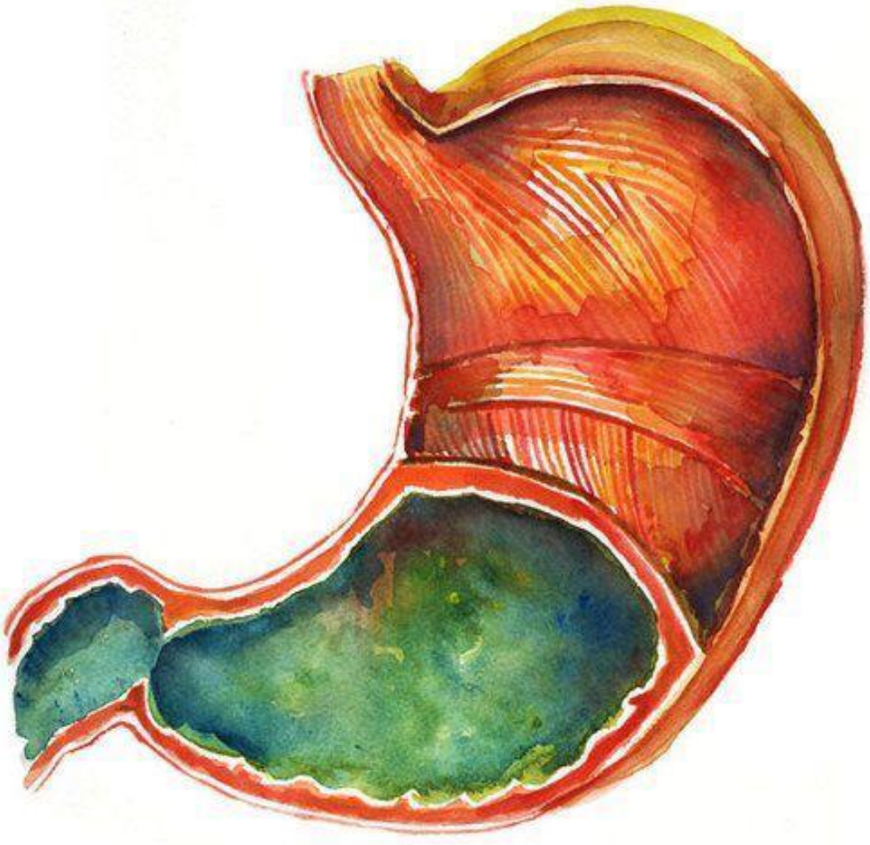
MED437
KING SAUD UNIVERSITY

Team 437 Pathology Practical



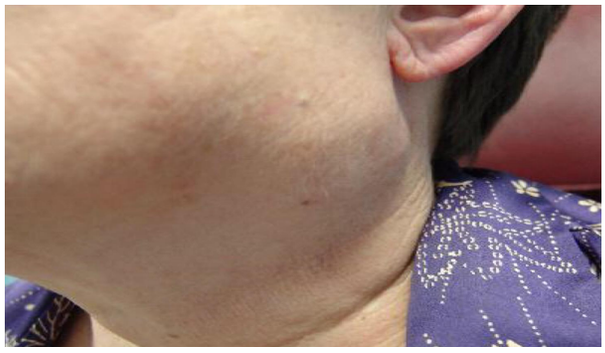
Pathology Practical

GNT Block

Grey: Notes
Pink: only in girls slides

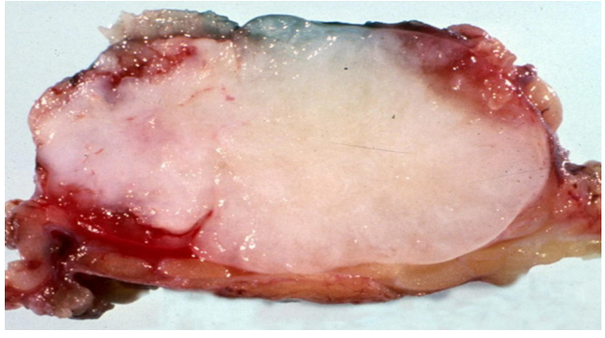
Case 1: Pleomorphic adenoma (Mixed tumor)

Gross:



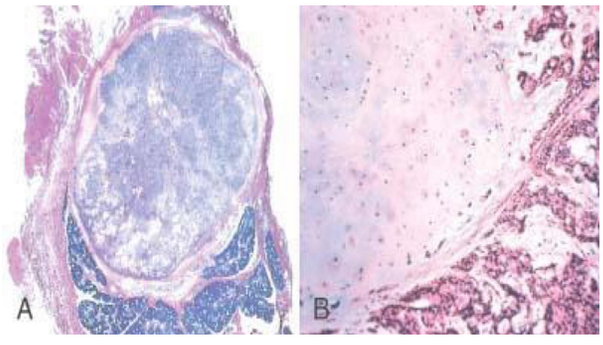
The classic place for any visible **parotid swelling or tumor** is present between the **tip of the ear** and **the tip (angle) of the mandible**

This picture shows:
- Parotid swelling

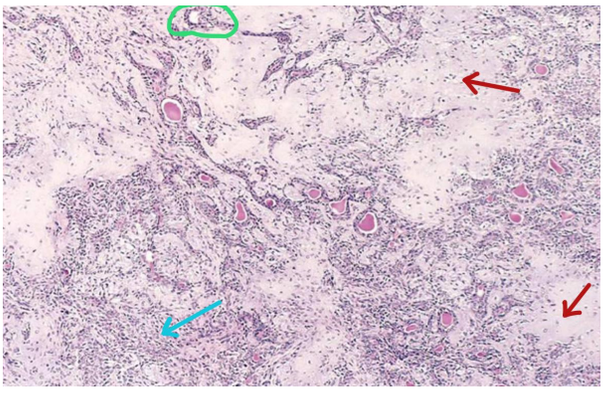


Pleomorphic adenoma (Mixed tumor)
'Benign tumor"

Microscopic view:

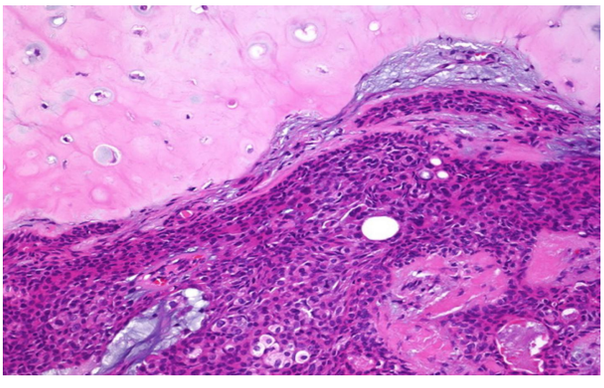


Mixed tumors are generally benign, they are:
- Have connective tissue (i.e., usually cartilagenous) components
- Have glandular components
- Generally look and feel like little round soft cartilage balls.
- Well defined encapsulated mass



Mixed tumor of the parotid gland contains:
- Epithelial cells forming ducts
- Myoepithelial cells (spindle cells)
- Chondromyxoid stroma

The same cell can differentiate into multiple types of cells like epithelial, myoepithelial cells, CT or cartilage. That's why it give the pleomorphic appearance.



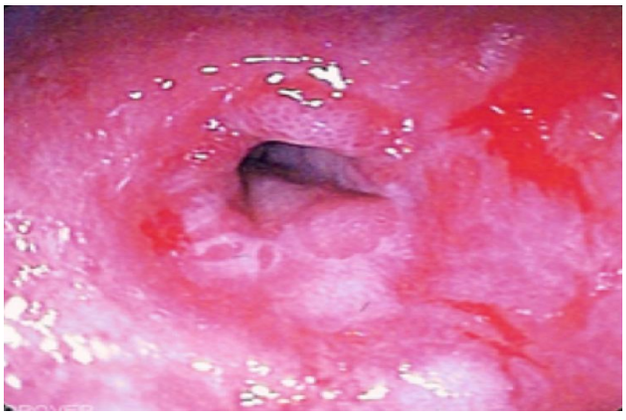
- Tumour shows mixed cellular components like epithelial, myoepithelial, chondriod and myxoid elements.
- Epithelial areas shows small ducts, acini and strands or sheets of cells.
- Myxoid areas are formed of loose myxomatous tissue and chondroid areas consist of pale blue matrix.

Case 2: Gastroesophageal Reflux Disease (GERD)

GERD:

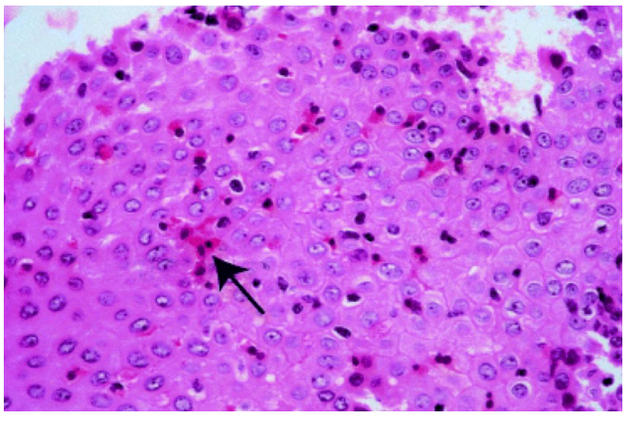
- Inflammatory Cells: Eosinophils, Neutrophils and Lymphocytes
- Basal zone hyperplasia
- Lamina Propria papillae elongated and congested

Endoscopy:



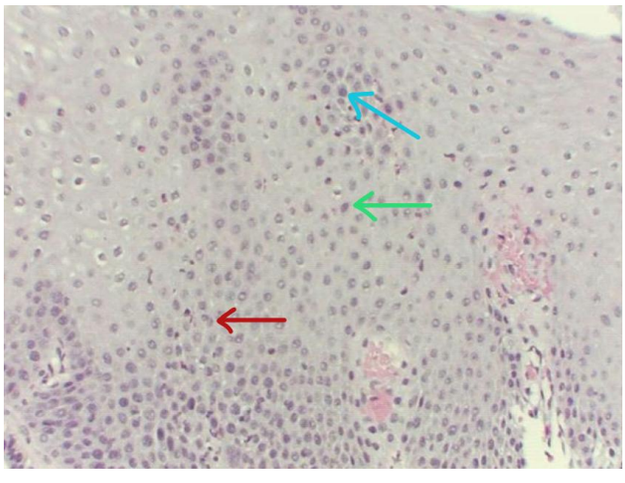
Necrosis of esophageal epithelium causing ulcers near the junction of the stomach and esophagus

Microscopic view:



It shows:
- Intraepithelial eosinophils (arrow)
- Basal cell hyperplasia

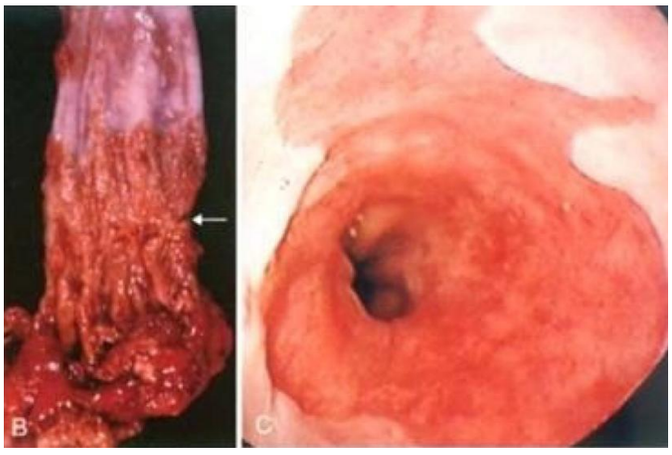
(high power, H/E stain).



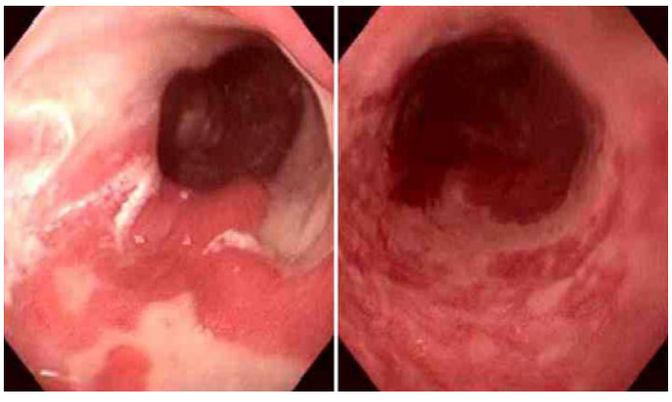
It shows:
- Inflammatory cells infiltration mainly eosinophils and neutrophils
- Basal zone hyperplasia
- Lamina Propria papillae elongated and congested

Case 3: Barrett's Esophagus

Gross/Endoscopy:

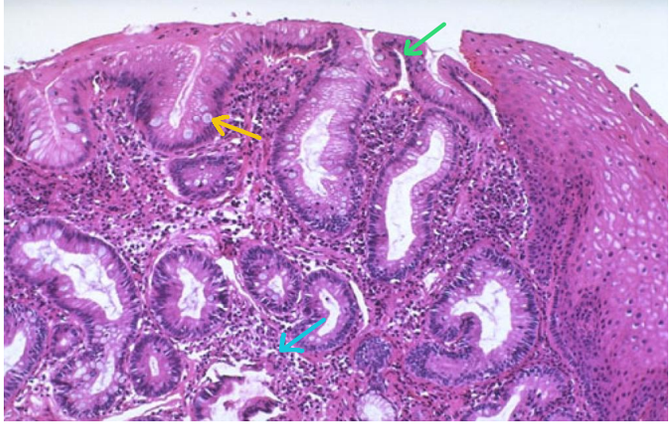


- Intestinalized metaplastic mucosa is at risk for glandular dysplasia.
- Searching for **dysplasia** when BARRETT's is present is of utmost importance.
- Most/All **adenocarcinomas** arising in the esophagus arise from previously existing BARRETT's.
- Newly named Columnar lined esophagus



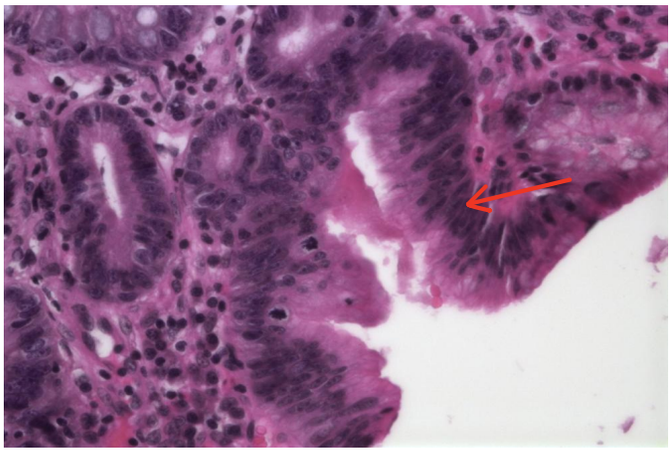
These two endoscopic views demonstrate Barrett esophagus, they shows:
- Areas of mucosal erythema of the lower esophagus
- Islands of normal pale esophageal squamous mucosa.

Microscopic view:



There is gastric-type mucosa above the gastroesophageal junction.
Typical Barrett's mucosa shows:
- Intestinal metaplasia
- Chronic inflammation
- Goblet cells in the columnar mucosa

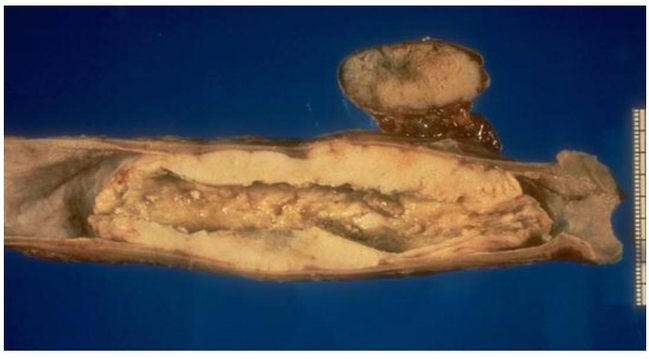
Left: columnar epithelium
Right: squamous epithelium



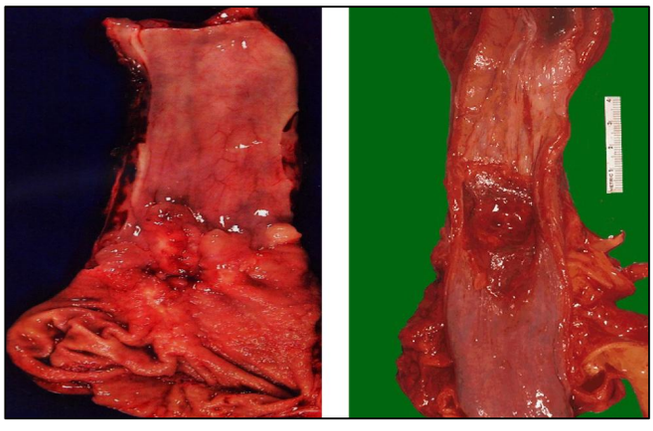
This section shows:
- Dysplasia "glandular"

Case 4: Carcinoma of Esophagus

Gross:



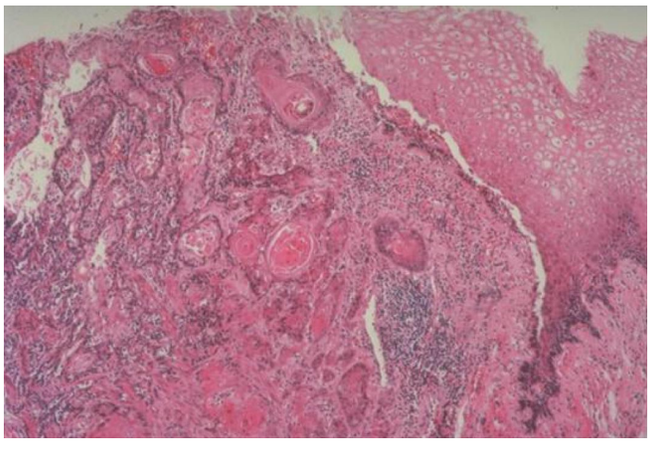
- Squamous cell carcinoma of the esophagus in a patient who presented with progressive dysphagia.
- The oval structure adjacent to the esophagus represents metatastic squamous cell carcinoma within a lymph node.
- There is Ulceration of the surface epithelium



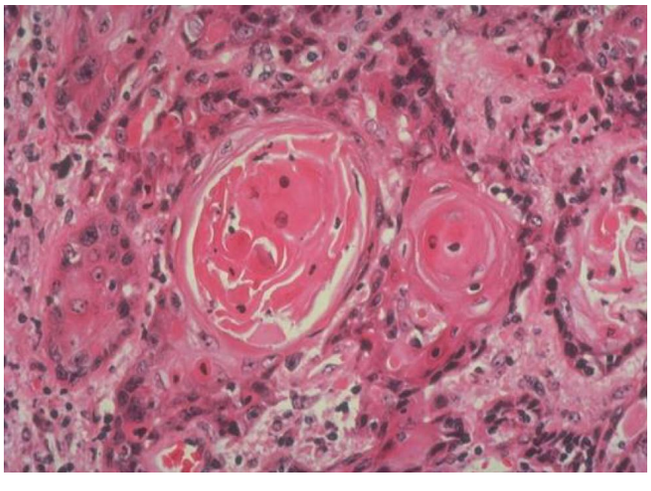
Mucosal surface is a squamous cell carcinoma shows:
- Irregular reddish
- Ulcerated exophytic mid-esophageal mass

Endoscopic views of an ulcerated mid-esophageal squamous cell carcinoma causing **luminal stenosis**
Predisposing factors: Acalasia(most important), Smoking, Alcohol, Caustic oesophageal injury, Plummer-Vinson syndrome

Squamous cell carcinoma Microscopic view:

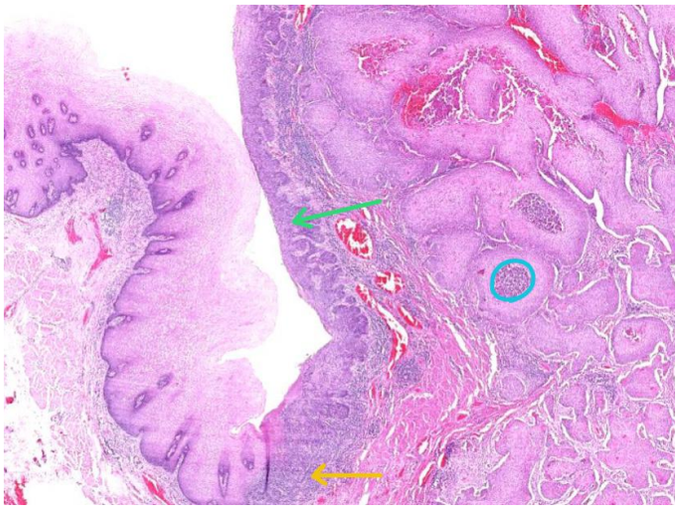


Infiltrating nests of neoplastic cells (Desmoplastic response "Inflammation surrounded by fibrosis')

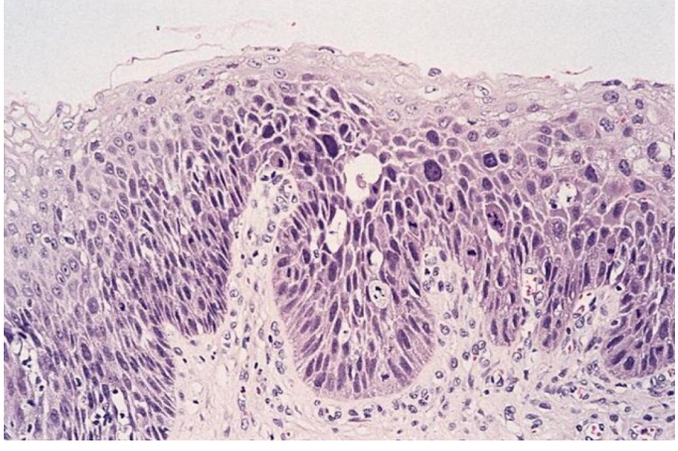


(Keratin pearls)
Solid nests of neoplastic cells having:
- Abundant pink cytoplasm
- Distinct cell borders
- Keratin material
- fibrosis with chronic inflammation

Squamous Dysplasia of the Esophagus Microscopic view:



It shows:
- Atypical squamous cells with disorganized architecture and abnormal differentiation within the epithelium (These features are obvious in **high grade dysplasia**)
- The nuclei are larger and more hyperchromatic than normal
- Increased mitotic activity
- Invasive squamous cell carcinoma arraigned in nests of malignant cells
- Chronic Inflammatory cells and fibrosis

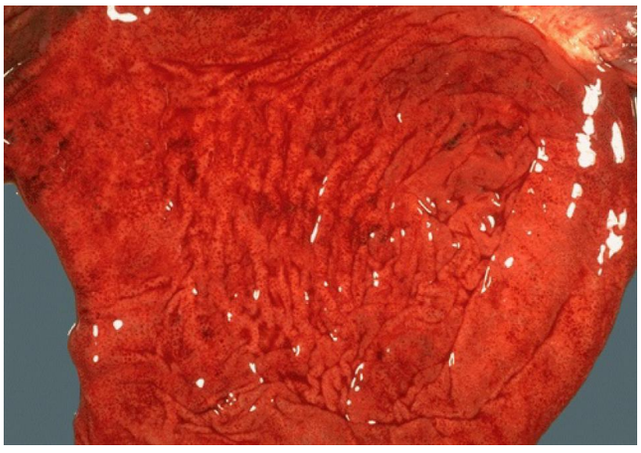


Squamous dysplasia of the esophagus may develop with time into squamous cell carcinoma

There is:
- Pleomorphism
- Hyperchromatism
- Increased C ratio and mitosis
- Involvement of the entire thickness “severe dysplasia”
- No invasion

Case 5: Acute Gastritis "Only in male slides"

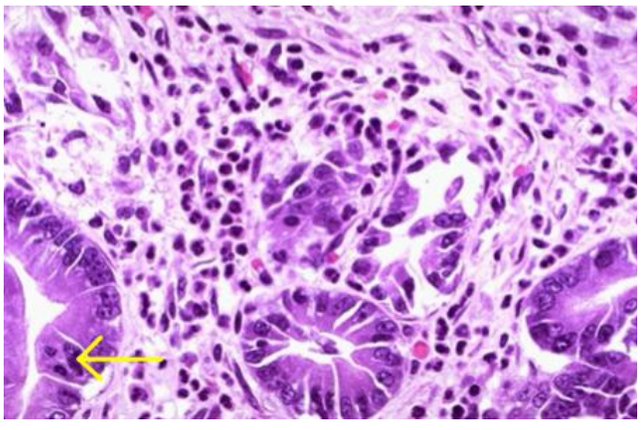
Gross:



This is a more typical acute gastritis with a diffusely hyperemic gastric mucosa.

There are many causes for acute gastritis, examples:
- Alcoholism
- Drugs
- Infections

Microscopic view:



Gastric mucosa demonstrates infiltration by neutrophils.

(At HPF)

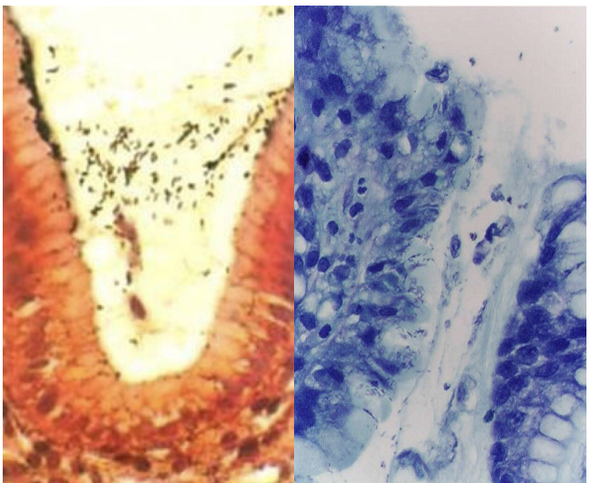
Case 6: Chronic Gastritis

Microscopic view:

There is:
- No erosions, no hemorrhage.
- Some neutrophils.
- Lymphocytes , lymphoid follicles.
- Regenerative changes:
  - Metaplasia (intestinal)
  - Atrophy: mucosal hypoplasia "thinning"
  - Dysplasia

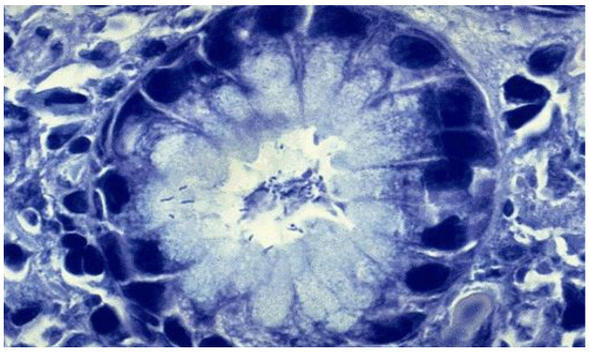
Case 7: Helicobacter-induced gastritis

Helicobacter pylori:



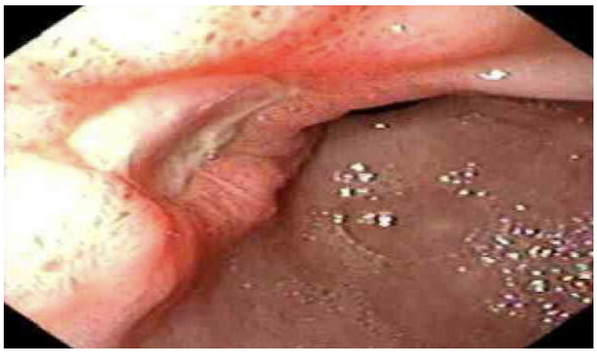
Gastric biopsy using:
- Left: Silver stain
- Right: Giemsa stain

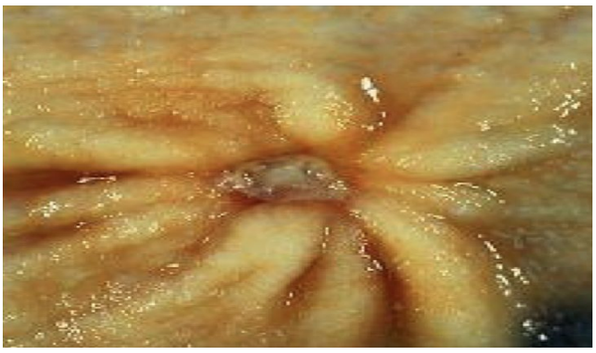
Microscopic view:



- Gastritis is often accompanied by infection with **Helicobacter pylori**.
- 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**

Peptic ulcer:





- "PEPTIC" implies acid cause/aggravation
- Ulcer vs. Erosion (muscularis mucosa intact in Erosion)
- Mucosa → Submucosa → Muscularis → Serosa

Ulcers are:
- Chronic
- Solitary (usually)
- Adults

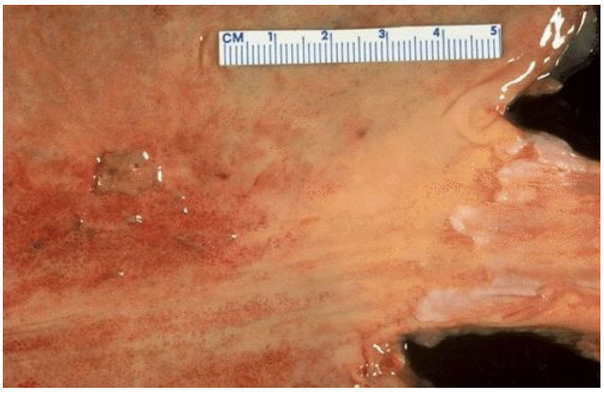
In female slides this info. was mentioned with chronic gastric ulcer case (because they are chronic)

Causes:
- 80% caused by H. pylori
- NSAIDs
- Stress

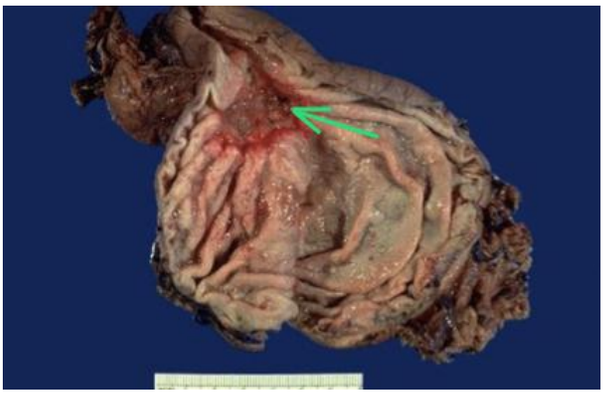
Only in male slides

Case 8: Acute gastric ulcer

Gross:



Acute Gastric ulcer in the upper fundus that is:
- **Benign** 1 cm.
- Shallow and sharply demarcated
- Surrounded by hyperemia.
- It is probably benign.

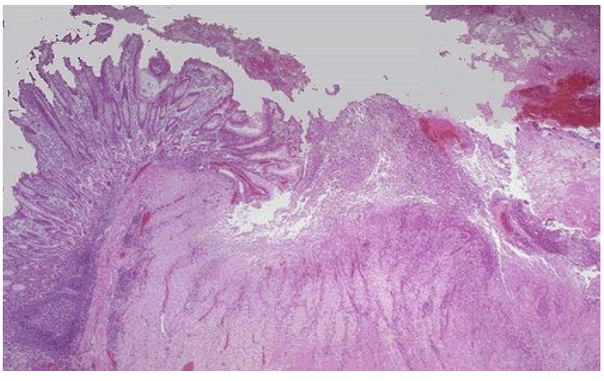


Stomach resection shows:
- **Malignant,** much larger 3 x 4 cm gastric ulcer (which led to the resection)
- The ulcer is much deeper with more irregular margins.

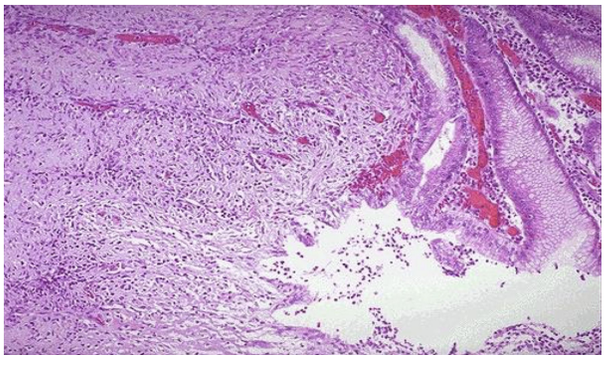
All gastric ulcers (either benign or malignant) should be biopsied to rule out a malignancy.

Complications of gastric ulcers (either benign or malignant):
- Pain. - Bleeding. - Perforation. - Obstruction.

Microscopic view:



LPF microscopy shows:
- The ulcer here is sharply demarcated
- **On the left:** Normal gastric mucosa falling away into a deep ulcer whose base contains inflamed, necrotic debris.
- An arterial branch at the ulcer base is eroded and bleeding.

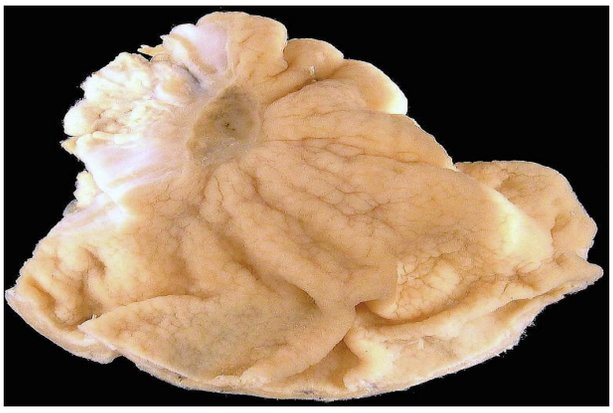


HPF microscopy shows:
- At the upper right: The mucosa merges into the ulcer at the left which is eroding through the mucosa.
- Ulcers will penetrate over time if they do not heal.
- Penetration leads to pain.

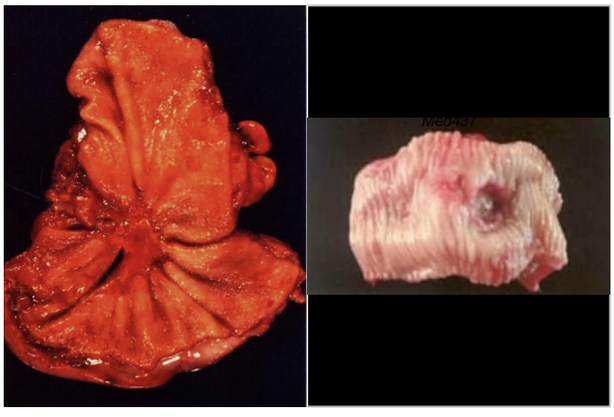
Perforated ulcer: If the ulcer penetrates through the muscularis and through adventitia, and leads to an acute abdomen.

Case 9: Chronic Gastric Ulcer

Gross:



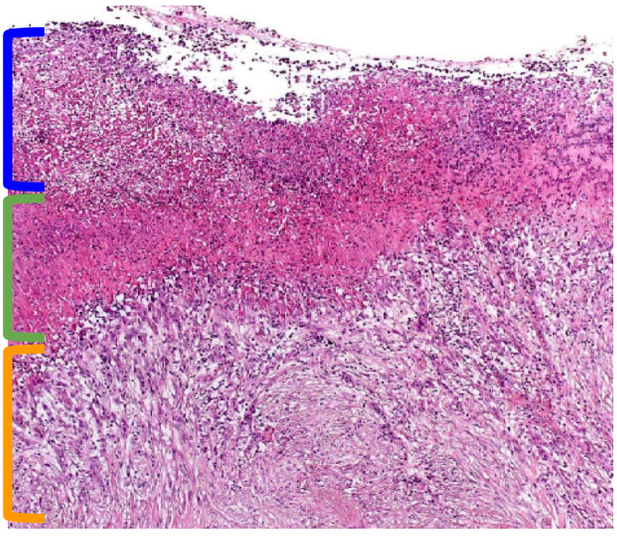
The specimen has been cut to show the submucosa, muscle coat and adventitial connective tissues in the region of the ulcer
It shows:
- Irregular portion of gastric wall.
- Oval shape ulcer
- Deeply penetrating ulcer
- Necrotic debris covers the base.



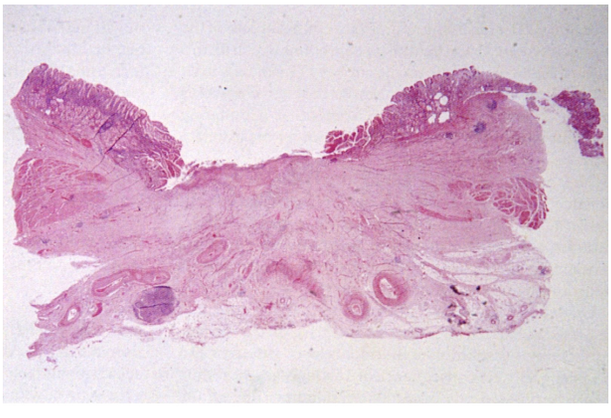
The (Left) picture shows:
- Well defined
- It is about 1 to 2 cm in size (can be 10 Cm)
- Smooth and red surface
- Necrotic based
- Usually it's solitary

The (Right) picture Nothing was mentioned

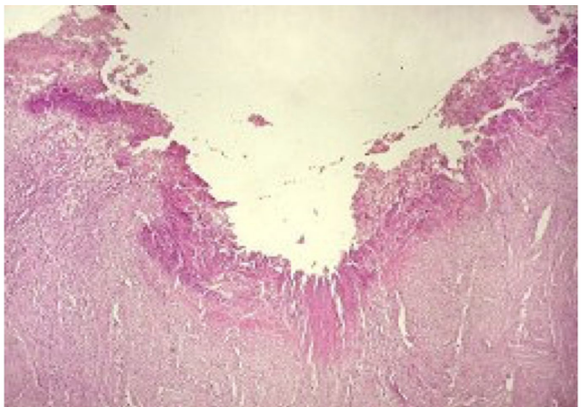
Microscopic view:



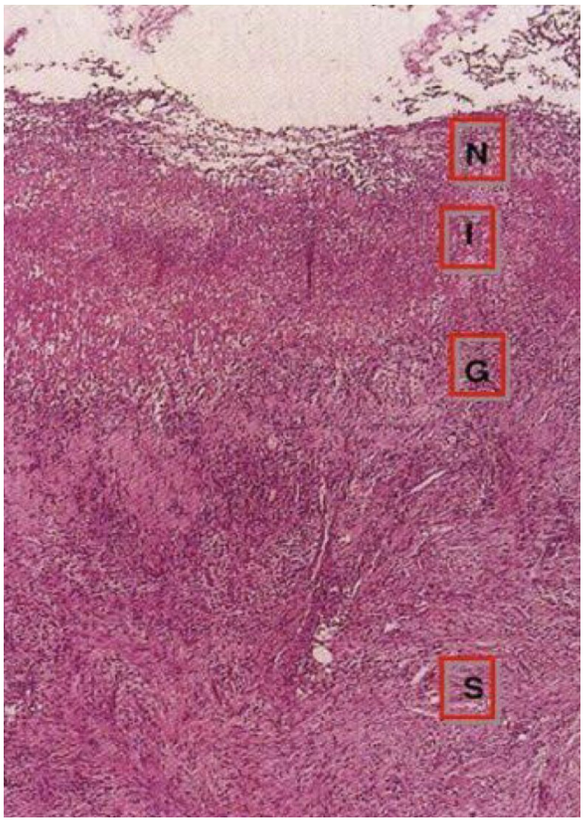
Microscopic examination shows the typical features of a chronic peptic ulcer in the antrum.
The picture shows:
-Cellular Debris: Numerous viable and degenerate polymorphs.
-Fibrinoid Necrosis: Inflammatory cells and granulation tissue.
-Granulation Tissue: Variable sized capillary channels are separated by fibroblastic connective tissue heavily infiltrated with lymphocytes, neutrophils, and eosinophils.



It shows:
- Shallow margin
- Gradual loss of mucosa then full thickness loss of mucosa (ulceration)
- The base contains (necrosis + inflammation + granulation + fibrosis)



This picture shows the base which contains:
- Fibrinopurulent exudate
- Inflammation
- Granulation
- Fibrosis

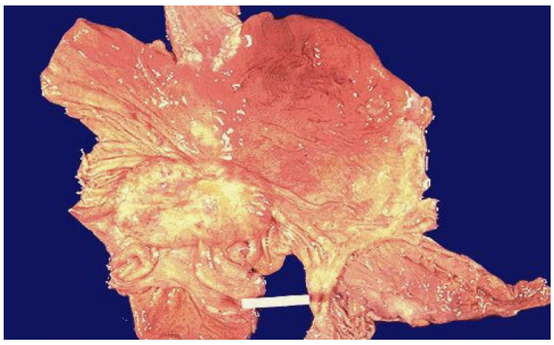


The Base of Non-perforated chronic peptic Ulcer:
- Necrosis (N)
- Inflammation (I)
- Granulation tissue (G)
- Scar (Fibrosis)(S)

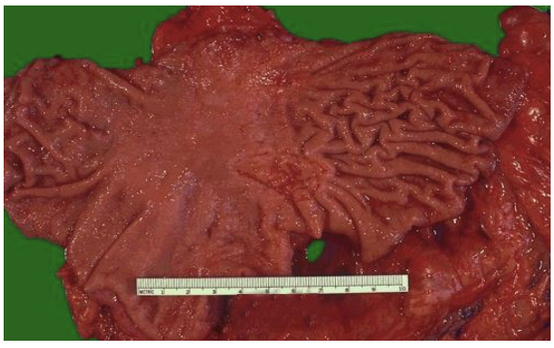
(Top -luminal surface, Bottom muscular wall)

Case 10: Carcinoma of the stomach

Gastric Adenocarcinoma Gross:

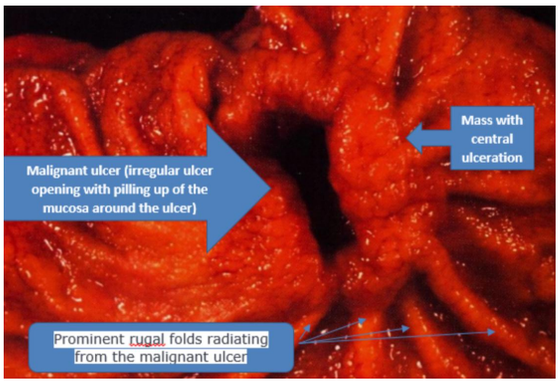


- Gastric Neoplasia is common
- It is **not** possible to rule out a malignancy from gross appearance alone
- Biopsy should be done to differentiate between benign and malignant
- **This picture shows:** gastric adenocarcinoma.

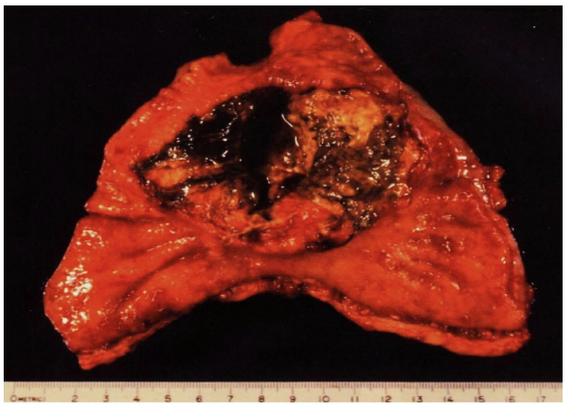


Gastric Adenocarcinoma with ulcer , shows:
- Gastric ulcer in the center
- It is shallow
- It is about 2 to 4 cm in size

On biopsy this ulcer proved to be malignant, so the stomach was resected as shown here

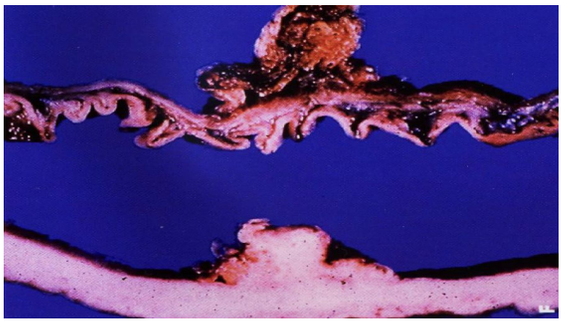


It is adenocarcinoma with raised mass have central irregular ulcer with raised pilling up everted edges.



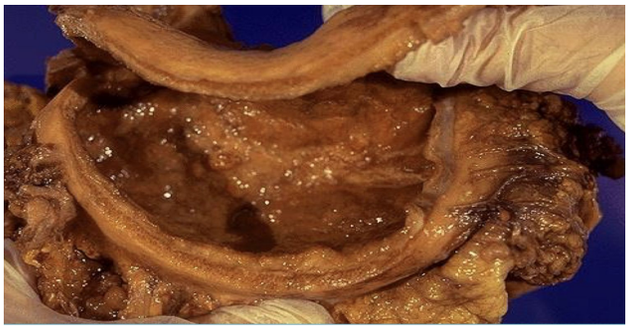
The picture shows:
- localized adenocarcinoma
- Single mass with ulceration
- in the center of the stomach

-The mass may contains :
H.pylori Infection , gastritis, Atrophy, dysplasia, without cancer



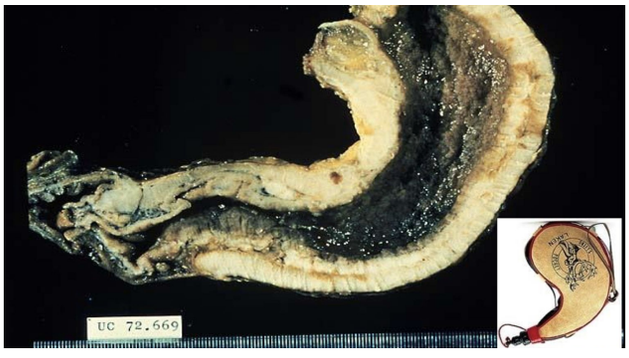
The picture shows:
- Normal stomach wall (the upper part)
- Diffuse type which involve All layers of the stomach (the lower part)

Gross: Gastric Adenocarcinoma (Lentis Plastica)



There is:
- A shrunken "leather bottle" stomach due to **diffuse infiltrative gastric adenocarcinoma**
- Extensive mucosal erosion
- Markedly thickened gastric wall

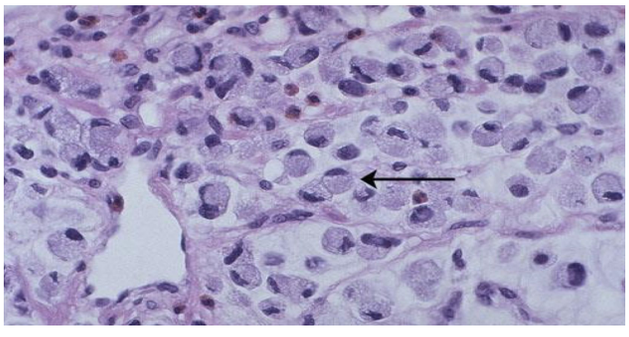
This type of carcinoma has a very poor prognosis



LINITIS PLASTICA Is the most spectacular and feared
There is :
- Grows diffusely through all layers
- Greatly thickening its wall
- A classic leather bottle appearance.

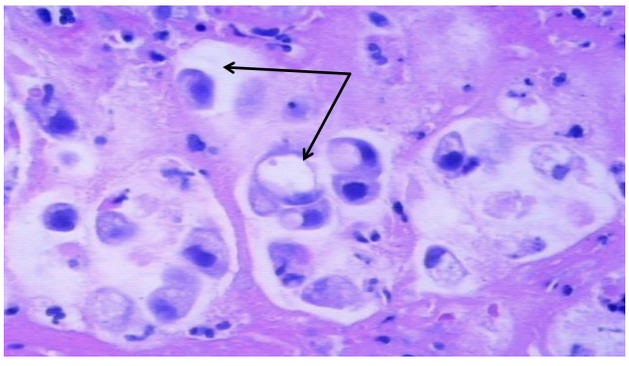
It has a horrible prognosis.

Microscopic: Gastric Adenocarcinoma



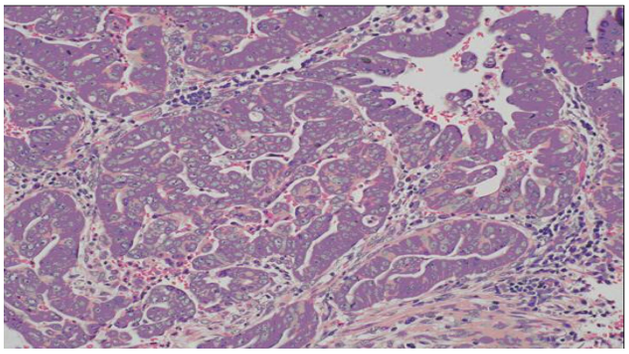
Signet Ring Cell -HPF

There is :
- A signet ring cell pattern
- Filled cell with **mucin (not fat)** vacuoles push the nucleus peripherally (shown at the arrow.)



Signet Ring Cell -HPF

There is :
- Poorly differentiated signet ring cell (often seen with Lentis Plastica)
- Large holes in the cytoplasm(shown at the arrow.) represents intracellular mucin which push the nucleus peripherally

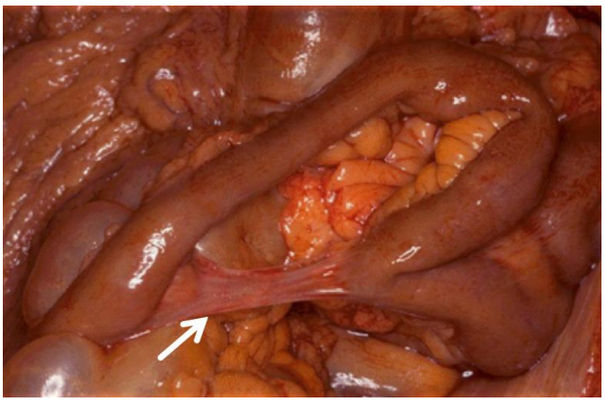


Photomicrograph of a poorly differential intestinal type adenocarcinoma of the stomach

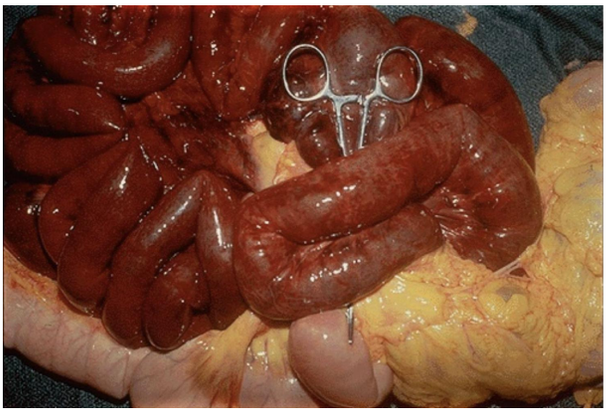
Only in male slides

Case 11 : Small intestinal infarction

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



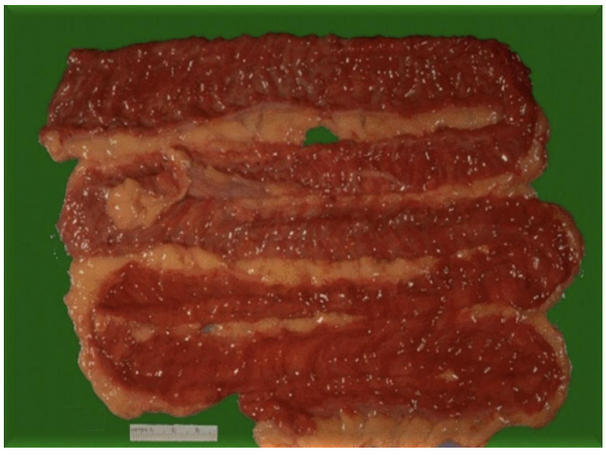
-The dark red infarcted small intestine contrasts with the light pink viable bowel.

-The forceps extend through an internal hernia in which a loop of bowel and mesentery has been caught.

-This is one complication of adhesions from previous surgery. The trapped bowel has lost its blood supply.

Ischemic Enteritis

Gross :

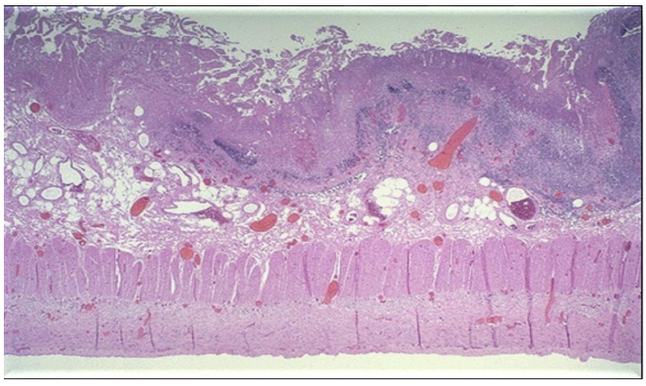


The small intestinal mucosa demonstrates marked hyperemia as a result of ischemic enteritis.
Such ischemia most often results from:
- Hypotension (shock), from Cardiac failure
- Marked blood loss
- Loss of blood supply from mechanical obstruction (as with the bowel strangulated in a hernia or with volvulus or intussusception).

If the blood supply is not quickly restored, the bowel will infarct.

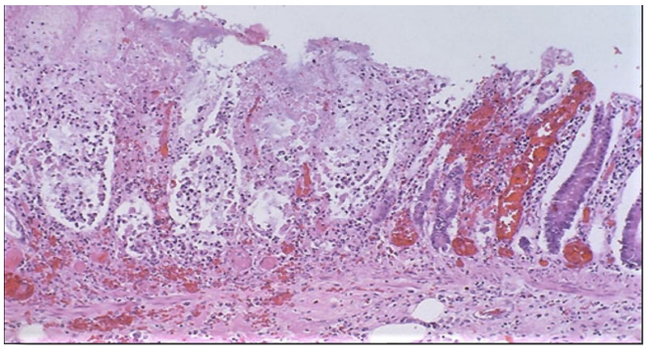
- On closer inspection, early ischemic enteritis involves the tips of the villi.
- Bowel is hard to infarct from atherosclerotic vascular narrowing or thromboembolization because of the widely anastomosing blood supply.
- Most cases of bowel ischemia and infarction result from generalized hypotension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

Microscopic view:



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

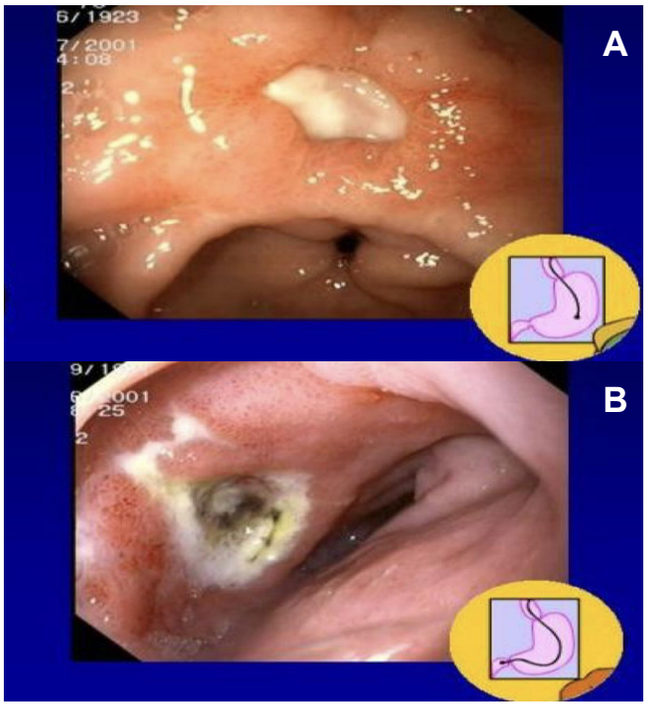
(LPF)



At higher magnification with more advanced necrosis, the small intestinal mucosa shows:
- Hemorrhage
- Acute inflammation

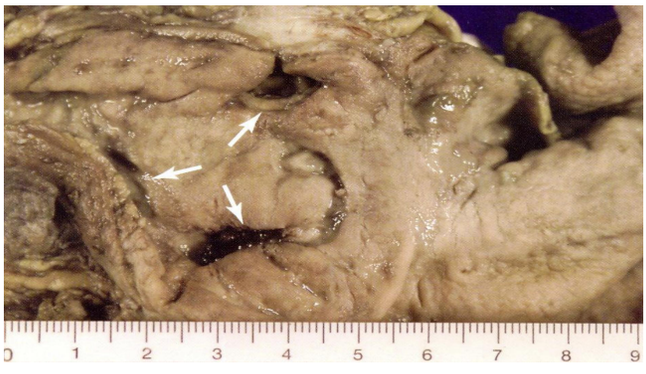
Case12 : Chronic duodenal ulcer

Gross :



A/ Gastric Ulcer:
The ulcer has a clean white base and some swelling around its edges

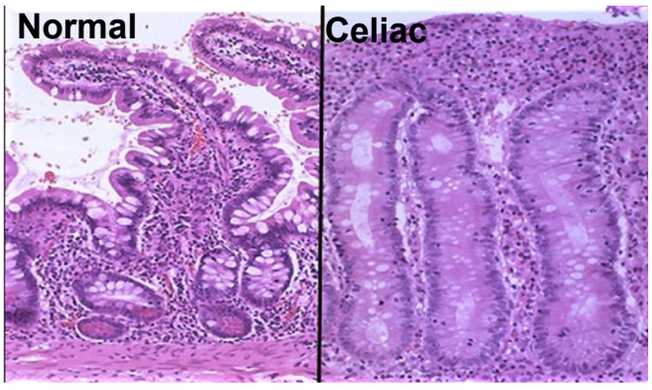
B/ Duodenal Ulcer
The white base of the ulcer is marked by a blackish area signaling a recently bleeding vessel



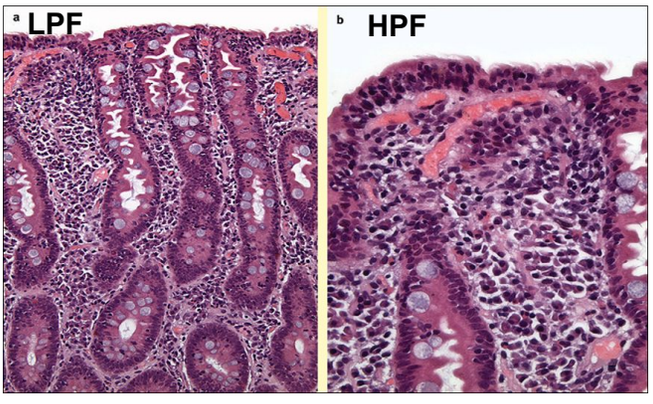
- The ulcer occur mainly in the first part of duodenum.
- It related to H.pylori infection.
- It appear as single ulcerated area.

Case 13: Celiac disease

Microscopic view:

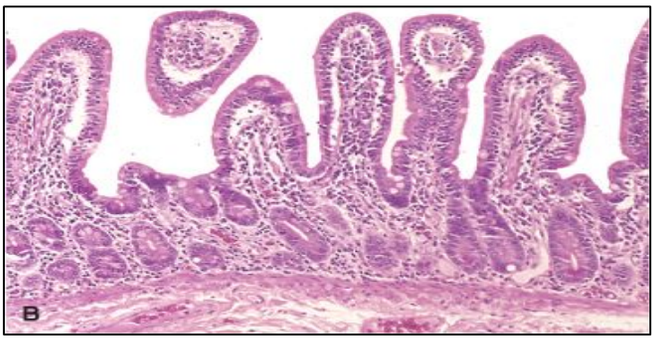


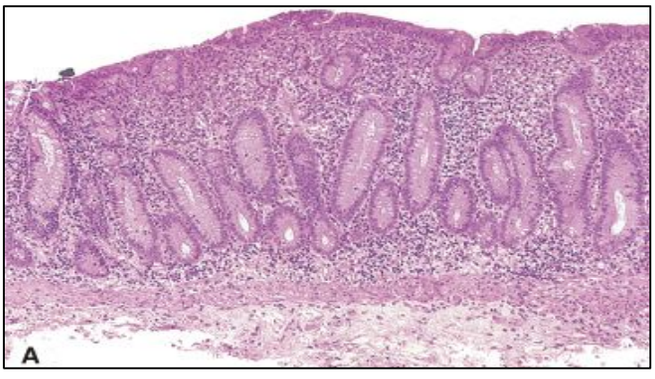
- Normal small intestinal mucosa is seen at the left.
- The mucosa involved by celiac disease (sprue) at the right has blunting and flattening of villi.
- Celiac disease most often becomes apparent either in infancy, or in young to middle age adults.



Low-power view (LPF) of fully developed sprue-type changes. Note the:
- Elongated crypts
- Complete lack of villi.

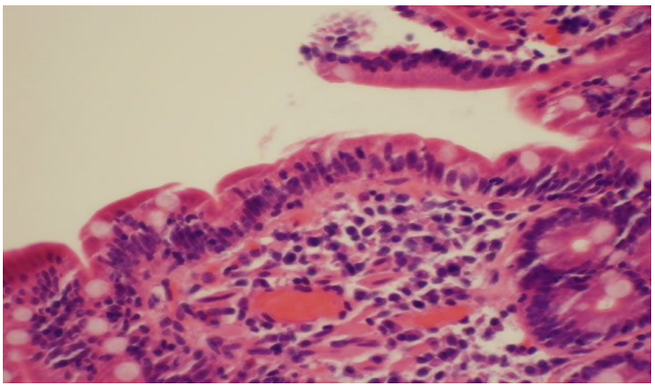
High-power view (HPF) showing:
- Damaged surface epithelium
- Large numbers of intraepithelial lymphocytes.





B/ Villous length to crypt length 3/1

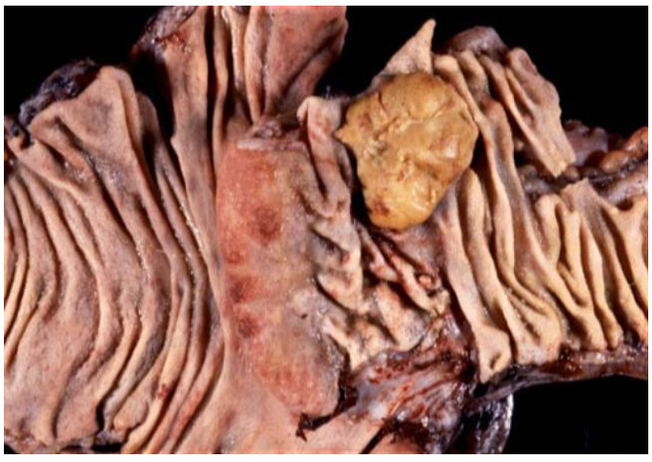
In celiac disease:
- In the beginning there will be attack to the lining epithelium by lymphocyte
- Injury to the lining epithelium then regenerate like surface without vill,So there will be **villous atrophy like in A picture**



Three important features:
- Intraepithelial lymphocytosis
- Loss of the villi
- Hyperplasia of the crypt with elongation of the crypt.

Case 14: Carcinoid tumor of small intestine

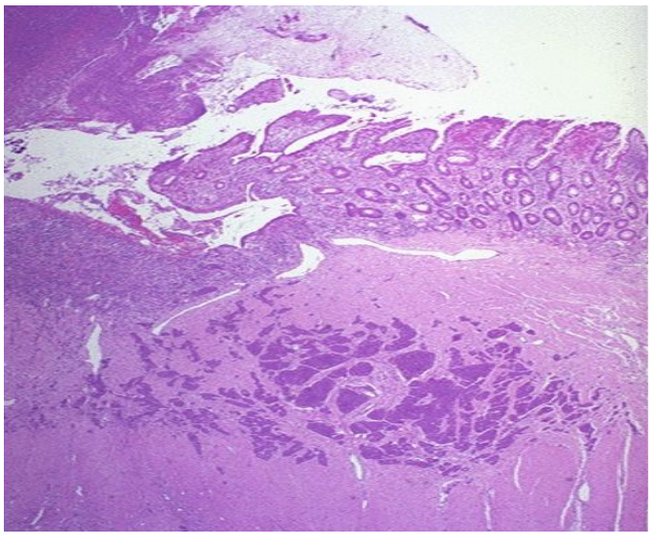
Gross:



Neoplasms of the small intestine are uncommon. Benign tumors can include: leiomyomas, fibromas, neurofibromas, and lipomas.

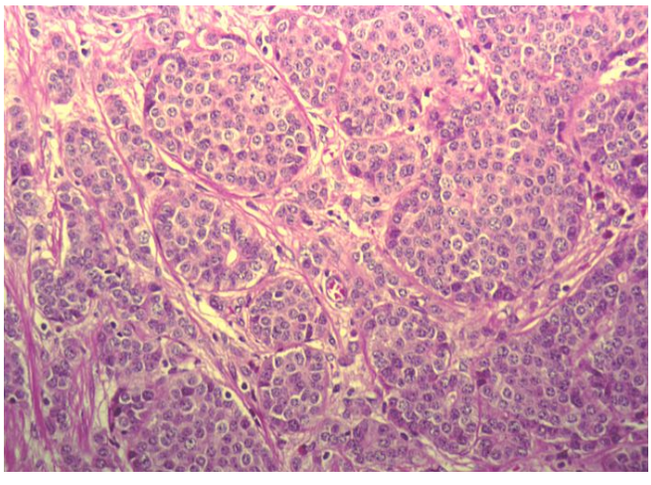
- Seen here at the ileocecal valve is another tumor that has a faint yellowish color.
- This is a carcinoid tumor.
- Most benign tumors are incidental submucosal lesions, though rarely they can be large enough to obstruct the lumen.

Microscopic view:



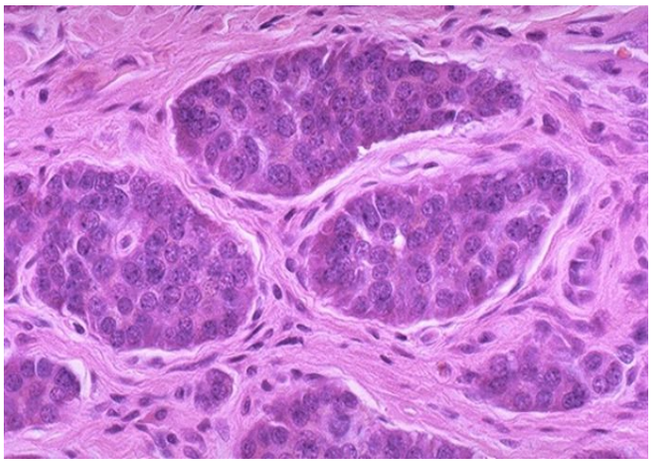
The carcinoid tumor is seen here to be a discreet, though not encapsulated, mass of multiple nests of small blue cells in the submucosa.

(LPF)



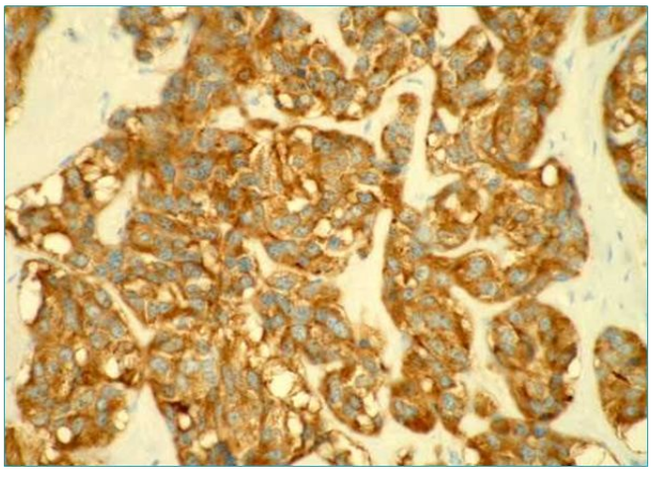
Tumour consists of alveolar groups and clumps of small uniform polygonal cells (arranged in nests) having:
- Centrally placed round nuclei
- Abundant granular cytoplasm (Pink cytoplasm)

If checked with an electron microscope we will see neurosecretory granules.
(MPF)



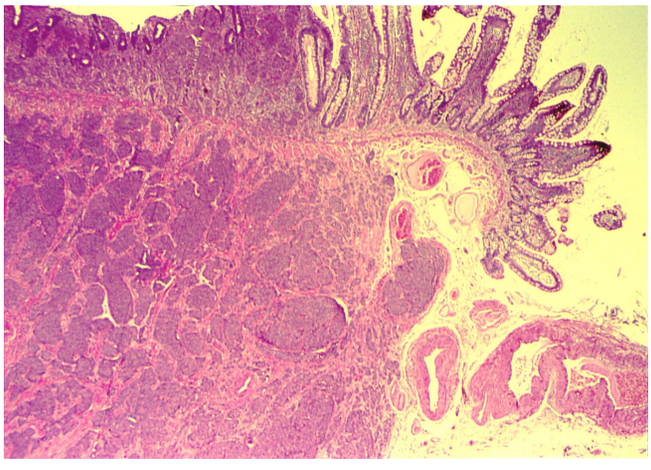
At high magnification,the nests of carcinoid tumor have:
- Typical endocrine appearance
- Small round cells having small round nuclei and pink to pale blue cytoplasm.

Rarely, a malignant carcinoid tumor can occur as a large bulky mass. Metastatic carcinoid to the liver can rarely result in the carcinoid syndrome.



Carcinoid tumor showing strong positive staining with the synaptophysin immunohistochemical stain (IHC stain).

This finding confirms the neuroendocrine nature of this neoplasm.

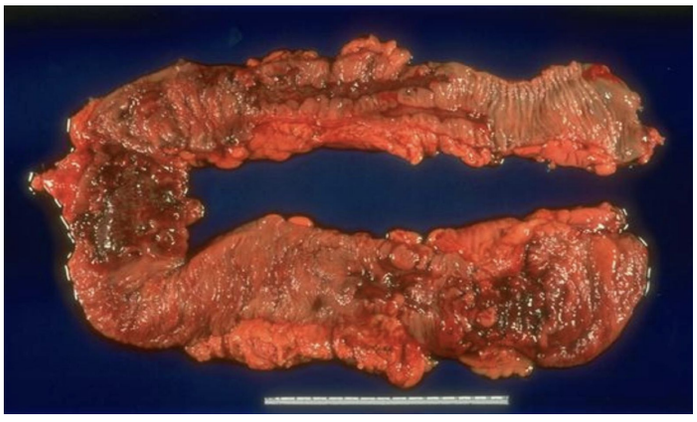


Carcinoid tumor is the most common tumor of small intestine,It is a malignant tumor arising from neuroendocrine cells (produce hormones) along intestinal Tract.

Section of small intestine shows:
- Surface ulceration
- Infiltrating tumour mass in mucosa and submucosa.

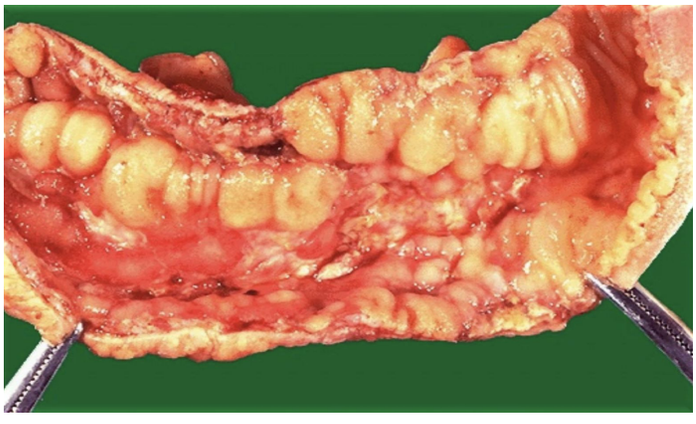
Case 15: Crohn's disease

Gross:

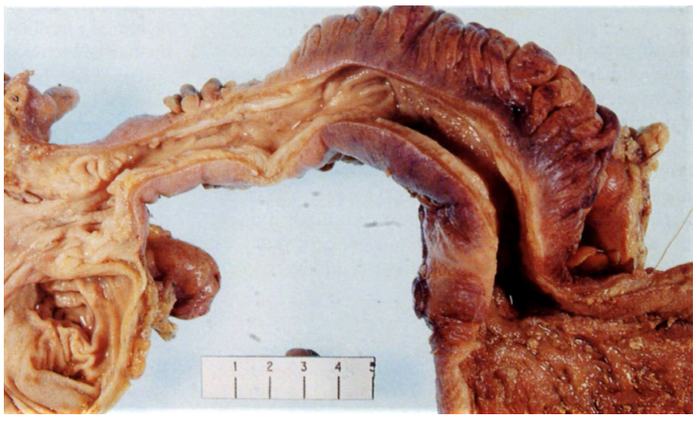


The inflammation has produced d that are:
- Large
- Irregularly shaped to rake-like
- Separated from each other by mucosa that appears close to normal.

Skip lesion



The mucosal surface of the small intestine demonstrates:
- An irregular nodular appearance
- Hyperemia
- Focal superficial ulceration.

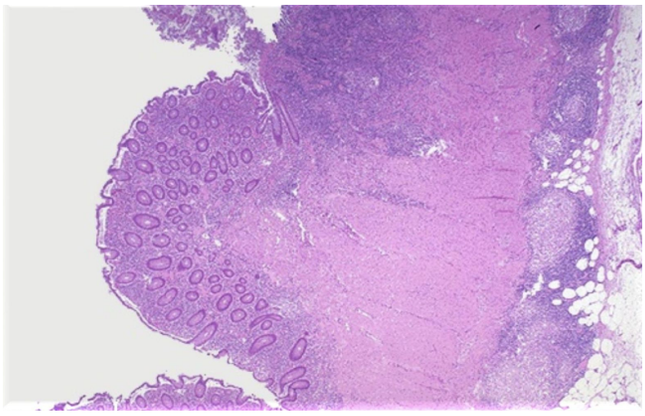


Section of large bowel shows alternating normal and ulcerating mucosa
- Transmural inflammation
- Thickened wall

- Linear mucosal ulcers and thickened intestinal wall.

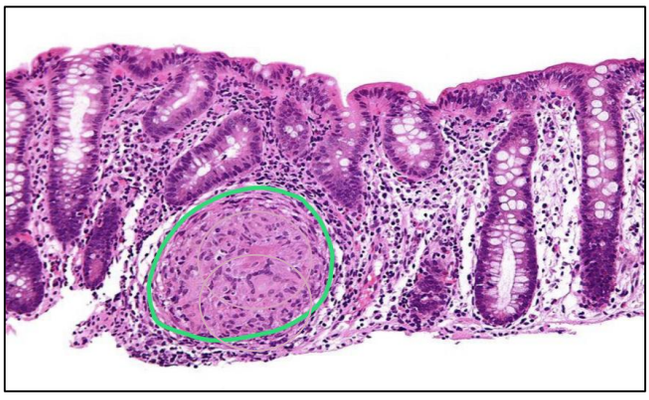
- Longitudinal irregular ulcers.

Microscopic view:



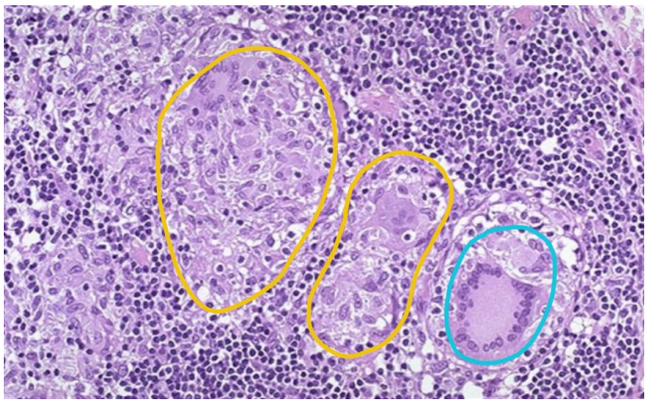
Crohn's disease is characterized by transmural inflammation.

LPF microscopy shows:
- Inflammatory cells (the bluish infiltrates) extend from mucosa through submucosa and muscularis
- It appear as nodular infiltrates on the serosal surface with pale granulomatous centers.



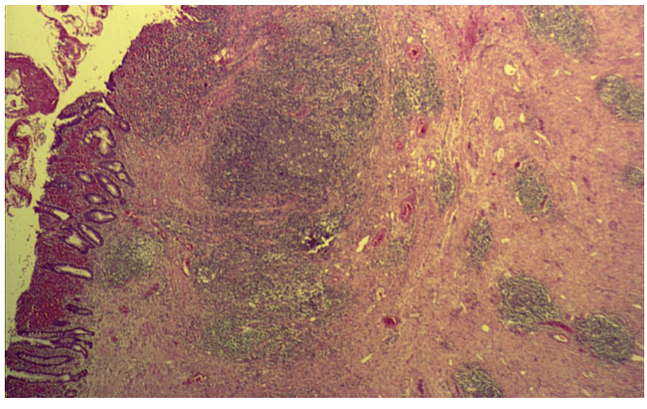
HPF microscopy of all layers of intestinal wall show:
- Transmural chronic inflammatory cell infiltrate.
- Lymphoid aggregates
- Mild fibrosis.

Subserosa contains few non-necrotizing (non-caseating) epithelioid granulomas



Granulomatous nature of the inflammation of Crohn's disease is demonstrated here with (At HPF):
- Epithelioid cells
- Giant cells
- Many lymphocytes.

Special stains for organisms are negative.

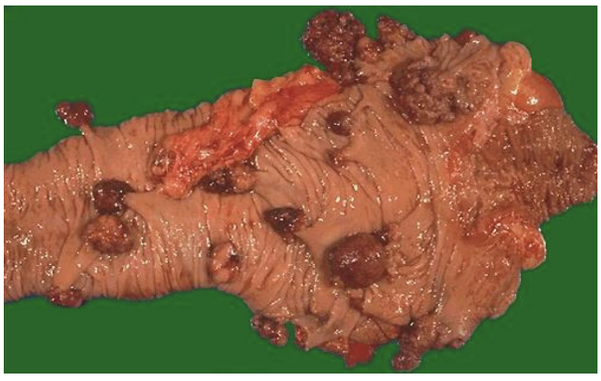


In the Large bowel:
- Lymphoid aggregate (The bluish area)

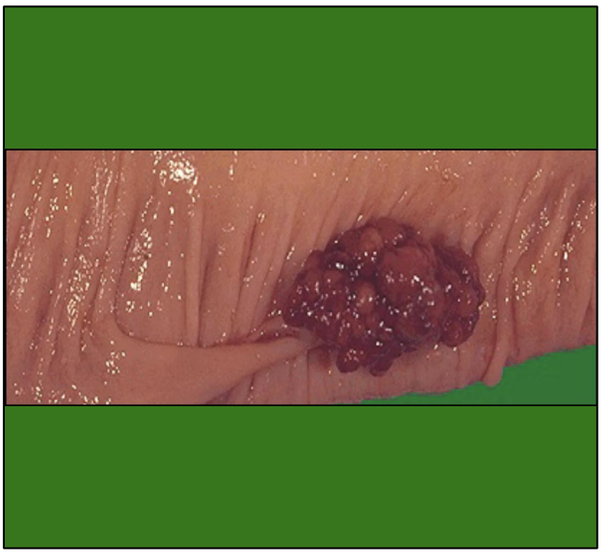
Extra-hepatic manifestations:
- Uveitis
- Pyoderma nodosum
- Migratory polyarthritis

Case 16: Adenomatous polyps of rectum / colon

Adenomatous polyp of the colon-Gross



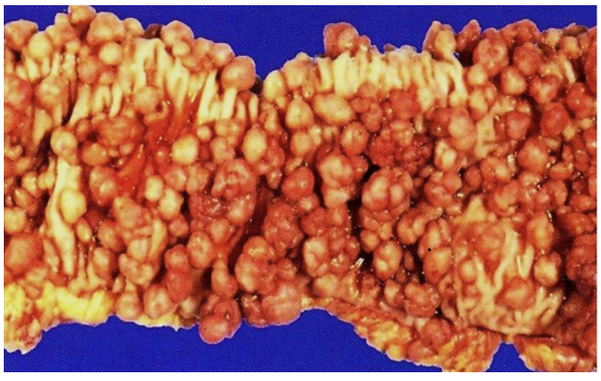
Multiple adenomatous polyps (tubulovillous adenomas) of the cecum are seen here in a case of **familial adenomatous polyposis**, a genetic syndrome in which an abnormal genetic mutation leads to development of multiple neoplasms in the colon.



This adenomatous polyp (projection above the surface) **has:**
- a hemorrhagic surface (which is why they may first be detected with stool occult blood screening)
- 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

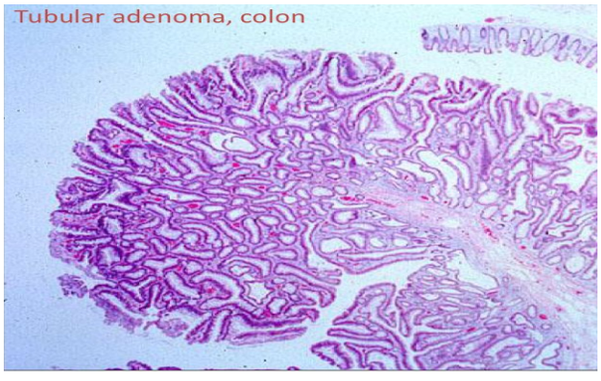
The polyp May occur due to **infection**, **inflammation**, **hyperplastic**, **neoplastic** .



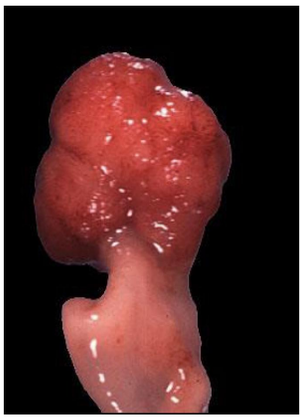
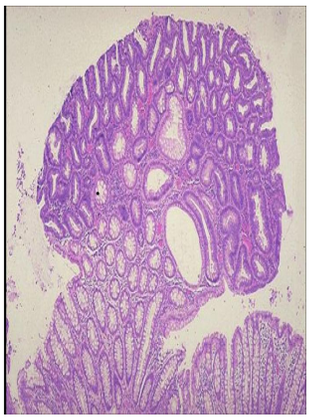
Familial polyposis of the colon
- It is caused by mutations of the adenomatous polyposis coli , or **APC** gene , in chromosome 5
- The major complication is development of **adenocarcinoma** of the colon.

Familial multiple polyps vary in size , and should be removed by surgery to avoid cancer .

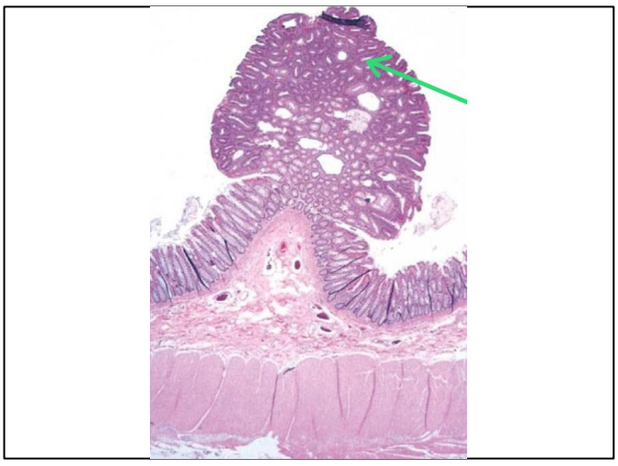
Microscopic view:



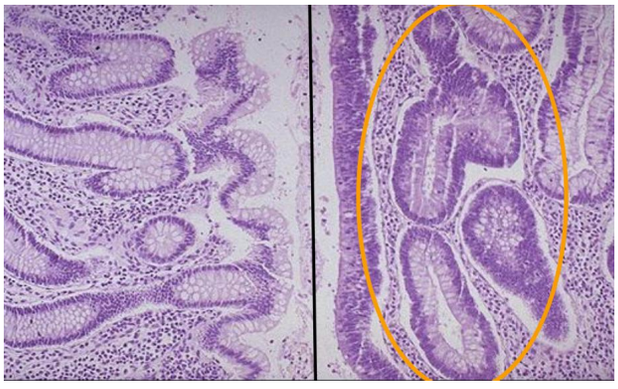
- Darker lining epithelium due to dysplasia, hyperchromasia, more crowded cells so it's **adenoma**
- **Benign** because there is no invasive component

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.
- Less Goblet cells
- Cells lining the glands of the polyp are hyperchromatic nuclei.

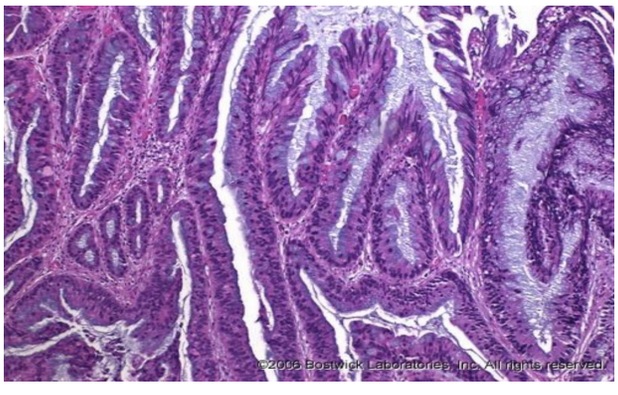


Adenoma has:
- Small neck (pedunculated)
- Darker lining epithelium due to dysplasia
- Larger nuclei
- More crowded cells



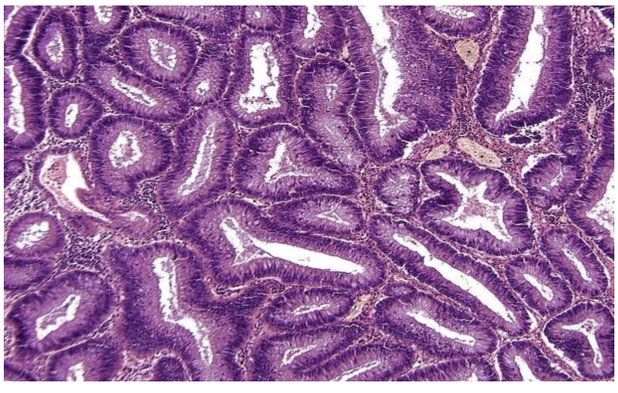
Left: Normal colonic mucosa
Right: An adenomatous polyp (tubular adenoma)

The neoplastic glands are:
- More irregular
- Have darker (hyperchromatic) and more crowded nuclei (dysplastic epithelium)



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

- The growth appear as a finger like projection, it has high grade to progress to cancer.
- Multiple villi have thin fibrovascular core .

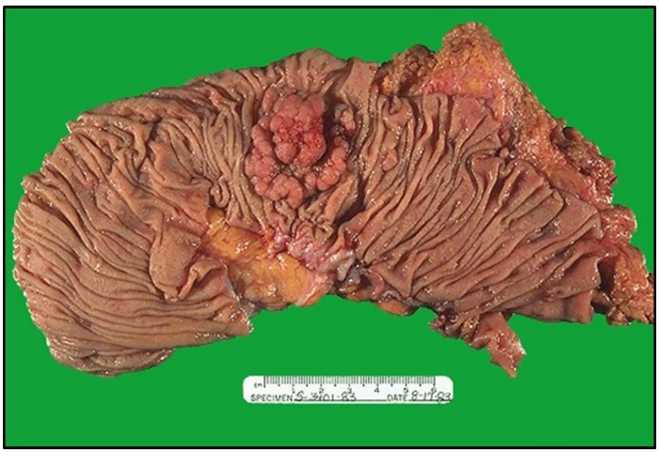


TUBULAR adenoma with:
- Crowded dysplastic glands
- Chronic inflammation.

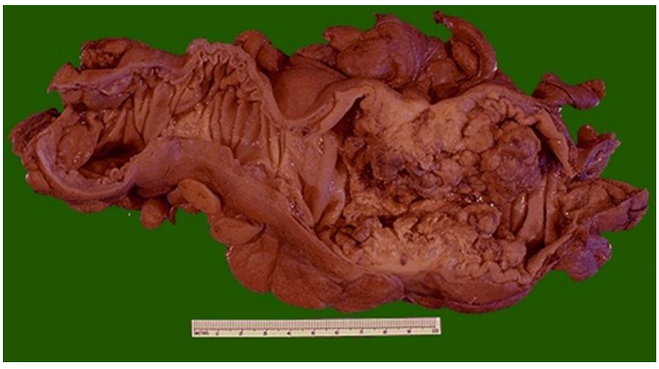
Its lining epithelial is crowded, hyperchromatic with increased nuclear cytoplasmic ratio and increased mitosis (features of dysplasia)

Case 17 :Adenocarcinoma of the large intestine

Adenocarcinoma of the Colon - Gross

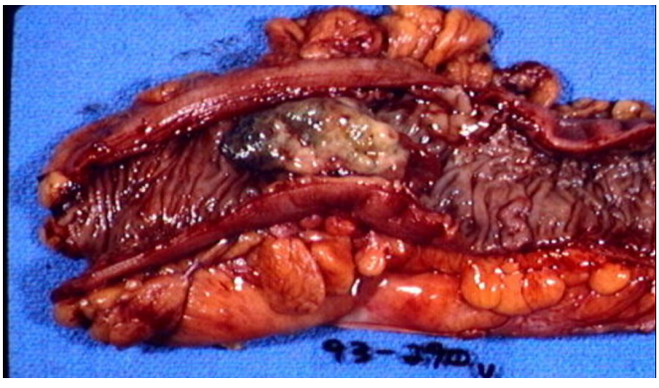


- This is an **adenocarcinoma** arising in a **villous adenoma.**
- The surface of the neoplasm is polypoid and reddish pink.
- Hemorrhage from the surface of the tumor creates a guaiac positive stool.
- This neoplasm was located in the **sigmoid colon**.



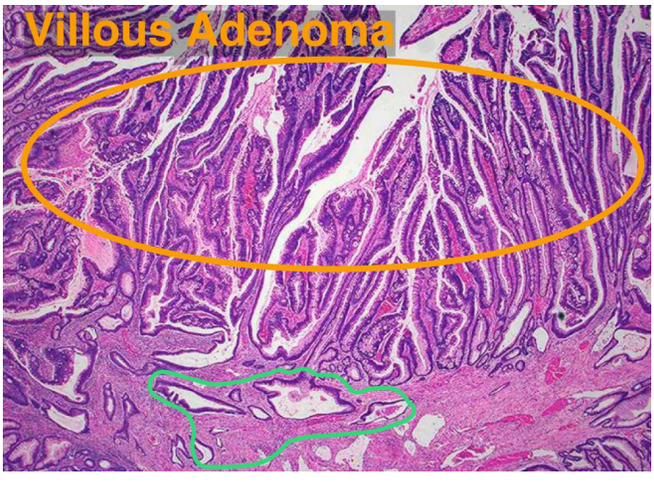
Organ: Colon
Dx: adenocarcinoma

- There is Raised mass with central ulceration
- It Usually arise on a top of a polyp (whether villous or tubular adenoma)



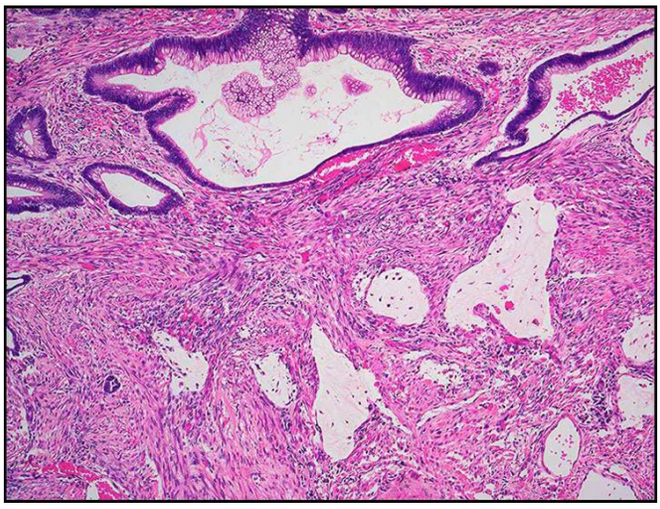
- Ulcerated adenocarcinoma with big ulceration
- Tumor involve the whole thickness of the bowel wall
- All infiltrated by the tumor.

Microscopic view:



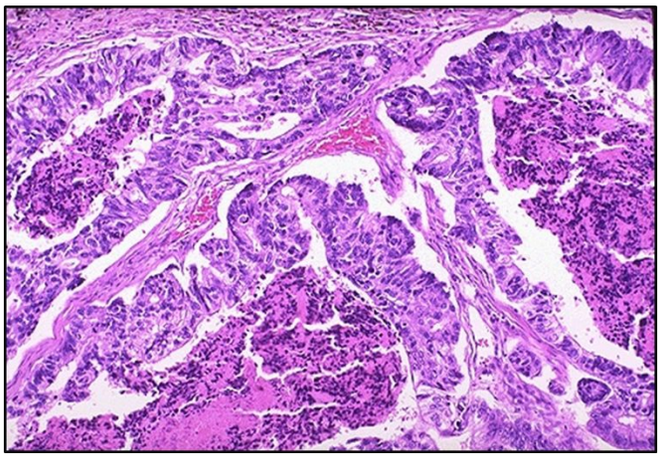
Tumour consists of crowded irregular malignant acini separated by thin fibrovascular stroma.

- Moderately differentiated adenocarcinoma arising on top of villous adenoma.
- Infiltrative dysplastic malignant gland.
- Villous adenoma base showing carcinoma.
- Invasive irregular gland going deep into the tissue

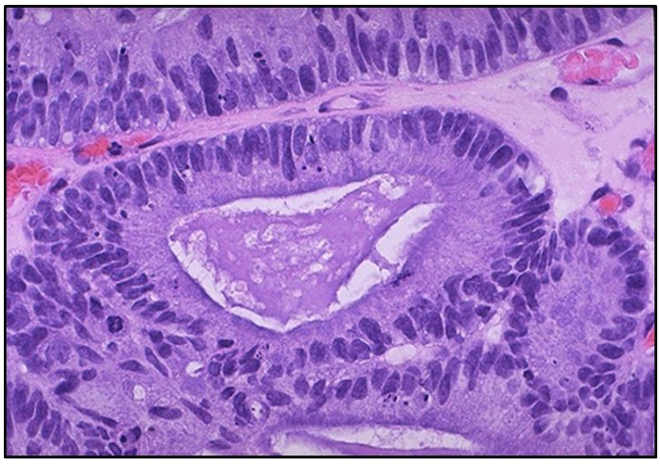


A tumour mass at one end, and a normal mucosa on the other side:

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

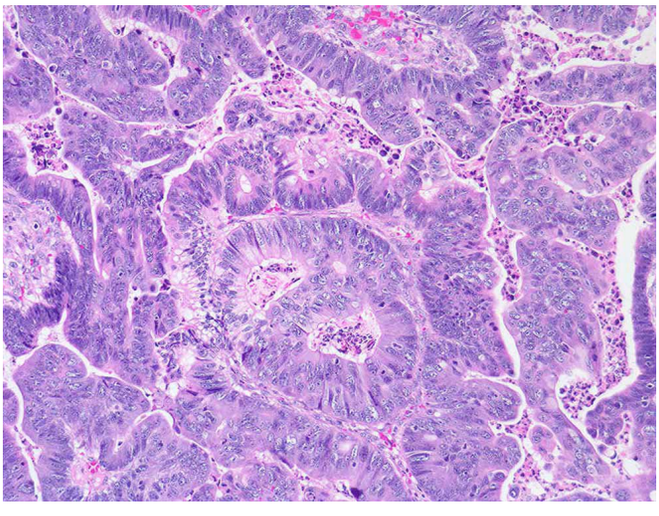


Here is an adenocarcinoma in which the glands are:
- Much larger
- Filled with necrotic debris.



At high magnification, the neoplastic glands of adenocarcinoma have crowded nuclei with:
- Hyperchromatism
- Pleomorphism.

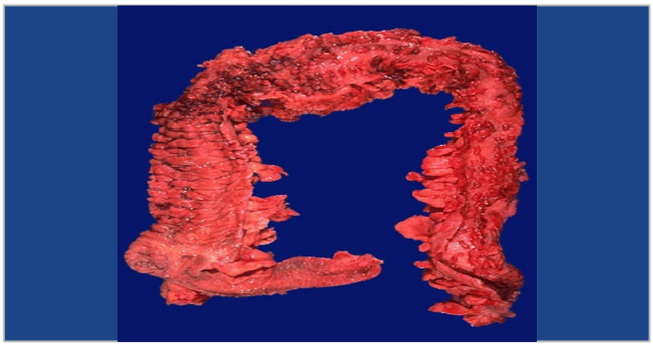
No normal goblet cells are seen



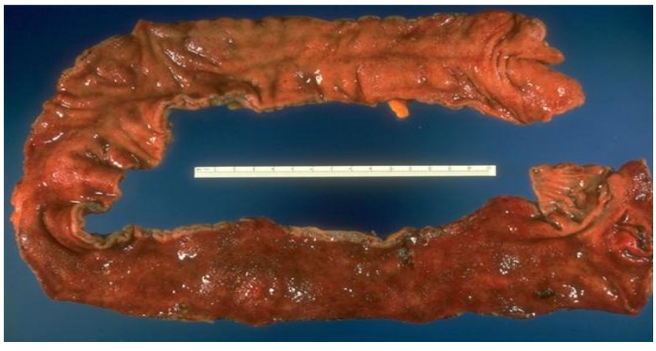
The Nuclei is hyperchromatic pleomorphic with increased C ratio , overcrowded and infiltrating lamina propria.

Case 18: Ulcerative colitis

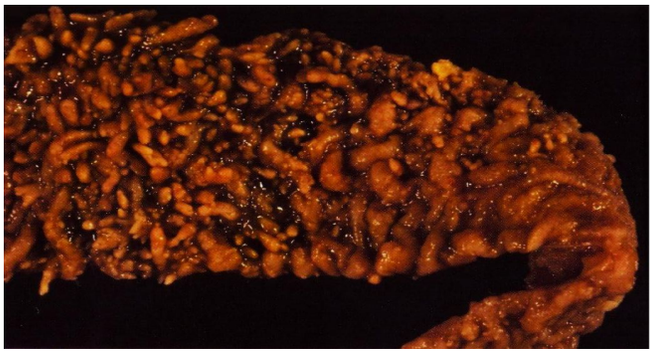
Gross:



- Intense inflammation begins at the sigmoid colon (Right) and extends upward and around to the ascending colon.
- Ileocecal valve with a portion of terminal ileum that is not involved (At the lower left).

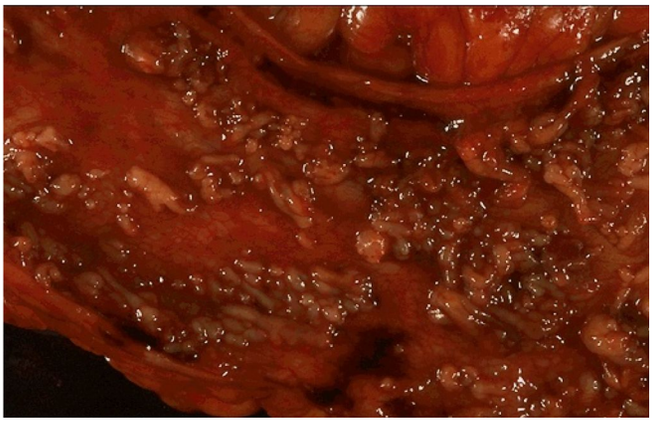


In this picture:
- The entire colon is abnormal,
- The usual transverse rugal folds have been almost completely effaced
- Diffuse continuous inflammation with no skip area in colon mucosa
- Redness and hyperemia in rectum and descending colon



It shows pseudo polyps formation with contenous ulceration

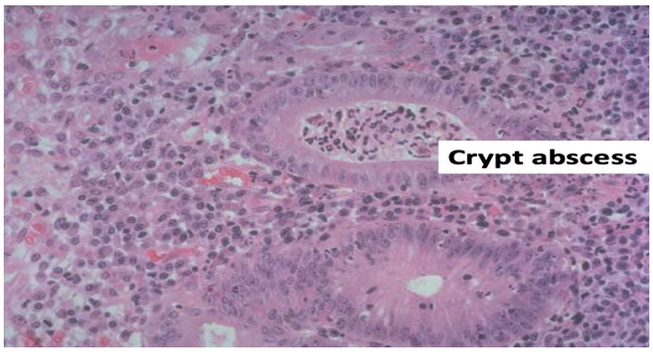
The Main complications are:
- Toxic mega colon
- Glandular dysplasia
- Adenocarcinoma



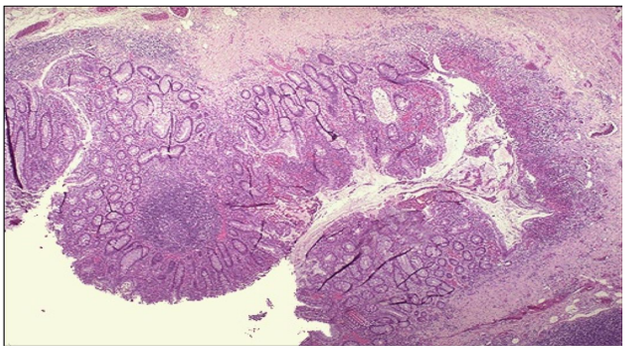
- Severe ulcerative colitis shows Pseudopolyps
- The remaining mucosa has been ulcerated away and is hyperemic

Extraintestinal manifestations include:
- Uveitis
- Migratory polyarthritis
- Sarco-ileitis ankylosing spondylitis
- Erythema nodosum
- Clubbing of the fingers
- Adenocarcinoma of the colon

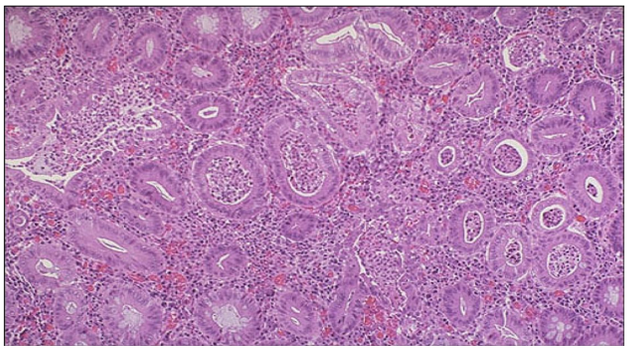
Microscopic view:



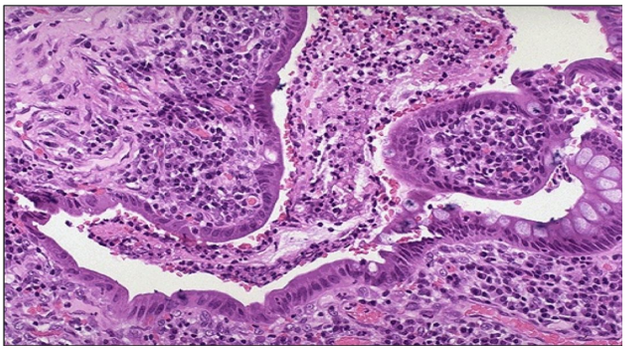
- Infiltration of neutrophils attacking the epithelium (active colitis)
- Accumulation of neutrophils in lumen known as **crypt abscess**



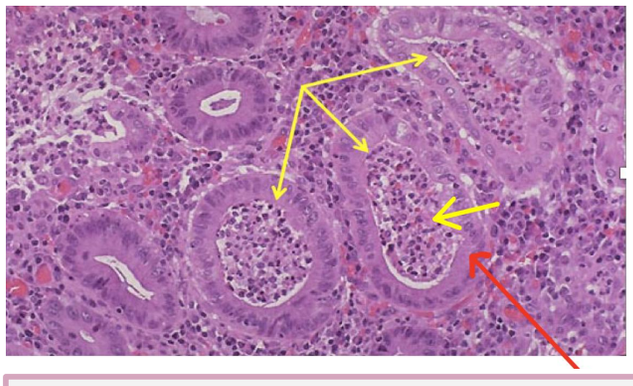
- The inflammation of ulcerative colitis is confined primarily to the mucosa
- The mucosa is eroded by an ulcer that undermines surrounding mucosa



The section shows:
- Crypt abscesses in the colonic mucosa of active ulcerative colitis (in which a neutrophilic exudate is found in glandular lumens)
- Intense inflammation in the submucosa
- The glands demonstrate loss of goblet cells and hyperchromatic nuclei with inflammatory atypia.



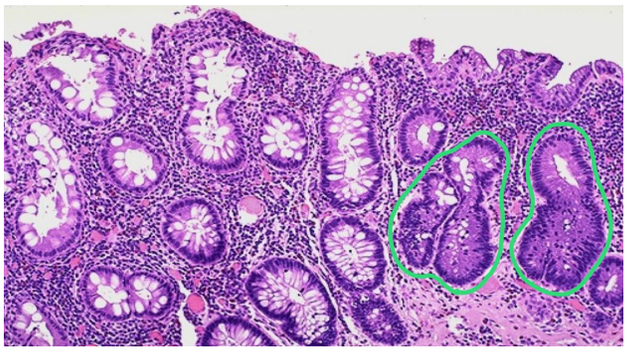
The section shows:
- Intense inflammation of mucosa
- loss of goblet cells in the colonic mucosa
- An exudate is present over the surface
- acute and chronic inflammatory cells are present



- Crypt abscesses are a histologic finding more typical with ulcerative colitis
- Unfortunately, not all cases of IBD can be classified completely in all patients
- Neutrophilic attack epithelium (active colitis)

Ulcerative colitis: Section of large bowel wall show a 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.



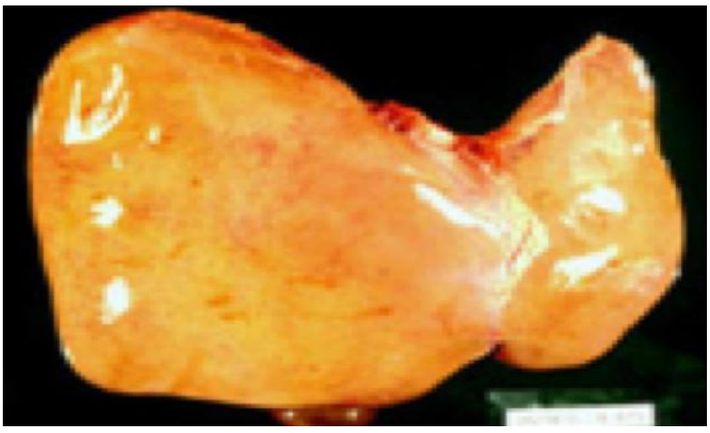
Over time, there is a risk for adenocarcinoma with ulcerative colitis.

- Normal glands are seen at the left
- Glands demonstrate dysplasia at the right
- The first indication that there is a move towards neoplasia

Only in female slides

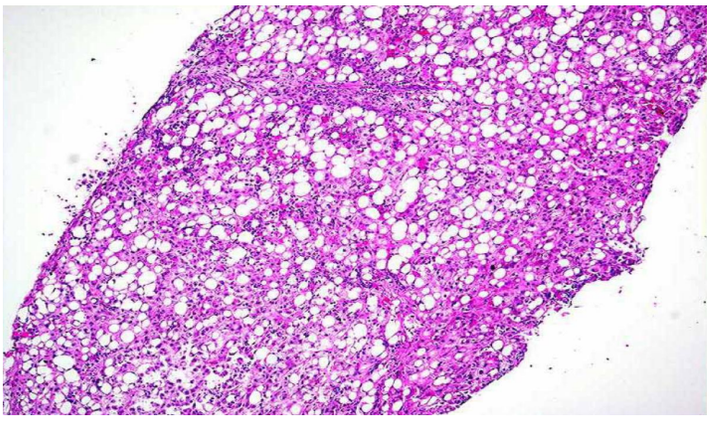
Case 19: Fatty liver

Gross:

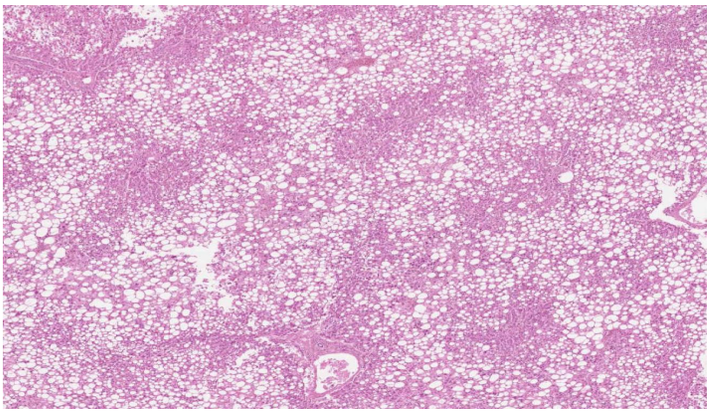


Yellow fatty liver

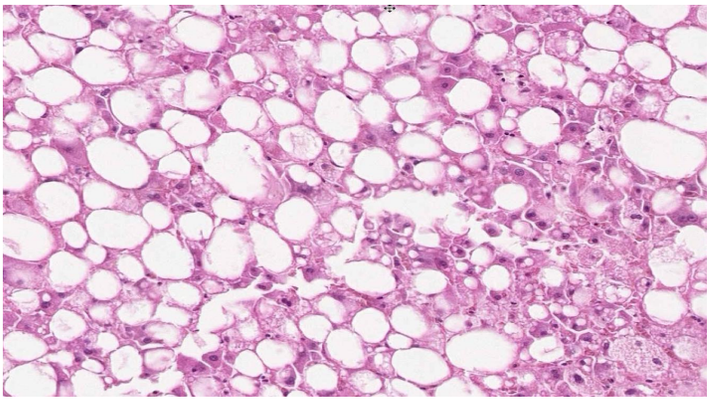
Microscopic view:



- Vacuoles in the Hepatocytes pushes the nucleus to the edge (steatosis)
- Inflammatory cells in between (non-alcoholic steatohepatitis)



- Big area of accumulation of the fat in liver (fat cyst)



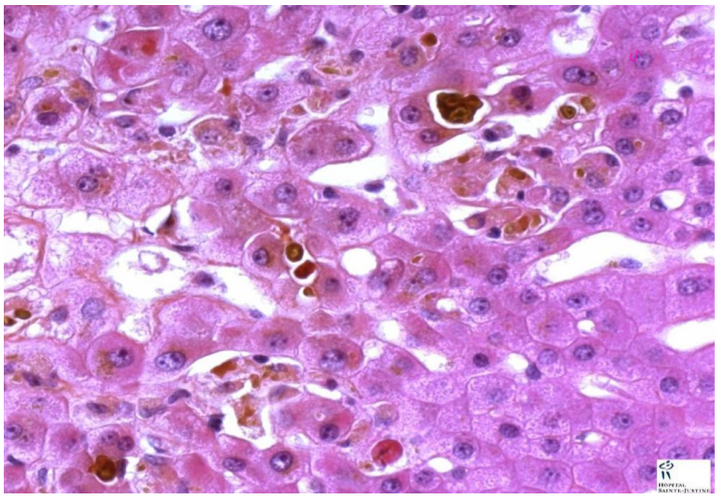
Section of the liver shows:
- Cystic spaces
- Fat rupture into the tissue
- Normal lobular architecture
- The liver cells are distended by clear vacuoles of dissolved fat with displacement of the nuclei to the periphery
- No inflammation and no fibrosis.

Only in female slides

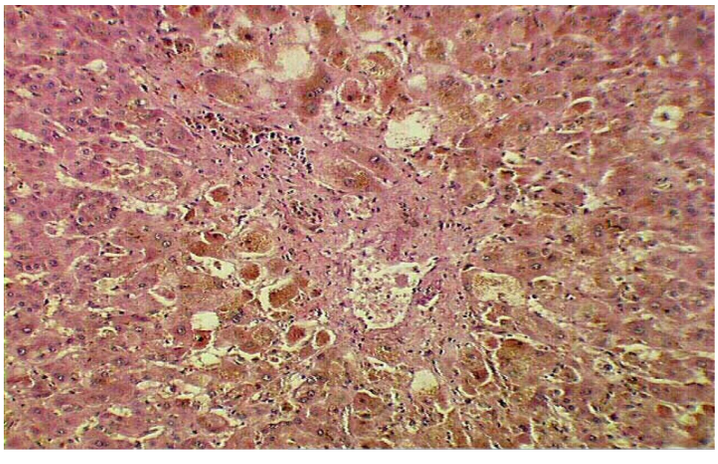
Case 20: Cholestasis

Could be mechanical or functional (obstructive and nonobstructive)
Changes in: Lobular parenchyma, Portal tracts
Bilirubin accumulation in liver lobule:
– starts in centrilobular zone
– pigment granules in parenchymal cells: (hepatocellular bilirubin stasis)
– inspissated bilirubin-stained bile plugs in dilated intercellular canaliculi: (canalicular bilirubin) stasis
Characteristic lab finding is elevated Alkaline phosphatase and GGT
Bile accumulation in the liver.

Microscopic view:



- Accumulation of bile in Hepatocytes
- Bile plugs (lakes)



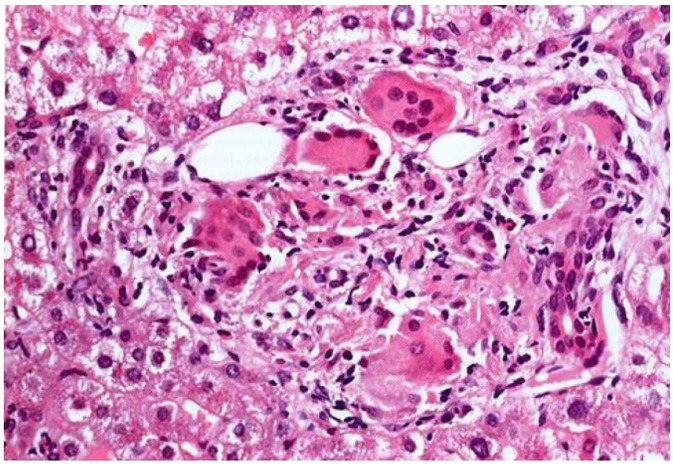
More amount of bile accumulated in hepatocyte

Only in female slides

Case 21: Drug toxicity

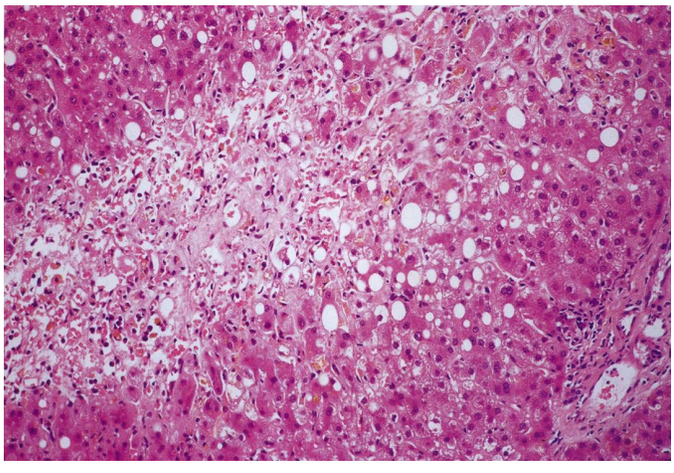
Liver injury due to medications or other toxic agents.
Can resemble any liver,process; clinical,correlation essential in diagnosis.
Histopathology: Changes that can be seen are:
• Cholestasis
• Steatosis
• Granulomas
• Hepatocellular Necrosis
• Predictable(intrinsic) or unpredictable(idiosyncratic)

Microscopic view:



A portal tract contains:
- Granulomas
- Multinucleated Giant cells

The patient became jaundiced after taking phenylbutazone
(Needle biopsy, H&E)



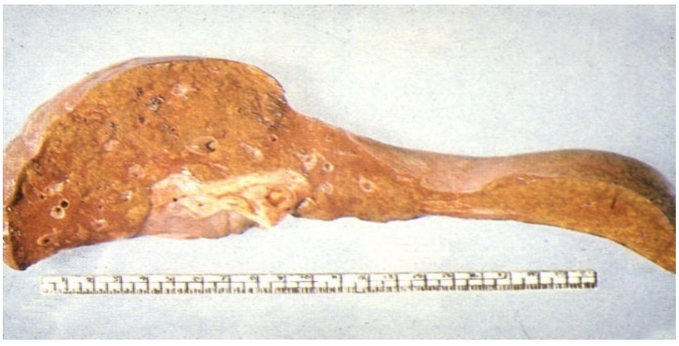
Drug-induced liver injury hepatitis type,in this acute hepatitis attributed to indometacin:
- Necrosis in acinar zone 3
- Steatosis
- Very mild portal inflammation (below right)

(Needle biopsy, H&E)

Only in female slides

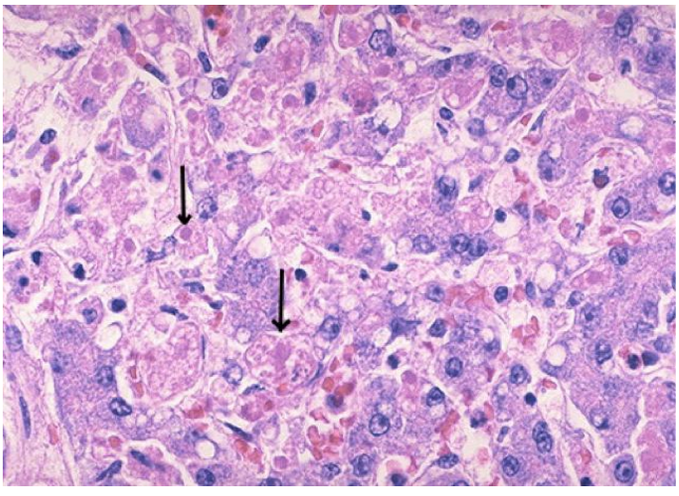
Case 22: Acute Viral Hepatitis

Gross:



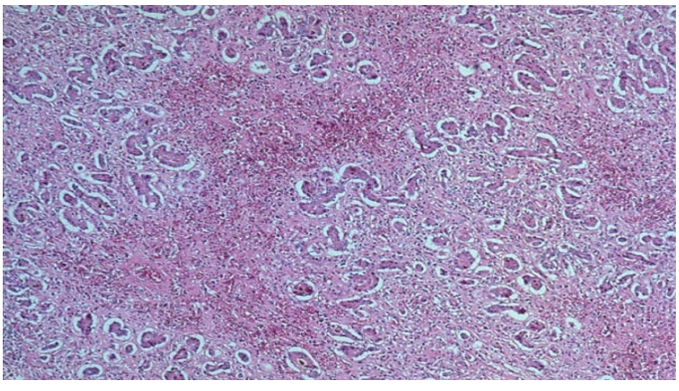
Fulminant Hepatitis shows:
- Necrosis of Hepatocytes
- Collapsed and thin area in the liver

Microscopic view:

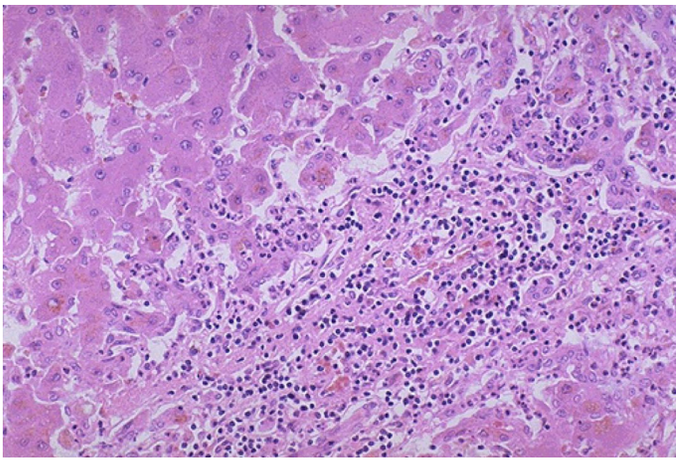


- Lymphocytic infiltration
- Necrosis of Hepatocytes

When we have neutrophilic infiltration? In Alcoholic Hepatitis



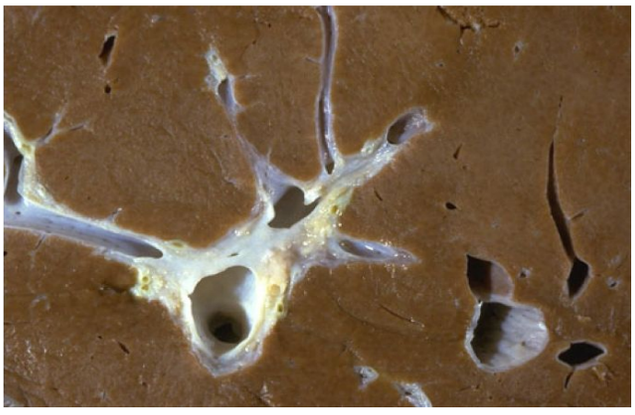
This section shows:
- Extensive necrosis
- lymphocytes infiltration
- Remaining bile ducts



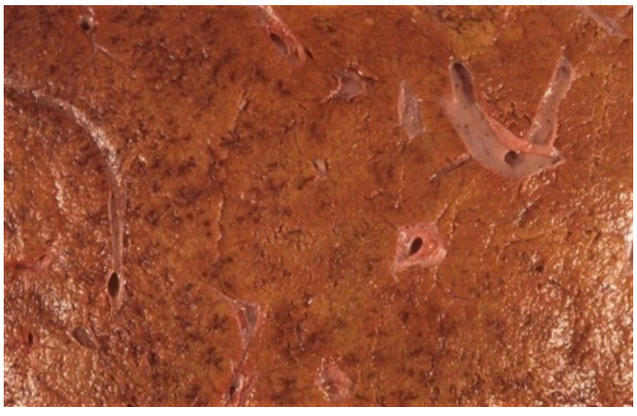
- Lymphocytic infiltration
- Necrosis of Hepatocytes

Case 23: Chronic viral hepatitis.

Gross:

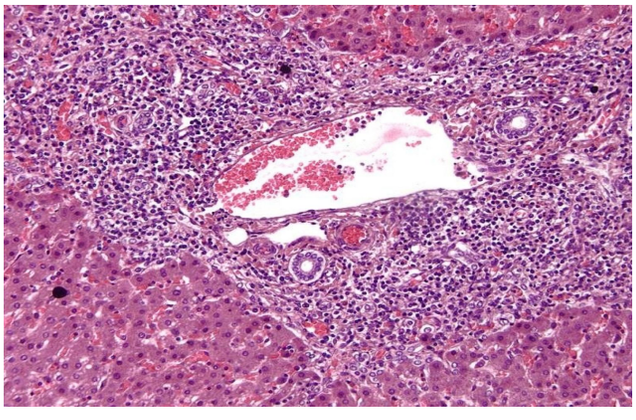


Normal Liver
- Brown color
- Near the hilum we found portal area (portal vein, hepatic artery, bile ducts)
- Portal vein carrying blood to the liver, which branches at center left, and draining to inferior vena cava from the lower right branch of hepatic vein.

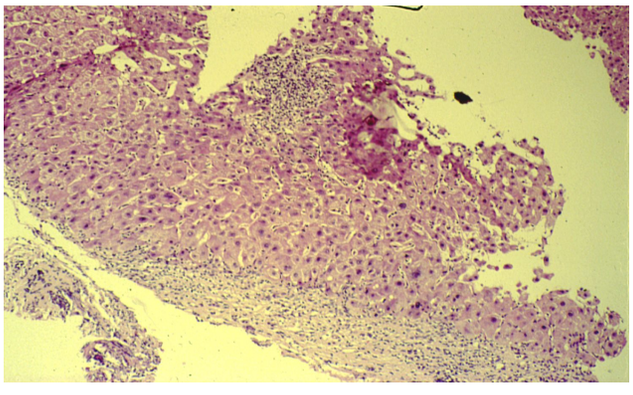


Chronic Hepatitis shows:
- Necrosis
- Lobular collapse
- Hemorrhage
- Irregular furrows
- Granularity

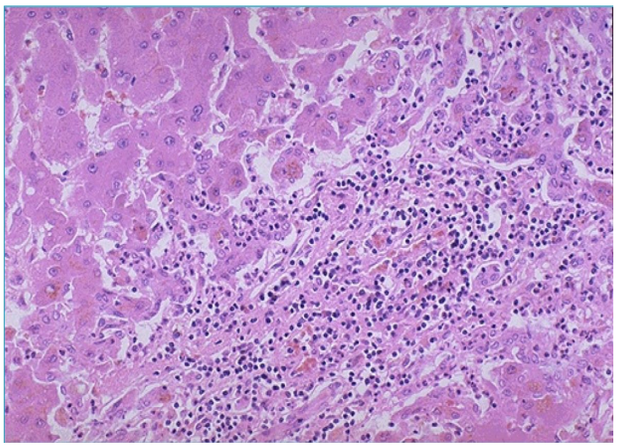
Microscopic view:



Chronic Viral Hepatitis B
- Portal infiltration by lymphocyte (portal inflammation)
- Hepatocyte necrosis (piecemeal necrosis)
- Ground glass appearance

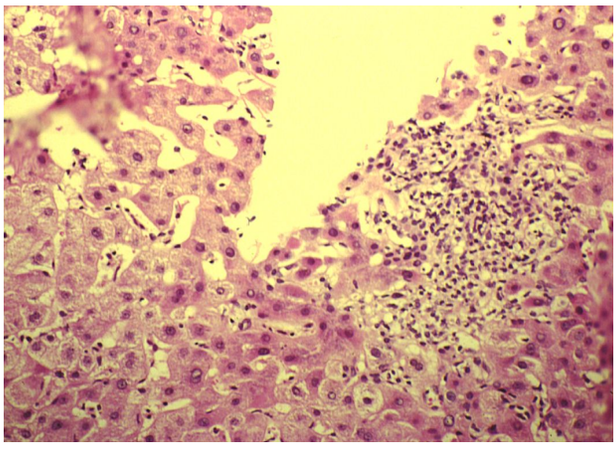


- Aggregation of lymphocyte
- Piecemeal necrosis
- Septic formation with inflammation

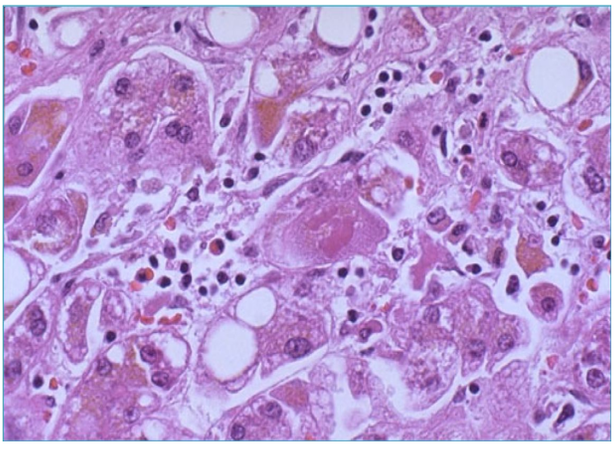


Chronic Viral Hepatitis B:
- Liver cell destruction
- A mononuclear inflammatory cell infiltrate extends from portal areas and disrupts the limiting plate of hepatocytes
- Hepatocytes necrosis (piecemeal necrosis)

In this case, hepatitis B surface antigen (HBsAg) and hepatitis B core antibody (HBcAb) were positive.

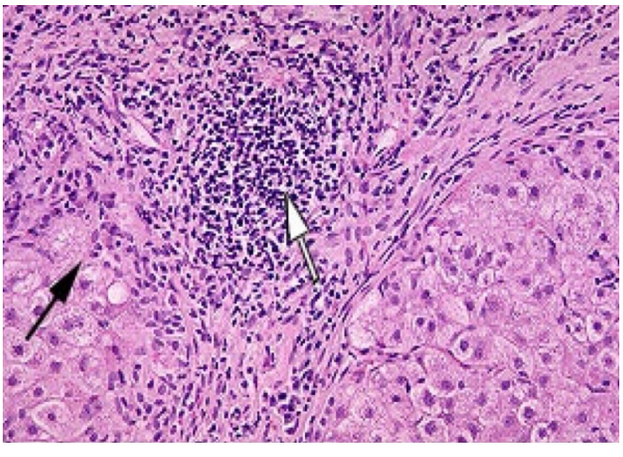


Chronic Viral Hepatitis B shows:
- Moderate chronic inflammatory cells infiltration (lymphocytes and histiocytes) in both portal tracts and liver parenchyma
- Piecemeal necrosis
- Hepatocytes swelling and “spotty” hepatocytes necrosis
- No evidence of cirrhosis or malignancy



Chronic Viral Hepatitis C Leads to chronic liver disease in 50% of the cases, here it shows:
- Necrosis and inflammation are prominent
- Steatosis

Chronic hepatitis:
- Graded by the degree of activity (necrosis and inflammation)
- Staged by the degree of fibrosis (portal fibrosis, septal fibrosis and portal to central fibrosis and finally cirrhosis)



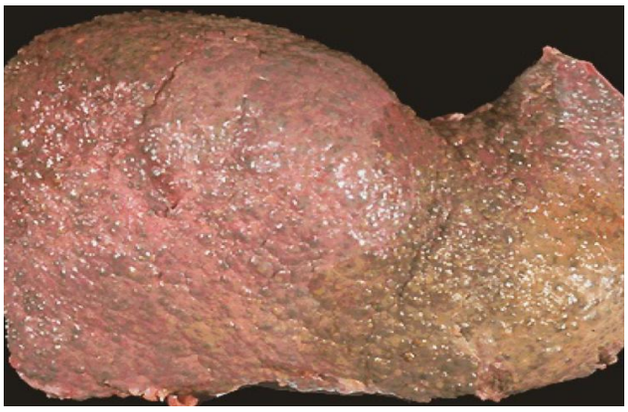
Portal Inflammation in Chronic Hepatitis

- Severe portal infiltrates by lymphocyte (white arrow)

- Sinusoidal infiltrates by lymphocyte (black arrow)

Case 24: hepatic cirrhosis

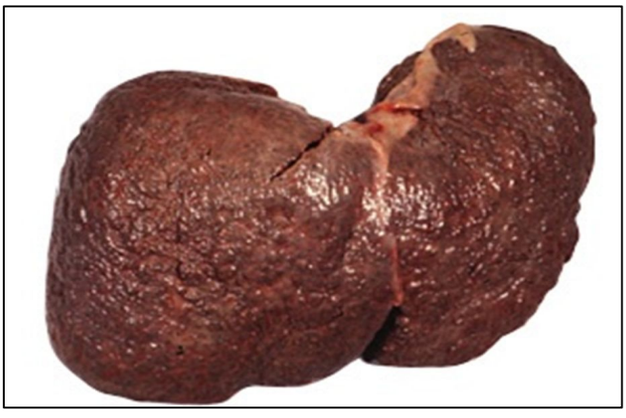
MRI:



This is an example of a micronodular cirrhosis
it shows:
- Regenerative nodules are quite small (averaging less than 3 mm in size).

The **most common cause** for this is**: chronic alcoholism.**
The process of cirrhosis develops over many years**.**

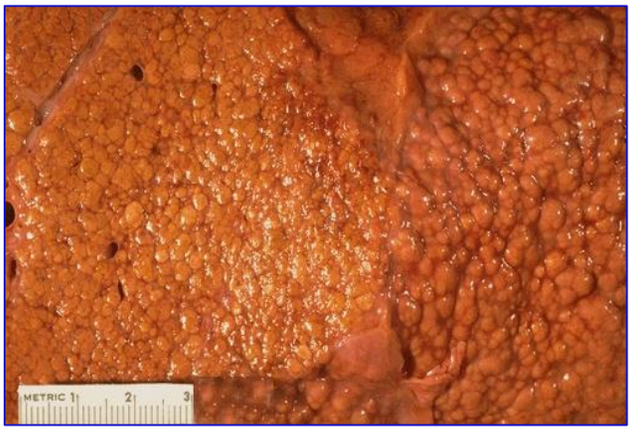
Gross:



This liver shows:
- Nodular surface due to cirrhosis

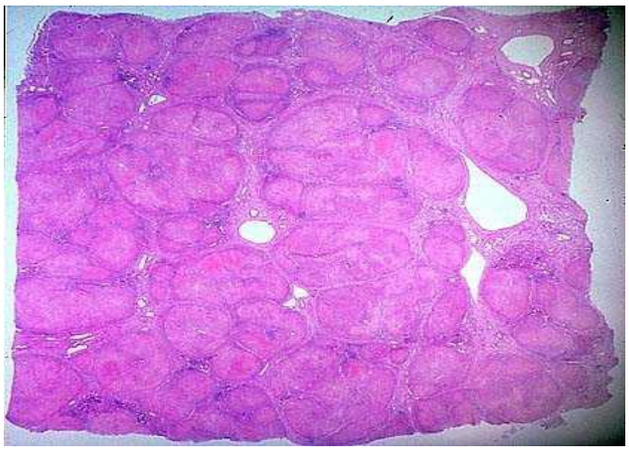
macronudular cirrhosis (HBV)
it shows:
- Multiple nodules of variable sizes
- Surrounded by Fibrosis

The major complications are:
- Portal hypertension with esophageal varices
- Hepatic failure (including hepatic encephalopathy and hepatic renal syndrome)
- Hepatocellular carcinoma

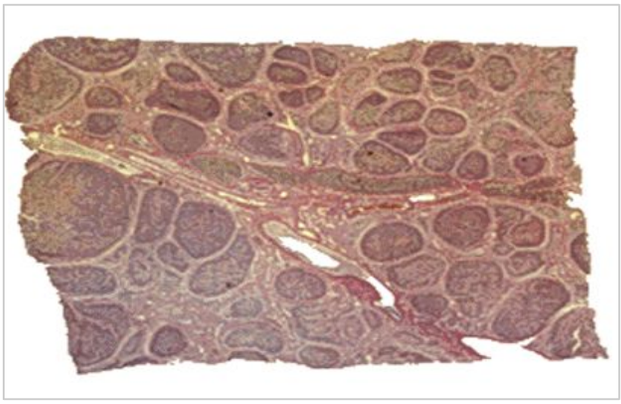


- A close-up view of a **micronodular cirrhosis** in a liver with fatty change demonstrates the small, yellow nodules.
- Micronodular cirrhosis may also be seen with **Wilson's disease**, **primary biliary cirrhosis**, and **hemochromatosis**.

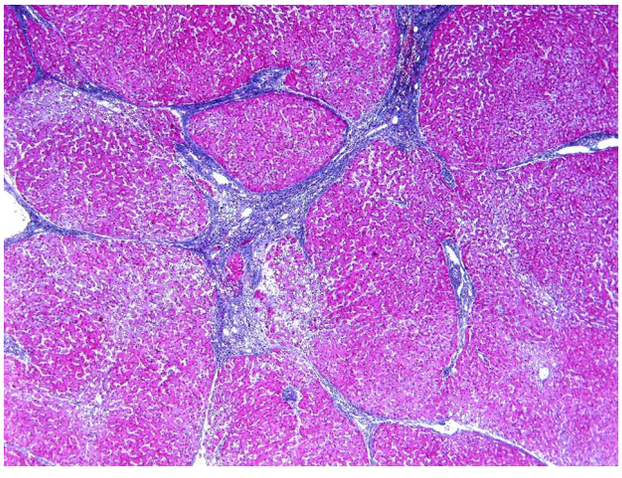
Microscopic view:



Irregular nodules separated by Portal to Portal fibrous bands

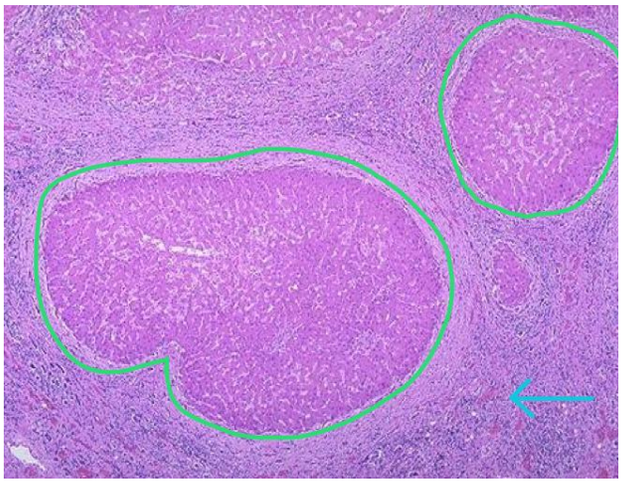


Hepatic Cirrhosis - LPF:
- The parenchyma shows darker tan nodules of varying sizes.
- These nodules are composed of hepatocytes.
- The paler areas in between are collagen.

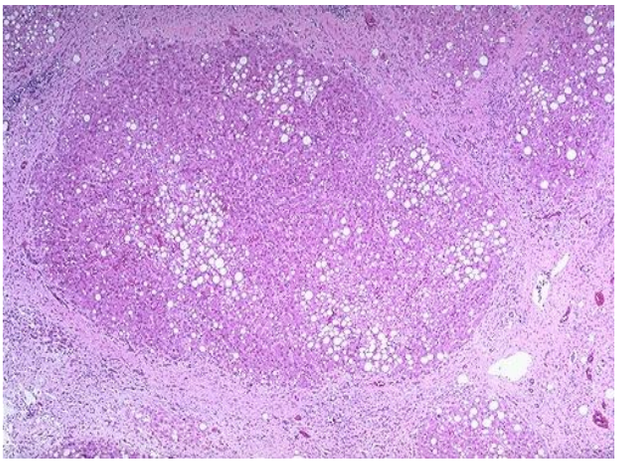


It shows:
- Loss of lobular architecture
- Formation of regenerative nodules of variable size and shape.
- Surrounded by fibrous tissue.
- Each nodules consists of liver cells without any arrangement and with no central vein.
- Large number of proliferated bile ducts and chronic inflammatory cells are present in fibrous tissue.

(Mason Trichome stain)

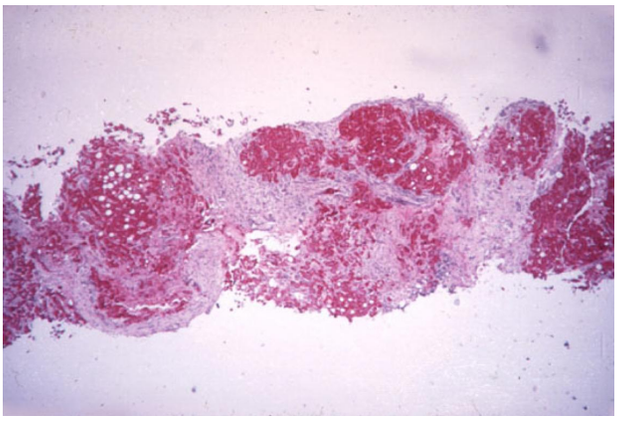


Microscopically with cirrhosis shows:
- Regenerative nodules of hepatocytes which surrounded by fibrous connective tissue that bridges between portal tracts and between portal tract and central vein
- Within this collagenous tissue are scattered lymphocytes and Proliferation of bile ducts

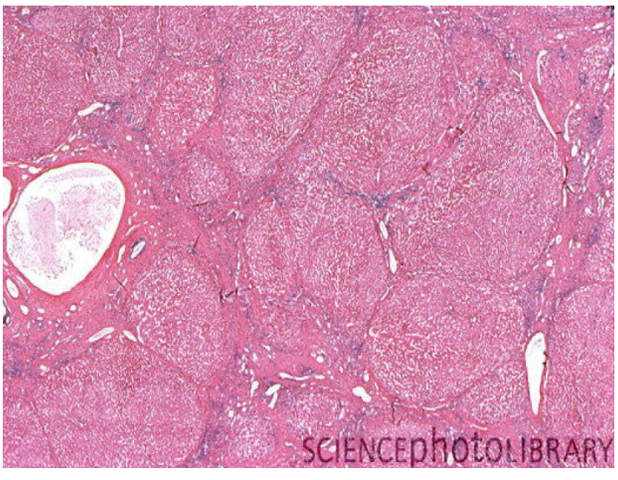


Micronodular cirrhosis with fatty liver- LPF

- Micronodular cirrhosis is seen along with moderate fatty change. (Steatosis)
- Note the regenerative nodule surrounded by fibrous connective tissue extending between portal regions.



Core Biopsy of the liver shows:
- Variably size nodules
- Separated by fibrous tissue

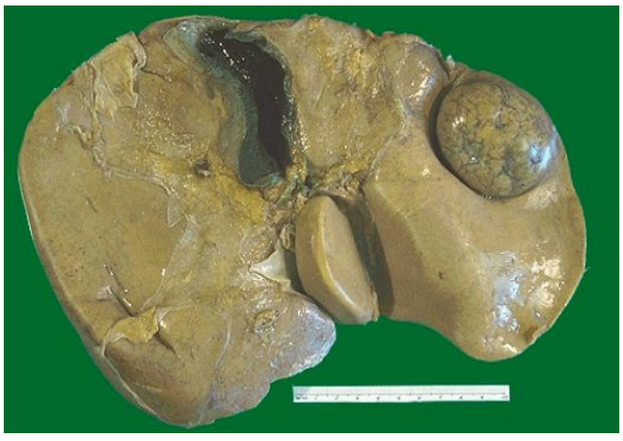


Hepatic Cirrhosis - LPF:
- The parenchyma shows nodules of varying sizes.
- These nodules are composed of Hepatocytes.
- The areas in between are collagen with mononuclear cell infiltration.

Only in male slides

Case 25: hepatic adenoma

Gross:



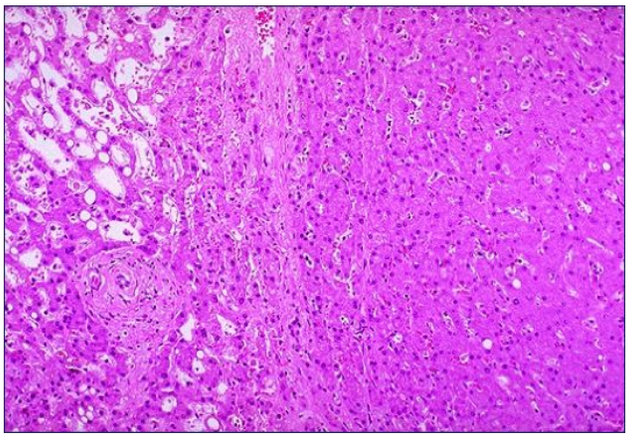
At the upper right is a well-circumscribed neoplasm that is arising in liver.
This is an hepatic adenoma.

Cut Section Gross:

- The cut surface of the liver reveals the hepatic adenoma. Note how well circumscribed it is.

- The remaining liver is a pale yellow brown because of fatty change from chronic alcoholism.

Microscopic view:

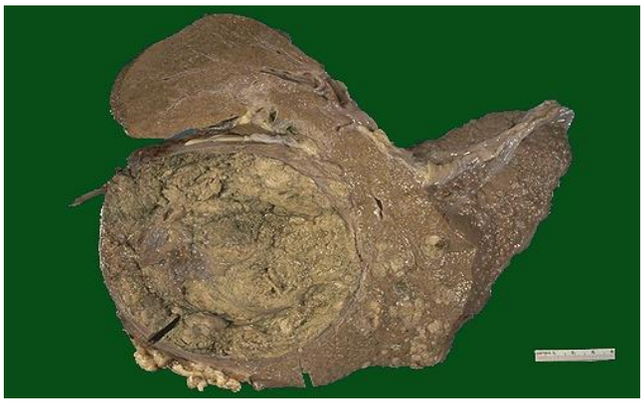


Left: Normal liver tissue with a portal tract
Right: Hepatic adenoma and it is composed of cells that closely resemble normal hepatocyte

but the neoplastic liver tissue is :
- Disorganized hepatocyte cords
- Does not contain a normal lobular architecture and no portal tract.

Case 26: Hepatocellular Carcinoma

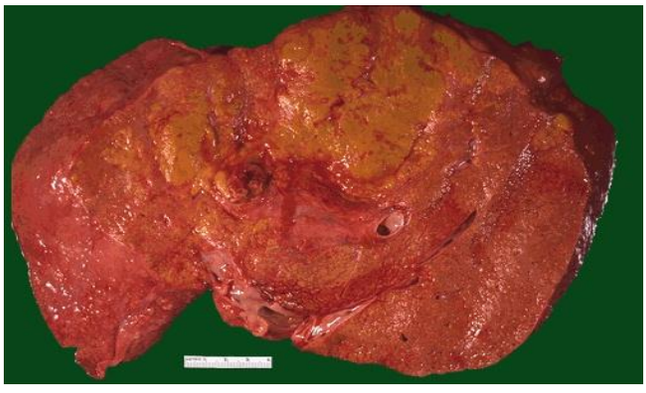
Gross:



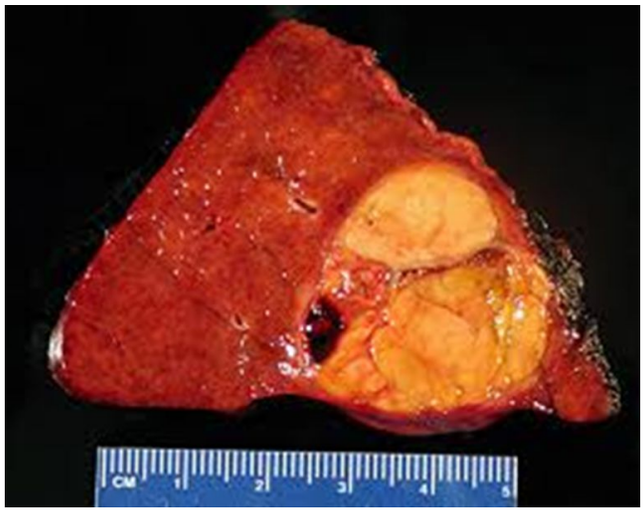
Such liver cancers arise in the setting of cirrhosis. Worldwide, viral hepatitis is the most common cause, but in the U.S., chronic alcoholism is the most common cause.

The neoplasm is:

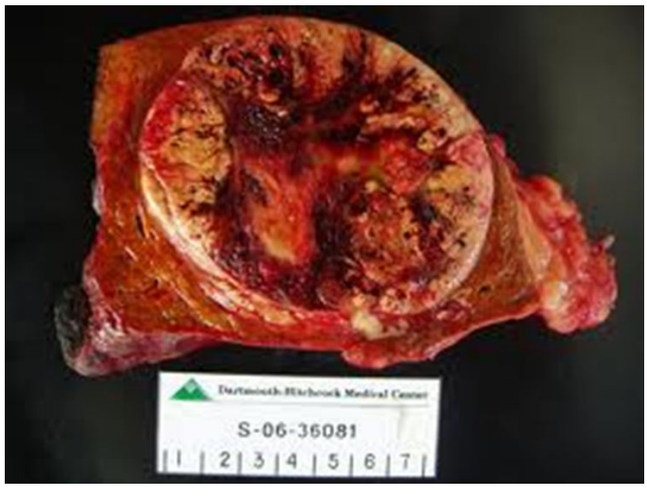
- Large and bulky
- Has a greenish cast because it contains bile
- To the right of the main mass there are smaller satellite nodules.



- HCC with a greenish yellow hue.
- Clue to the presence of such a neoplasm is an elevated serum alpha-fetoprotein.
- Such masses may also focally obstruct the biliary tract and lead to an elevated alkaline phosphatase

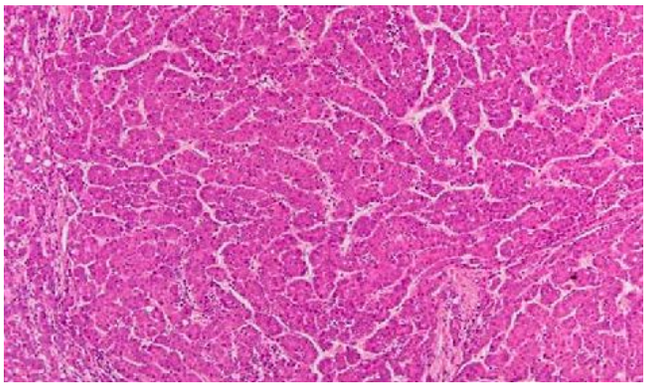


- Big nodule
- Area of Necrosis
- Area of Hemorrhage

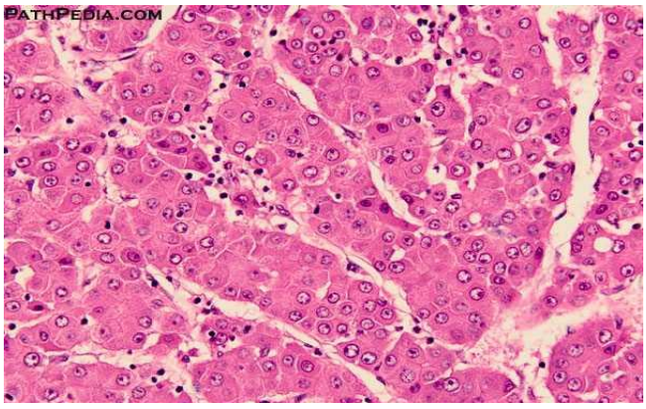


- Large nodule
- Could be solitary or multiple
- The Surrounding liver could have cirrhosis or not according to the cause

Microscopic view:

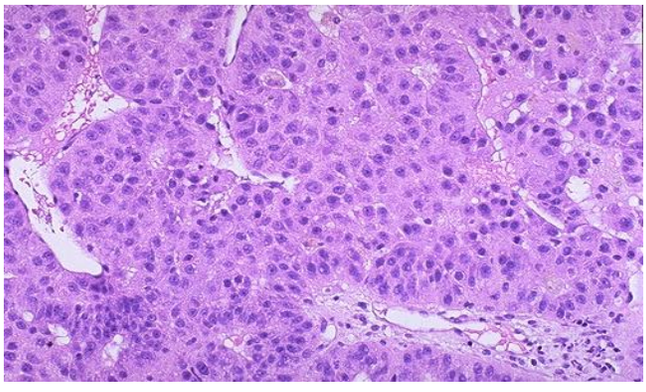


This example of well-differentiated HCC shows:
- Trabecular pattern
- Intervening sinusoids.
- Thick cords of Hepatocytes
- Disturbance of the normal sinusoidal architecture

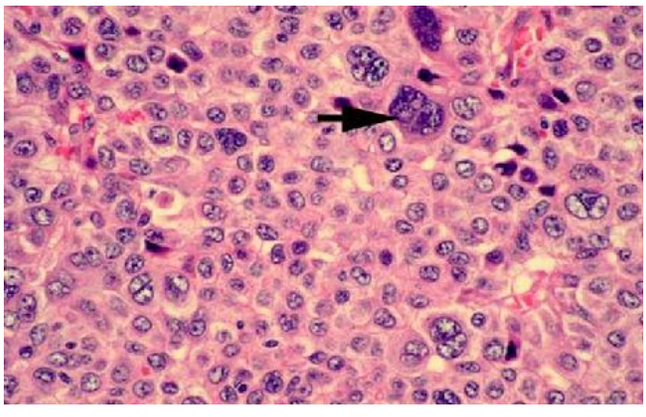


The key to the identification of HCC is:
- Its resemblance to hepatocytes
- Presence of more than 2-3 cell-thick hepatocellular plates/cords
- Nuclear atypia
- Absence of portal tracts

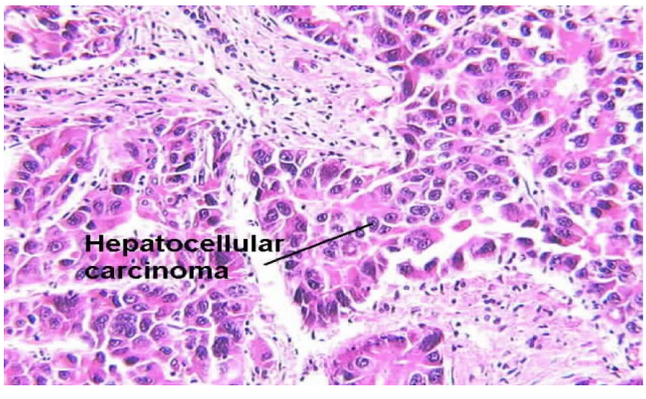
Note the hepatic plates are separated from each other by sinusoids.
(It's well differentiated)



Note that this hepatocellular carcinoma is composed of:
- Liver cords that are much wider than the normal liver plate that is two cells thick.
- There is no discernable normal lobular architecture.
- Though vascular structures are present.



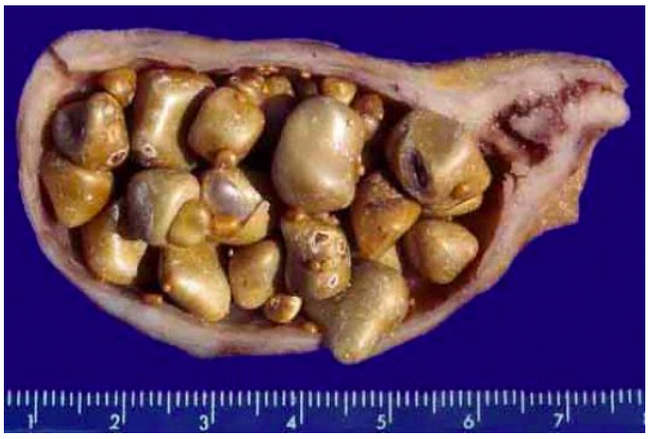
Anaplastic tumor giant cells can be seen in poorly differentiated HCC (arrow), it shows:
- Numerous mitoses.
- Malignant liver cells are pleomorphic, binucleated or forming giant cells with hyperchromatic nuclei.
- Areas of haemorrhage and necrosis are present
- Thick cords, trabeculate and nests of malignant liver cells separated by sinusoidal spaces.



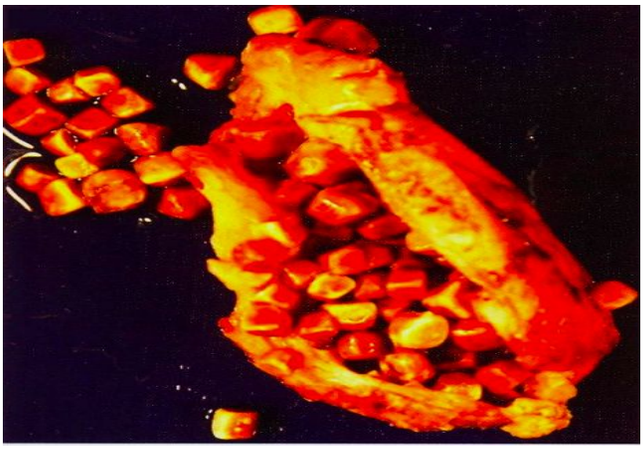
- Alveolar structure more like acini
- Dysplasia
- Moderately differentiated

Case 27: chronic cholecystitis with stones

Gross:

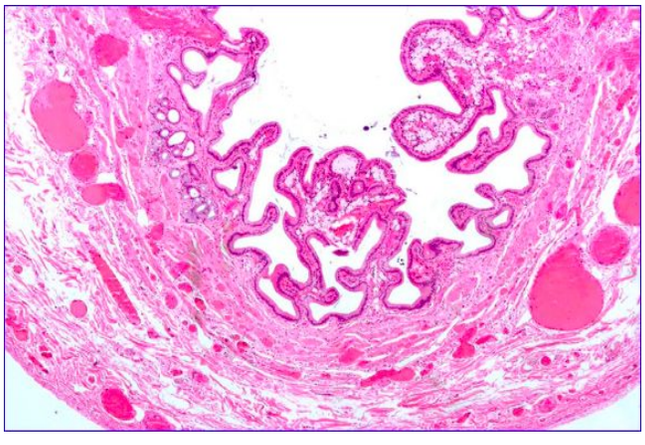


Gross appearance of gallbladder after sectioning longitudinally shows:
- Thickness of gallbladder wall
- Abundant polyhedric stones and small papillary tumor in the cystic duct.



- Multi-phase yellow Cholesterol stones (yellow).
- The wall is thickening and fibrosis.

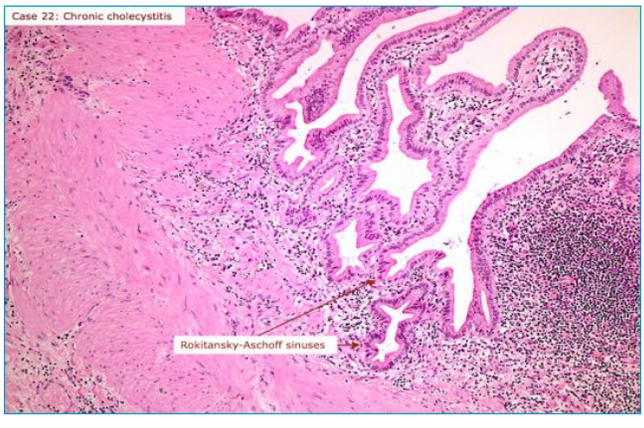
Microscopic view:



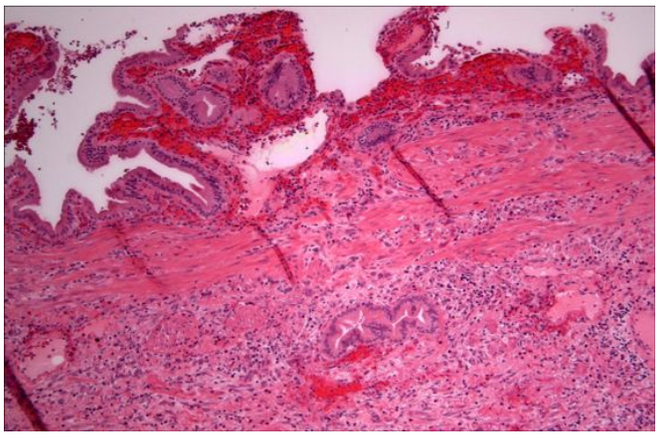
- Dead lipid laden macrophages (foam cells) are seen in the finger-like projections into the gallbladder lumen.

- It should be apparent that this is gallbladder, as no muscularis mucosae is present (as elsewhere in the gastrointestinal tract).

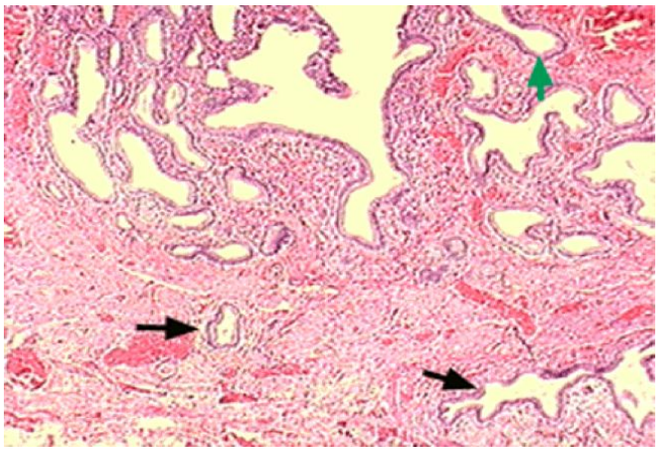
- The blood vessels are congested and the subserosa edematous.



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



- 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.



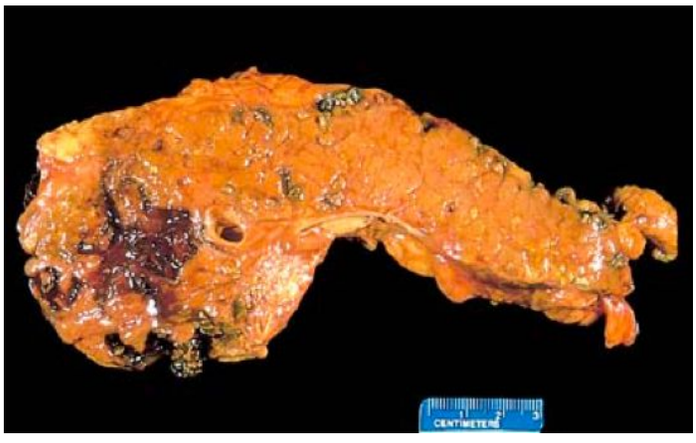
The surface epithelium has lost its normal delicate-papillary appearance **(green arrow)** with an increase in fibrous tissue and mild chronic inflammation in the lamina propria
Rokitansky-Aschoff sinuses are seen in the muscularis **(black arrow)**The degree of chronic inflammation is quite variable and as in this case surprisingly mild

Only in male slides

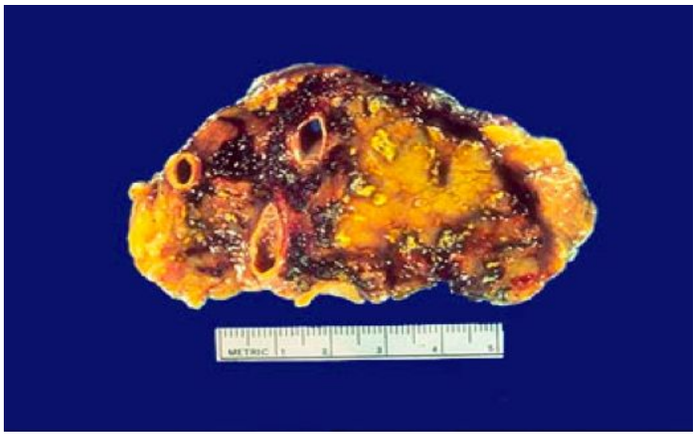
Case 28: Acute pancreatitis

Causes: Alcoholism, Bile reflux(gallstones),Medications (thiazides),Hypertriglyceridemia, hypercalcemia,Acute ischemia,Trauma, blunt, 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 estimation.
Consequences: edema,fat necrosis,acute inflammatory infiltrate ,pancreas autodigestion, blood vessel destruction ,“saponification”

Gross:

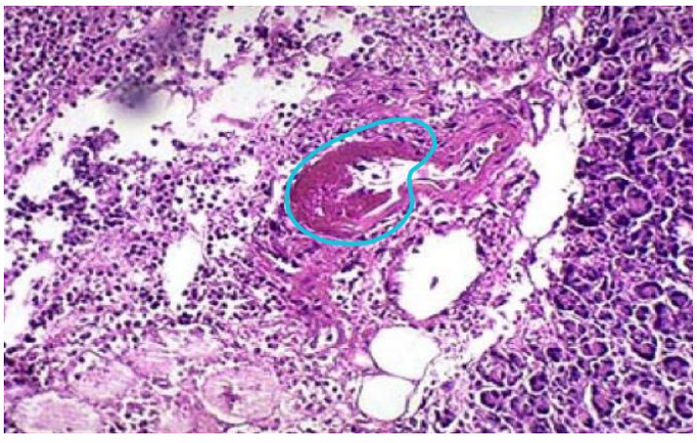


Fat necrosis appears as chalky white calcium soaps.



Severe acute pancreatitis characterized by:
- Black areas of hemorrhage
- chalky, yellow-white areas of fat necrosis.
- Pancreatic parenchyma is soft and gray-white due to necrosis

Microscopic view:

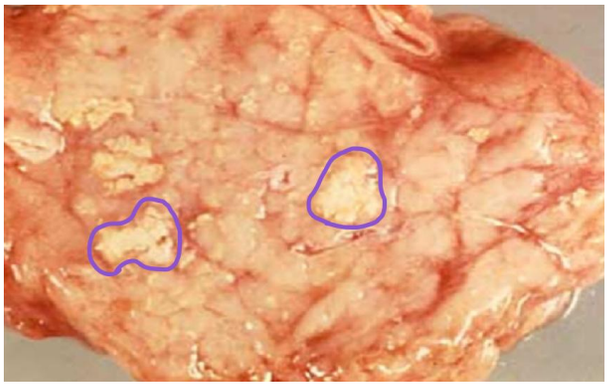


- Severe acute pancreatitis shows area of acute inflammation with necrosis.
- Within the necrotic area is a blood vessel showing **fibrinoid necrosis** of the vessel wall leads to severe, **hemorrhagic**, **acute pancreatitis**.

Common causes of acute pancreatitis are:
- Alcoholism, Gall stones impaction, Traumatic, Hereditary and Idiopathic .

Case 29: Chronic pancreatitis

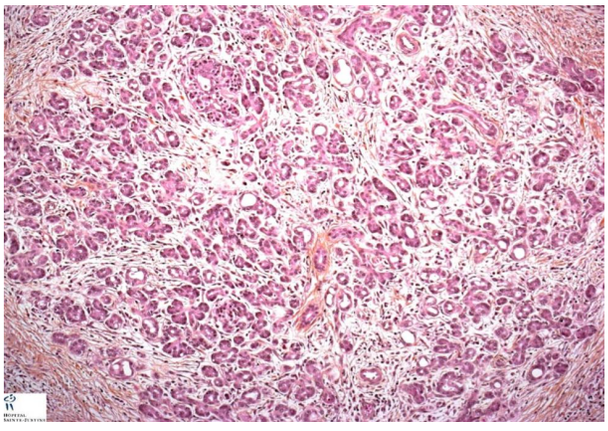
Gross:



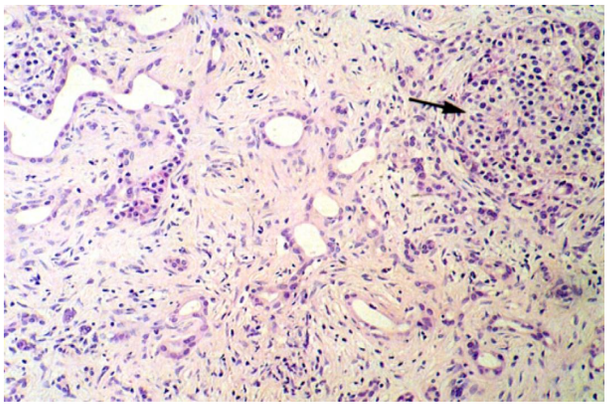
There is:
- Soap like calcium deposits
- Calcium deposition is secondary to fat necrosis and dystrophic calcification

Possible causes of chronic pancreatitis are:
- Gallstones, Alcoholism, Tropical , Hereditary and Idiopathic .

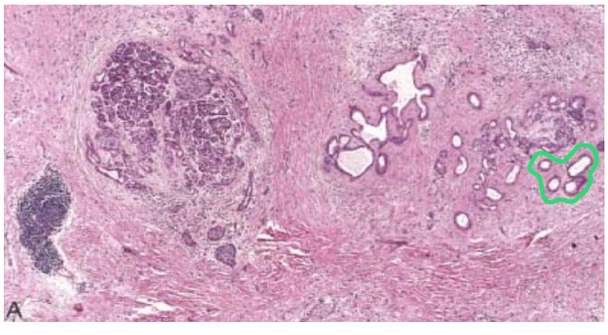
Histology:

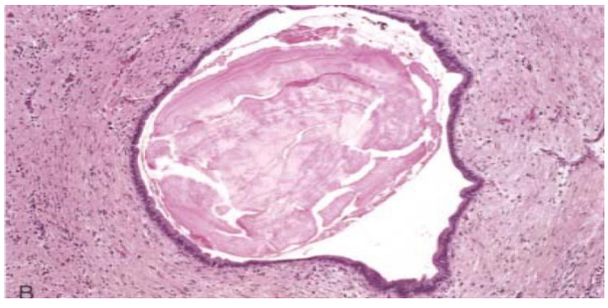


Unfortunately dense fibrosis is a feature BOTH of chronic pancreatitis as well as adenocarcinoma.



Chronic Pancreatitis shows:
- Parenchymal fibrosis
- Chronic inflammatory infiltrate
- Reduced number and size of acini
- Variable dilatation of pancreatic ducts
- Relative sparing of islets of langerhans (arrow)
- Lining epithelium is not dysplastic (no nuclear pleomorphism or hyperchromatism)





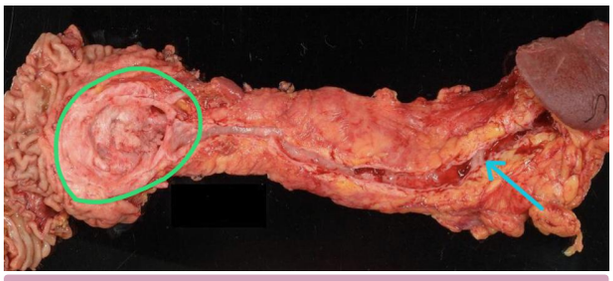
A/ Residual islets of langerhans

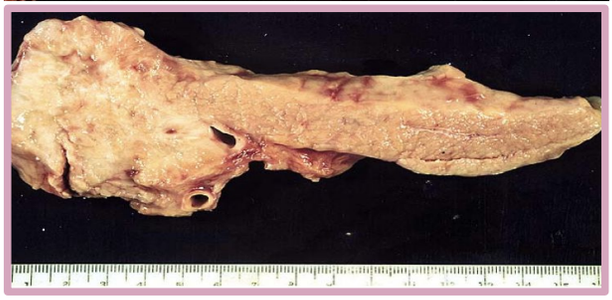
B/ Dilated duct

Fibrosis and inflammation in the background

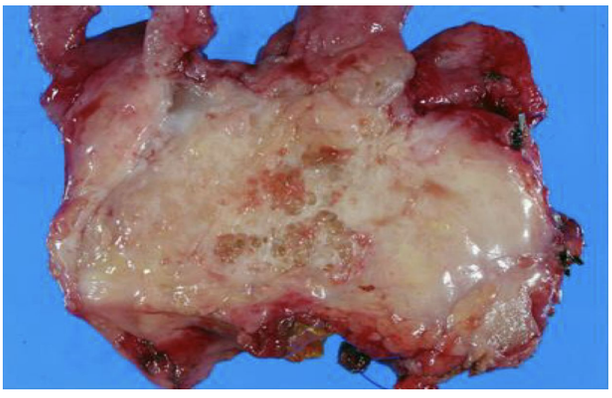
Case 30 : Pancreatic adenocarcinoma

Gross:



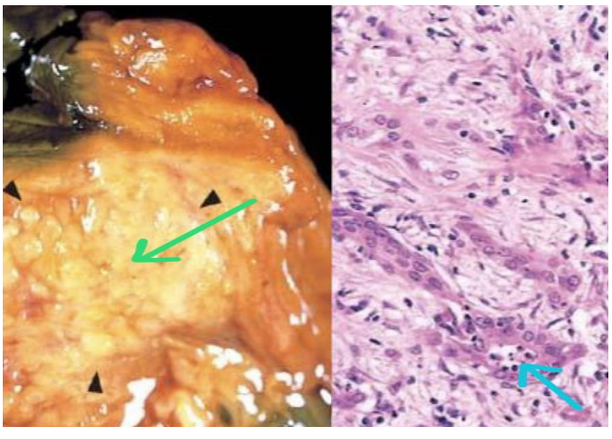


Horizontal section of pancreas , It shows:
- Well circumscribed tumor nodule at the head
- Dilated main pancreatic duct
- Part of the duodenum on the left
- Spleen on the right side.



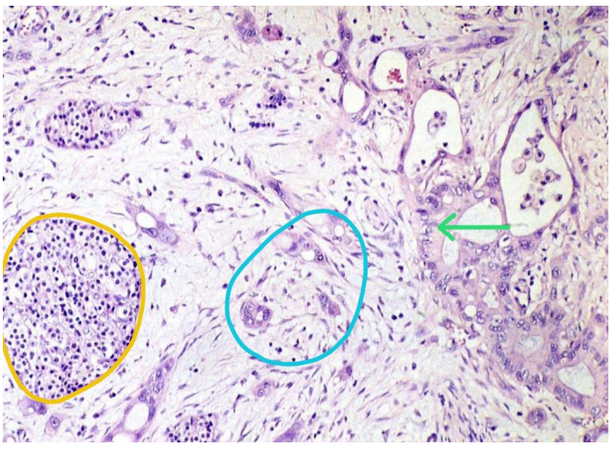
Cut surface shows:
- Large duct type ductal adenocarcinoma

A microcystic pattern with cysts measuring from millimeters up to 1 cm.



Gross (left) picture shows:
- ill defined pale and firm pancreatic mass
- Irregular edges
- Fibrosis

Microscopic (right) picture shows:
- Acini surrounded by desmoplastic fibrous stroma
- Malignant glands
- Glands lined by hyperchromatic nuclei
- Fibrosis (desmoplastic reaction)



There is :
- Deeply infiltrative growth pattern of glands with Irregular shape and distribution
- Desmoplasia
- Nuclear pleomorphism with nucleoli
- Loss of polarity and Mitotic figures
- Fibrosis with infiltrating malignant glands showing nuclear atypia (Hyperchromatic and Increase Mitosis)
- Islet of langerhans are preserved

MED437
KING SAUD UNIVERSITY

Team 437
Pathology Practical

Team Leaders:

Dimah Al Araifi - Fayez Aldarsouni

Team Members:

Laila Alsabbagh
Marwah Alkhalil
Ghada E.Almuhanna
Lujain Alzaid
Fatimah albassam
Ahad Algrain
Shahad Alzahrani