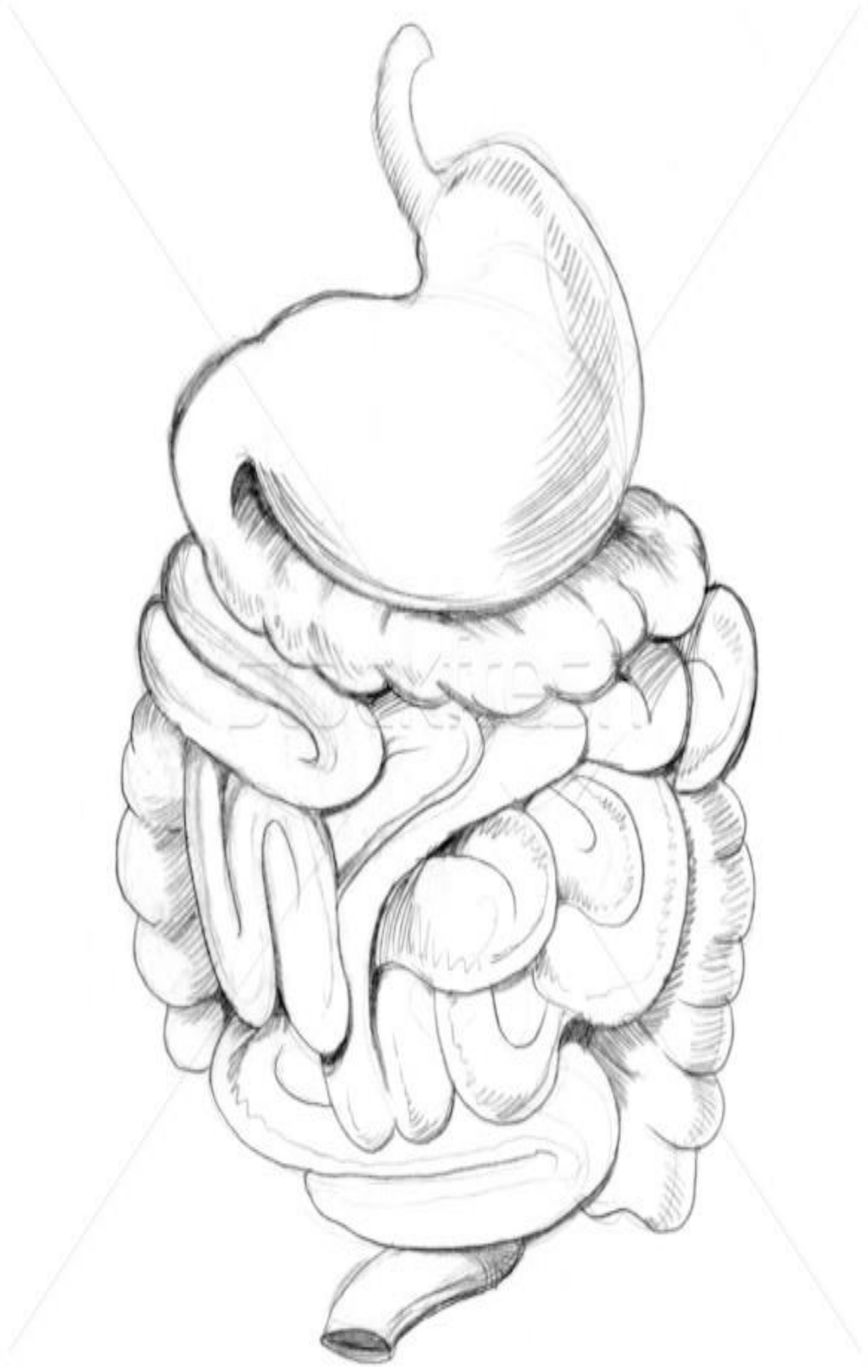
This work is based on Females notes and slides ONLY.



Pathology Practical

Med435.

Color codes:

Important

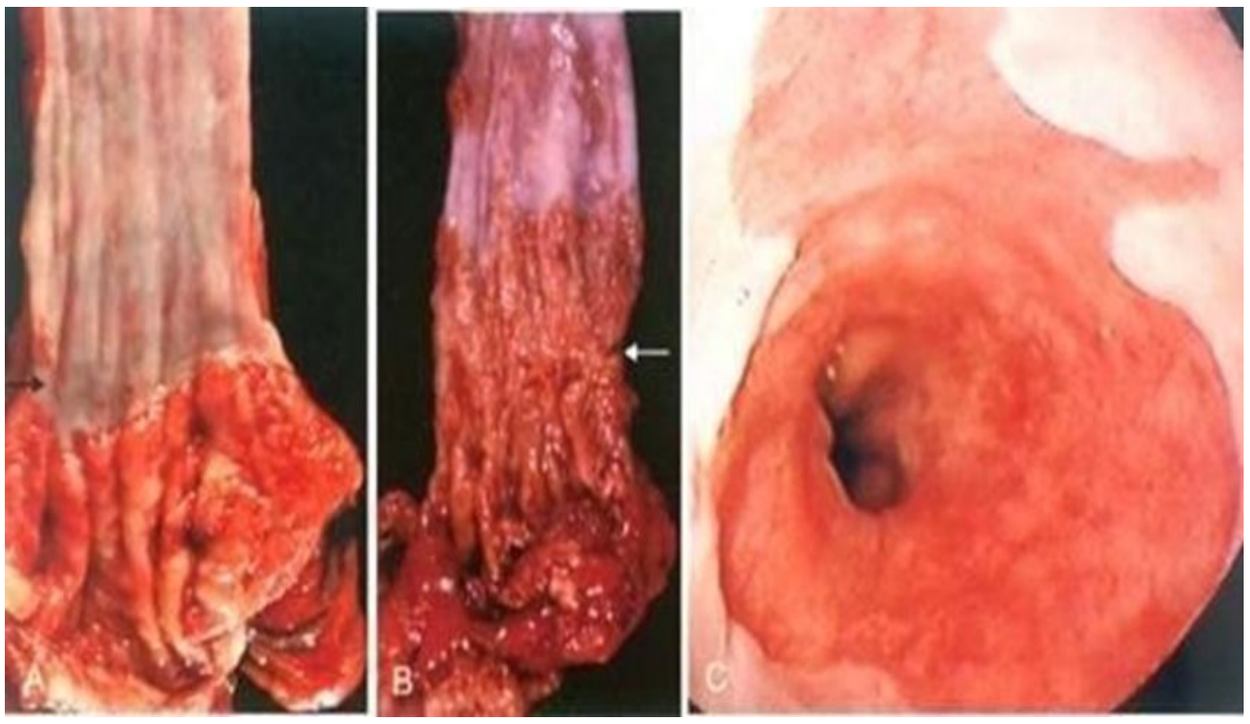
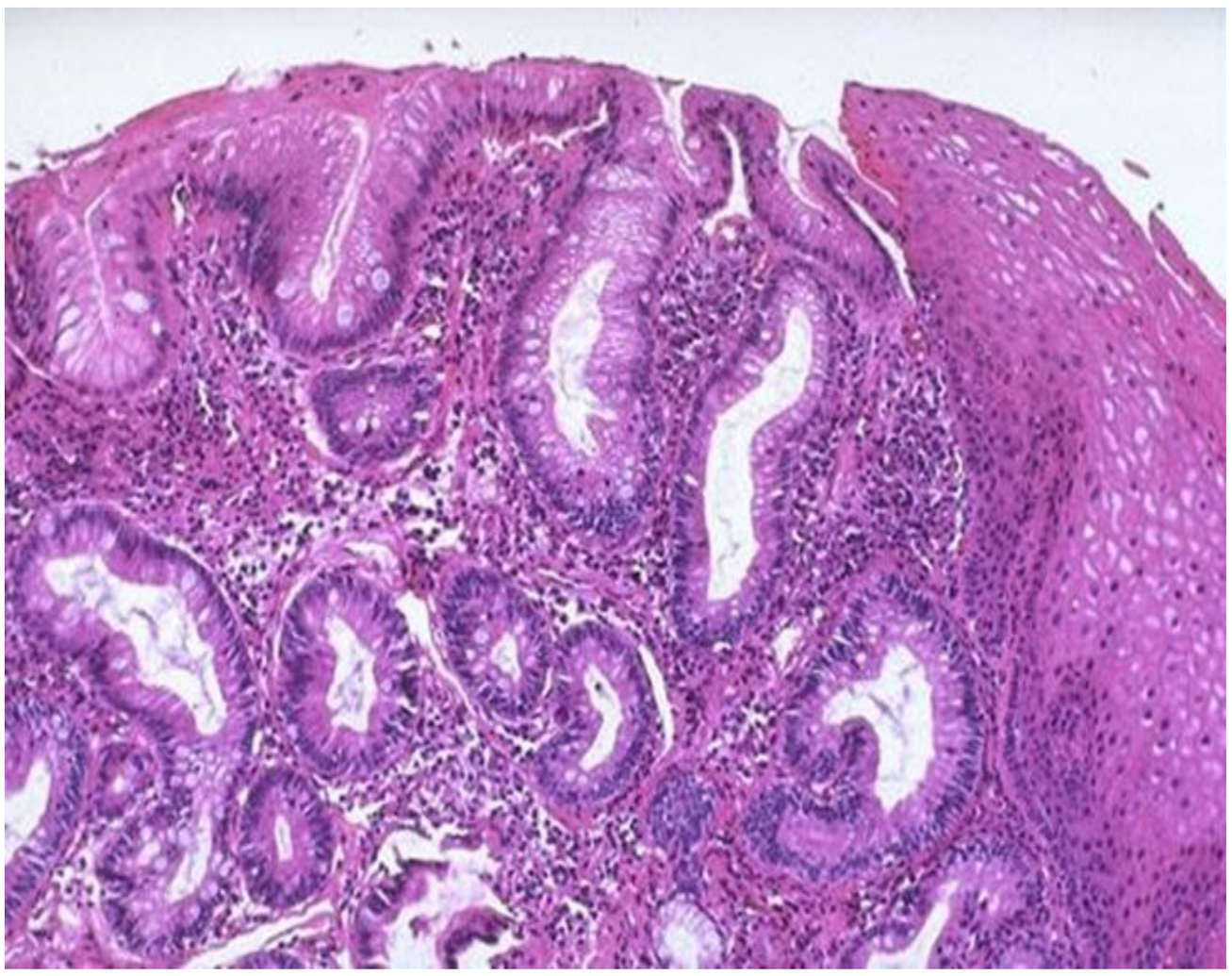
doctors notes

The doctor loves it.

Exam notes:
6 to 8 Cases in the exam.
Most important cases:
1-Barrett's esophagus
2-Celiac disease
3-Carcinoid tumor
4-Ulcerative colitis
5-Hepatic cirrhosis
6-Pancreatic ductal adenocarcinoma
7-Gastric adenocarcinoma

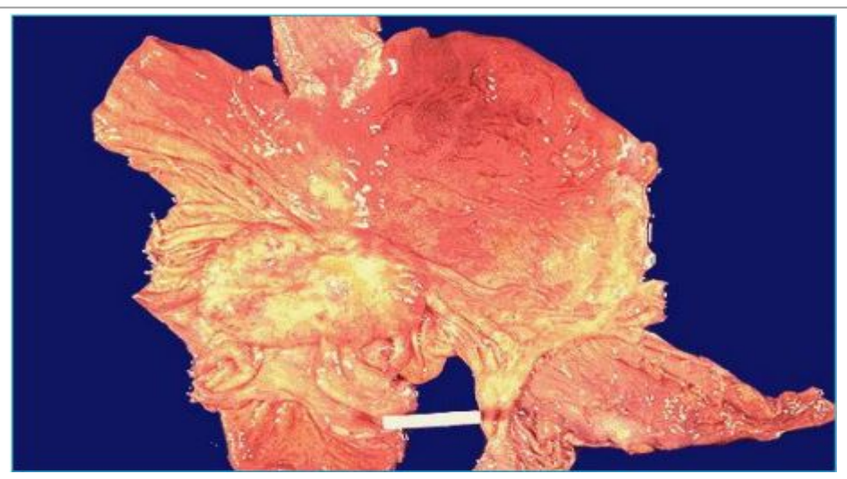
Case #1:BARRETT'S ESOPHAGUS ★

- Dysplasia is a pre neoplastic condition, If not treated can lead to adenocarcinoma.
- **Metaplasia: from Squamous cells to Columnar cells.**
- **Complication: Adenocarcinoma**

BARRETT'S ESOPHAGUS	**Barrett's Esophagus – Endoscopic view**
	
- Most/All adenocarcinomas arising in the esophagus arise from previously existing BARRETT's .	**1.Mucosal erythema and Redness** **2. Residual Pale-white squamous mucosa.**
Barrett's esophagus – Microscopic view	Glandular " Dysplasia" - HPF
	**1.**Chronically inflamed gastric type mucosa. **2. Intestinal metaplasia with goblet cells**. **3.** Columnar epithelium on the left and squamous epithelium to the right.

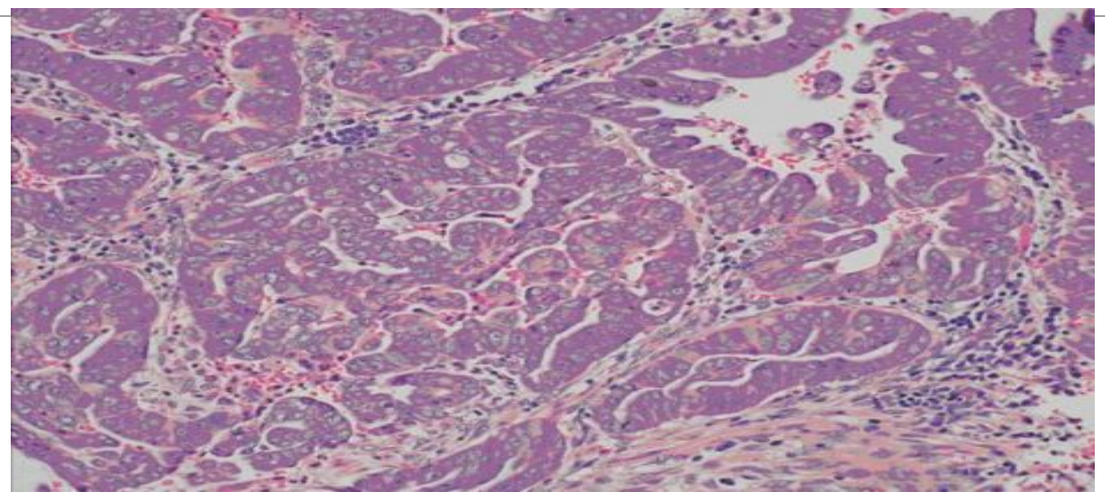
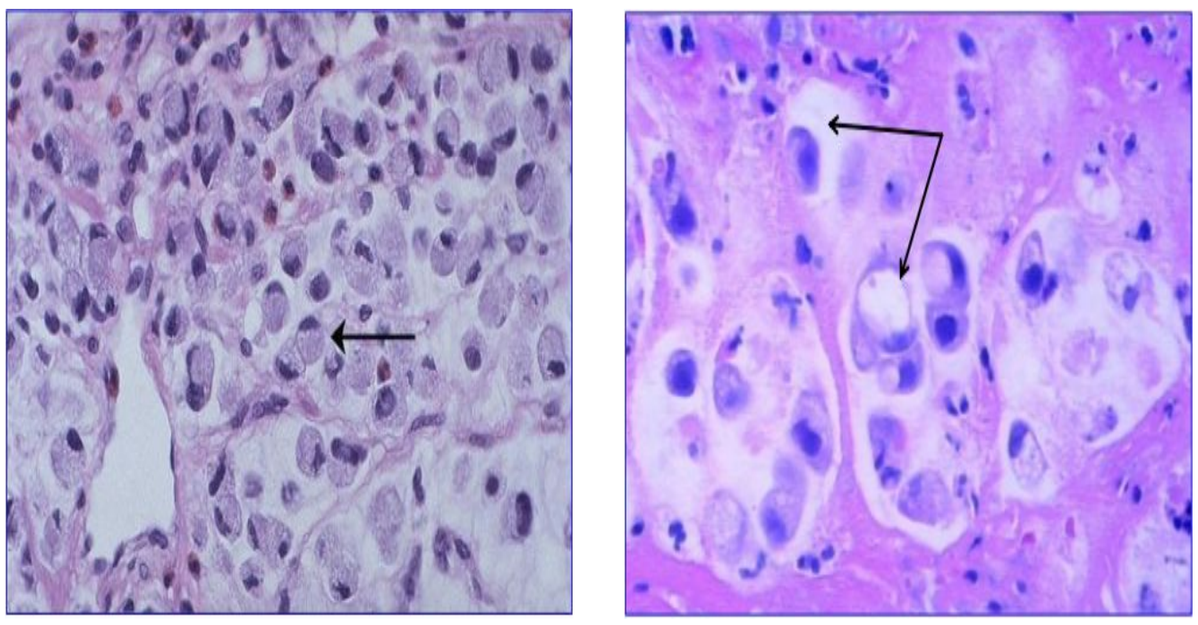
Case #2: Gastric Adenocarcinoma

Most common Cancer of the stomach: **Adenocarcinoma**
Gastric Neoplasia is not uncommon.
Linitis Plastica has a very poor prognosis!

Gastric Adenocarcinoma - Gross	Gastric Adenocarcinoma with ulcer - Gross
	
ALL gastric ulcers and ALL gastric masses must be biopsied, because it is not possible to tell from gross appearance alone which are benign and which are malignant.	1-Here is a gastric ulcer in the center of the picture. 2-It is shallow and is about 2 to 4 cm in size. 3-This ulcer on biopsy proved to be malignant, so the stomach was resected as shown here.

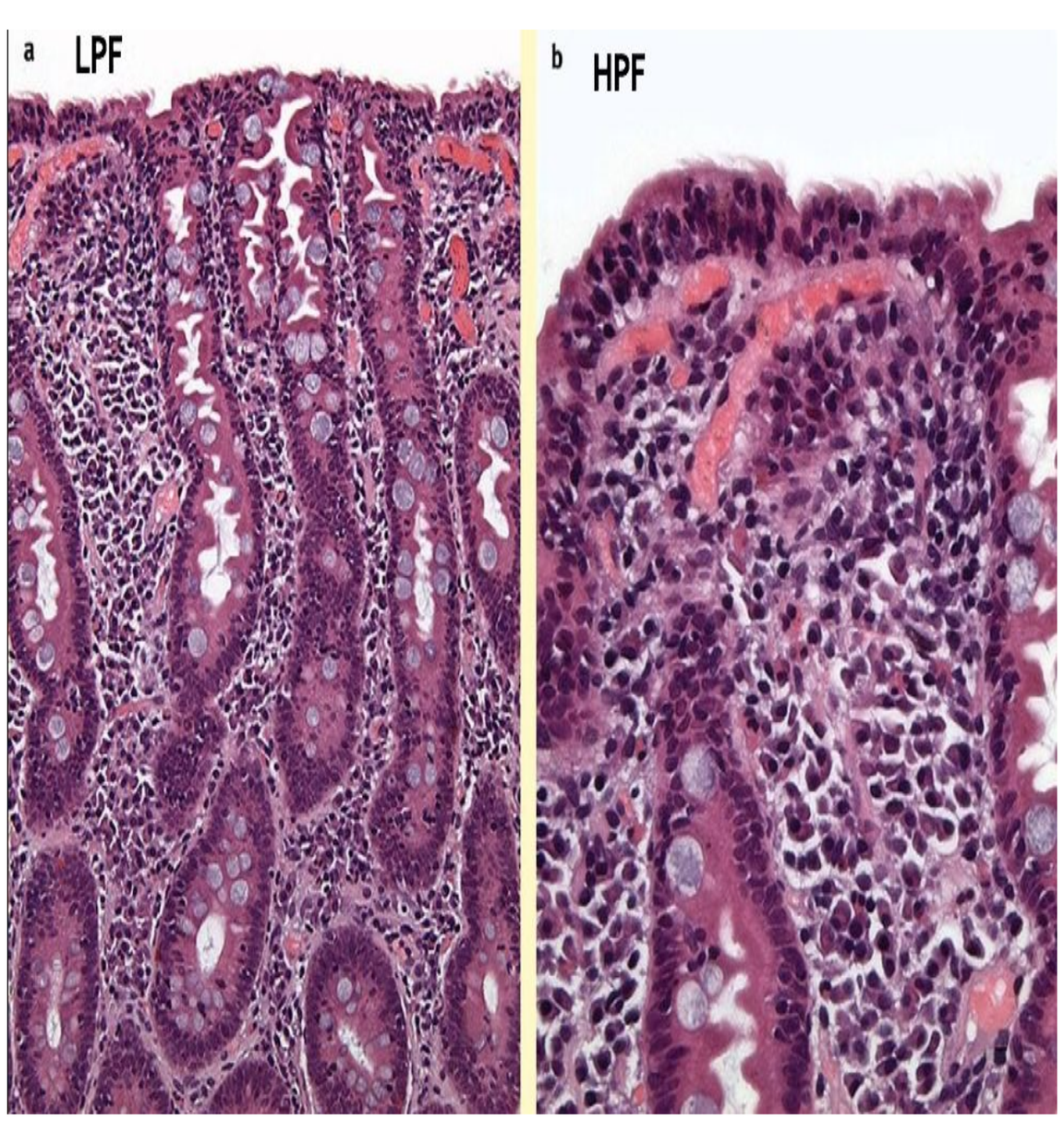
Gastric Adenocarcinoma ; **Linitis Plastica-** Gross

1.Diffuse infiltrative gastric adenocarcinoma which gives the stomach a shrunken "leather bottle" appearance.
2.It grows diffusely through all layers of the stomach
3. extensive mucosal erosion with markedly thickened gastric wall.

Gastric Adenocarcinoma- Intestinal Type	Gastric Adenocarcinoma- Signet Ring Cell -HPF
	
Photomicrograph of a poorly differential intestinal type adenocarcinoma of the stomach	**1.Signet ring cells** are poorly differentiated adenocarcinoma cells, and are often seen with Linitis Plastica.

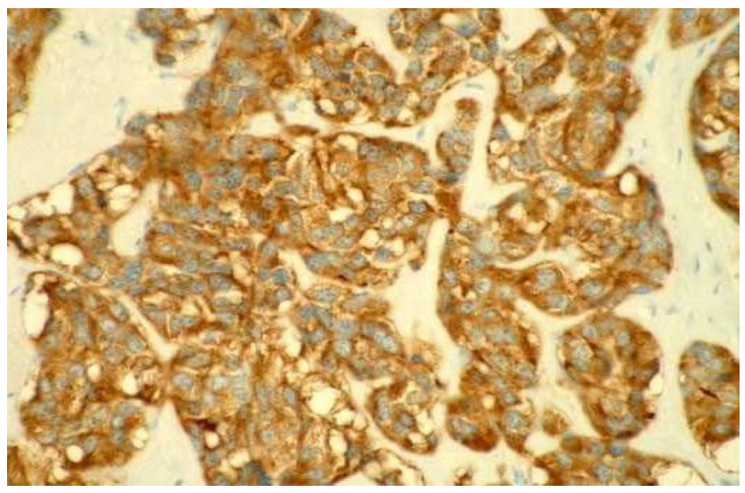
Case #3:Celiac Disease ★

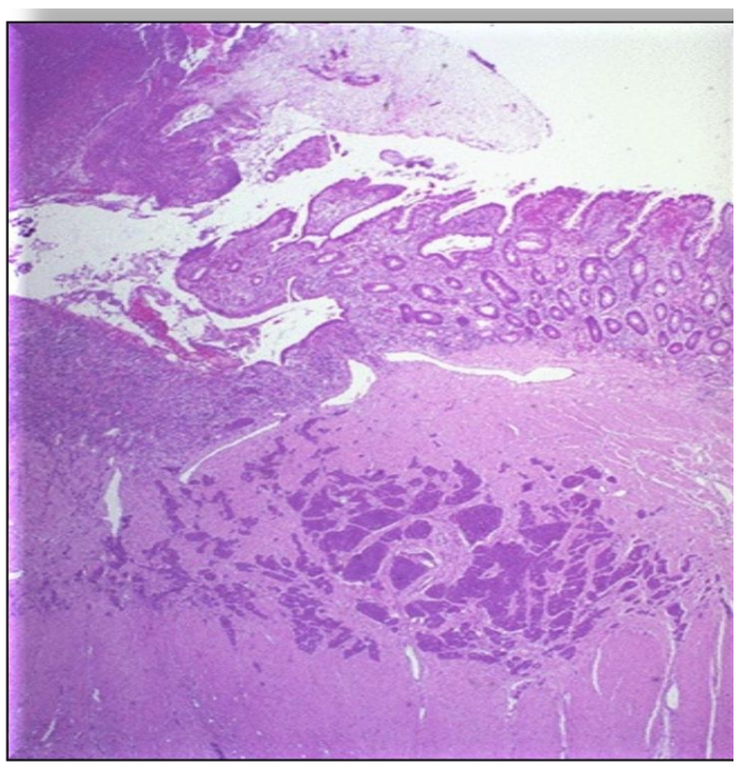
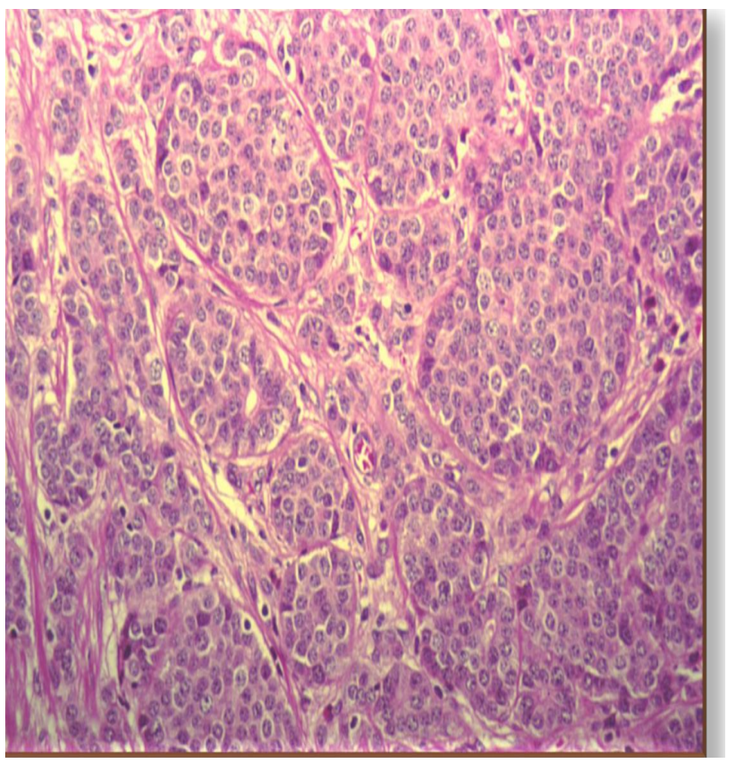
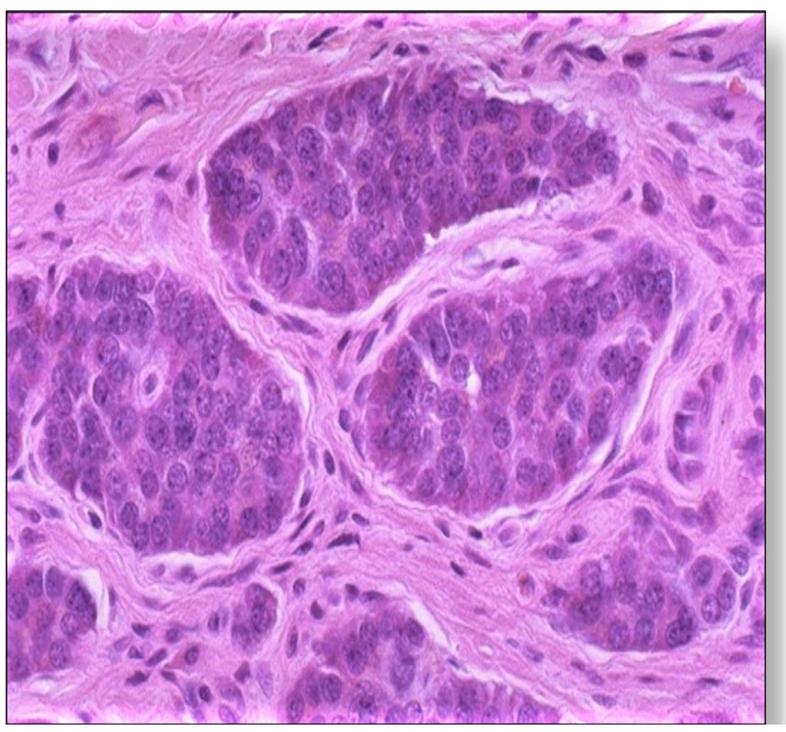
Most common benign disease of duodenum? Celiac disease.
Celiac disease most often becomes apparent either in infancy, or in young to middle age adults.
confirmed by Biopsy in addition to serology.
-Patient usually present with: Diarrhea, Steatorrhea.
-Serology done to confirm diagnosis: Anti-gliadin antibodies - Anti-enterocytes antibodies
-Major types of diarrheal disease in general:
a - Exudative.
b - Secretory.
c- Osmotic.
d- Deranged motility.
e- Malabsorption related. **Like in celiac disease.**

Normal vs Celiac Disease (Sprue) **– LPF**	***Celiac Disease (Sprue)*** **– LPF & HPF**
	
Normal small intestinal mucosa is seen at the left. The mucosa involved by celiac disease (sprue) at the right has blunting and flattening of villi.	**1.Elongated mucosal crypts** **2.Complete lack of villi (villi atrophy)** **3.Increased intraepithelial Lymphocytes.** **4.Inflammation of lamina propria**

Case #4:Carcinoid Tumor of Small intestine

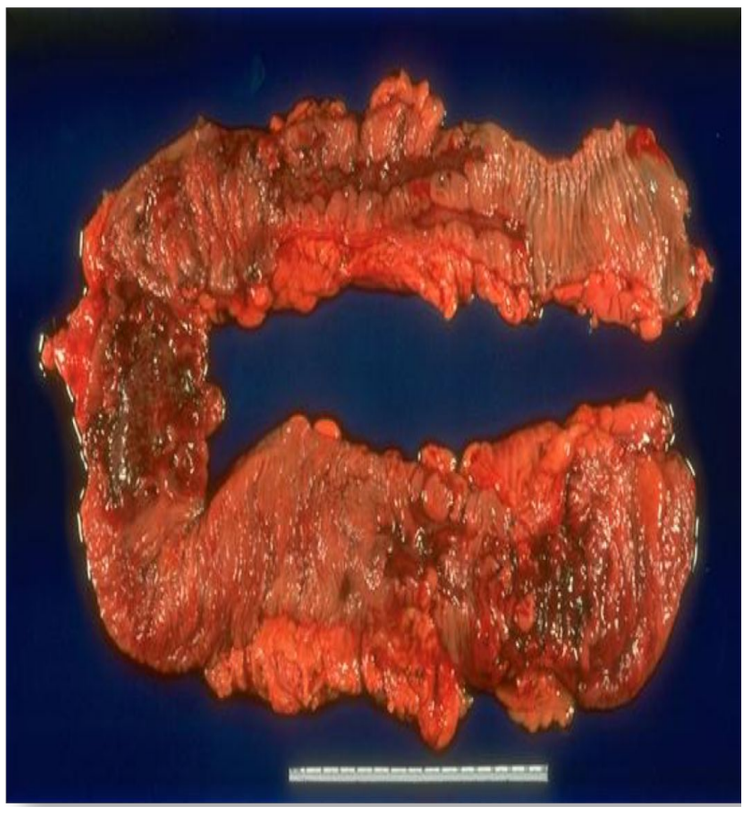
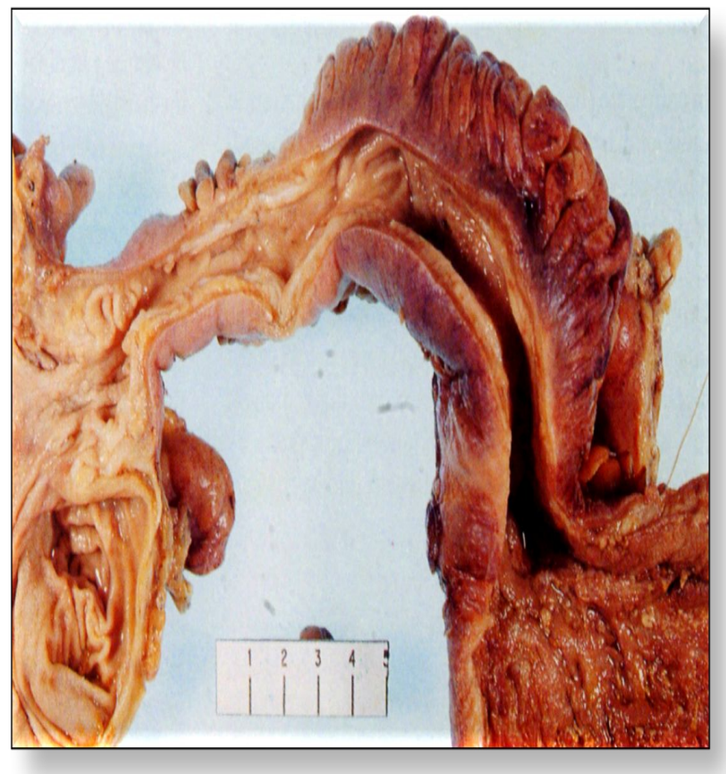
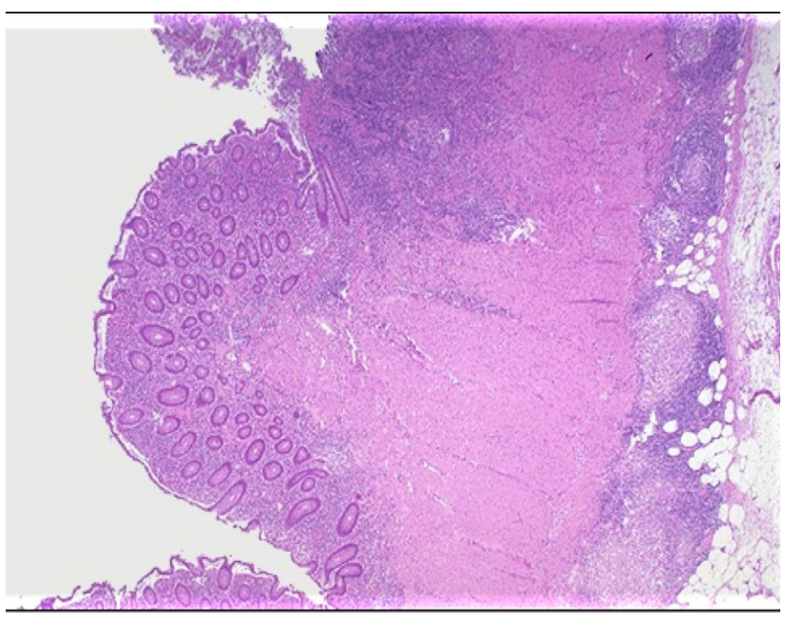
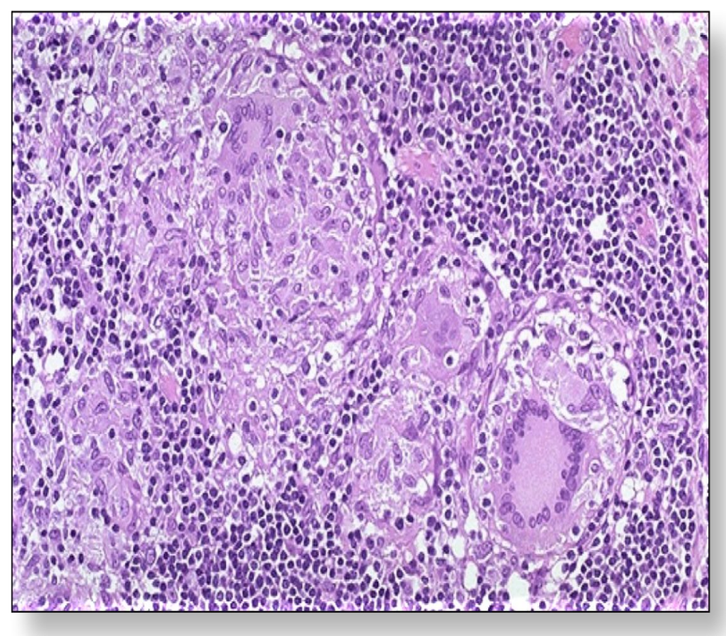
Most common type of small intestine tumor: carcinoid. "a benign neuroendocrine tumor"
Neoplasms of the small intestine are uncommon. Benign tumors can include leiomyomas, fibromas, neurofibromas, and lipomas.
Metastatic carcinoid to the liver can rarely result in the **carcinoid syndrome**.
The patient with **carcinoid syndrome** which can cause:
a- Cutaneous flushes. b- Asthmatic attacks. c- Diarrhea, nausea, vomiting.
d- Signs and symptoms related to pulmonary and tricuspid valve stenosis.

Carcinoid tumor of small intestine - Gross	Carcinoid tumor of small intestine – IHC stain
 faint **yellowish** tumor	 strong positive staining with the **synaptophysin** immunohistochemical stain (IHC).

Carcinoid tumor of small intestine - LPF	**Carcinoid tumor of small intestine - MPF**	**Carcinoid tumor of small intestine - HPF**
 **1.** mass of multiple nests of small blue cells in the submucosa	 **1. A group of neoplastic cells** **2.** Abundant cytoplasm **3.** Nucleus showing s**alt and pepper** chromatin.	 **1.**Group and clusters of tumor cells. **2.**Small uniform nuclei showing salt and pepper chromatin. **3.**Granular cytoplasm.

Case #5: Crohn's disease

Crohn's disease is a chronic inflammatory condition of the GI tract.

Gross	Gross	Gross
		
1.Large, irregularly shaped to rake-like ulcers which we describe as skipping lesion (patchy).	Mucosal surface demonstrates an irregular nodular appearance with hyperemia and focal superficial ulceration	Section of large bowel shows alternating normal and ulcerating mucosa
Histopathology	**Histopathology**	**Histopathology**
		
1.transmural inflammation. Here, inflammatory cells (the bluish infiltrates) with pale **2.**granulomatous centers (on the left). **3.**lymphoid aggregates and mild fibrosis.	the granulomatous nature of the inflammation here: 1-epithelioid cells. 2-giant cells. 3-lymphocytes. Special stains for organisms are negative .	

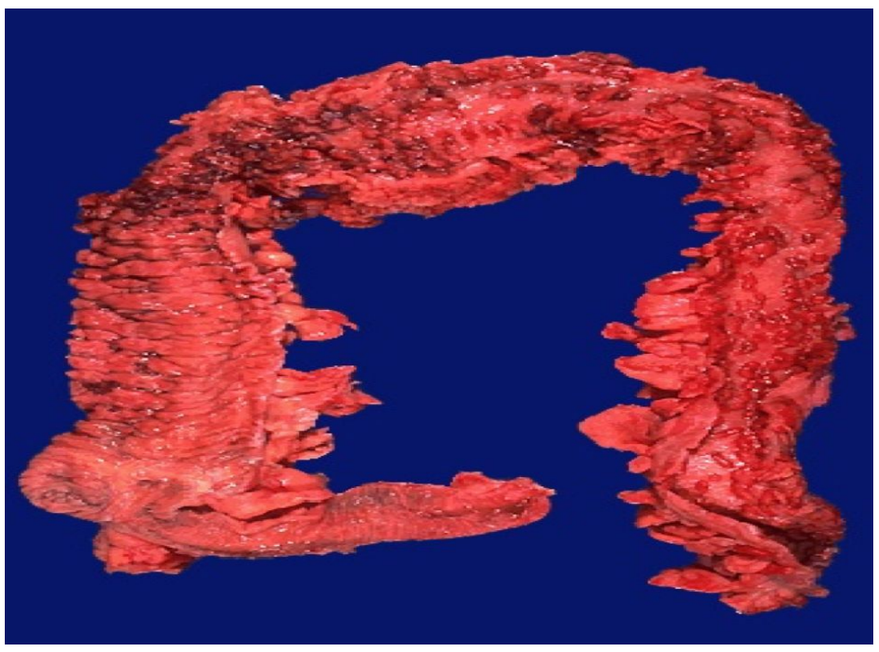
Case #6:Ulcerative colitis

The most intense inflammation begins at the sigmoid colon and extends upward and around to the ascending colon .

UC complications :

1- increase risk of carcinoma and adenocarcinomas. 2-toxic megacolon and glandular dysplasia. 3-haemorrhage. 4 -perforation and peritonitis. 5-electrolytes imbalance due to diarrhea

Gross

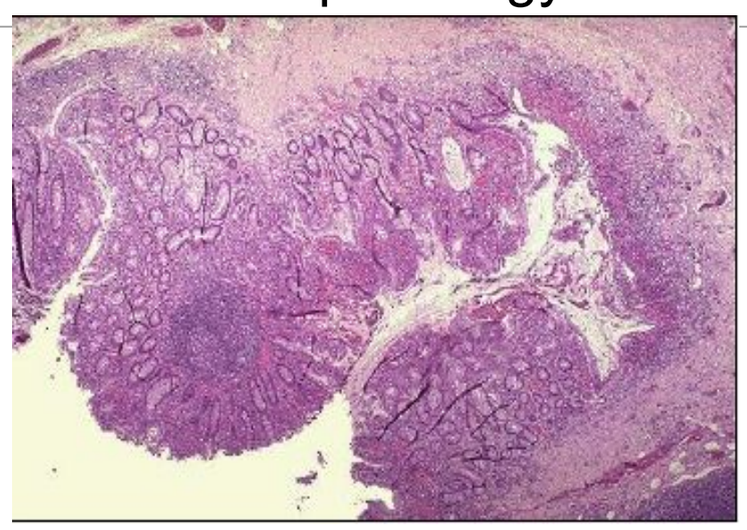


ileocecal valve with a portion of terminal ileum that is not involved

Gross

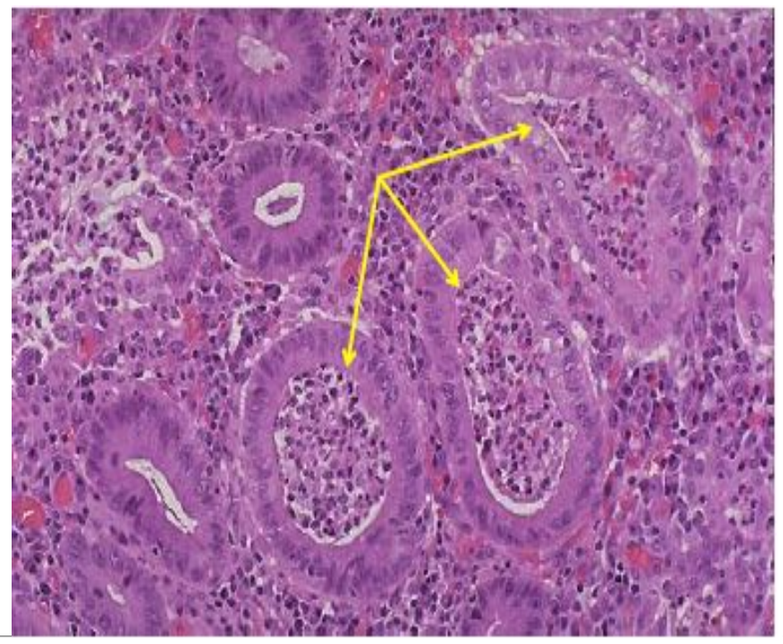
Dilated ulcerated colon showing numerous inflammatory pseudopolyps.

Histopathology

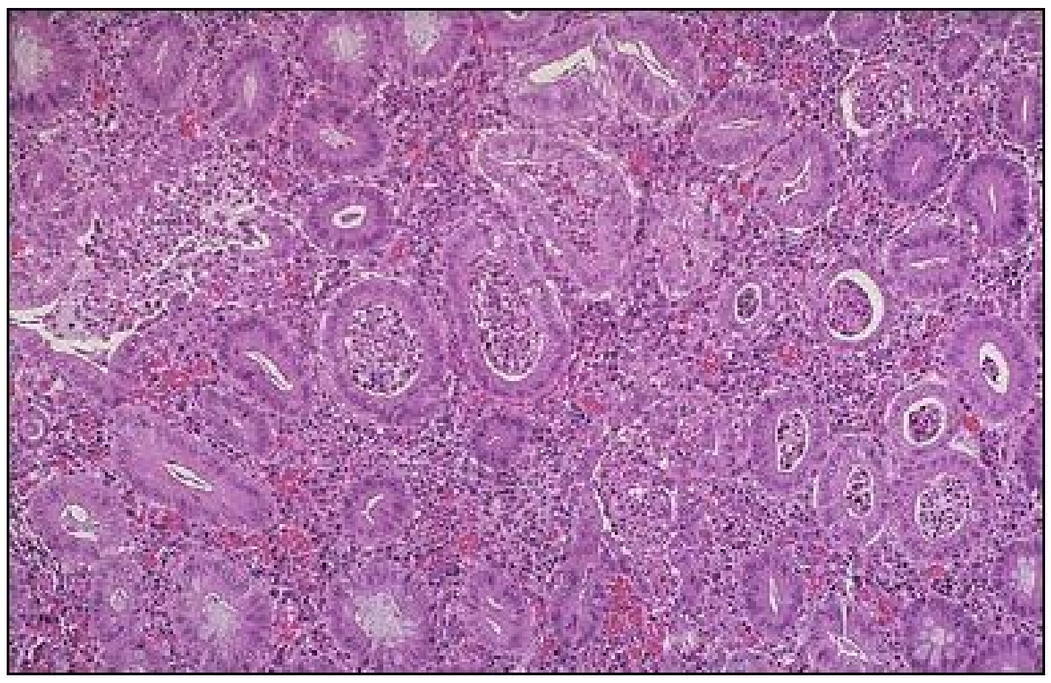


The inflammation is confined primarily to the mucosa.

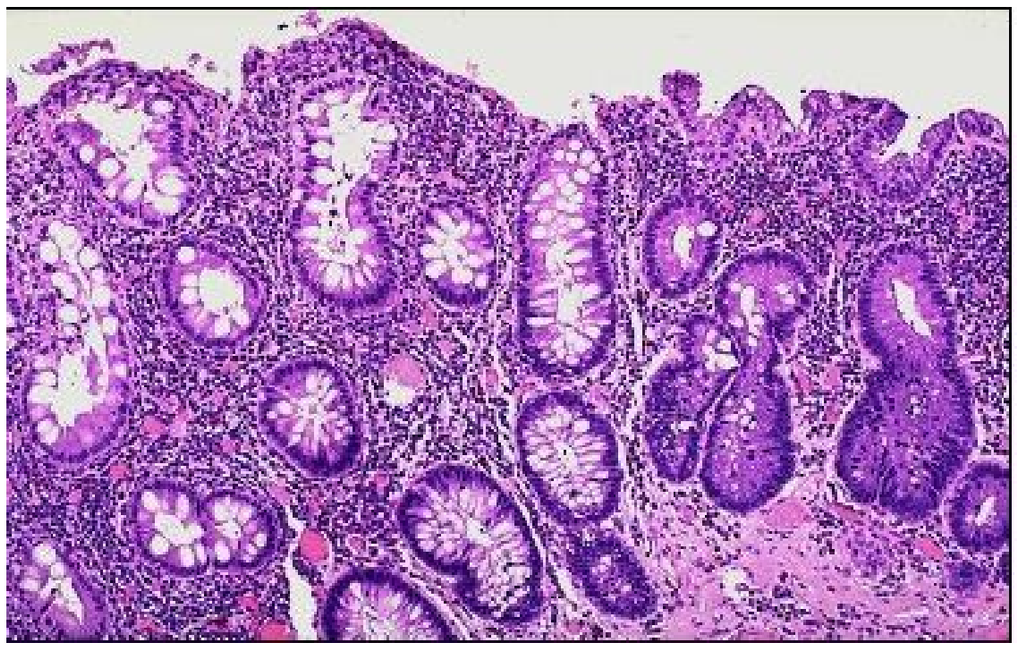
Histopathology



Crypt abscesses: histologic finding more typical with ulcerative colitis.



a- Crypt abscesses. b- Goblet cells depletion.
c- Marked acute on chronic inflammation in lamina propria.



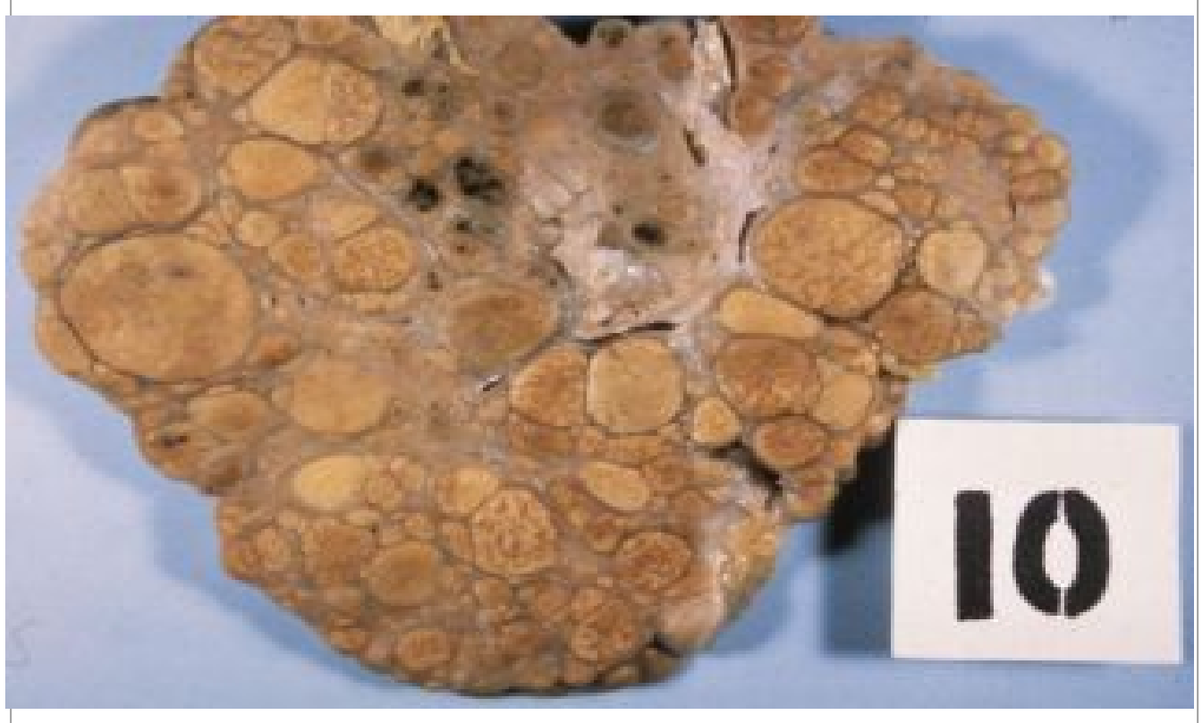
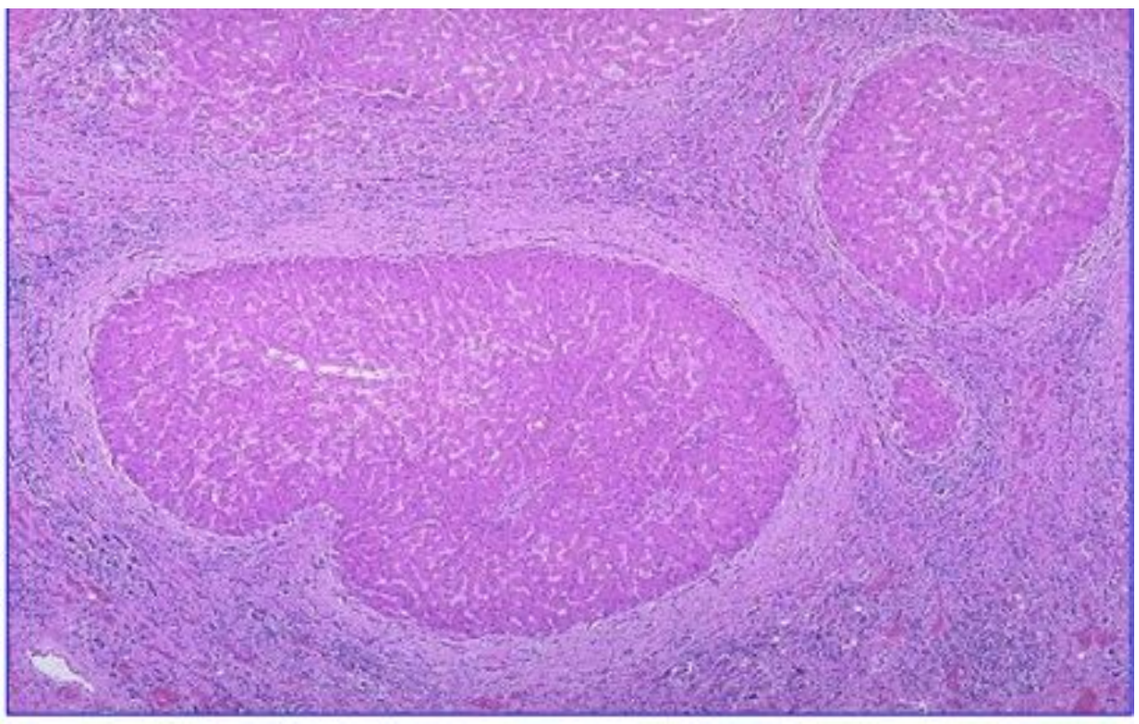
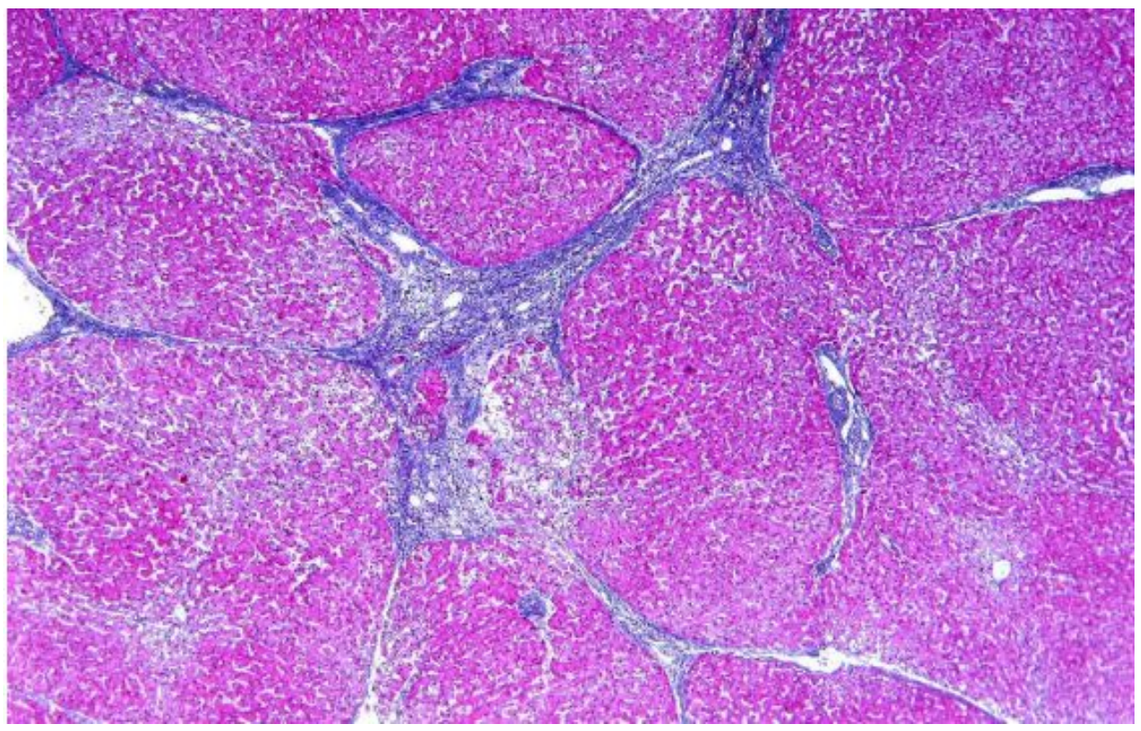
glands at the right demonstrate dysplasia, the first indication that there is a move towards neoplasia.

Case #7: hepatic cirrhosis

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 **no central vein.**

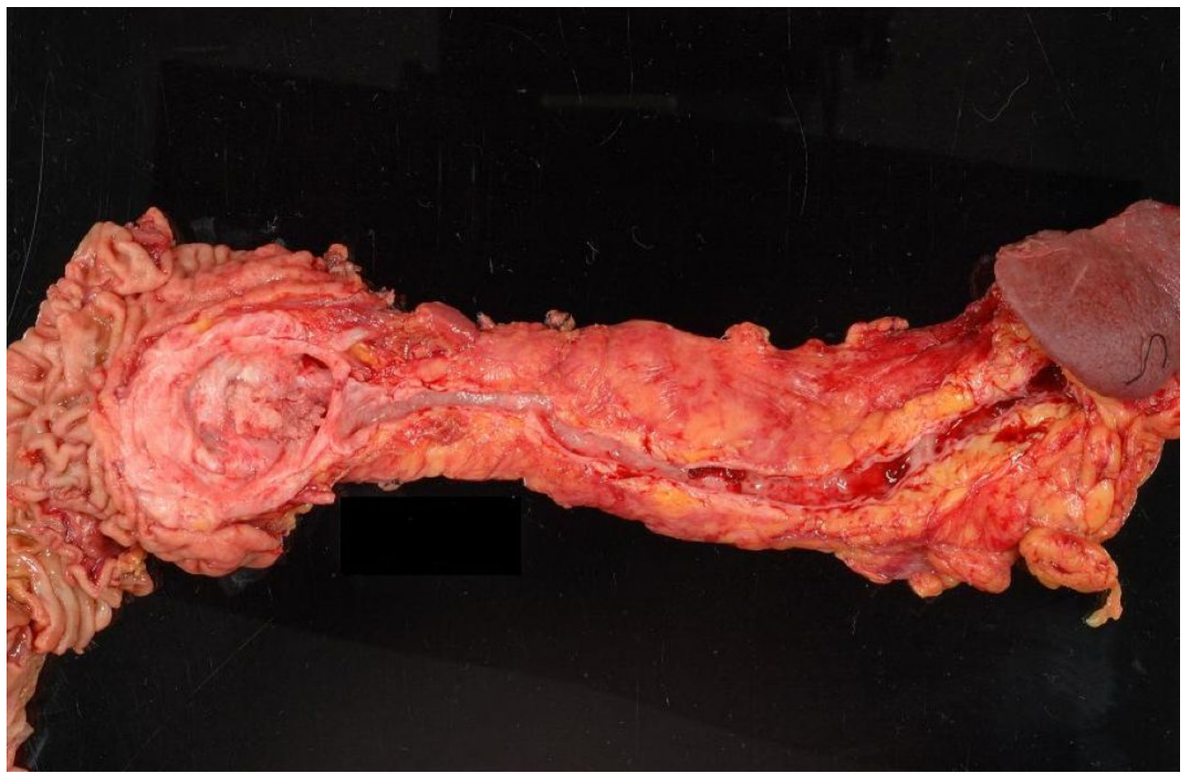
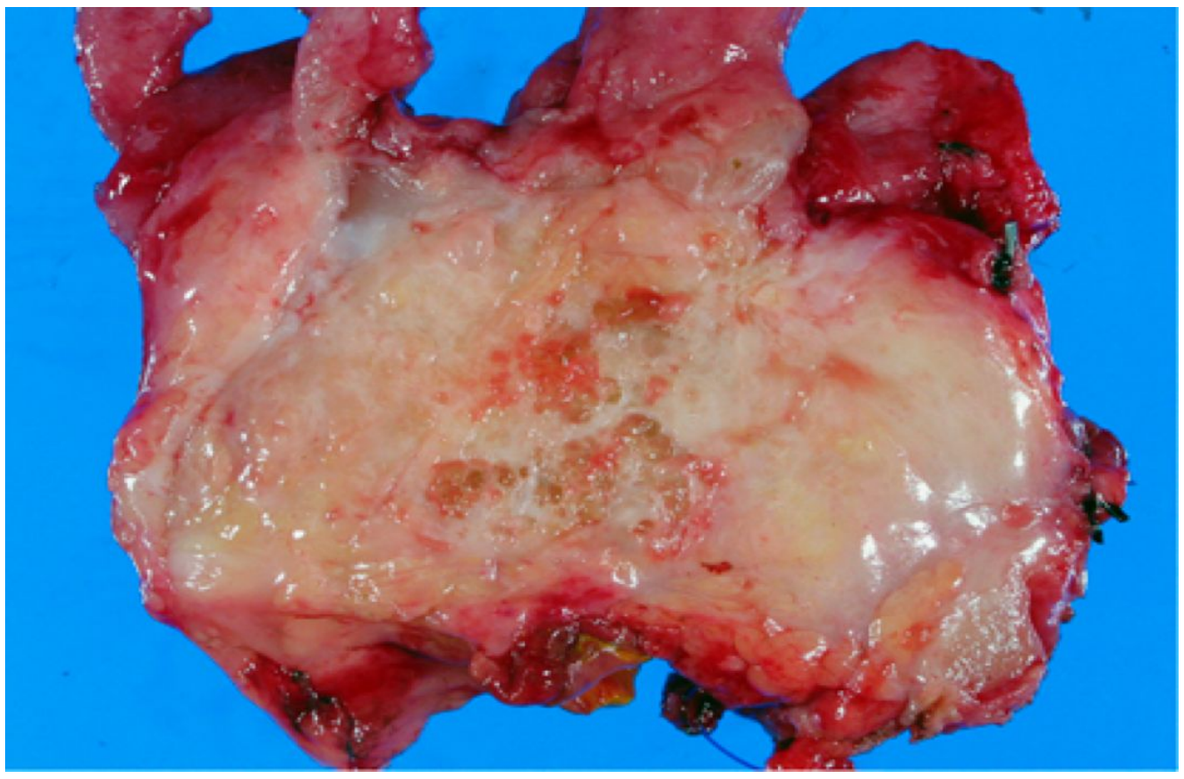
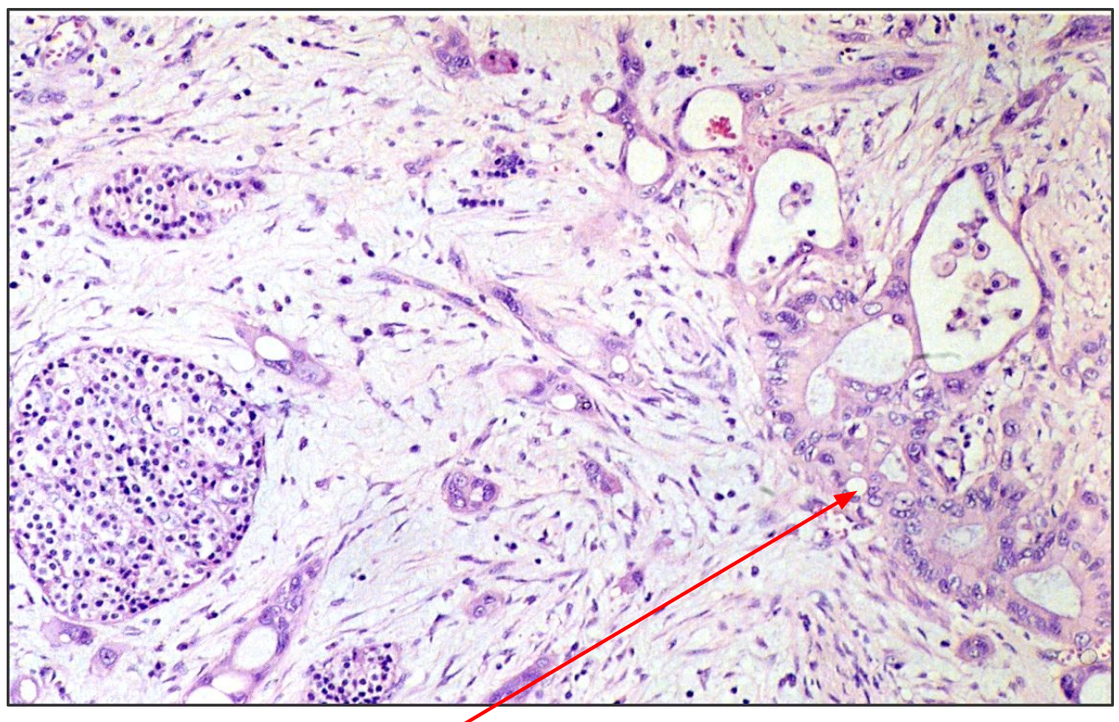
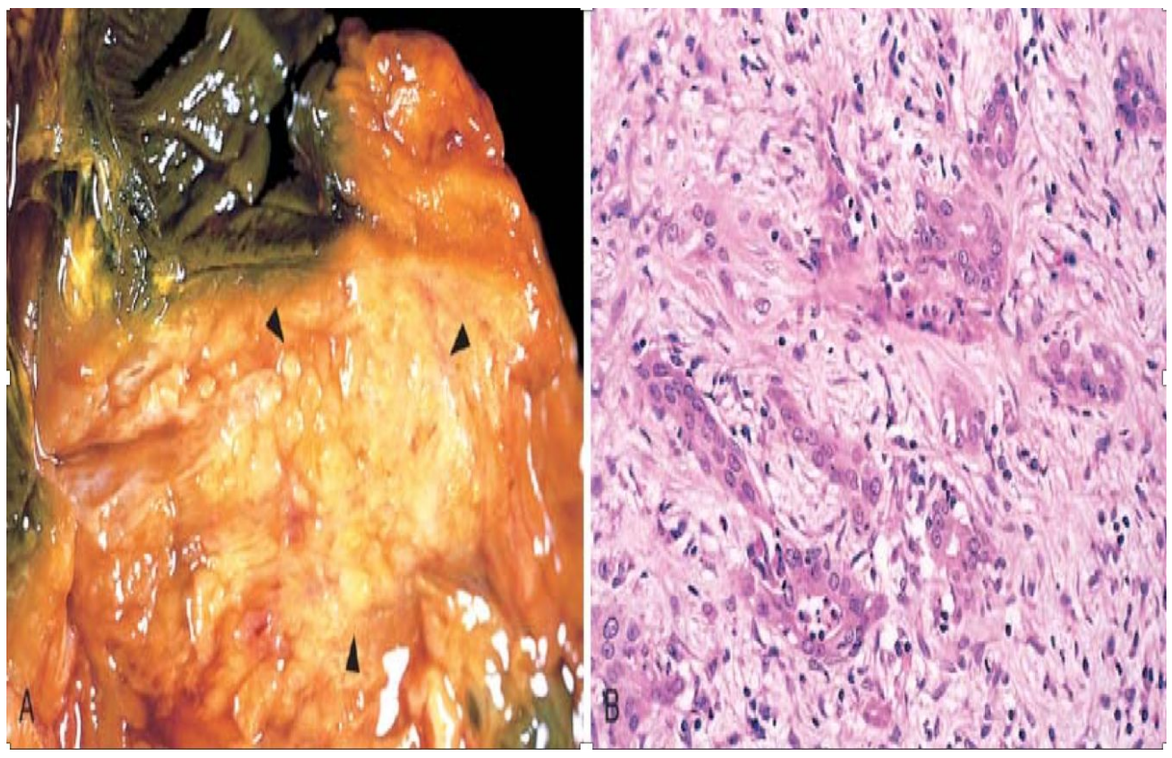
- **Complications that can occur in cirrhosis following portal HTN:**

a- Ascites. b- Oesophageal varices. c- Hepatic encephalopathy. d- Caput medusa. e- Splenomegaly. f. Hemorrhoids. g. Malnutrition. h. Skin spider angiomata.

Gross	Gross
 **1.**Hepatic nodules of variable sizes **2.** Fibrous bands between modules.	 Nodular liver surface
Histopathology	Histopathology
 **1.**Regenerative hepatocytes nodules. **2.** Fibrous bands between the regeneration nodules. **3.**Proliferating bile ducts and chronic inflammatory cells within the fibrous bands.	 **MASSON'S TRICHROME STAIN** showing: Fibrosis around liver nodules

Case #8: pancreatic adenocarcinoma

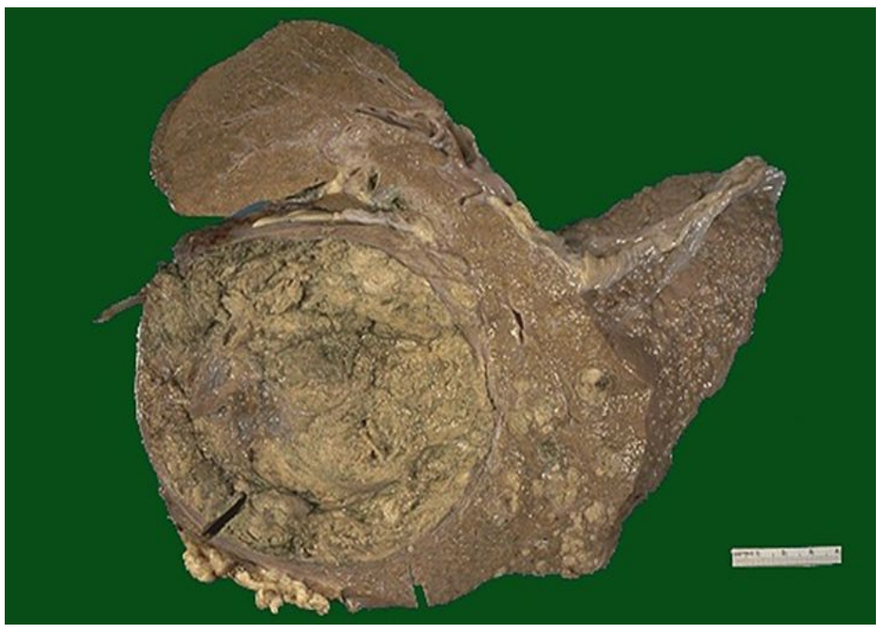
- **Treatment:** whipple procedure, Removal of the pancreas, and part of the duodenum.
- **Genes: a- KRAS. b- P16 c- TP53 d- SMAD4**

PANCREATIC ADENOCARCINOMA – Gros	Gross
	
1.well circumscribed tumor nodule at the head of pancreas. **2.**dilated main pancreatic duct. **3.**Part of the duodenum is seen on the left and the spleen on the right side	**Ill-defined or poorly circumscribed pale and firm tumor mass most probably at the head of pancreas.**
Histopathology	Histopathology
	
1.Malignant glands lined by atypical cells. **2.Desmoplastic fibrous tissue reaction around tumor cells**	Gross picture shows ill defined pale and firm pancreatic mass (left). Microscopic picture shows malignant glands or acini surrounded by desmoplastic fibrous stroma (right) .

Case #9:Hepatocellular carcinoma

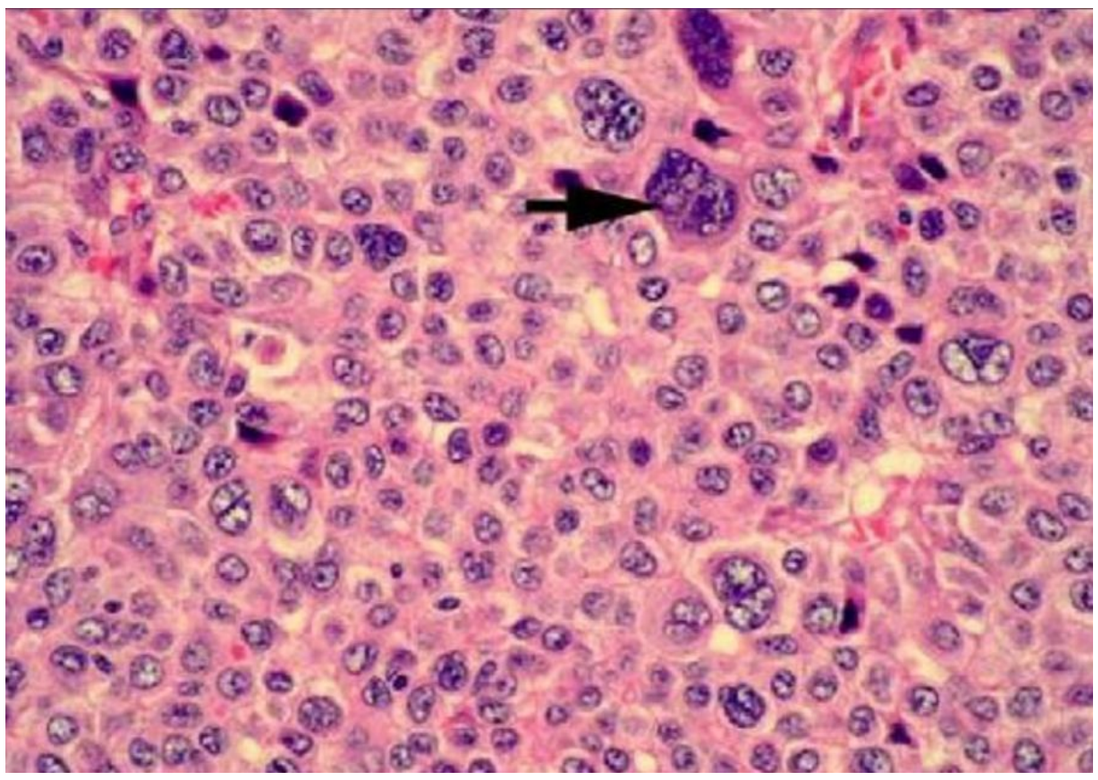
- **Aetiologic factors which leads to HCC:** Hepatitis B or C - Chronic alcoholism - Aflatoxin exposure.

Gross



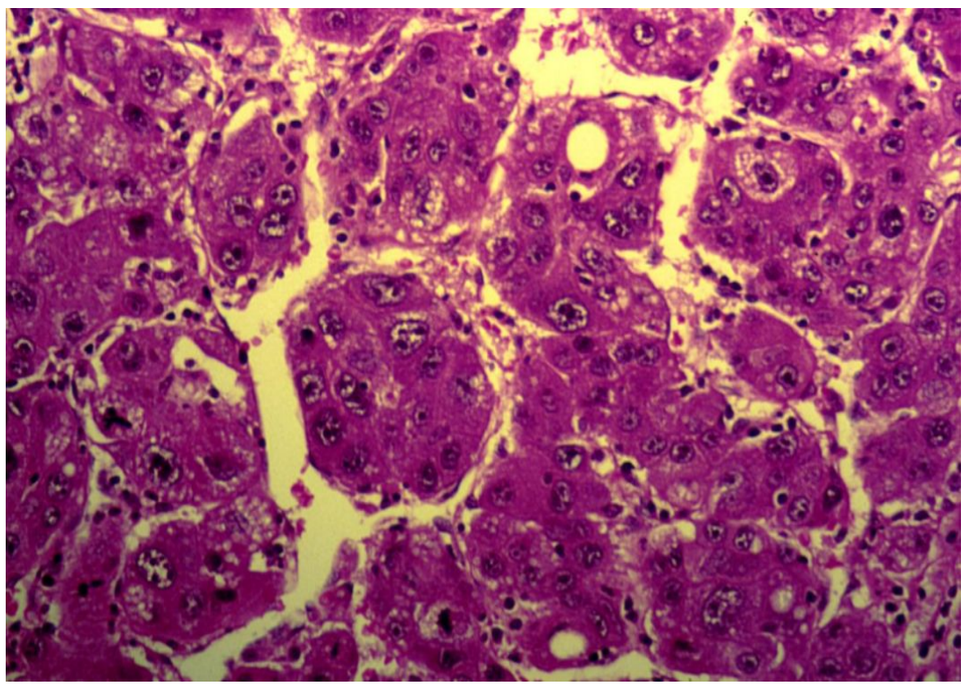
Well circumscribed hepatic mass showing **partly pale** and **partly hemorrhagic** cut surface.

Histopathology



1. **Malignant cells.**
2. **Hepatocytes with prominent nucleoli and nuclear pleomorphism.**
3. **Loss of normal hepatic architecture.**

Histopathology



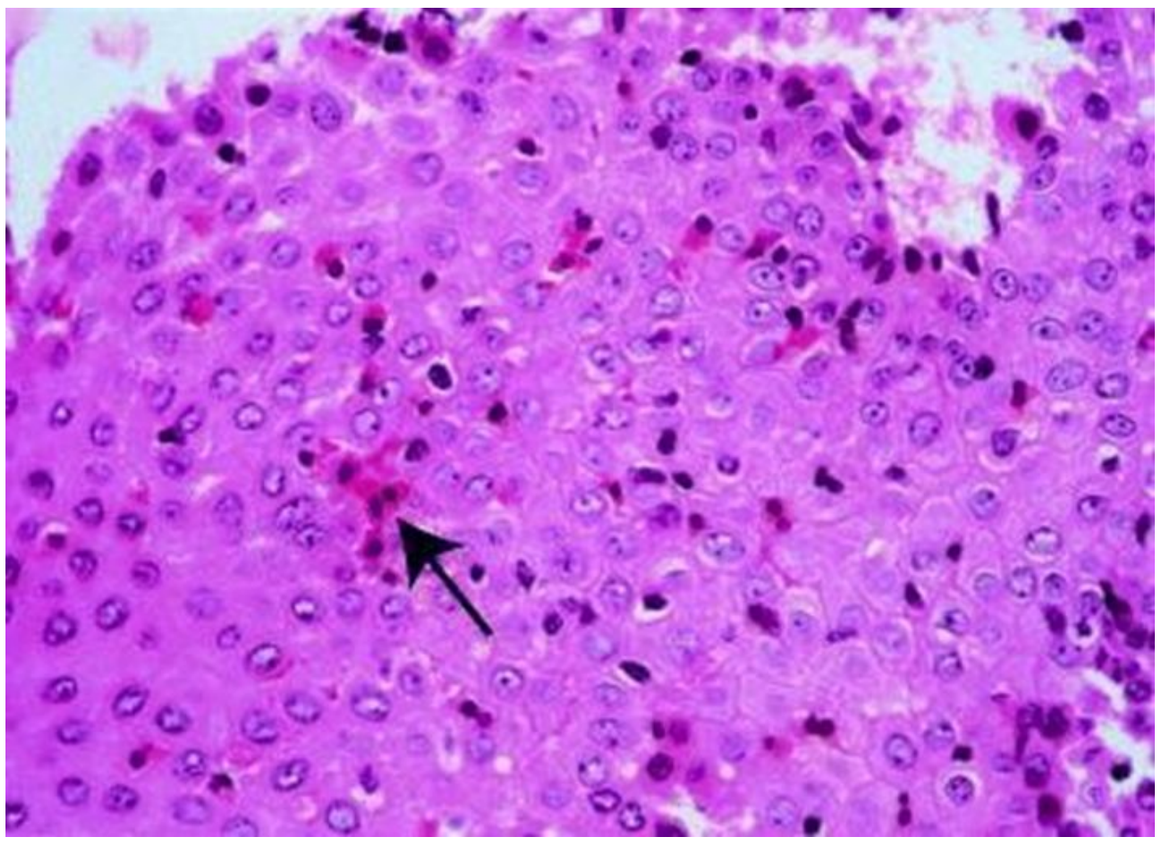
- Thick cords, trabeculae and nests of malignant liver cells separated by sinusoidal spaces..
- Pleomorphic, binucleated or forming giant cells with hyperchromatic nuclei, Mitoses are numerous.
- haemorrhage and necrosis.

Case #10: GASTROESOPHAGEAL REFLUX DISEASE(GERD)

GASTROESOPHAGEAL REFLUX DISEASE (GERD) is characterized by:

1. **Inflammatory Cells: Eosinophils, Neutrophils, Lymphocytes**
2. **Basal zone hyperplasia**
3. **Lamina Propria papillae elongated and congested**

- **Endoscopy** is done to define the cause of GERD. e.g. hernia,or acid reflux. Biopsies are commonly taken almost every time (for confirmation).
- Should be differentiated from Eosinophilic gastroenteritis.

GERD – Endoscopy view	GERD – Microscopically HPF
	
Reflux esophagitis (GERD): **Necrosis of esophageal epithelium** causing ulcers near the junction of the stomach and esophagus due to constant reflux.	Squamous epithelium of lower esophagus w/ inflammation: **1.** Inflammatory cells: **eosinophils** (arrow), PMN and lymphocytes. **2.** **Basal cell hyperplasia**. **3.** Lamina Propria papillae elongated and congested

Case #11: acute pancreatitis.

- **Causes:** Alcoholism, bile reflux, Viral infection "mumps", hyperthermia, Gallstones, medications (thiazides), hypertriglyceridemia, hypercalcemia, acute ischemia, trauma, iatrogenic, Genes: PRSS1, SPINK1, Idiopathic 10-20%,
- **Clinical features:** Severe abdominal pain. Extreme Emergency Situation. High Mortality.
- **The MOST important lab test is: α – AMYLASE.**
- **Consequences:** EDEMA. FAT NECROSIS, ACUTE INFLAMMATORY INFILTRATE, PANCREAS AUTODIGESTION, BLOOD VESSEL DESTRUCTION, SAPONIFICATION.

Gross

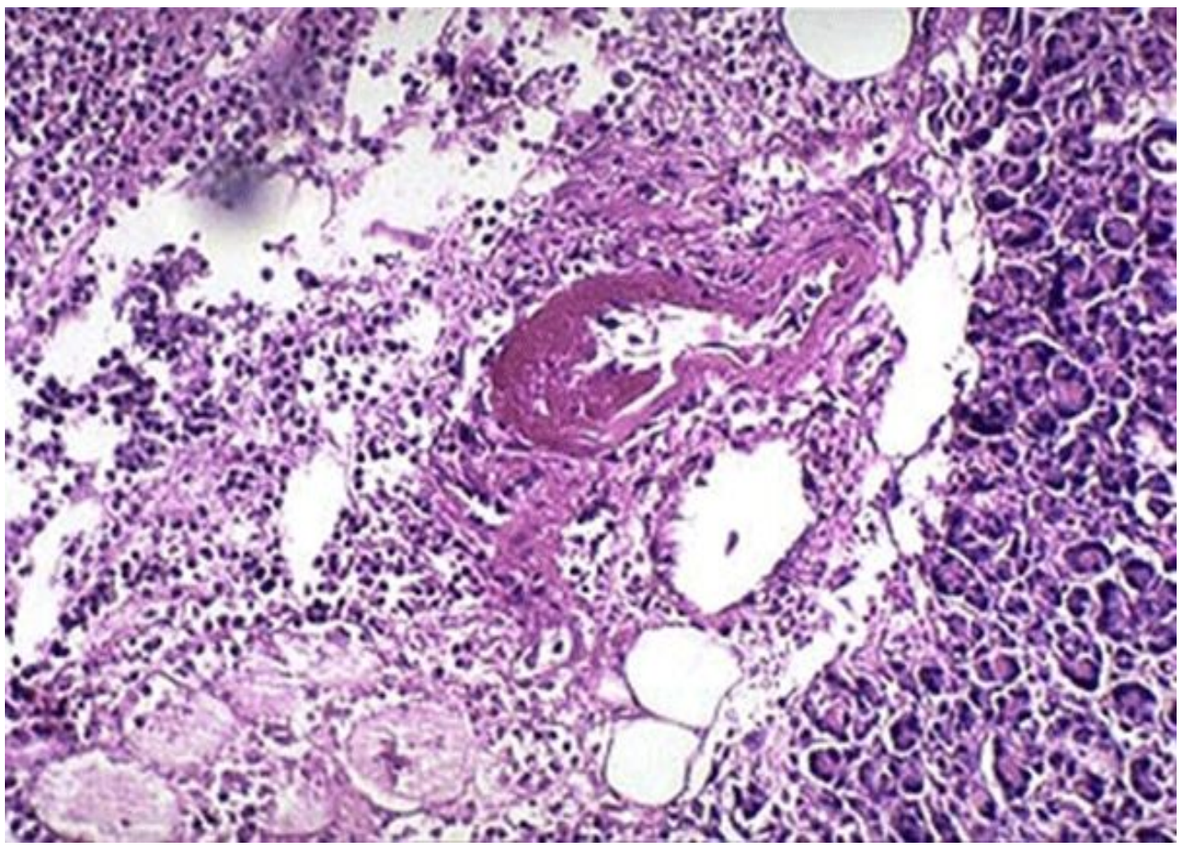
1. black areas of hemorrhage.

2.chalky, yellow-white areas of fat necrosis.

3. Pancreatic parenchyma is soft and gray-white due to necrosis.

Fat necrosis appears as chalky white calcium soaps.

Histopathology



1.area of acute inflammation with necrosis.

2.fibrinoid necrosis of the vessel wall leads to severe, hemorrhagic, acute pancreatitis.

Case #12: Peptic Ulcers

- "PEPTIC" implies acid cause/aggravation.
- Ulcer vs. Erosion (muscularis mucosa intact)
- It is chronic, solitary (usually) & affects adults.
- Why do they come with hematemesis or melena? Because the inflammatory cells eat up the blood vessels.
- Causes:
  - 80% caused by H. pylori
  - NSAIDs
  - Stress

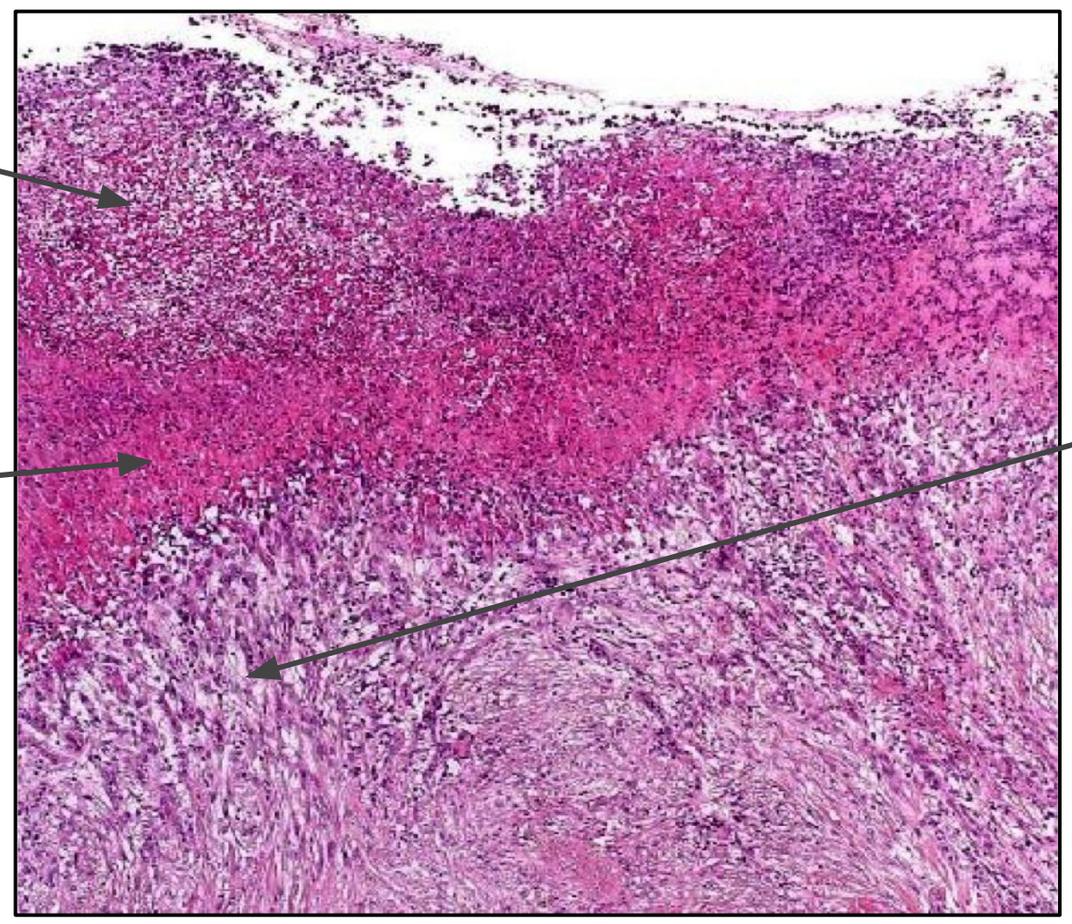
Gross

- The specimen consists of an irregular portion of gastric wall.
- The ulcer is oval in shape and deeply penetrating.
- Necrotic debris covers the base.
- The specimen has been cut to show the submucosa, muscle coat and adventitial connective tissues in the region of the ulcer.

Histopathology

Cellular Debris:
Numerous viable and degenerate polymorphs.

Fibrinoid Necrosis:
Inflammatory cells and granulation tissue.



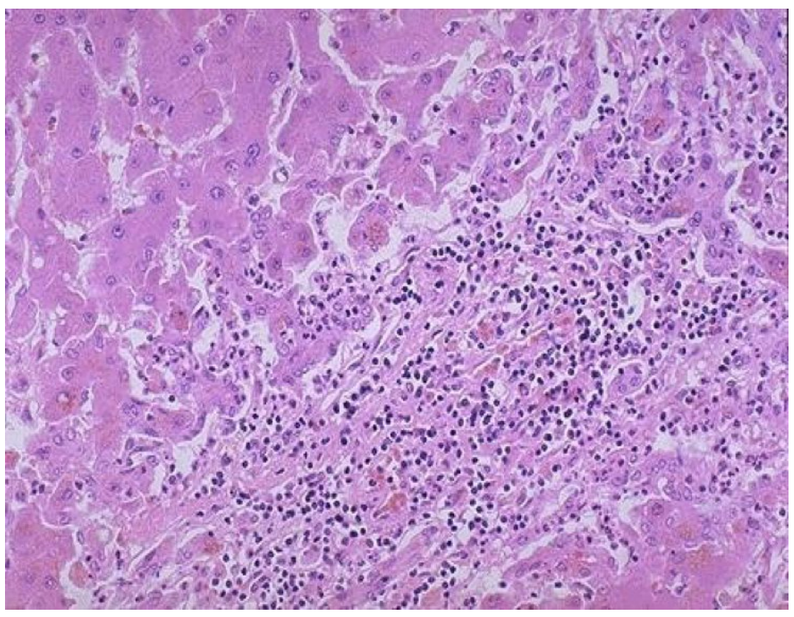
Granulation Tissue:
Variable sized capillary channels are separated by fibroblastic connective tissue heavily infiltrated with lymphocytes, neutrophils, and eosinophils.

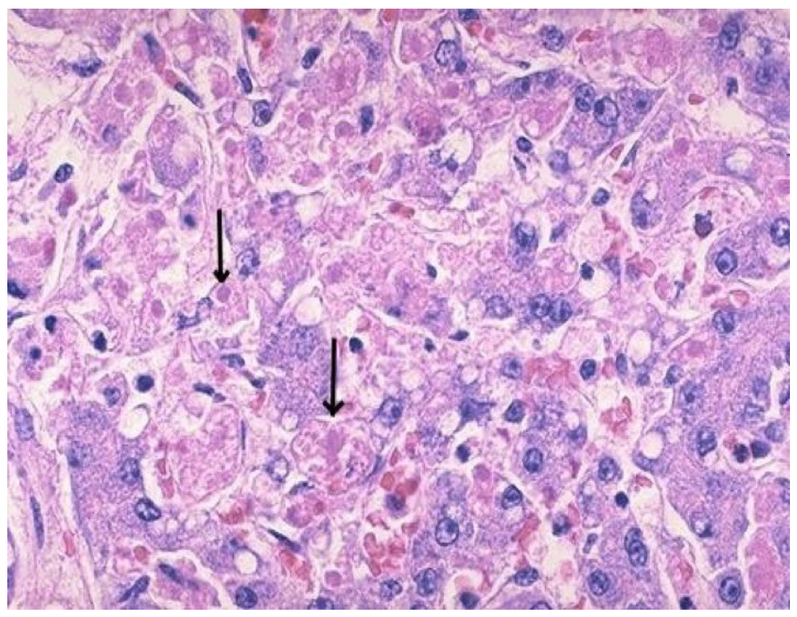
Microscopic examination shows the typical features of a chronic peptic ulcer. The ulcer is located in the antrum

Case #13:Acute viral hepatitis

- **Diagnosis:** by enzyme and serological testing ,NO NEED for biopsy.

Acute viral hepatitis histopathology



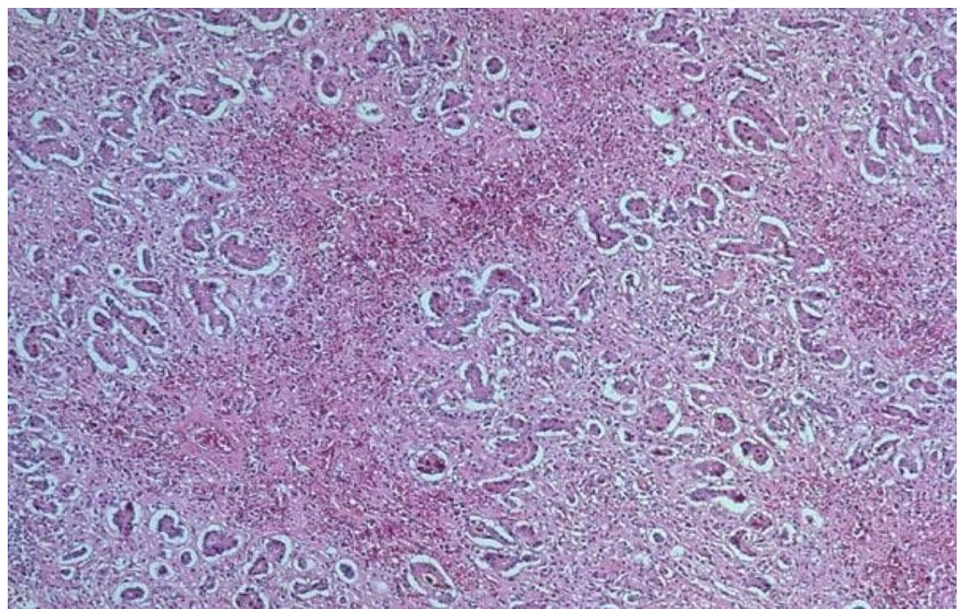


1.Extensive necrosis
2.Ballooning degeneration"arrow".
3.Councilman body a dying hepatocyte is seen shrinking down to form an eosinophilic

FULMINANT HEPATITIS- Gross

Hepatitis giving rise to liver failure.

FULMINANT HEPATITIS

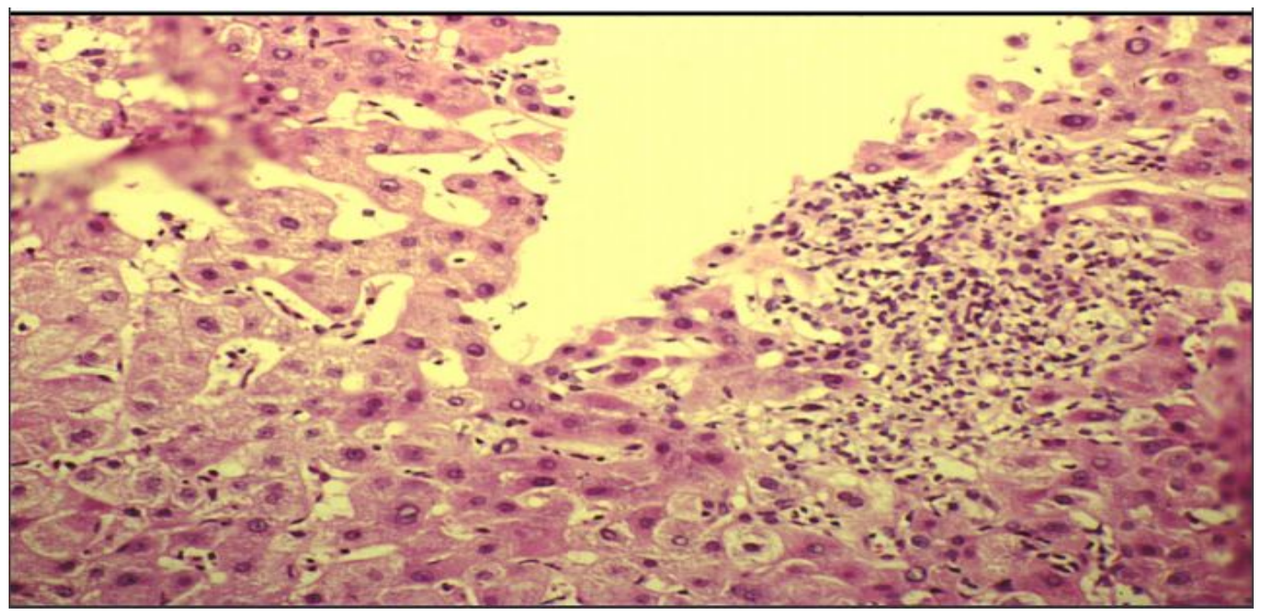


1.Massive hepatic necrosis ,often results in death.

Case #13: Chronic hepatitis

CHRONIC HEPATITIS- Gross

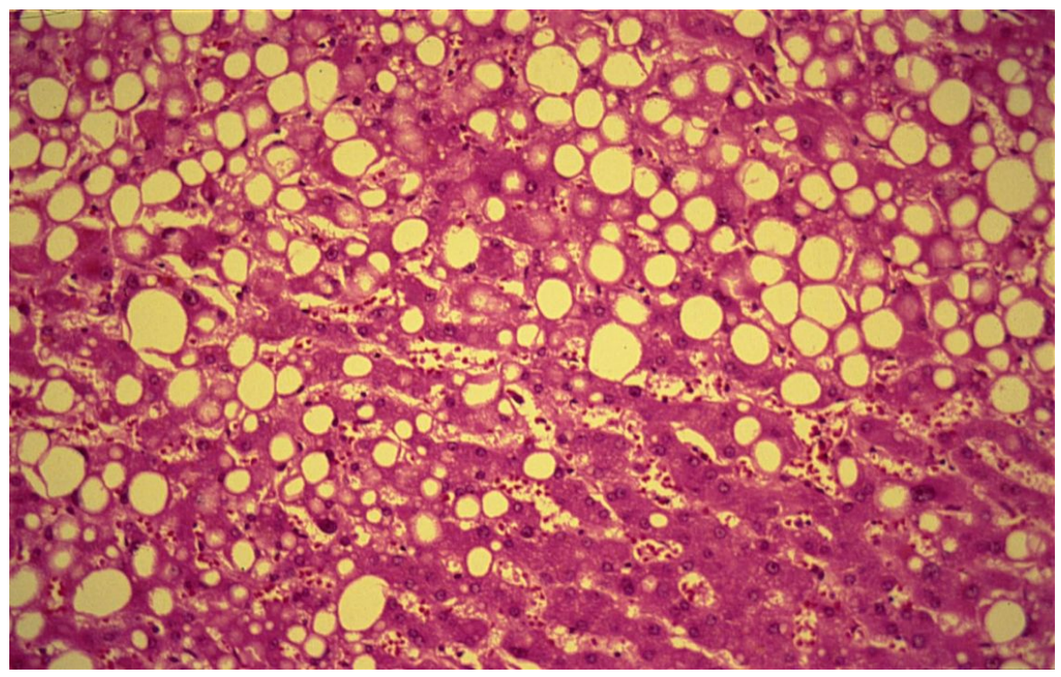
CHRONIC HEPATITIS- HISTOPATHOLOGY



1.Chronic inflammatory cells infiltration in both portal tracts and liver parenchyma.
2.Piecemeal necrosis, hepatocytes swelling, "spotty" hepatocytes necrosis.
No evidence of cirrhosis or malignancy noted.

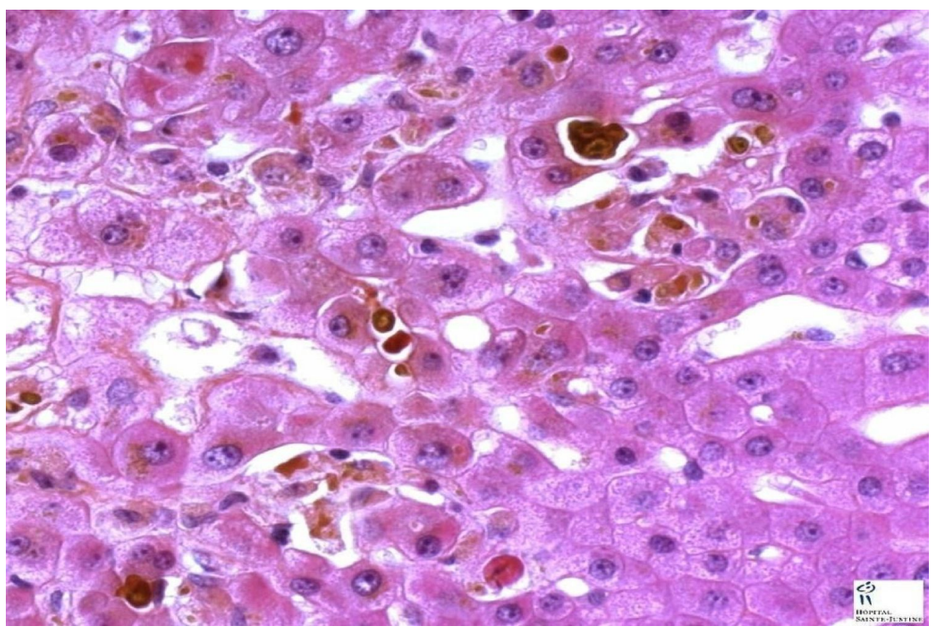
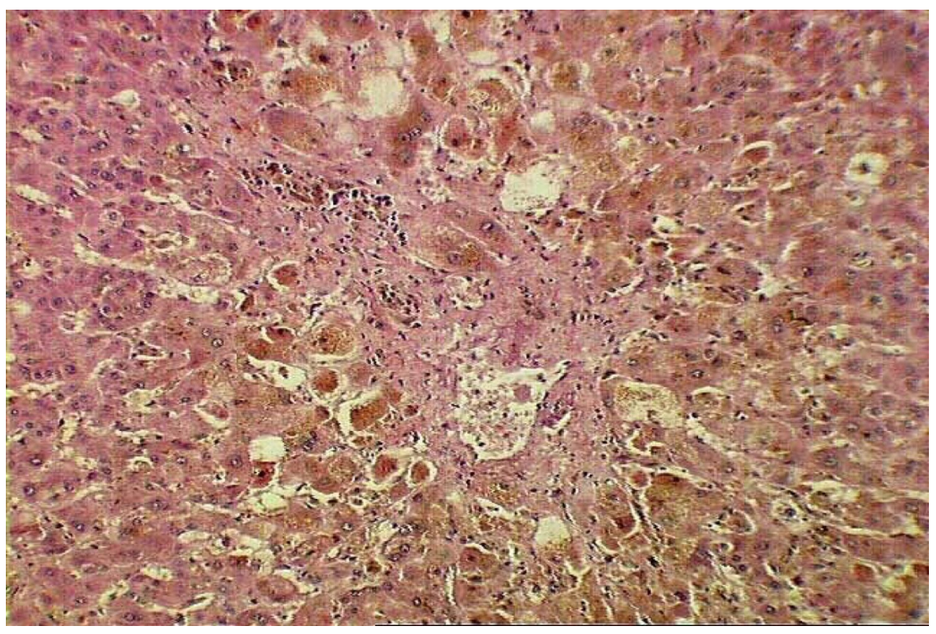
Case #14: Fatty Liver

- **What's the normal lobular architecture?** 1.Central vein in the center 2.Portal tract at the periphery containing portal vein. 3.Hepatocytes.
- **Lobular architecture:** normal in fatty liver, and absence in Liver cirrhosis.

Liver- Gross	Fatty changes in the liver
	
1.Normal liver on the left **2.**Fatty liver on the right "Steatosis"	**1.**Normal lobular architecture. **2.** Liver cells are distended by clear vacuoles of dissolved fat with displacement of the periphery. **3.**Fatty cysts may be seen. **4.**No inflammation and no fibrosis.

Case #15: Cholestasis

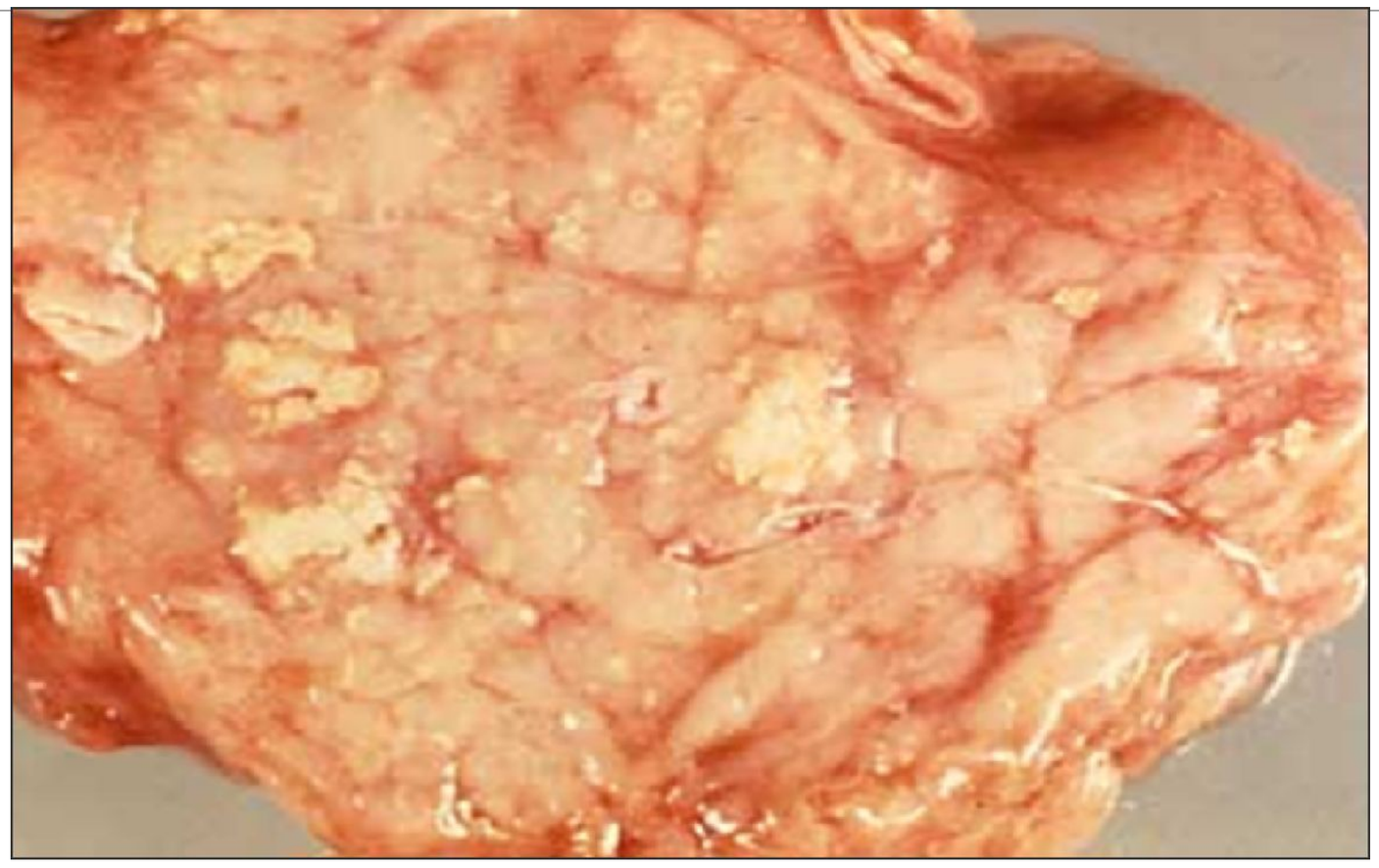
- **What is cholestasis?** Bile accumulation in the liver. "hepatocytes"
- **Could be:** mechanical or functional (obstructive and non-obstructive)
- **Changes in**: Lobular parenchyma or portal tracts
- **Characteristic lab finding:** **elevated Alkaline phosphatase and GGT**

Cholestasis histopathology.	Cholestasis histopathology
	
1.Bile leaks or plugs **2.**High pigments in the hepatocytes.	**1.**Brownish pigments -> bile. **2.** Hepatocytes and portal vein,

Case #16: chronic pancreatitis

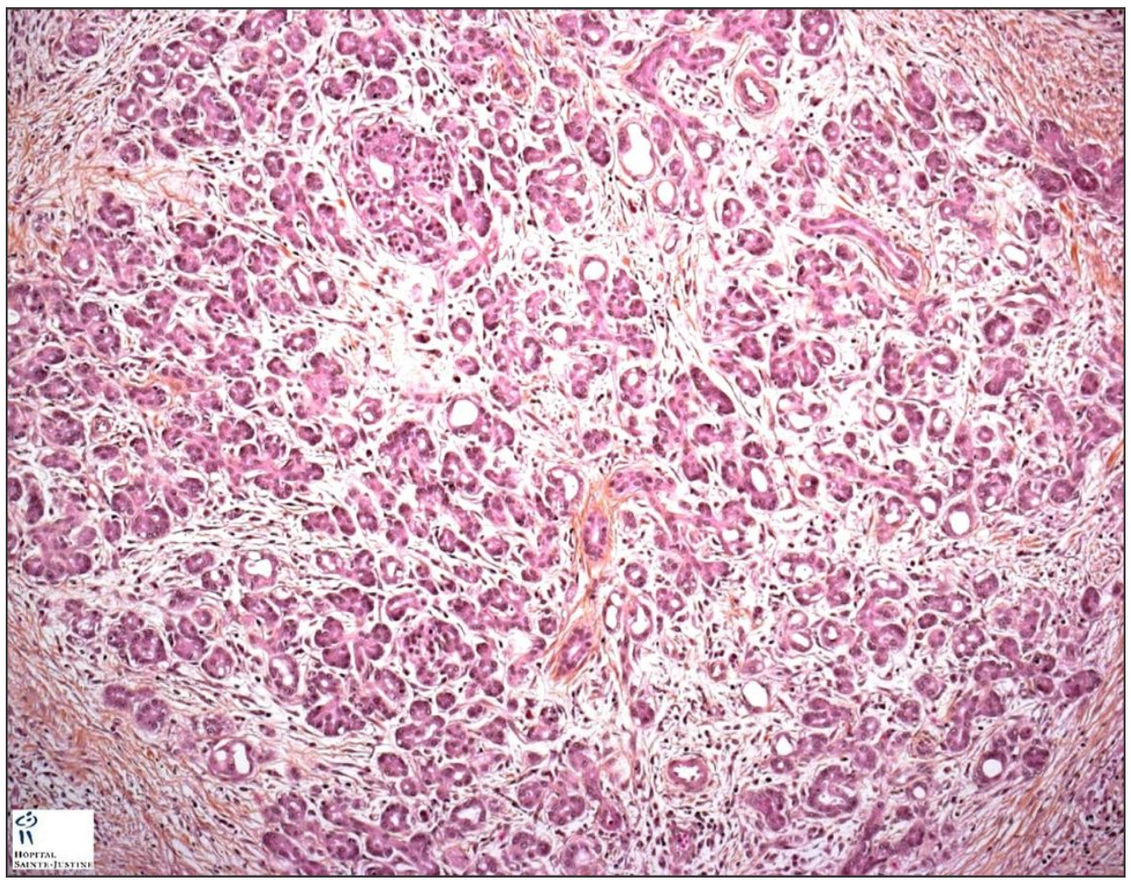
- **causes of chronic pancreatitis**: gallstones , alcoholism, tropical , hereditary and idiopathic.
- **Patient presents with:** Diabetes and fat in the stool.

Chronic pancreatitis- Gross



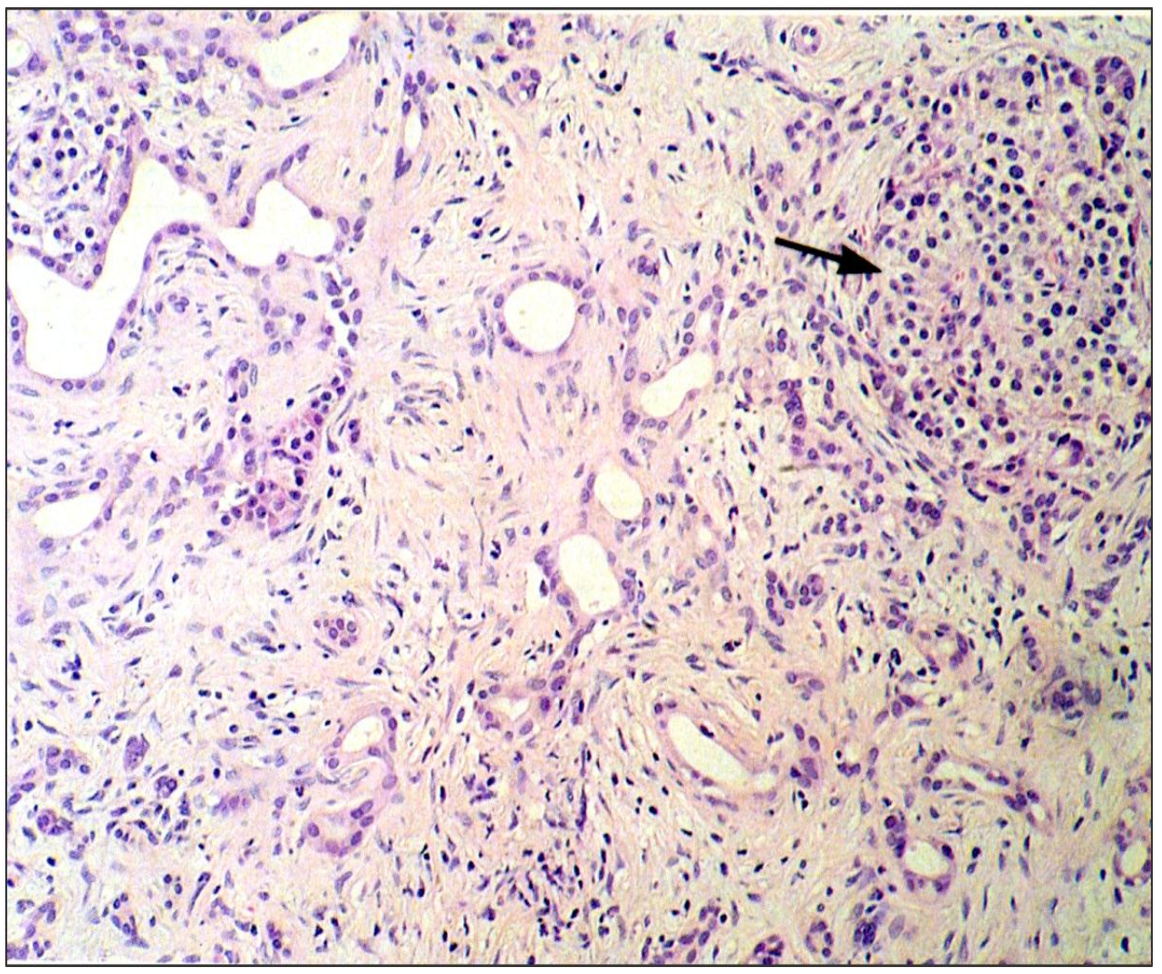
1.Soap
2.Calcium, Calcium deposition is secondary to fat necrosis and dystrophic calcification

Chronic pancreatitis- LPF



dense fibrosis is a feature BOTH of chronic pancreatitis as well as adenocarcinoma

Histopathology

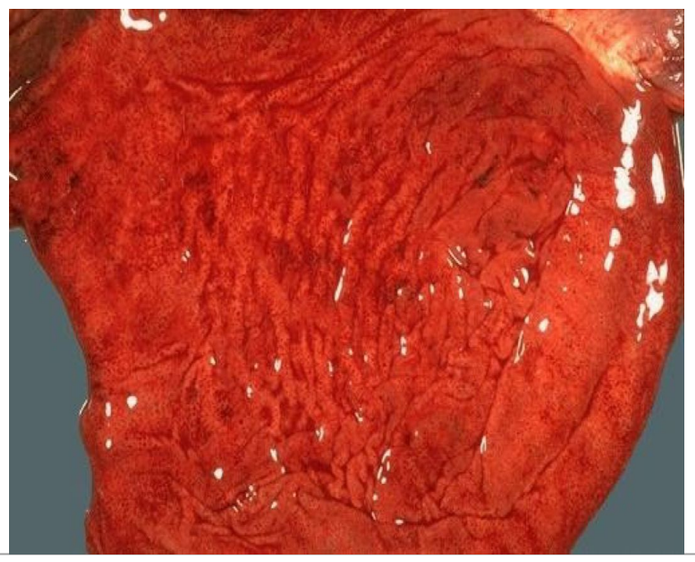


1.Parenchymal Fibrosis
2.Chronic Inflammatory infiltrate
3.Loss of acini

Case #17:Acute Gastritis

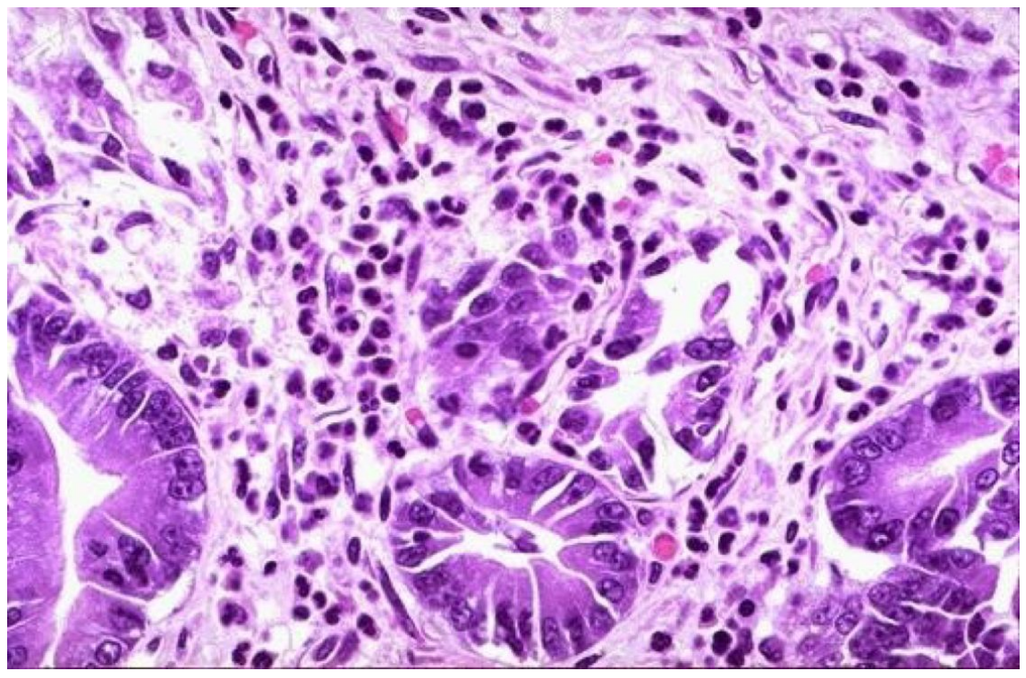
Causes of **Acute Gastritis: 1. Alcoholism 2.Drugs 3. Infections.**

Gross



This is a more typical acute gastritis with a diffusely **hyperemic gastritis. It is also edematous**

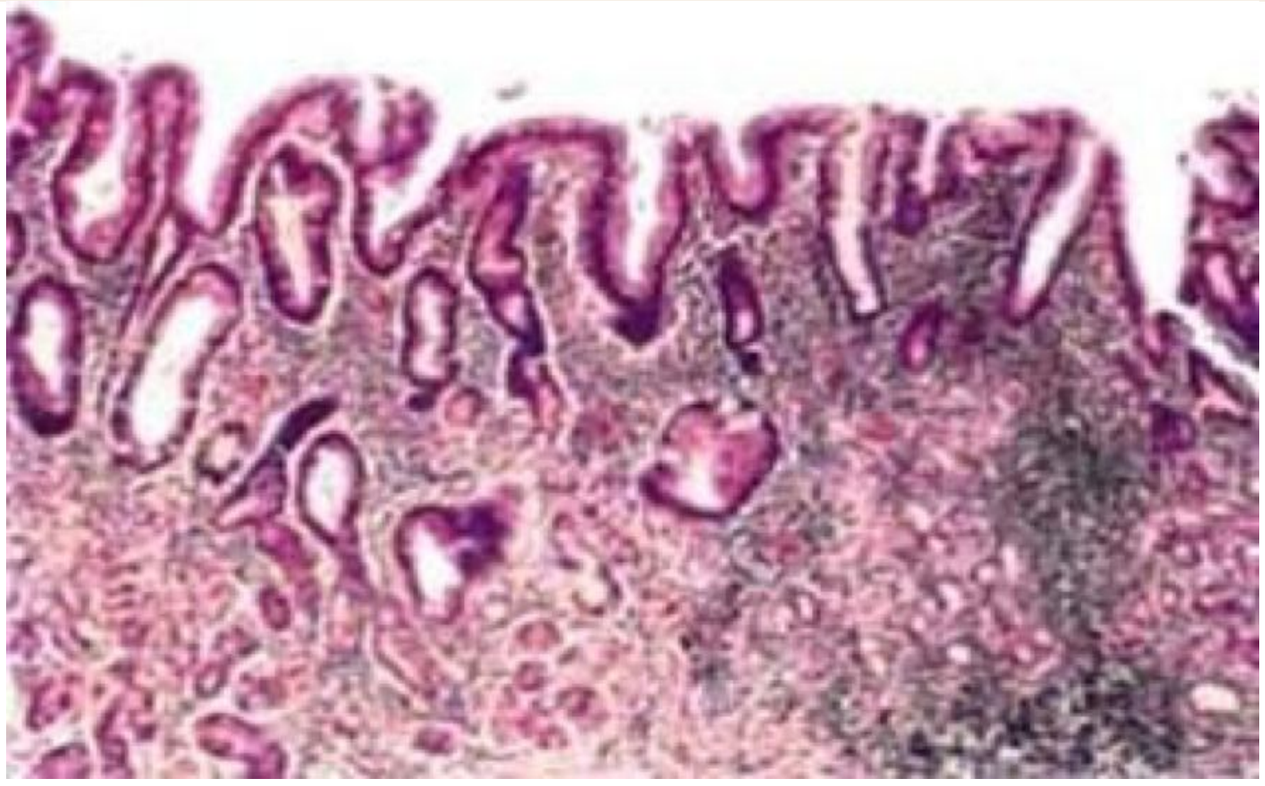
Histopathology HPF



Gastric mucosa demonstrates infiltration by **neutrophils**

Case #17:Chronic Gastritis

1. Chronic **NO erosions, NO hemorrhage**. (because it is long standing)



Histopathology

- Perhaps some neutrophils
- Lymphocytes, lymphoid follicles

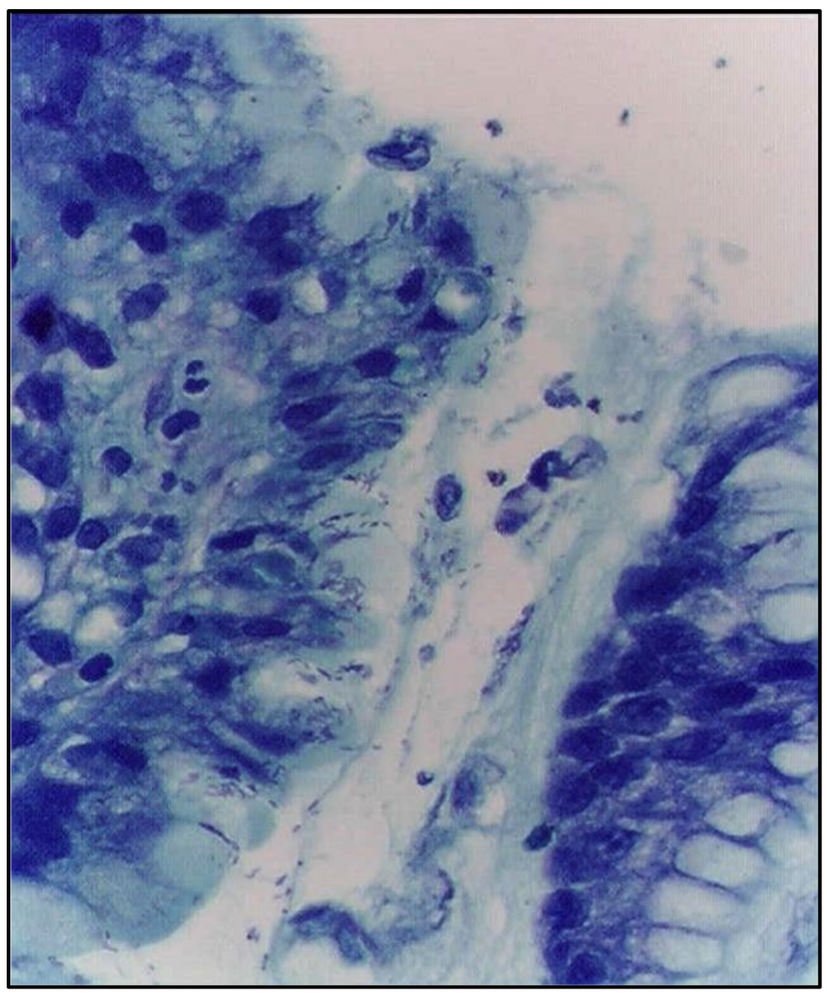
Regenerative Changes:

1. Metaplasia (intestinal)
2. Atrophy: Mucosal Hypoplasia “thinning”
3. Dysplasia

Case #18:Helicobacter-induced Gastritis

- It is the most common type of Gastritis.
- Gastritis is often accompanied by infection with Helicobacter pylori.
- Bacteria is attacking the mucosa
- Antibiotic is the treatment.

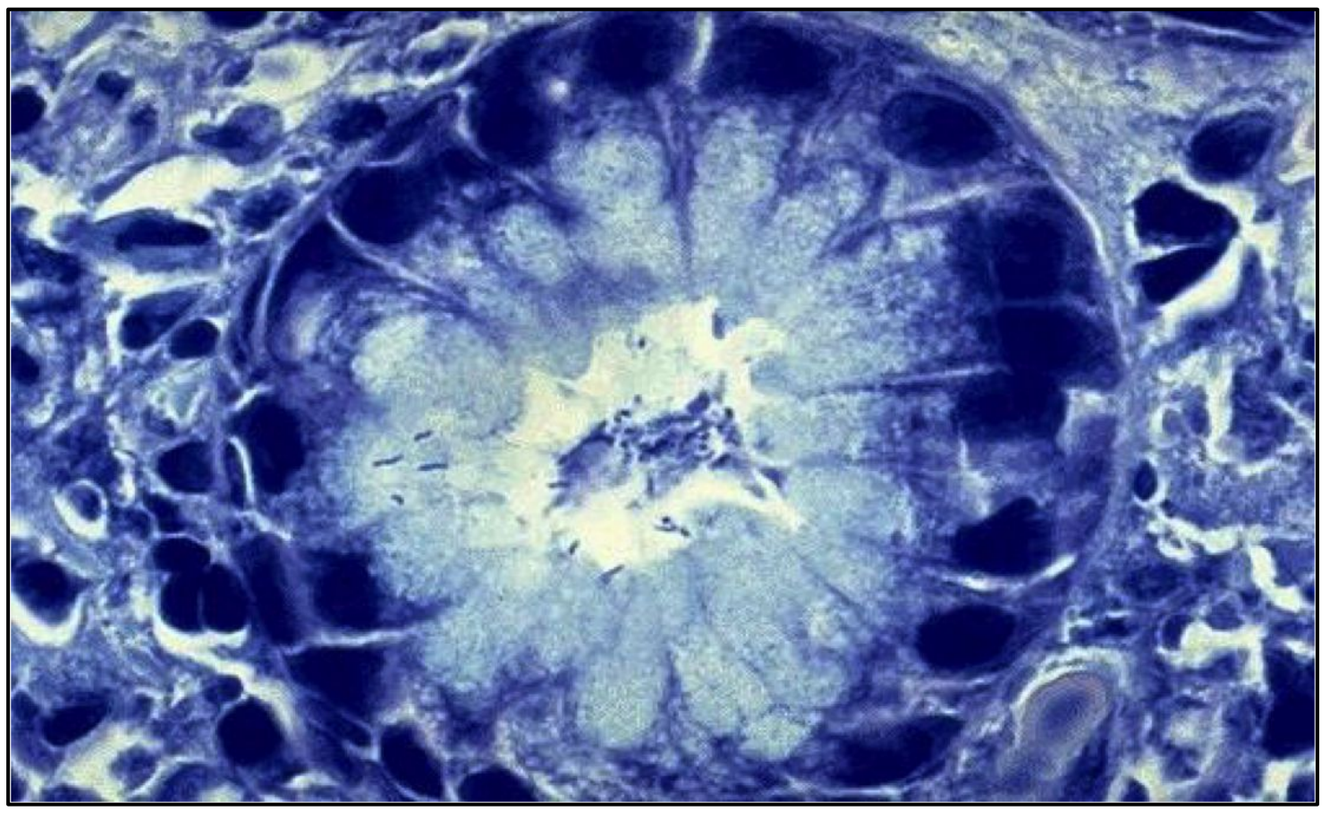
Helicobacter pylori, gastric biopsy



Silver stain on left,
Giemsa stain on right.

You don’t need to know the stains.

Microscopic View

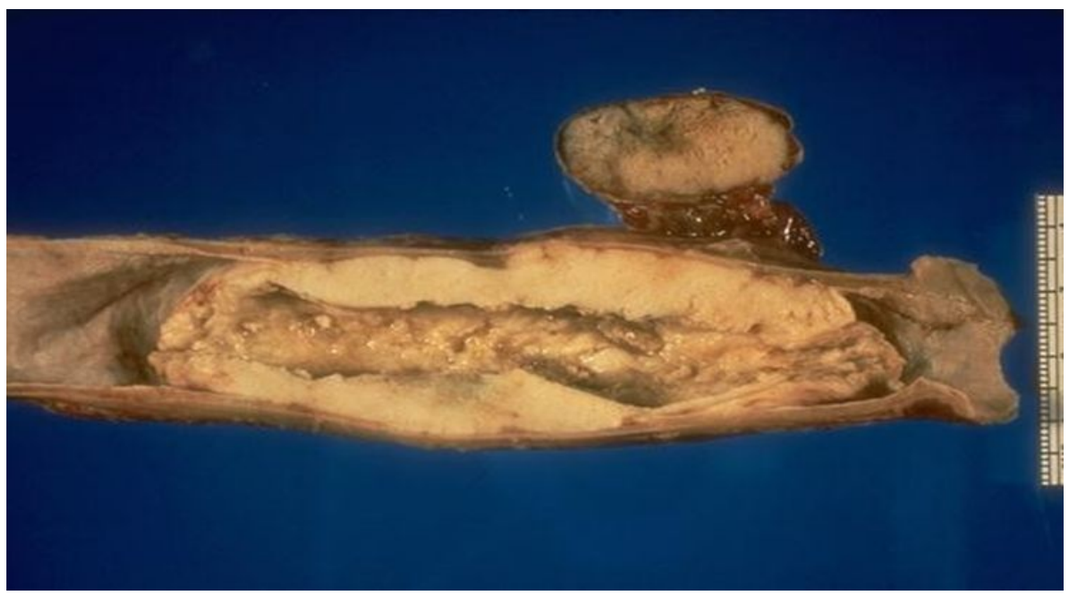


This small curved to spiral rod-shaped bacterium is found in the surface epithelial mucus of most patients with active gastritis. The rods are seen here with a methylene blue stain

Case #19:Carcinoma of the Esophagus

- **Squamous Cell Carcinoma is the most common carcinoma of the esophagus, Usually seen in elderly patients.**
- **The usual complain of the patient:** they present with progressive **dysphagia** which means that the patient first has difficulties in swallow the solid food then it progress to have difficulties in swallow also the liquid . WHY? Because the lumen is progressively narrowed . **In addition to loss of appetite and losing weight**
- It could be Poor, moderate, or well differentiated depending on the amount of the keratin formation because squamous cells produce keratin so if more Keratin it is well differentiated and if there are less keratin it is poor differentiated.

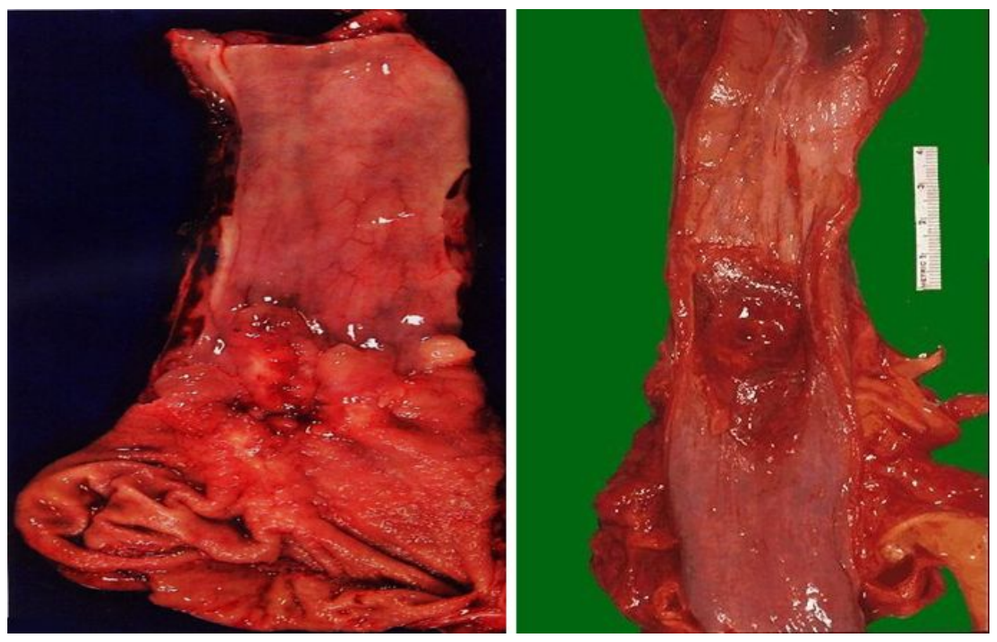
Carcinoma of the Esophagus - Gross



1. Squamous cell carcinoma of the esophagus within a lymph node.

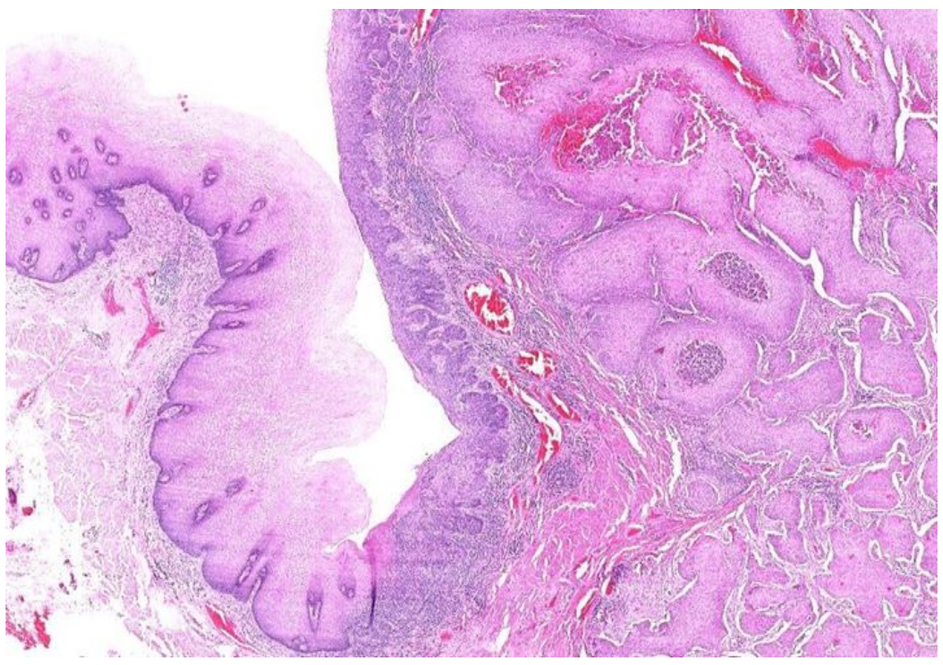
2.The oval structure adjacent to the esophagus represents metastatic

Carcinoma of the Esophagus - Gross

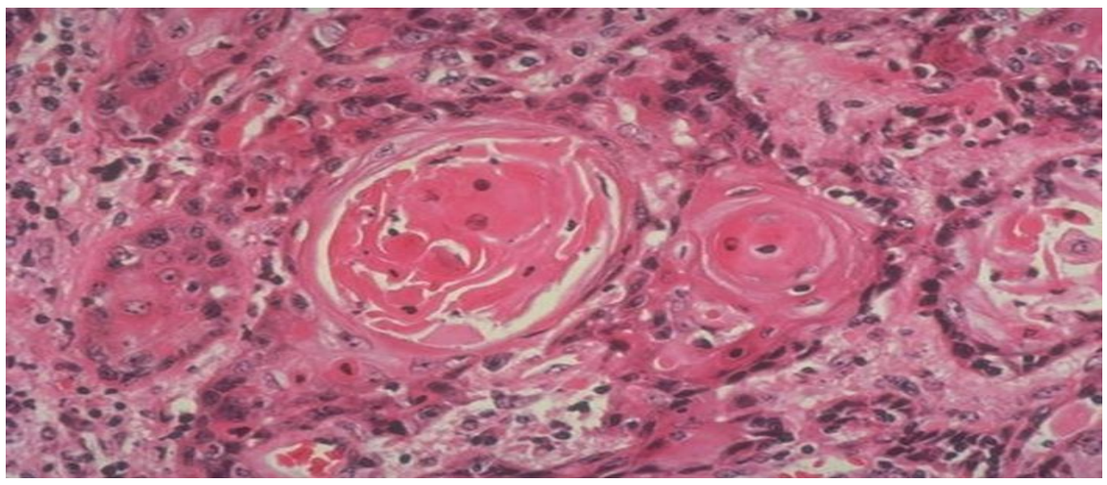


1. squamous cell carcinoma causing luminal stenosis .

Squamous Dysplasia of the Esophagus - LPF

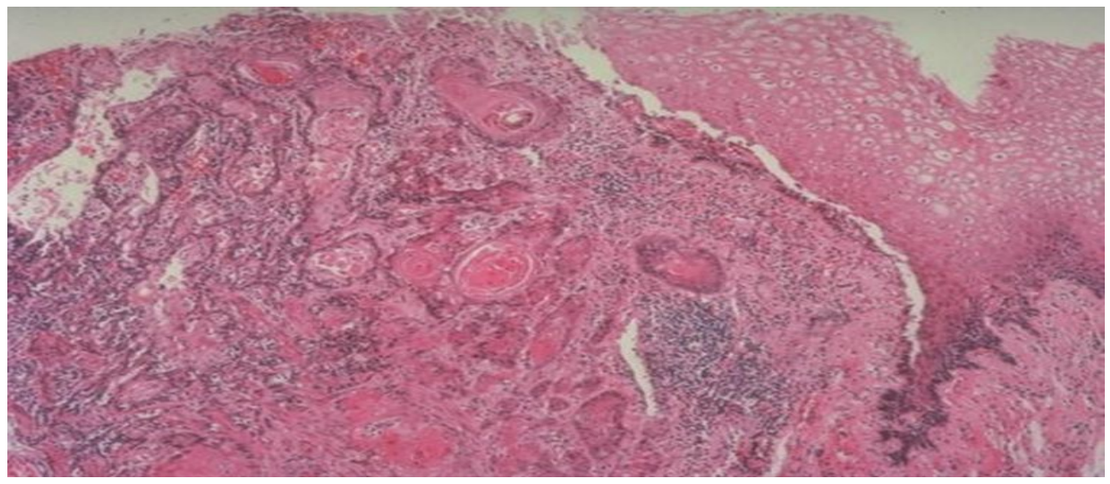


1. **From left to right Normal then dysplastic then infiltrating.**
2. **high grade dysplasia.**
3. **nuclei are larger and more hyperchromatic than normal, and there is increased mitotic activity**



Solid nests of neoplastic cells having abundant pink cytoplasm and distinct cell borders

Malignant cells producing keratin keratin is pinkish material)



Infiltrating nests of neoplastic cells

(deep down the lamina propria)

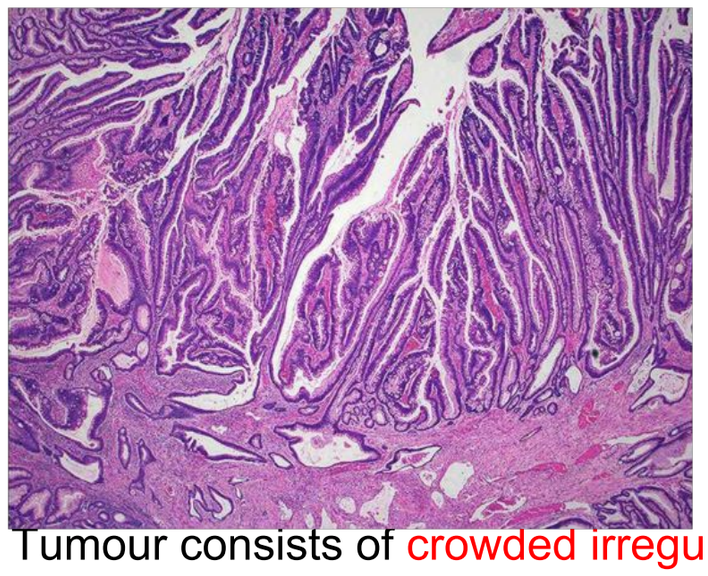
This is a well differentiated squamous cell carcinoma

Case #20:Adenocarcinoma of the colon

Adenocarcinoma of the Colon - Gross

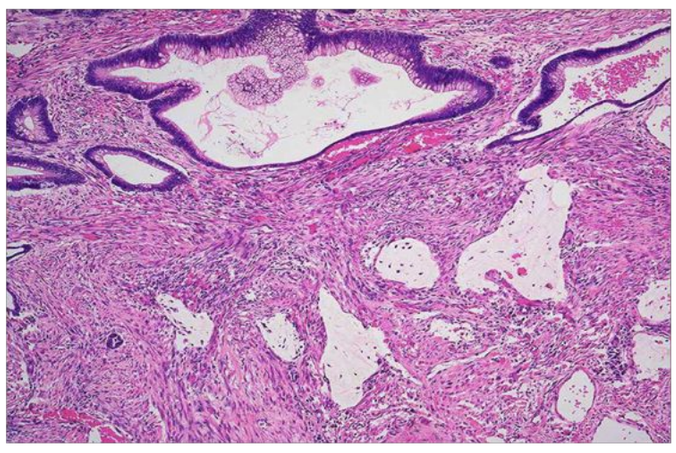
This is an adenocarcinoma arising in a **villous adenoma.**The surface of the neoplasm is polypoid and reddish pink. Hemorrhage from the surface of the tumor creates a guaiac positive stool. This neoplasm was located in the sigmoid colon

Adenocarcinoma of the Colon - LPF



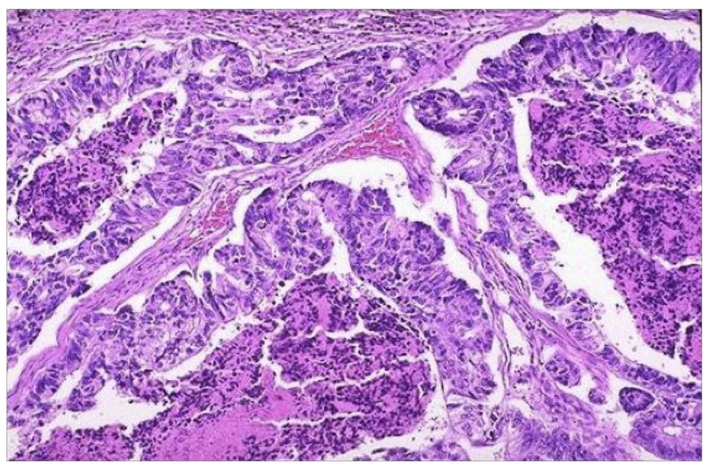
Tumour consists of crowded irregular malignant acini separated by thin fibrovascular stroma.

Adenocarcinoma of the Colon - LPF



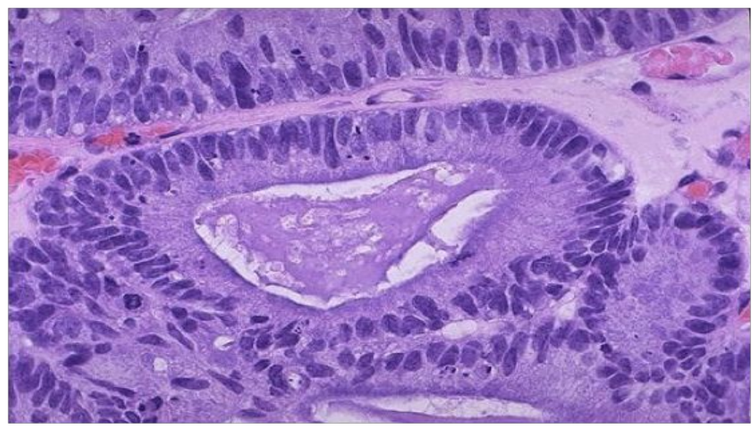
The acini are lined by one or several layers of neoplastic cells with papillary projection showing pleomorphism, hyperchromatism and few mitoses.

Adenocarcinoma of the Colon - MPF



Here is an adenocarcinoma in which the glands are much larger and filled with necrotic debris.

Adenocarcinoma of the Colon - HPF



At high magnification, the neoplastic glands of adenocarcinoma have crowded nuclei with hyperchromatism and pleomorphism. No normal goblet cells are seen

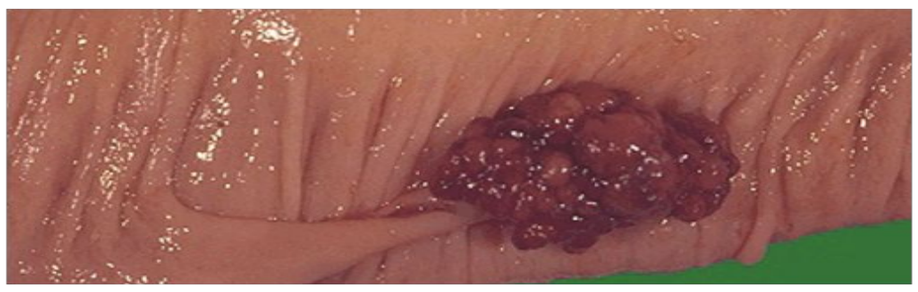
Case #21:Adenomatous polyp & familial polyposis

Familial adenomatous polyposis:

- a genetic syndrome in which an abnormal genetic mutation leads to development of multiple neoplasms in the colon.
- It is caused by mutations of the adenomatous polyposis coli , or APC gene .
- The major complication is development of adenocarcinoma of the colon.

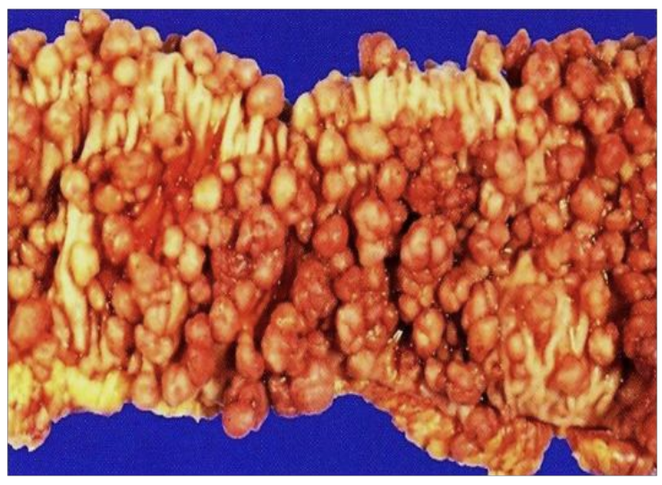
Adenomatous polyp of the colon - Gross

Multiple adenomatous polyps (tubulovillous adenomas) of the cecum are seen here in a case of familial adenomatous polyposis,

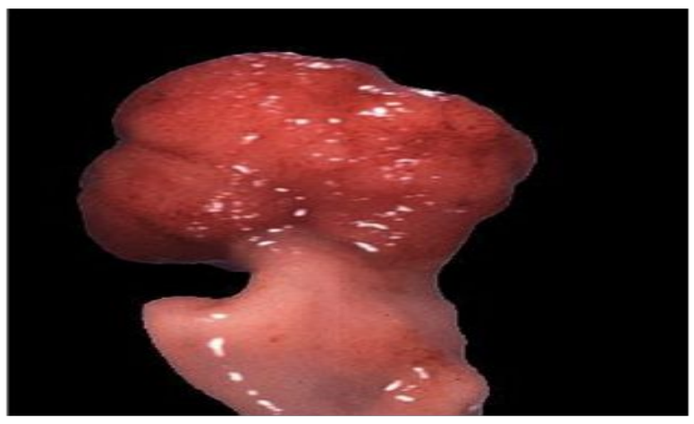


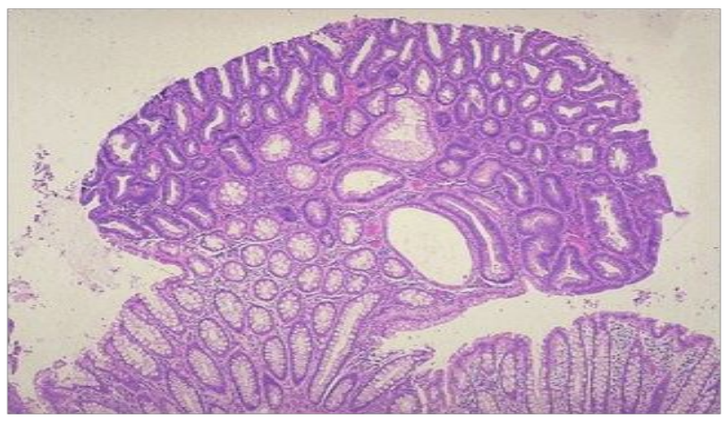
This adenomatous polyp has a hemorrhagic surface (which is why they may first be detected with stool occult blood screening) and a long narrow stalk. The size of this polyp--above 2 cm--makes the possibility of malignancy more likely, but this polyp proved to be benign

Familial polyposis of the colon - Gross



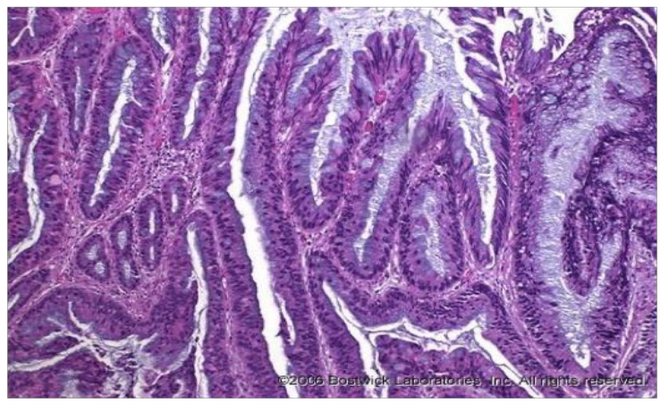
Adenomatous polyp of the colon - LPF





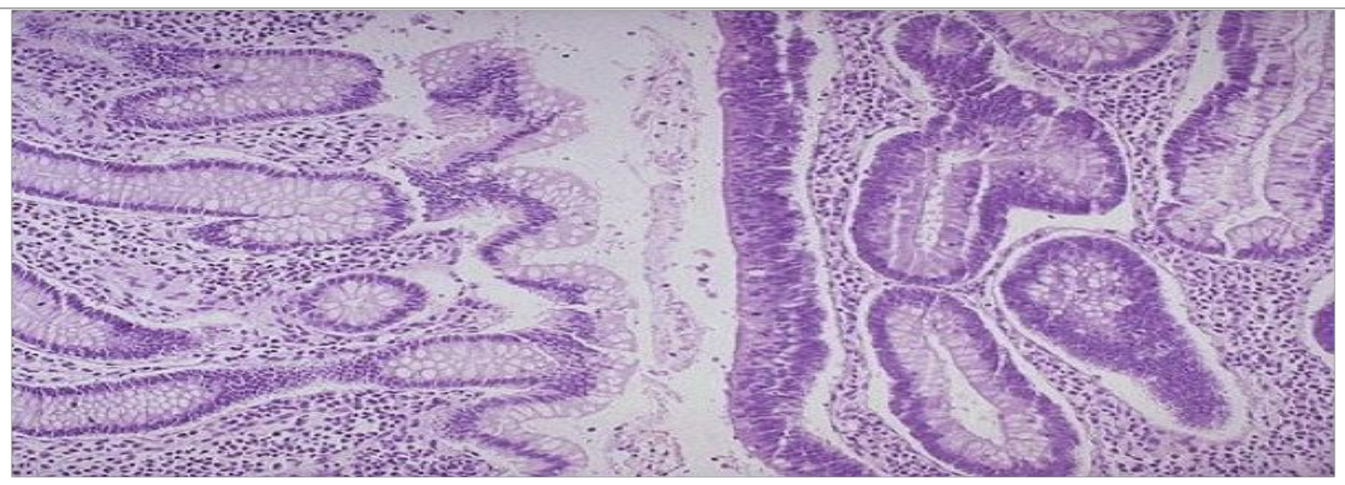
This small adenomatous polyp (tubular adenoma) on a small stalk is seen microscopically to have more crowded, disorganized glands than the normal underlying colonic mucosa. Goblet cells are less numerous and the cells lining the glands of the polyp have hyperchromatic nuclei

Adenomatous Polyp(Villous) - MPF



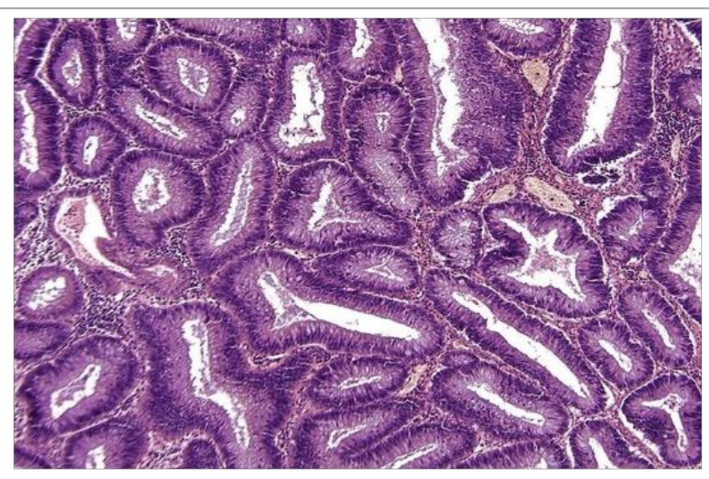
Villous adenomas behave more aggressively than tubular adenomas.They have a HIGHER rate of developing into frank adenocarcinomas than the “tubular” patterns.

Normal vs Adenomatous polyp of the colon - MPF



A microscopic comparison of **normal** colonic mucosa on the **left** and that of an **adenomatous polyp (tubular adenoma)** on the **right** is seen here. The neoplastic glands are more irregular with darker (**hyperchromatic**) and more crowded nuclei.

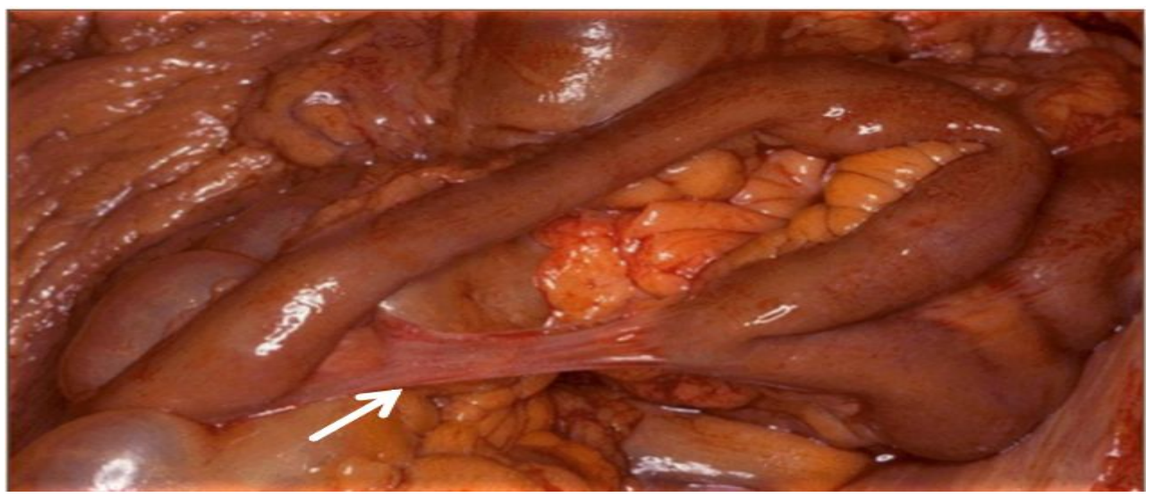
Adenomatous Polyp(Tubular) - MPF



TUBULAR adenoma with crowded dysplastic glands and chronic inflammation.

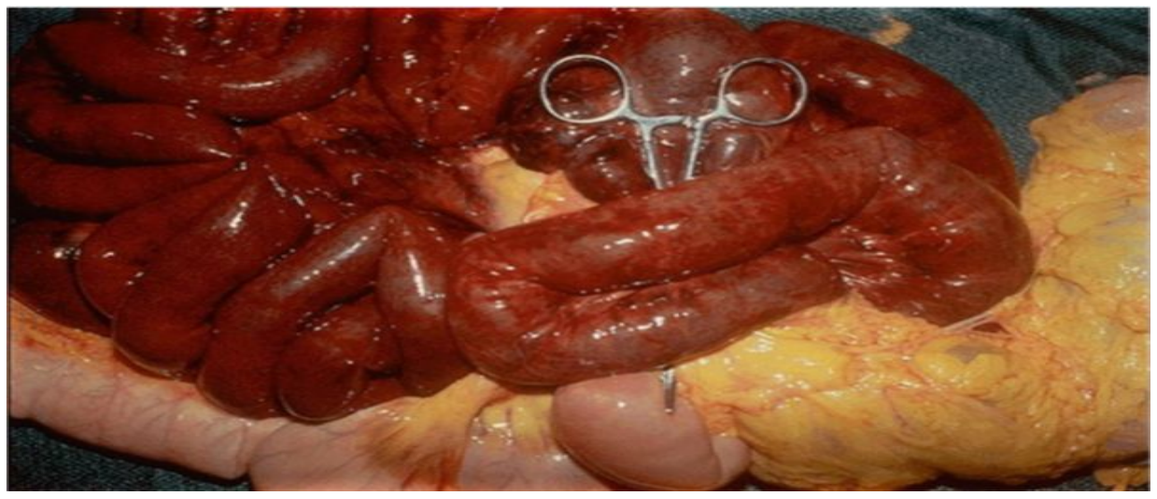
Case #24:Small intestinal infarction & ischemic enteritis

Adhesions, peritoneum, small intestine - Gross



-This is an adhesion between loops of small intestine.
-Such adhesions are typical following abdominal surgery.
- More diffuse adhesions may also form following peritonitis.

Small intestinal infarction - Gross



-benign infarction due to any obstruction of the blood vessels like infection, or after any surgery.
Not important to know in the exam.

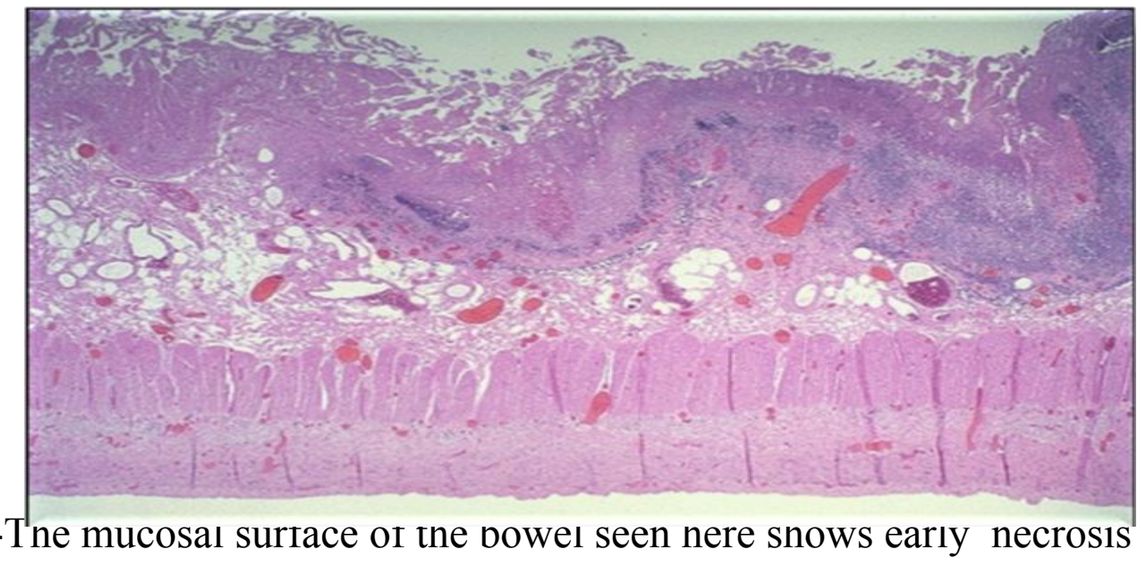
Ischemic Enteritis - Gross

-The small intestinal mucosa demonstrates marked hyperemia as a result of ischemic enteritis.
-Such ischemia most often results from hypotension (shock) from cardiac failure, from marked blood loss, or from loss of blood supply from mechanical obstruction (as with the bowel strangulated in a hernia or with volvulus or intussusception). -If the blood supply is not quickly restored, the bowel will infarct.

Ischemic Enteritis – Gross [ENDOSCOPY]

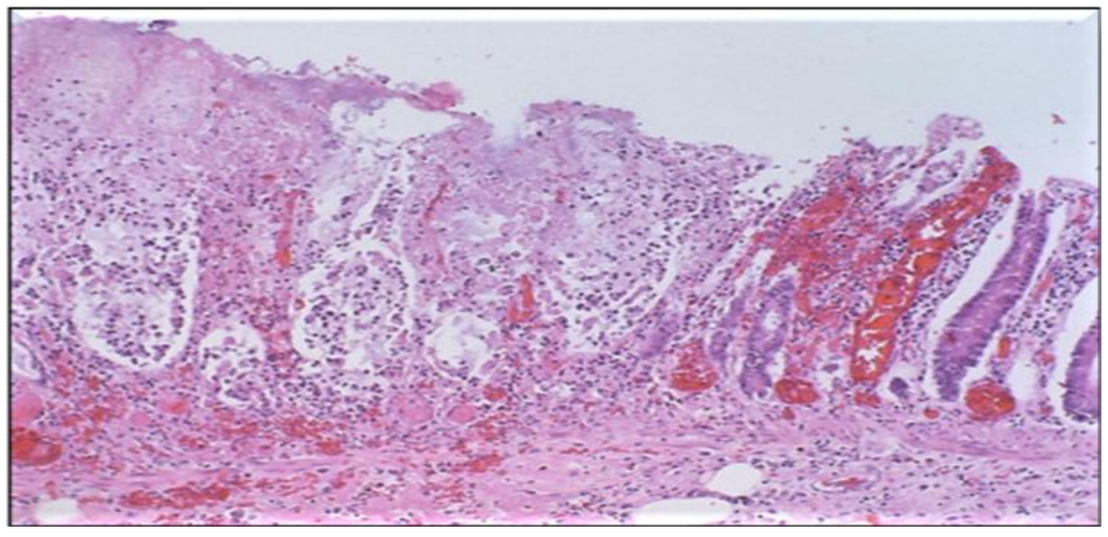
-On closer inspection, early ischemic enteritis involves the tips of the villi.
- In general, bowel is hard to infarct from atherosclerotic vascular narrowing or thromboembolization because of the widely anastomosing blood supply.
-Thus, most cases of bowel ischemia and infarction result from generalized hypotension and decreased cardiac output.

Histopathology
Ischemic Enteritis – LPF



-The mucosal surface of the bowel seen here shows early necrosis with hyperemia extending all the way from mucosa to submucosal and muscular wall vessels.
-The submucosa and muscularis, however, are still intact.

Histopathology
Ischemic Enteritis – MPF



-At higher magnification with more advanced necrosis, the small intestinal mucosa shows hemorrhage with acute inflammation in this case of ischemic enteritis.

Case#23:PLEOMORPHIC ADENOMA

PAROTID GLAND SWELLING – **Clinical**

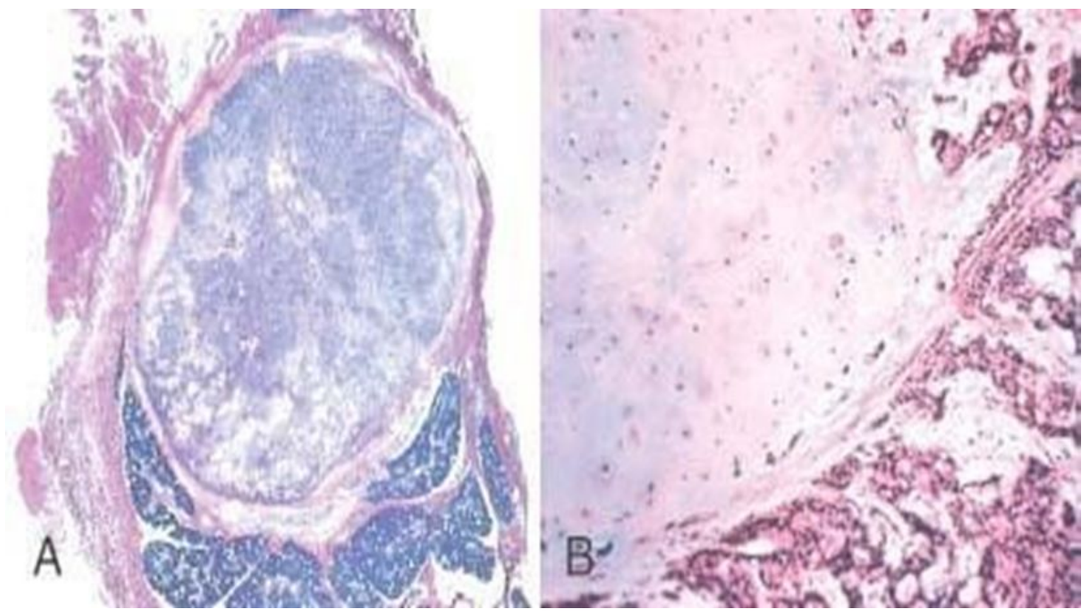
The classic place for any visible parotid **swelling** or tumor is present **between the tip of the ear** and the tip **(angle) of the mandible.**

- **Lymph node is the most common cause**

PLEOMORPHIC ADENOMA
(MIXED TUMOR) - Gross

Pleomorphic adenoma (white area of Cartilage-like tumor.

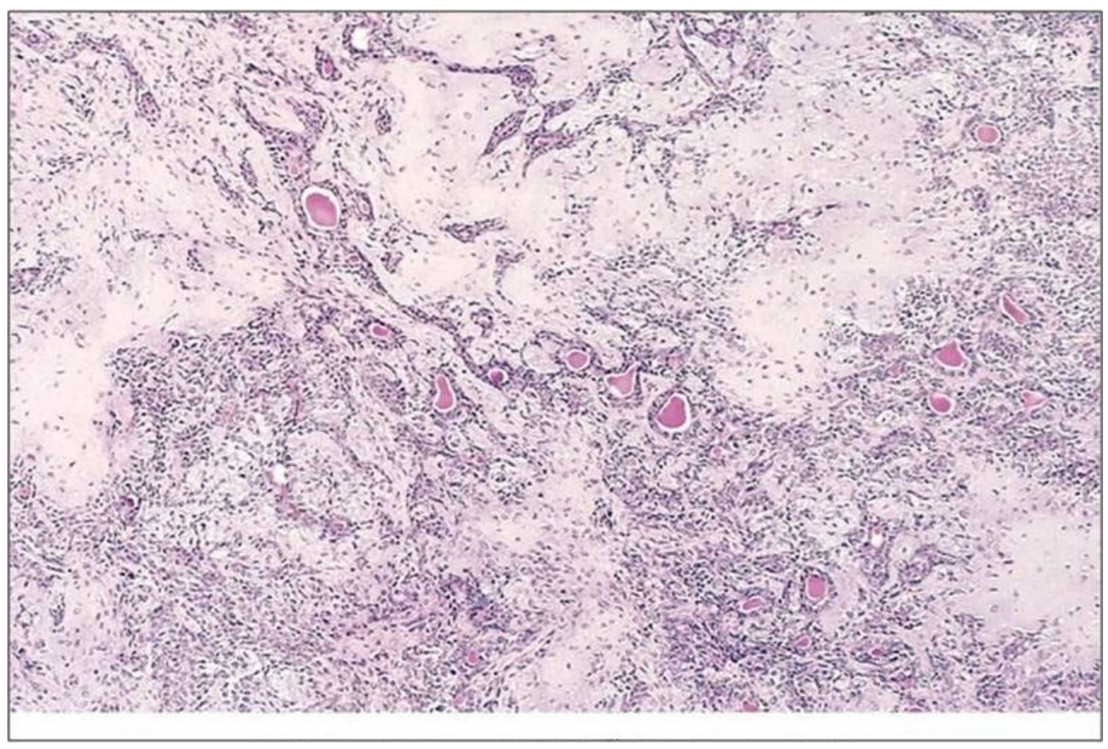
PLEOMORPHIC ADENOMA (MIXED TUMOR)

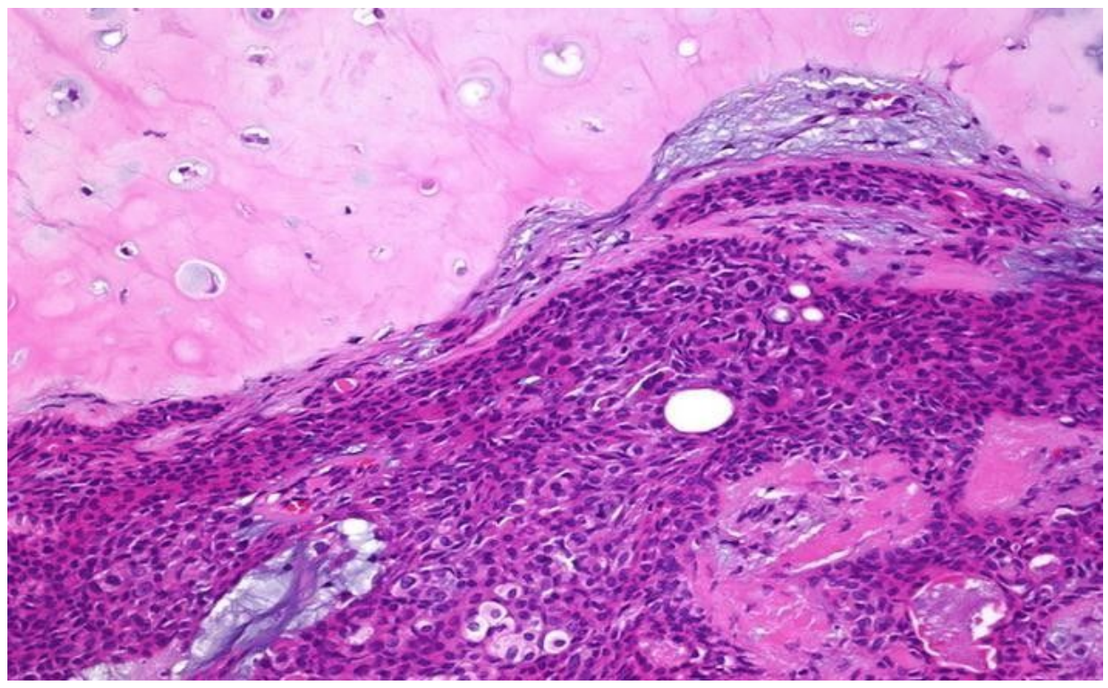


Mixed tumors are generally **benign**, have BOTH:

- Connective tissue components (i.e., usually cartilaginous)
- Glandular components = hence the name "pleomorphic or mixed", they generally look and feel like **little round soft cartilage balls**.

PLEOMORPHIC ADENOMA - Microscopically





1. Epithelial cells
2.Myoepithelial cells
3.Chondromyxoid stroma

Thank you for checking our work.

Done by:

Munerah alOmari

Lina Alshehri

Shahad AlEnezi

Noura AlTawil

Sarah AlMutawa

Nouf Altwaijri

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