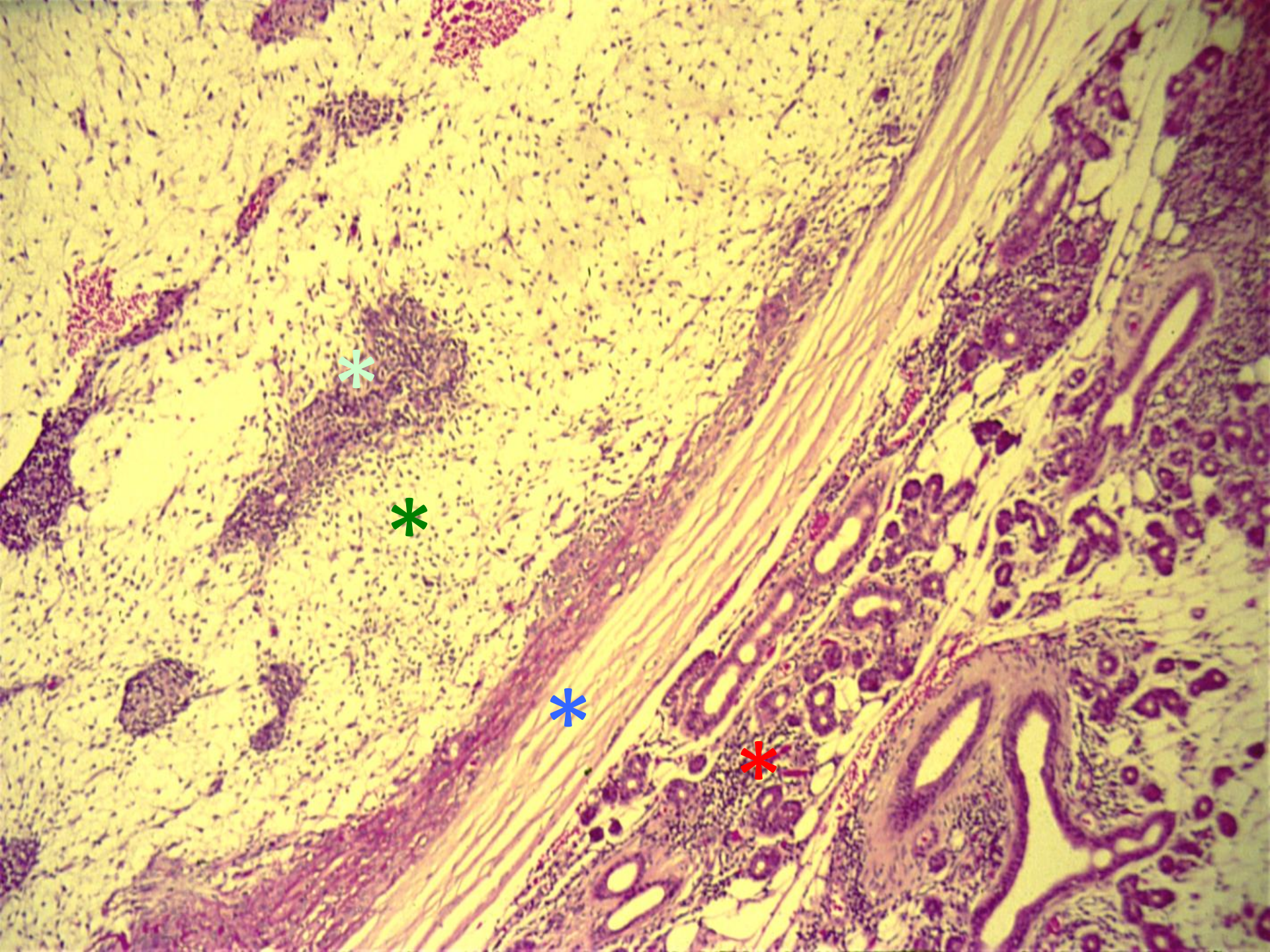
Digestive system block-Pathology practical. Revision

Pathology Team

Case 1

Pleomorphic Adenoma of The Salivary Gland

Most common place: parotid gland



Pleomorphic adenoma of the salivary gland:

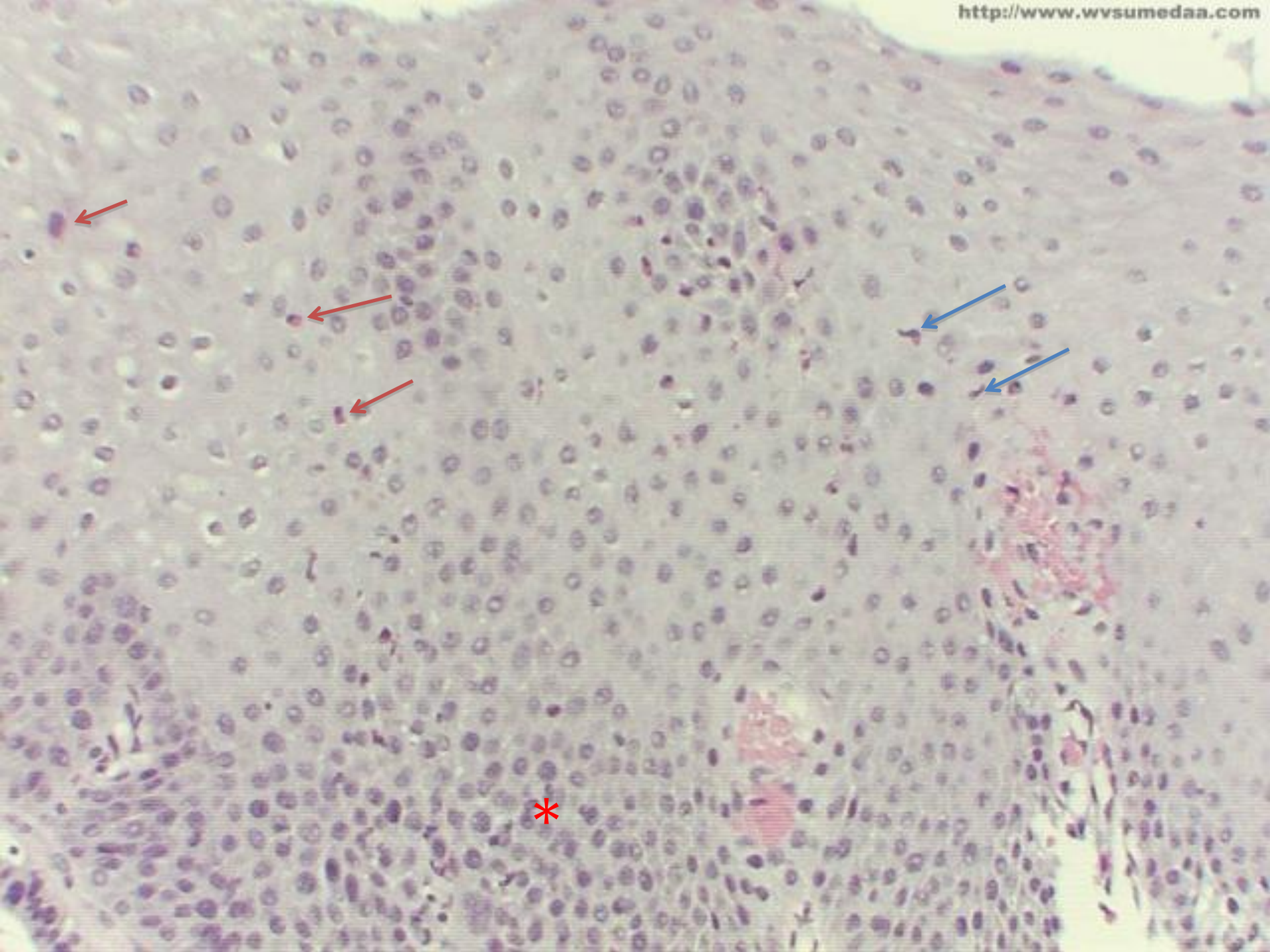
Section shows an incomplete fibrous capsule * separating the tumour from normal salivary * gland:

- Tumor shows mixed cellular components usually benign:
  - Epithelial and myoepithelial that are lining the glands and acini →

    (this constitute the epithelial component *.
  - chondriod (cartilagenous) } Mesenchymal components
  - myxoid (loose) elements.
- Epithelial areas shows small ducts, acini and strands or sheets of cells.
- Myxoid areas are formed of loose myxomatous tissue * and chondriod areas consists of pale blue matrix.

Case 2

REFLUX/GERD


http://www.wvsumedaa.com

Microscopically:

1. Intraepithelial inflammatory Cells
    - Eosinophils (red arrow)
    - Neutrophils (blue arrow)
    - Lymphocytes
2. Basal zone hyperplasia *
3. Lamina Propria papillae elongated and congested (not clear in the slide)

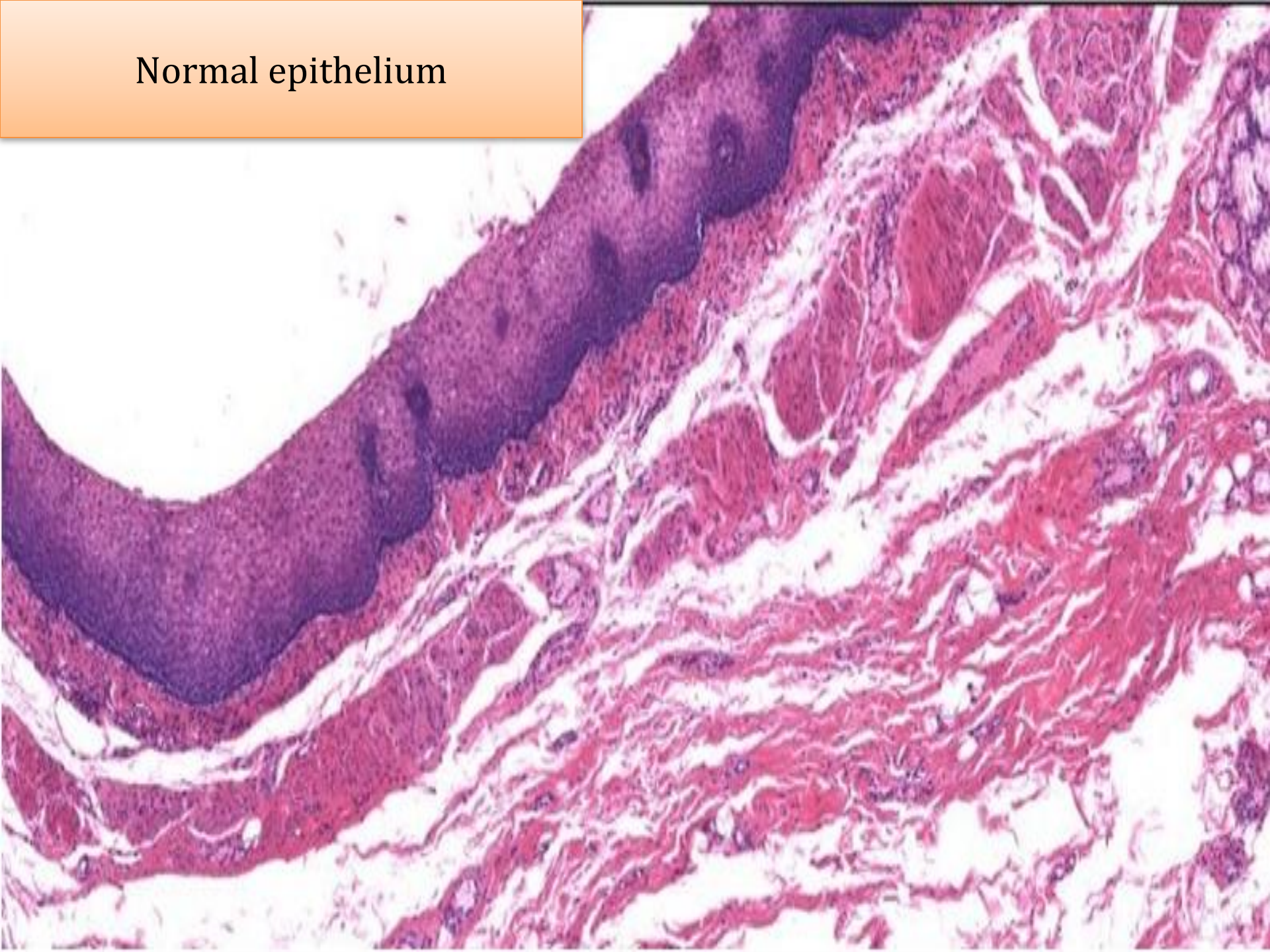
Complications:

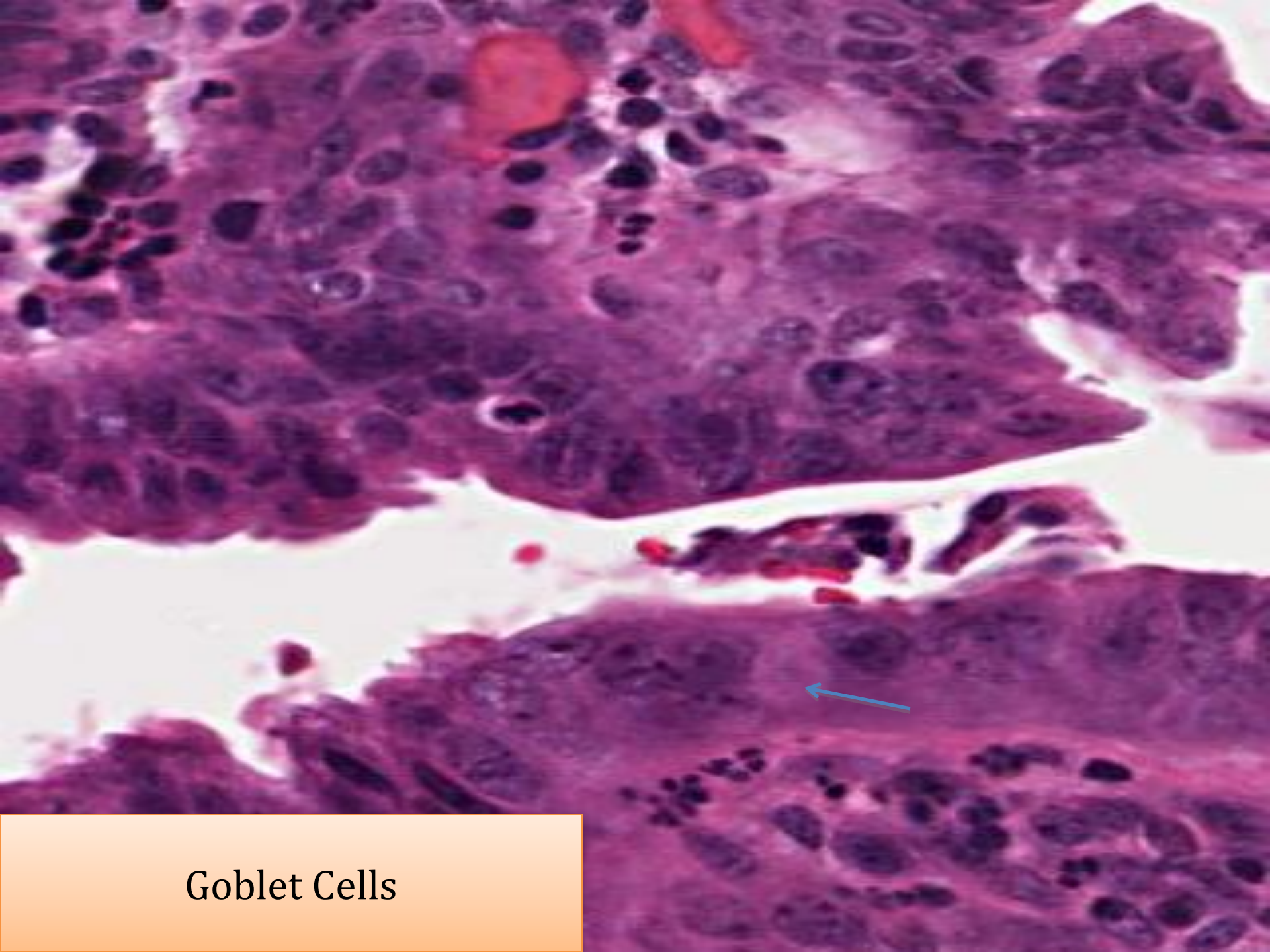
If GERD becomes chronic → Barrett 'esophagitis (changes of the lining epithelium of the esophagus from squamous to columnar cells)

Case 3

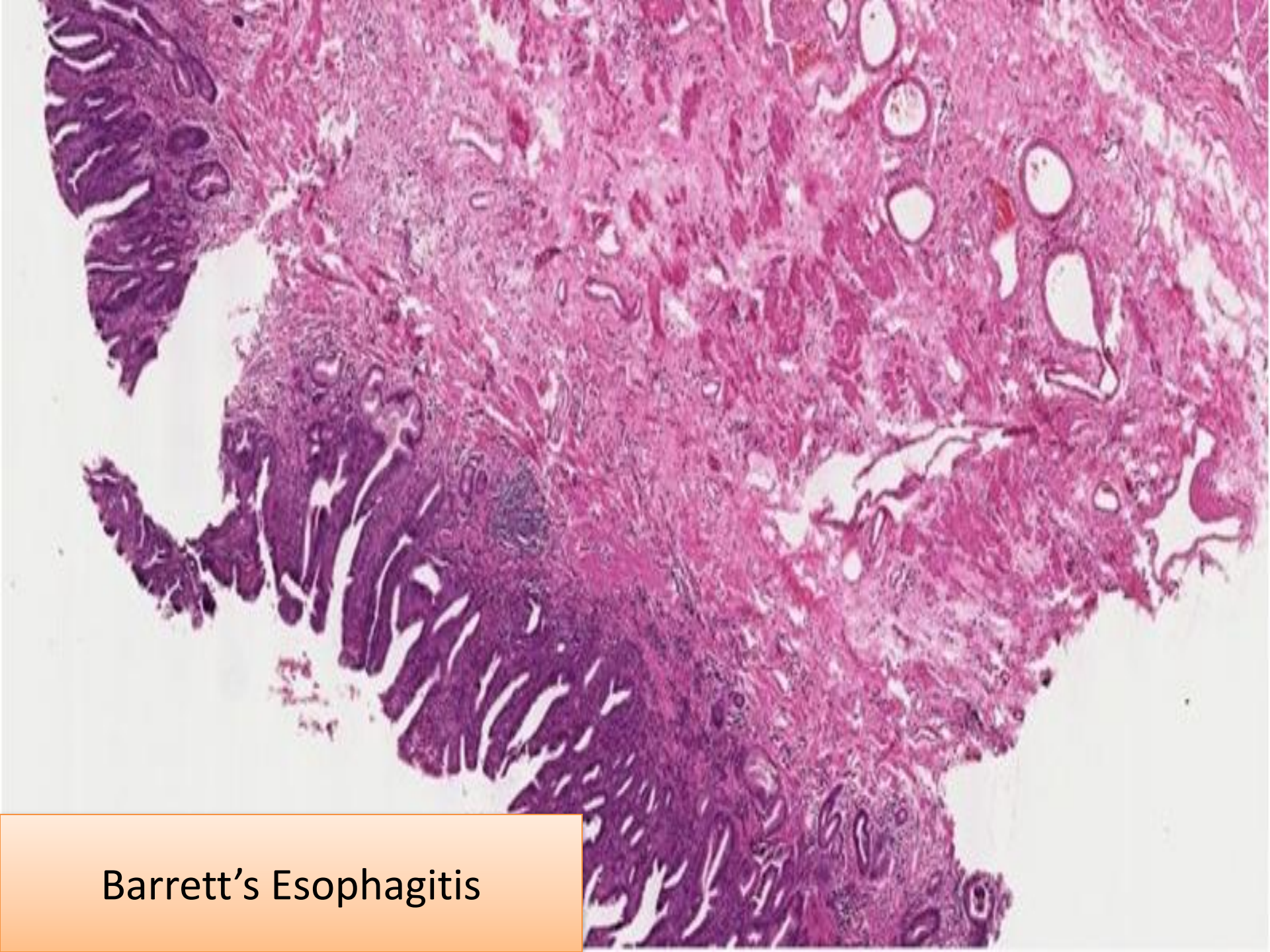
Barrett ’s Esophagitis

Normal epithelium





Goblet Cells



Barrett’s Esophagitis

Cause: duo to chronic GERD

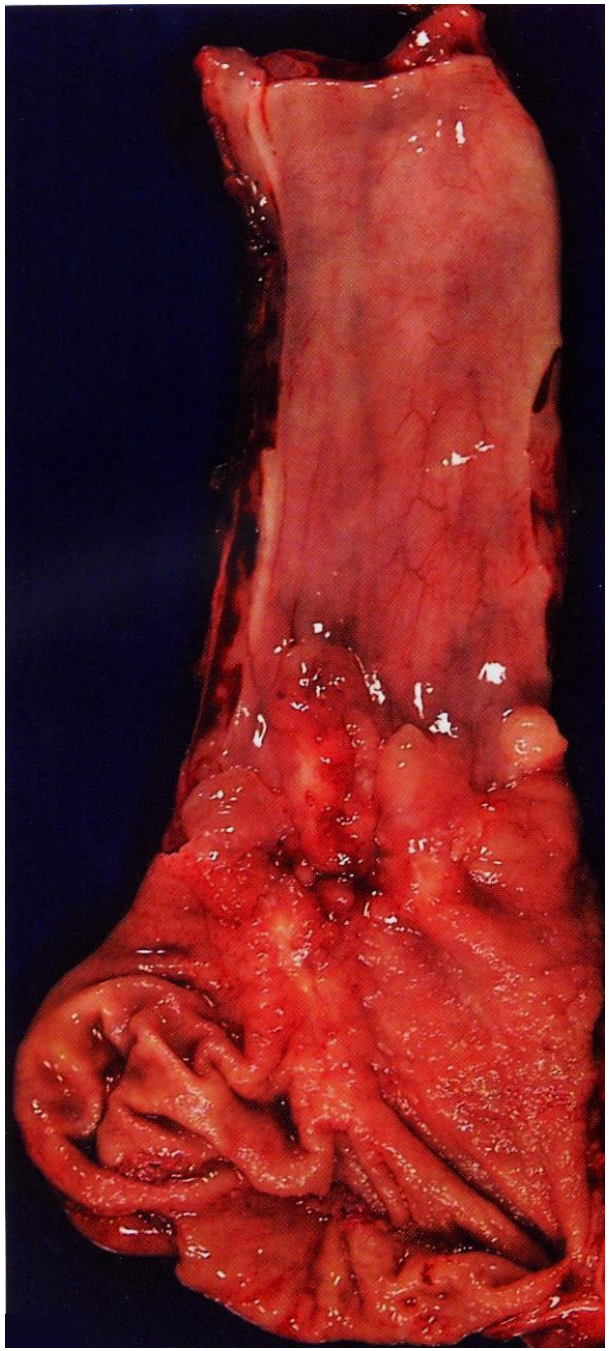
Microscopically:

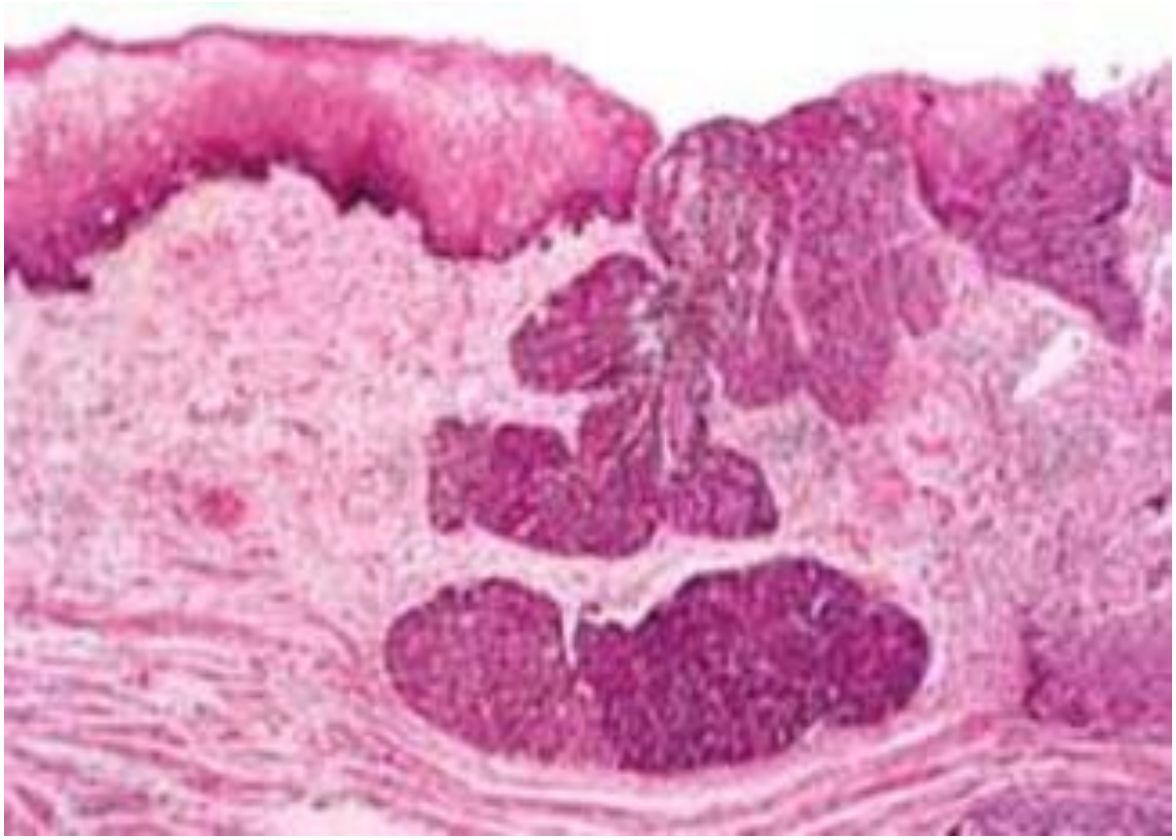
Replacement of the normal squamous cell epithelium of the esophagus into a columnar epithelium

Complication: Barrett's esophagitis can be complicated to adenocarcinoma.

Case 4

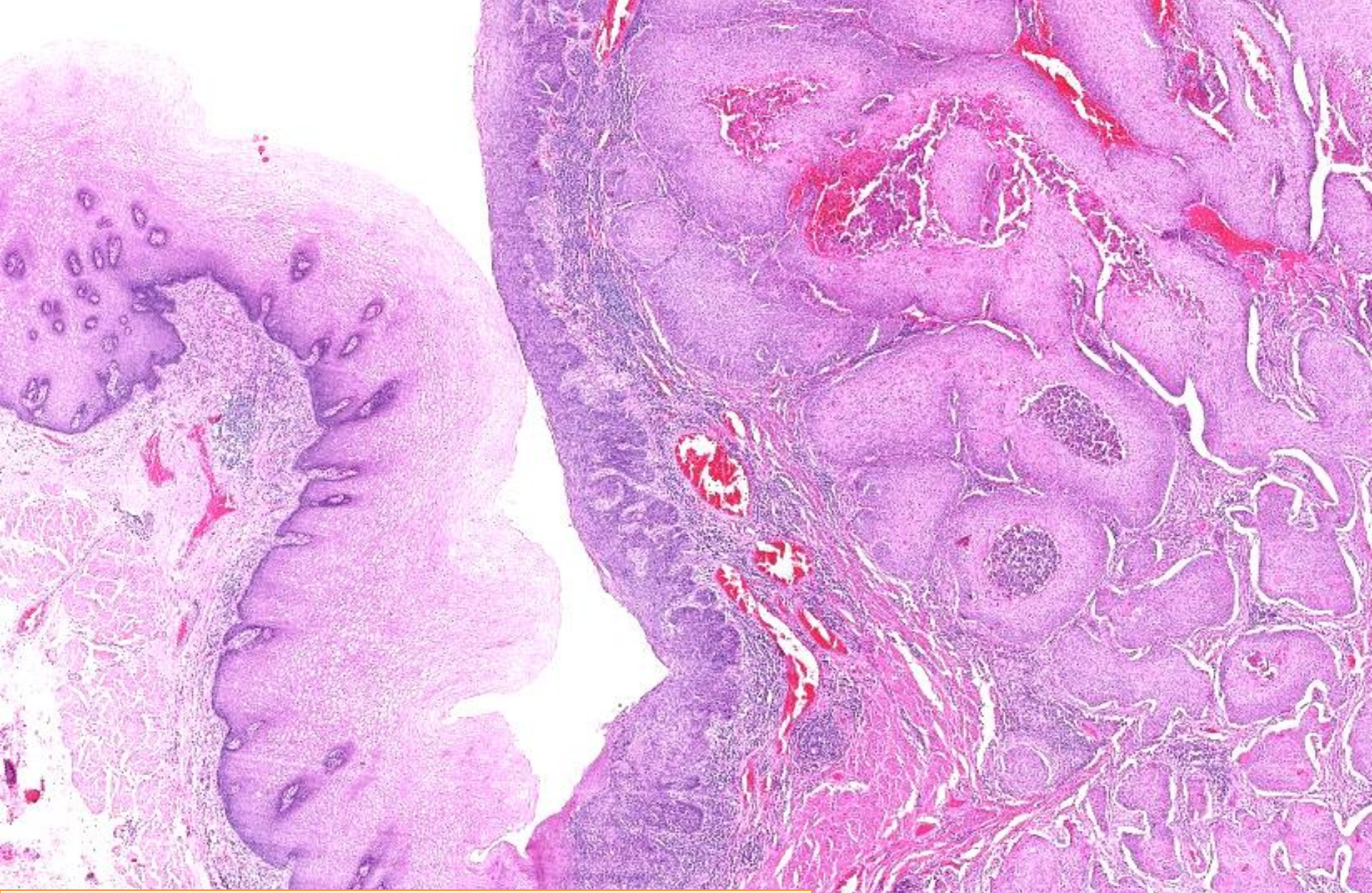
Squamous Carcinoma of Esophagus





DYSPLASIA→IN-SITU→INFILTRATION

The most common cancer of the esophagus is squamous cell carcinoma



Slide showing invasion of the sub-mucosa by moderately differentiated malignant squamous cells.

The most common type of esophageal carcinomas is squamous cell carcinoma then adenocarcinoma (barrett's esophagitis)

Risk factors: alcohol and tobacoo use , caustic esophageal injury ,achalasia and frequent consumption of very hot beverages

Case 5

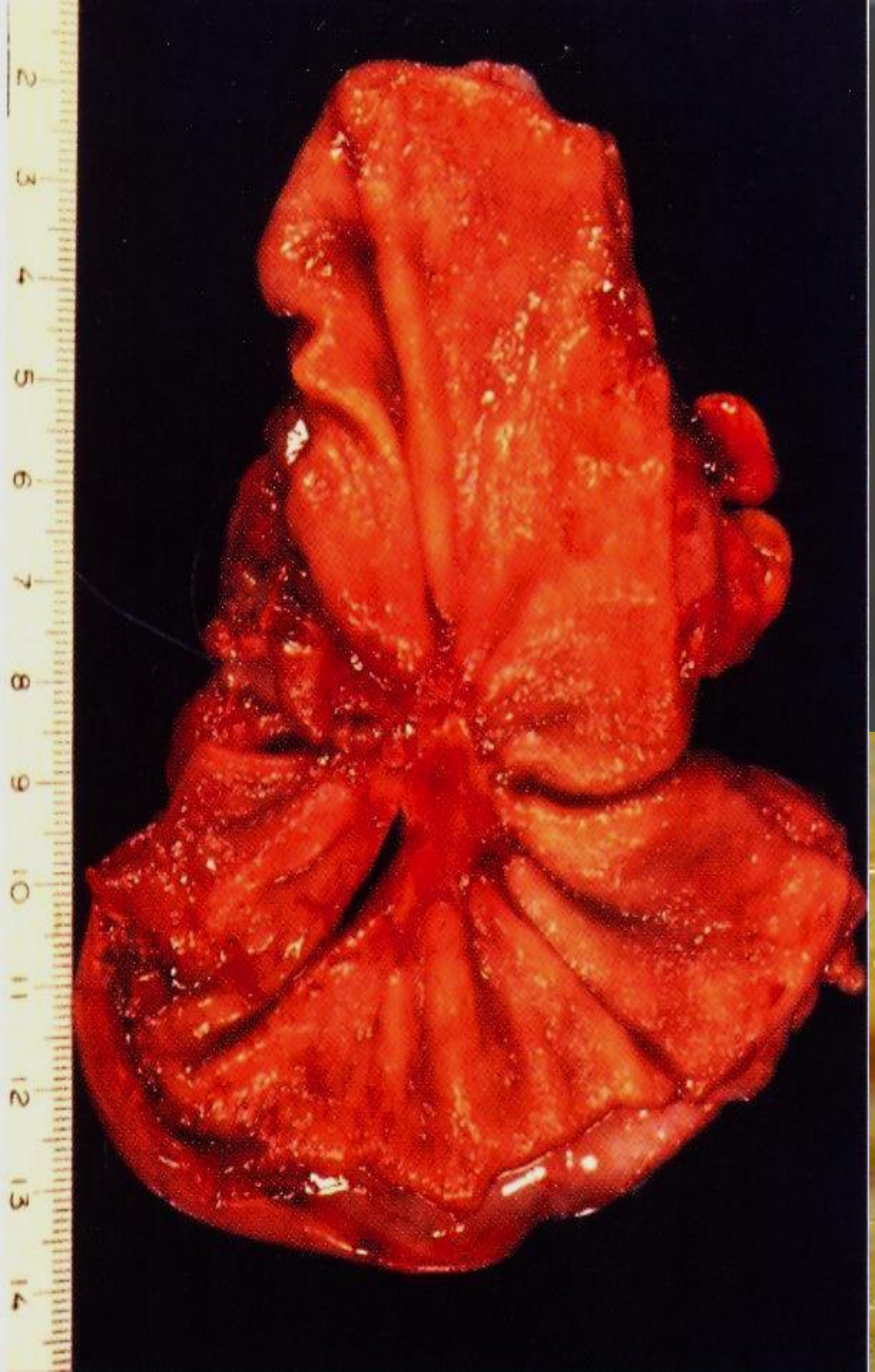
Peptic ulcer

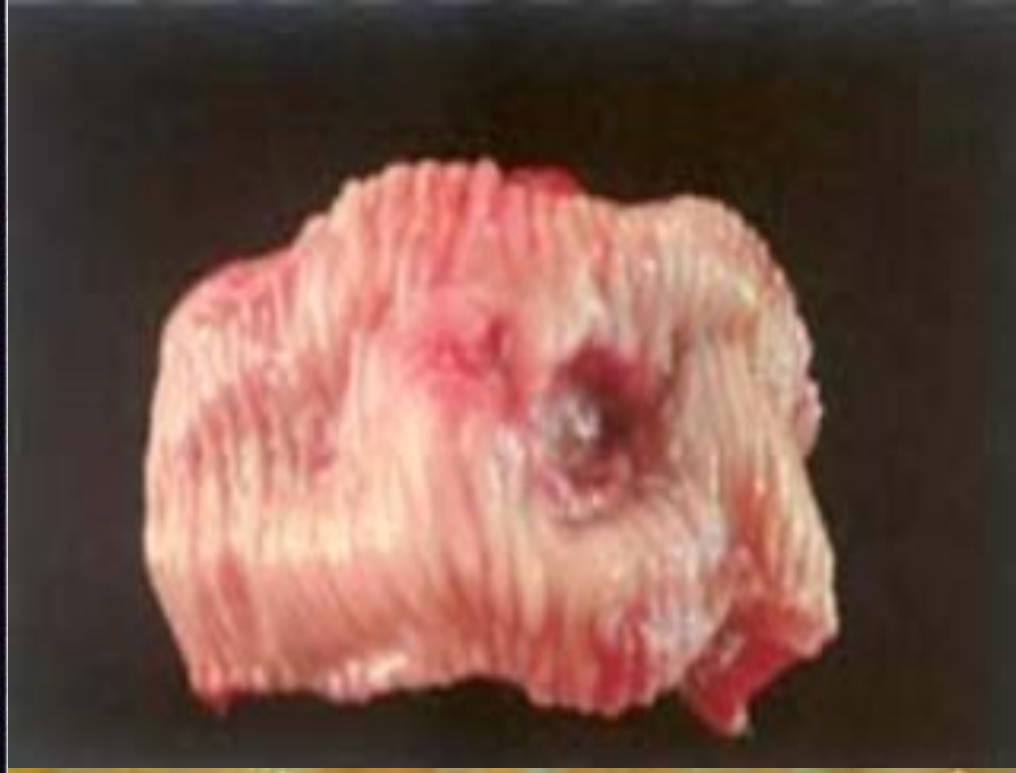
Characteristics:

- “PEPTIC” implies acid cause/aggravation
- ULCER vs. EROSION (muscularis mucosa intact)
- MUC→SUBMUC→MUSCULARIS→SEROSA
- Chronic, solitary (usually), adults.

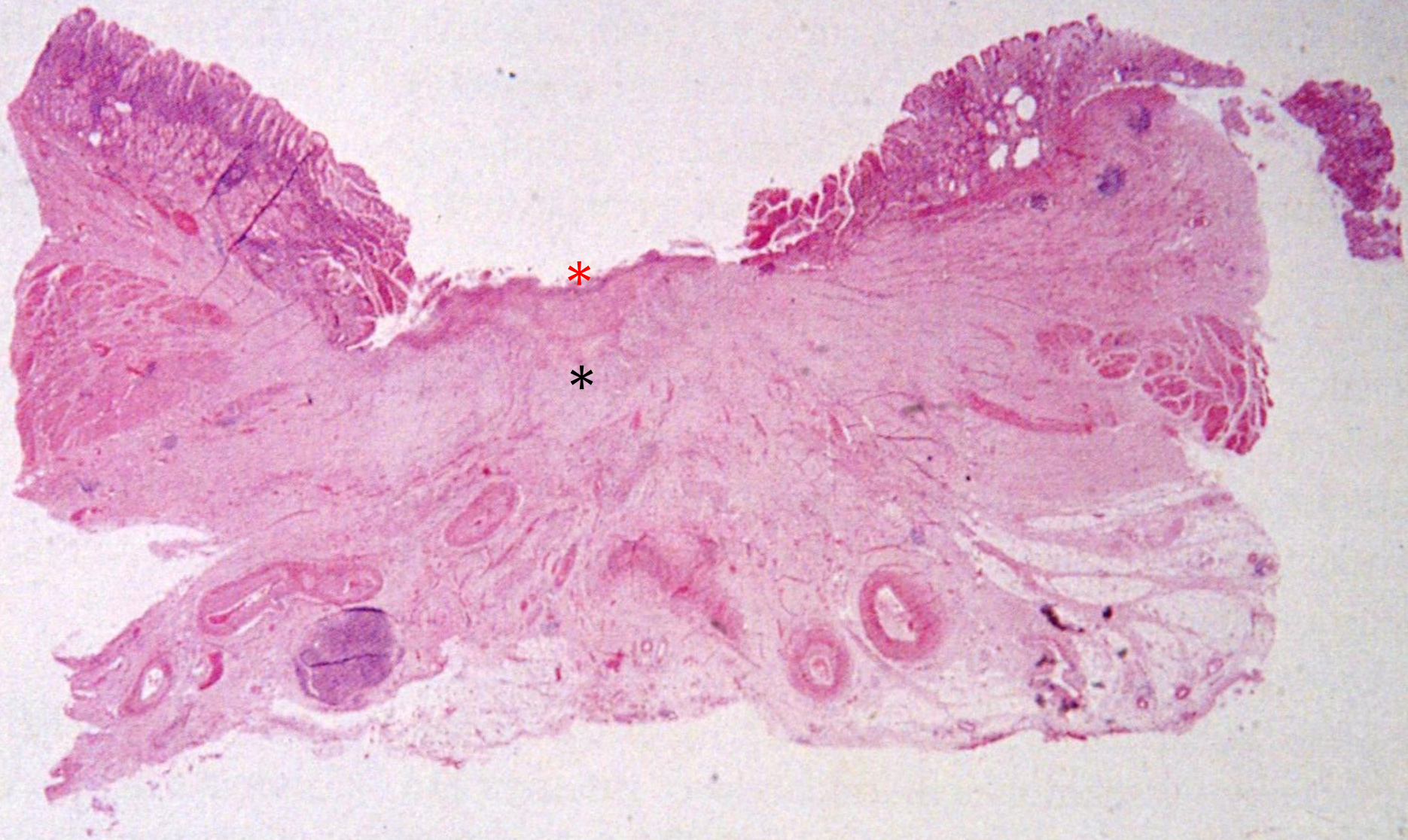
Causes:

- 80% caused by H. pylori in gastric
- 100% caused by H. pylori in duodenum
- NSAIDS
- STRESS





Ulcerted areas of the mucosa



Microscopic sections show:

1. Necrotic tissue (ulceration) in the superficial layer " mucosa" *
2. acute inflammatory exudates *
3. vascular granulation tissue *
4. fibrosis *

Complication:

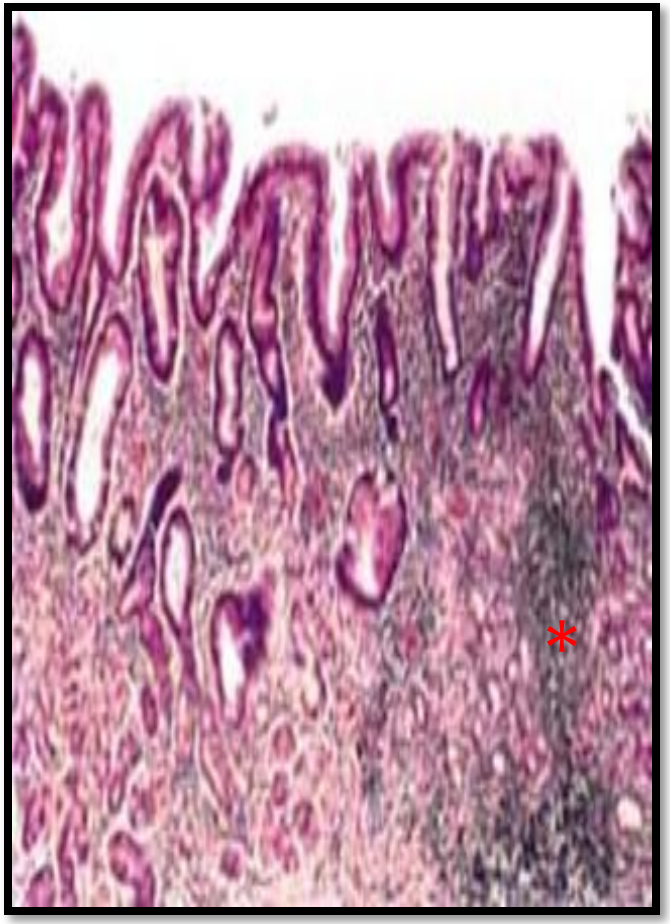
Perforation , hemorrhage , pyloric obstruction and penetration

Case 6

Gastritis

Causes:

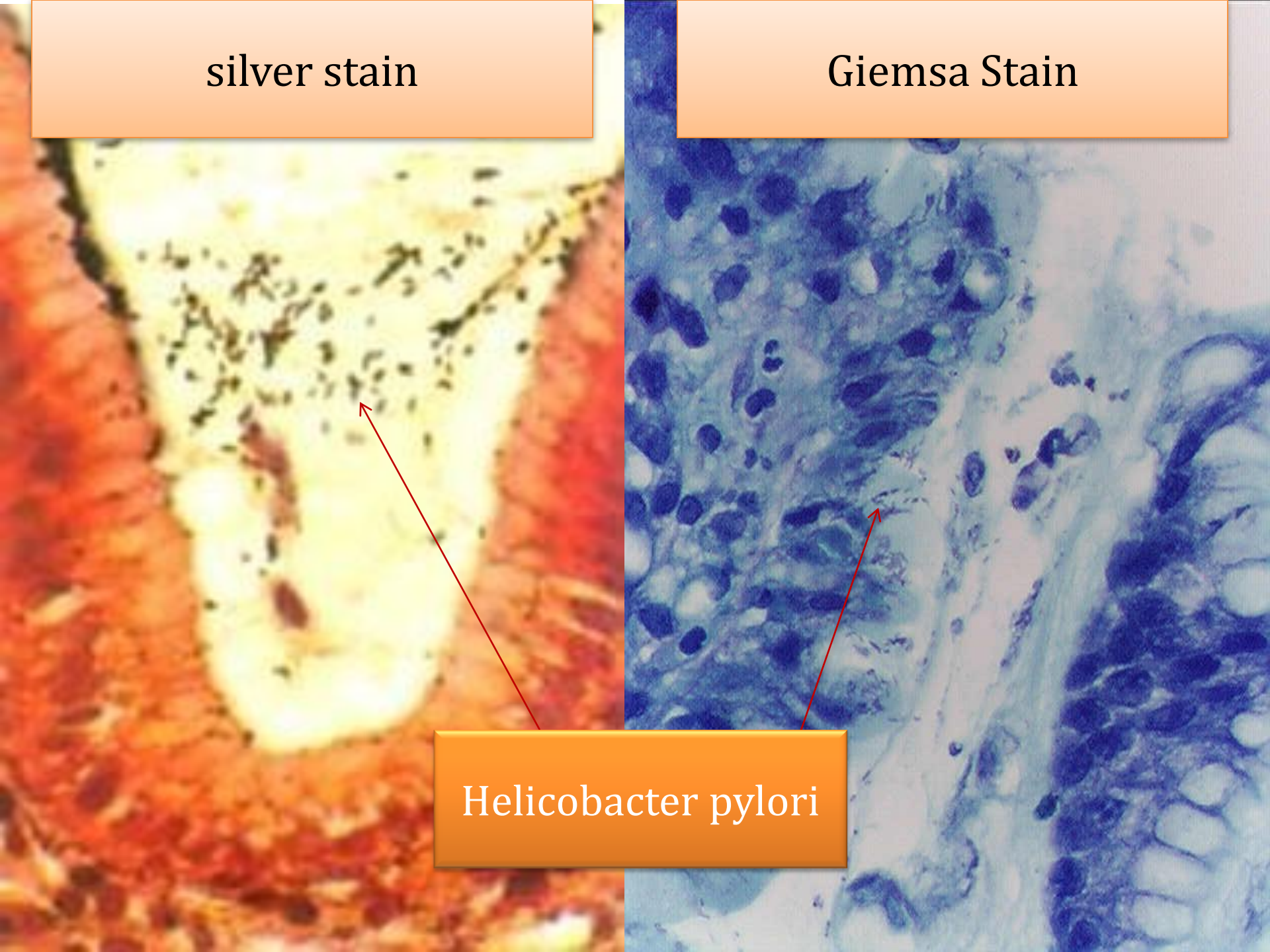
Most commonly H.pylori



Characteristics:

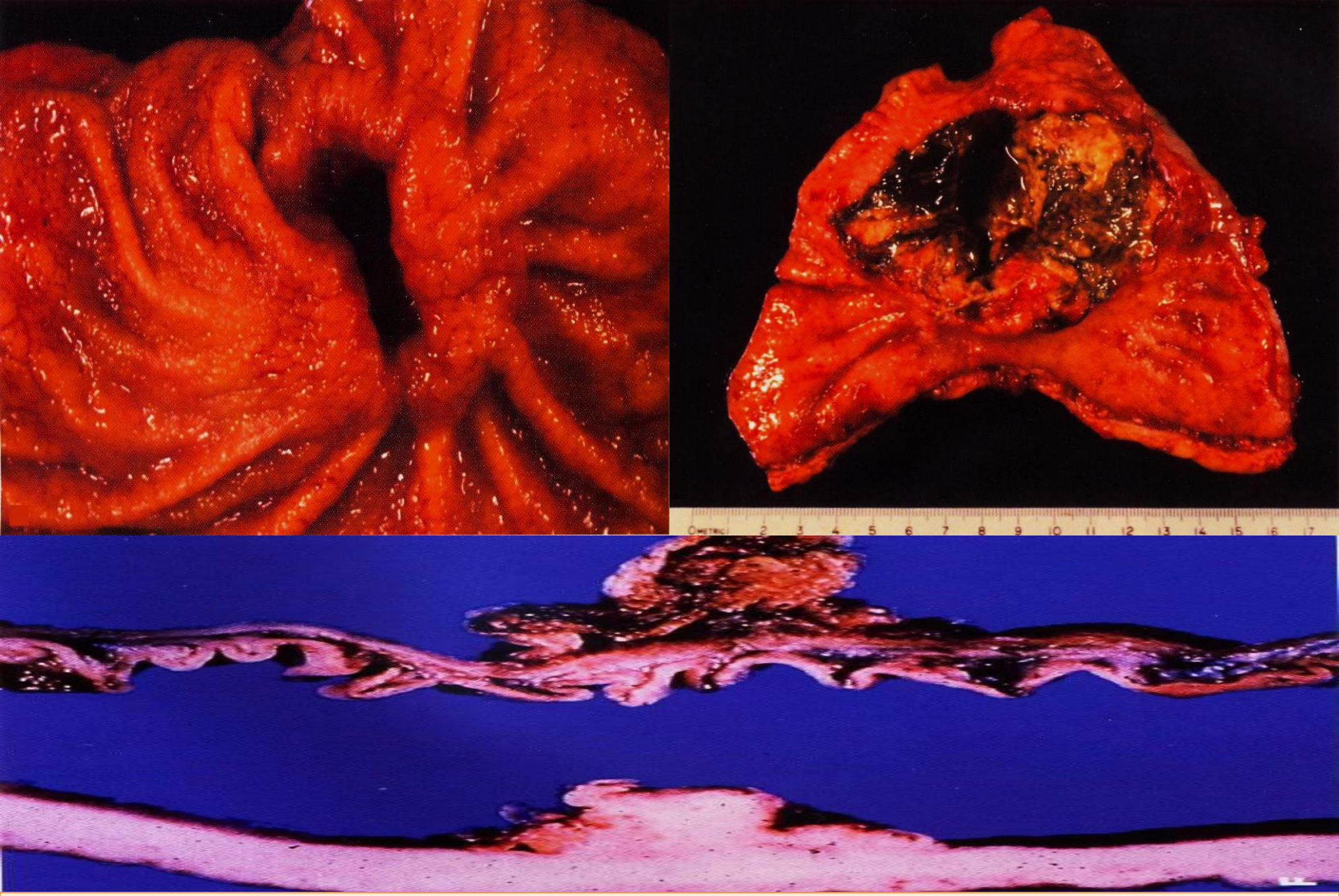
Chronic, no erosions, no hemorrhage

- Perhaps some neutrophils
- Lymphocytes, lymphoid follicles *
- Regenerative changes (not seen):
  - Metaplasia, intestinal
  - Atrophy, mucosal hypoplasia, “thinning”
  - Dys-plasia


silver stain
Giemsa Stain
Helicobacter pylori

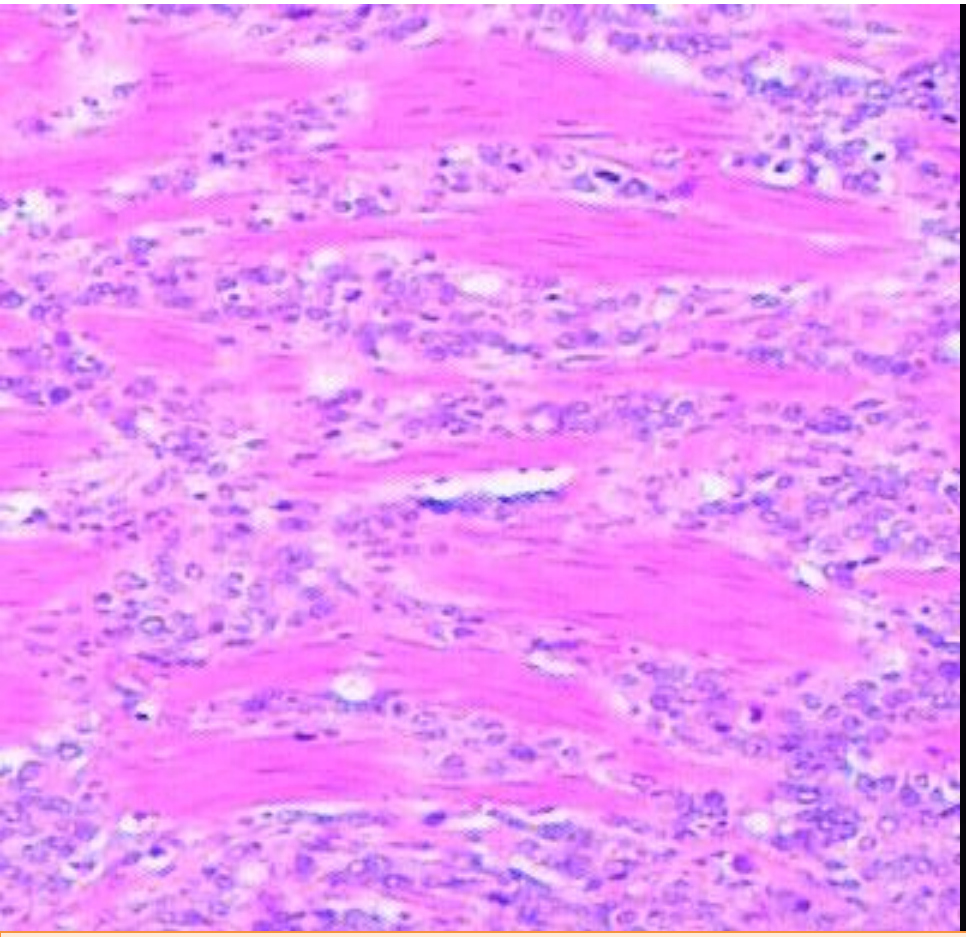
Case 7

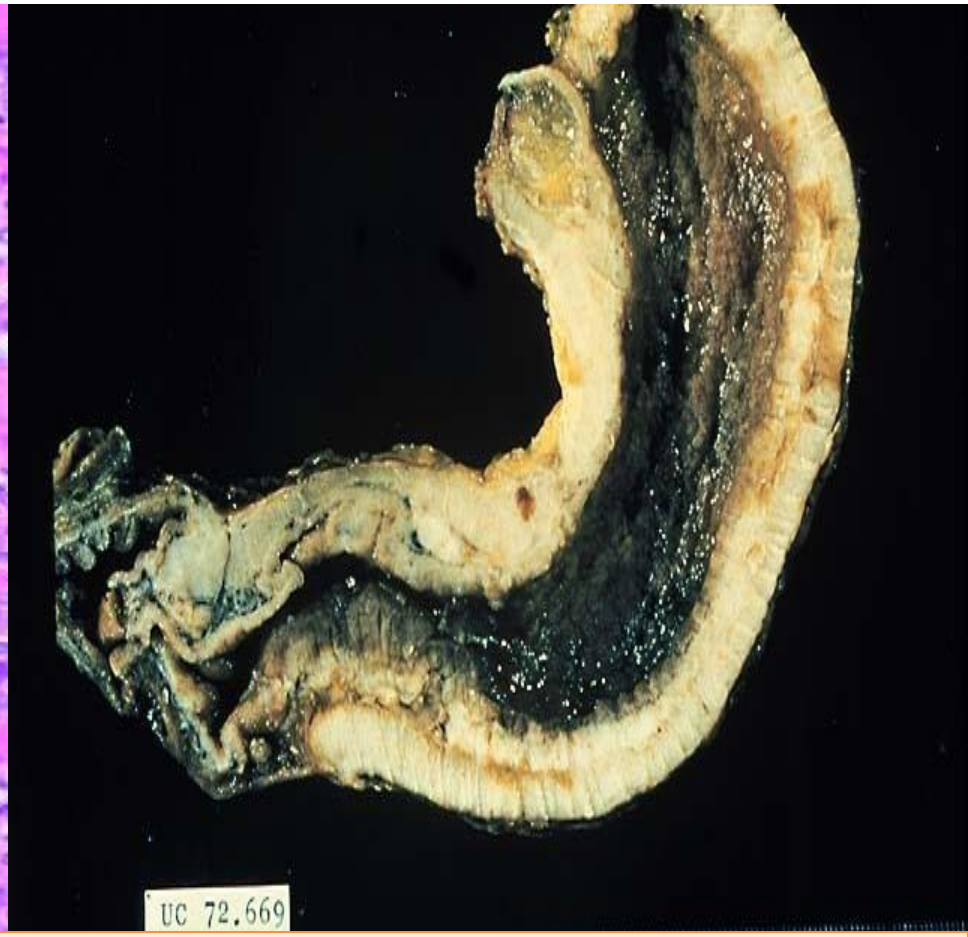
Carcinoma of the stomach



a) Eroding ulcer B) fungating mass c) diffuse infiltration (linitcs plastica)

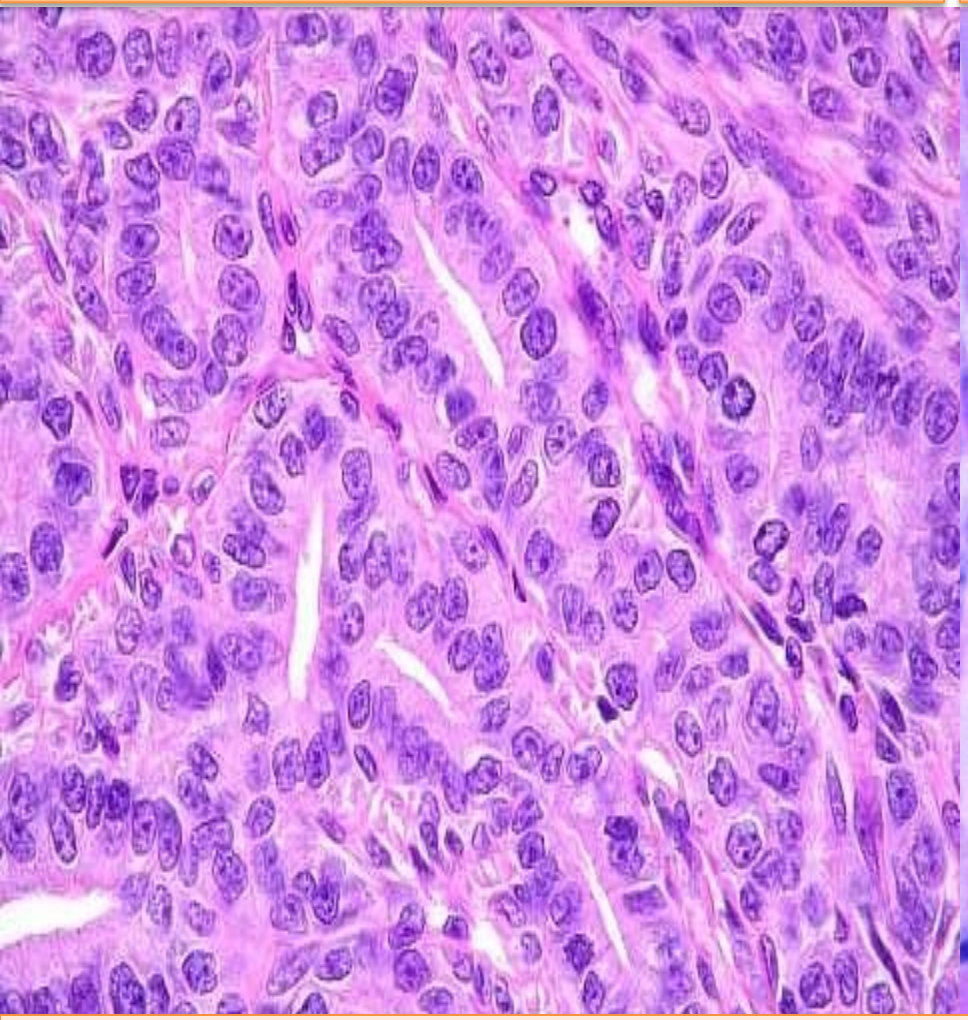
ADENOCARCINOMA-GROWTH PATTERNS



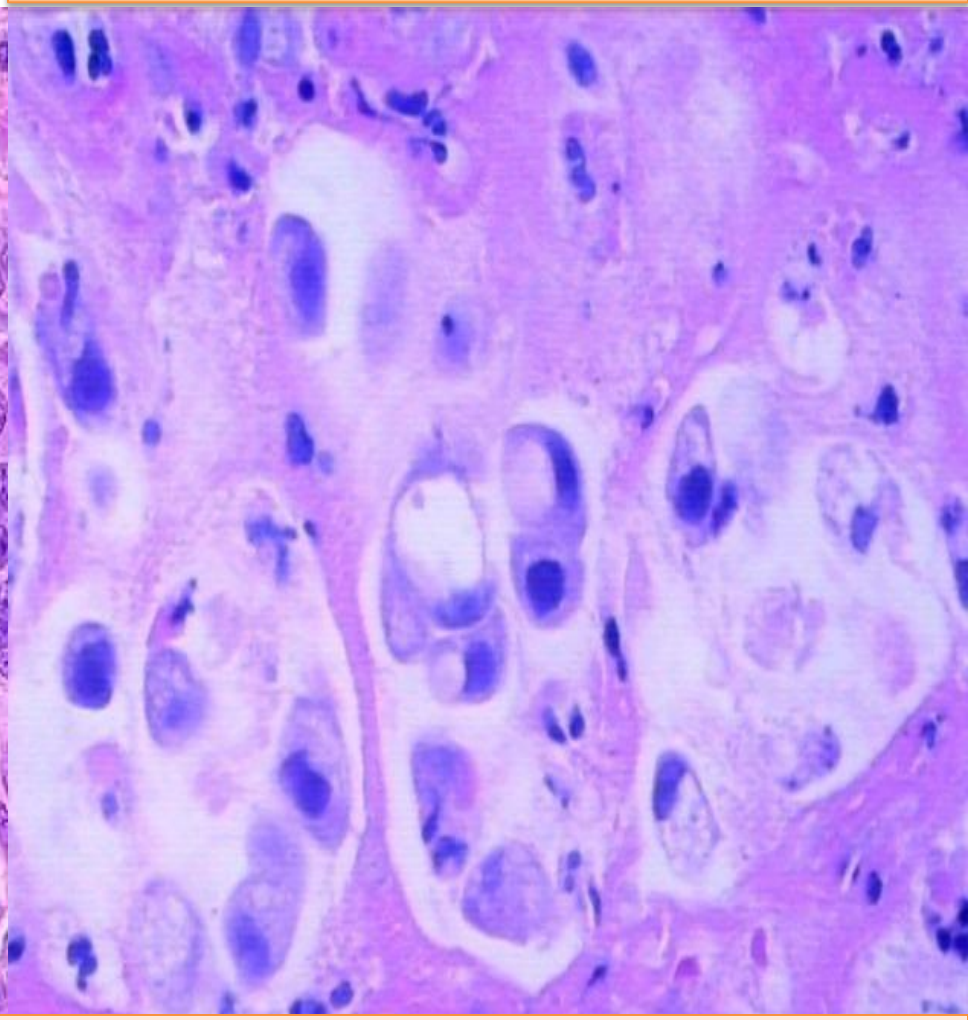


The LINITIS PLASTICA is the most SPECTACULAR, and most FEARED, of all gastric adenocarcinomas. It grows DIFFUSELY through all layers of the stomach, greatly thickening its wall, and giving the stomach a classic LEATHER BOTTLE appearance. It has a horrible prognosis.

Gastric adenocarcinoma - intestinal type



Gastric adenocarcinoma -- diffuse signet ring cell type

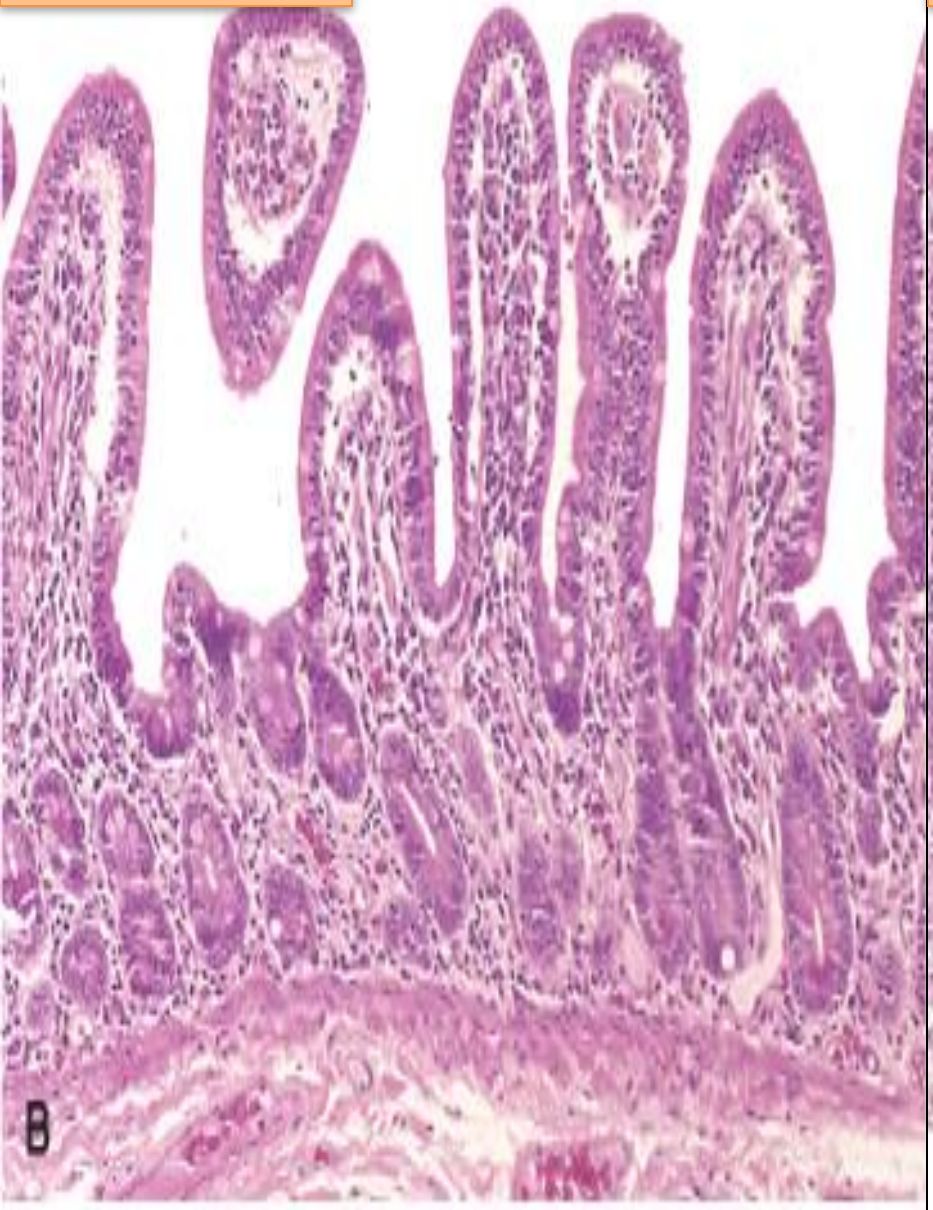


Signet ring cells are POORLY differentiated adenocarcinoma cells, and are OFTEN seen with linitis plastica. Could those large “holes” in the cytoplasm possibly be mucicarmine positive” Answer: YES

Case 8

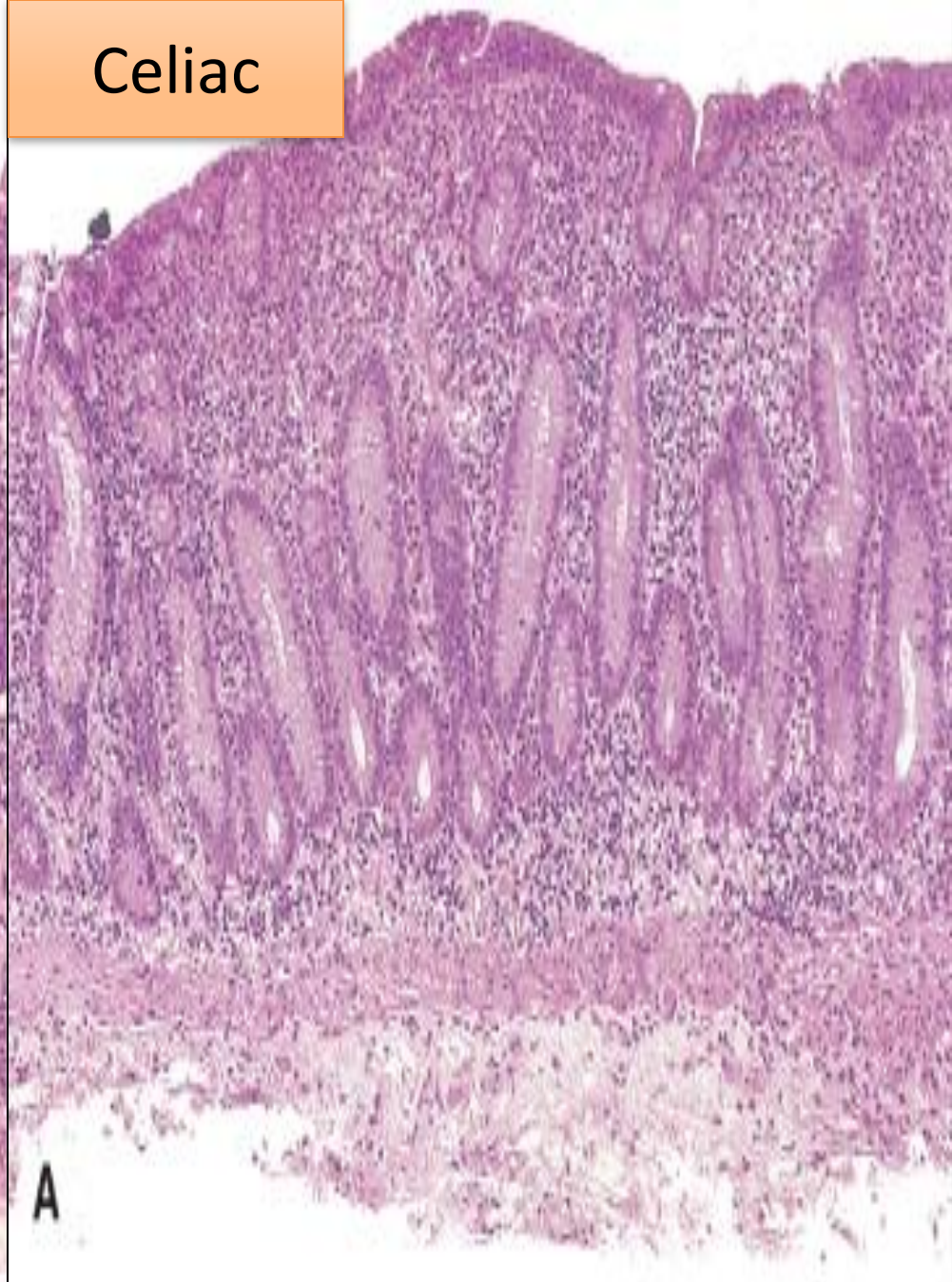
Celiac disease

Normal



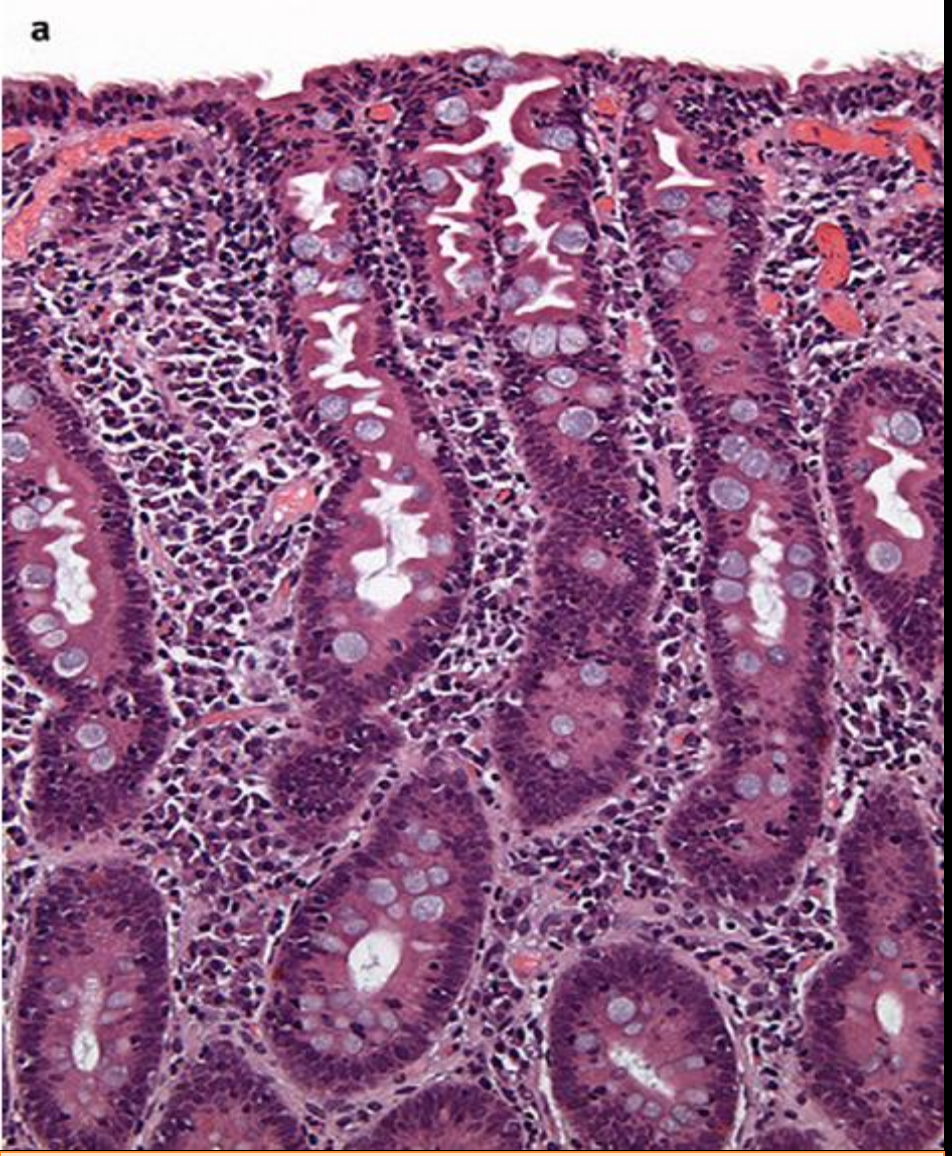
Villous length to crypt length 3/1

Celiac

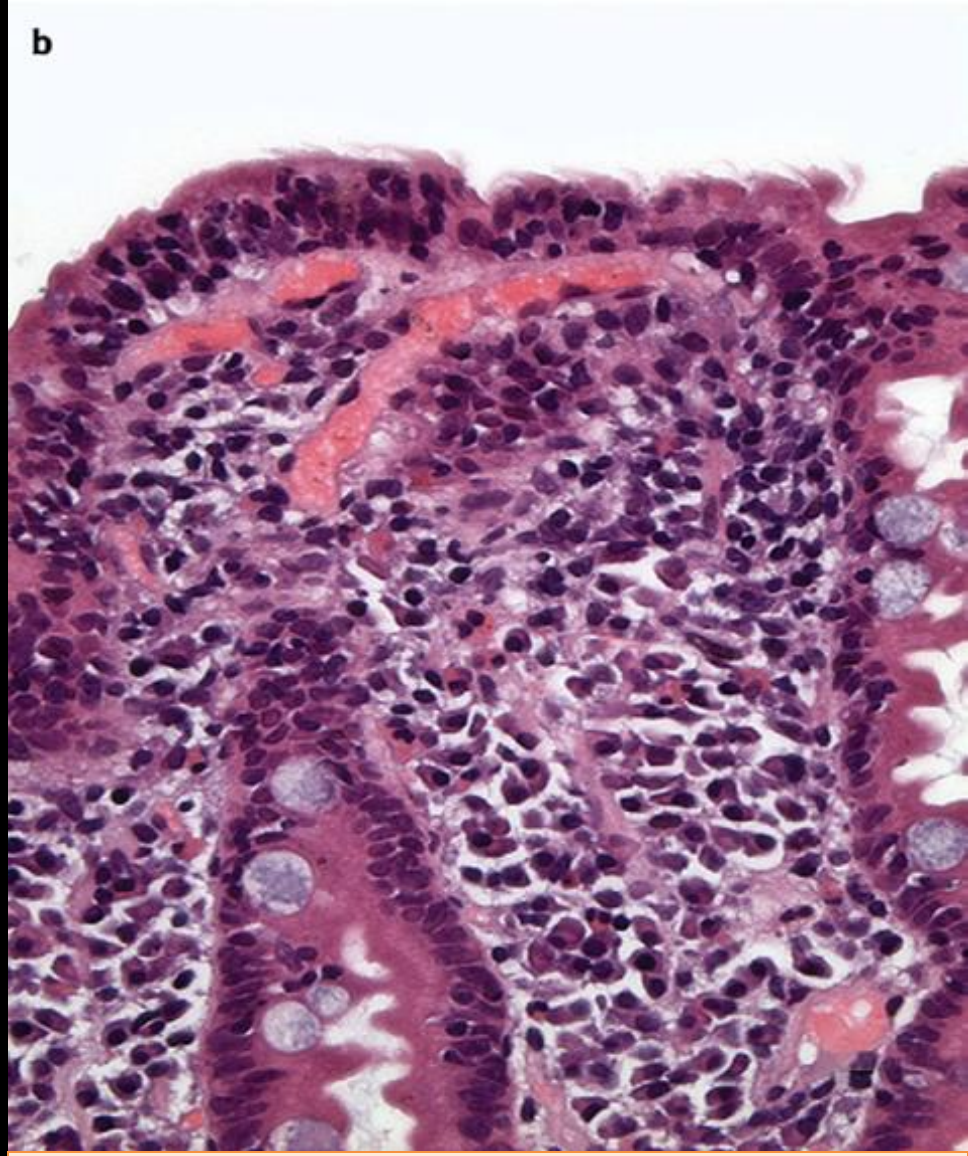


Histopathological findings:

- Villous atrophy
- Intraepithelial lymphocytes (IEL)
- Crypt Hyperplasia
- Plasma cells in the lamina propria



A Low-power view of fully developed sprue-type changes. Note the elongated crypts with complete lack of villi.



B High-power view showing damaged surface epithelium with large numbers of intraepithelial lymphocytes.

Case 9

Carcinoid Tumor of Small Intestine

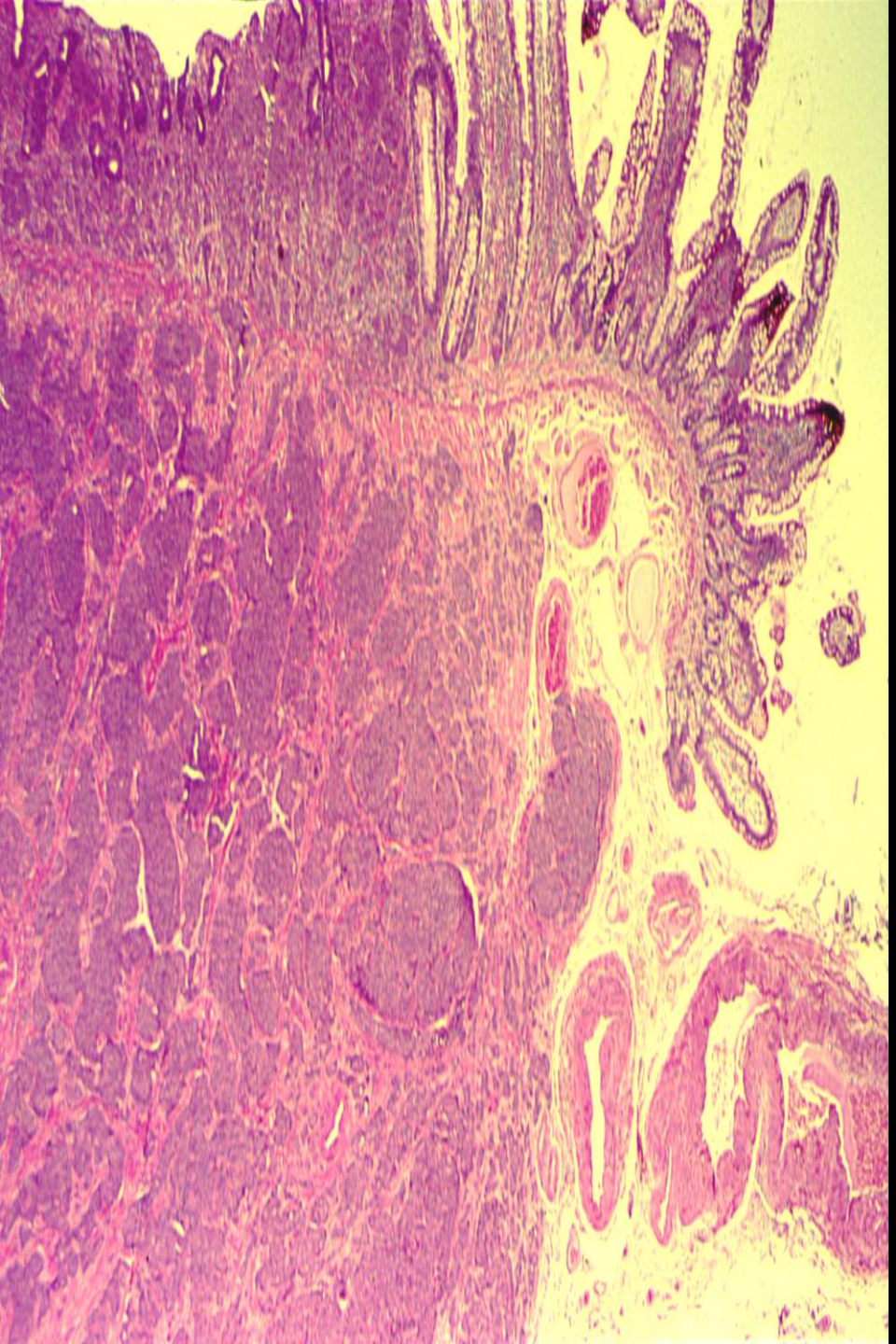
originate from:

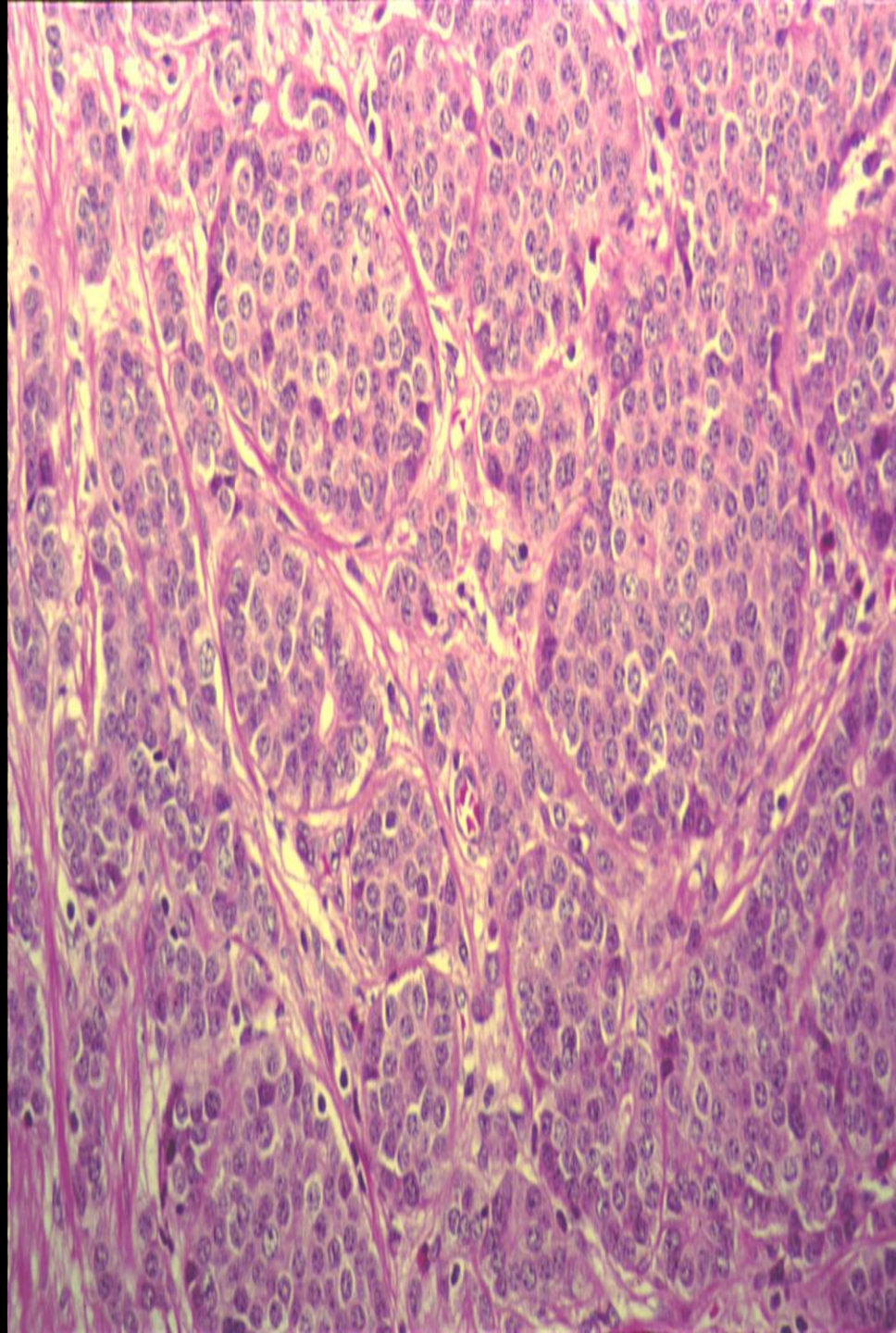
neuroendocrine cells found in lung as well as the GI

Clinical presentation:

might be presented by carcinoid syndrome where 5HT is not degraded in the liver duo to liver metastasis causing the serotonin to be found in the blood circulation

Remember that 5Ht is a vasodilator, so patients will be clinically presented by abdominal pain , cramps , flushing , diarrhea and heart valve lesion .





Section of small intestine shows:

- surface ulceration * and an infiltrating tumor mass in mucosa and submucosa *
- Tumor consists of alveolar groups and clumps of small monomorphic (uniform) polygonal cells *
- The cells have centrally placed round nuclei with salt and pepper like nuclear chromatin and esinophilic abundant granular cytoplasm.

Case: 10

IBD: Crohn's disease

IBD: are idiopathic diseases

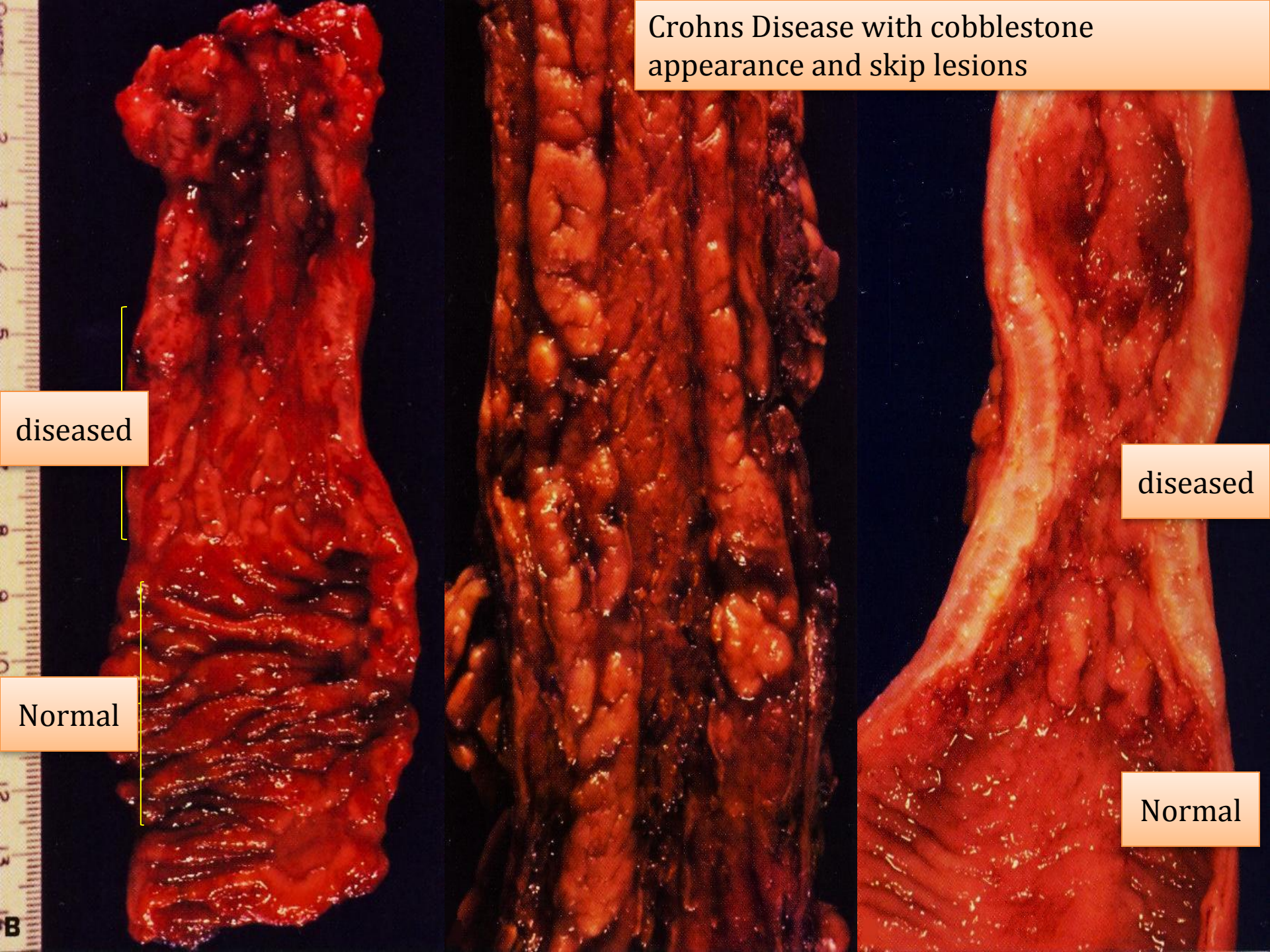
Site affected: ileum.

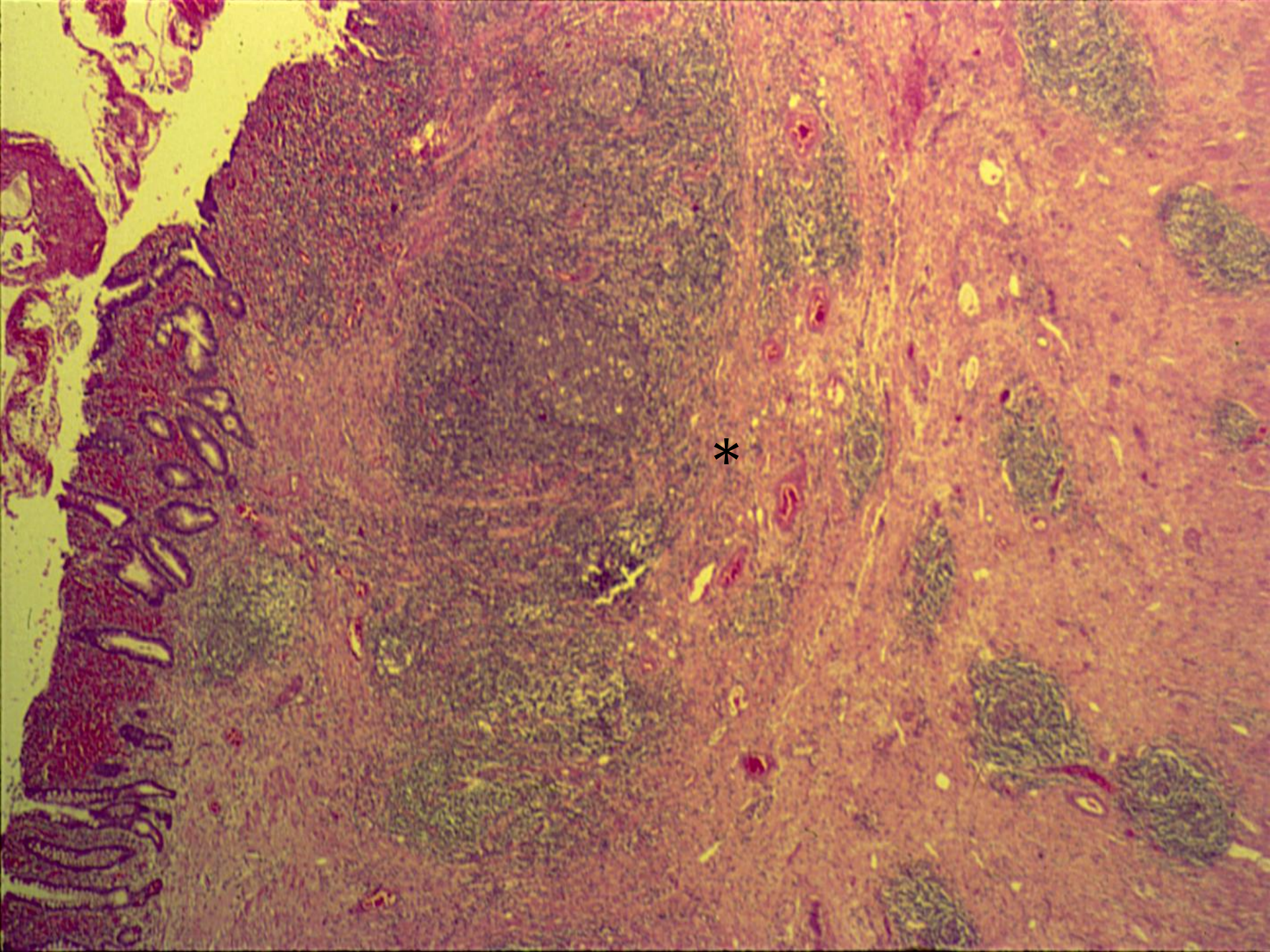
Characteristics:

- skip lesion
- Coublestoning (because it is not continues)

Complications:

obstruction , malabsorption , anal fistula (deep ulcer), fissure (continuation with another organ), and sinuses and low incidence of malignant transformation .


Crohns Disease with cobblestone appearance and skip lesions
diseased
Normal
diseased
Normal



Section of small intestine shows:

- alternating normal and ulcerating mucosa (grossly seen):
- All layers of intestinal wall show transmural chronic inflammatory cell infiltrate, lymphoid aggregates and mild fibrosis (*).
- Subserosa contains few epithelioid granulomas.

Case 11

IBD: Ulcerative colitis

Cause:

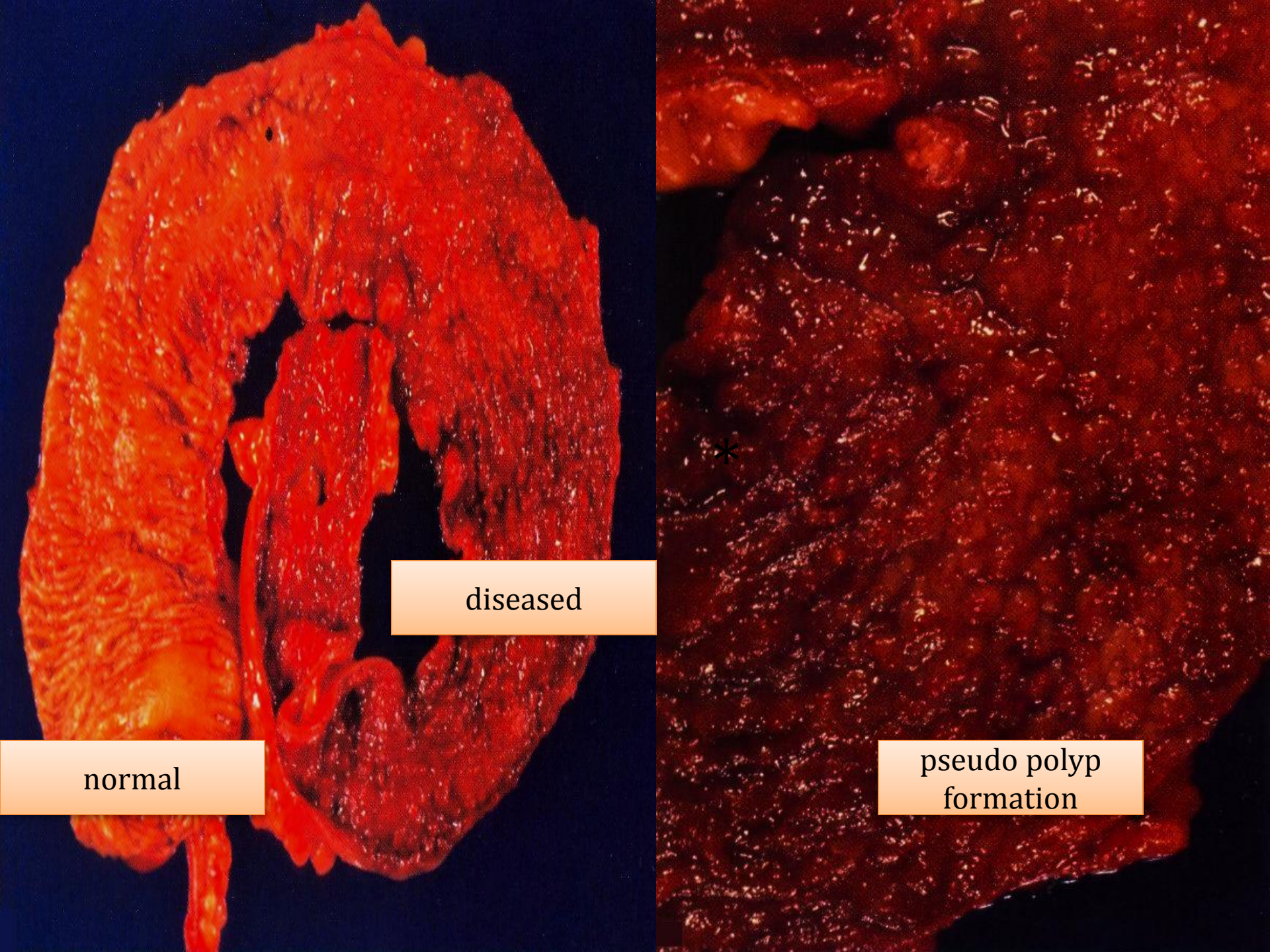
Idiopathic

Site:

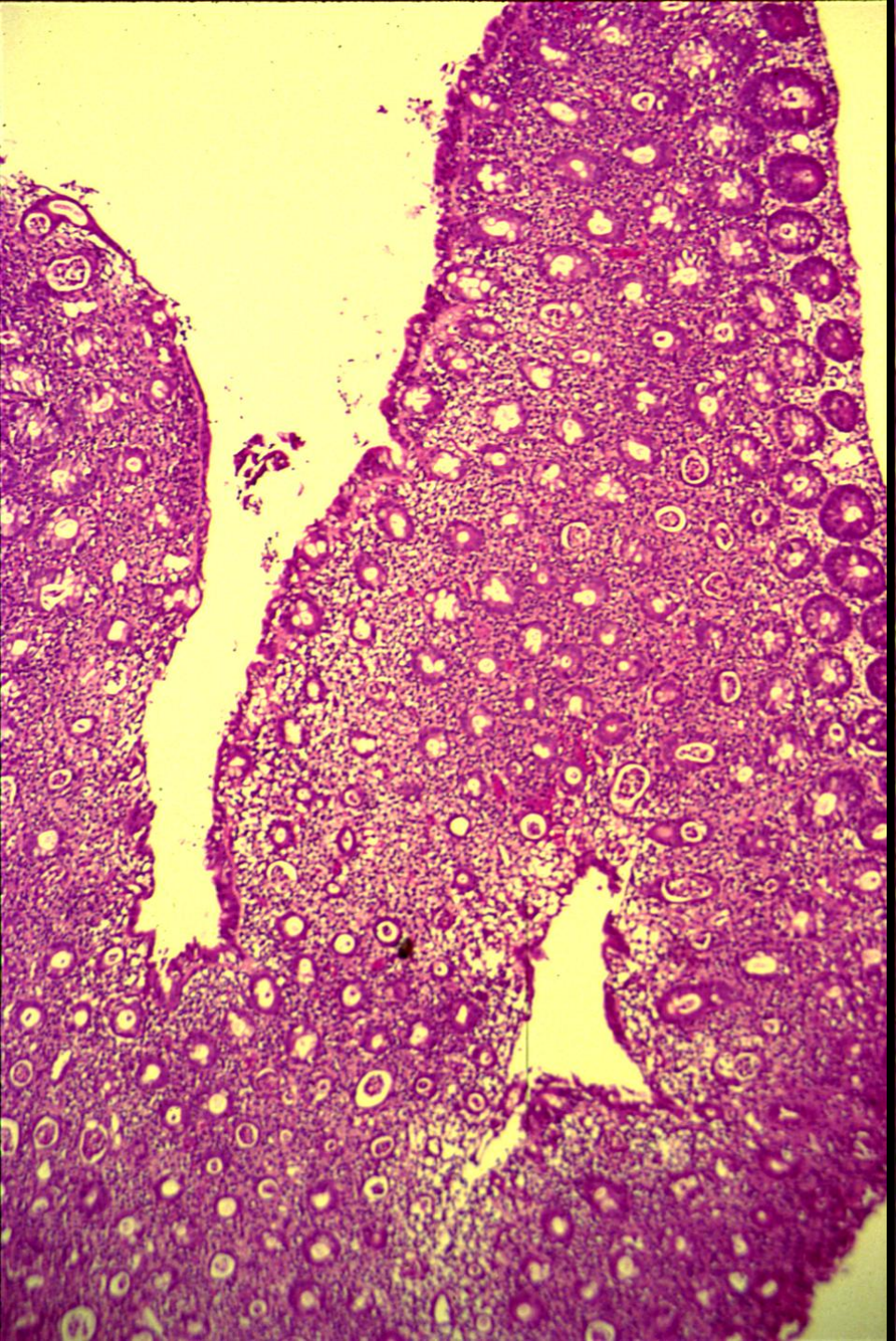
Rectum and above

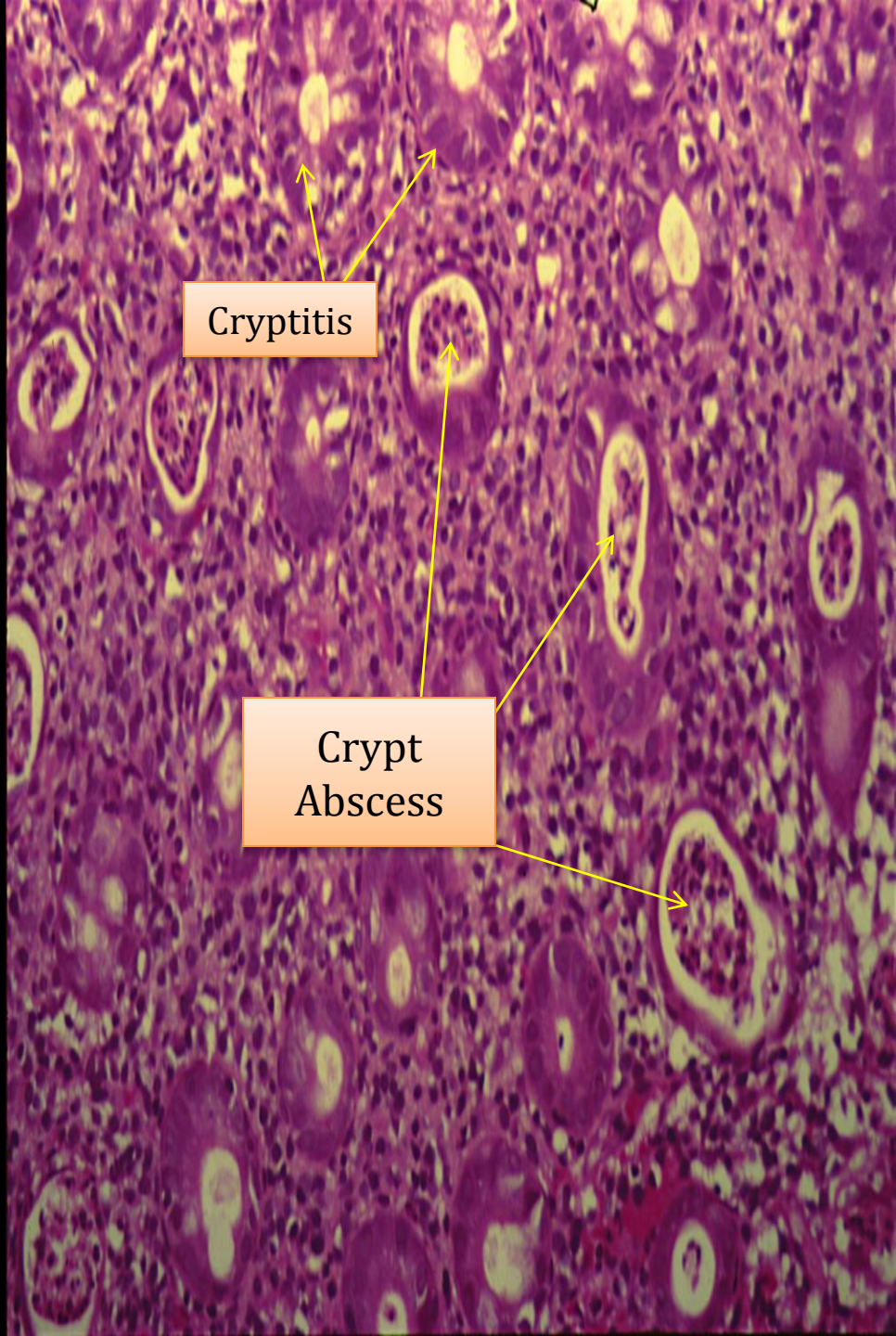
Characteristics:

- Continues lesion
- No granulomas
- Ulceration, crypt abscess (neutrphils in the lumen), cryptitis (inflammatory cells on the epithelium).


diseased
normal
*
pseudo polyp formation


pseudo polyp
formation




Cryptitis
Crypt
Abscess

Section of large bowel wall show:

- few relatively superficial ulcers lined by acute inflammatory exudate. Marked oedema and vascular congestion are seen in lamina propria
- The mucosa adjacent to the ulcers contains several crypt abscesses and there is evidence of goblet cells depletion in many glands.
- No granulomas or glandular dysplasia are noted.

Case 12

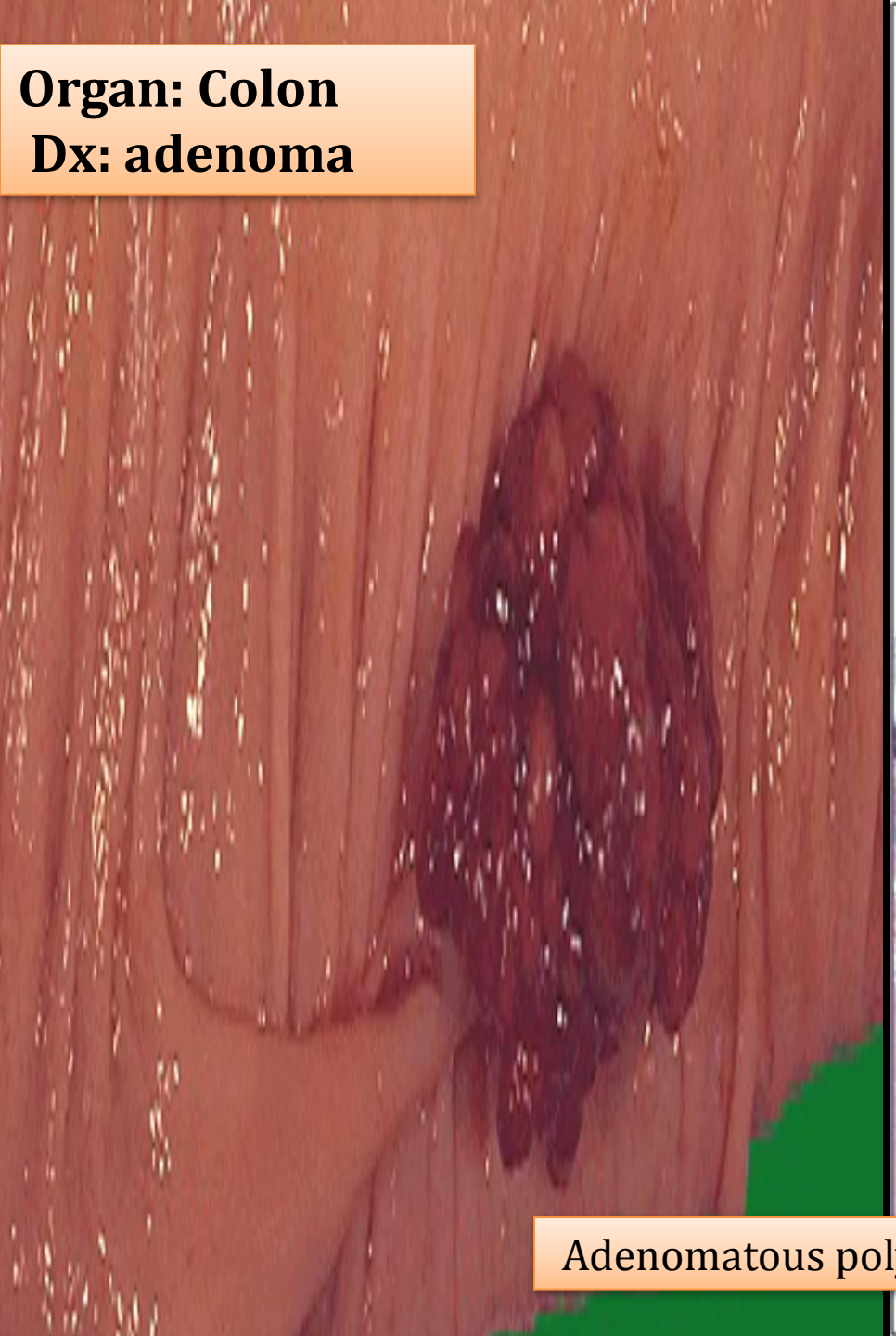
Adenomatous Polyps of Large Intestine

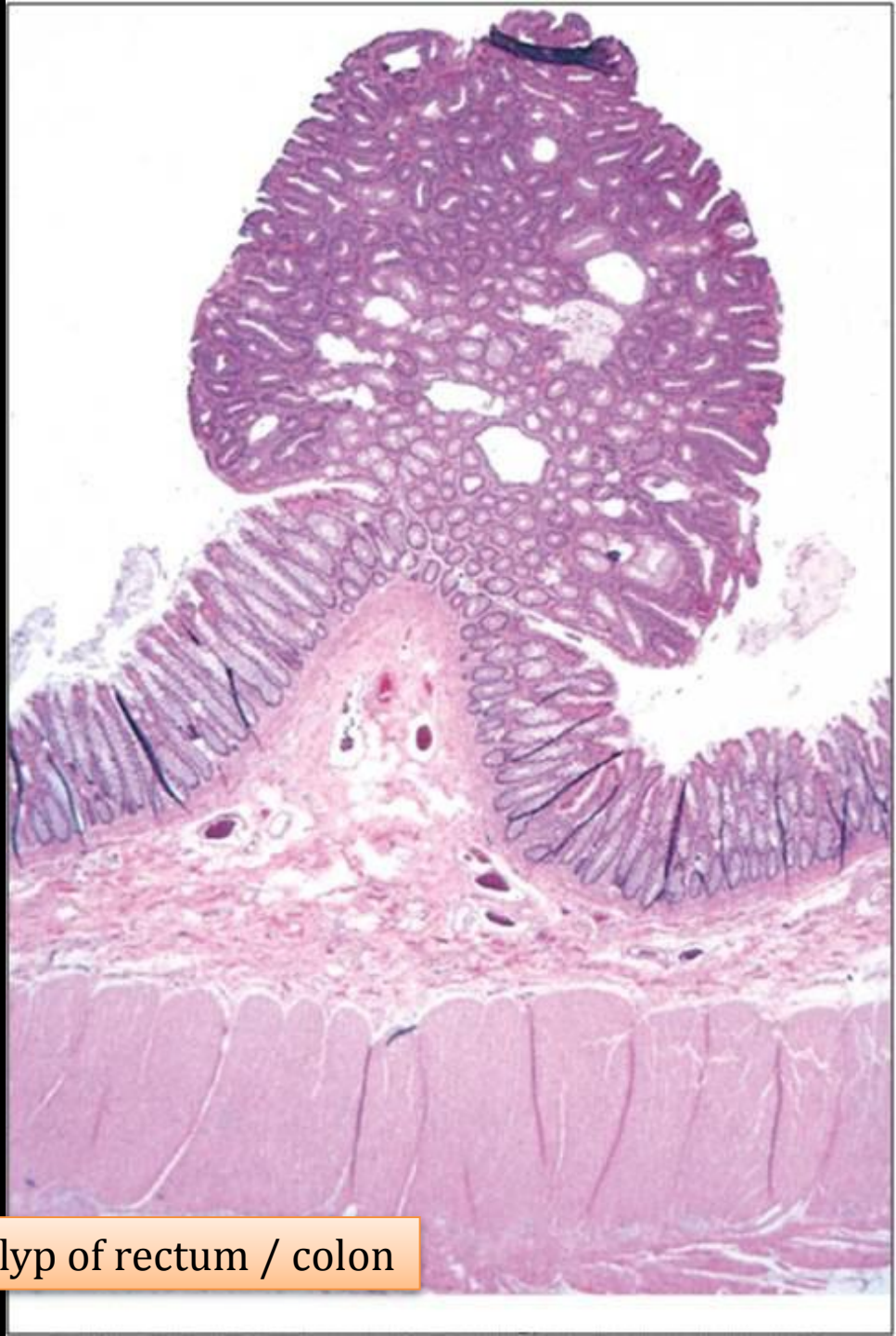
Types of adenoma:

1. Tubular adenoma
2. Villous adenoma (more prone to develop cancer)
3. Tubulovillus adenoma
4. Lymphoid adenoma
5. hyperplastic adenoma
6. juvanile adenoma

Latter three don’t transform into cancer

Organ: Colon
Dx: adenoma



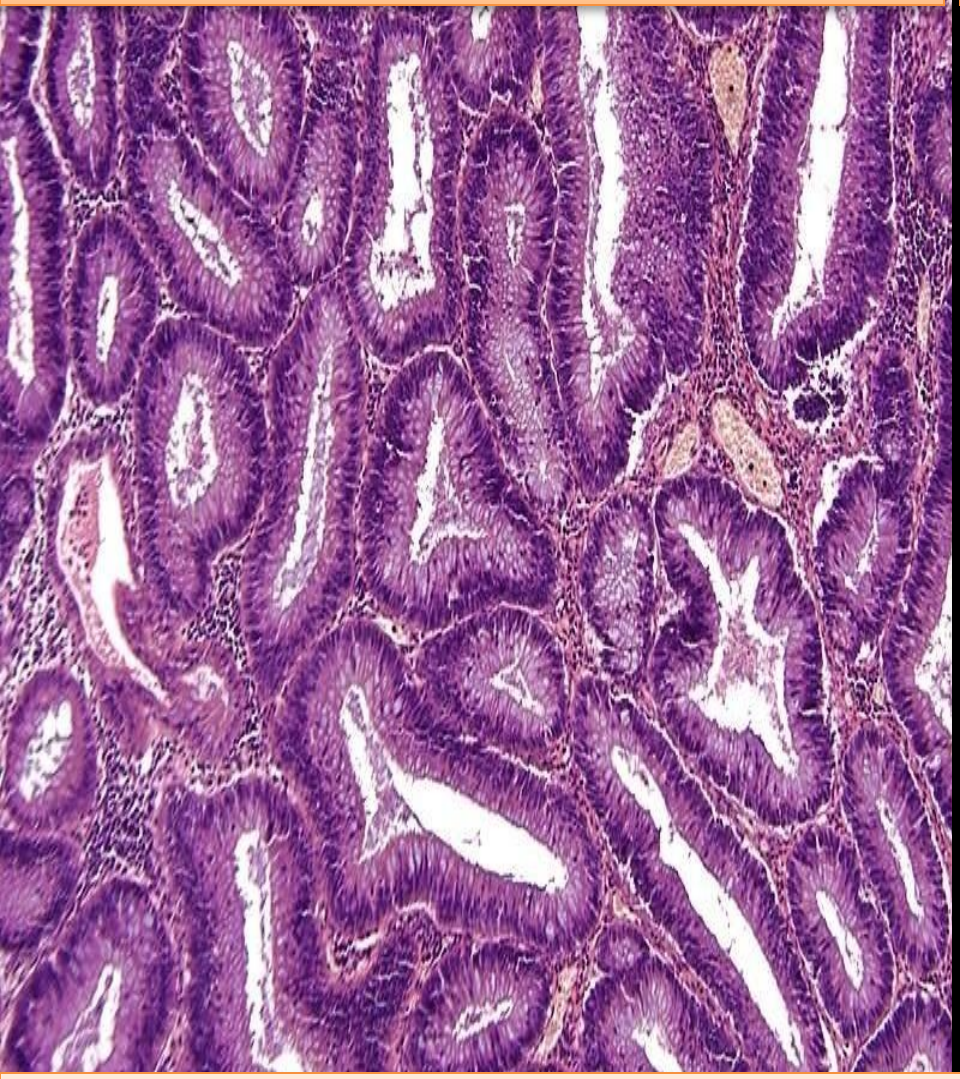


Adenomatous polyp of rectum / colon

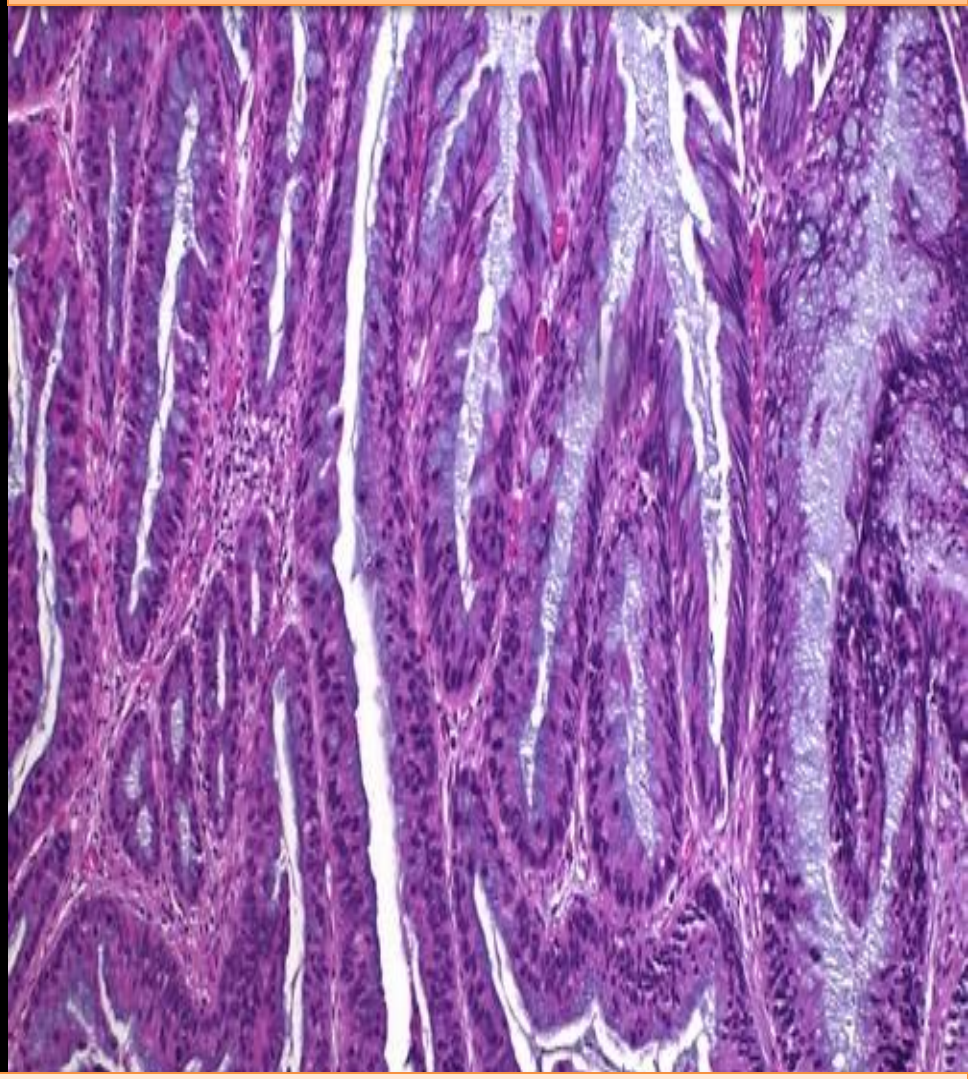
Microscopic sections show

- Pedunclated growth
- Dysplastic glands
- Increased number of glands
- Absence of stalk invasion (no infiltration) .

Adenomatous Polyp (Tubular)



Adenomatous Polyp (Villous)

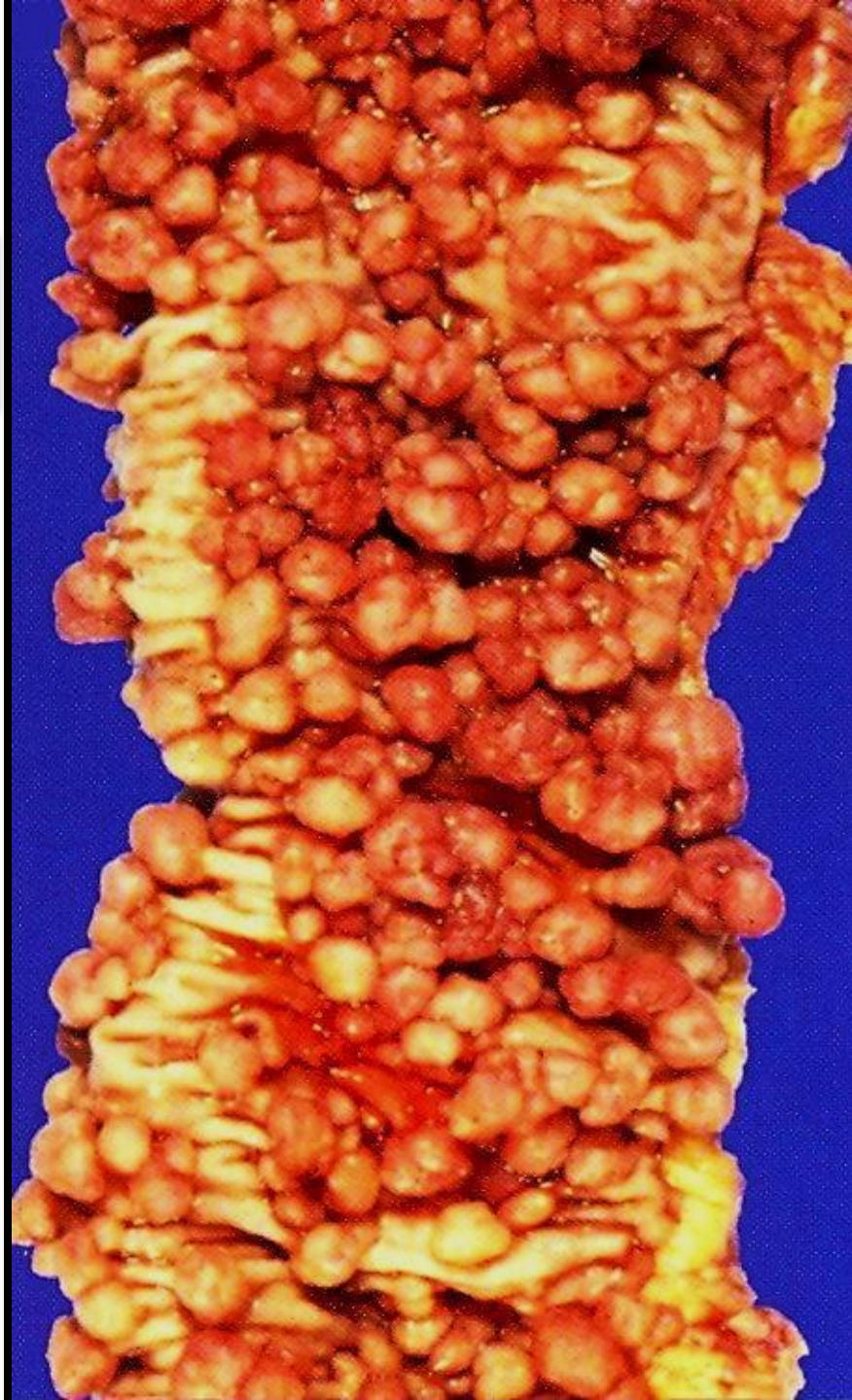


TUBULAR adenoma, note how all the epithelial (glandular) cells look the same.
Villous adenomas behave more aggressively than tubular adenomas. They have a HIGHER rate of developing into frank adenocarcinomas than the “tubular” patterns. Tubulovillous adenoma , Hyperplastic polyp ,Lymphoid polyp and Juvenile polyp are other types of large bowel polyps .

Familial Adenomatous Polyposis

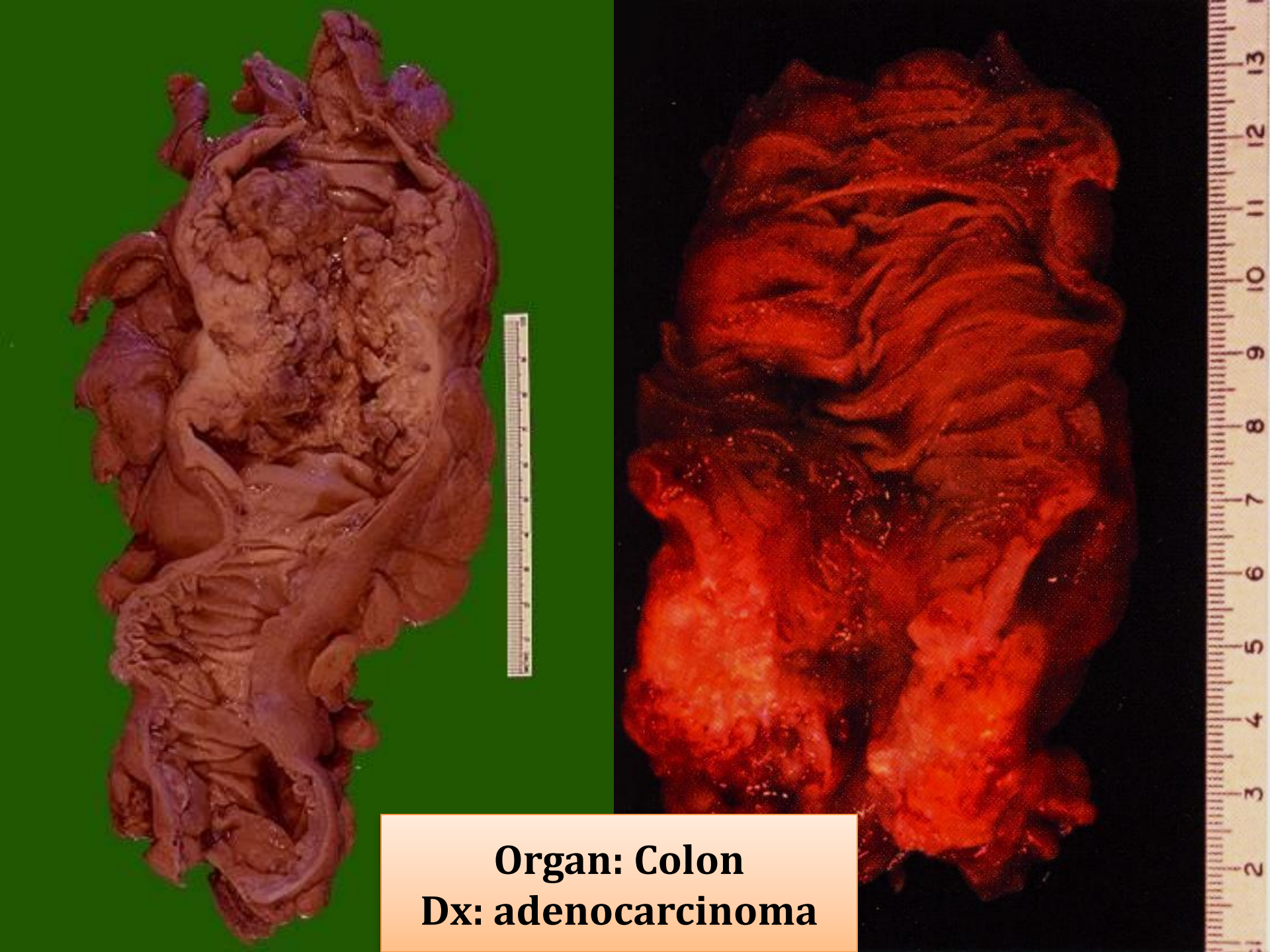
It is caused by mutation

of APC gene .

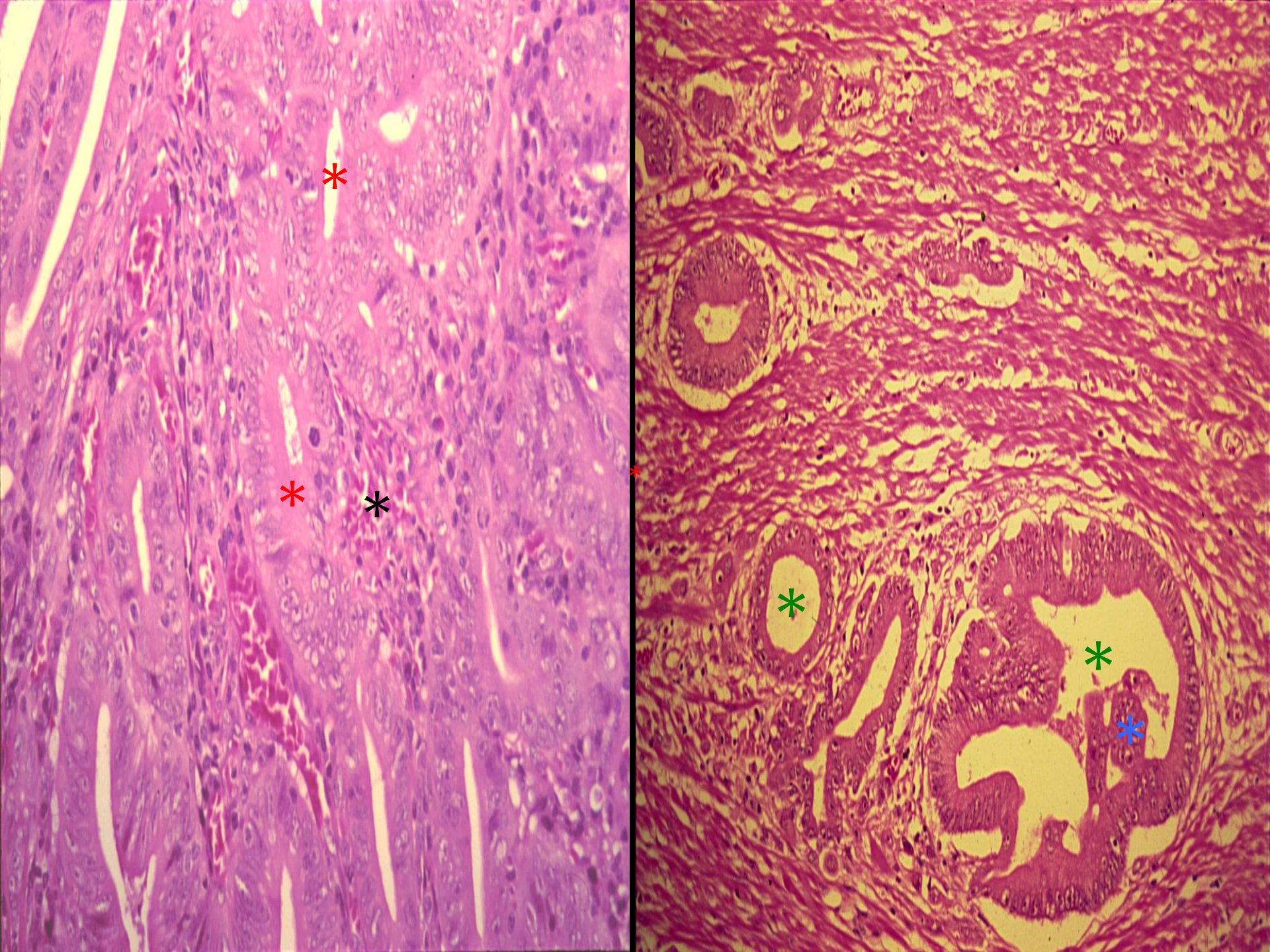


Case 13

Adenocarcinoma



Organ: Colon
Dx: adenocarcinoma

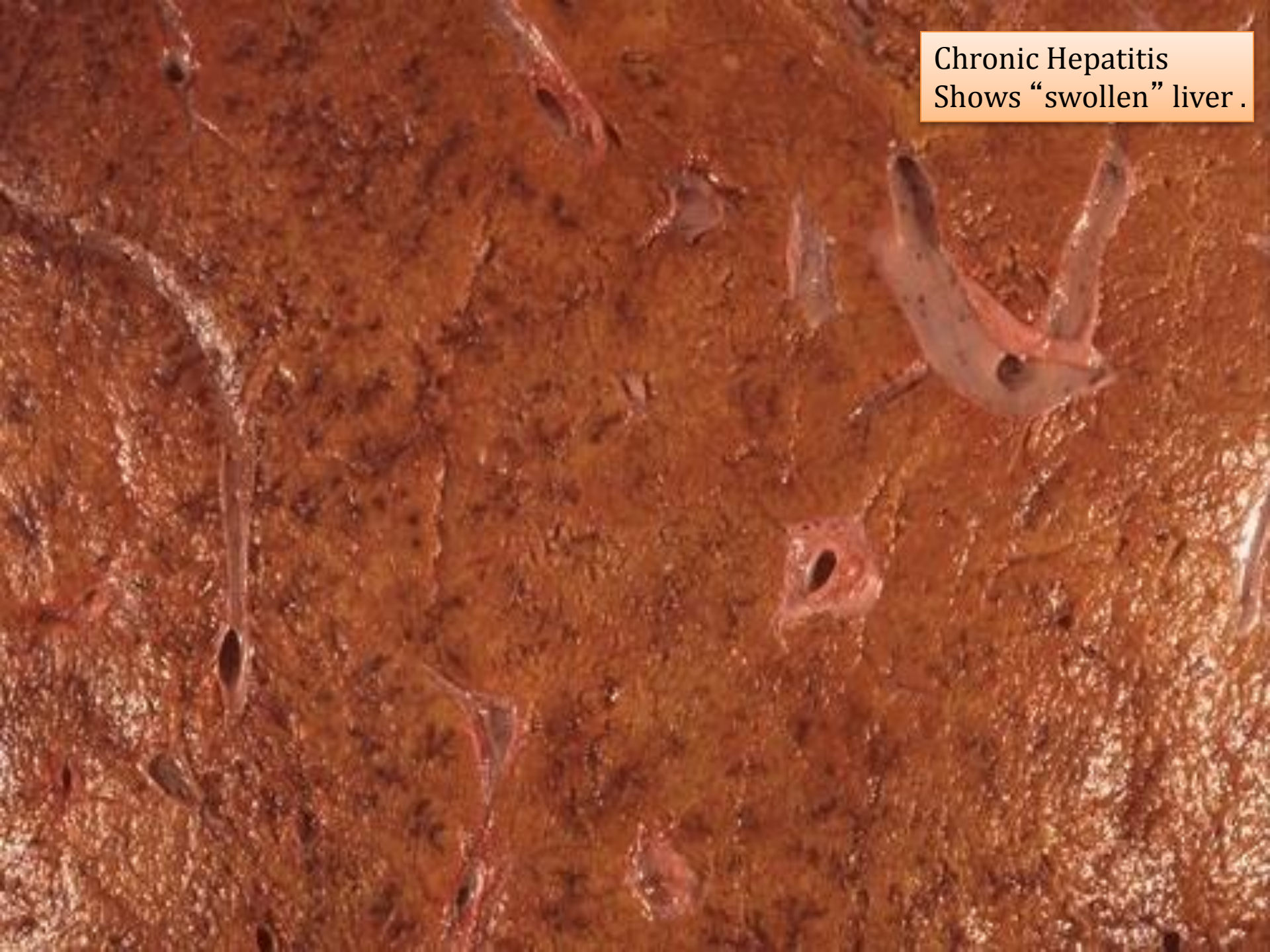


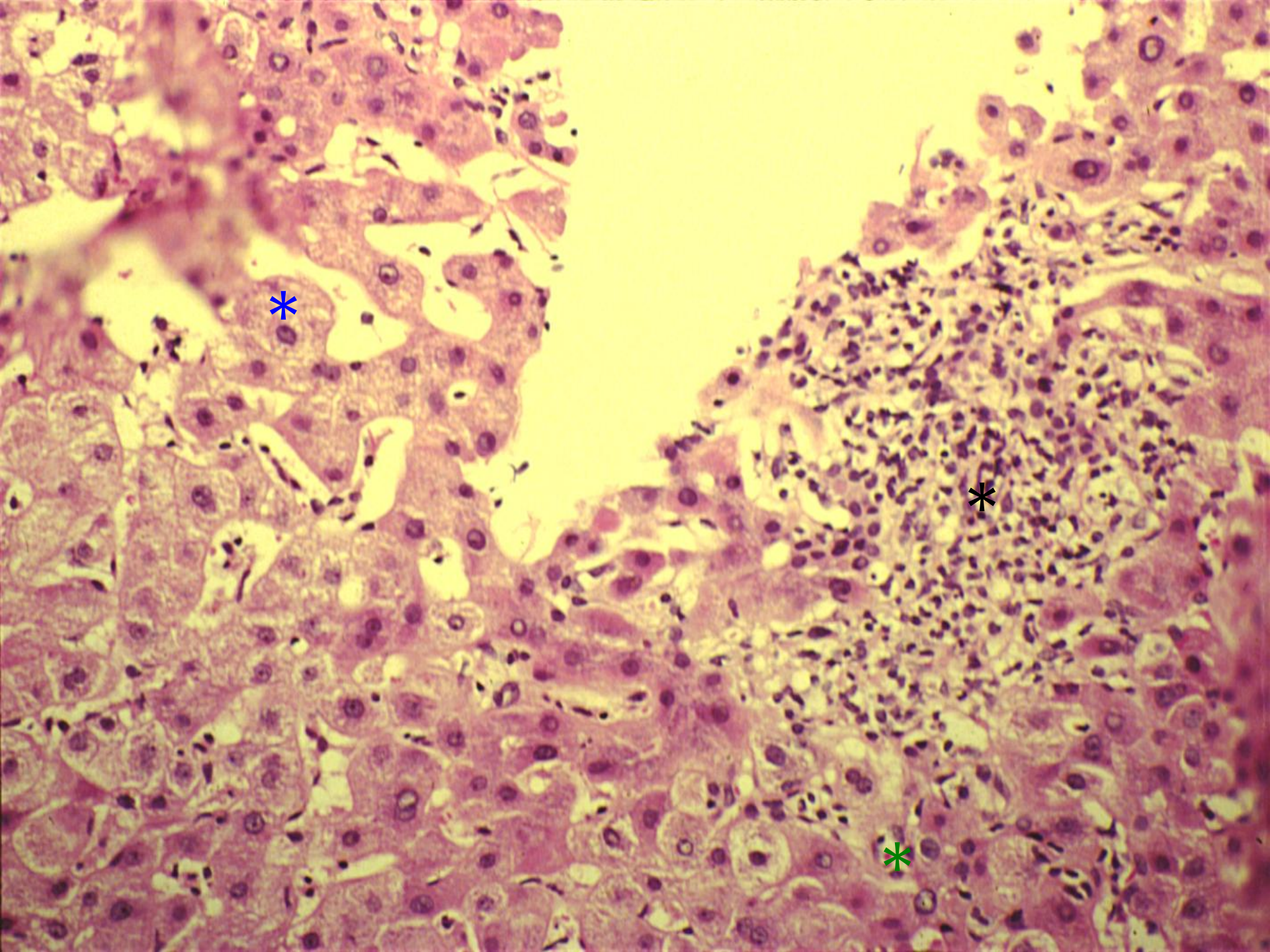
Section of large intestine shows:

- Tumour consists of crowded irregular malignant acini (*)separated by thin fibrovascular stroma. (*)
- The acini are lined by one or several layers of neoplastic cells with papillary projection (*) showing pleomorphism, hyperchromatism and few mitoses.
- Muscle coat is invaded by neoplastc glands. (*)

Case 14

Chronic hepatitis


Chronic Hepatitis
Shows “swollen” liver .



Section from this liver biopsy show:

- Moderate chronic inflammatory cells infiltration consisting of lymphocytes and histiocytes in both portal tracts and liver parenchyma. (*)
- Piecemeal necrosis (*) (necrosis around the portal triad), interface hepatitis (necrosis protruding to the hepatocytes), hepatocytes swelling and "spotty" hepatocyte necrosis are also noticed as well as ballooning degeneration (*).
- No evidence of cirrhosis or malignancy noted.

Complications:

Cirrhosis

Hepatocellular carcinoma and hepatic failure

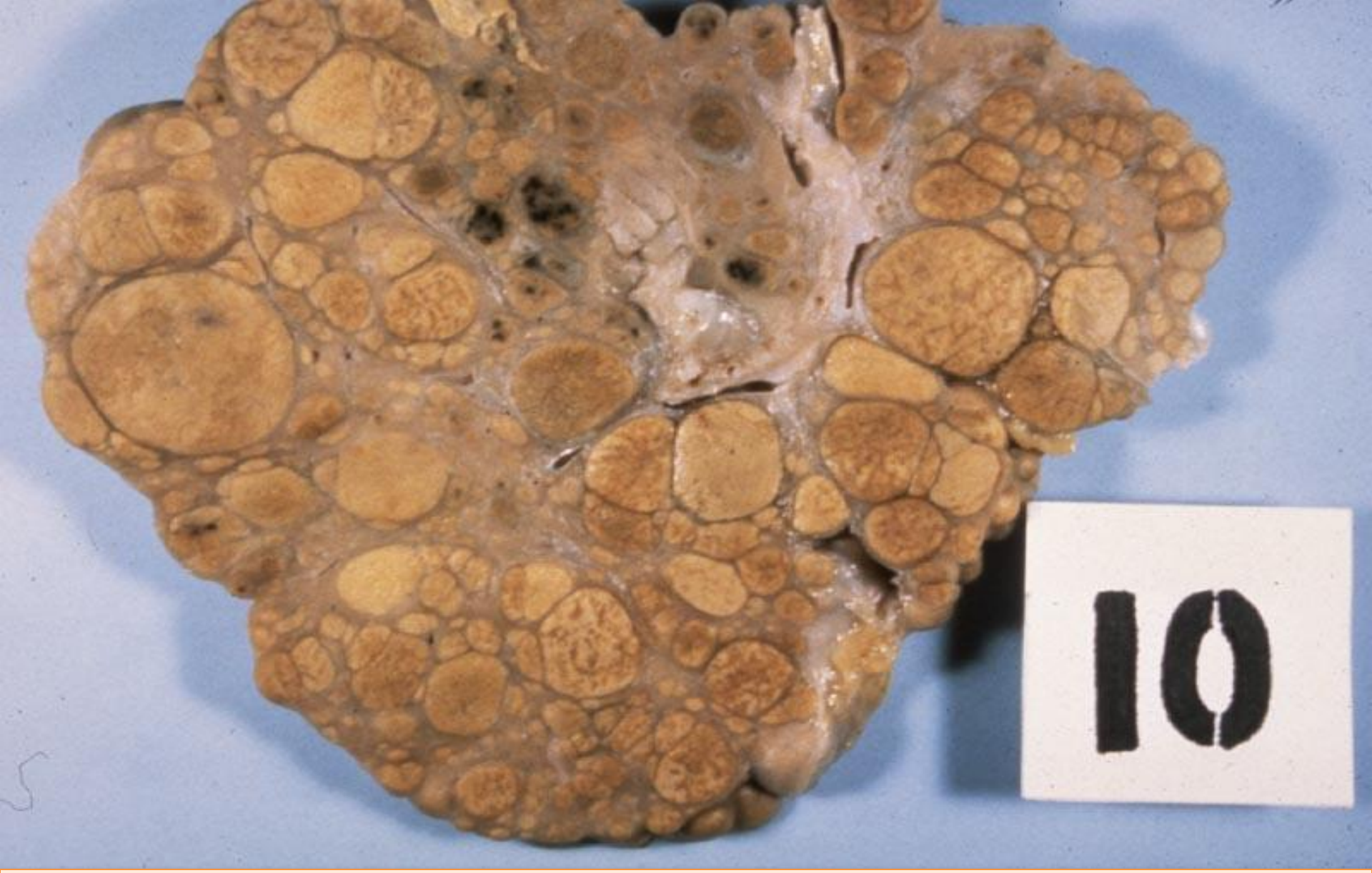
Case 15

cholestasis

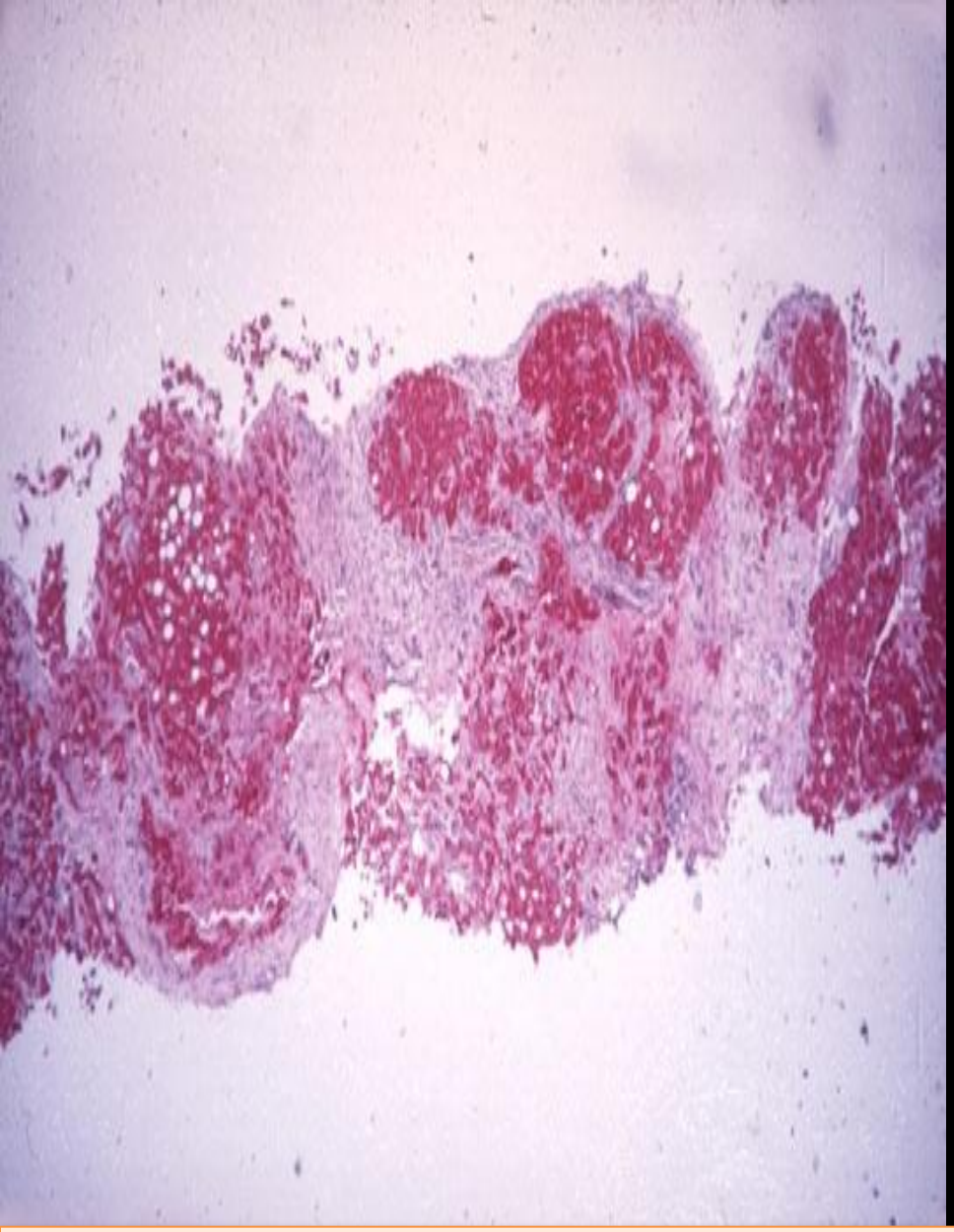
Cholestasis might be duo to pancreatic cancer

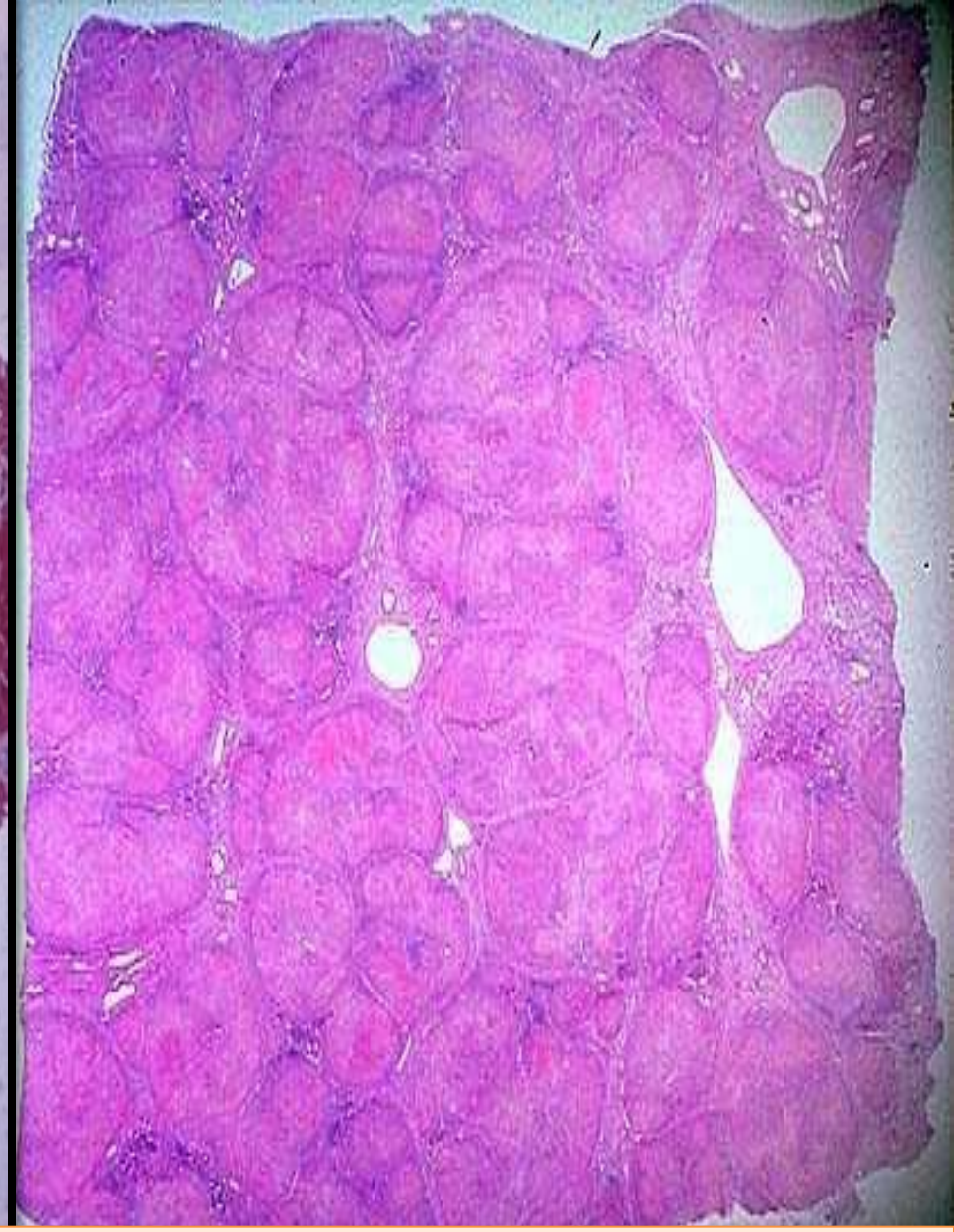
Case 16

Cirrhosis



Organ: Liver Dx: macronudular cirrhosis (HBV)





Irregular nodules separated by portal-to-portal fibrous bands

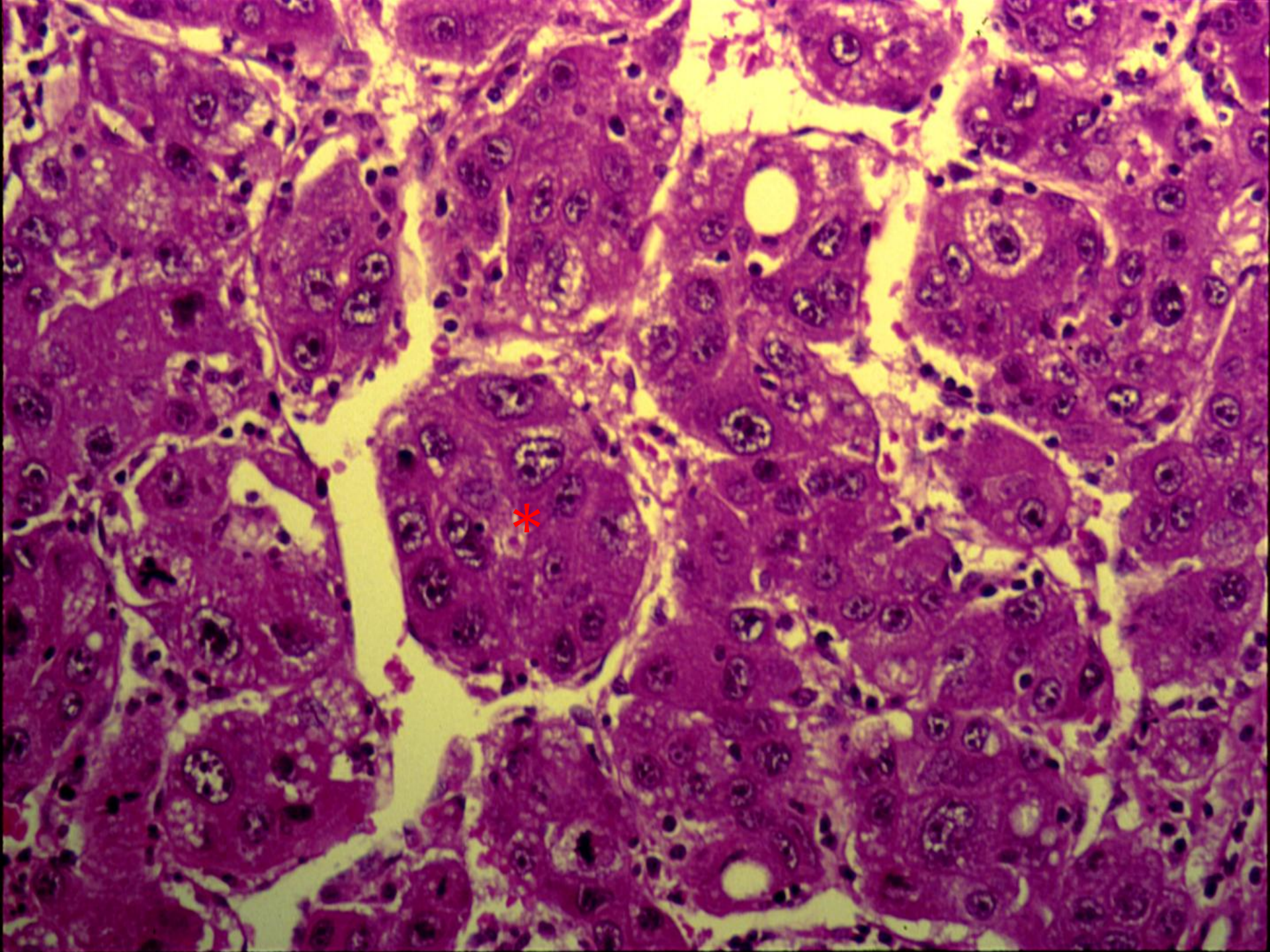
Cirrhosis, trichrome stain

Section of liver show:

- Loss of lobular architecture and formation of regenerative nodules of variable size and shape, surrounded by fibrous tissue.
- Each nodules consists of liver cells without any arrangement and with loss of central vein.
- Large number of proliferated bile ducts and chronic inflammatory cells are present in fibrous tissue. (not seen)

Case 17

Hepatocellualr Carcinoma



Section show tumour consisting of:

- Thick cords, trabeculae and nests of malignant liver cells (*) separated by sinusoidal spaces.
- Malignant liver cells are pleomorphic, binucleated or forming giant cells with hyperchromatic nuclei.
- Mitoses are numerous.
- Areas of haemorrhage and necrosis are present. (not seen)

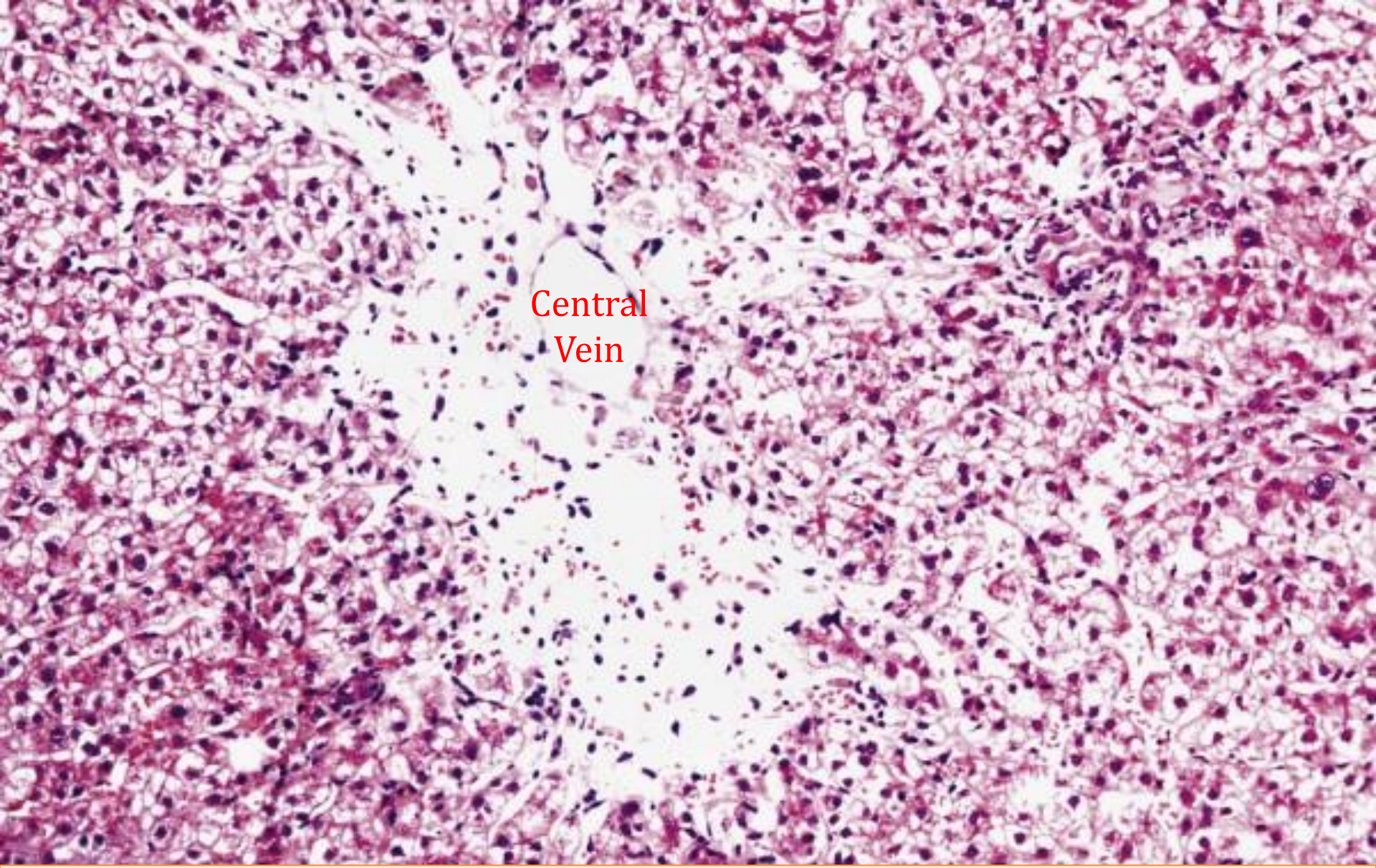
Clinical presentation: ascites

Laboratory finding:

Elevated alpha- fetoprotien

Case 18

Drug- induced liver injury

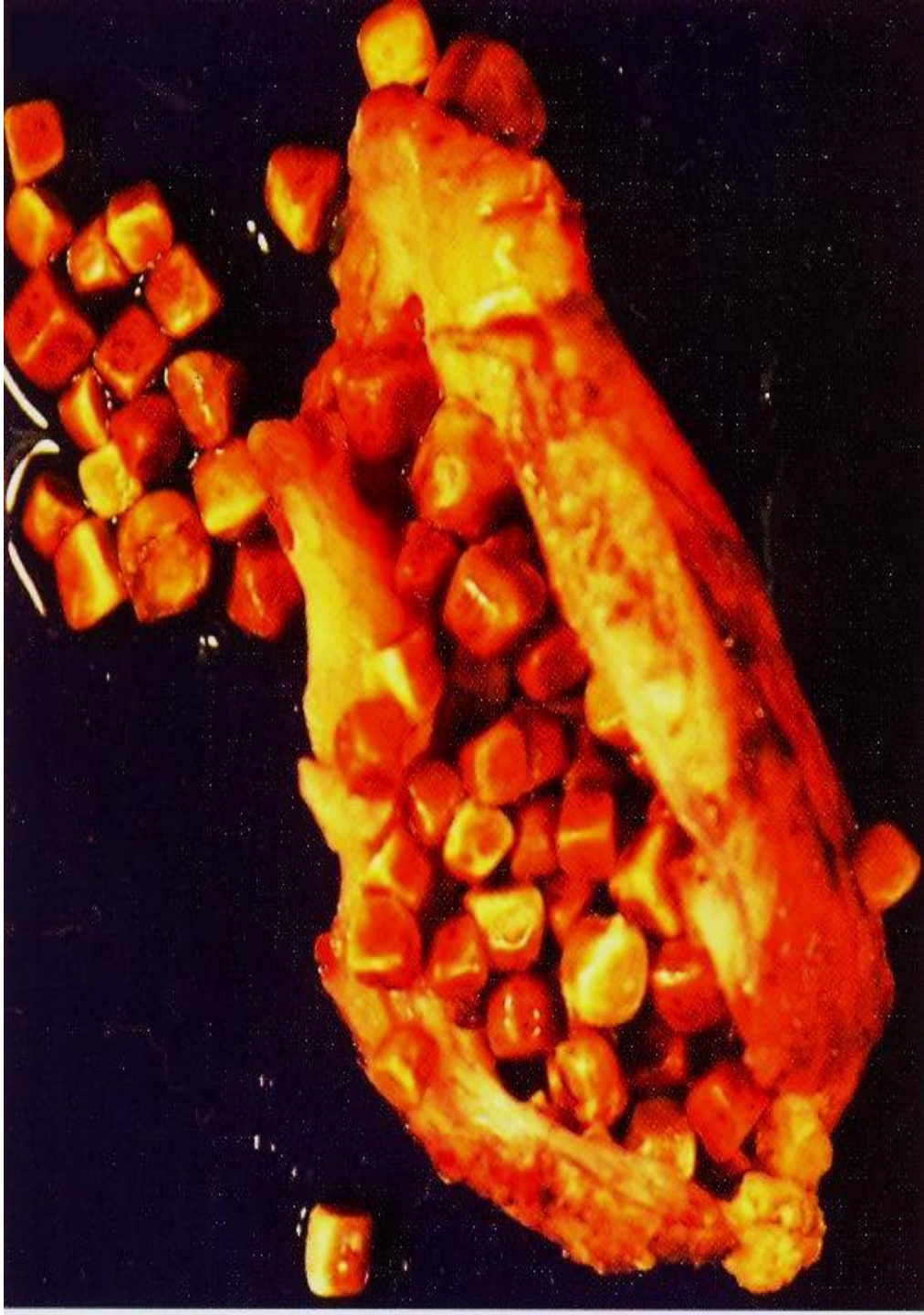


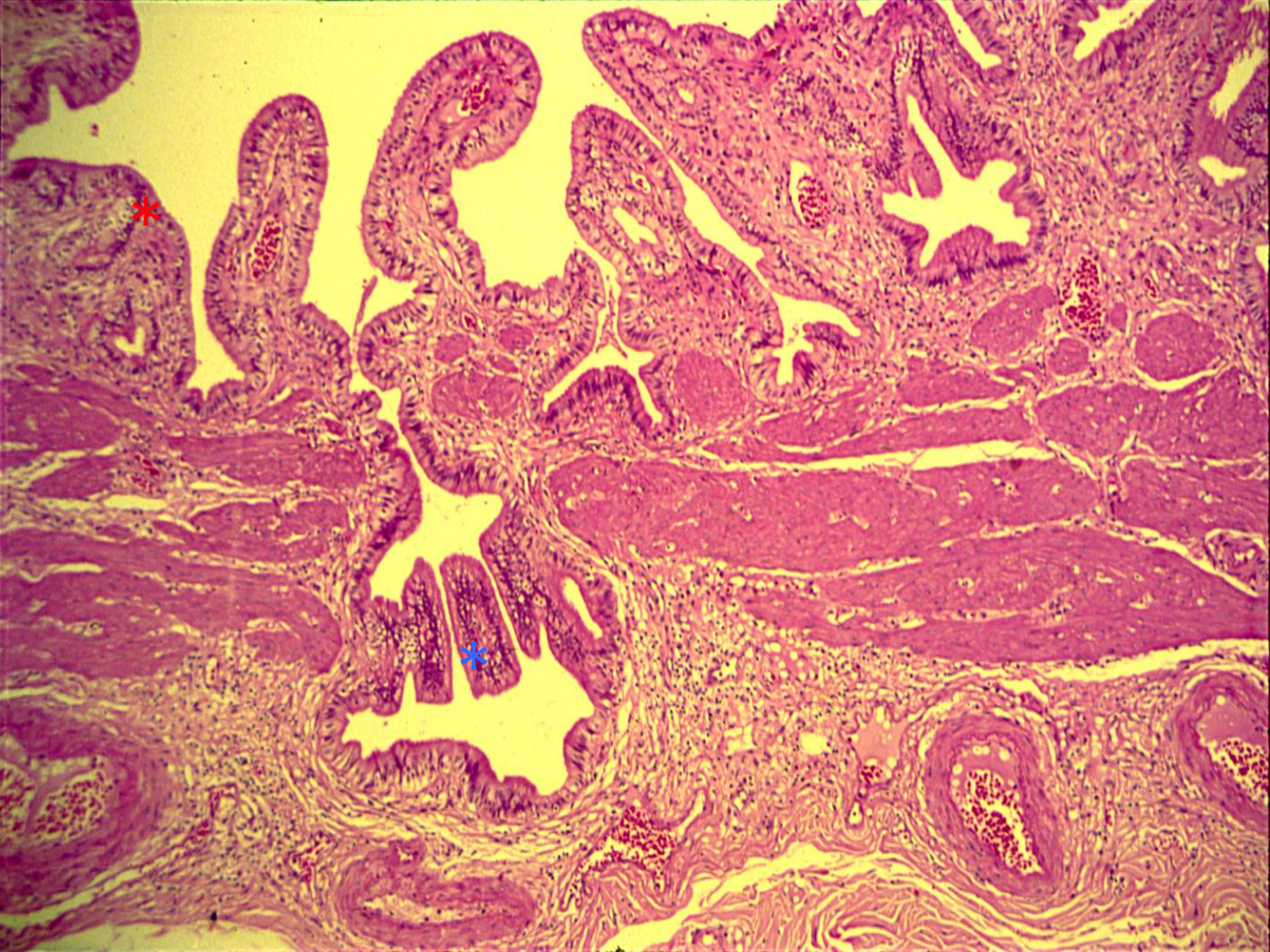
Hepatocellular necrosis due to paracetamol (acetaminophen). Confluent necrosis with little inflammation is seen in a perivenular area. (Needle biopsy, H&E.)

Case 19

Chronic Cholecystitis

Chronic cholecystitis with stones



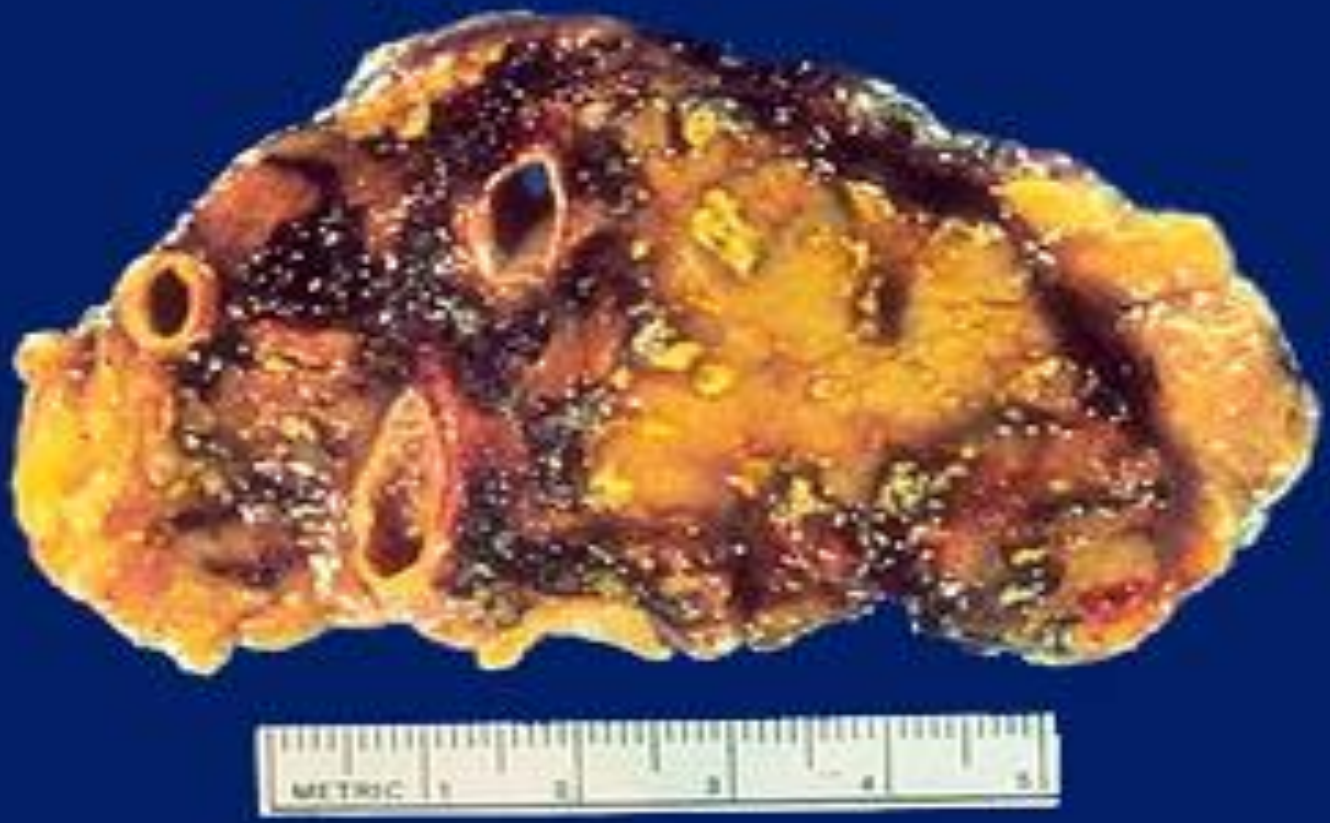


Chronic cholecystitis:
Section of gallbladder wall shows:

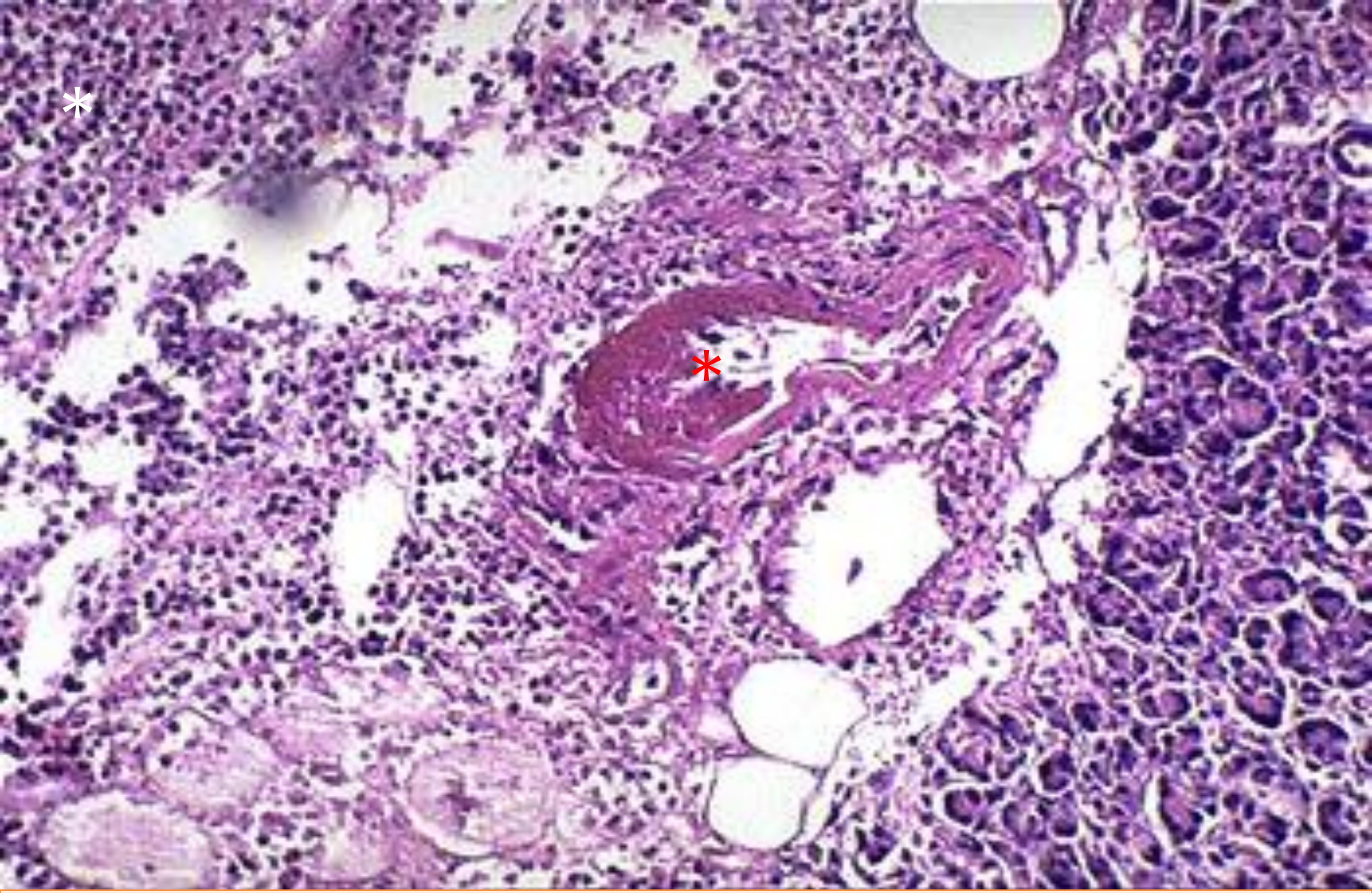
- Irregular mucosal folds and foci of ulceration (*) in mucosa.
- Wall is penetrated by mucosal glands which are present in muscle coat (Rokitansky- Aschoff sinuses) (*).
- All layers show chronic inflammatory cells infiltration and fibrosis.

Case 20

Acute pancreatitis



In severe acute pancreatitis, black areas of hemorrhage are present within the pancreas as well as chalky, yellow-white areas of fat necrosis. Pancreatic parenchyma is soft and gray-white due to necrosis.



This image of severe acute pancreatitis shows an area of acute inflammation (*) with necrosis. Within the necrotic area is a blood vessel showing fibrinoid necrosis (*) of the vessel wall. Damage such as this leads to severe, hemorrhagic, acute pancreatitis.

Morphology

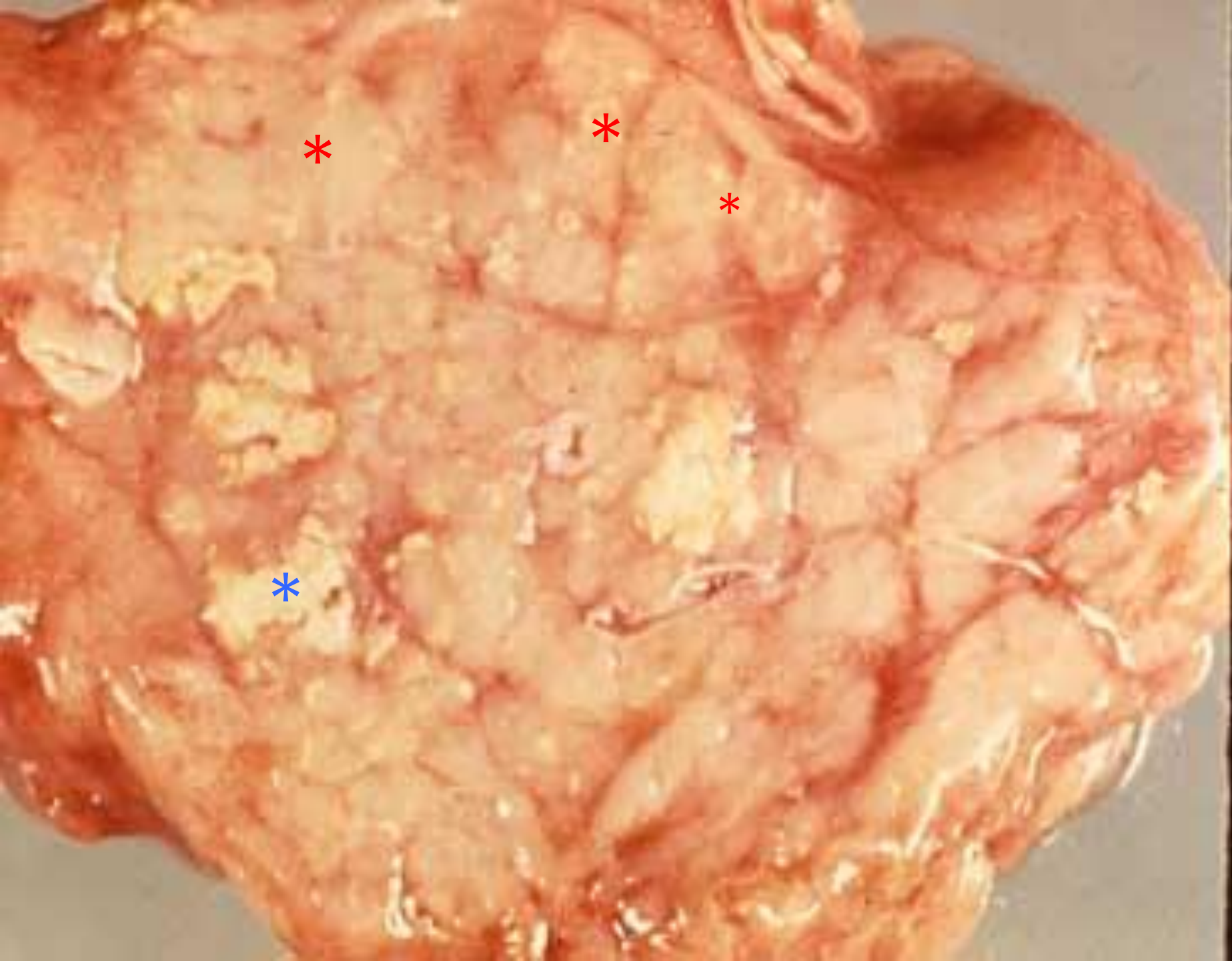
- Edema
- Fat necrosis
- Acute inflammatory infiltrate
- Pancreas autodigestion
- Blood vessel destruction
- “Saponification”

Common causes: alcoholism , gall stones impaction , traumatic , hereditary and idiopathic .

Test: most important lab test is amylase .

Case 21

Chronic Pancreatitis

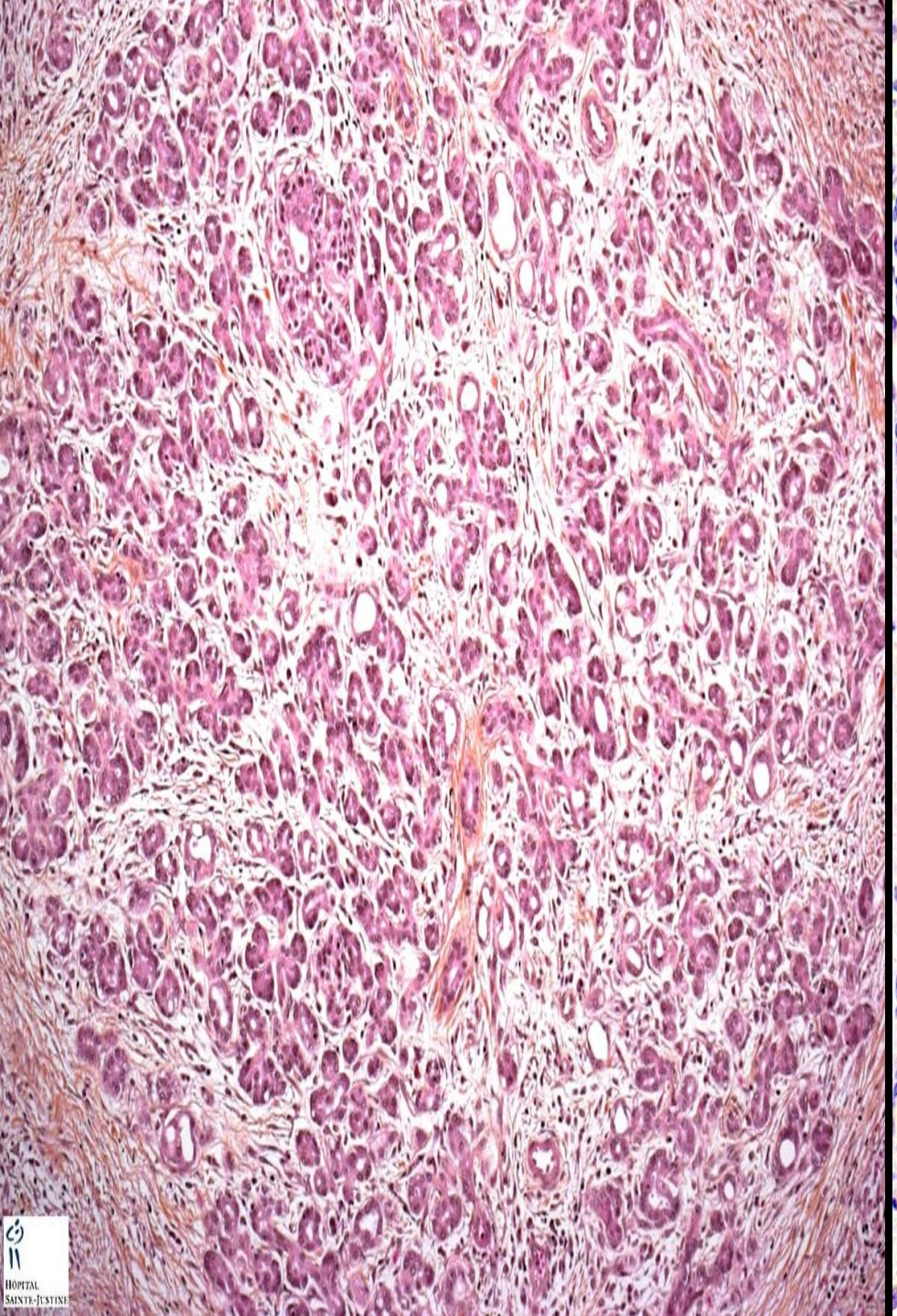


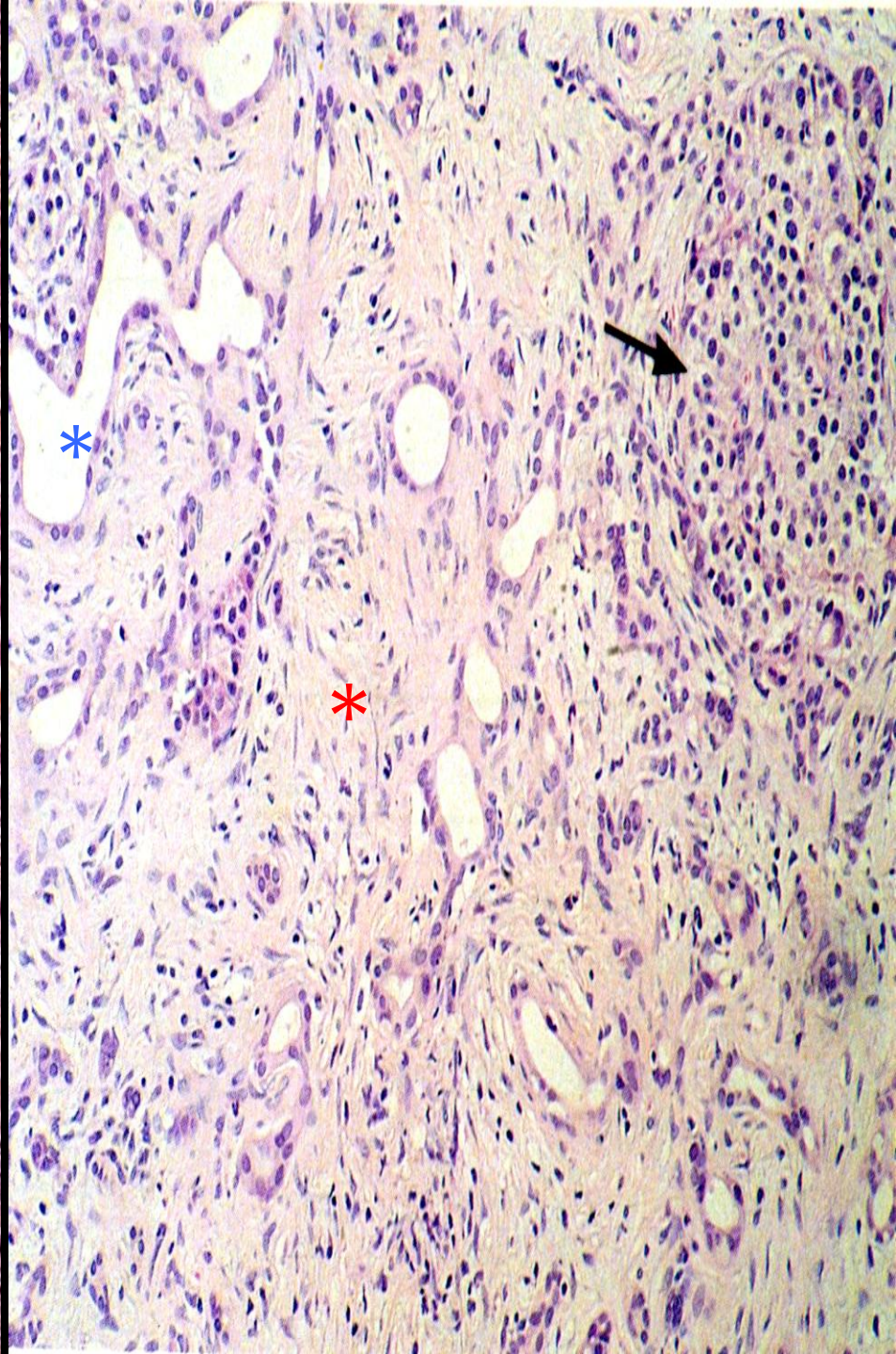
- Find the “soap” (*), find the calcium (*).

Calcium deposition is secondary to fat necrosis and dystrophic calcification .

Possible causes:

gall stones , alcoholism, tropical , hereditary and idiopathic.


HOPITAL
SAINTE-JUSTINE

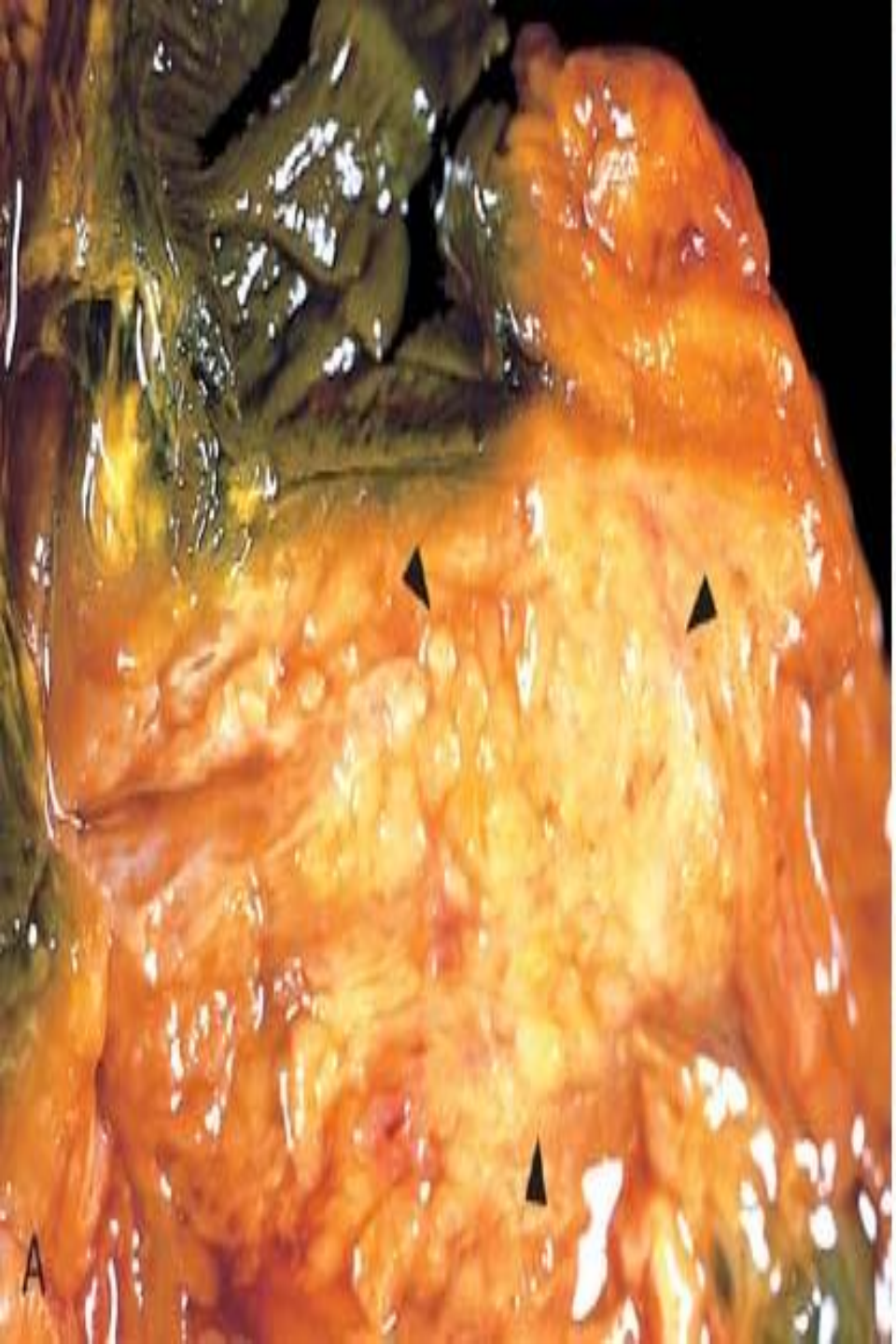


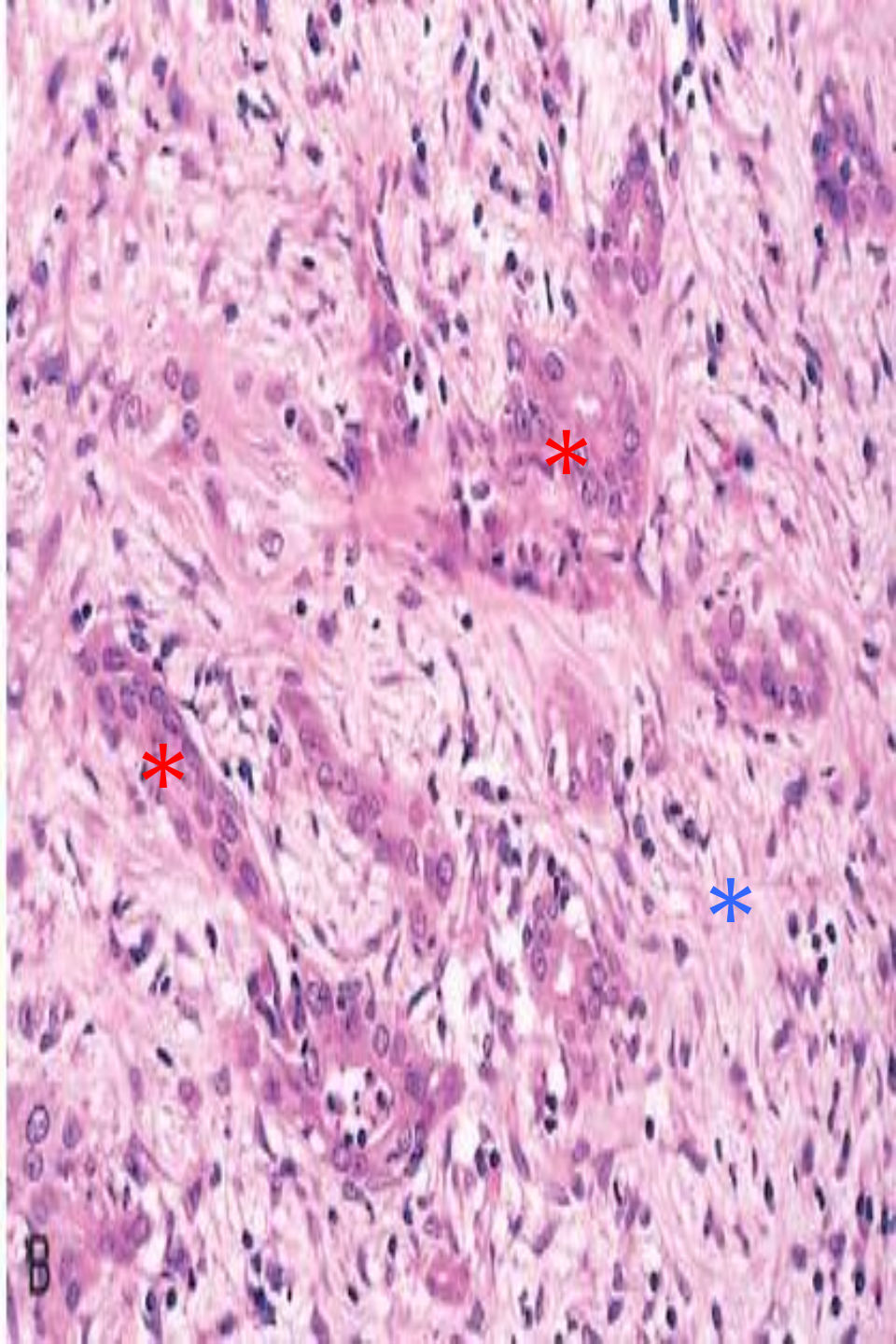
Microscopic sections show:

- Parenchymal fibrosis (*)
- Chronic inflammatory infiltrate
- Reduced number and size of acini (left picture)
- Variable dilatation of pancreatic ducts (*)
- Relative sparing of islets of langerhans (arrow)

Case 22

Pancreatic Adenocarcinoma


A


B

Charactristics:

- Abortive tubular structures (immature, circle tubules) (*)
- Deeply infiltrative growth pattern with irregular shape and distribution (*)
- Desmoplasia (*) (a tool to differentiate between benign and malignant and it is the growth of fibrous or connective tissue)
- Marked nuclear pleomorphism with nucleoli (not clearly seen in this picture)
- Loss of polarity (loss of organization)
- Mitotic figures
- Perineurial invasion in more than 90% of cases