

Practical pathology of GIT Block



تنبيه:

- fatty liver and acute viral hepatitis cases.
ذكرت في المحاضرة ولم يتم ذكرها في المراجعة من قبل الدكتور مرعي
- Cholecystitis and gall stones cases.
موجودة في ملف مراجعة الدكتور مرعي ولم يتم ذكرها في المراجعة والمحاضرة ابدا

لذلك لم نضعهم في الملف

شارك في العمل:

رنيم العتيبي.

روان البديع.

فراص الفواز.

عبدالعزيز المسعود.

عبدالله الزهراني.

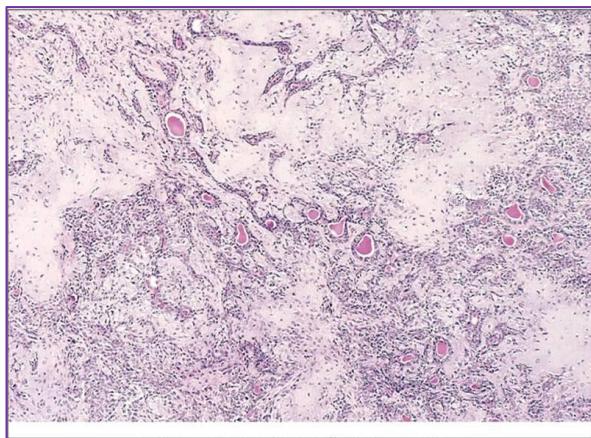
Case 1-Pleomorphic Adenoma



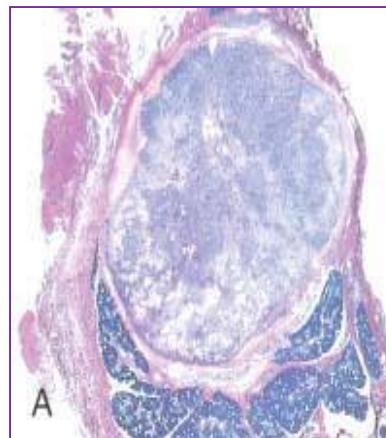
-Left side neck swelling (in the parotid gland) with intact skin.



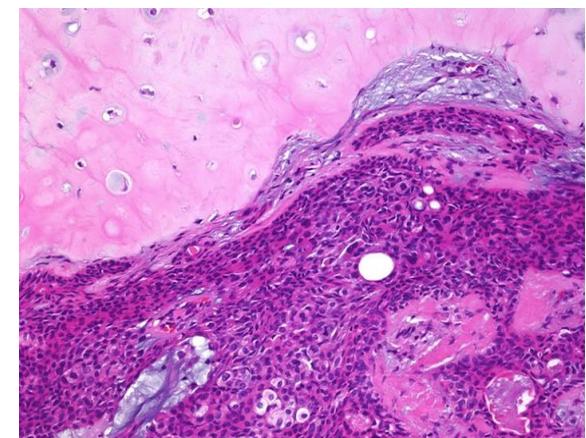
-Well circumscribed mass with pale tan cut surface area and hemorrhage



-epithelial cells forming ducts.
-myoepithelial cells.
-chondromyxoid stroma.



-Cartilaginous and glandular components.
-Connective tissue.



-Small ducts & acini.
-Pale blue matrix
-Myoepithelial.
-Chondriod(cartilage)
-Myxoid (epithelial)

Good prognosis

Case 2-GERD

Endoscopy:

-Edematous area around gastroesophageal junction.



Complications:

-barrett's esophagus

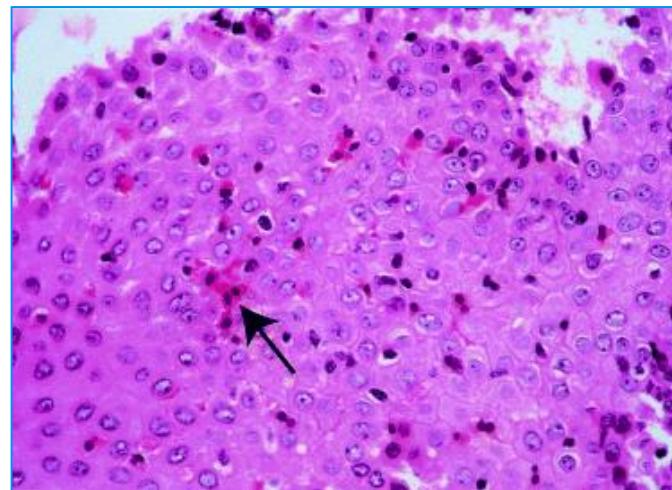
-Intraepithelial eosinophils (arrow).

-basal zone hyperplasia.

-Lamina propria elongation.

-Inflammatory Cells:

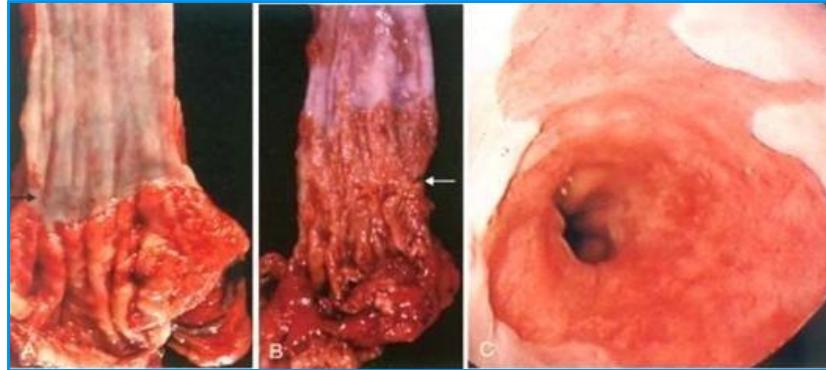
- Eosinophils
- Neutrophils
- Lymphocytes



Case 3- Barret's esophagus

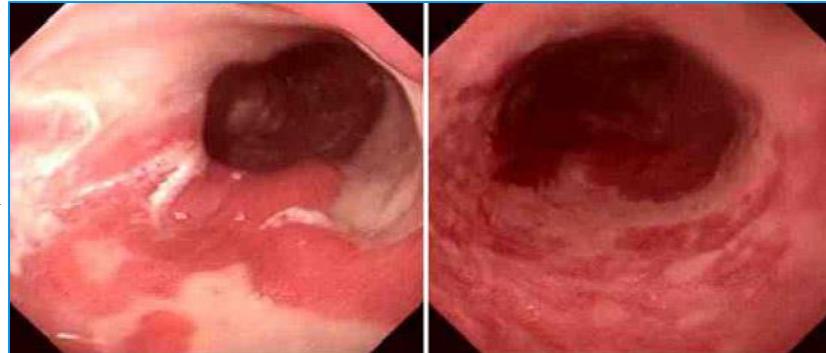
- Its risk factor of ADENOCARCINOMA.
- Columnar lined esophagus is a new name of Barrett's.

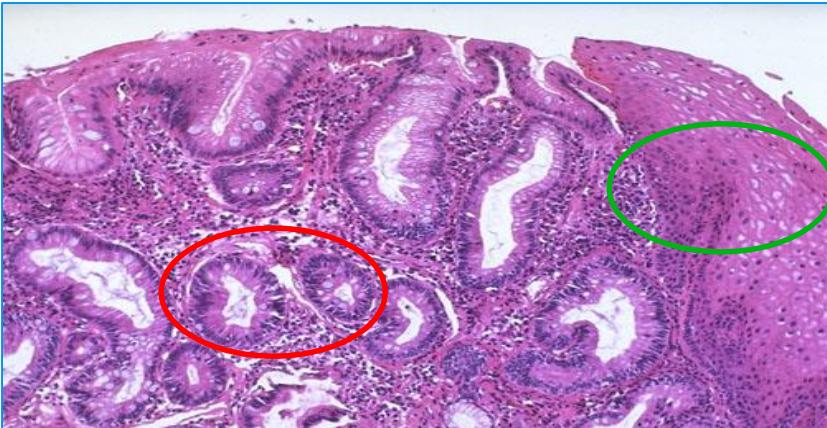
-Erythematous mucosa near the gastroesophageal junction.



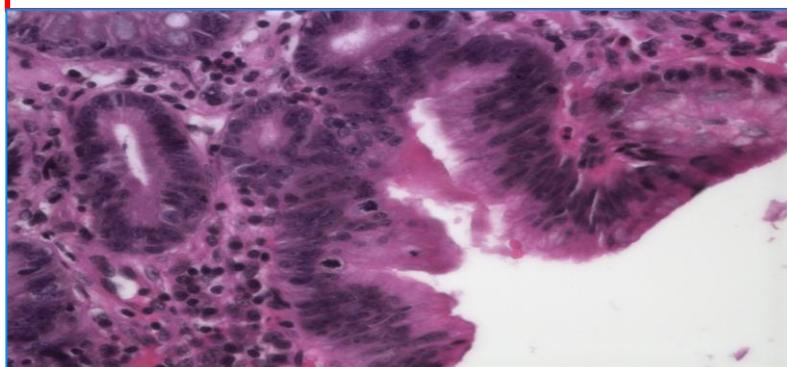
Endoscopy:

- Mucosal erythema of the lower esophagus.
- Islands of normal pale esophageal squamous mucosa.





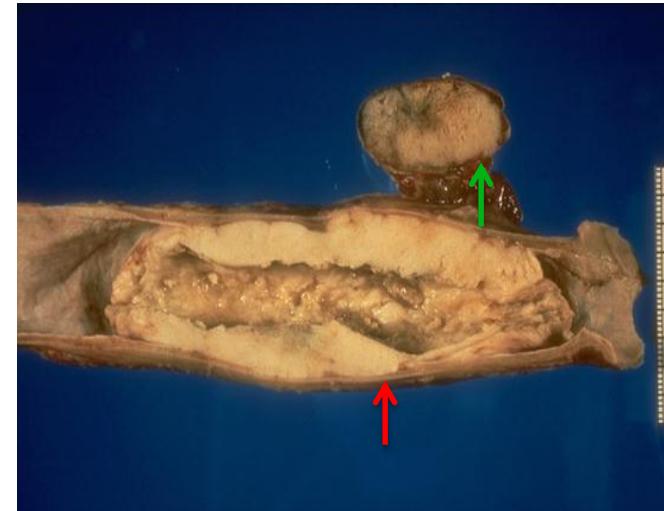
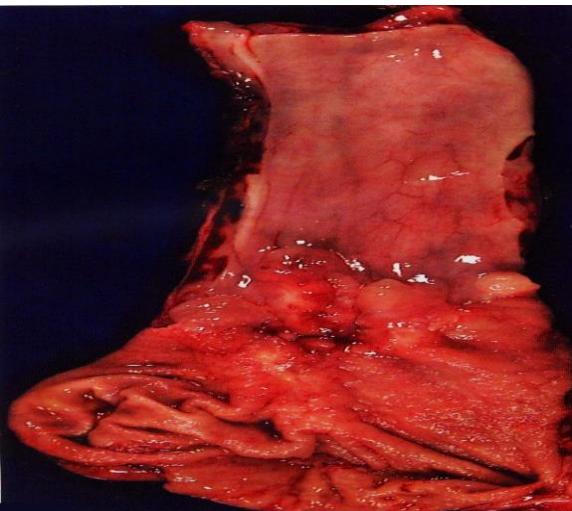
The glandular dysplasia stage



- GOBLET cells in the columnar Intestinal metaplasia (red).
- Normal squamous epithelium(green).
- Chronic inflammatory cells.

- Loss of polarity.
- Increase mitosis.

Case 4- Carcinoma of the esophagus

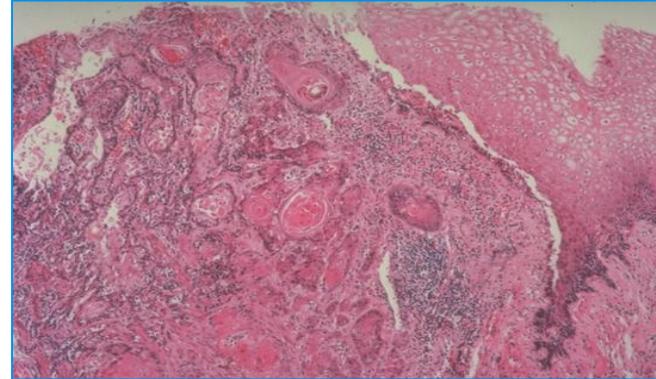
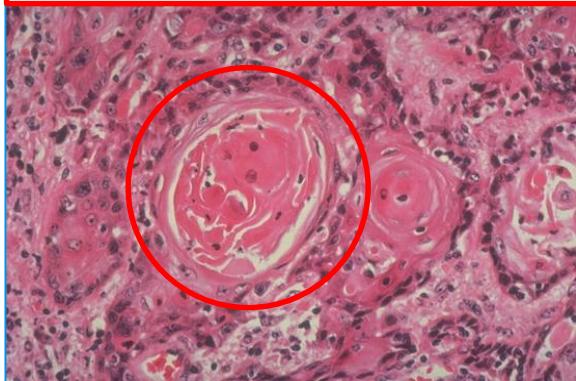


-Irregular reddish, ulcerated exophytic mid-esophageal mass.

- Mass infiltrating the lumen (red arrow).
- Paraesophagus metastatic lymph node (green arrow).

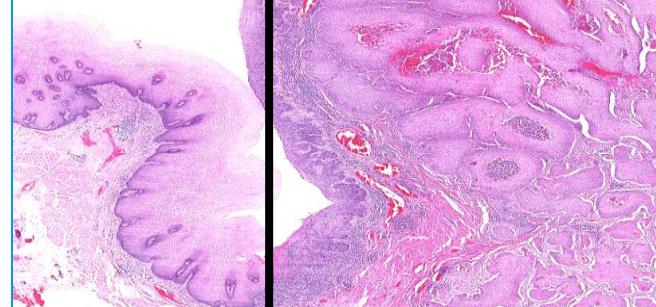
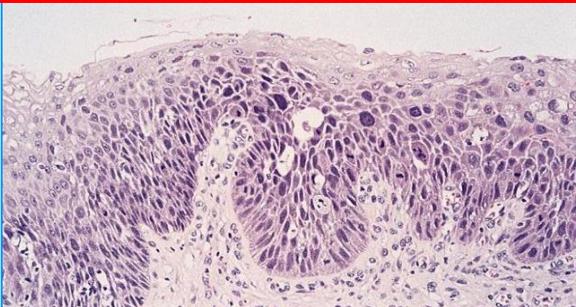
Cont.. Squamous cell carcinoma of the esophagus

Squamous Cell Carcinoma of the Esophagus.



- nests of malignant squamous cells
- pleomorphism, hyperchromatism, increase mitosis, and **invasion of the lamina propria**
- Keratinization** (red circle)

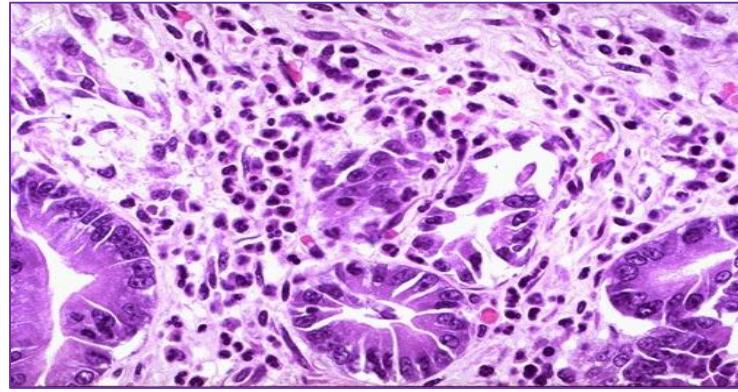
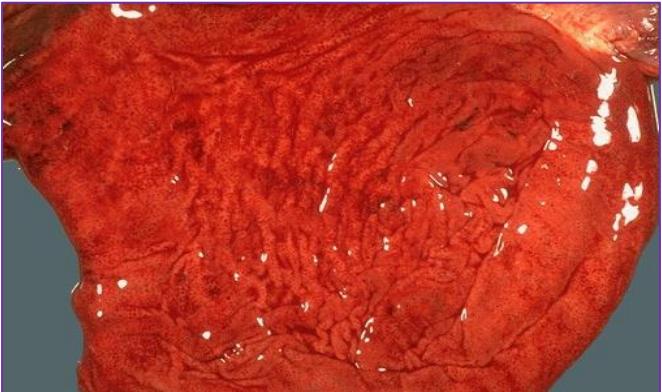
High grade squamous Dysplasia of the Esophagus



- loss of polarity.
- pleomorphism.
- More hyperchromatism.
- Increased mitosis.

- Left normal, right **invasion of the lamina propria**.
- pleomorphism, hyperchromatism, and increase mitosis

Case 5- Acute Gastritis



- diffuse hyperemic gastric mucosa.

- **Risk factors:**

alcoholism, drugs and infections

Case 6- Chronic Gastritis

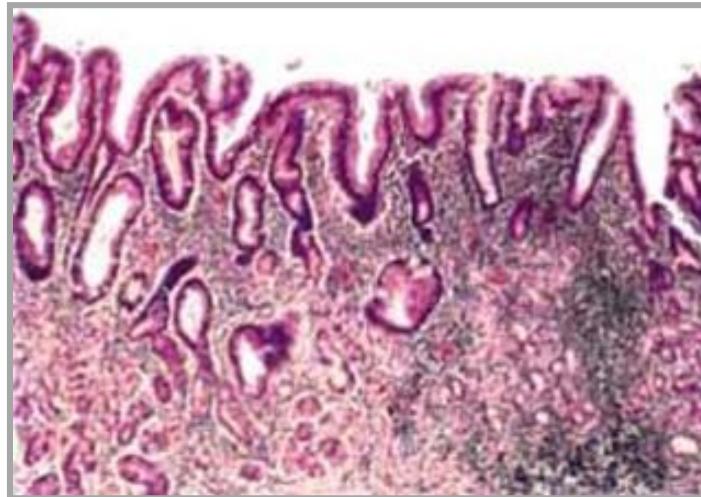
-Chronic, no erosions, no hemorrhage

-perhaps some neutrophils

-Lymphocytes, lymphoid follicles

-Regenerative changes:

- Metaplasia (intestinal)
- Atrophy: mucosal hypoplasia, “thinning”
- Dysplasia



Case 7- GASTRITIS Helicobacter-induced

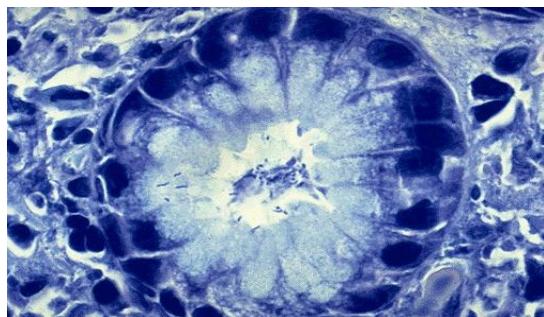
- **Helicobacter pylori, gastric biopsy:**



-Silver stain with numerous Helicobacter pylori.



-Giemsa stain with numerous Helicobacter pylori.



-Giemsa stain with numerous Helicobacter pylori.
-found in the surface epithelial mucus of most patients **with active gastritis**

- **Peptic ulcer**

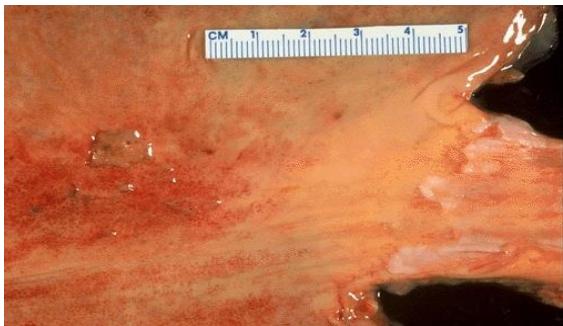


-Regular edges mucosal ulcer
-clear base

Note\H-pylori induced gastritis can cause cancer if not treated

Case 8- Acute gastric ulcer

- Benign

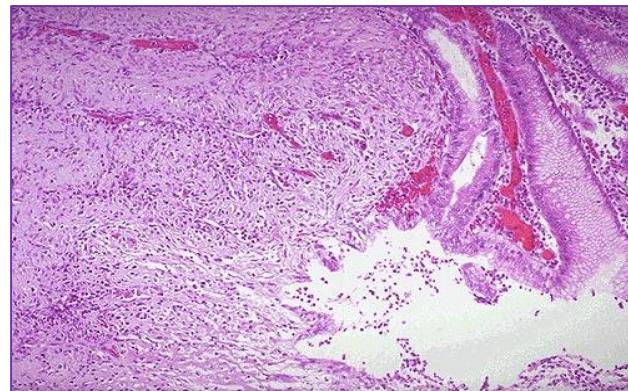
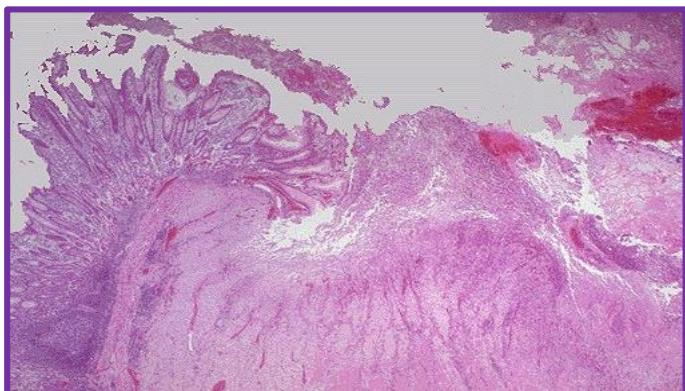


- regular ulcerative shallow ridge.
- sharply demarcated with clean base.

- Malignant



- irregular ulcerative deeper ridge.
- with dirty base.



- loss the epithelium over lining the ulcer.
- necrotic debris.
- Inflammatory collection.
- congestion of the capillaries.

Case 9- Chronic gastric ulcer

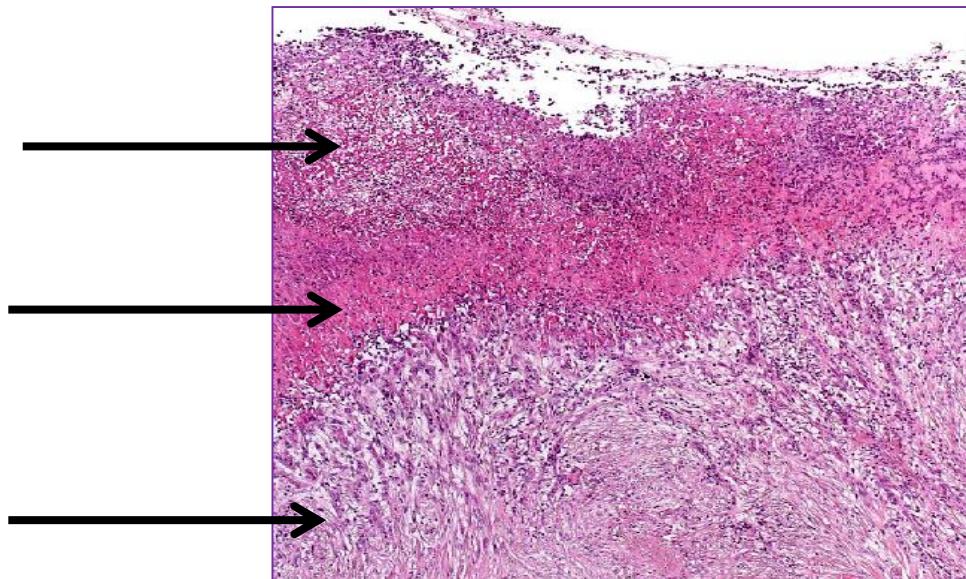
- Regular gastric mucosal ulcer.
- Sharply demarcated with necrotic debris.

Complications:

- Perforation, bleeding.
- Pyloric obstruction.
- Malignant transformation.



-Cellular Debris with neutrophils infiltration.



-Fibrinoid Necrosis and granulation tissue.

-Fibrosis.

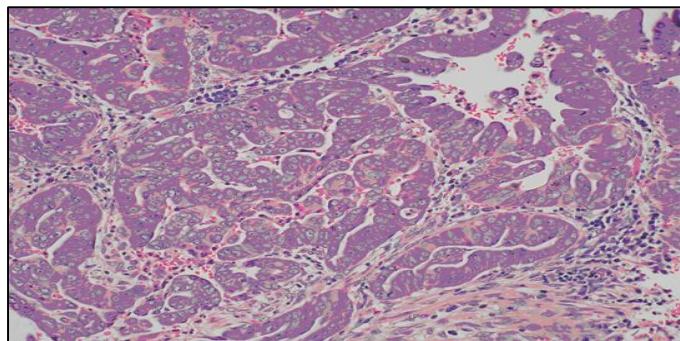
Case 10- Carcinoma of the stomach



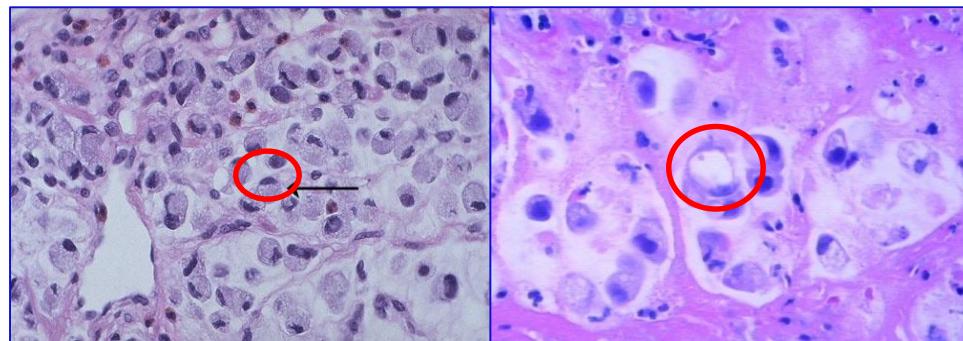
1-intestinal type: **Irregular mucosal mass**



2-diffused type: **Linitis plastica**
“diffuse infiltration and **thickening of the gastric wall**”.



1-intestinal type:
-Irregular crowded malignant glands.
-pleomorphism, hyperchromatism, and mitosis.



2-diffused type:
-**Malignant signet ring cell** (circle) infiltrating the lumen of the stomach
-Mucin vacular cytoplasm push the nucleus to the periphery

* Most common cause is *Helicobacter pylori*.

Case 11- Acute pancreatitis



- 1- Fat necrosis
- 2- Chalky appearance
- 3- area of hemorrhage

1- fibrinoid necrosis of blood vessel (circle).

2- Fat necrosis .

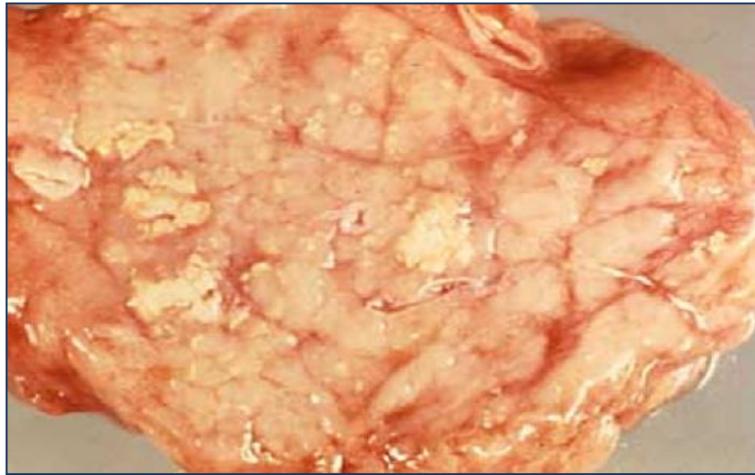
3- inflammatory cells .

(normal acini in right area of the picture)

Lab Test :elevated α -AMYLASE



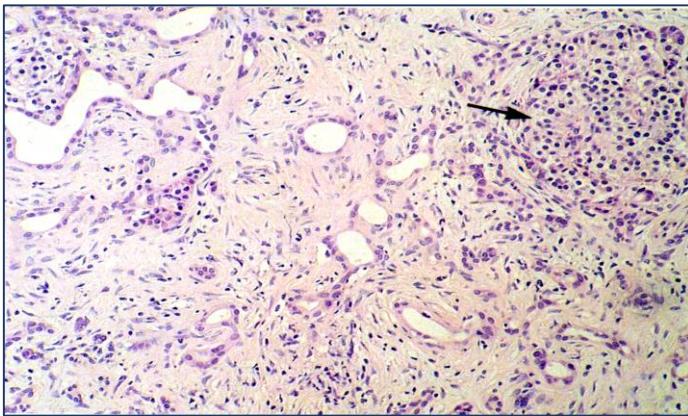
Case 12- chronic pancreatitis



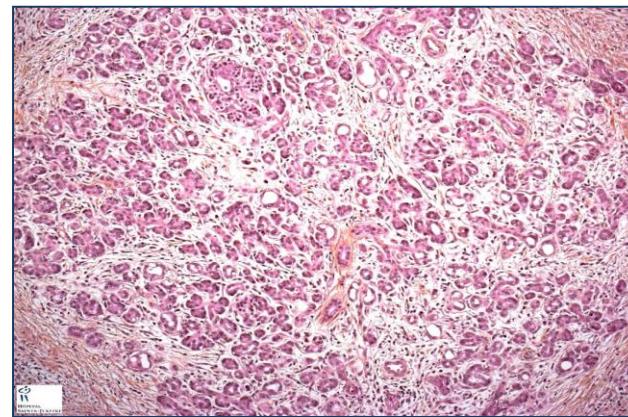
Causes of chronic pancreatitis :

- 1- gall stones.
- 2- alcoholism.

-Calcium depositions.



- Acinar atrophy.
- Fibrosis (stromal fibrosis)
- chronic inflammatory cells.
- Residual islets of langerhans(arrow).



- Fibrosis more prominent.
- Residual islets of langerhans

Case 13- pancreatic adenocarcinoma

-well-circumscribed tumor mass of the head of pancreas.

-dilatation of major pancreatic duct.

-normal duodenum and spleen.

-irregular grey-white mass.

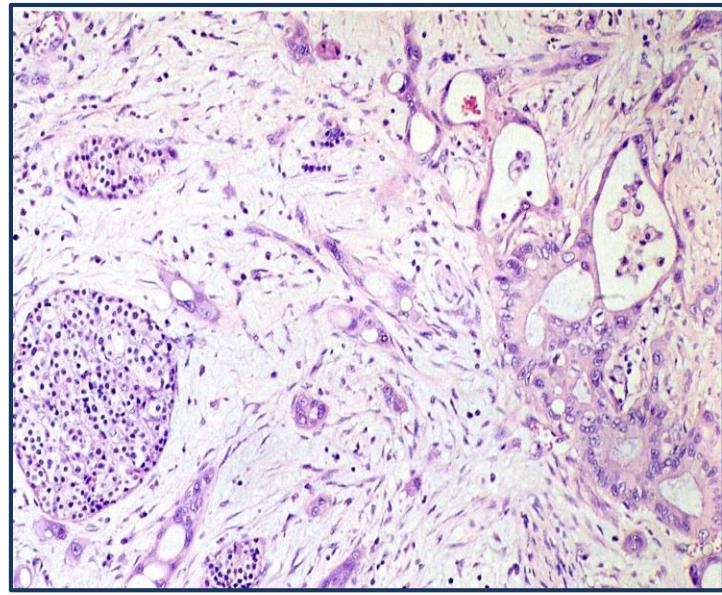
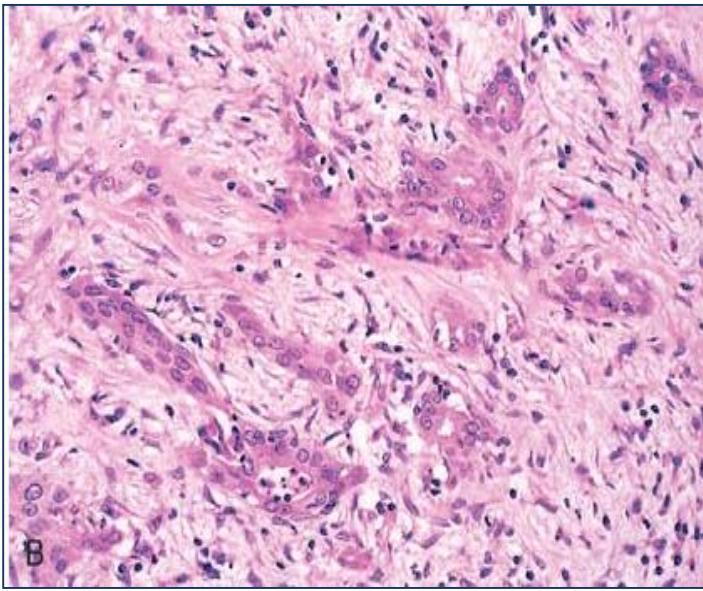
-area of hemorrhage.

-cyst.

-irregular infiltrated mass.



Cont.. pancreatic adenocarcinoma



- malignant glands infiltrative or acini surrounded by **desmoplastic fibrous stroma**.
- Malignant cells characteristics:
 - A- hyperchromatism.
 - B- pleomorphism.
 - C- mitosis.

Case14- small intestine peritoneal adhesion

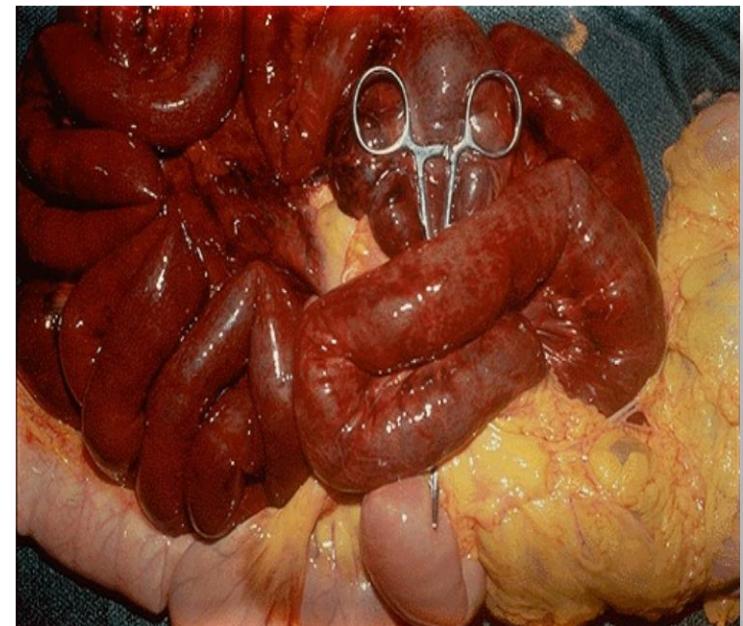
Occur following abdominal surgery or peritonitis.



Case 15- small intestine infarction

Dark red blackish wall of small intestine.

*one of the complication of adhesion



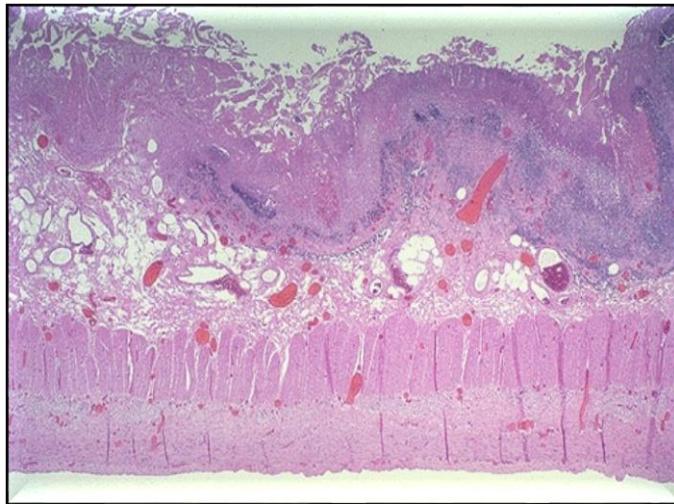
Case 16- Ischemic Enteritis



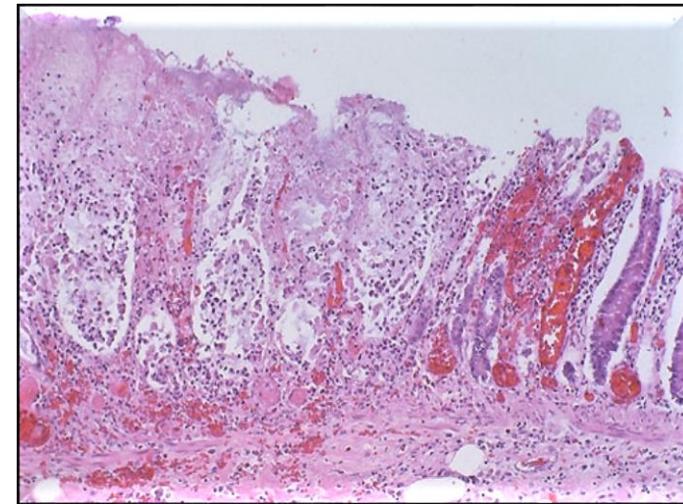
-Marked hyperemic mucosa



-Early ischemic enteritis, pale red hemorrhagic areas



-Extensive necrosis with hyperemia.
-exudate or neutrophilic cells
infiltrating mucosa and sub mucosa.

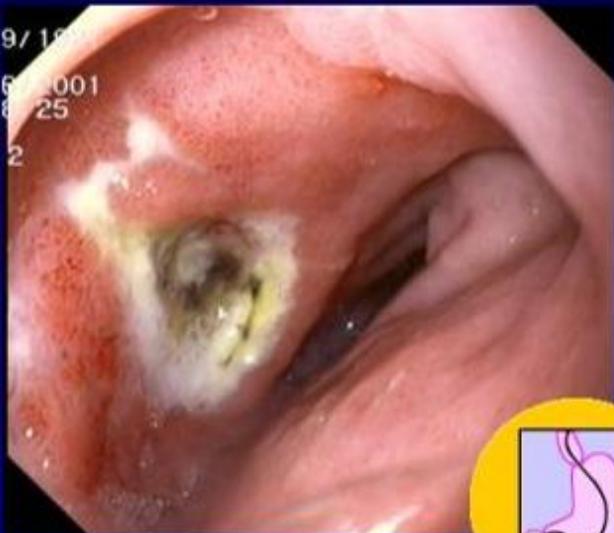


-Necrosis with hyperemia.
-Inflammatory cells in mucosa, submucosa.

Causes: Hypotension, hemorrhage, strangulation, valvulus, and intussusception.

Case 17- chronic duodenal ulcer

Duodenal Ulcer (DU)



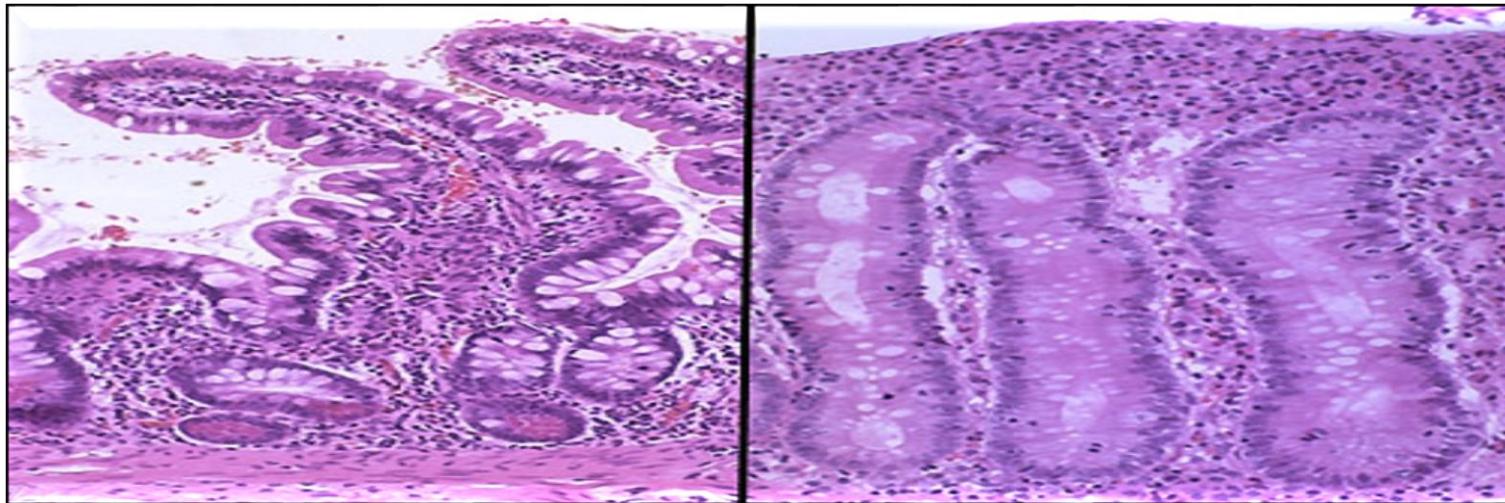
Gastric Ulcer (GU)



- White base ulcer with blackish area.

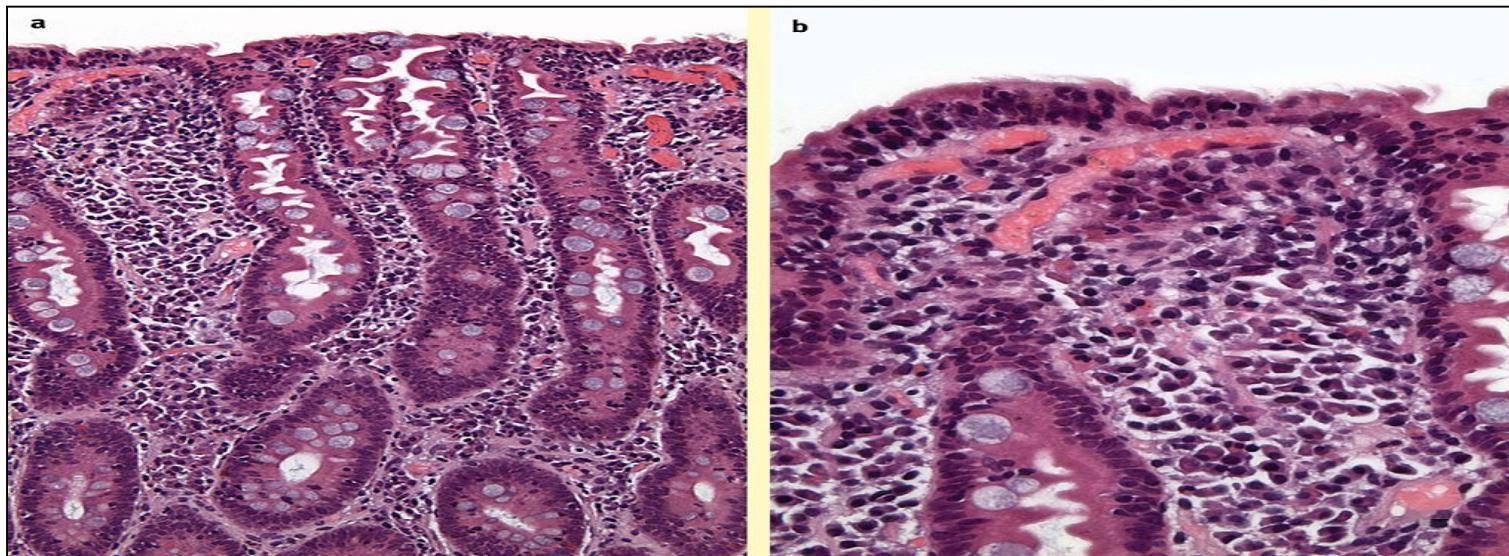
- White ulcerated area.
- swelling around its margin.

Case 18- celiac disease (Sprue)



- Normal

- Villous atrophy.
- Increased intraepithelial lymphocyte

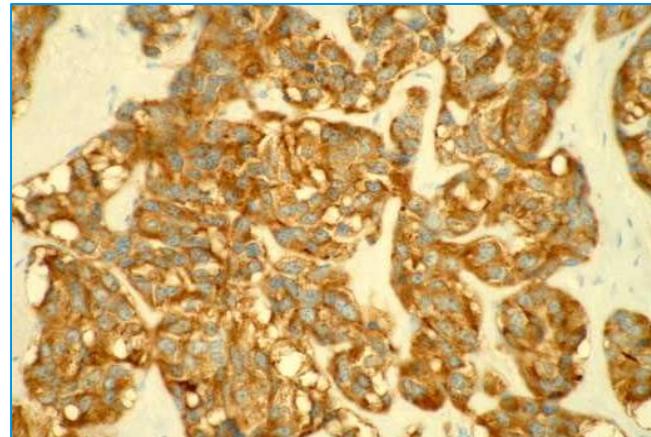


- Villous atrophy.
- Increased intraepithelial lymphocyte.

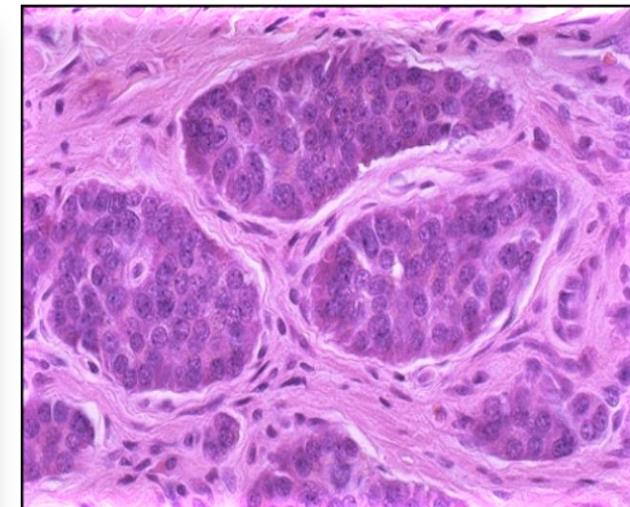
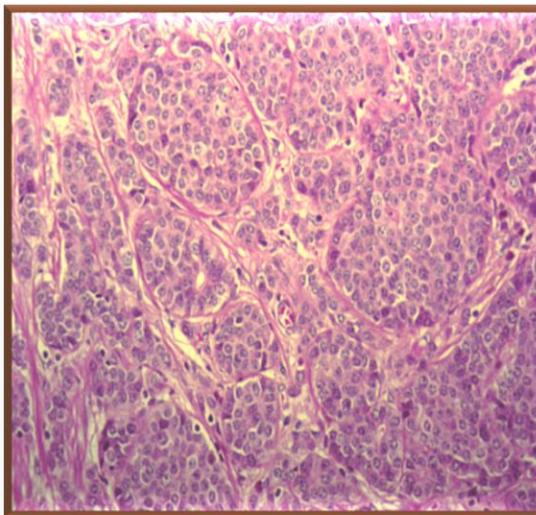
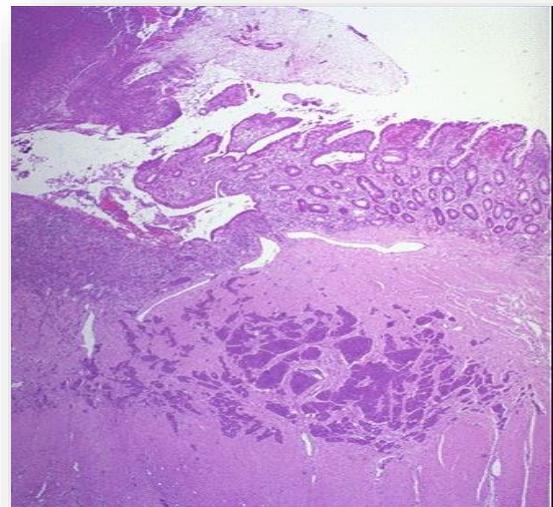
Case 19- carcinoid (neuroendocrine) tumor of small intestine



-nodular yellowish mass at the mucosa of small bowel



-synaptophysin immunohistochemical stain shows positive brownish cytoplasmic staining.



-Multiple nests of neuroendocrine tumor cells in the submucosa.

- Uniform oval polygonal cells.
- Salt and paper chromatin.
- Granular cytoplasm.

* EM will show neurosecretory granules in the cytoplasm.

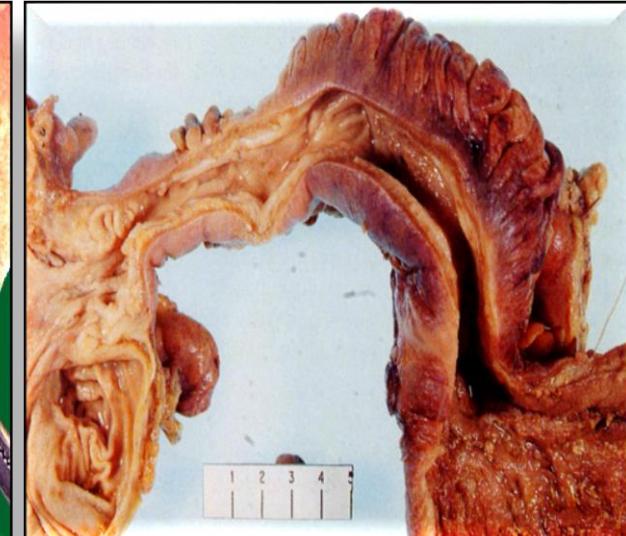
Case 20- crohn's disease



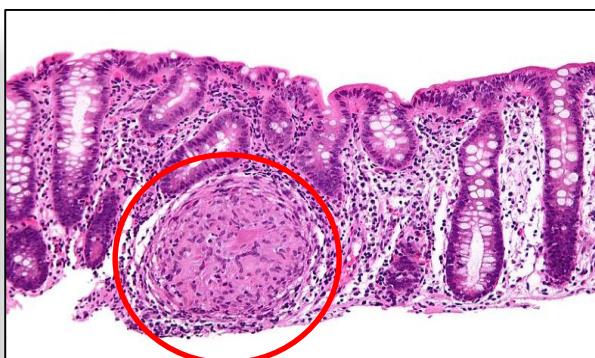
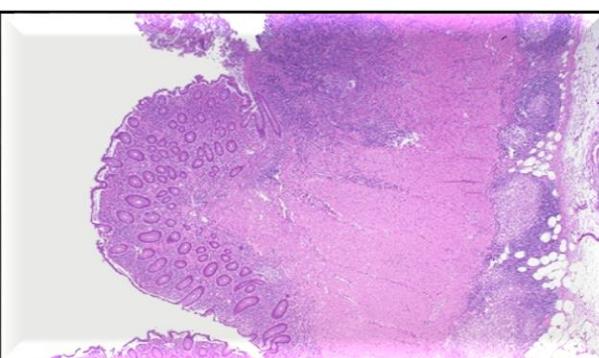
-Ulcerated inflamed wall
(skip lesion).



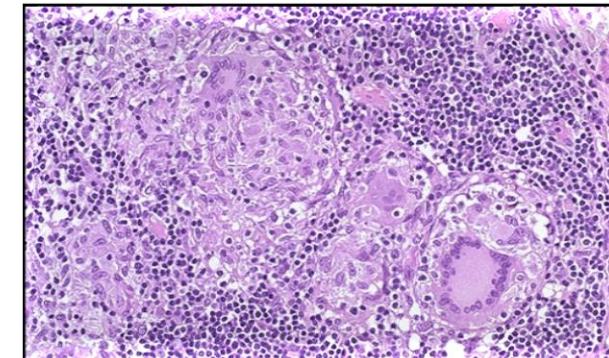
-cobble stone appearance.
-thick wall.
-superficial mucosal ulcer.
-narrowing of the lumen.



-Same feature + Creeping fat.



- Transmural chronic inflammation.
- non-necrotizing **granuloma** in the submucosa (circle).

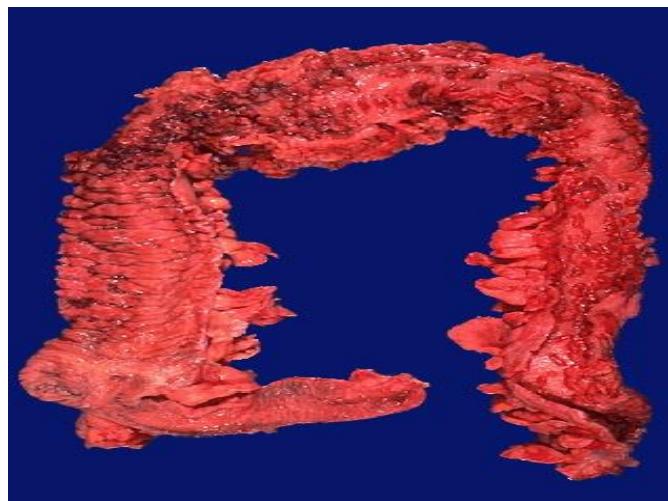


-non-necrotizing granuloma
consist of epithelioid cells, giant cells, and many lymphocytes

Complication of crohn's disease:

Fistula, sinuses, obstruction, perforation, and risk for malignancy

Case 21- Ulcerative colitis



-diffused irregular ulcerated inflammatory **mucosa** of the distal colon.

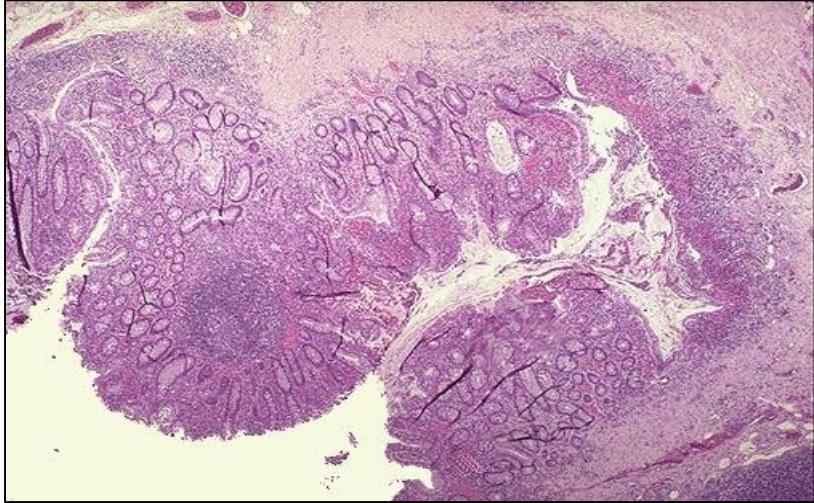


- Many pseudo polyps (inflammatory).

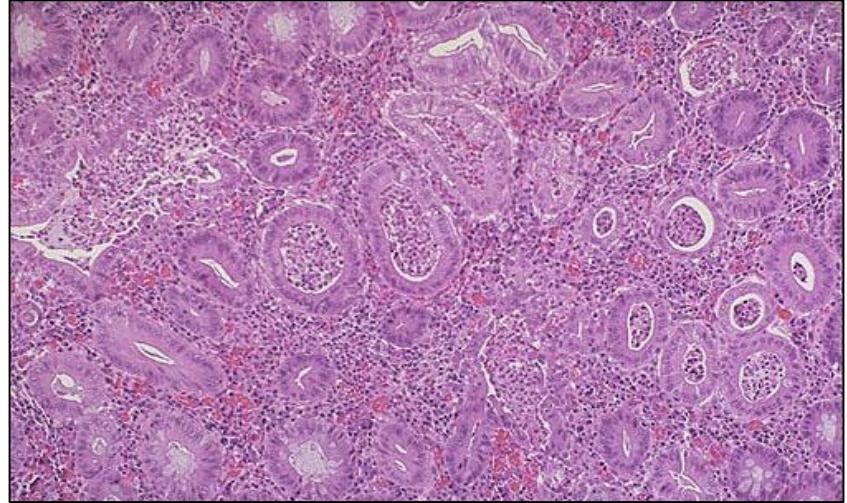
complications:

Toxic mega colon, glandular dysplasia and adenocarcinoma .

cont.. Ulcerative colitis

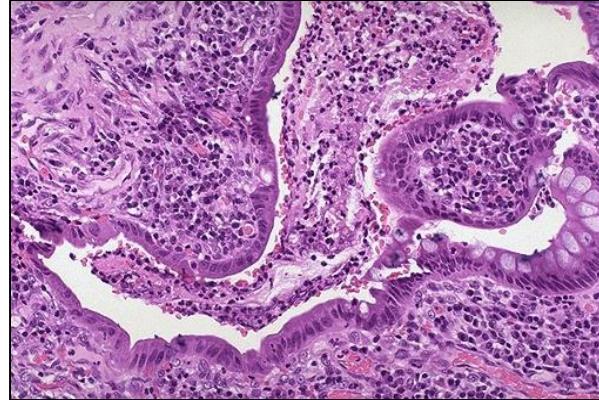


-Mucosal ulceration and inflammation.

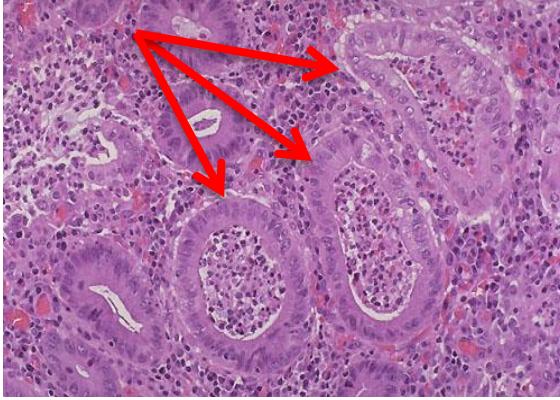


Mucosa shows : - cryptitis.

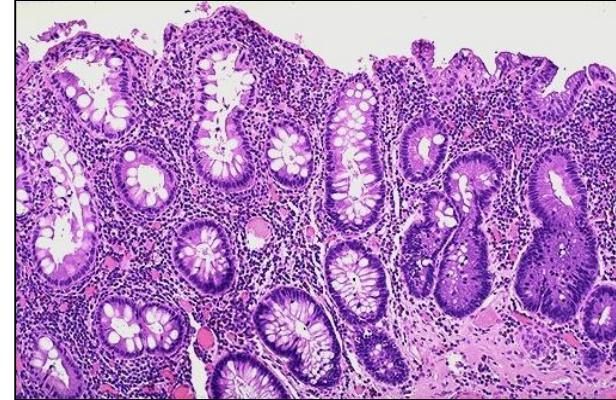
- crypt abscesses “ neutrophils inside the tubules”
- Loss of goblet cells.



-Chronic Inflammatory cells .
-Loss of Goblet cells.



-Crypt Abscesses.
-chronic inflammatory cells.



-Ulcerative colitis with some degree of dysplasia.

Case 22- Adenomatous polyps

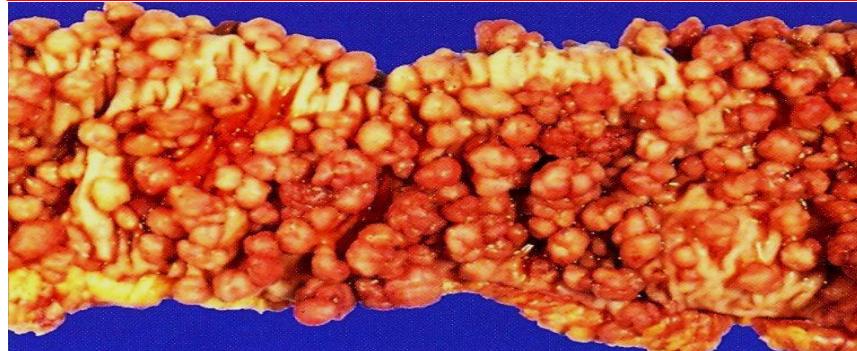


Multiple adenomatous polyps



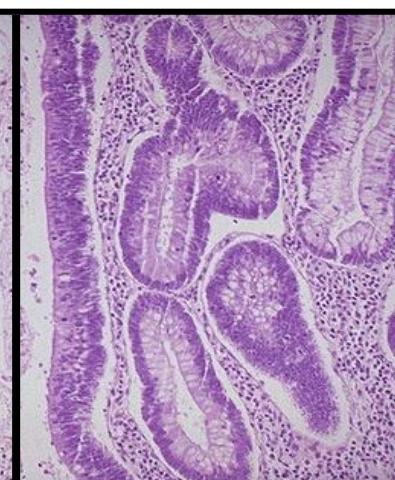
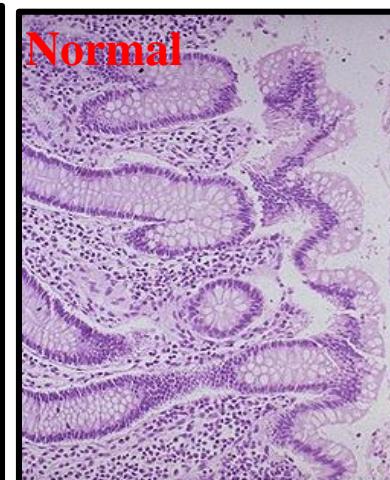
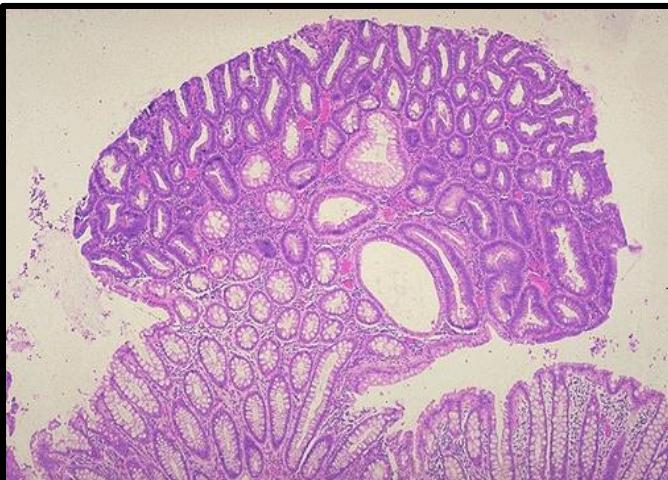
Large one Adenomatous polyp

Familial polyposis of the colon



-Hundreds of polyps.
Associated with APC gene mutation

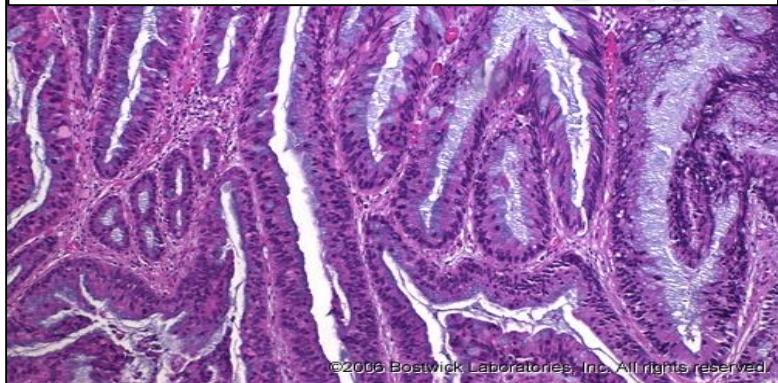
cont.. Adenomatous polyps



- Crowded tubular glands forming polypoid mass.
- Decreased goblet cells.
- Hyperchromatic epithelium, no invasion

- Adenomatous polyps.
- hyperchromatism.

Villous adenomatous polyp *



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- Crowded **villous** glands.
- Decreased goblet cells.
- Hyperchromatic nuclei

* have a **HIGHER** risk of developing into adenocarcinomas

Tubular adenomatous polyp

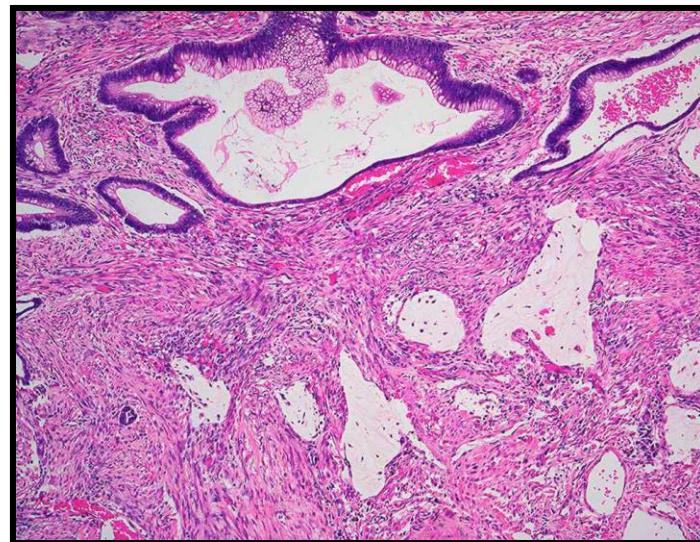


- Crowded **tubular** glands
- Decreased goblet cells.
- Hyperchromatic nuclei.

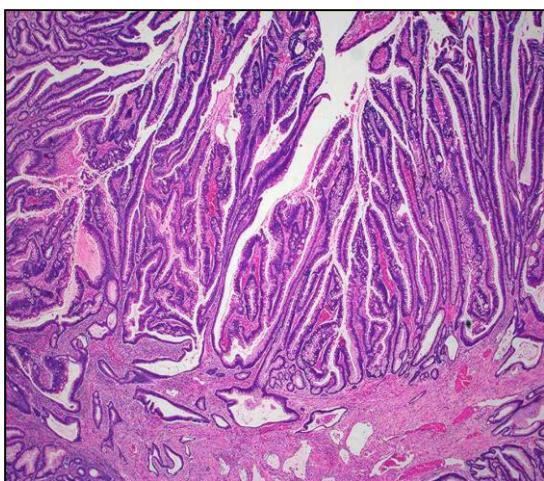
Case 23- Adenocarcinoma of the colon:



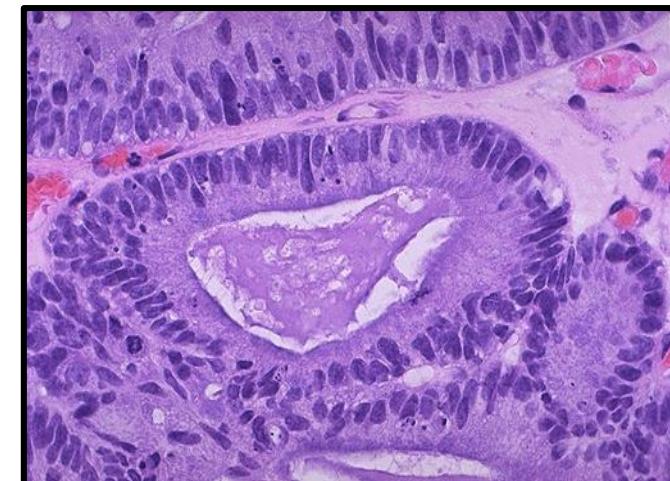
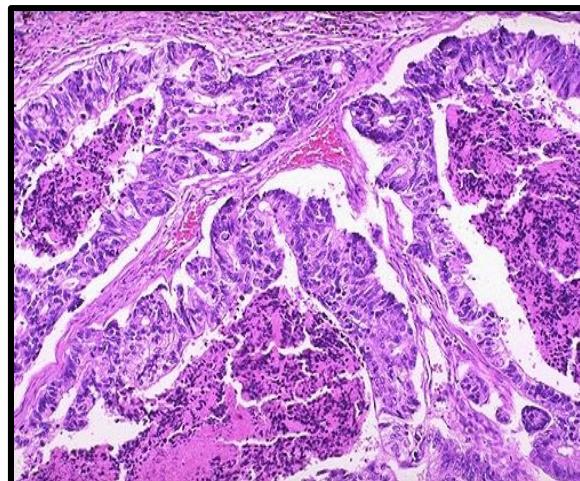
-Irregular fungaiting mass in the mucosa of the colon



-Same features, **muscle coat**.



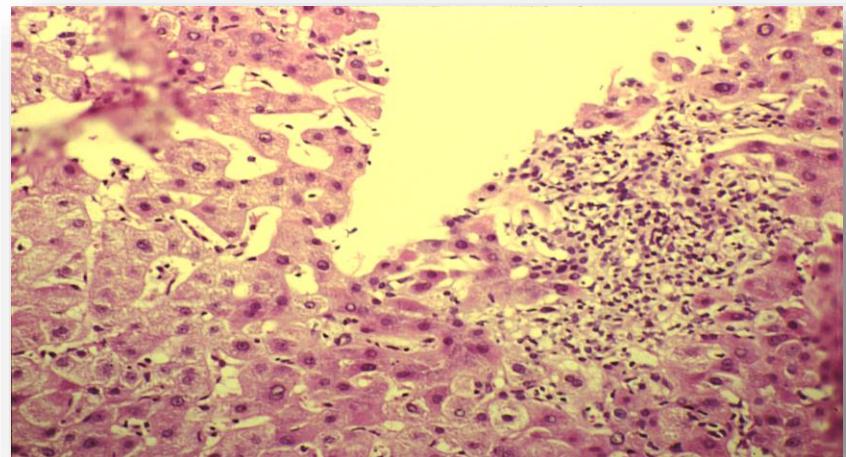
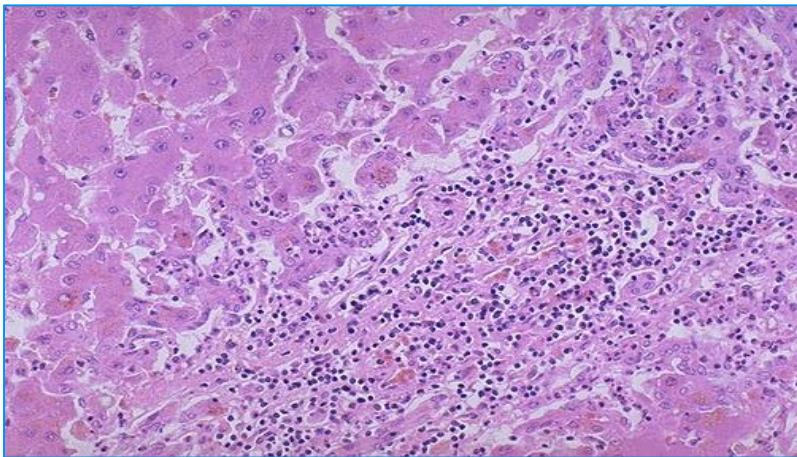
-Crowded malignant glands. -Decreased goblet cells. -Hyperchromatic nuclei. -invasion to the lamina propria.



Case 24- chronic viral hepatitis (HBV & HCV)

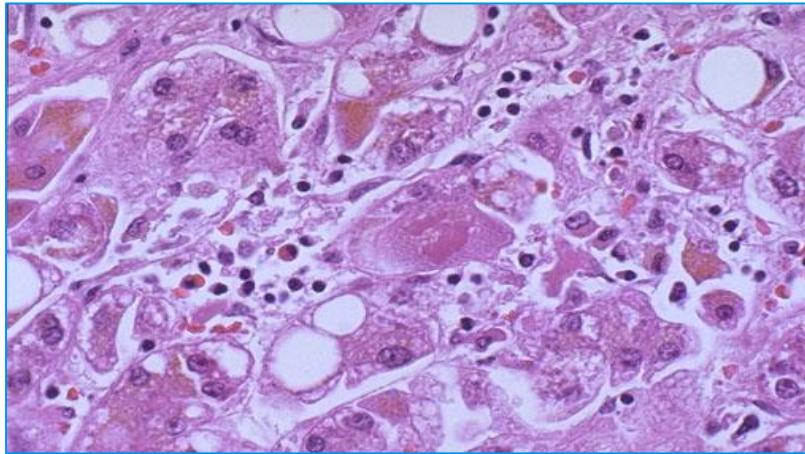


-pale hemorrhagic necrotic cut
surface area of the liver .

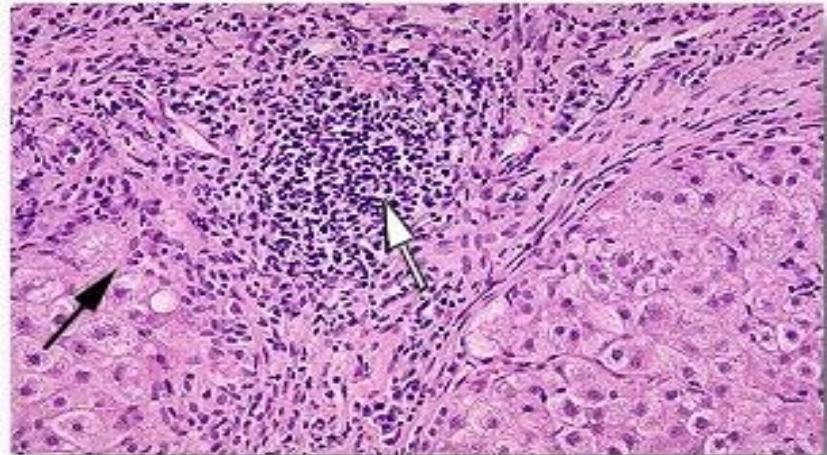


-chronic inflammatory cells. -piecemeal necrosis. -Individual hepatocyte necrosis.

Cont.. Chronic hepatitis

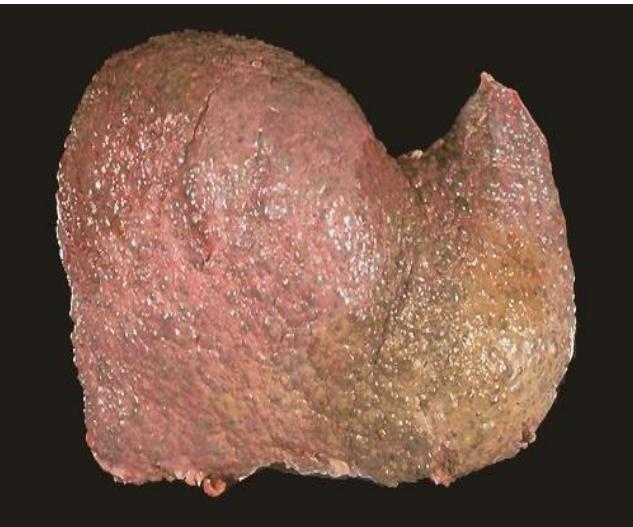


-Ballooning regeneration.
-chronic inflammatory cells.

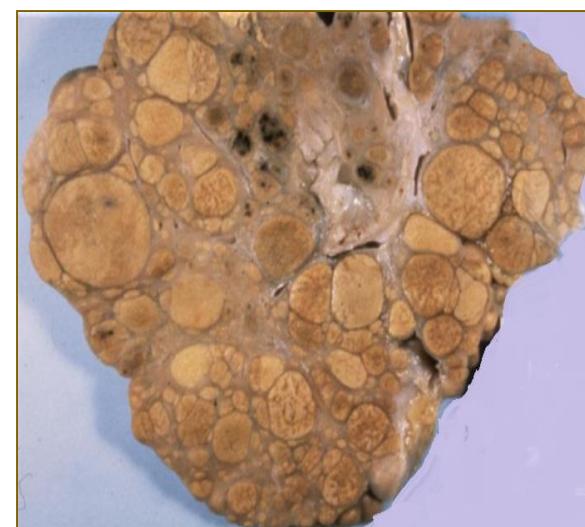


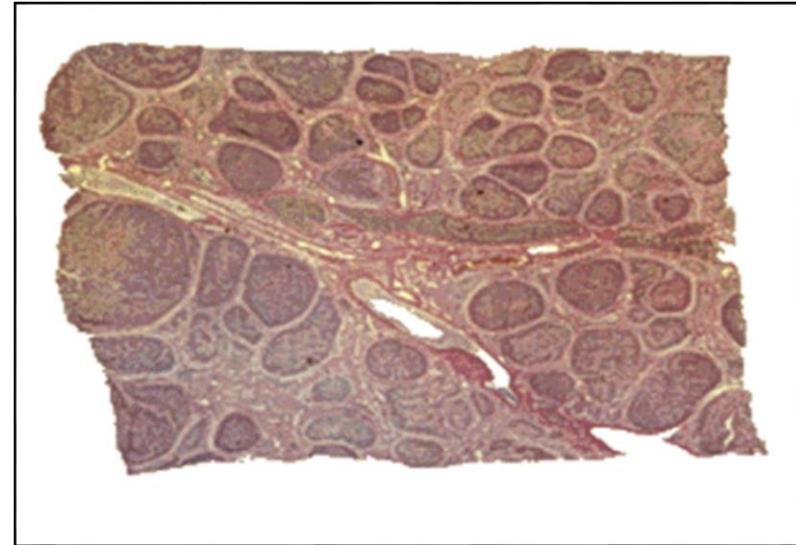
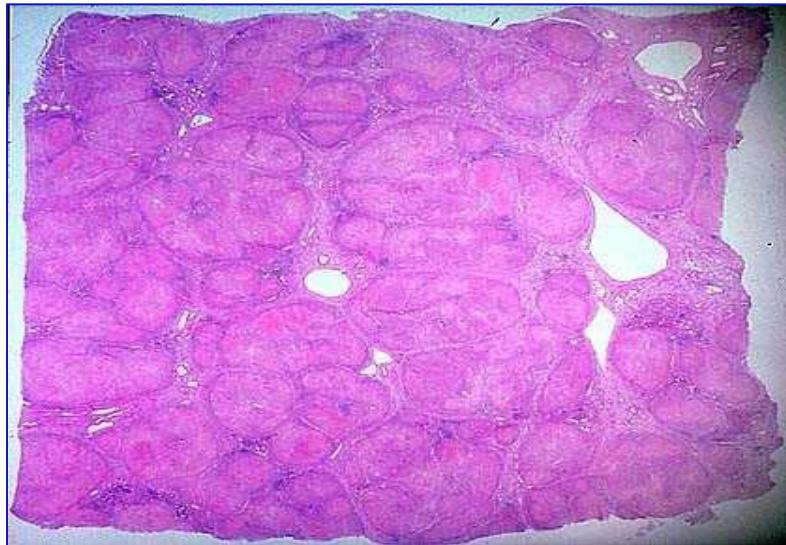
-More severe portal infiltrates with sinusoidal infiltrates.

Case 25- liver cirrhosis

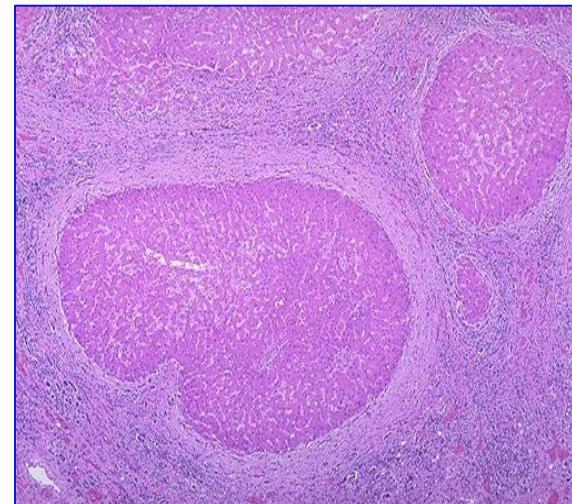
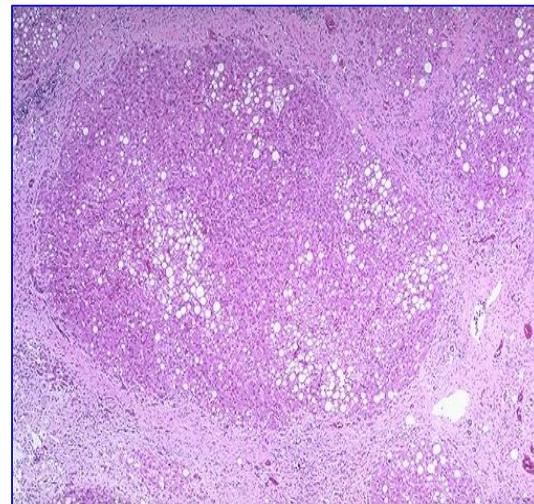
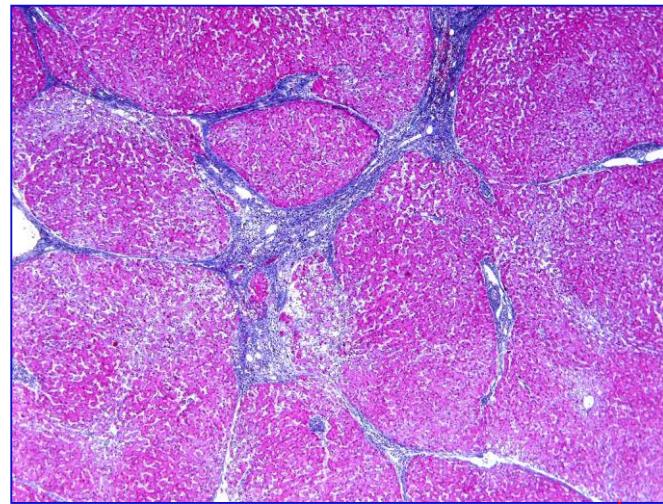


-Enlargement of the liver. -Nodular surface.





-Nodular arrangement. -separated by fibrous band.



-extensive blueish fibrosis around the nodules.

-Nodular regeneration of hepatocyte. -Surrounded by fibrosis

The stain in this slide **Masson's trichrome stain** (which make fibrosis appear blue)

Causes liver cirrhosis:

-chronic alcoholism. -hepatitis B & C.

Complication of liver cirrhosis:

-Portal HPT. -Liver failure. –HCC. –Hematemesis. -hepatic encephalopathy.

Case 26- hepatic adenoma



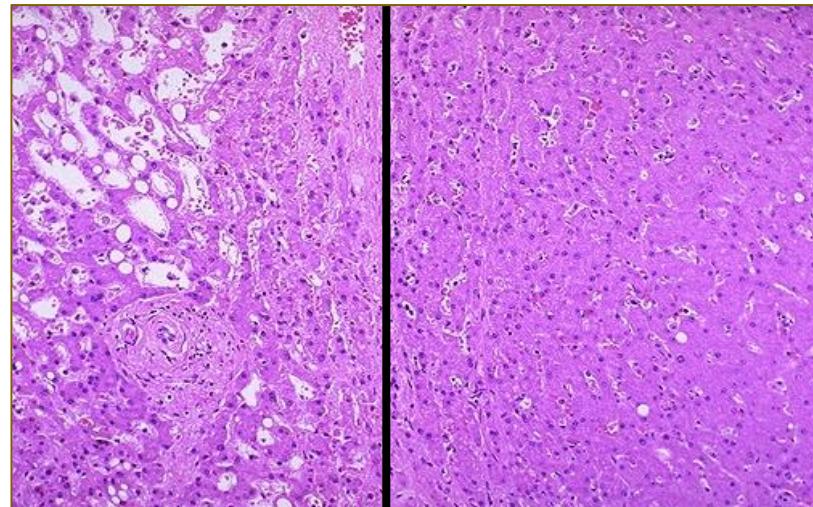
-Well-circumscribed mass.

-Normal liver tissue in the left.
-loss of lobular architecture on the right.



-well-circumscribed mass.
-pale yellow remaining liver.

***More common in female, associated with oral contraceptive.**



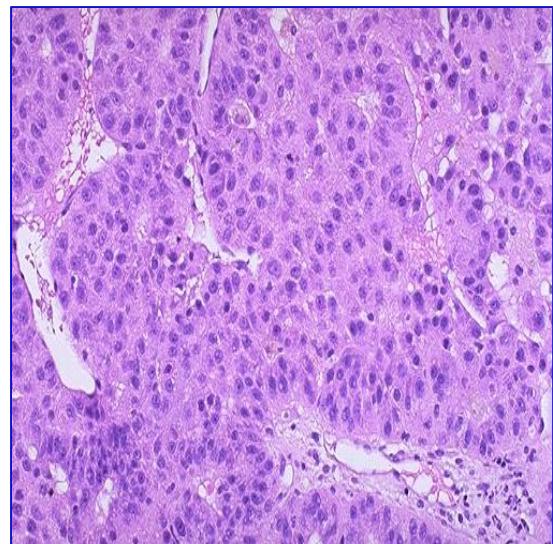
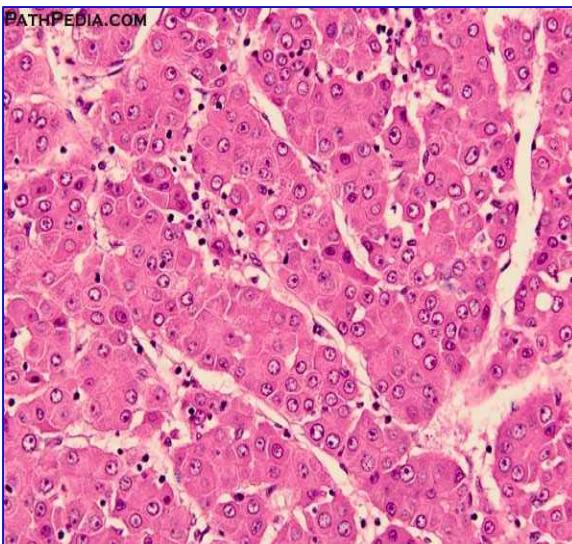
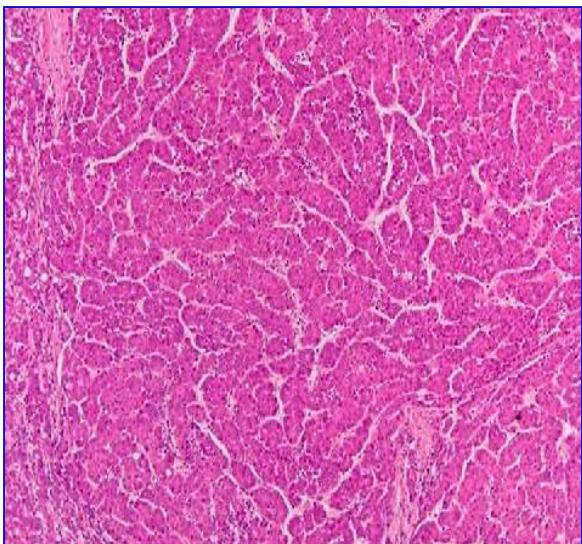
Case 27- Hepatocellular carcinoma



-well-circumscribed capsulated mass.
-pale hemorrhagic necrotic areas.

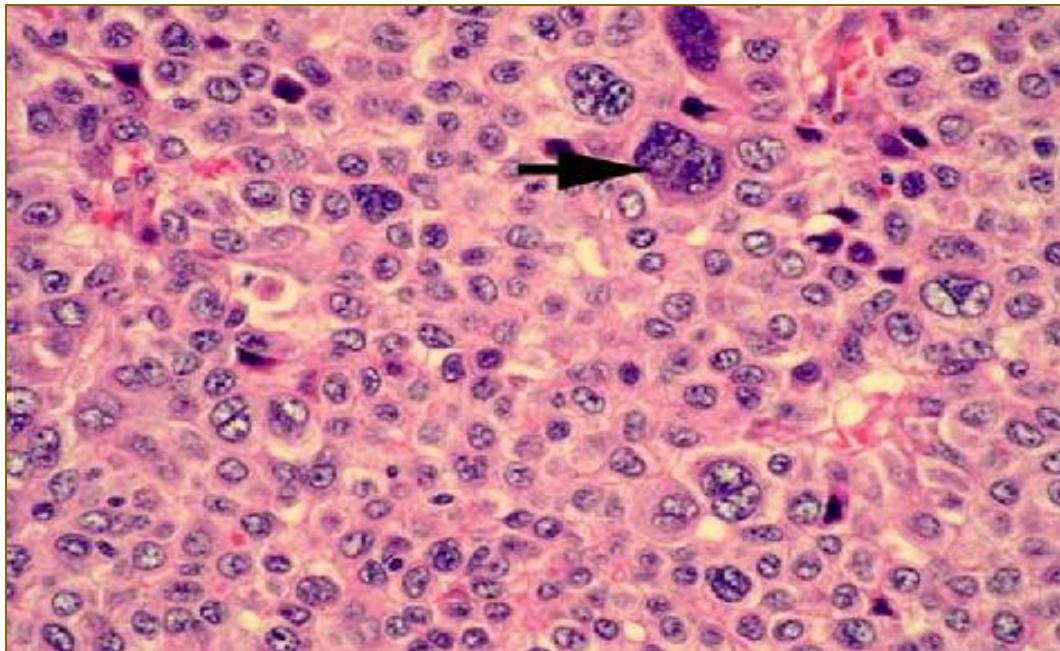


-hepatocellular carcinoma with greenish yellowish hue.



-Malignant Hepatocyte Cells -Pleomorphism. -Hyperchromatic. -Increase Mitosis

Cont.. Hepatocellular carcinoma



- Poorly differentiated.
- Malignant liver cells are:
 - Pleomorphic.
 - Forming giant cells (arrow).
 - Hyperchromatic nuclei.

Causes of HCC:

Most common causes: -Liver Cirrhosis -Viral Hepatitis (B,C) -Chronic Alcoholism.

Other etiological factors: -Alphatoxin. -Exposure. -Hemochromatosis. -Tyrosinemia.

* Associated with alpha-fetoprotein elevation.

THANK YOU