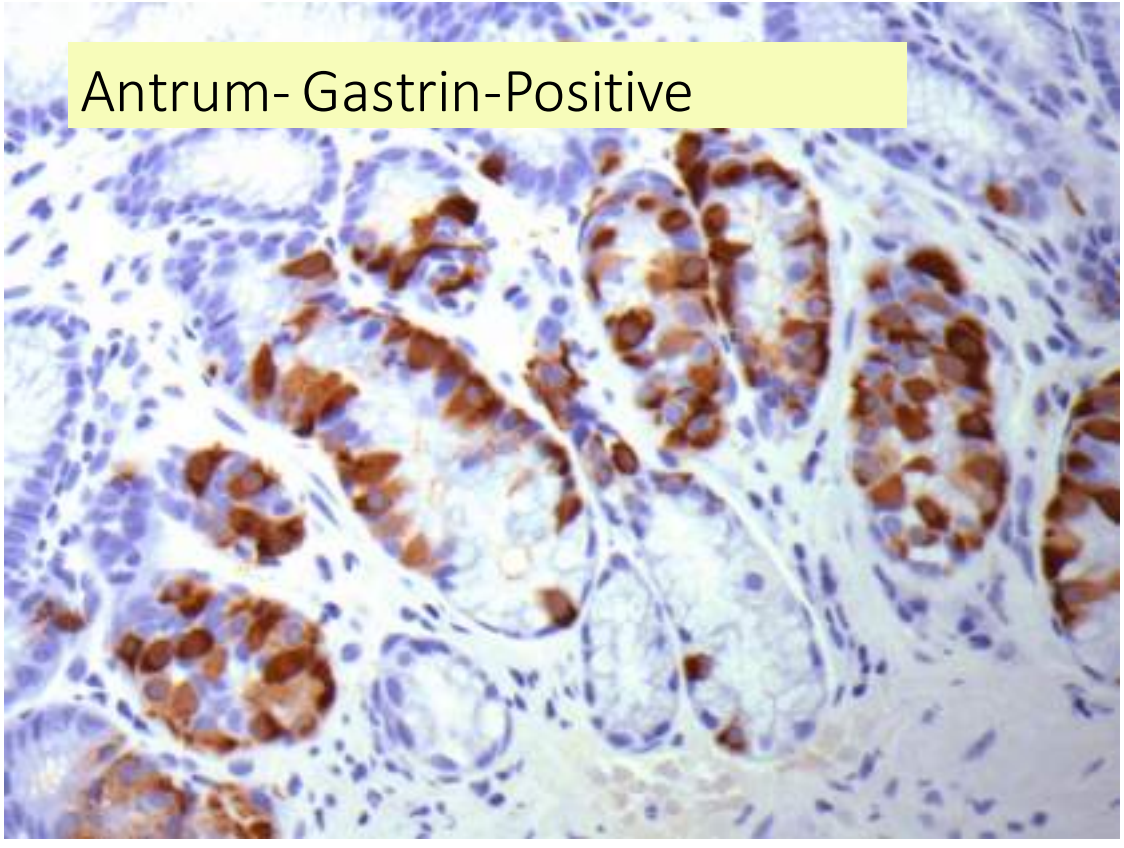
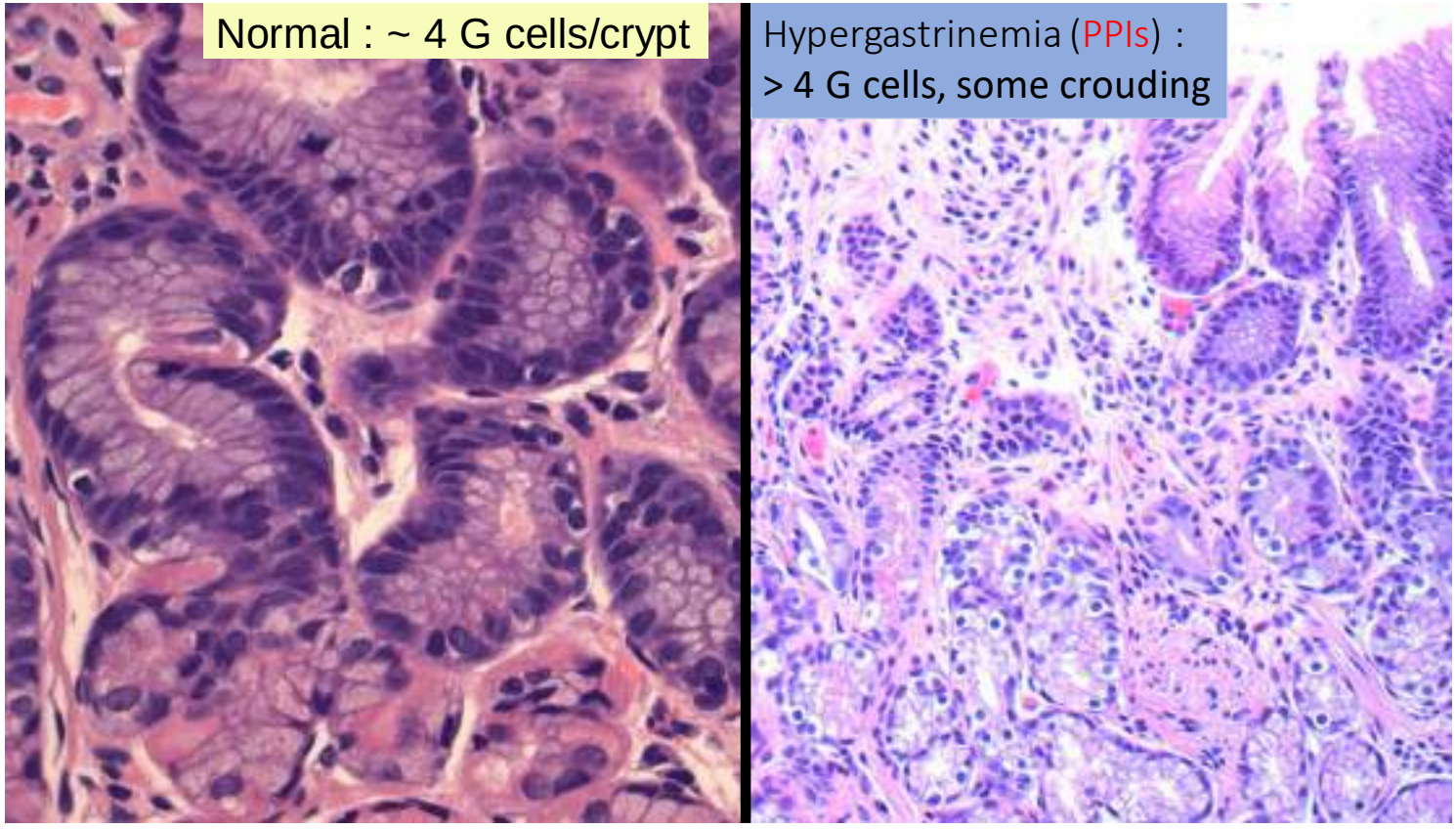


Antrum endocrine cells

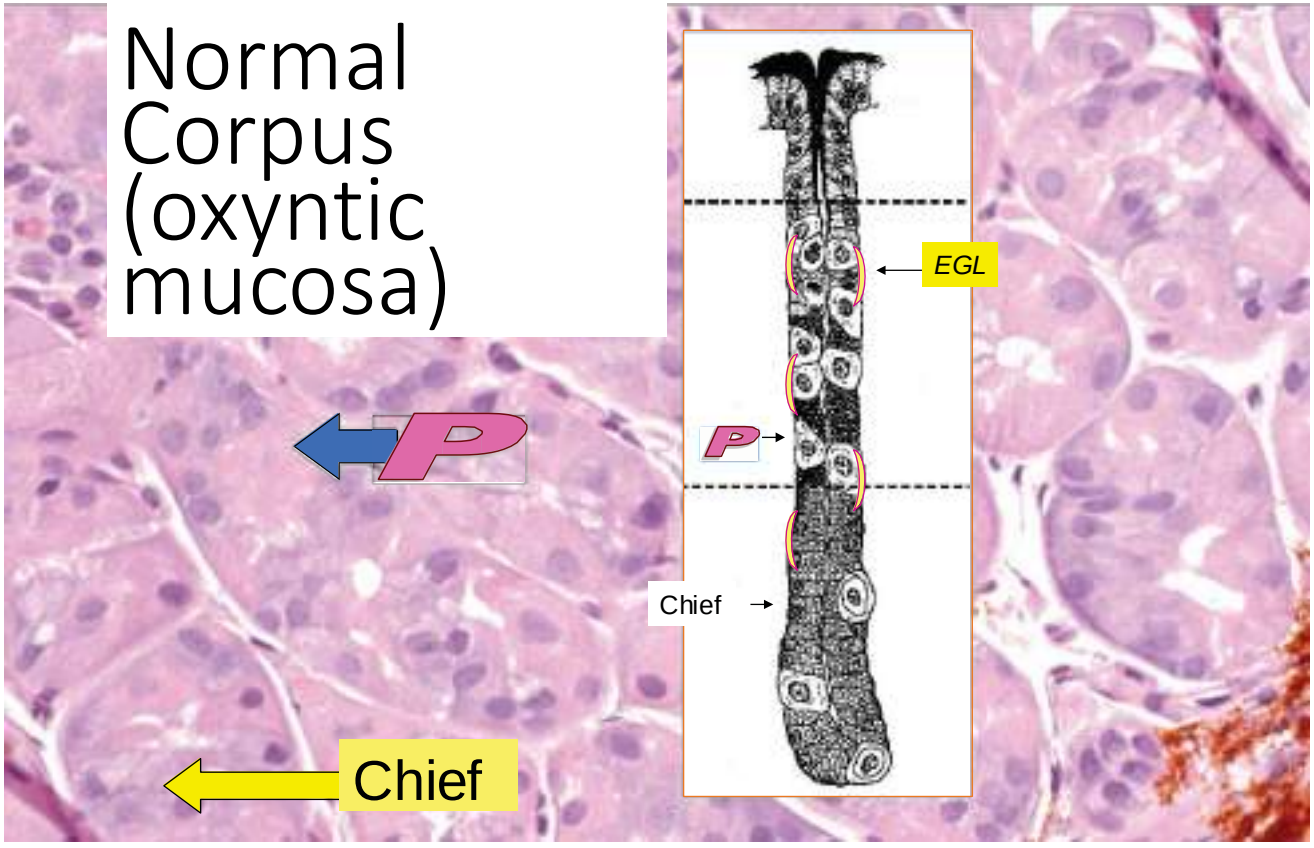


Heterogeneous staining pattern
Hence: best use immunostains

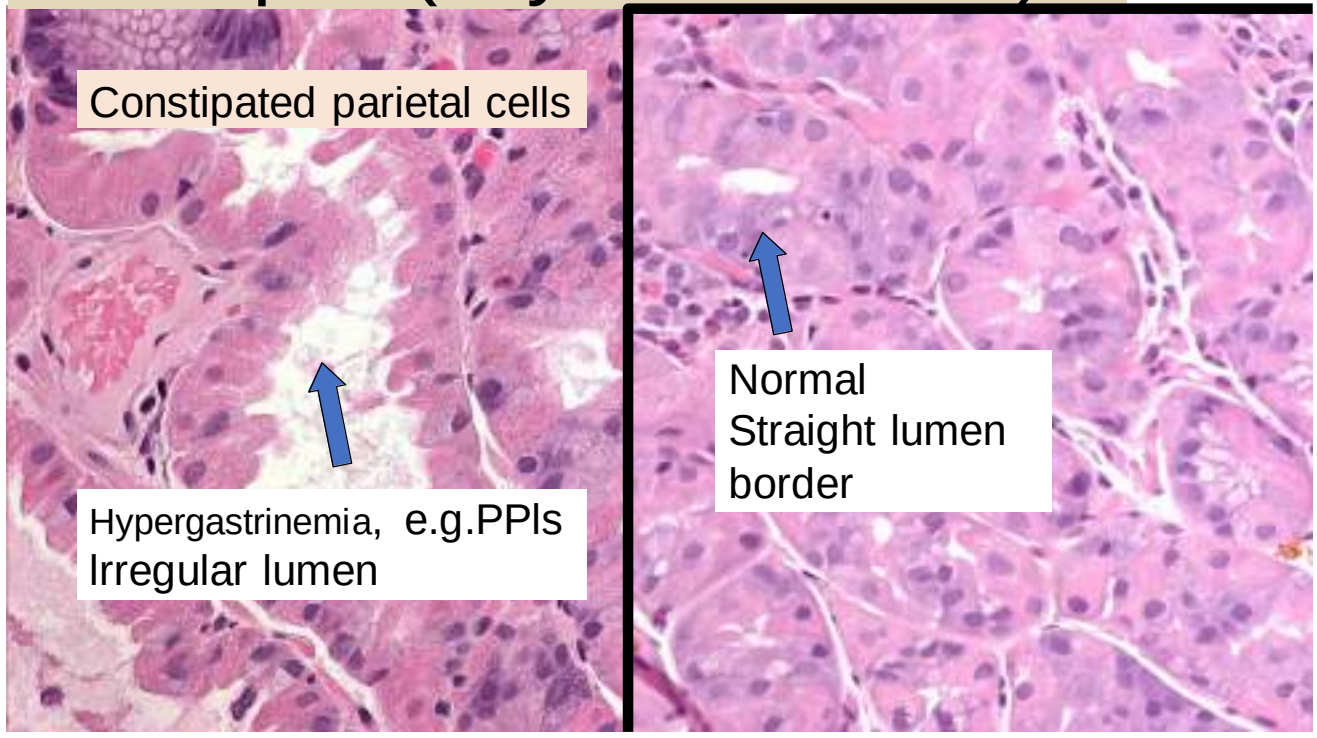

Antrum- Gastrin-Positive


Normal : ~ 4 G cells/crypt
Hypergastrinemia (PPIs) :
> 4 G cells, some crouding

Normal Corpus (oxyntic mucosa)

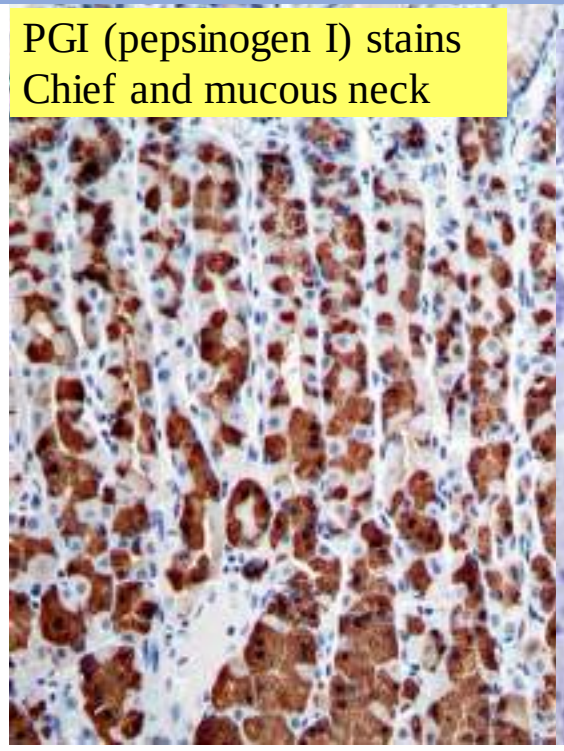


Corpus (oxyntic mucosa)



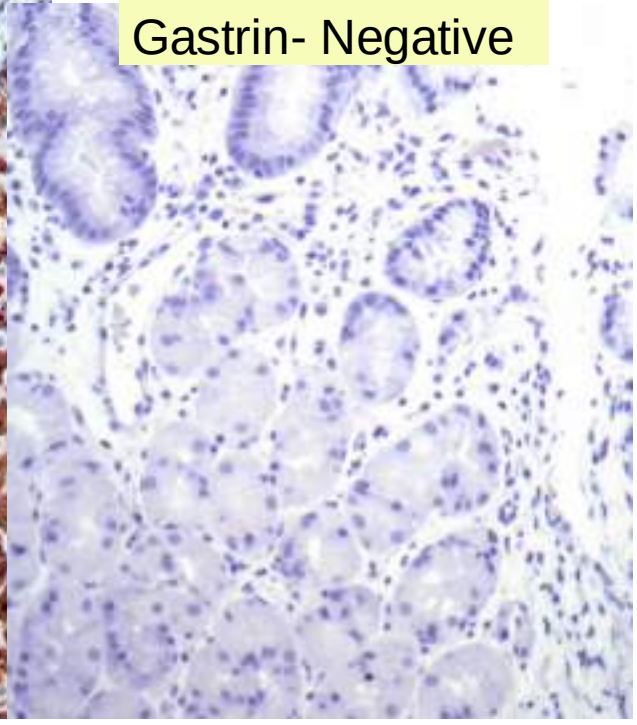
NORMAL CORPUS

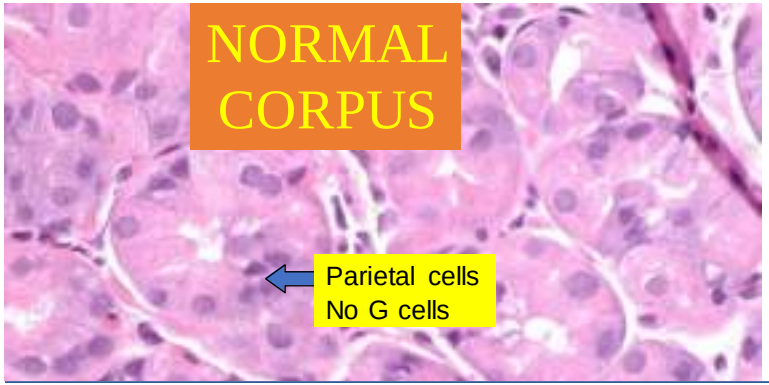
PGI (pepsinogen I) stains Chief and mucous neck



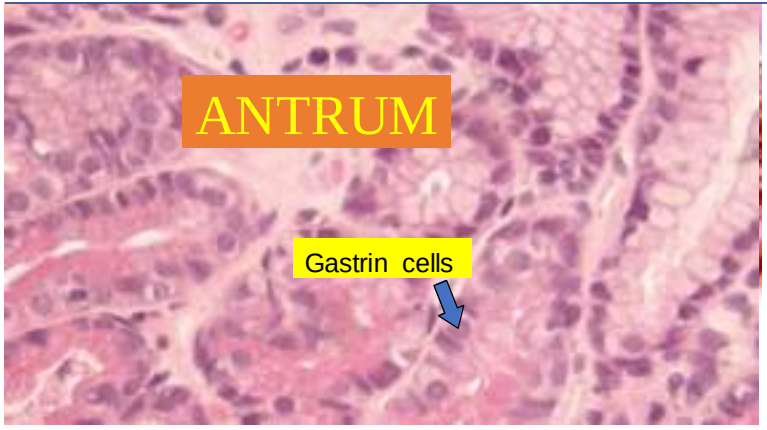
Oxyntic endocrine cells make histamine or ghrelin, not gastrin

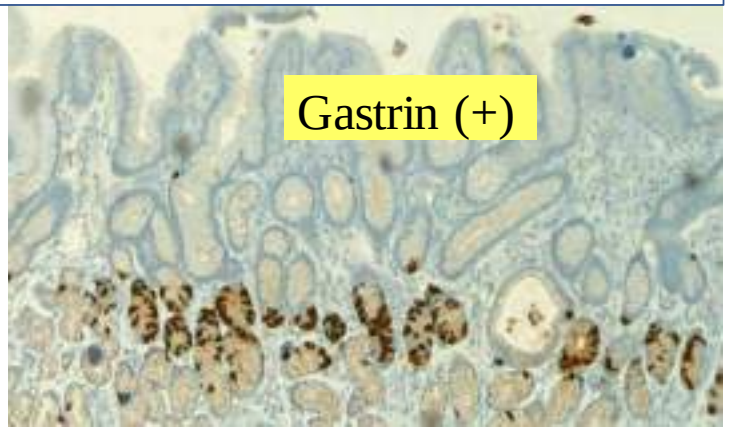
Gastrin- Negative



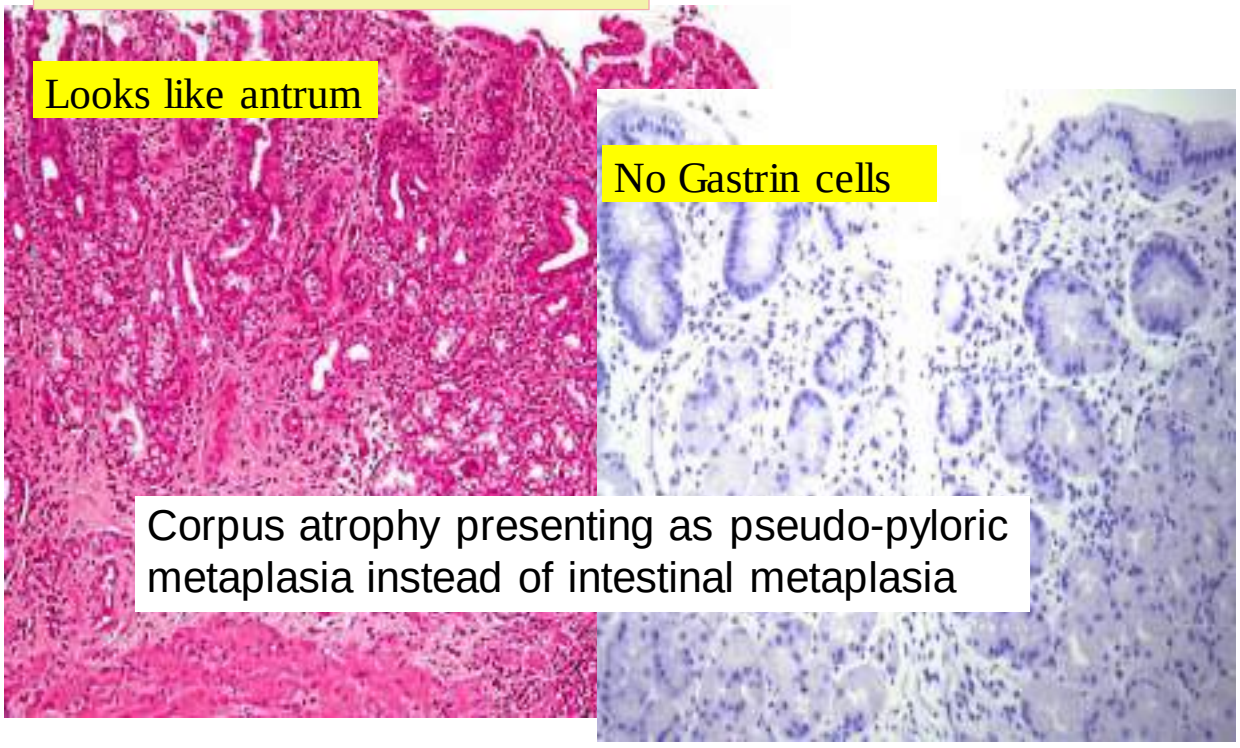

NORMAL CORPUS
Parietal cells
No G cells


Gastrin (-)


ANTRUM
Gastrin cells

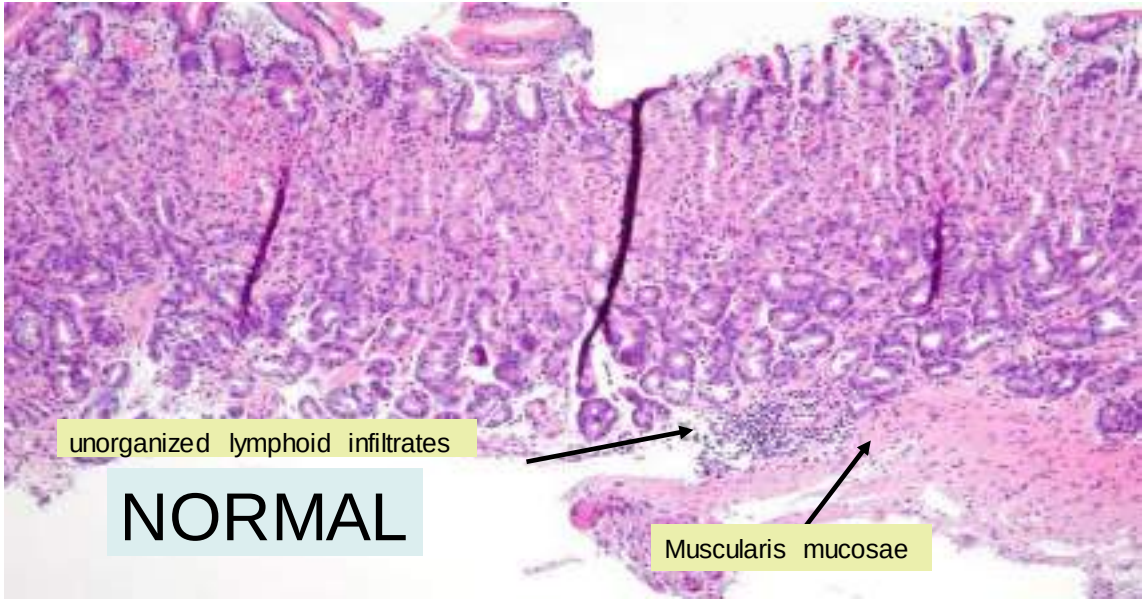

Gastrin (+)

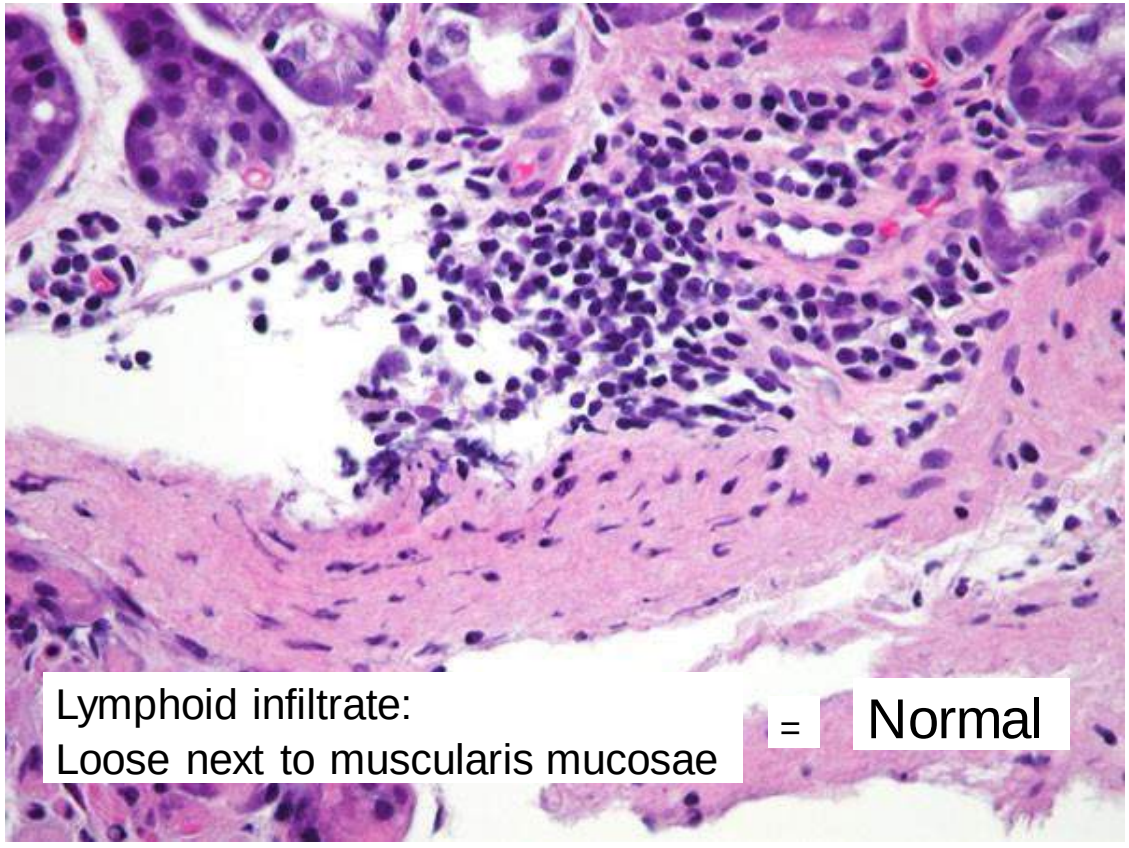
? Clinical relevance



Corpus atrophy presenting as pseudo-pyloric metaplasia instead of intestinal metaplasia

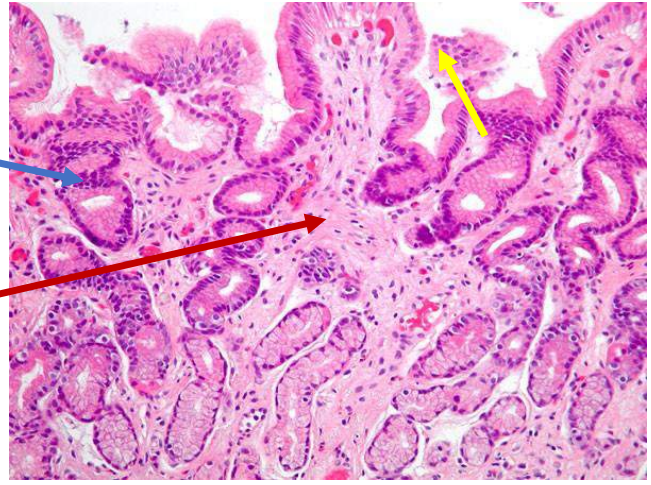
Landmark for NORMAL lymphoid infitrates
Musclaris mucosae



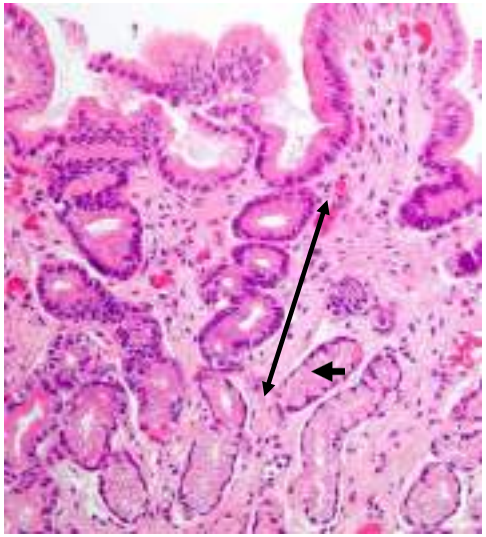

Lymphoid infiltrate:
Loose next to muscularis mucosae
=
Normal

Reactive/Chemical Gastropathy Triad

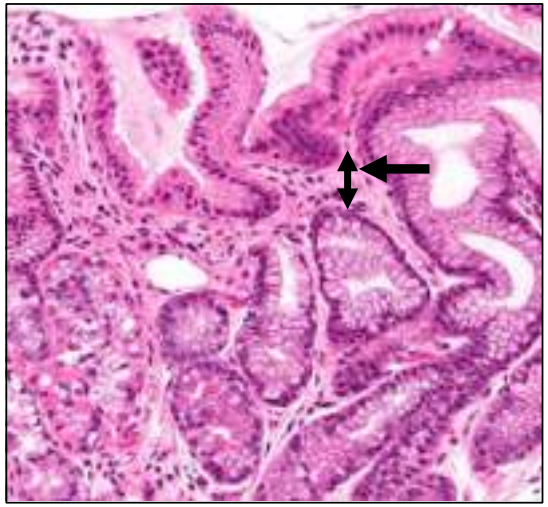
1. Foveolar hyperplasia
2. Smooth muscle fiber hyperplasia
3. Paucity of acute and chronic inflammatory cells



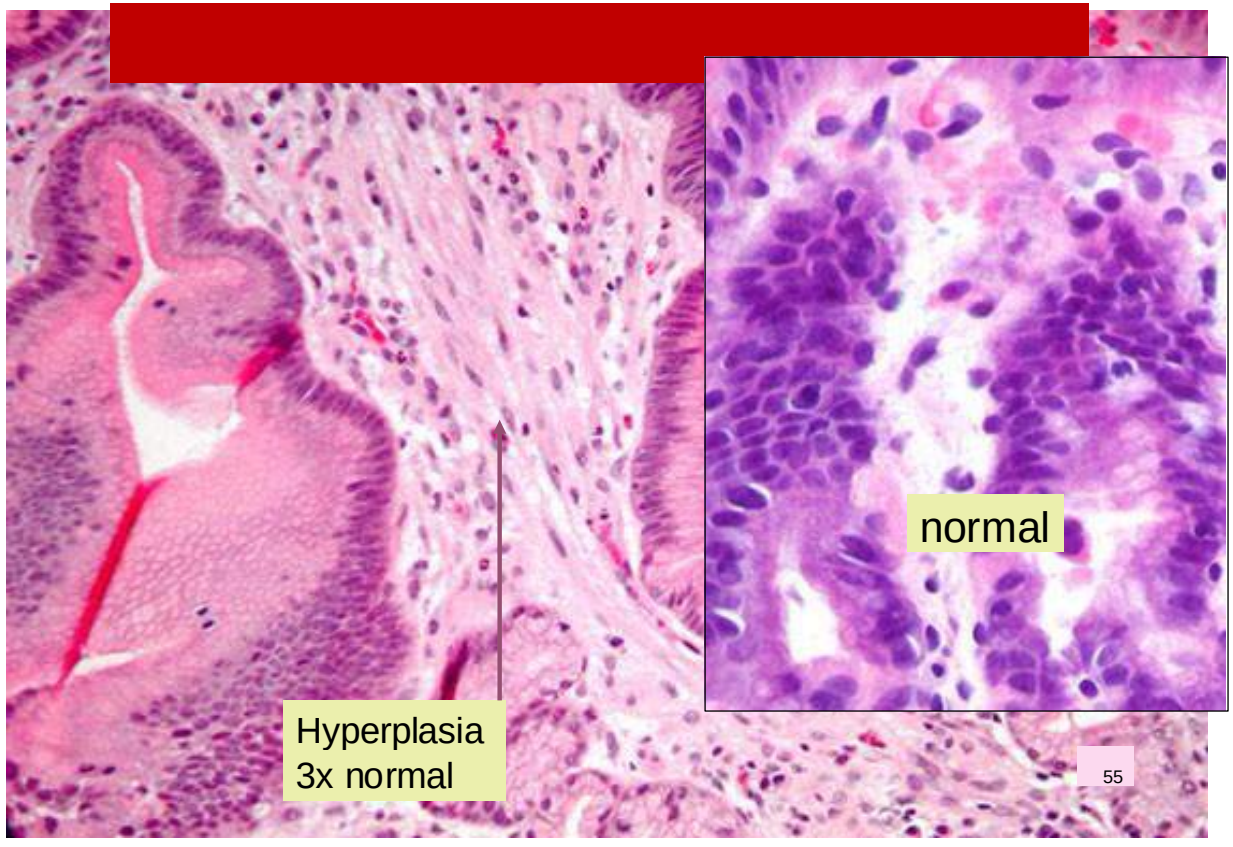
How to identify foveolar hyperplasia?
(length of neck region)

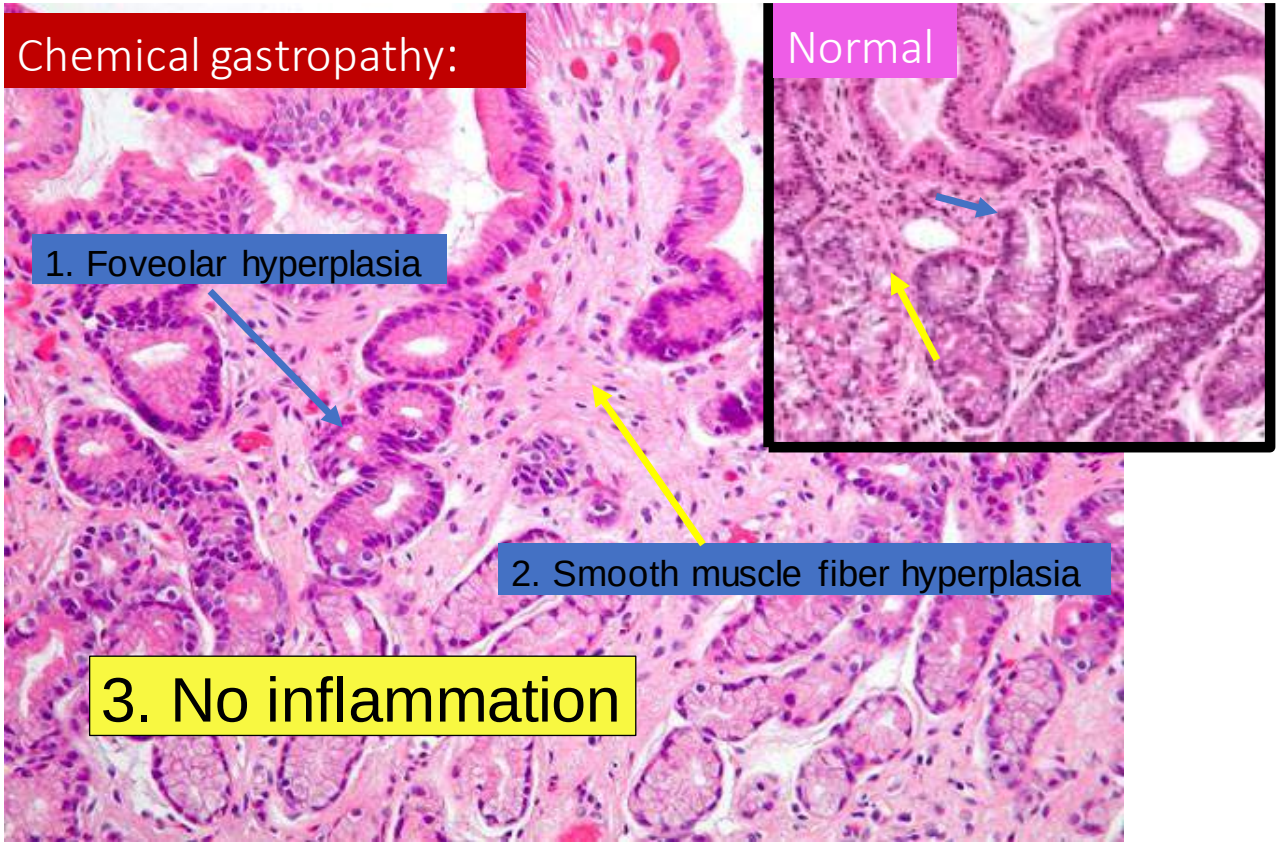


Foveolar hyperplasia
Long & tortuous
Up to 3 x normal



Normal

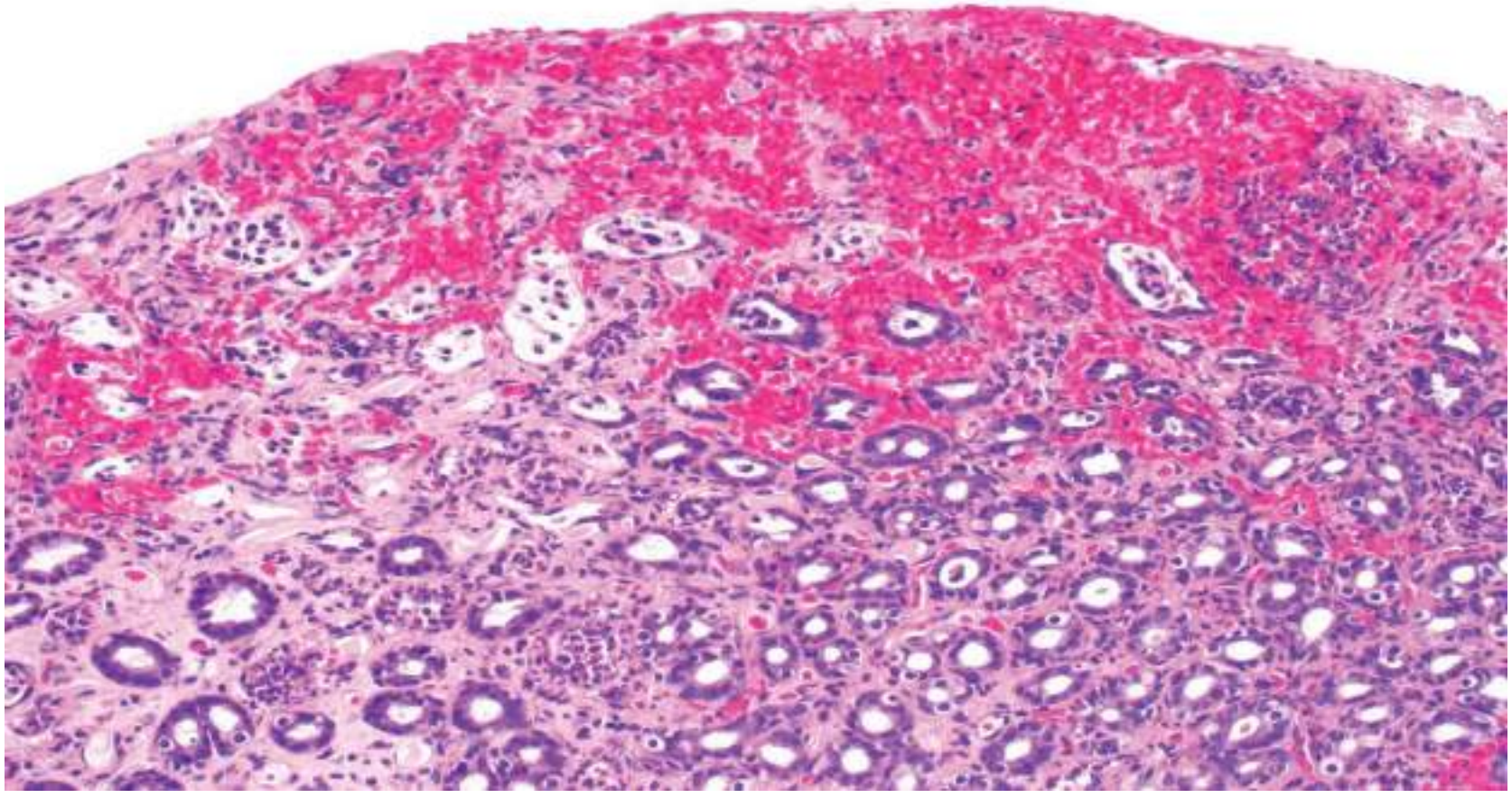

normal
Hyperplasia
3x normal


Chemical gastropathy:
Normal
1. Foveolar hyperplasia
2. Smooth muscle fiber hyperplasia
3. No inflammation

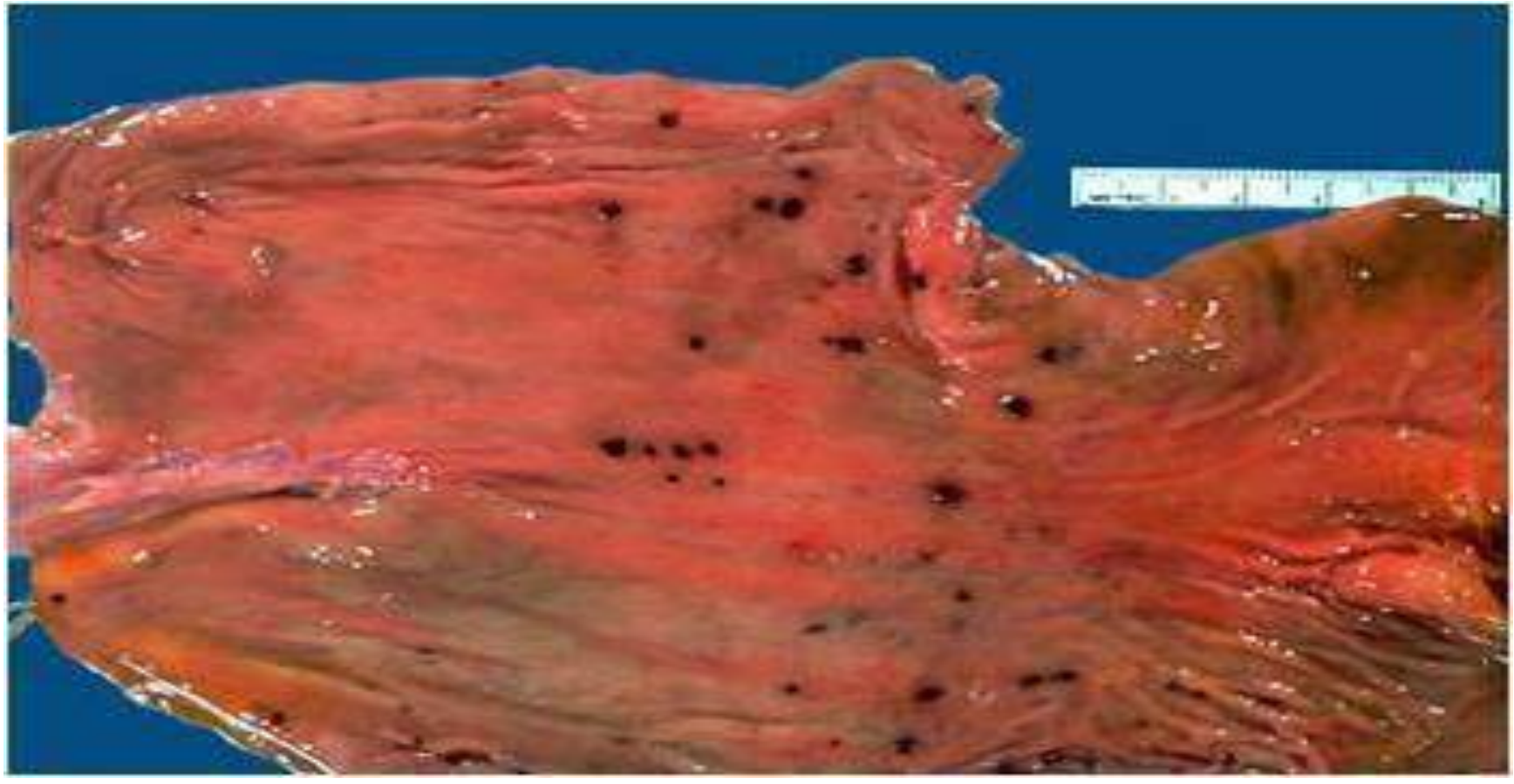
Acute gastritis

www.etttamen.com

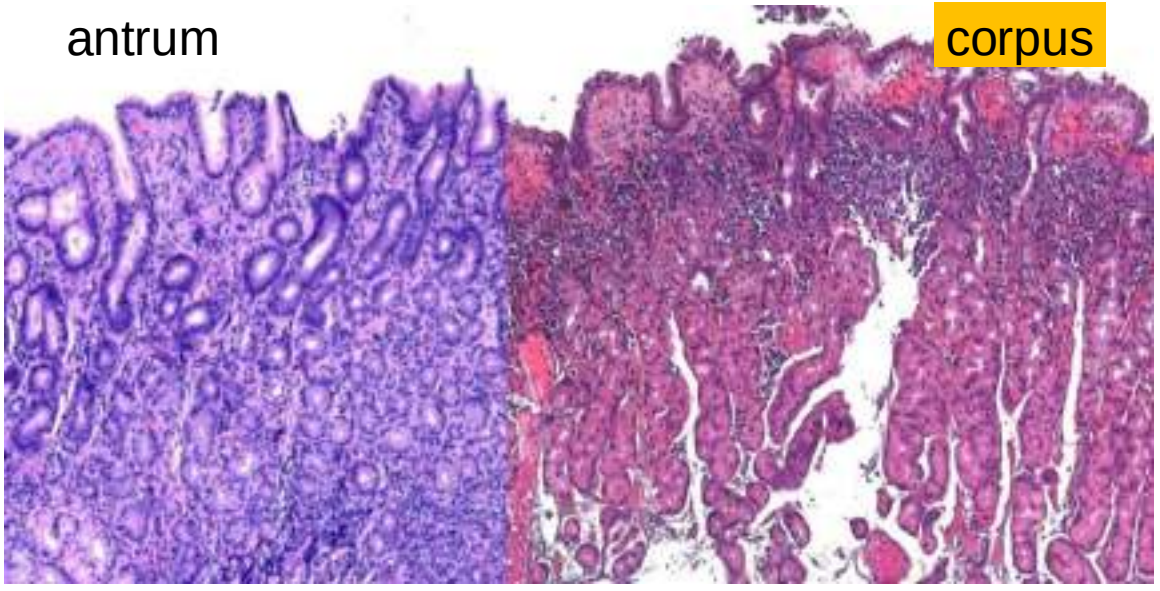
B



librepathology.org

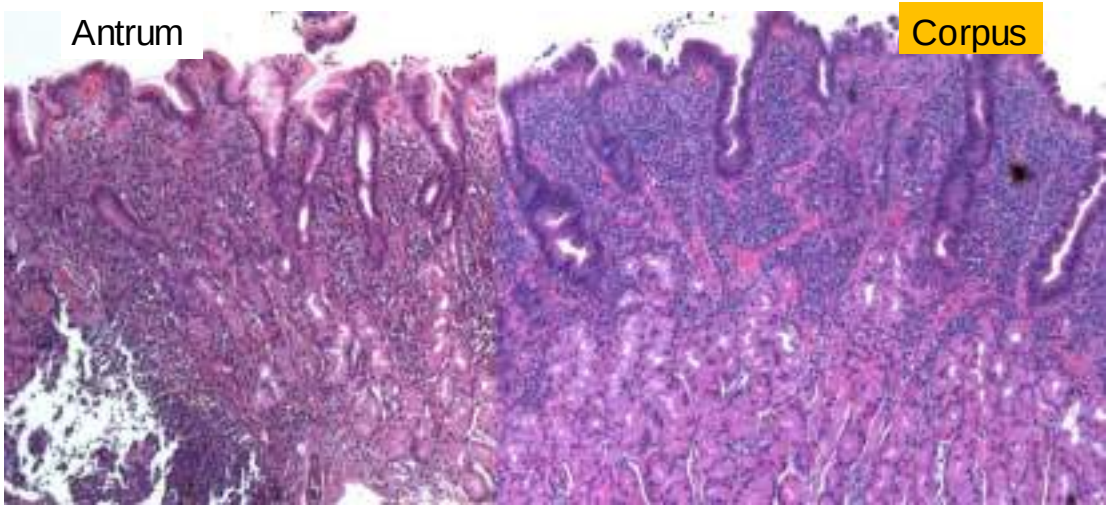


Sustained H. Pylori Gastritis



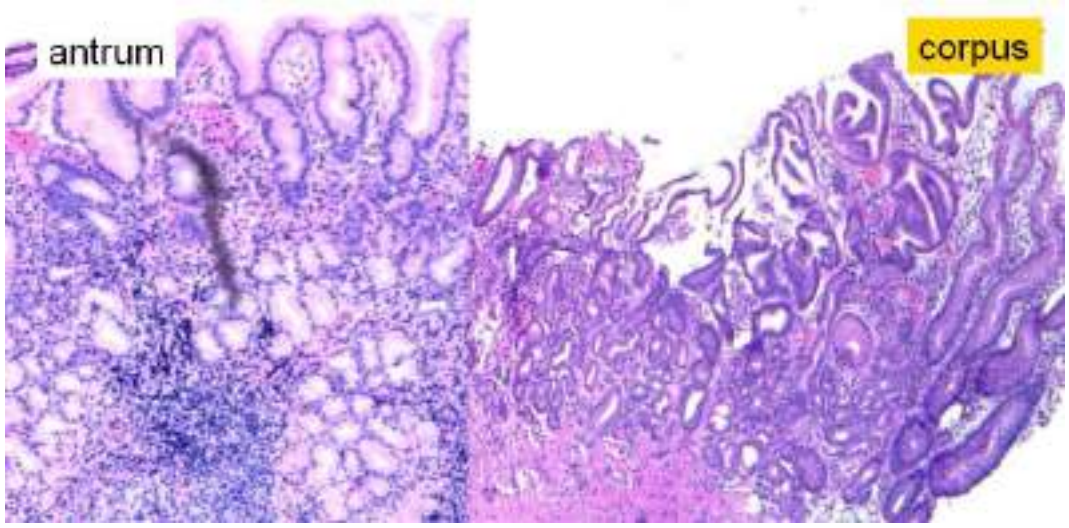
Pan Gastritis with superficial inflammation in the corpus

And later H. Pylori Gastritis

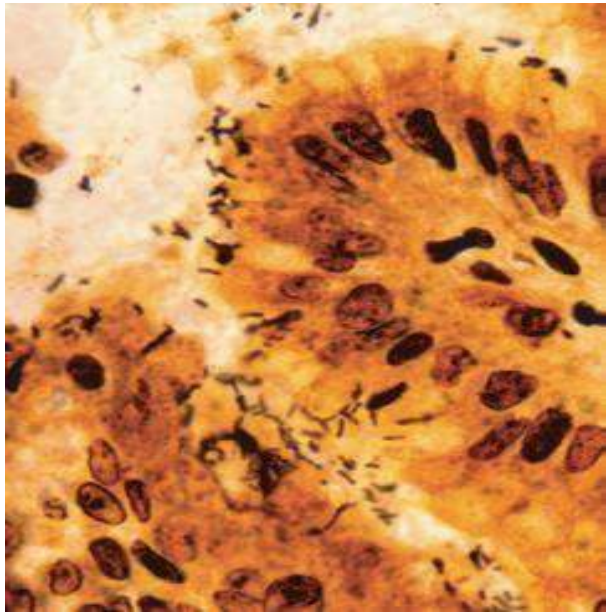


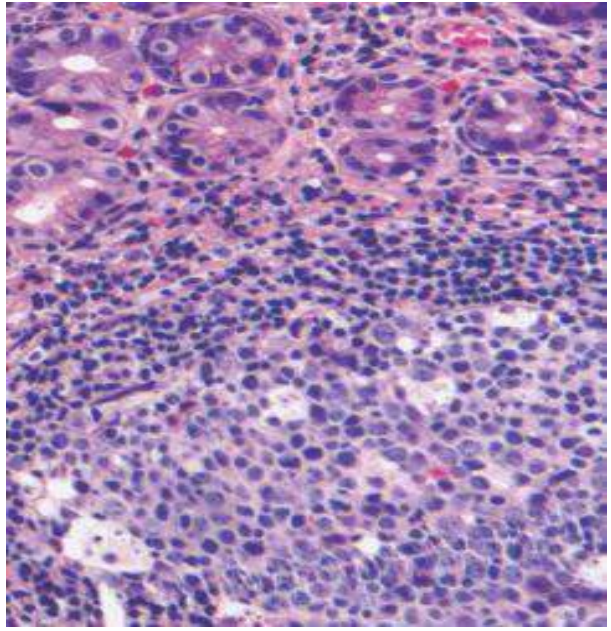
Pan-Gastritis with deeper inflammation in ! corpus

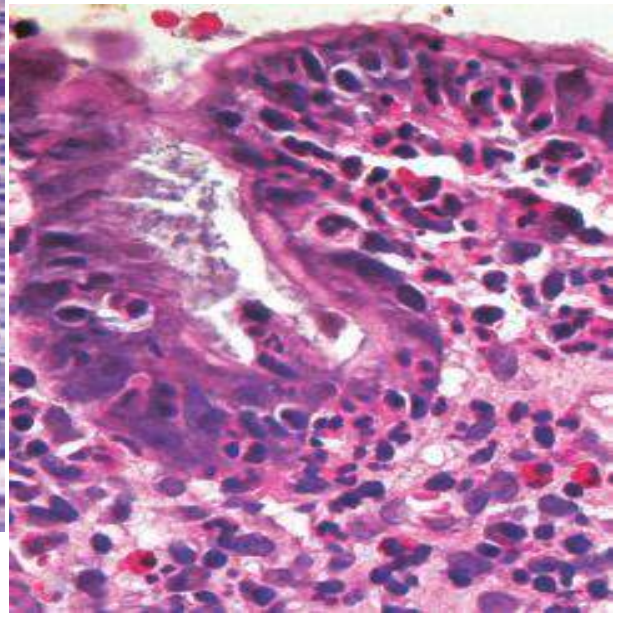
Too Late H. Pylori gastritis



Pan Gastritis with corpus atrophy
With increased risk for gastric carcinoma







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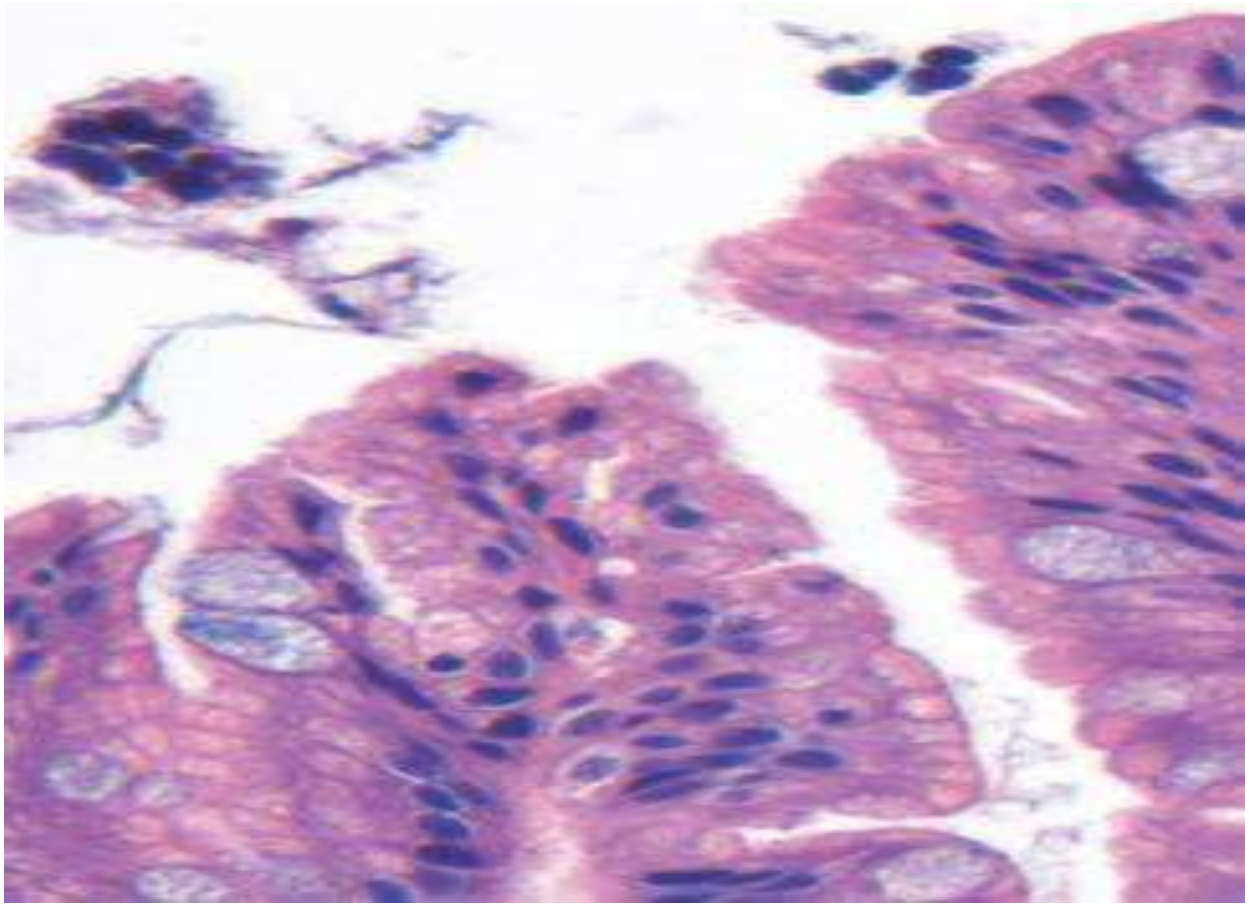
Intestinal metaplasia



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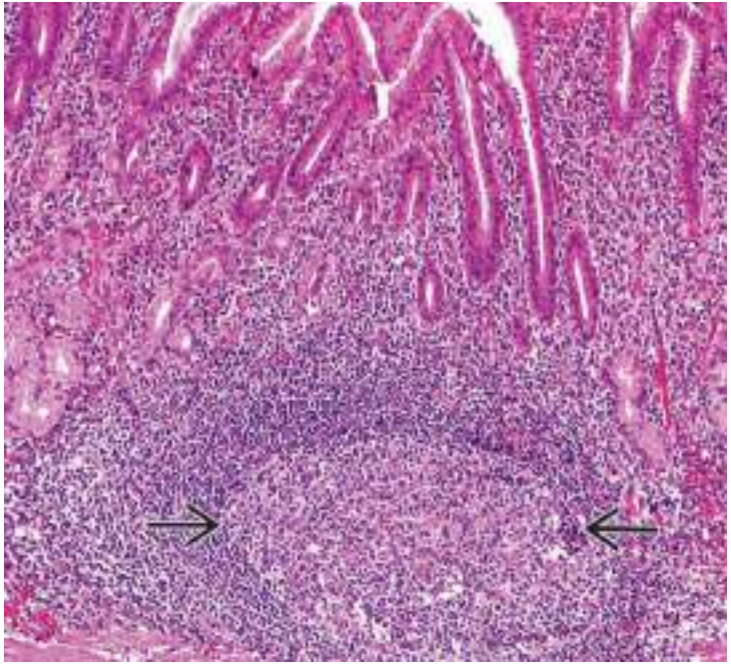
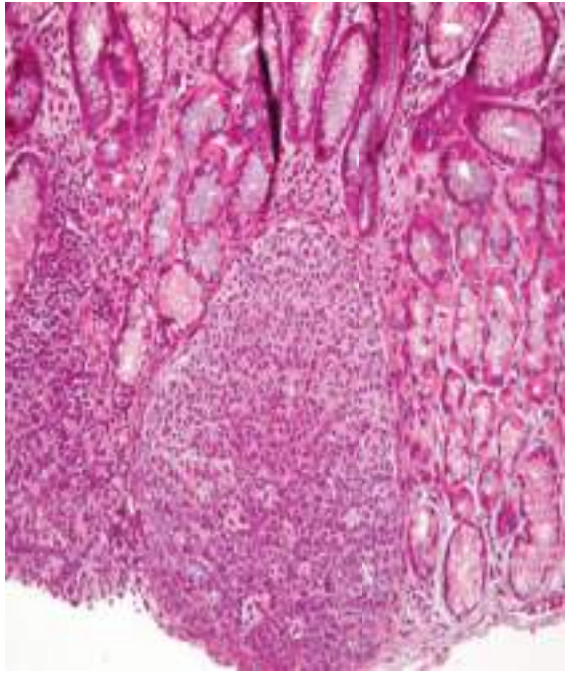
Lymphoid Follicles in *H. pylori* infection

Pre-treatment



Post treatment



PATHOLOGY OF THE STOMACH

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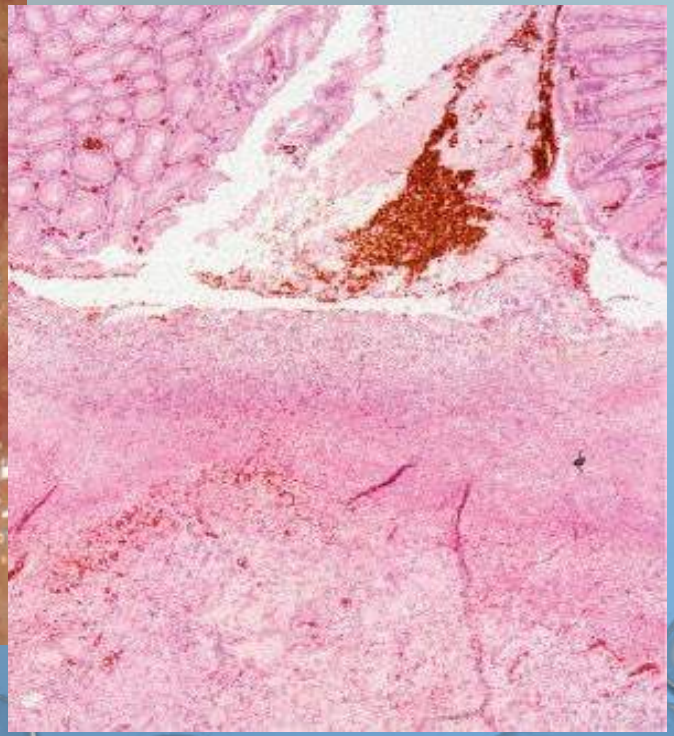
SCHOOL OF MEDICINE-PATHOLOGY DEPARTMENT

UNDERGRADUATE LECTURES 2023



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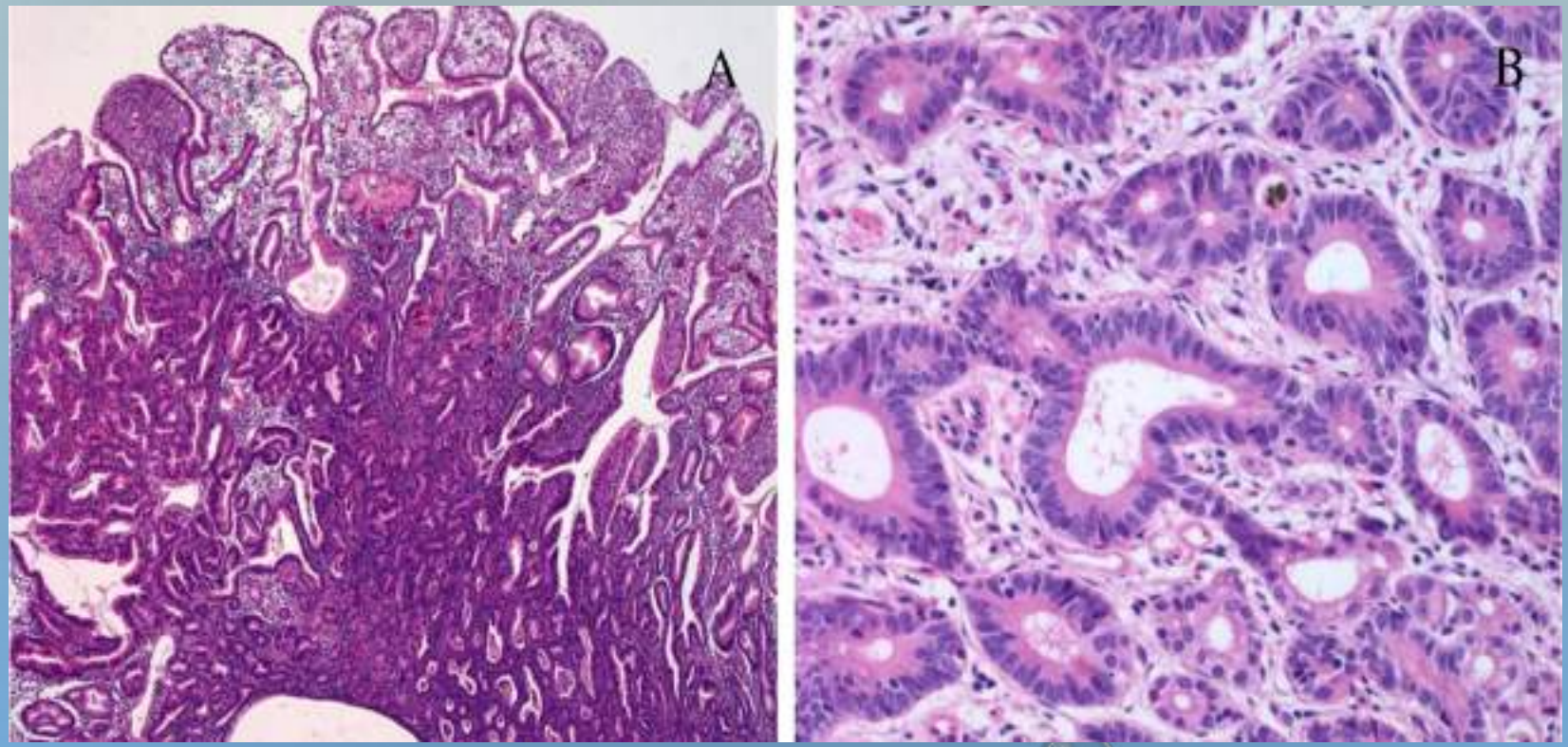
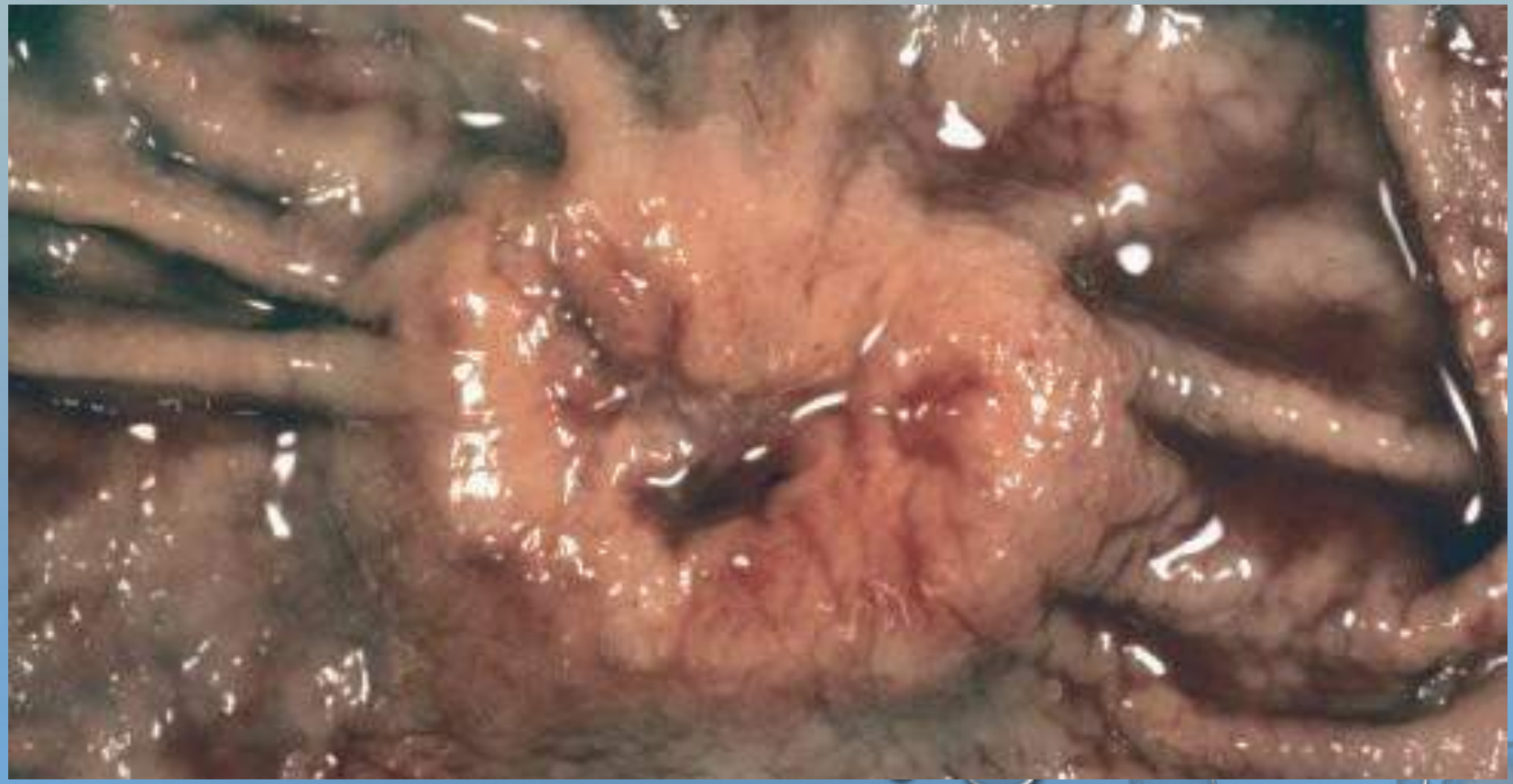
DUODENAL ULCER

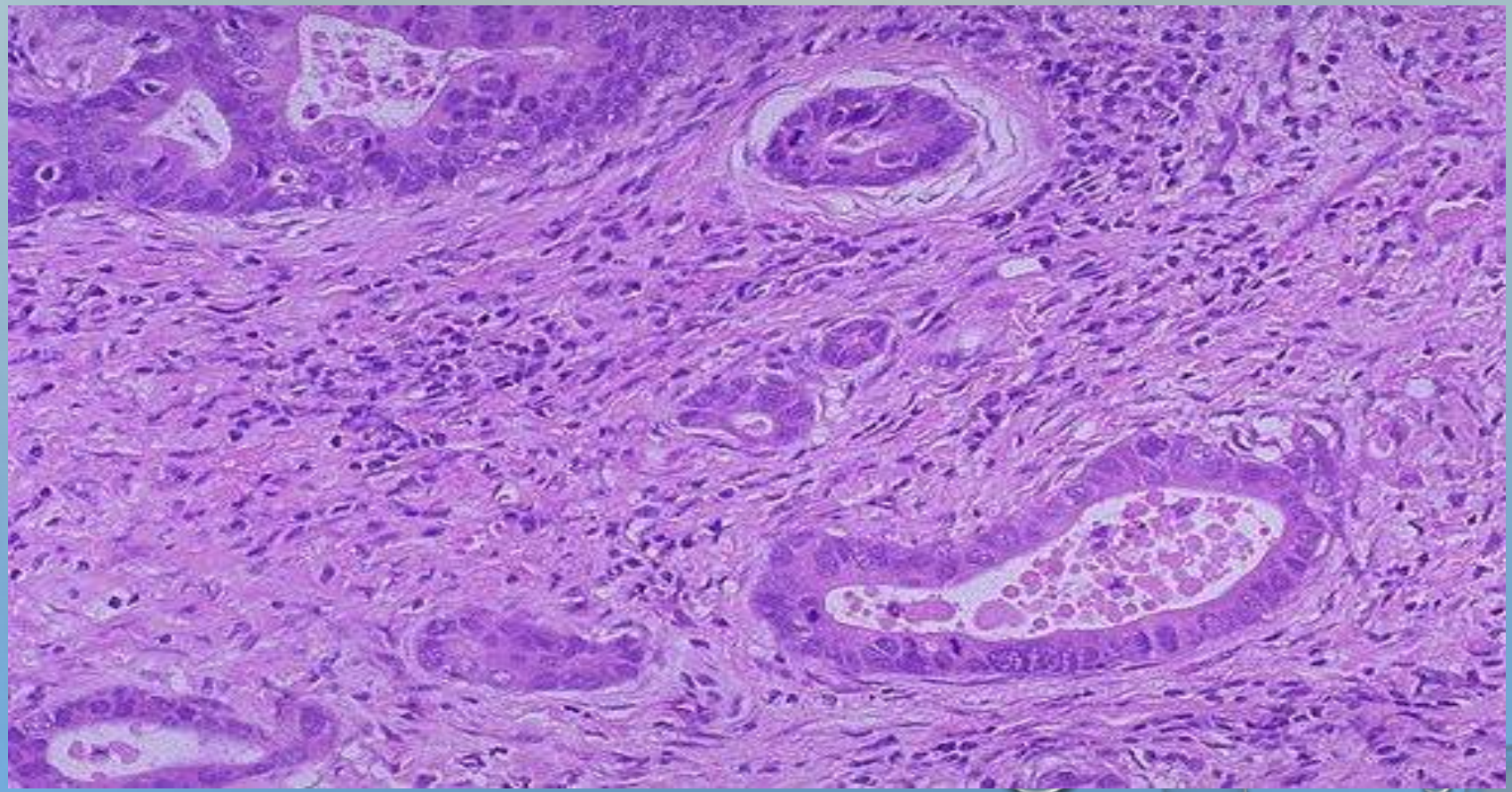
GASTRIC ADENOMA



INTESTINAL TYPE



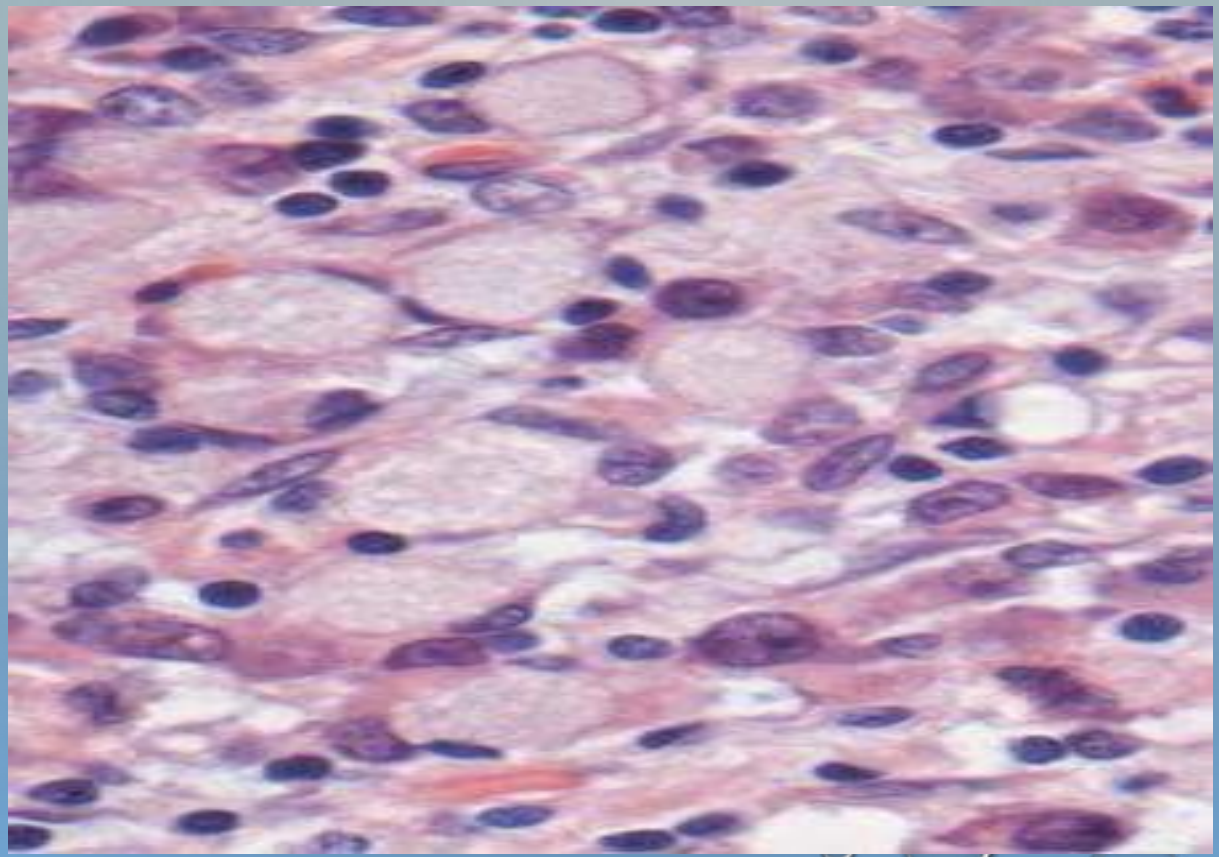
INTESTINAL TYPE



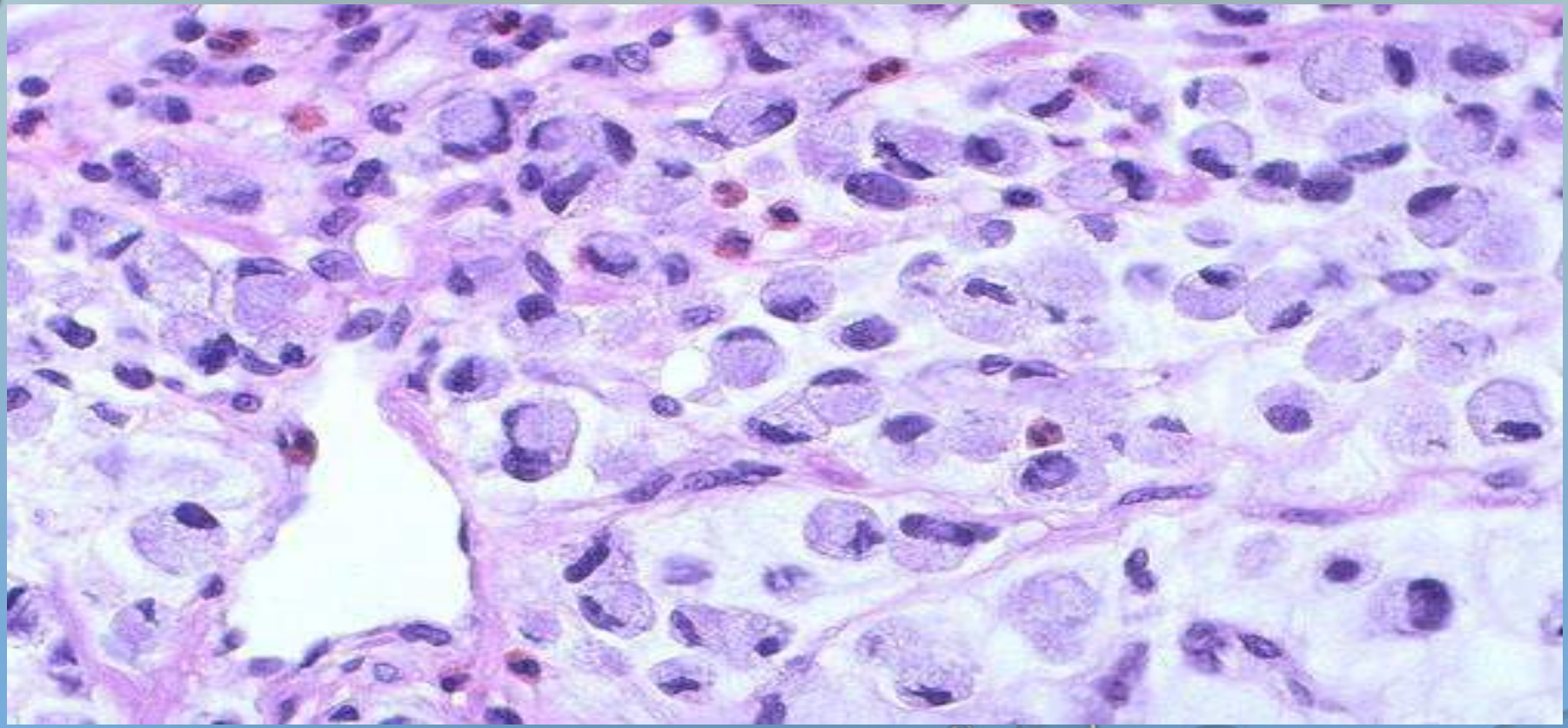
LINITIS PLASTICA

Signet ring cells:

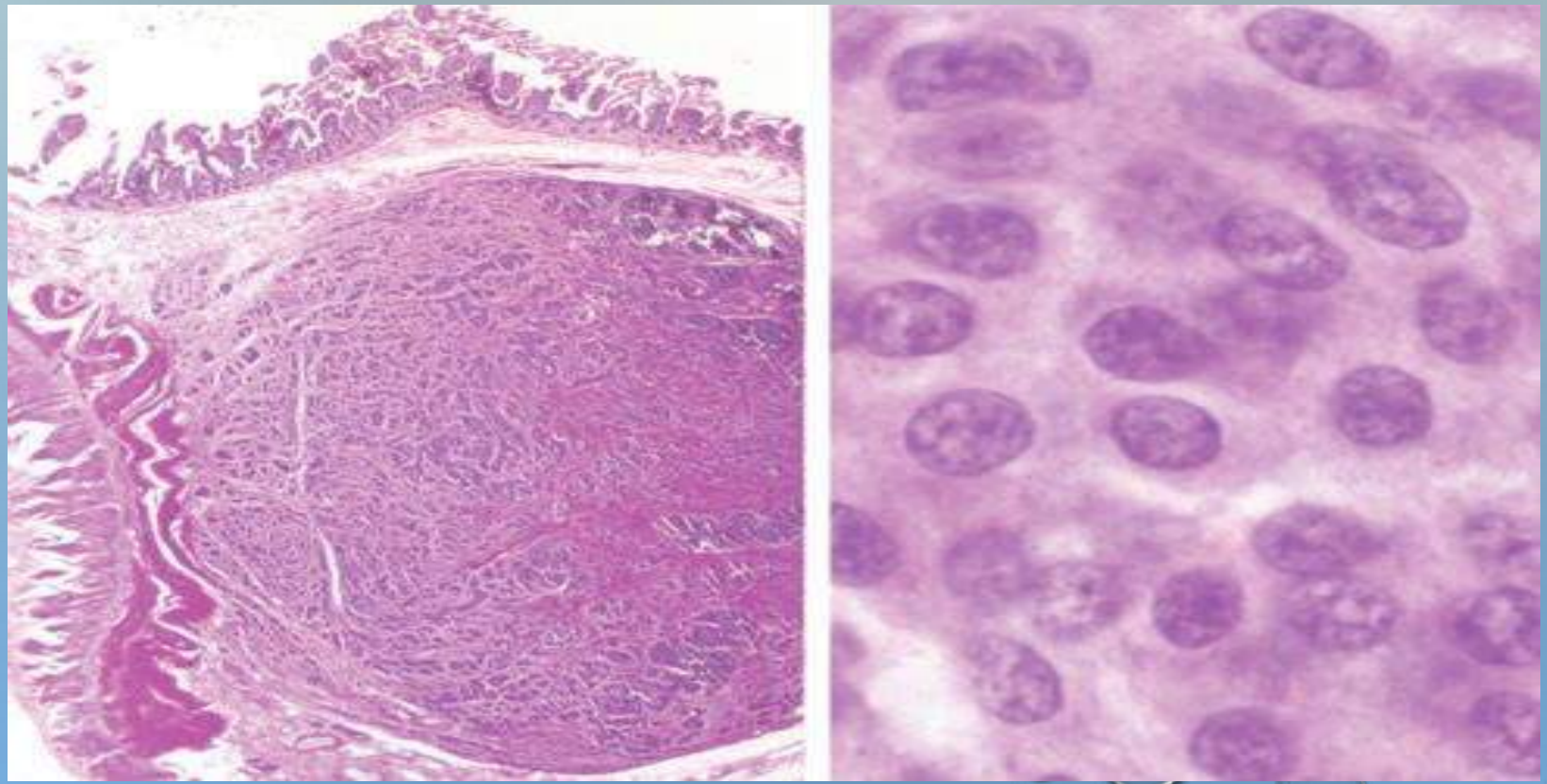
- large mucin vacuoles that expand the cytoplasm and push the nucleus to the periphery,



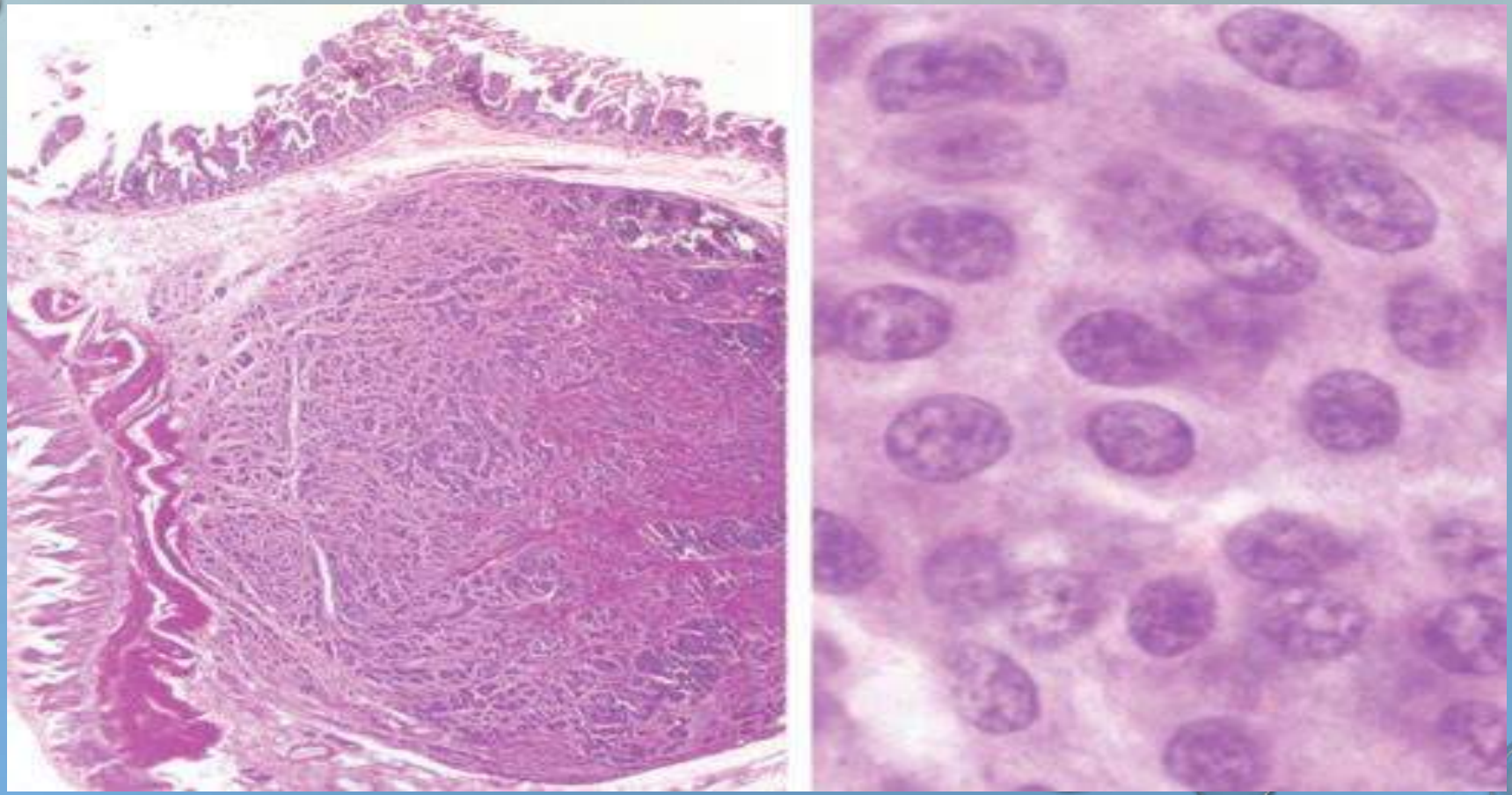
DIFFUSE TYPE, SIGNET RING CELLS



INTRAMURAL OR SUBMUCOSAL MASSES (SMALL POLYPOID LESIONS)



Islands, trabeculae, strands, glands, or sheets of uniform cells with scant, pink granular cytoplasm and salt and pepper chromatin.



Small and Large Intestinal pathology, part 1

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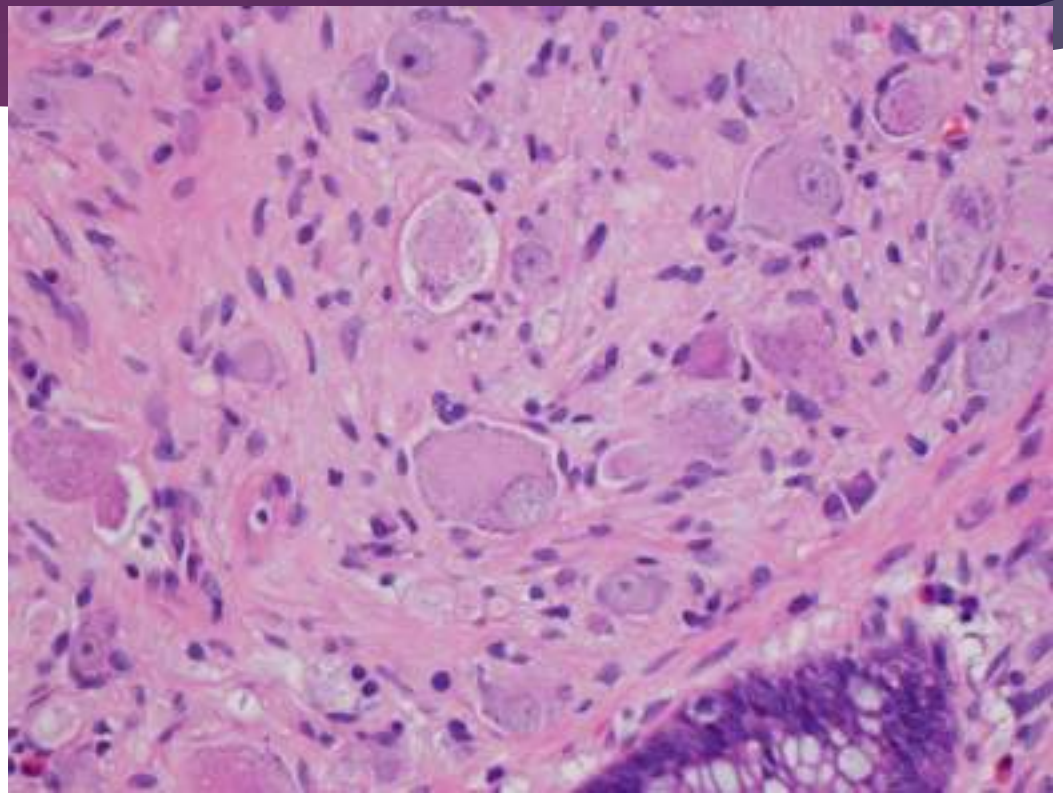
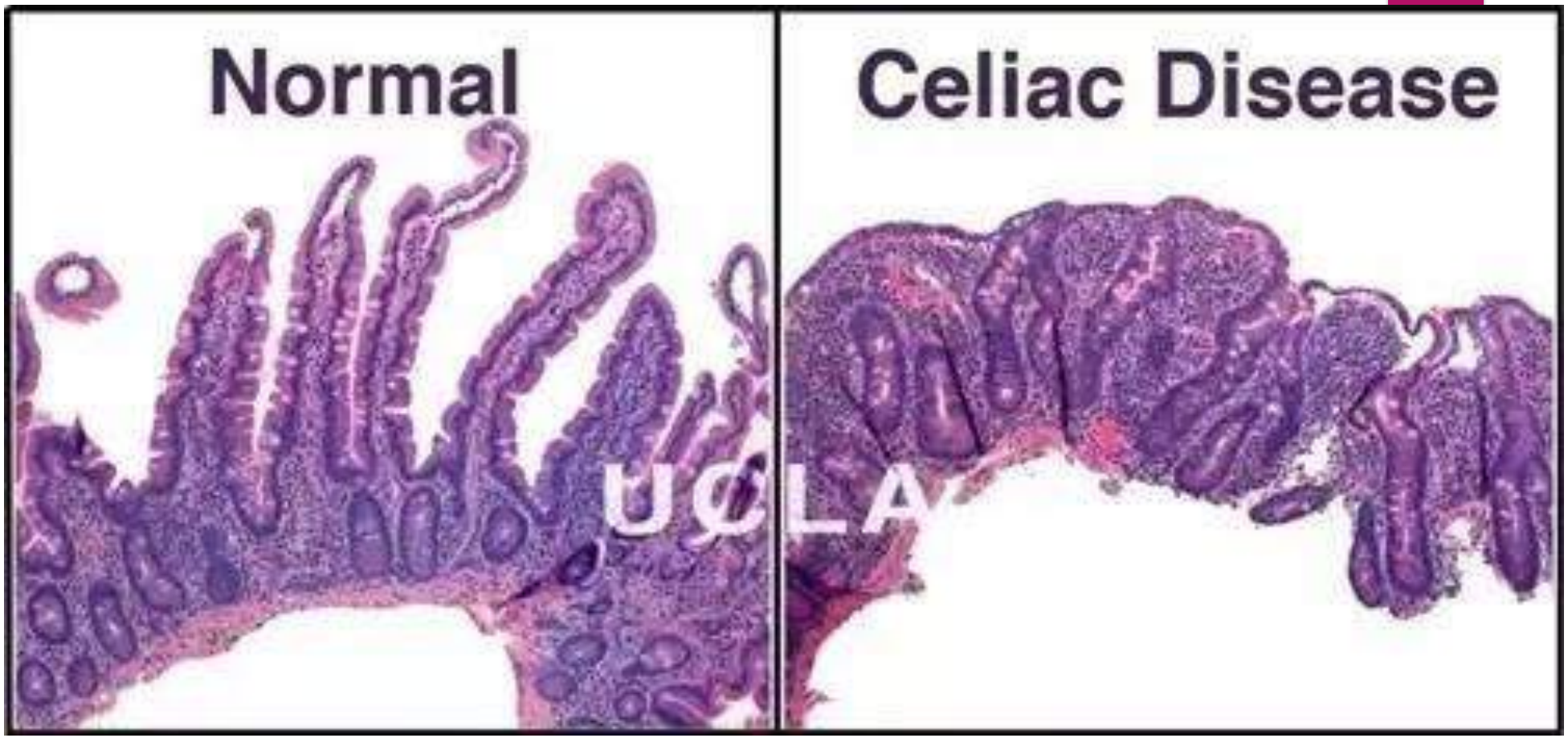
Robbins Basic Pathology 10th edition

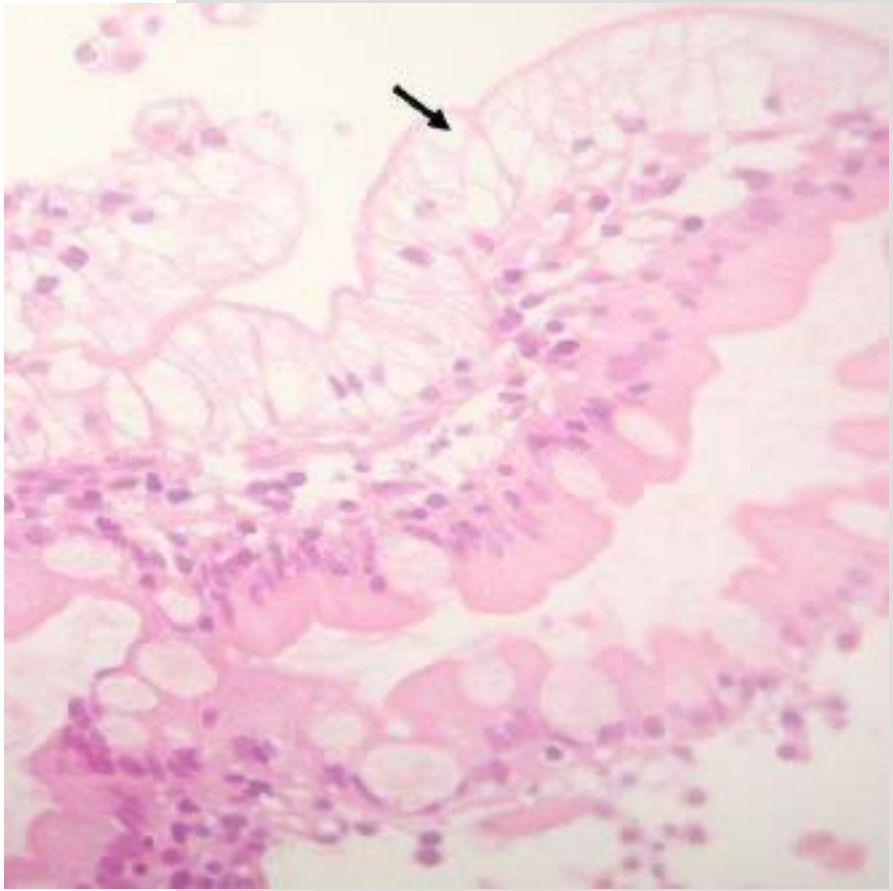
ganglion cells



http://www.pathologyoutlines.com


Normal
Celiac Disease
UCLA

Dermatitis herpetiformis.



Micrograph showing enterocytes with a clear cytoplasm (due to lipid accumulation) characteristic of abetalipoproteinemia.

Small and Large Intestinal pathology, part 2

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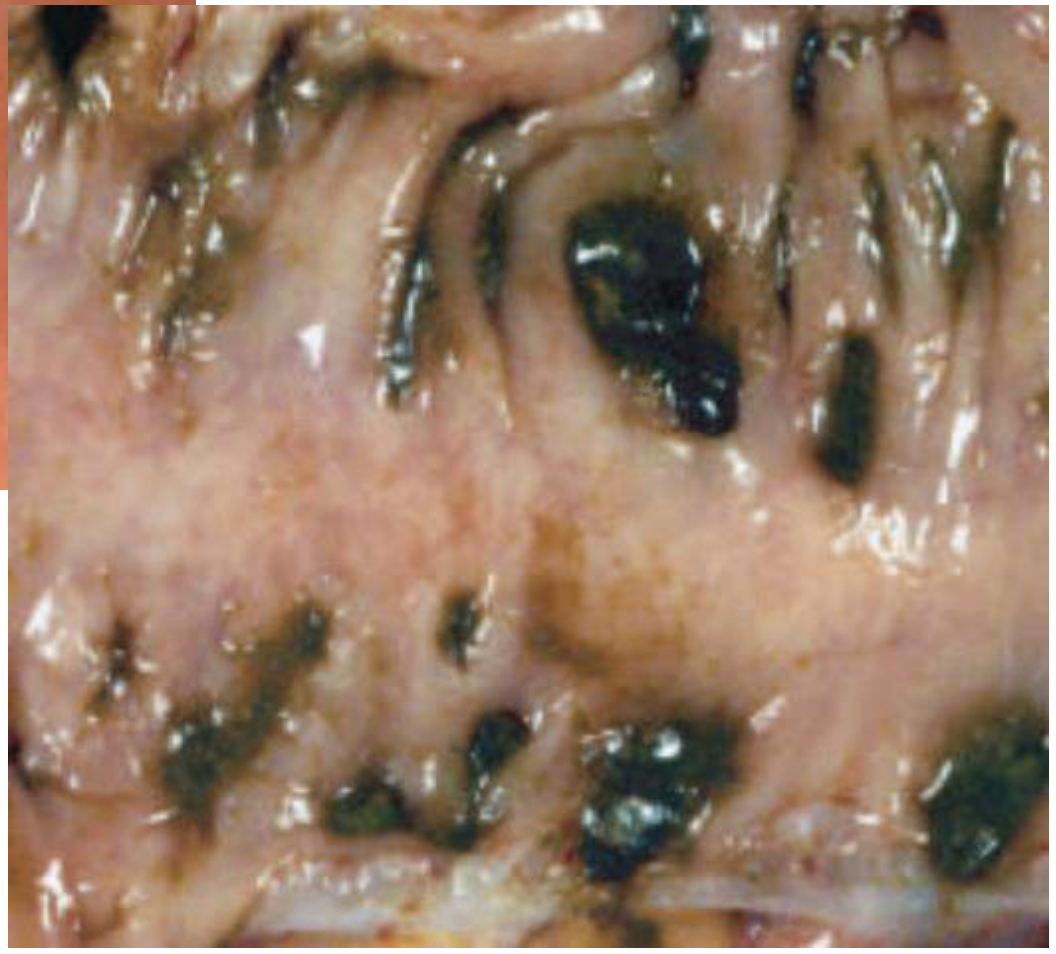
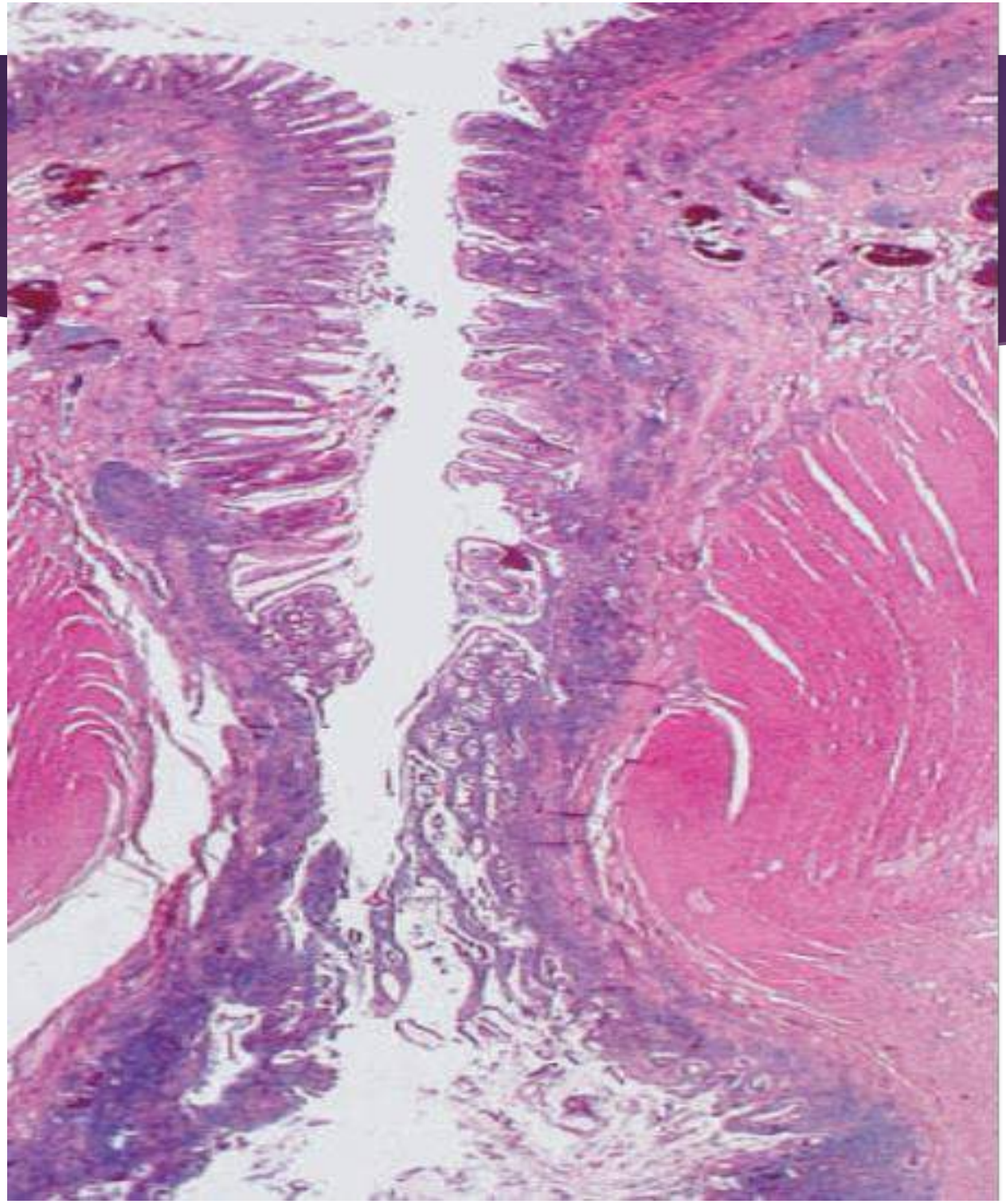
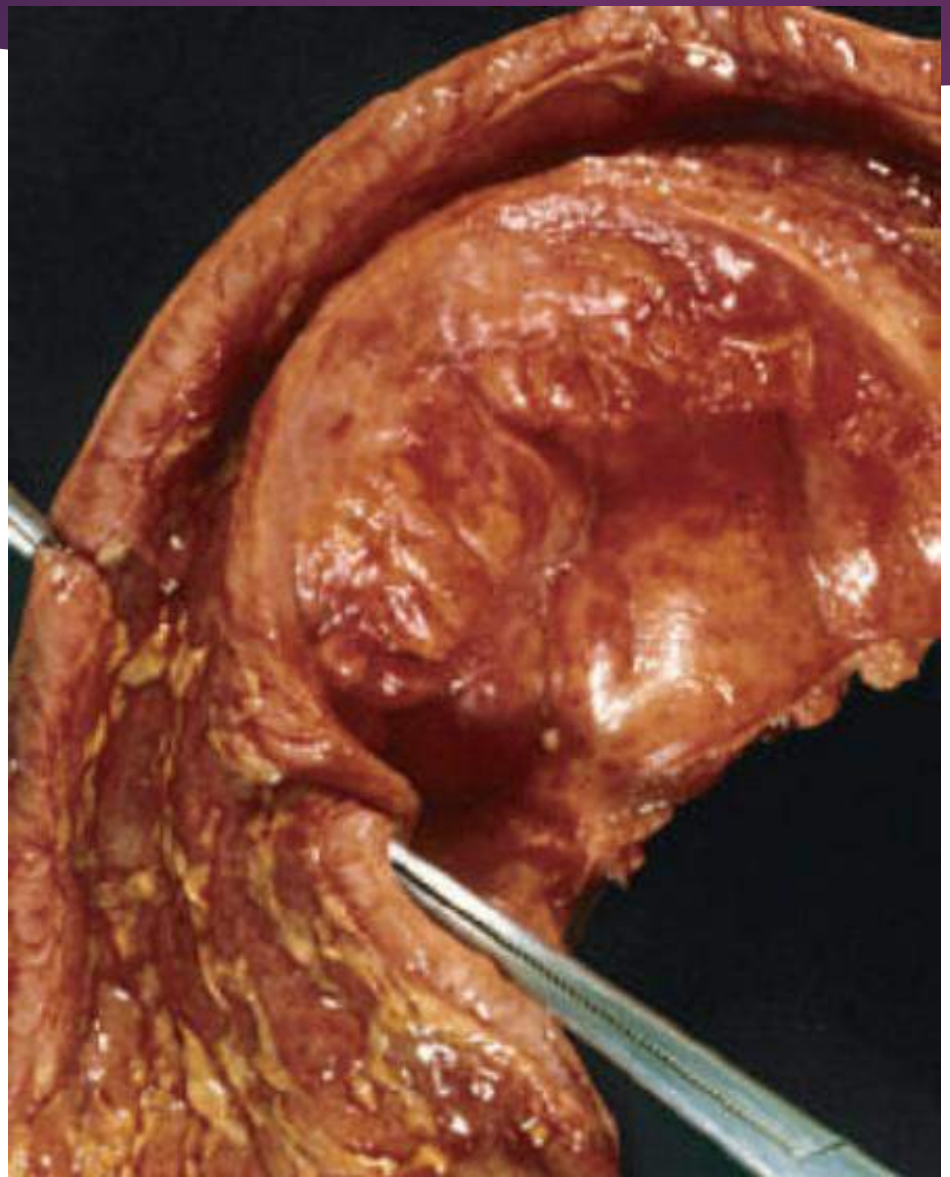
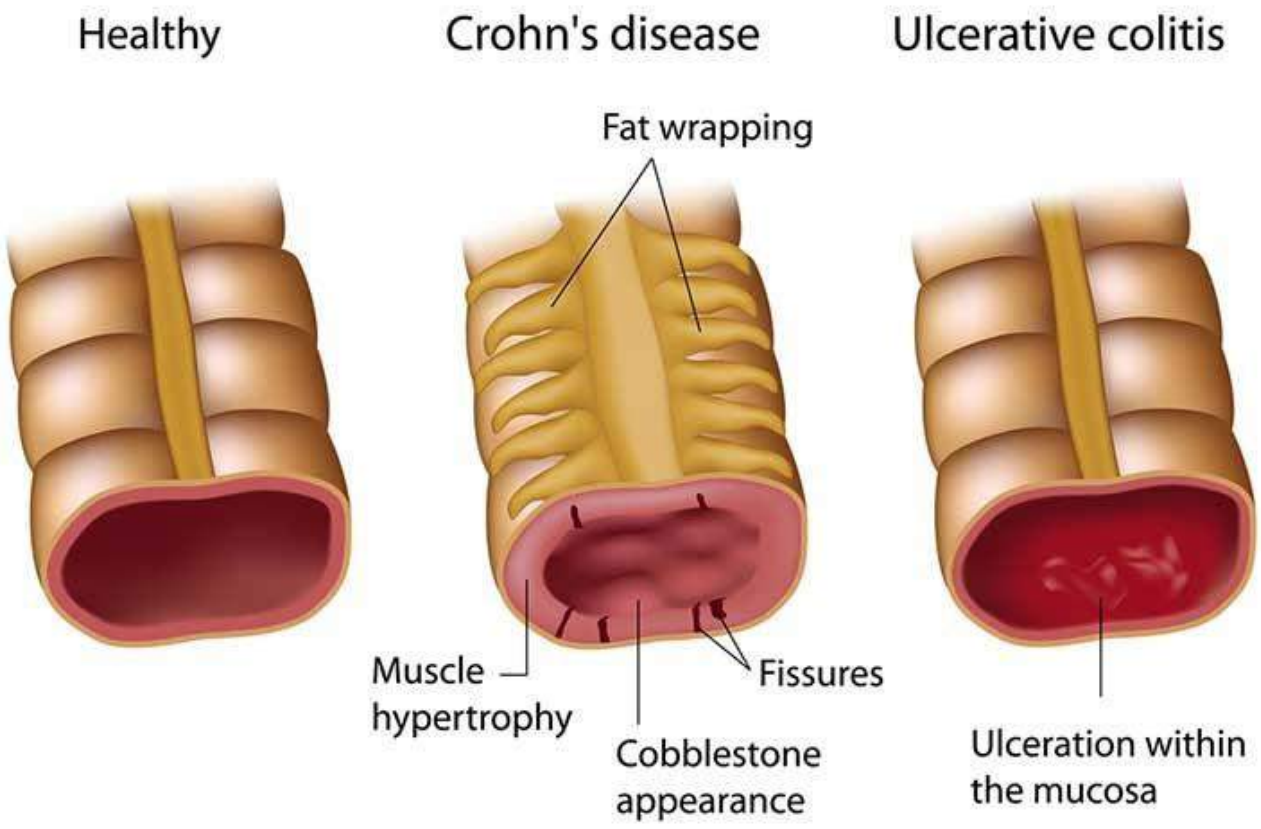
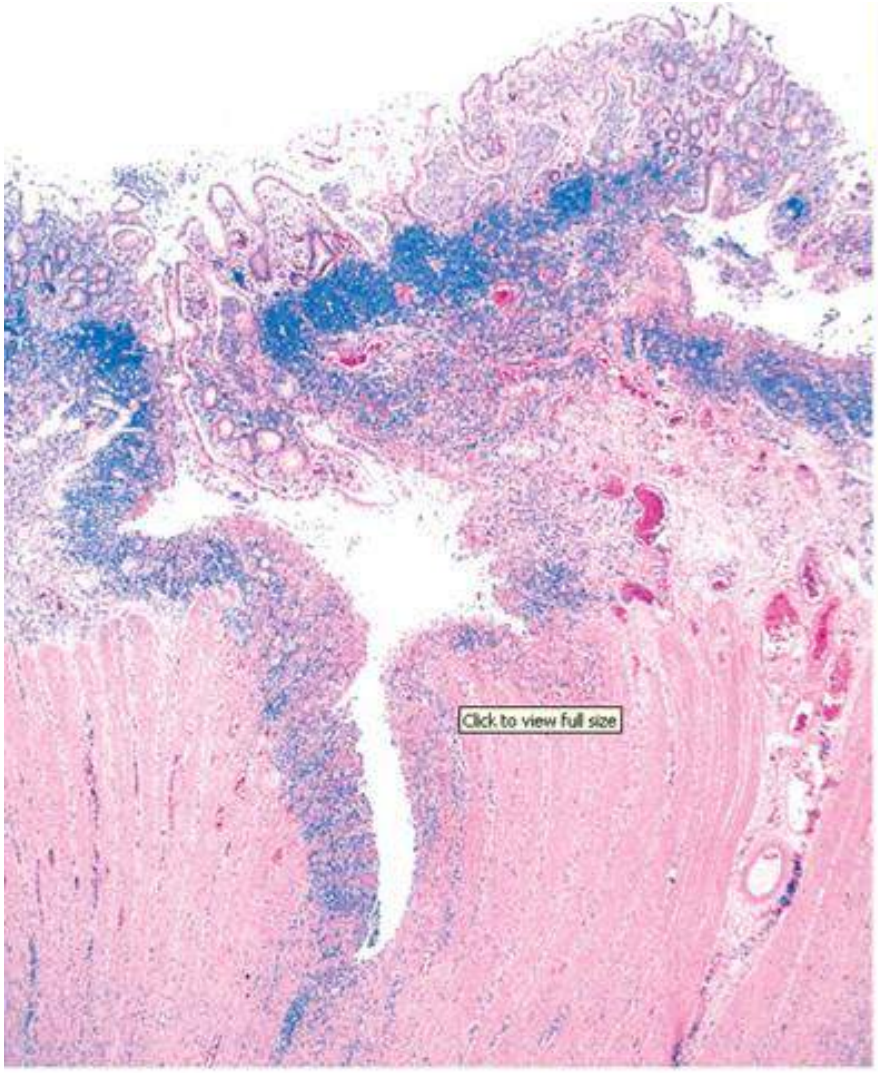




Small bowel stricture.

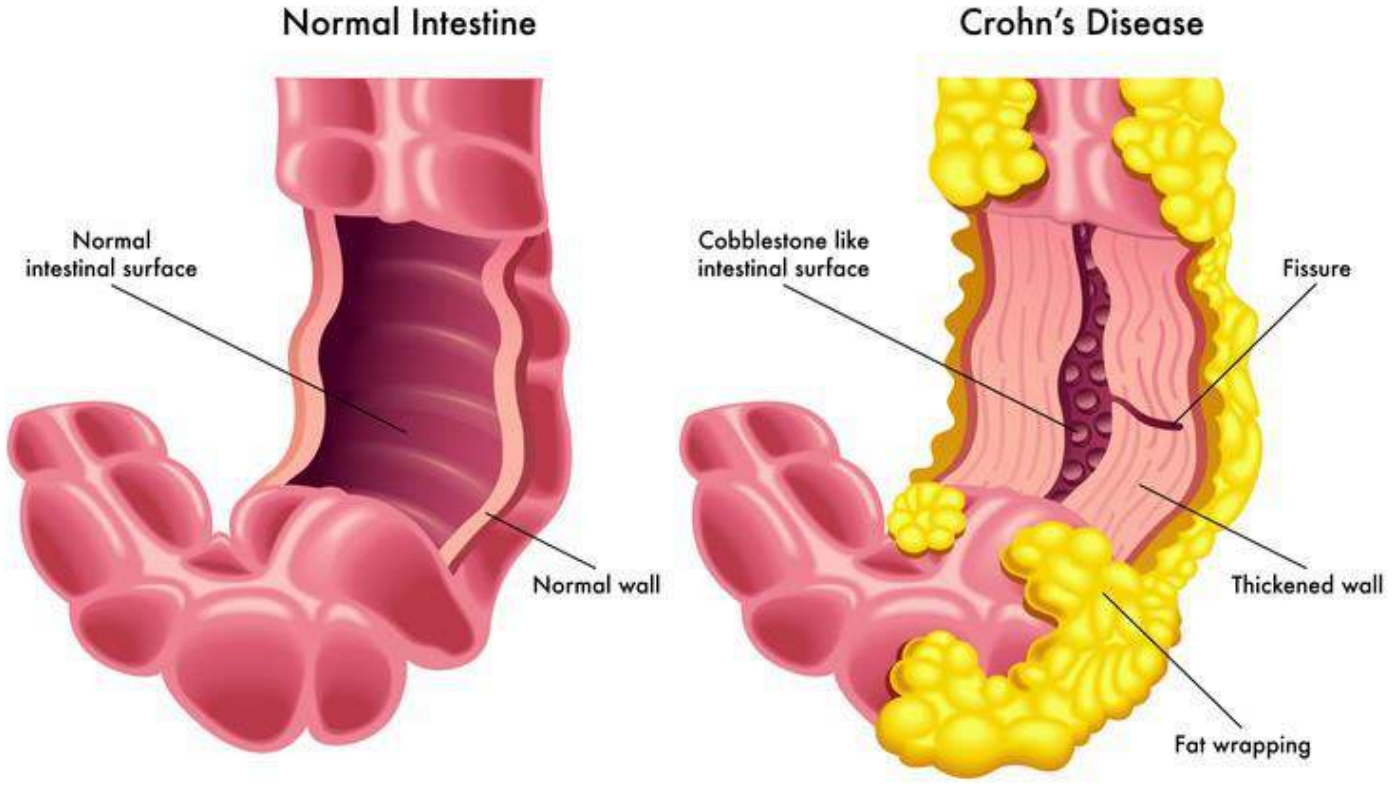



Healthy
Crohn's disease
Ulcerative colitis
Fat wrapping
Muscle hypertrophy
Cobblestone appearance
Fissures
Ulceration within the mucosa



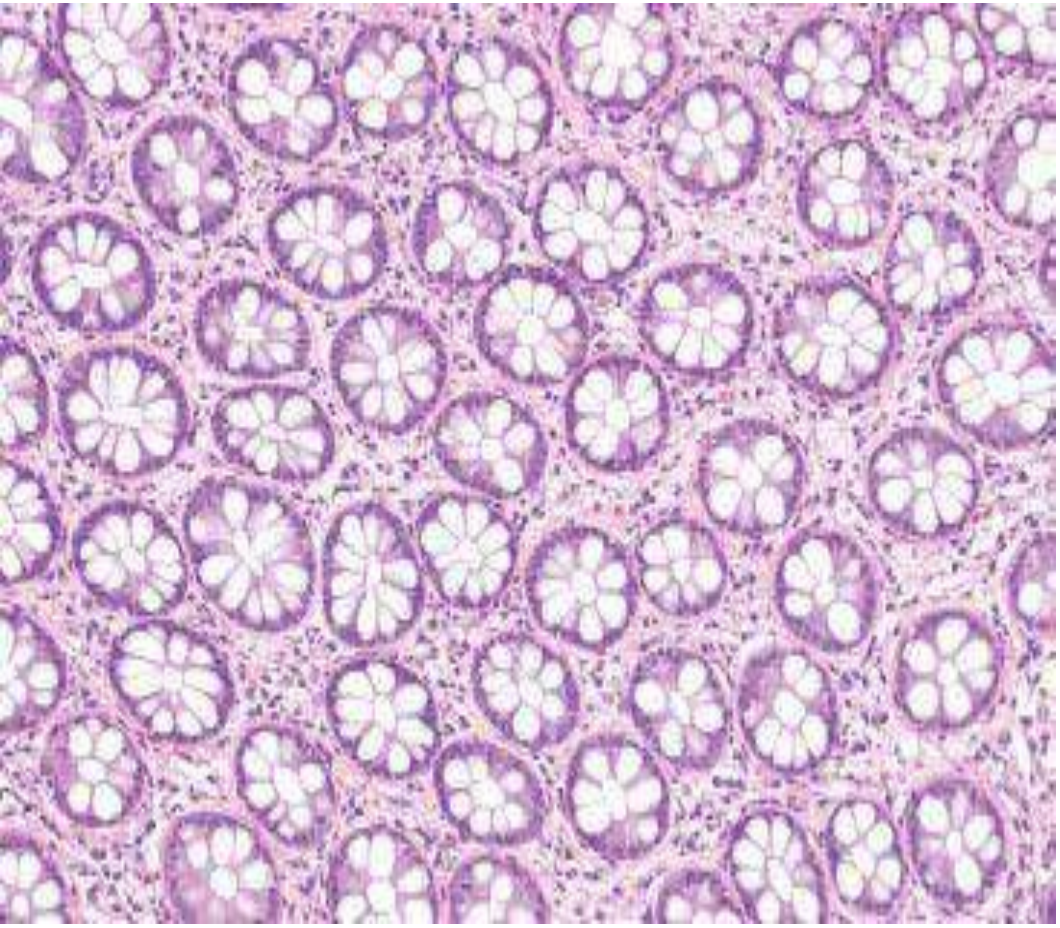
Crohn disease of the colon showing a deep fissure extending into the muscle wall, a second, shallow ulcer (upper right), and relative preservation of the intervening mucosa. Abundant lymphocyte aggregates are present, evident as dense blue patches of cells at the interface between mucosa and submucosa

Creeping fat



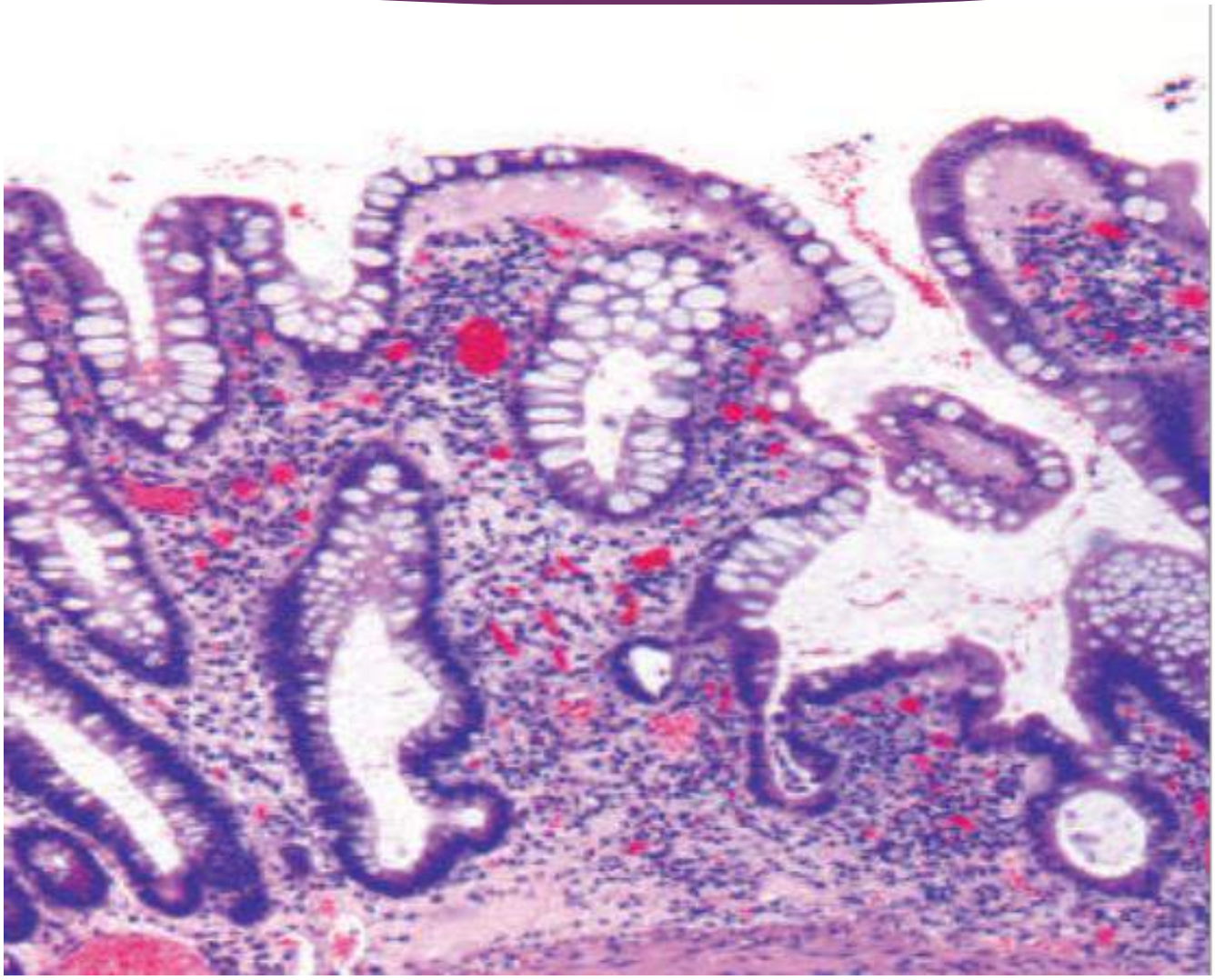
Cobblestone appearance

ResearchGate

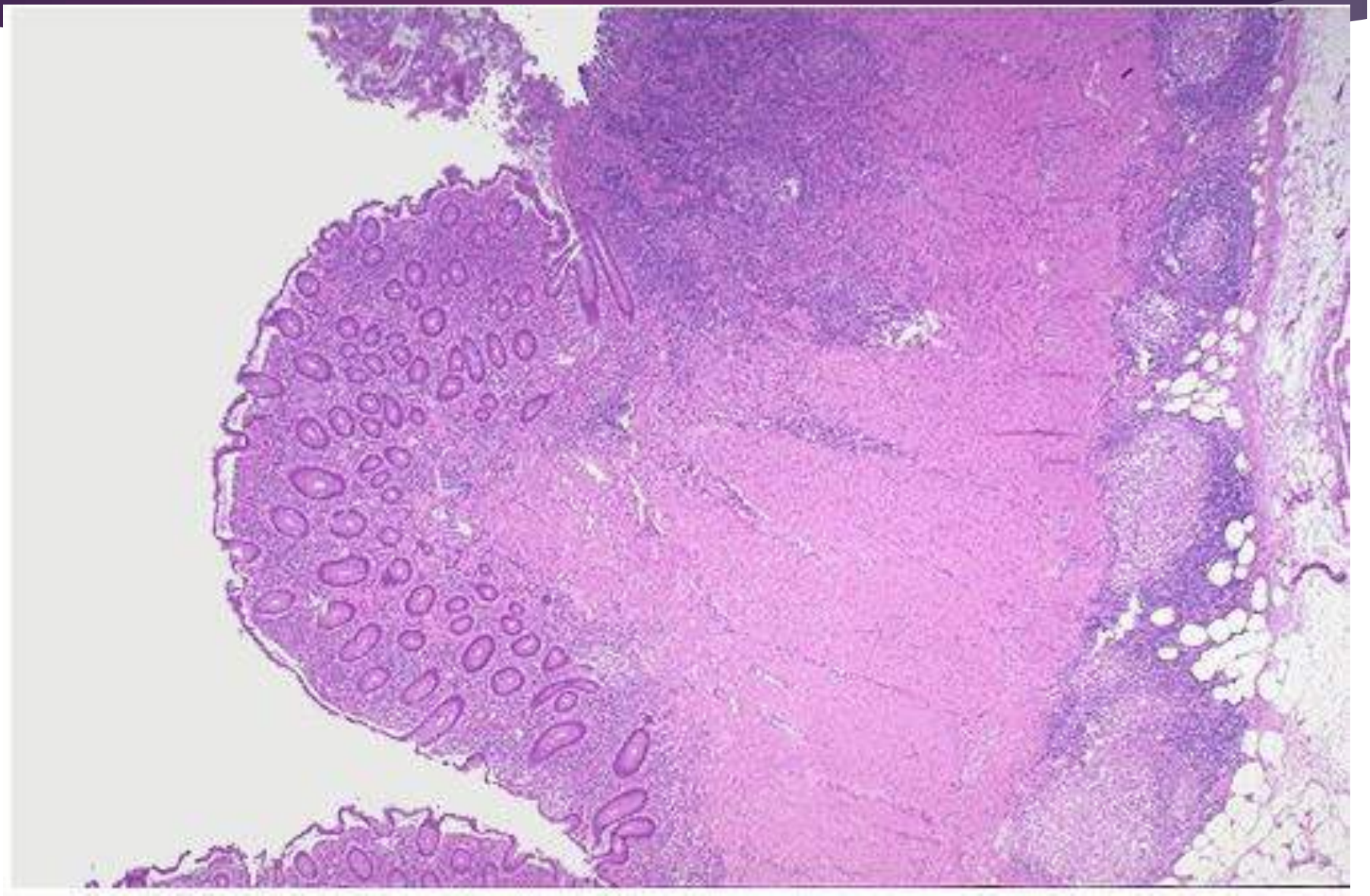



DIBOVPCR ISMJ www.medicalimages.com
06 Apr 2021

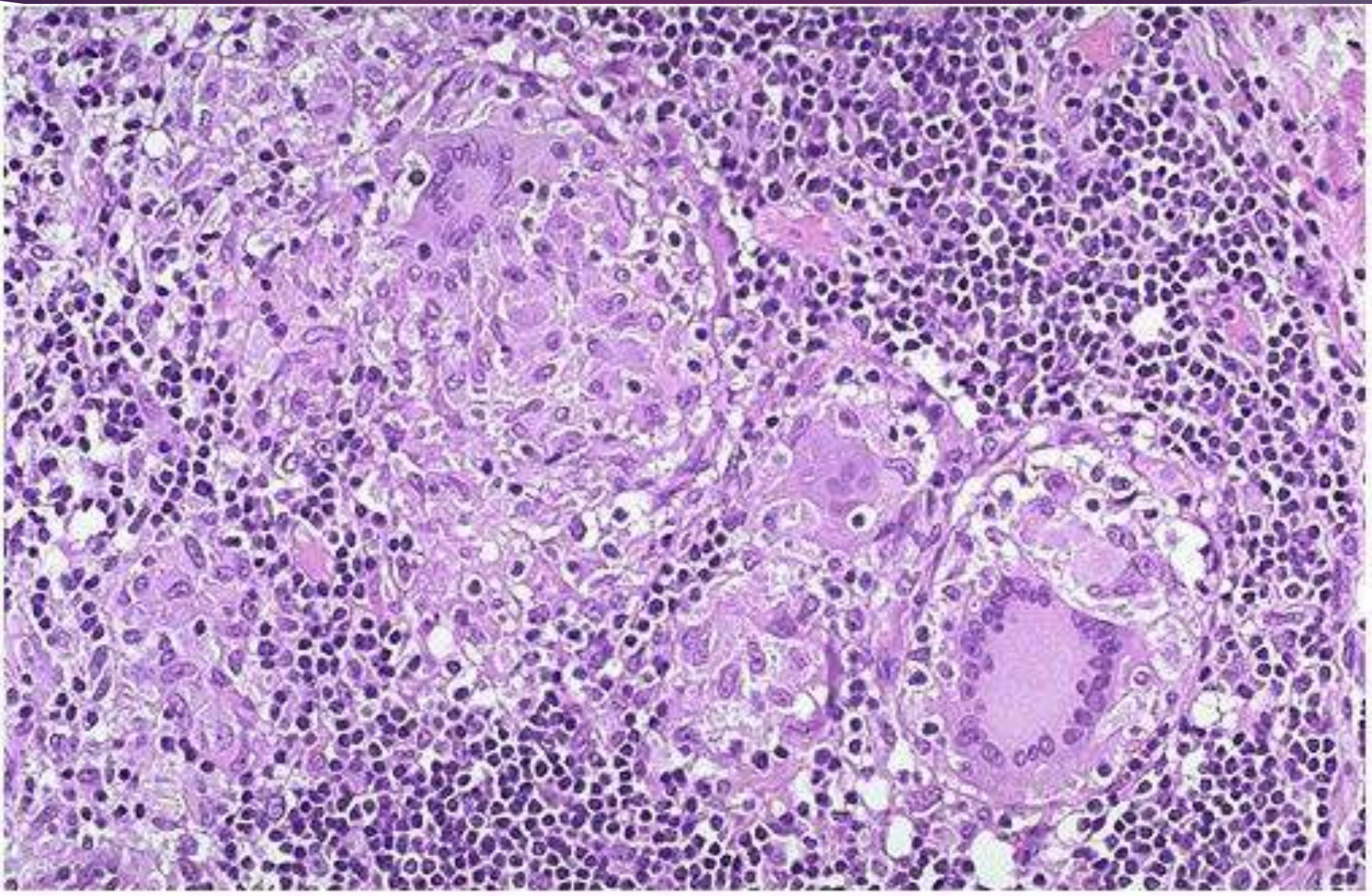
Haphazardly arranged crypts



Transmural inflammation.



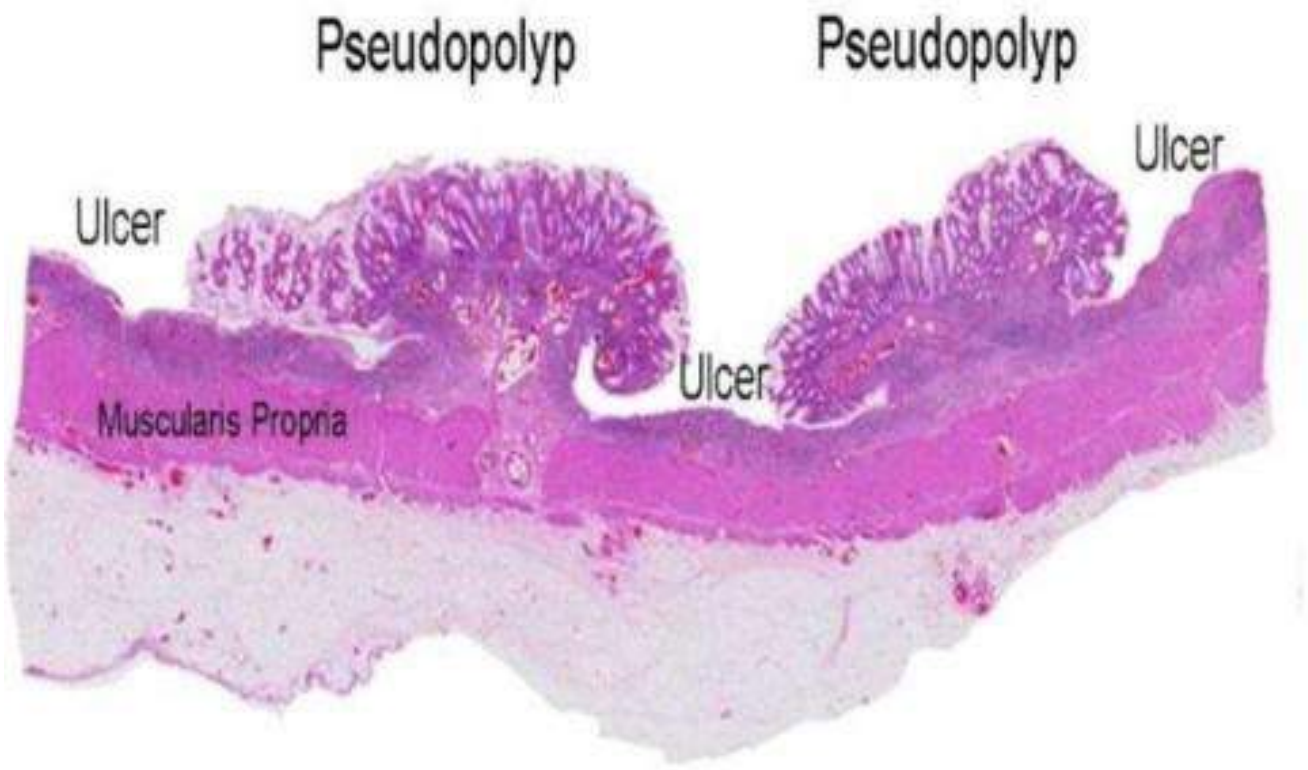
Non-caseating granuloma.



Erythema nodosum

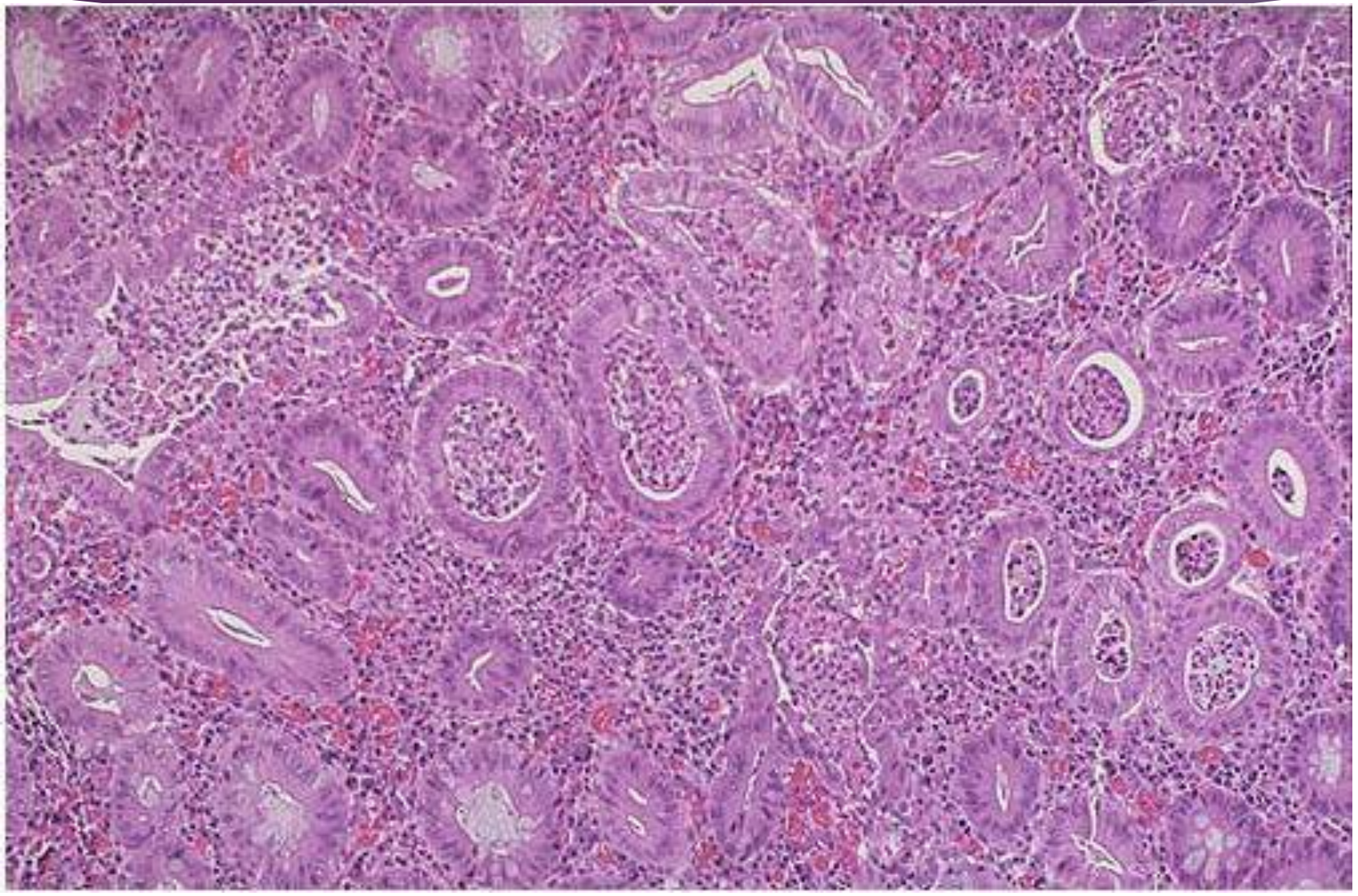
Clubbing

Wikipedia


Pseudopolyp
Pseudopolyp
Ulcer
Ulcer
Ulcer
Muscularis Propria

Toxic megacolon

Crypt abcesses.

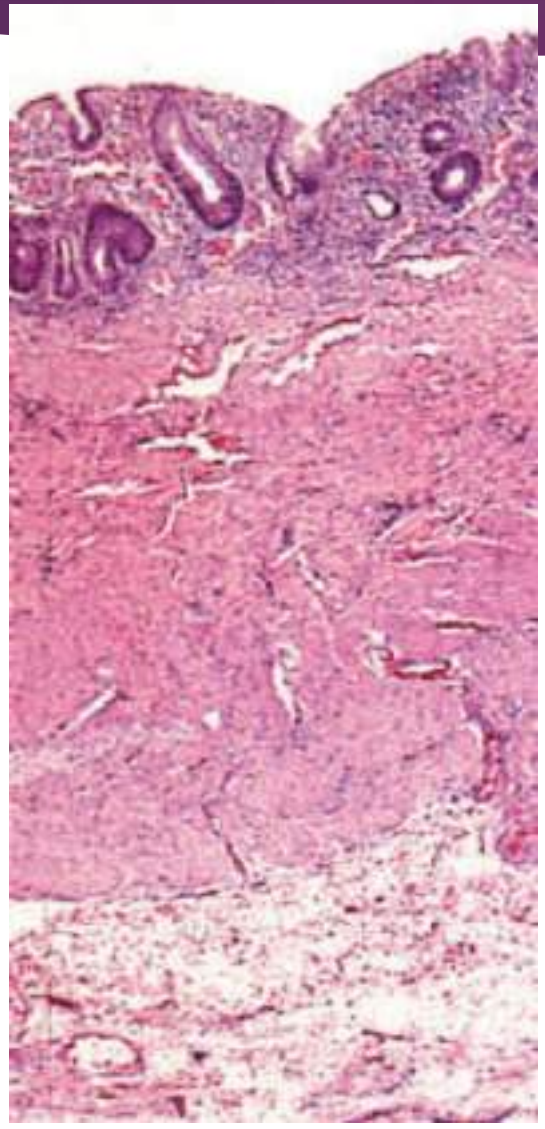


Mucopurulent material and ulcers.

Pancolitis.

Abrupt transition b/w normal and disease segment.

Limited to mucosa



Small and Large Intestinal pathology, part 3

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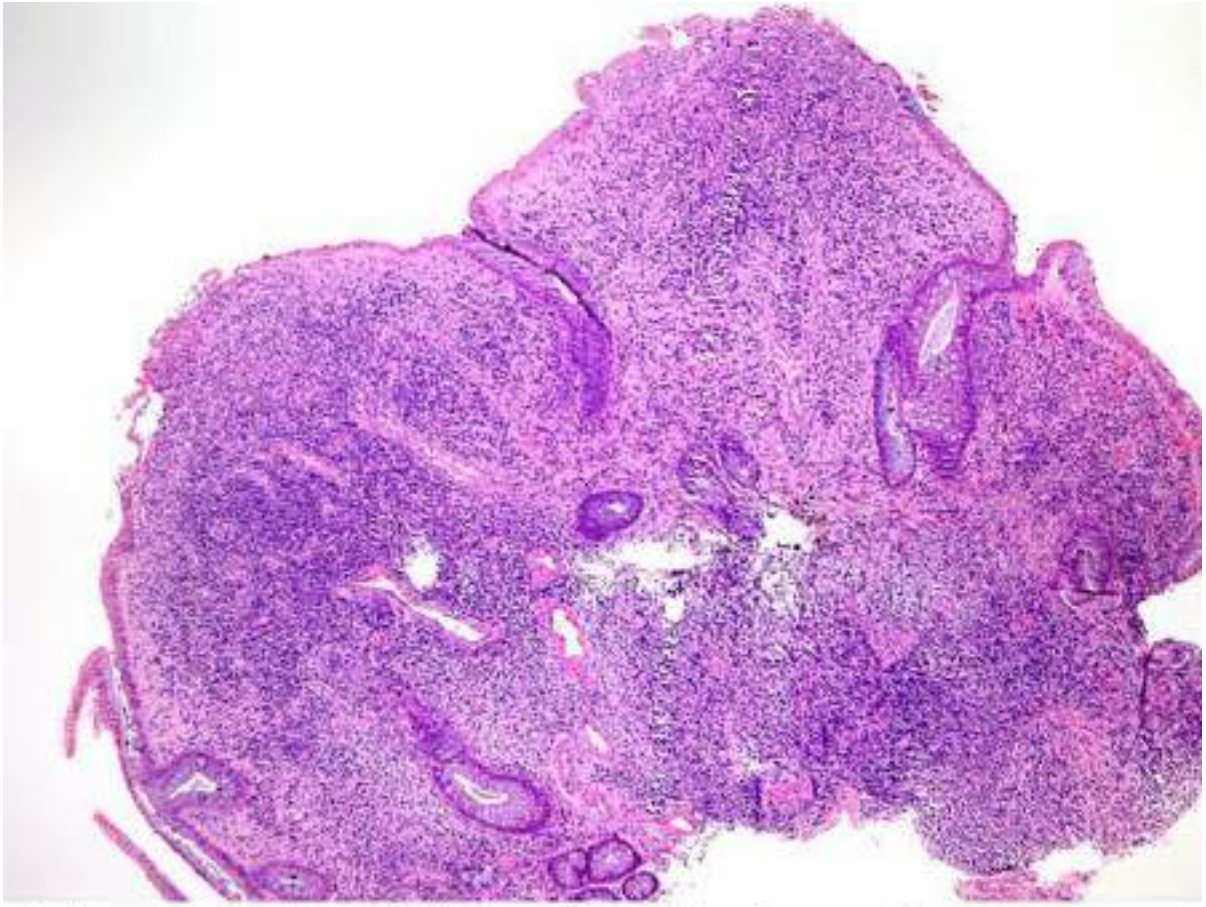
Undergraduate Lectures 2025



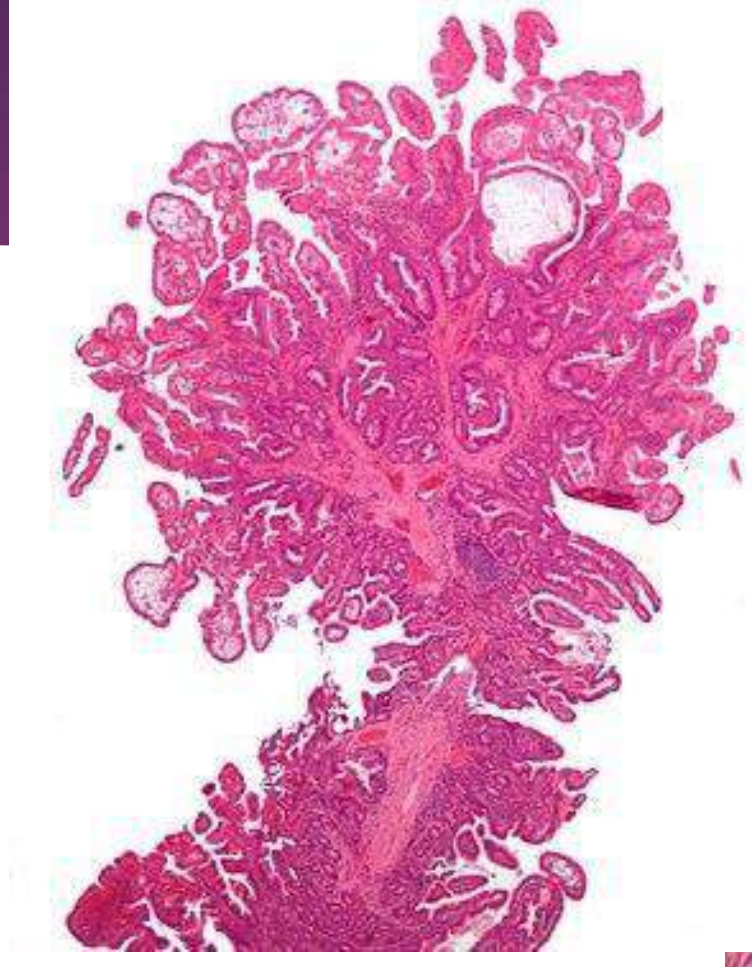
4x: low power, dense inflammation in lamina propria

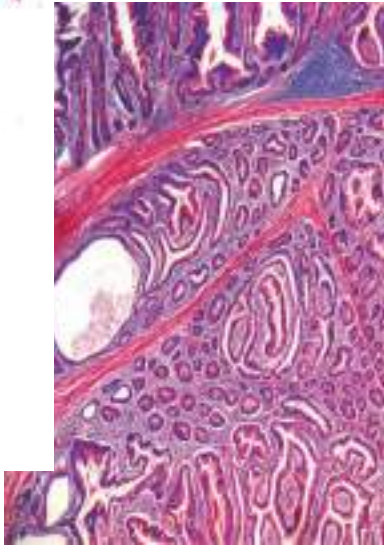
Juvenile Polyps

- Pedunculated
- Reddish lesions
- Cystic spaces on cut sections
- Dilated glands filled with mucin and inflammatory debris.
- Granulation tissue on surface.

Peutz-Jeghers polyp

- Large.
- Arborizing network of connective tissue, smooth muscle, lamina propria
- Glands lined by normal-appearing intestinal epithelium
- Christmas tree pattern.



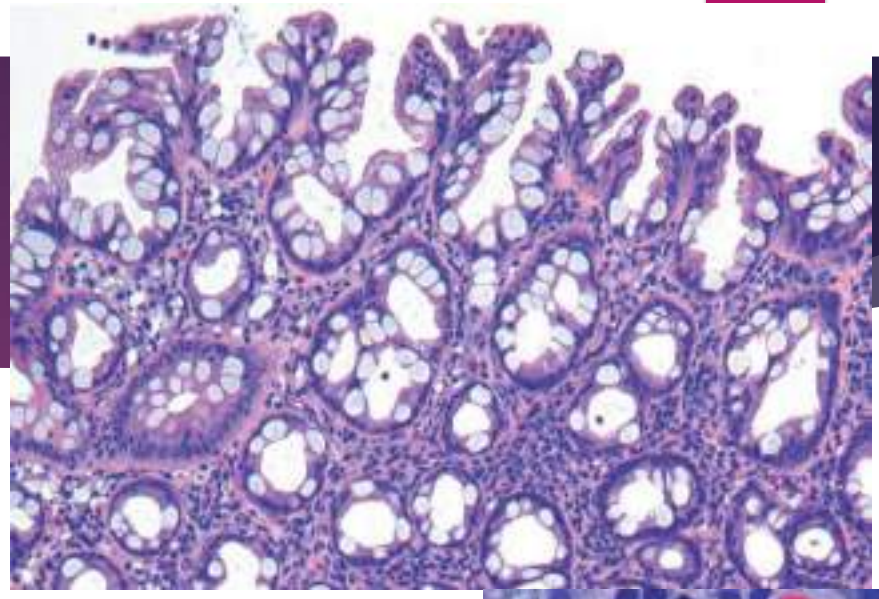


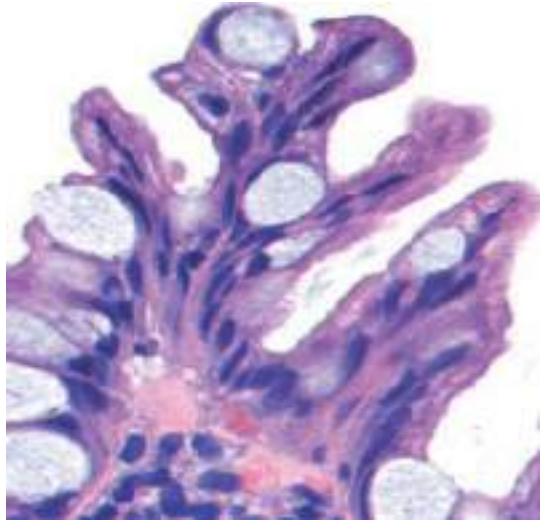
Mucocutaneous pigmentation

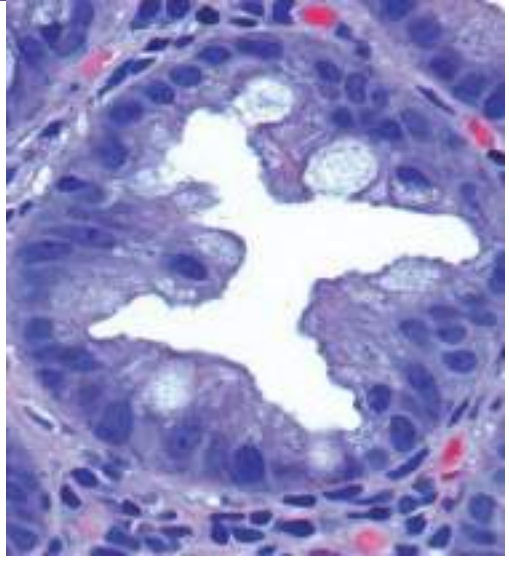
Hyperplastic polyp

- Left colon
- Rectosigmoid.
- Small < 5 mm
- Multiple

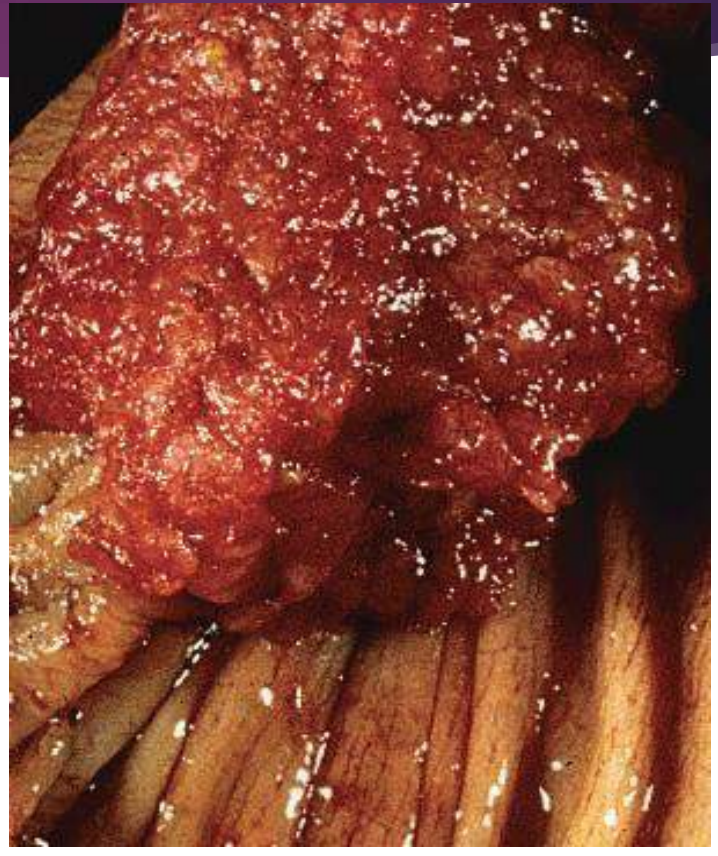
- Crowding of goblet & absorptive cells.





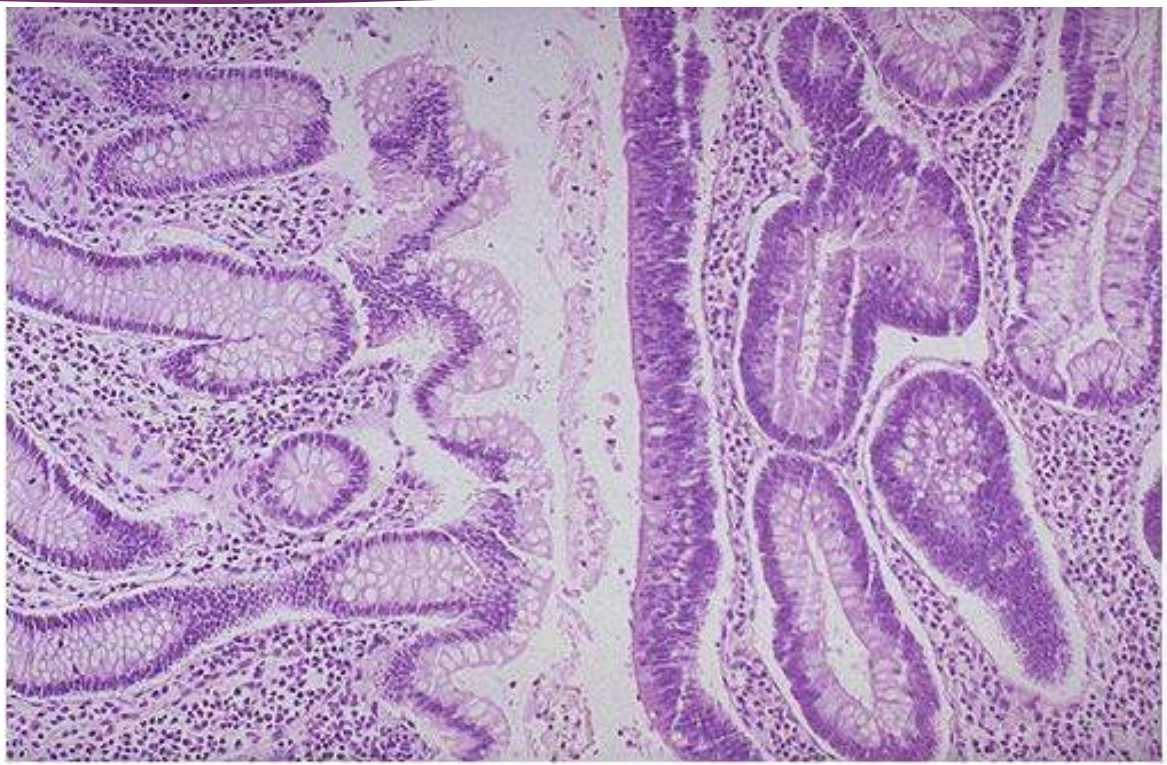


Pedunculated or sessile

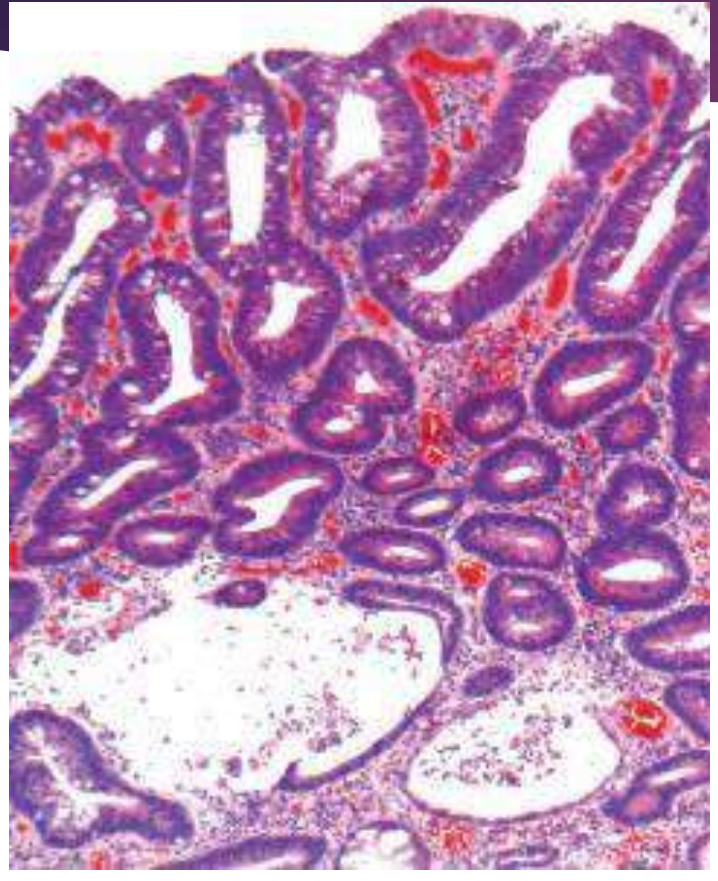


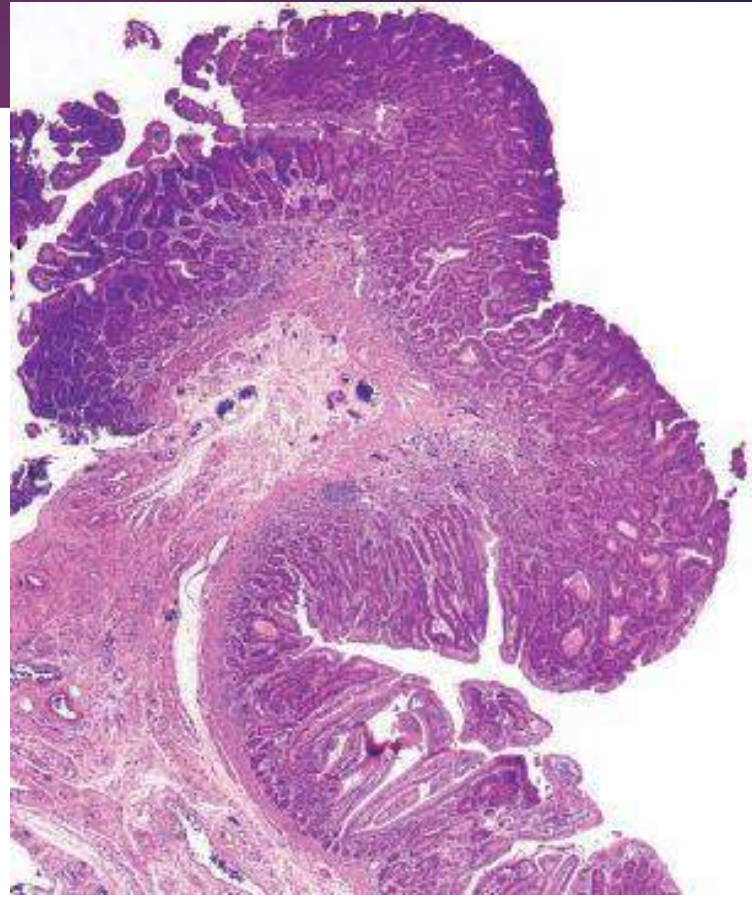
Colon adenoma

- **Hallmark: epithelial dysplasia**
- **Dysplasia**: nuclear hyperchromasia, elongation, stratification, high N/C ratio.
- **Size : most important correlate with risk for malignancy**
- **High-grade dysplasia is the second factor**

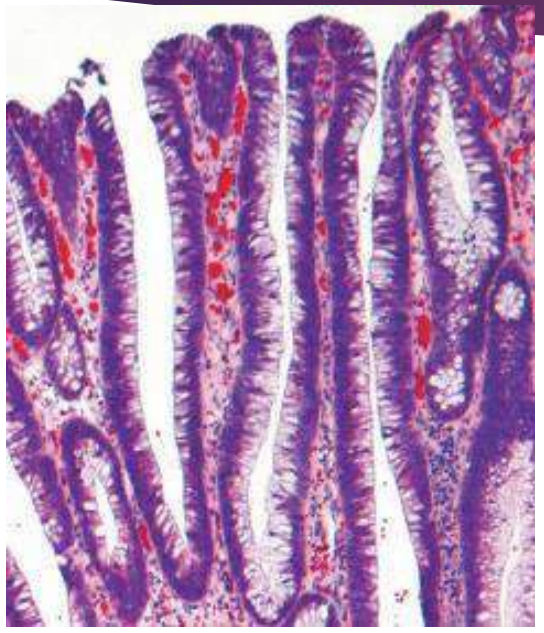


Tubular adenoma





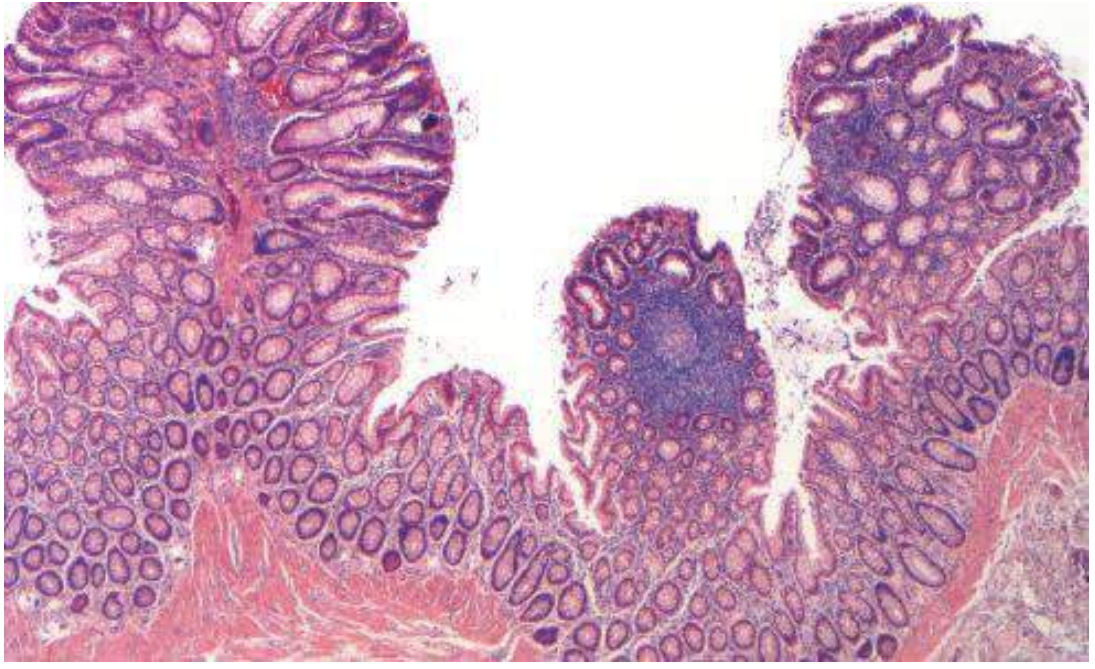
Villous adenoma.



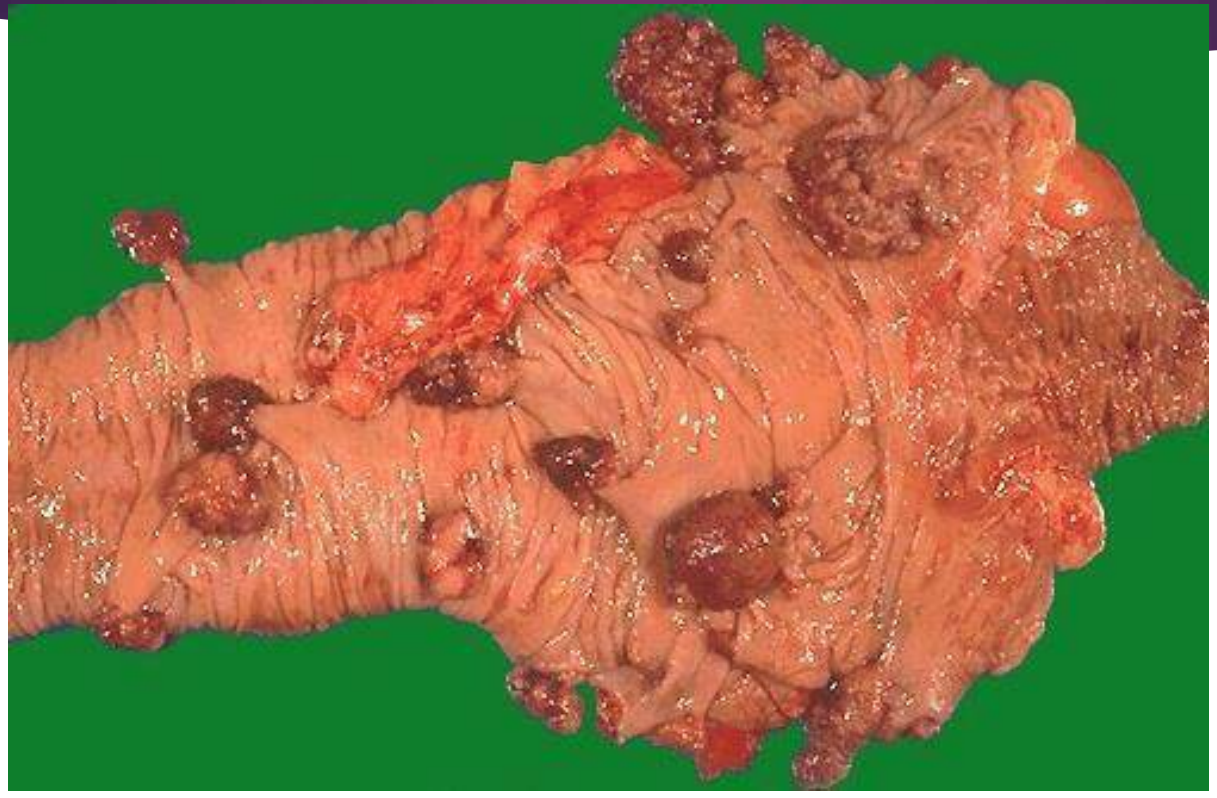
- Long slender villi.
- More frequent invasive foci

- **Architecture:**
- **Tubular.**
- **Tubulovillous.**
- **Villous.**

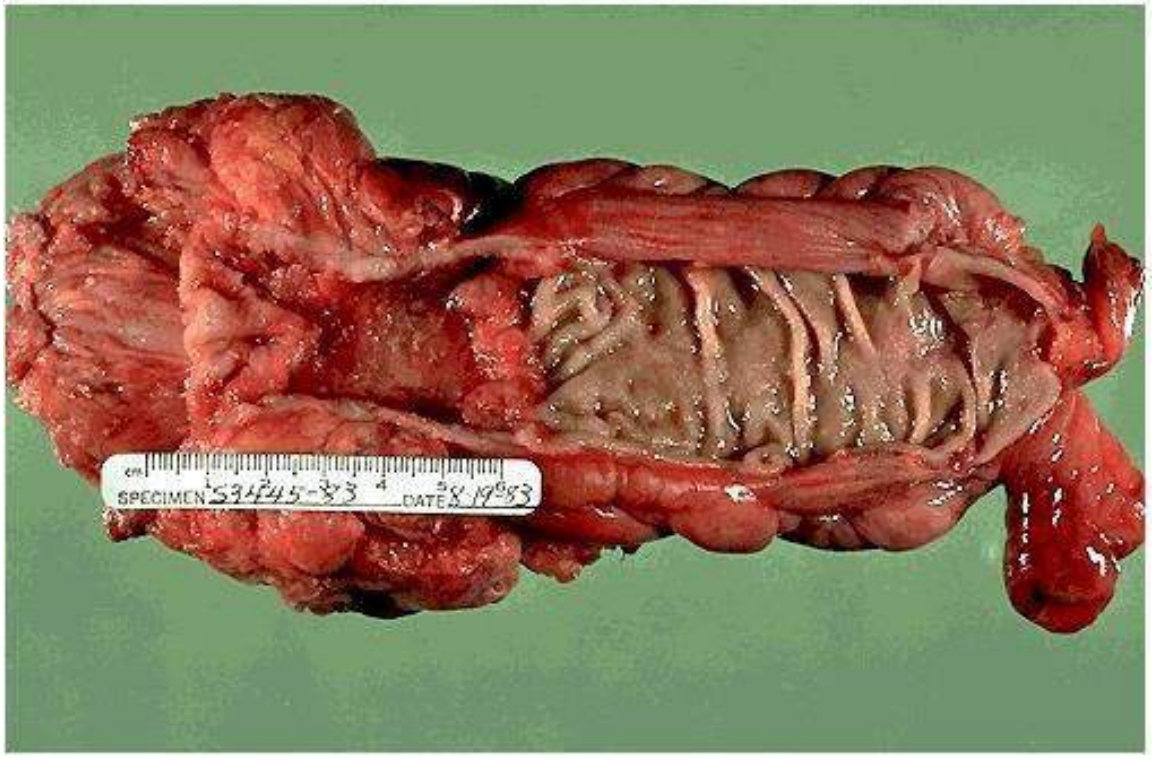
Villous adenoma



Cecal polyps in HNPCC.

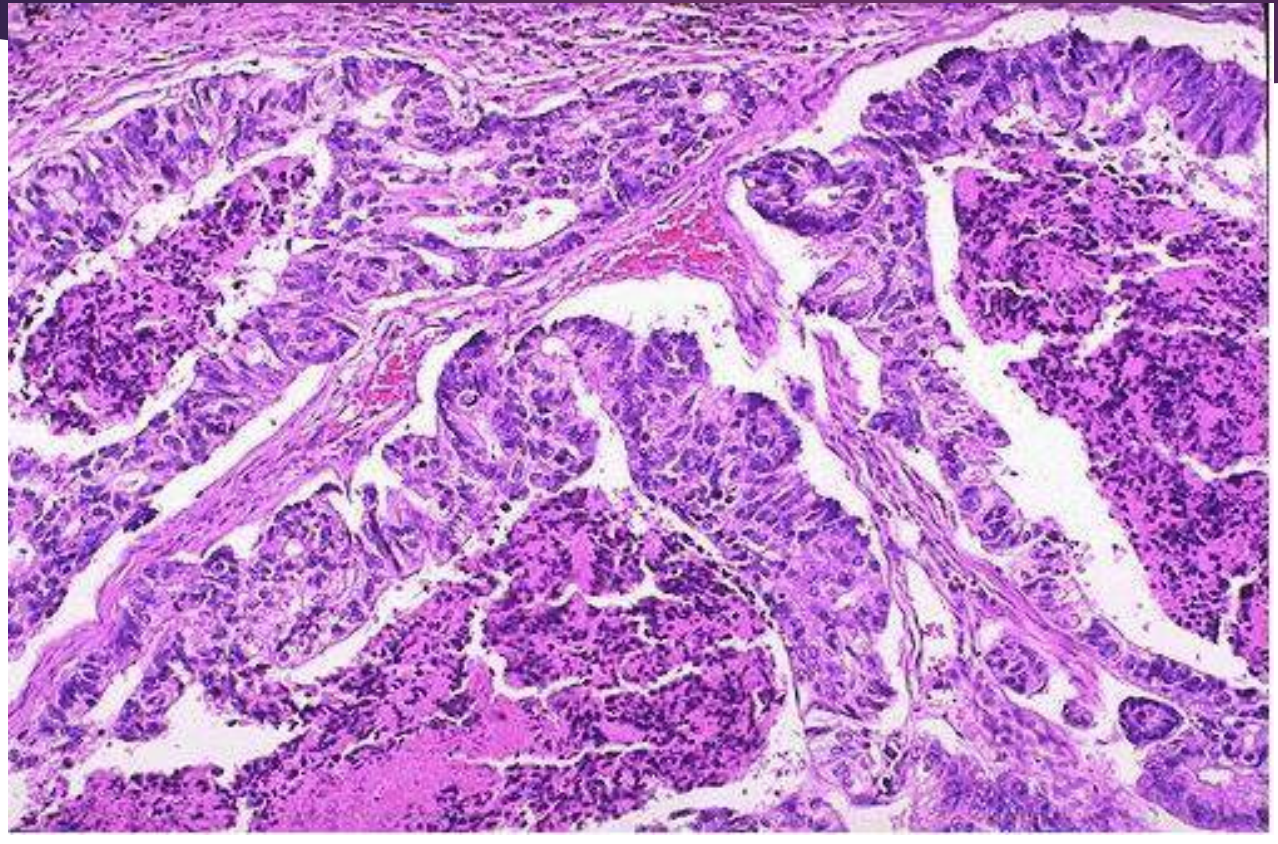


Rectosigmoid adenocarcinoma, napkin ring

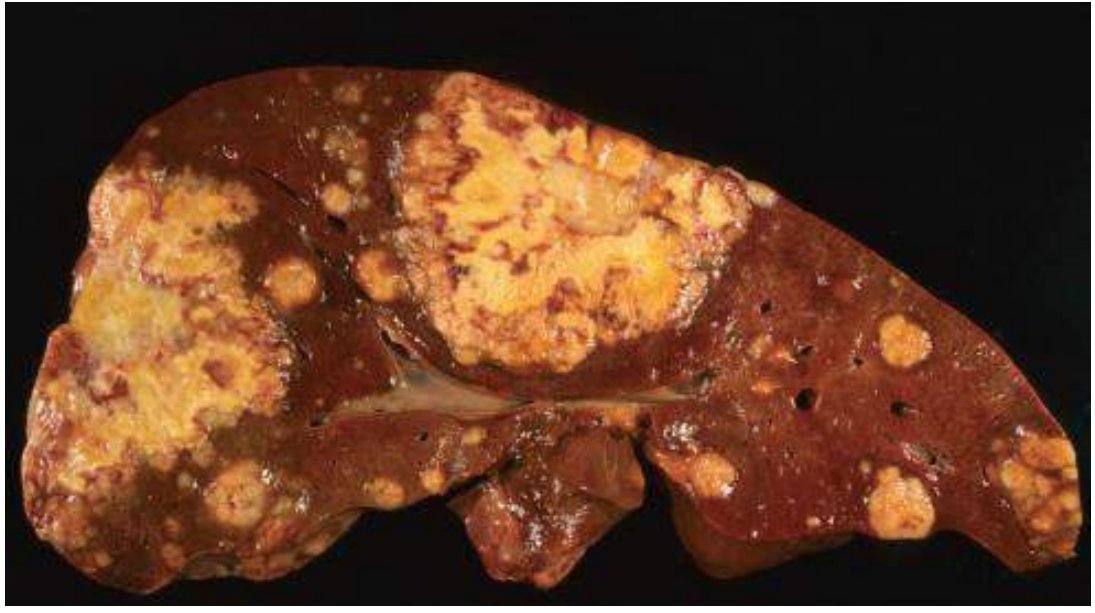


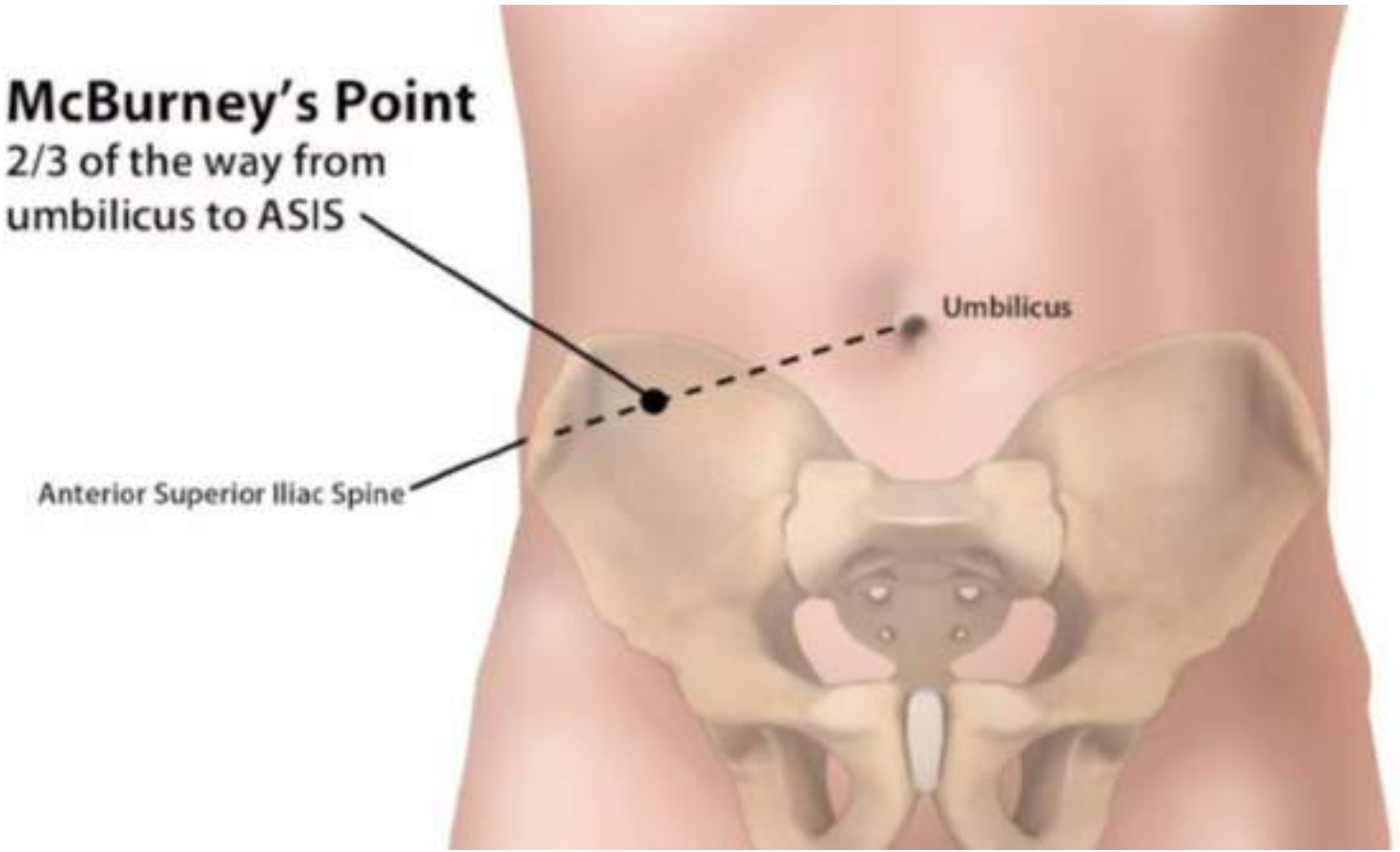
Exophytic adenocarcinoma

Adenocarcinoma with necrosis



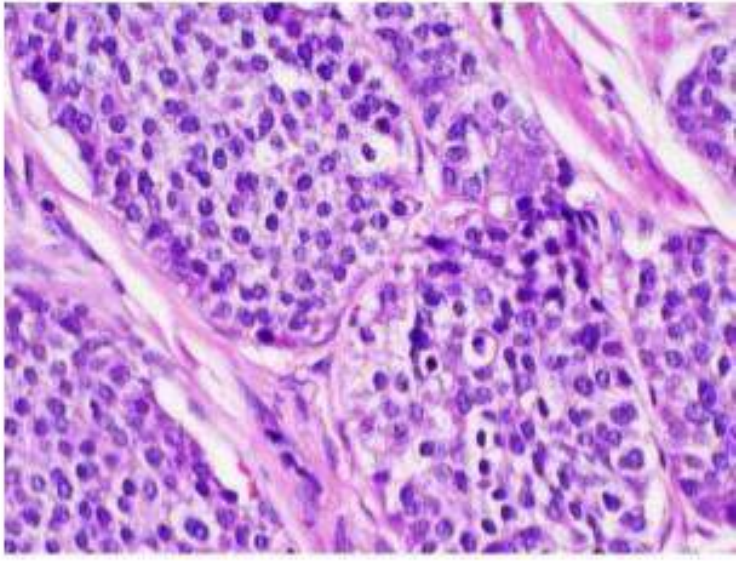
Liver metastasis.




McBurney's Point
2/3 of the way from
umbilicus to ASIS
Umbilicus
Anterior Superior Iliac Spine

Carcinoid tumor

Gross



Microscopic

PATHOLOGY OF LIVER I

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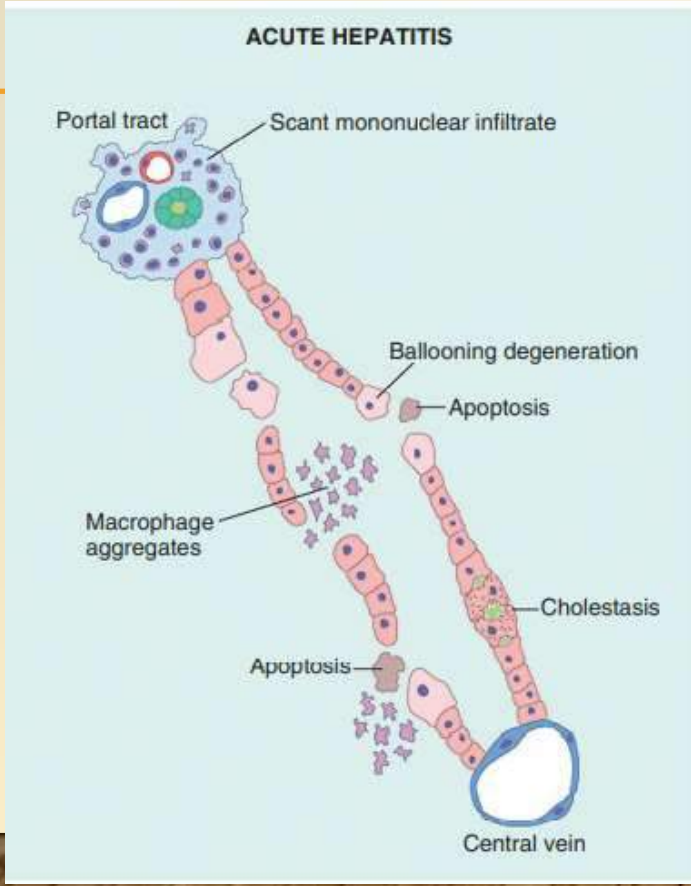
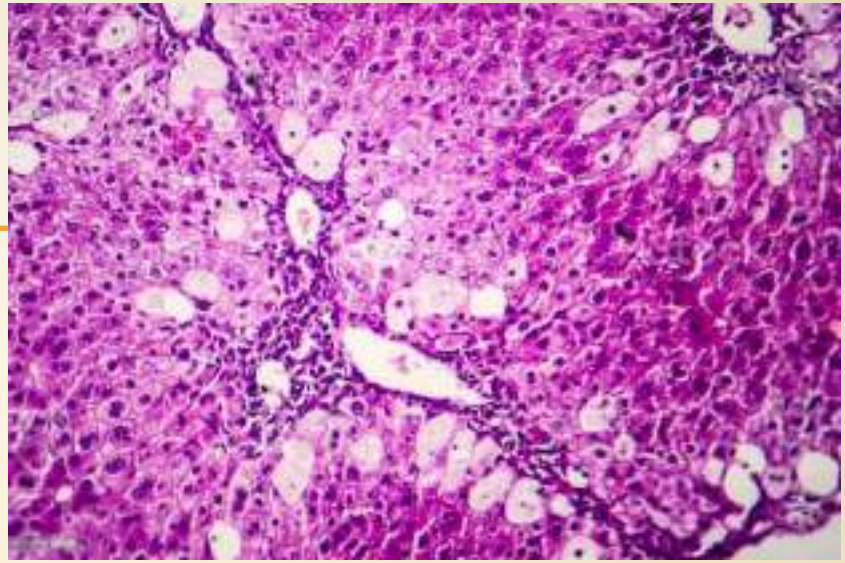
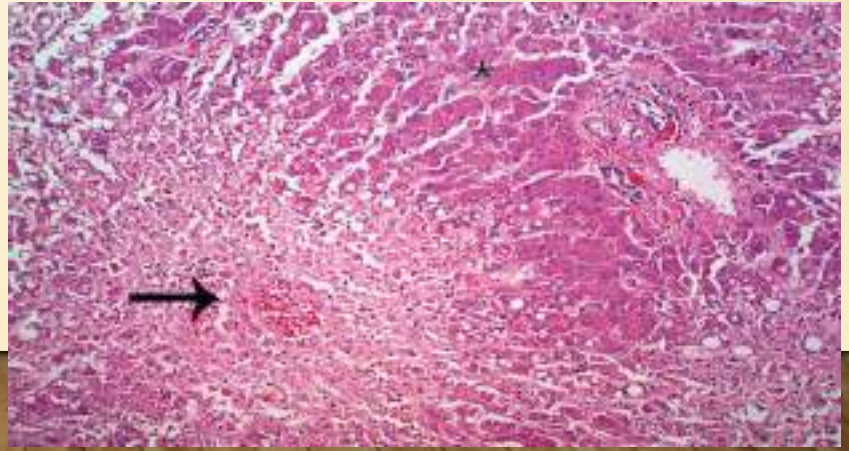
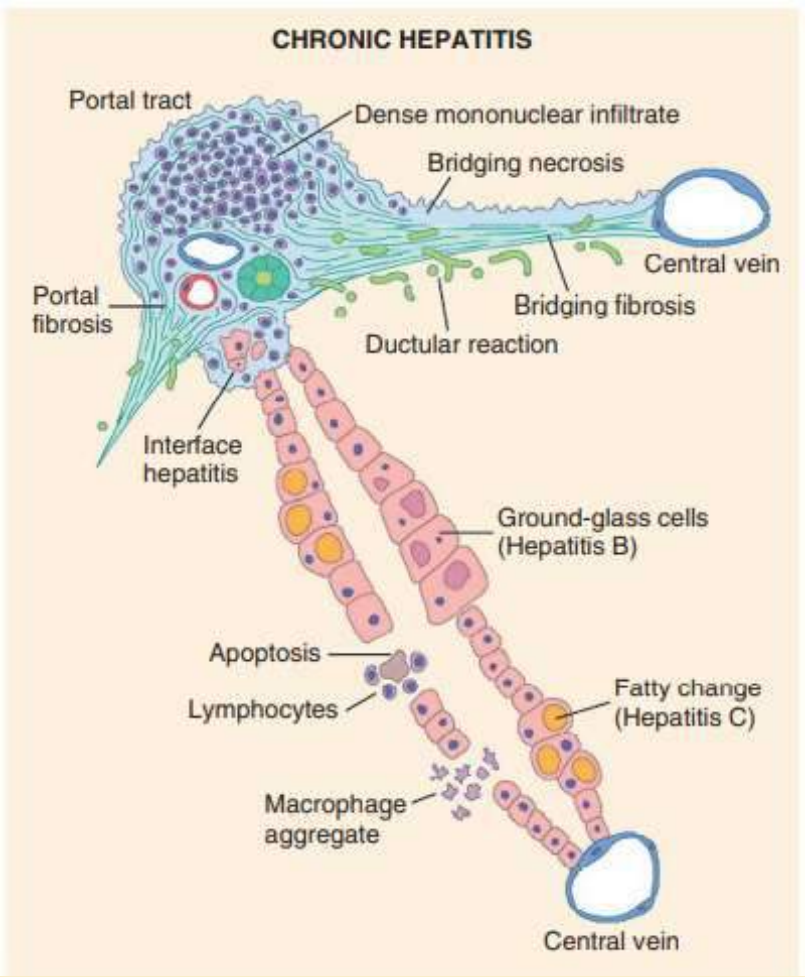
UNDERGRADUATE LECTURES 2025

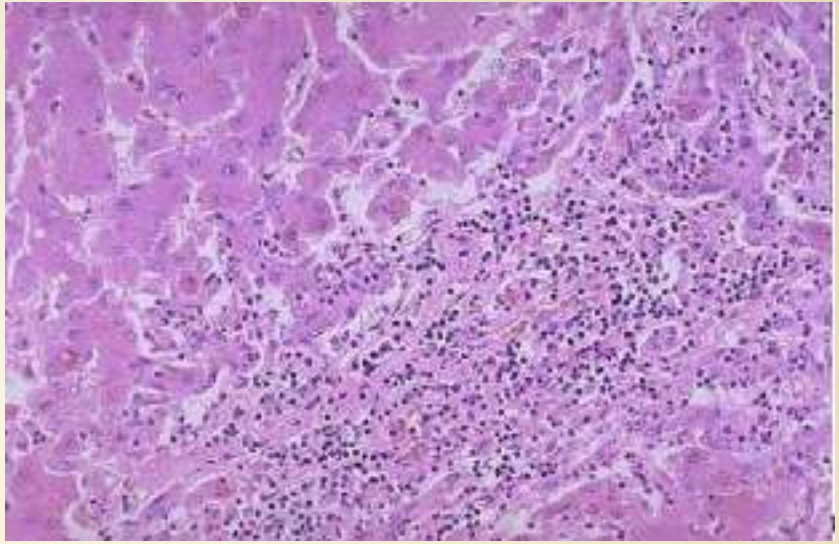
III. BIOPSY.


ACUTE HEPATITIS
Portal tract
Scant mononuclear infiltrate
Ballooning degeneration
Apoptosis
Macrophage aggregates
Cholestasis
Apoptosis
Central vein






CHRONIC HEPATITIS
Portal tract
Dense mononuclear infiltrate
Bridging necrosis
Central vein
Portal fibrosis
Bridging fibrosis
Ductular reaction
Interface hepatitis
Ground-glass cells (Hepatitis B)
Apoptosis
Lymphocytes
Fatty change (Hepatitis C)
Macrophage aggregate
Central vein



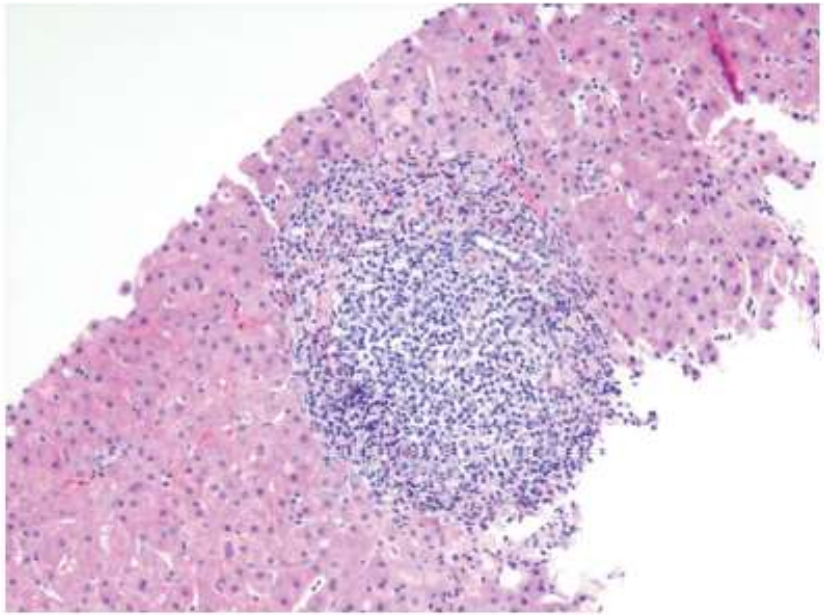


Fig. 16.15 Chronic viral hepatitis due to HCV, showing characteristic portal tract expansion by a dense lymphoid infiltrate.

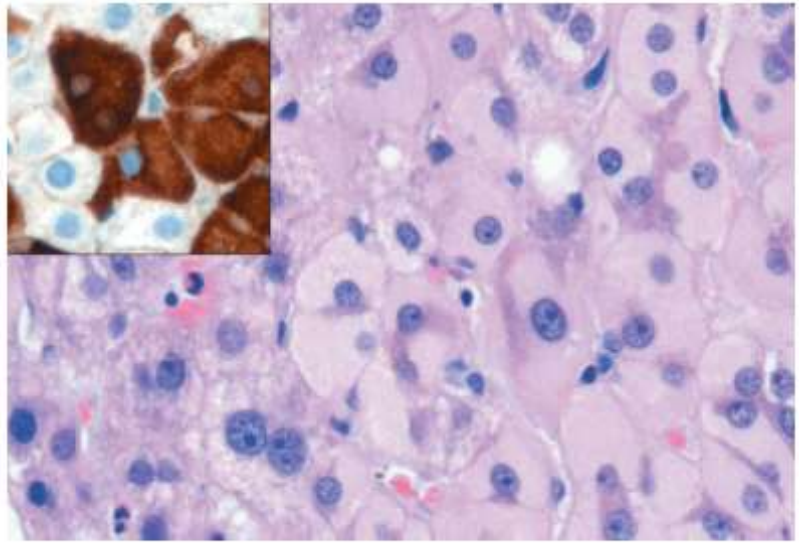
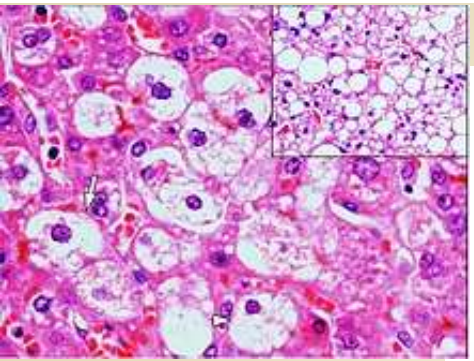
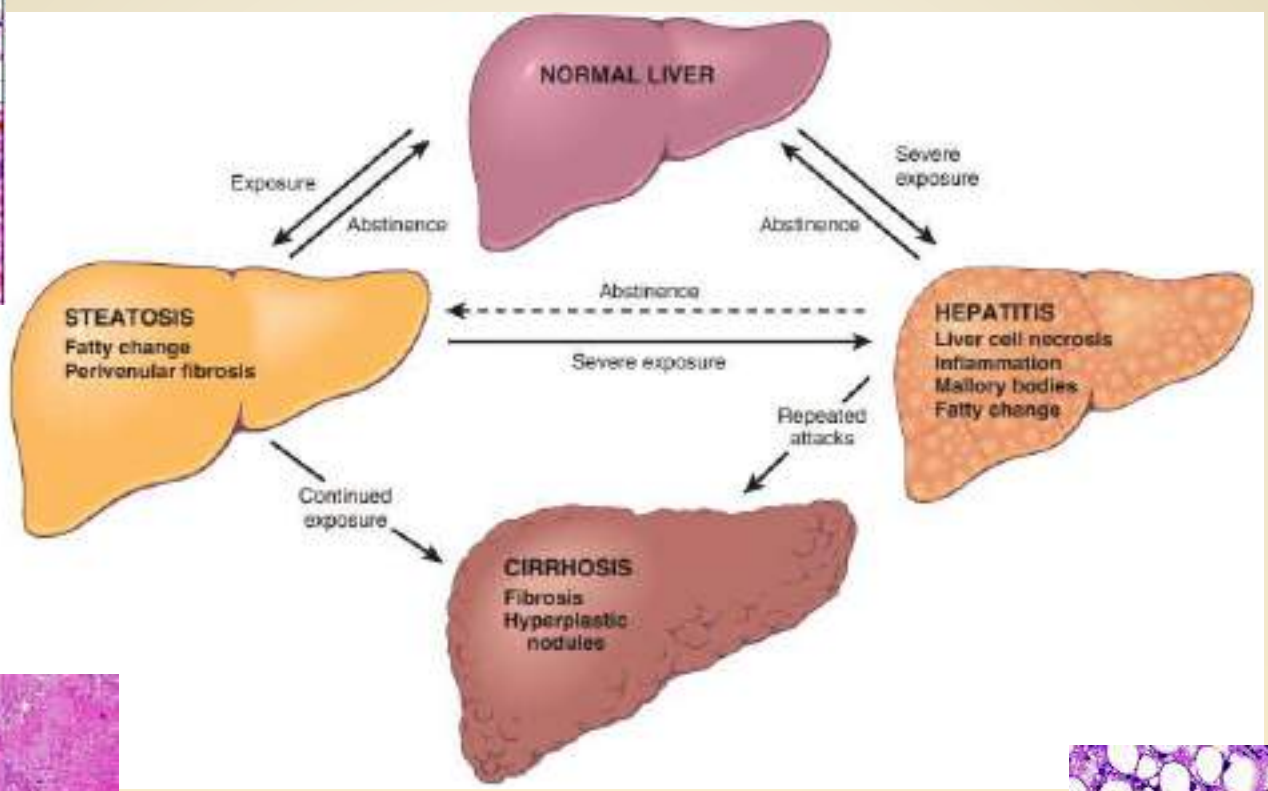
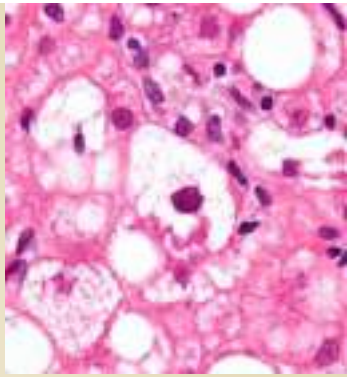
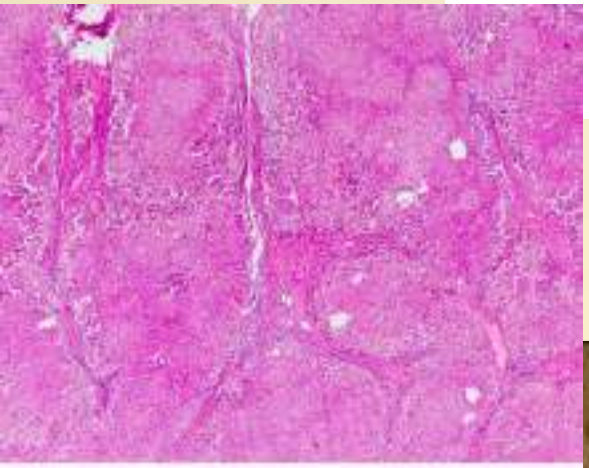


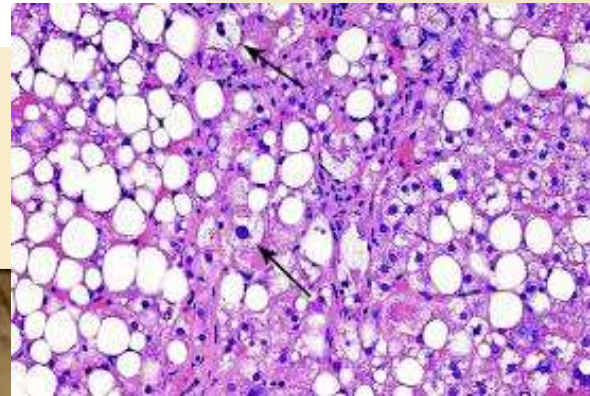
Fig. 16.14 Ground-glass hepatocytes in chronic hepatitis B, caused by accumulation of hepatitis B surface antigen. Hematoxylin-eosin staining shows the presence of abundant, finely granular pink cytoplasmic inclusions; immunostaining *(inset)* with a specific antibody confirms the presence of surface antigen *(brown)*.



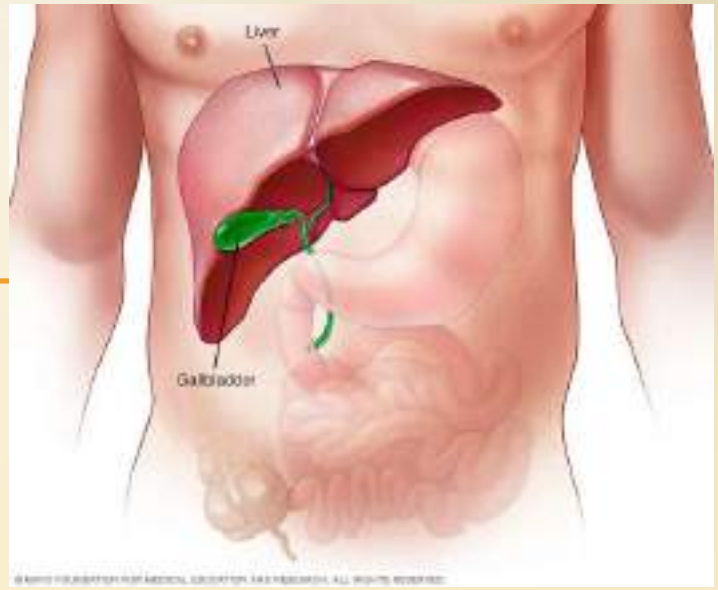

NORMAL LIVER
Exposure
Abstinence
Severe exposure
Abstinence
STEATOSIS
Fatty change
Perivenular fibrosis
Abstinence
Severe exposure
HEPATITIS
Liver cell necrosis
Inflammation
Mallory bodies
Fatty change
Repeated attacks
Continued exposure
CIRRHOSIS
Fibrosis
Hyperplastic nodules





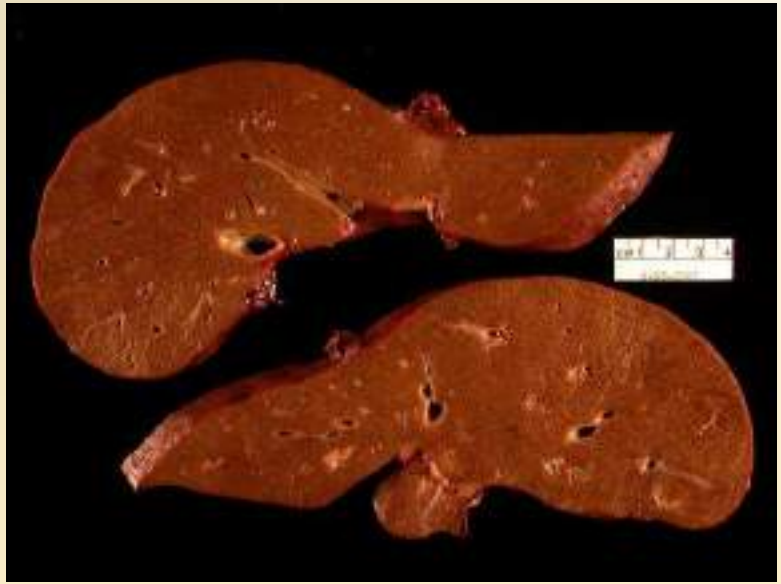


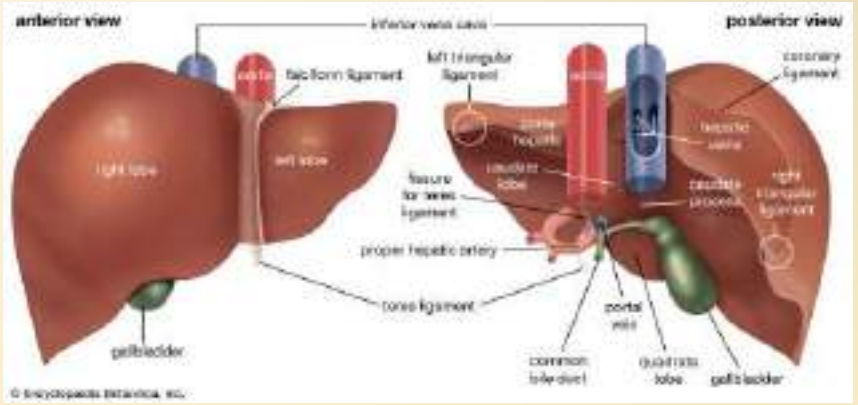
CIRRHOSIS AND CHOLESTASIS



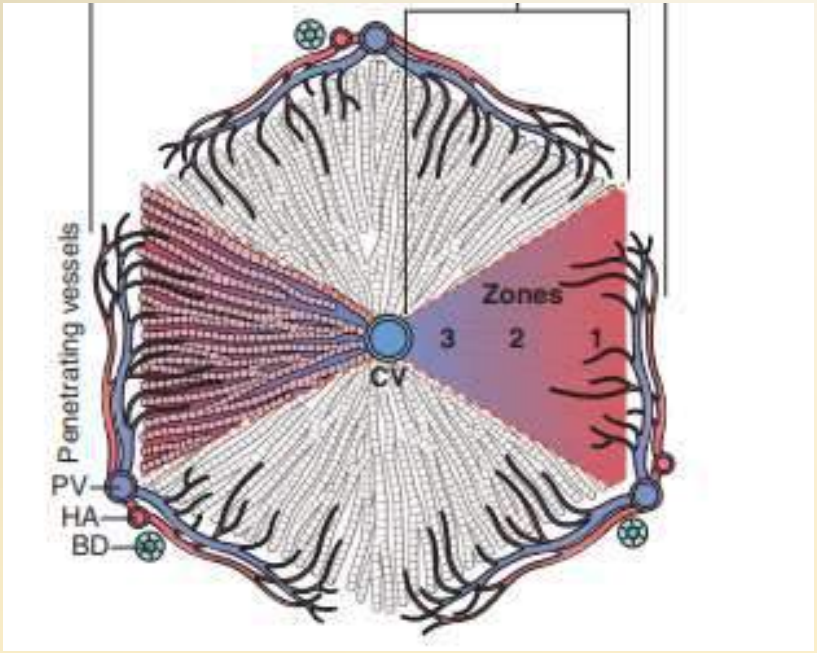
- The normal adult liver weighs 1400 to 1600 gm. It has a dual blood supply, with the portal vein providing 60% to 70% of hepatic blood flow and the hepatic artery supplying the remaining 30% to 40%.

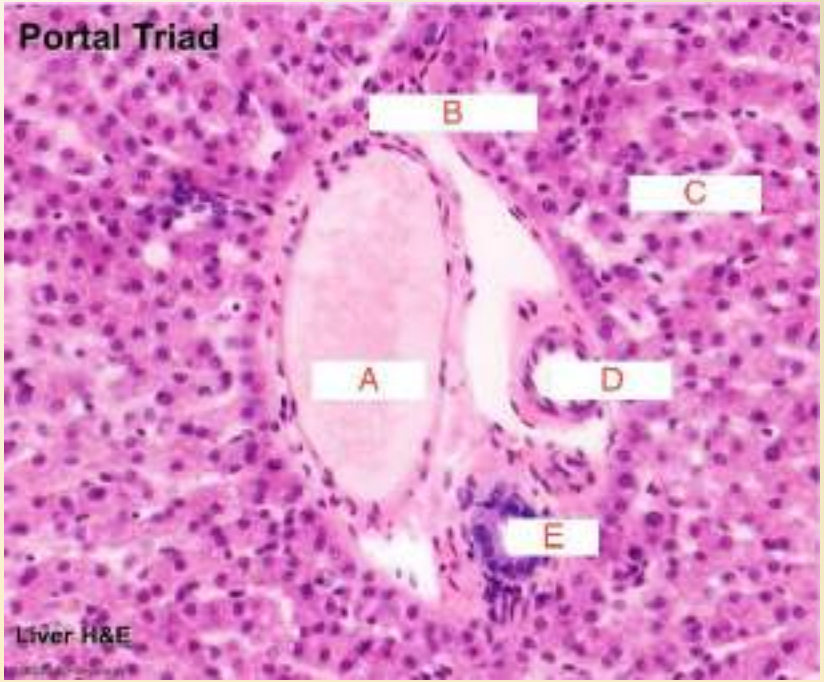
- Portal tract?

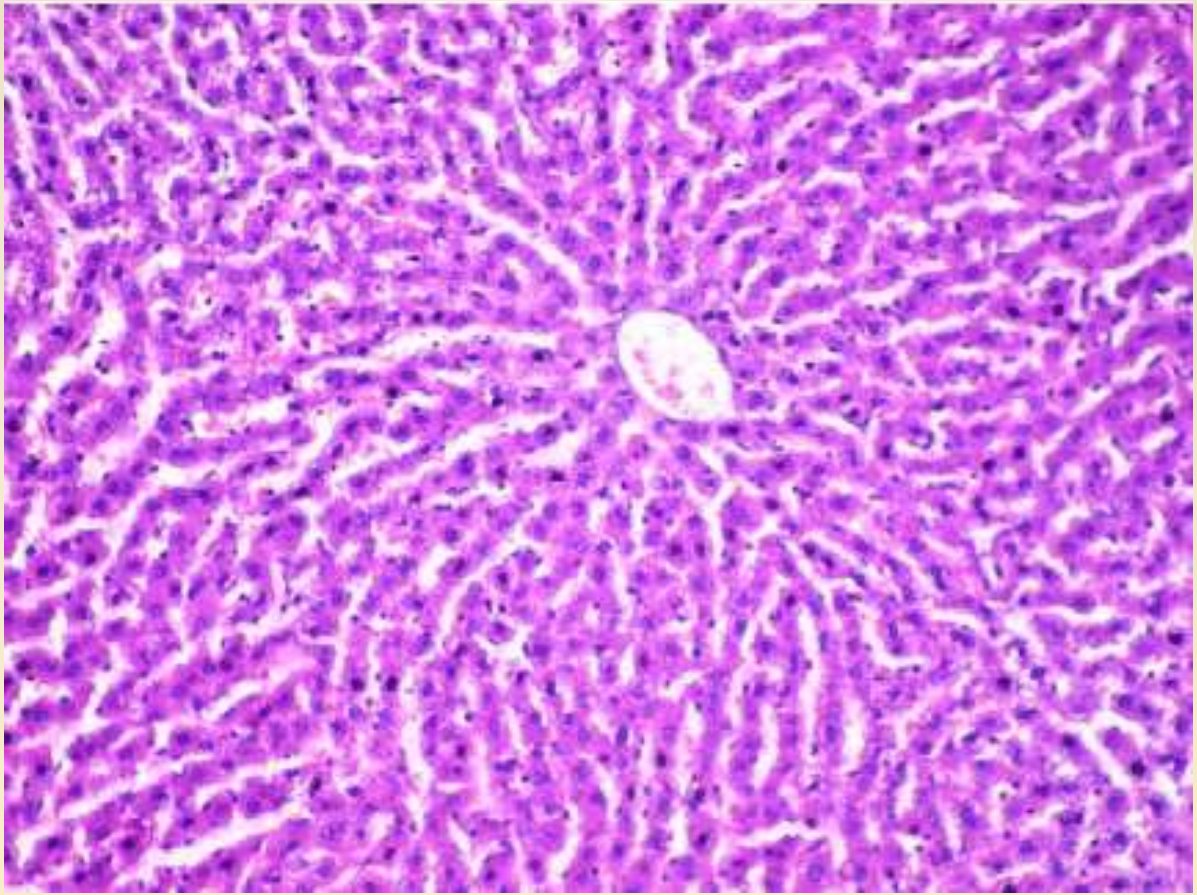




Models of liver anatomy







CIRRHOSIS

- Cirrhosis is the morphologic change most often associated with chronic liver disease; it refers to the diffuse transformation of the liver into regenerative parenchymal nodules surrounded by fibrous bands.

- The leading causes include:
  - chronic hepatitis B, C.
  - non-alcoholic fatty liver disease (NAFLD).
  - alcoholic liver disease
  - Drug induced liver injury
  - Cryptogenic (idiopathic) cirrhosis

DIAGNOSIS

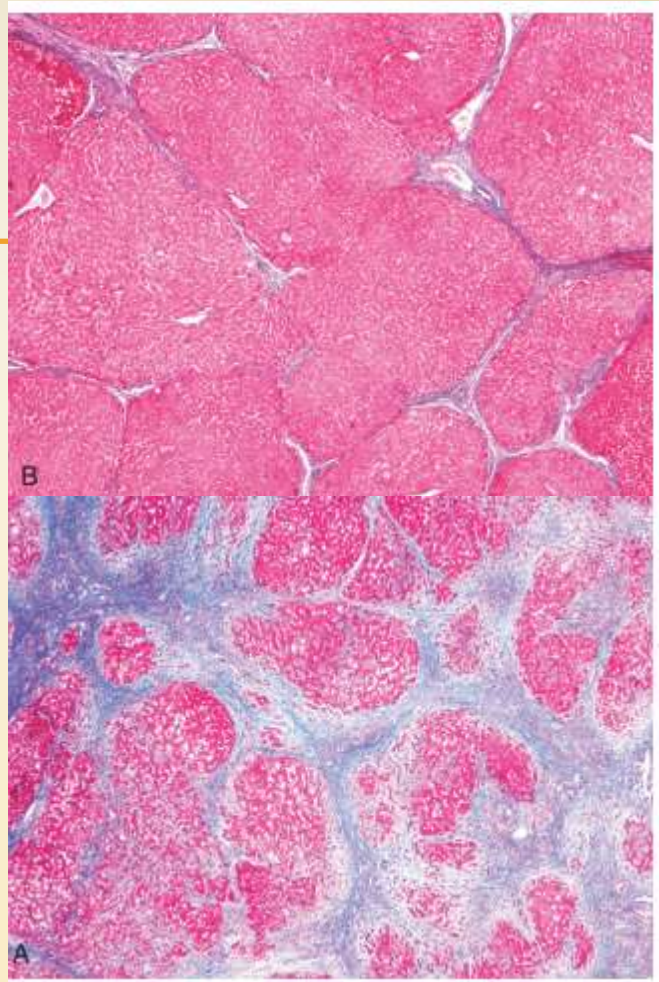
- 1. Liver function test.
- 2. Radiology.
- 3. Biopsy

HISTOPATHOLOGY

*diffuse transformation of the entire liver into regenerative parenchymal nodules surrounded by fibrous bands.

* ductular reactions.

* (Masson trichrome stain) highlights these fibrous septa.



CLINICAL FEATURES

- 1. 40% of individuals with cirrhosis are asymptomatic until the most advanced stages of the disease.
- 2. Non specific symptoms such as anorexia, weight loss, weakness.
- 3. signs and symptoms of liver failure e.g Jaundice, encephalopathy, and coagulopathy.
- 4. Pruritus, portal hypertention (intrahepatic vascular resistance).

5. Hyperestrogenemia:

- due to impaired estrogen metabolism in male patients with chronic liver failure can give rise to palmar erythema (a reflection of local vasodilatation) and spider angiomas of the skin. Such male hyperestrogenemia also leads to hypogonadism and gynecomastia.
- 6. hepatocellular carcinoma (HCC).

- Patients may have :

➢ Jaundice.

➢ Pruritus.

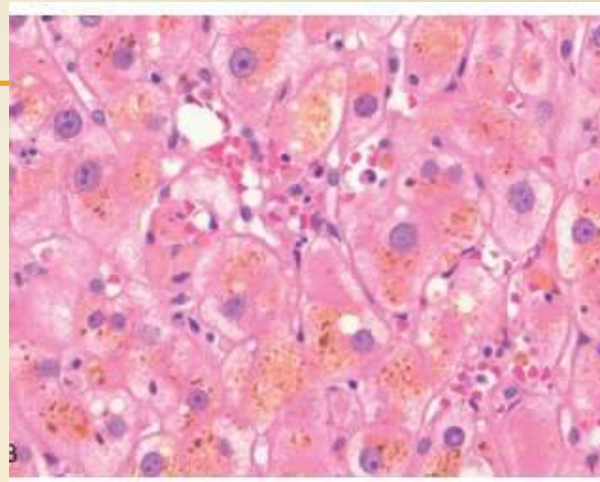
➢ skin xanthomas (focal accumulation of cholesterol).

➢ symptoms related to intestinal malabsorption, including nutritional deficiencies of the fat-soluble vitamins A, D, or K.

Lab:

elevated serum alkaline phosphatase and γ-glutamyl transpeptidase (GGT),

HISTOPATHOLOGY



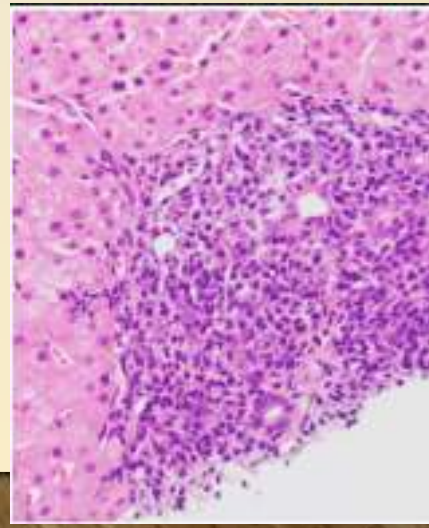
- accumulation of bile pigment within the hepatic parenchyma.
- Rupture of canaliculi leads to extravasation of bile, which is quickly phagocytosed by Kupffer cells.
- feathery degeneration:
- Droplets of bile pigment accumulate within hepatocytes, give them foamy appearance

CAUSES: A. BILE DUCT OBSTRUCTION.

- The most common cause of bile duct obstruction in adults is:
- extrahepatic cholelithiasis.
- malignant obstructions.
- postsurgical strictures.
- Obstructive conditions in children include :
- biliary atresia.
- cystic fibrosis.
- choledochal cysts.

C. PRIMARY BILIARY CHOLANGITIS.

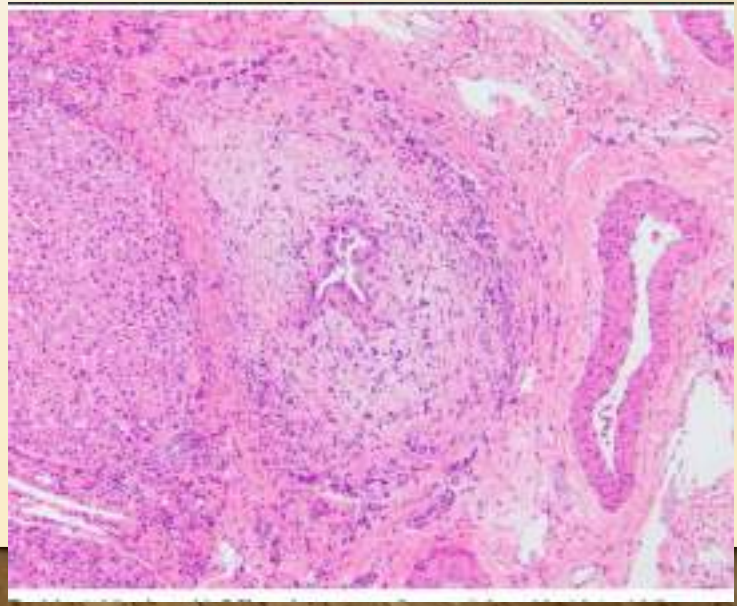
- autoimmune disease (Anti-mitochondrial antibodies) whose primary feature is nonsuppurative, inflammatory destruction of small- and medium-sized intrahepatic bile ducts.
- **Occur in** middle-age women, with a female-to-male ratio of 6:1. Its peak incidence is between 40 and 50 years of age.
- **Histology:**
- Dense lymphocytic infiltrate in portal tracts with granulomatous destruction and loss of medium sized interlobular bile ducts, focal and variable within the liver



D. PRIMARY SCLEROSING CHOLANGITIS

- Primary sclerosing cholangitis (PSC) is characterized by inflammation and obliterative fibrosis of intrahepatic and extrahepatic bile ducts, leading to dilation of preserved segments.

- Classic finding is "onion skin" fibrosis around affected bile ducts

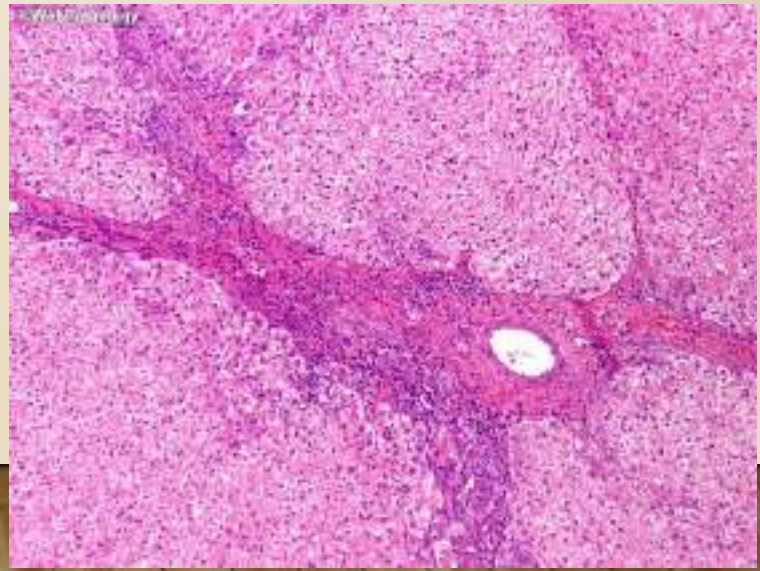


FOCAL NODULAR HYPERPLASIA: GROSS.

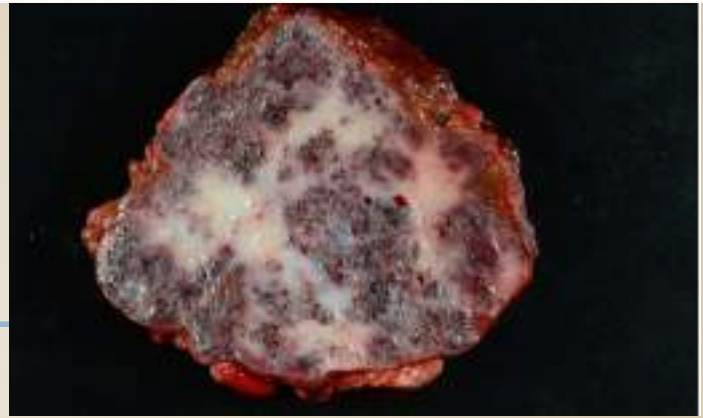
- well-demarcated, poorly encapsulated nodule in an otherwise normal liver.
- there is a central gray-white, depressed stellate scar from which fibrous septa radiate to the periphery.

FOCAL NODULAR HYPERPLASIA: MICROSCOPICALLY.

- The central scar contains large abnormal vessels and ductular reactions along the spokes of scar.
- The hyperplastic regions are composed of normal hepatocytes separated by thickened sinusoidal plates

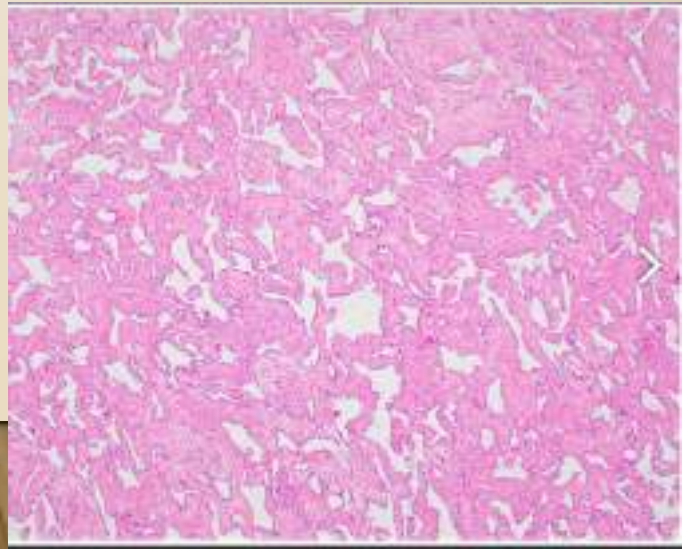


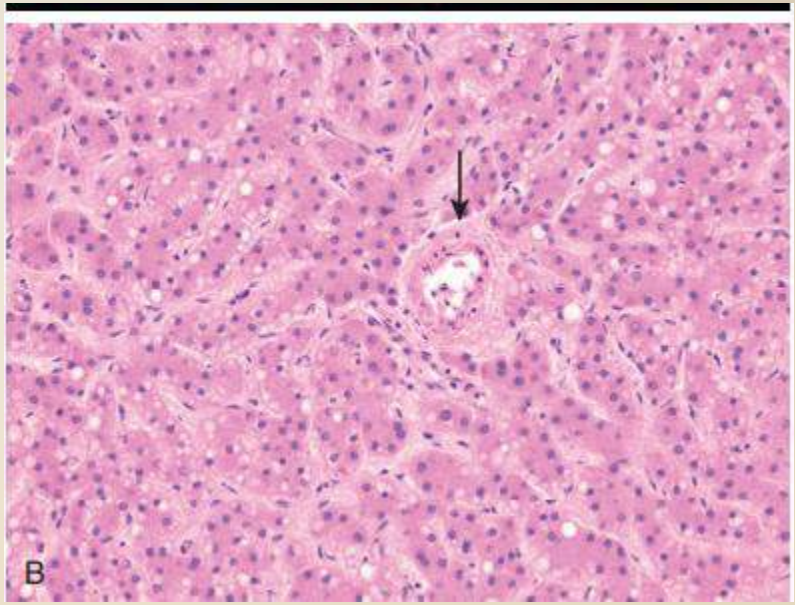
BENIGN NEOPLASMS



1. Cavernous hemangiomas:

- the most common benign tumor of the liver.
- Vast majority of hemangiomas are asymptomatic and require no intervention.
- **Gross description:**
- Well circumscribed with red-brown, spongy / honeycombed cut surface
- **Microscopic:**
- Circumscribed proliferation of variably sized, dilated and thin walled vessels





•solitary well circumscribed uncapsulated lesion.

Microscopic view showing cords of hepatocytes, with an arterial vascular supply (arrow) and no portal tracts.

HEPATOCELLULAR CARCINOMA (HCC)

- Primary malignancy of liver with hepatocellular differentiation.
- 80% of hepatocellular carcinoma cases arise in cirrhosis.
- Risk factors:
- Chronic liver disease leading to cirrhosis; most common etiologies leading to this include:

✓ chronic viral hepatits (HBV and HCV).

✓ heavy alcohol consumption.

✓ Metabolic syndrome: obesity, diabetes mellitus, and NAFLD .

✓ toxic injuries (aflatoxin, it synergizes with HBV (and perhaps also with HCV) to increase risk further.)..

✓ Inherited disorders, particularly hereditary hemochromatosis and α1AT deficiency, and to a lesser degree Wilson disease

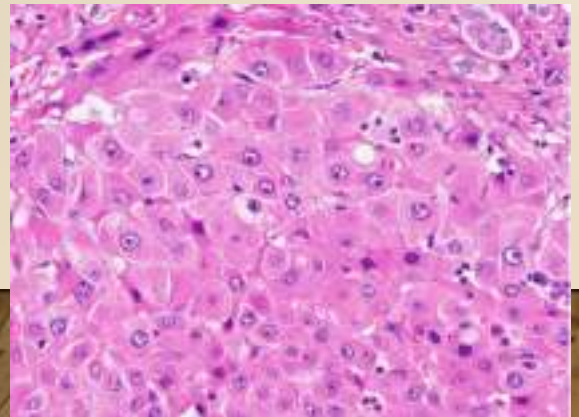
- HCC often appears to arise from premalignant precursors lesions:
- Hepatic adenoma.
- Chronic liver disease associated with cellular dysplasias :

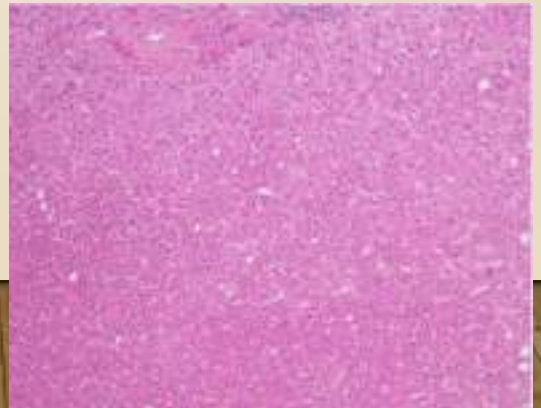
➢ large-cell change.:

❖ increase in both nuclear and cytoplasmic size, preserving nuclear to cytoplasmic ratio; nuclei are hyperchromatic, pleomorphic and frequently multinucleated.

➢ small-cell change:

❖ decreased cell volume, increased nuclear to cytoplasmic ratio, mild nuclear pleomorphism, hyperchromasia and cytoplasmic basophilia, giving the impression of nuclear crowding





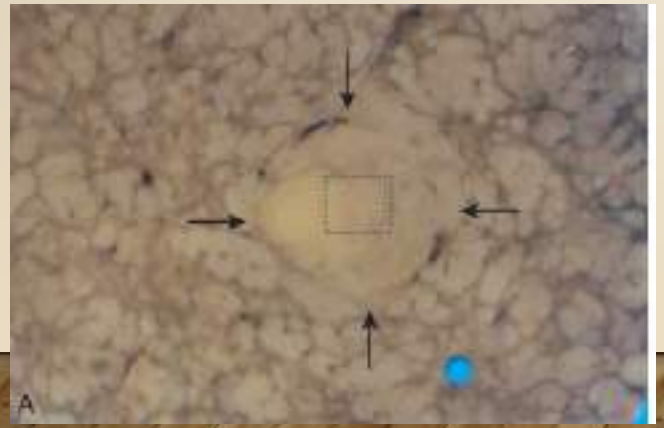
CLINICAL FEATURES

- ill-defined upper-abdominal pain, malaise, fatigue, weight loss.
- abdominal mass or abdominal fullness.
- Jaundice,, gastrointestinal or esophageal variceal bleeding.
- Metastatic : most commonly to the lungs.

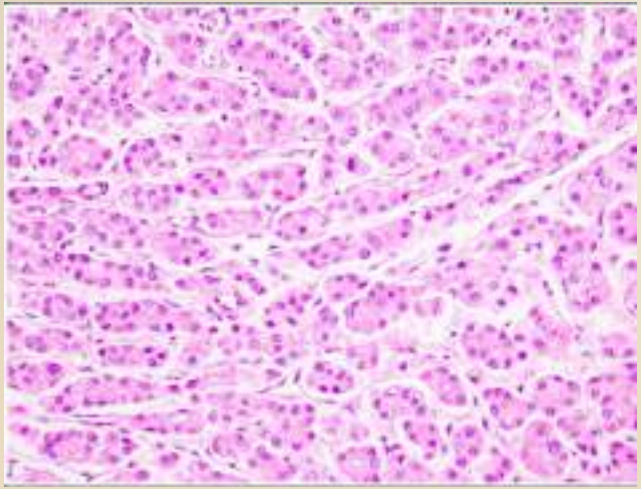
- Laboratory studies: Elevated serum levels of α-fetoprotein.
- imaging studies: Increasing arterialization during the development and progression of HCC .
- Death usually occurs from:
- (1) cachexia,
- (2) gastrointestinal or esophageal variceal bleeding
- (3) liver failure with hepatic coma.
- (4) rupture of the tumor with fatal hemorrhage

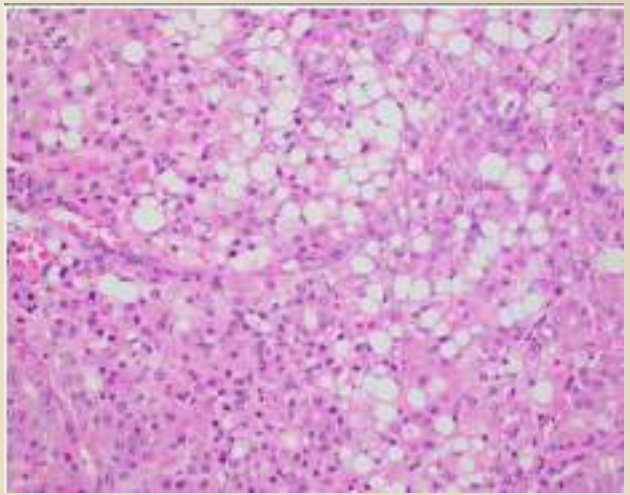
MORPHOLOGY

- HCC may appear grossly as:
- (1) a unifocal (usually large) mass.
- (2) multifocal, widely distributed nodules of variable size.
- (3) a diffusely infiltrative cancer,



- HCCs range from :
- well differentiated to highly anaplastic lesions.





Well-differentiated HCCs are composed of cells that look like normal hepatocytes and grow as thick trabeculae

tumor cells appear malignant on H&E and often cannot be distinguished from other poorly differentiated neoplasms;

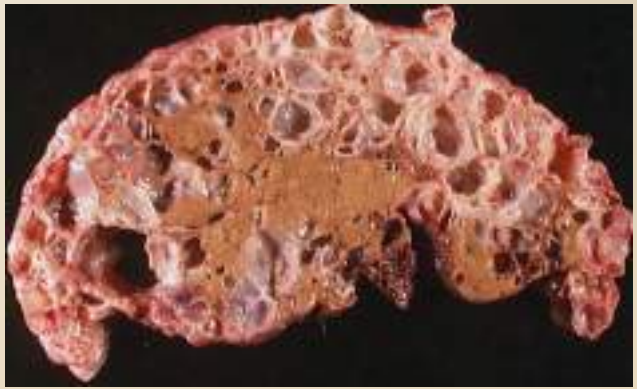
- The risk factors include :

✓ infestation by liver flukes .

✓ chronic inflammatory disease of the large bile ducts (such as primary sclerosing cholangitis),

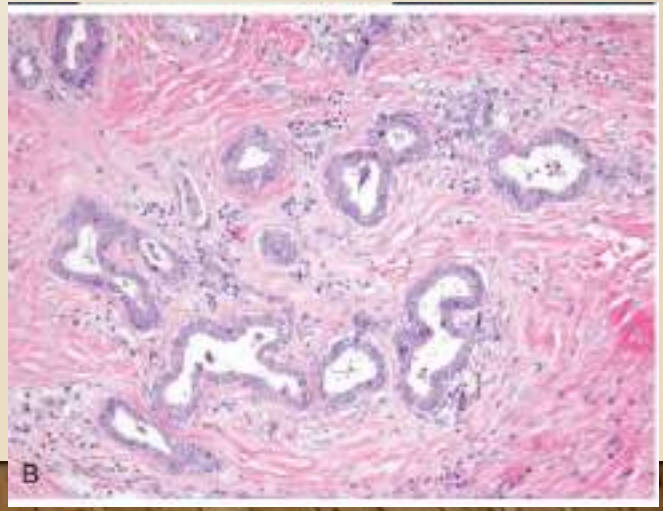
✓ hepatolithiasis.

✓ fibropolycystic liver disease.



MORPHOLOGY

- Most tumors appear as firm, gray nodules within the bile duct wall.
- Cholangiocarcinomas are typical mucin-producing adenocarcinomas. Most are well to moderately differentiated, growing as glandular/tubular structures lined by malignant epithelial cells.



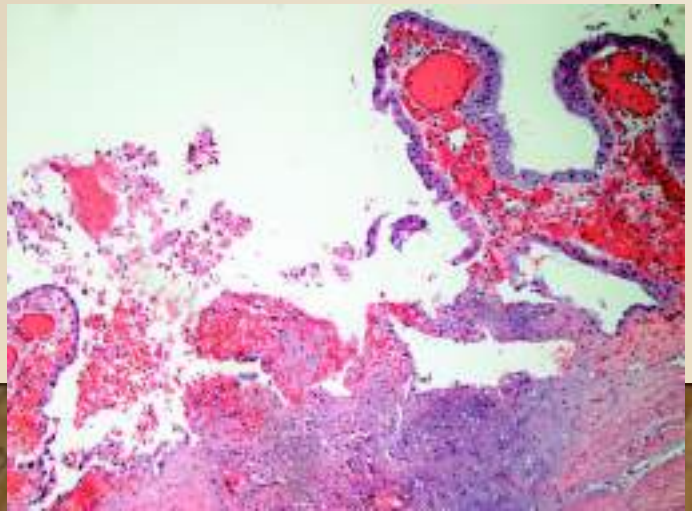
GALLBLADDER

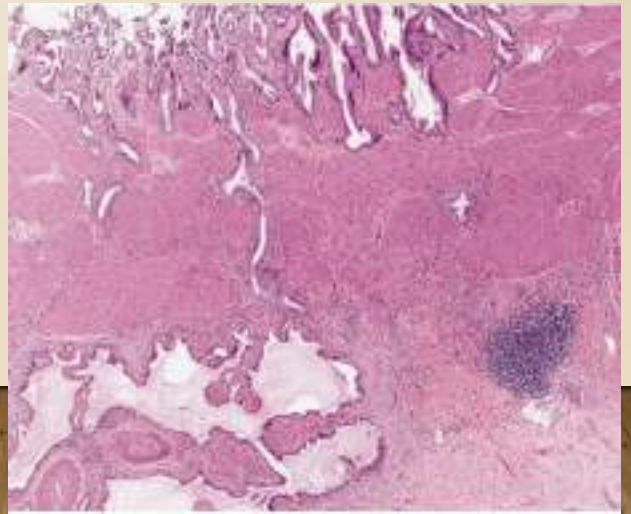
GALLBLADDER

- GALLSTONE DISEASE.
- CHOLECYSTITIS:

➢ Acute Calculous Cholecystitis: Acute inflammation of a gallbladder that contains stones.

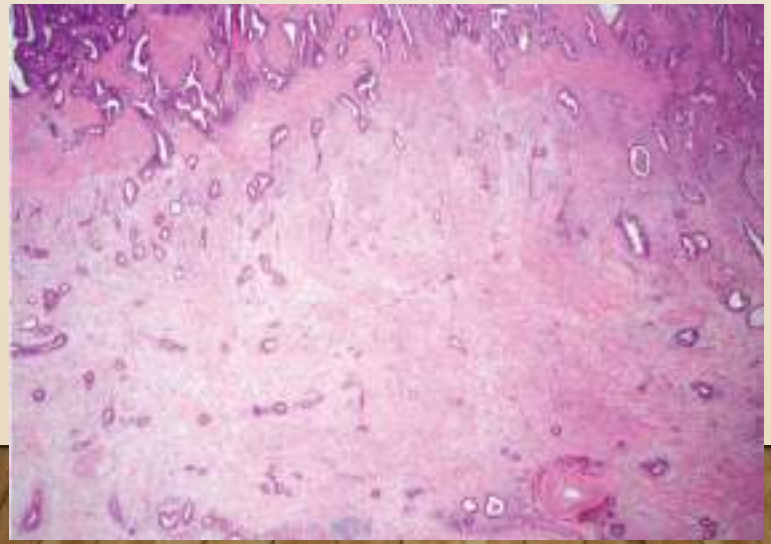
➢ Chronic Cholecystitis: occur due to repeated bouts of acute cholecystitis or de novo.





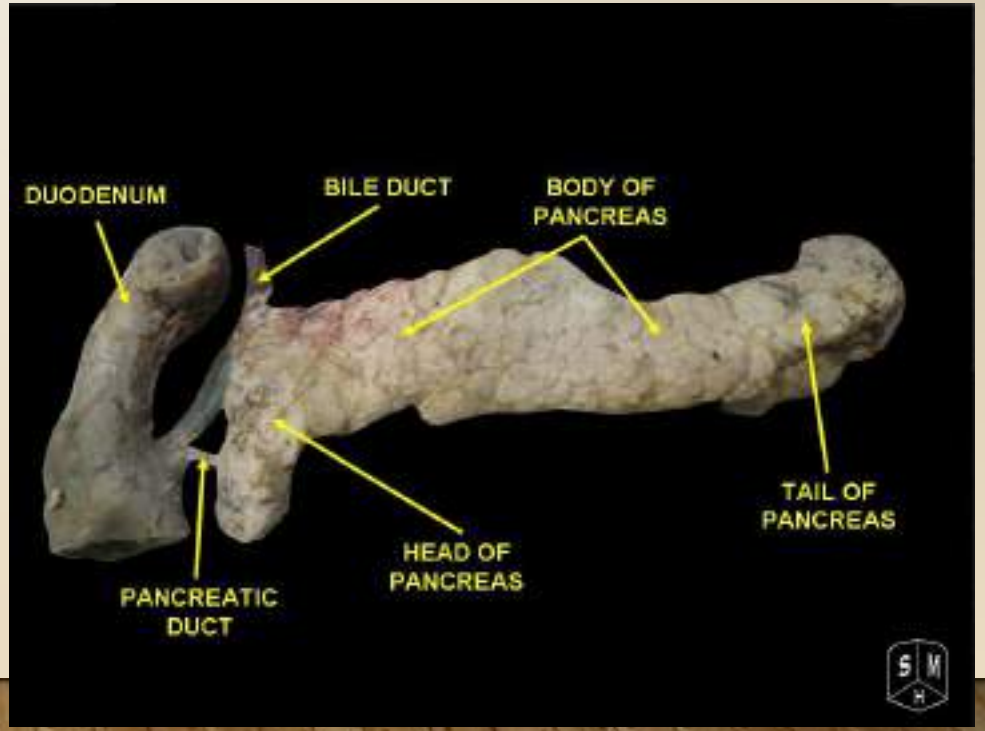
CARCINOMA OF THE GALLBLADDER:

- more common in women and occurs most frequently in the seventh decade of life.
- Presenting symptoms : abdominal pain, jaundice, anorexia, nausea and vomiting.
- Most carcinomas of the gallbladder are adenocarcinomas.



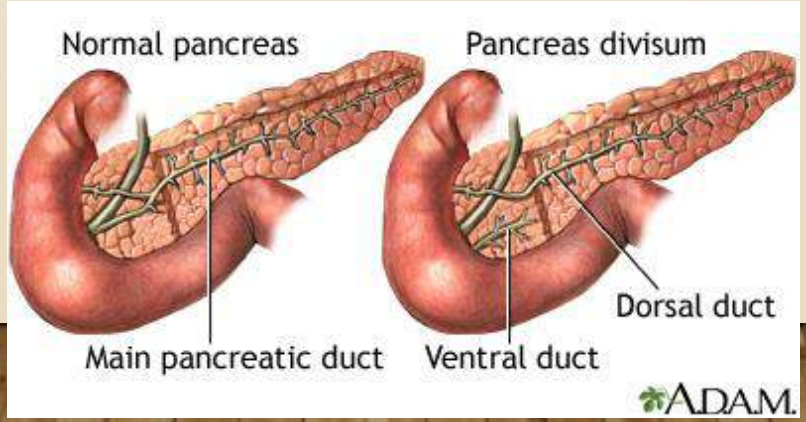
PANCREAS

- Congenital Anomalies.
- Pancreatitis.
- Pancreatic Cystic Neoplasms:
  - Pancreatic Carcinoma

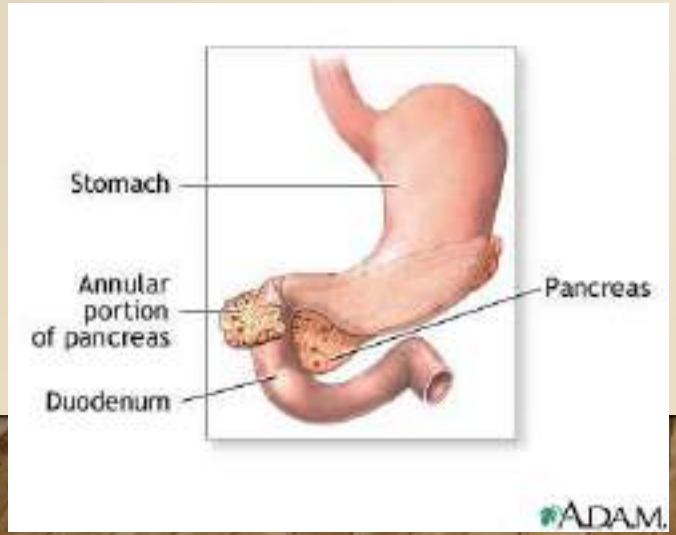


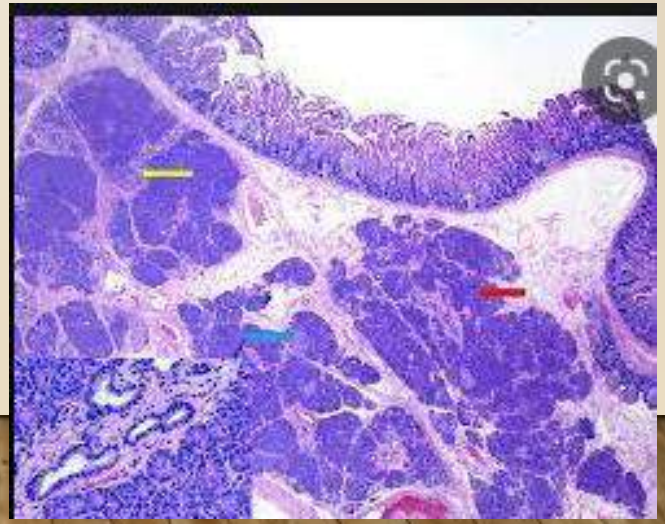
CONGENITAL ANOMALIES

- 1. Agenesis: the pancreas is totally absent.
- 2. Pancreas Divisum:
- most common congenital anomaly of the pancreas.
- caused by a failure of fusion of the fetal duct systems of the dorsal and ventral pancreatic primordia.



- 3. Annular Pancreas: ring of pancreatic tissue completely encircles the duodenum.
- 4. Ectopic Pancreas: favored sites are the stomach and duodenum, followed by the jejunum, Meckel diverticulum, and ileum.



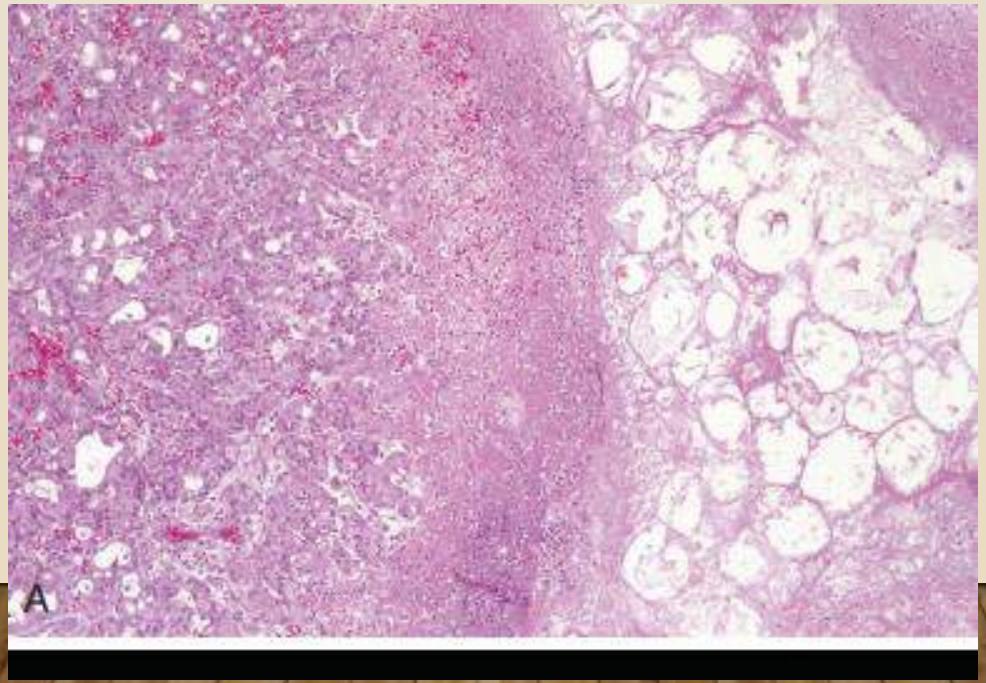


PANCREATITIS:ACUTE PANCREATITIS

- Acute pancreatitis is a reversible inflammatory disorder that varies in severity, from focal edema and fat necrosis to widespread hemorrhagic necrosis.
- Etiology:
  - Gallstones.
  - Non–gallstone-related obstruction.
  - Medications.
  - Infections.
  - Metabolic disorders

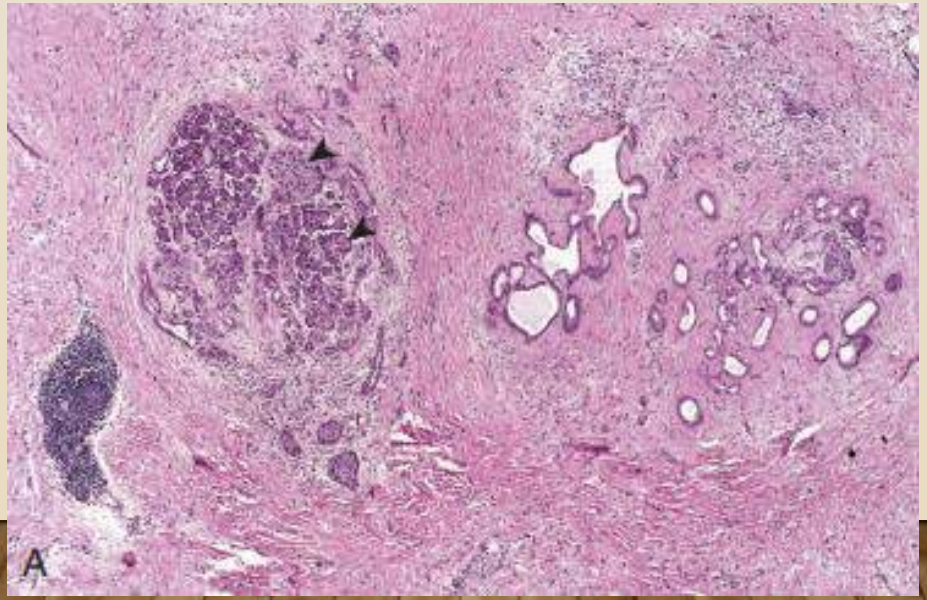
MORPHOLOGY

- acute inflammatory cell infiltrate admixed with edema and fibrinous exudate.
- patchy necrosis.



MORPHOLOGY

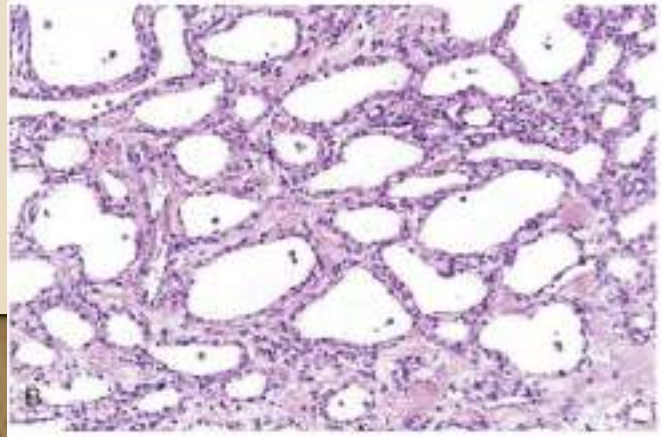
- Chronic pancreatitis is characterized by parenchymal fibrosis, reduced number and size of acini, and variable dilation of the pancreatic ducts



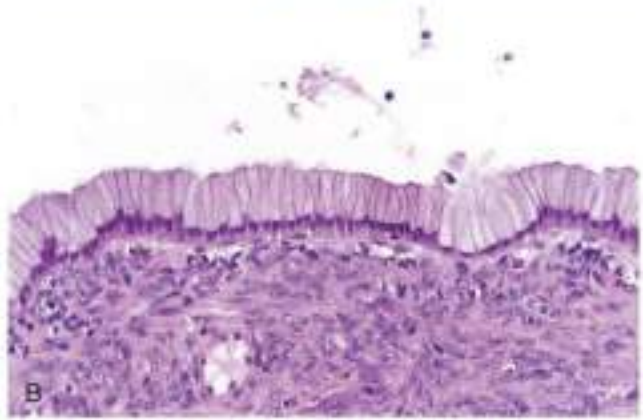
PANCREATIC NEOPLASMS: CYSTIC NEOPLASMS

- 1. Serous cystadenomas :
- composed of glycogen-rich cuboidal cells

surrounding small cysts containing clear, straw colored fluid.

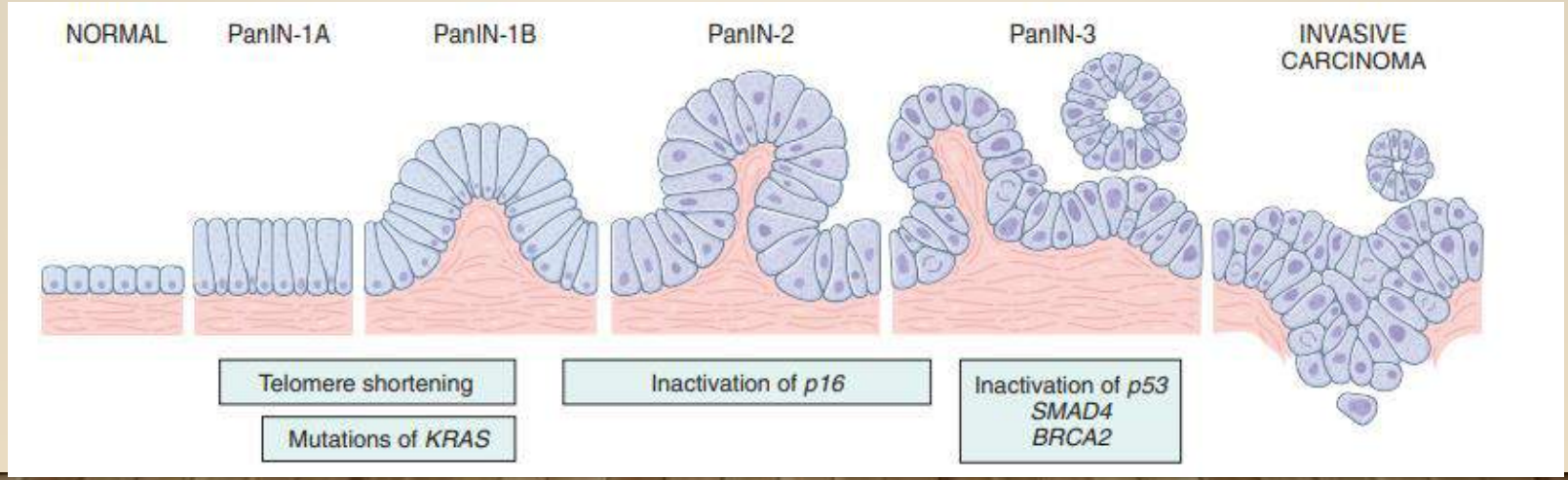


2. mucinous cystic neoplasm:
the cysts are lined by a columnar mucinous epithelium with an associated densely cellular stroma resembling that of the ovary.



PANCREATIC CARCINOMA

- pancreatic cancer arises as a consequence of inherited and acquired mutations in cancer-associated genes.



MORPHOLOGY

- Carcinomas of the pancreas usually are hard, gray-white, stellate, poorly defined masses.
- On microscopic examination,:
- pancreatic carcinoma usually

is a moderately to poorly differentiated adenocarcinoma

forming abortive glands with mucin secretion

or cell clusters and exhibiting an aggressive,

deeply infiltrative growth pattern