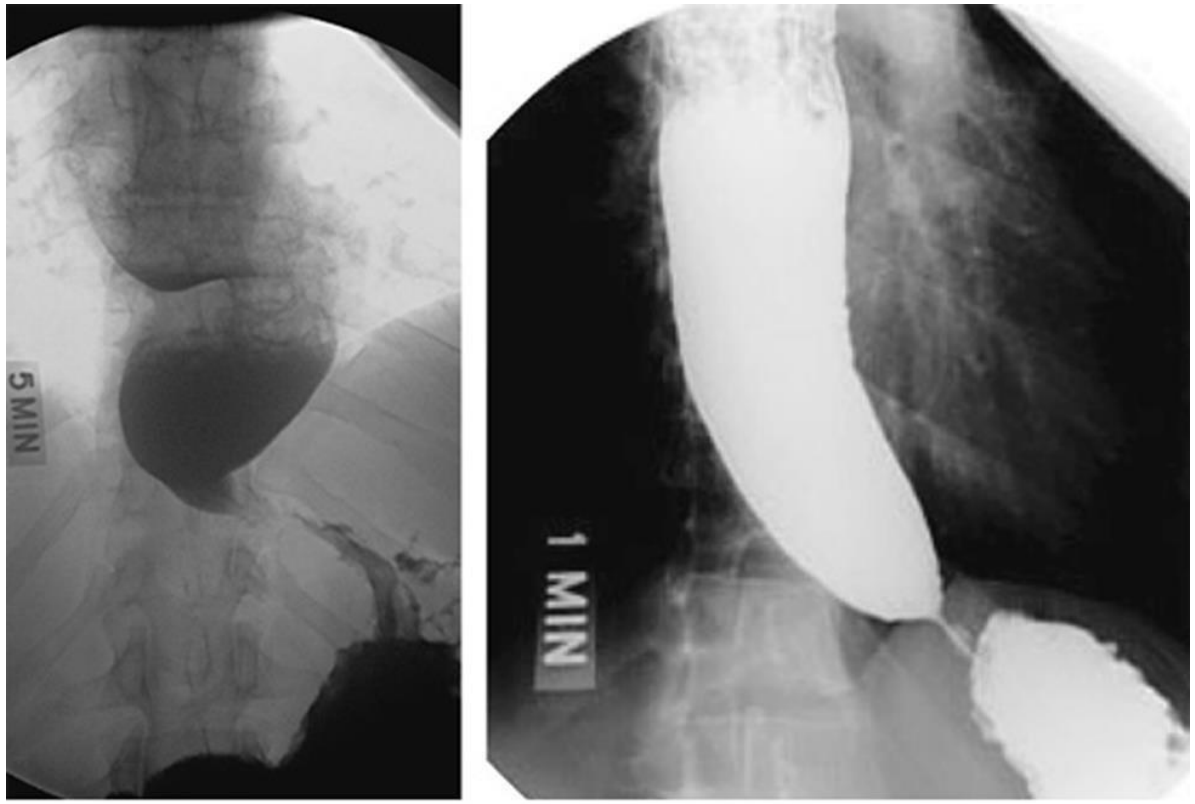
بسم الله الرحمن الرحيم

Gastrointestinal system pathology mid/final exam materials

Collected , written by Reenas Khresat

Achalasia



* Thin, non-canalized cord replaces a segment of esophagus.
* Most common location: at or near the tracheal bifurcation
* +-fistula (upper or lower esophageal pouches to a bronchus or trachea).

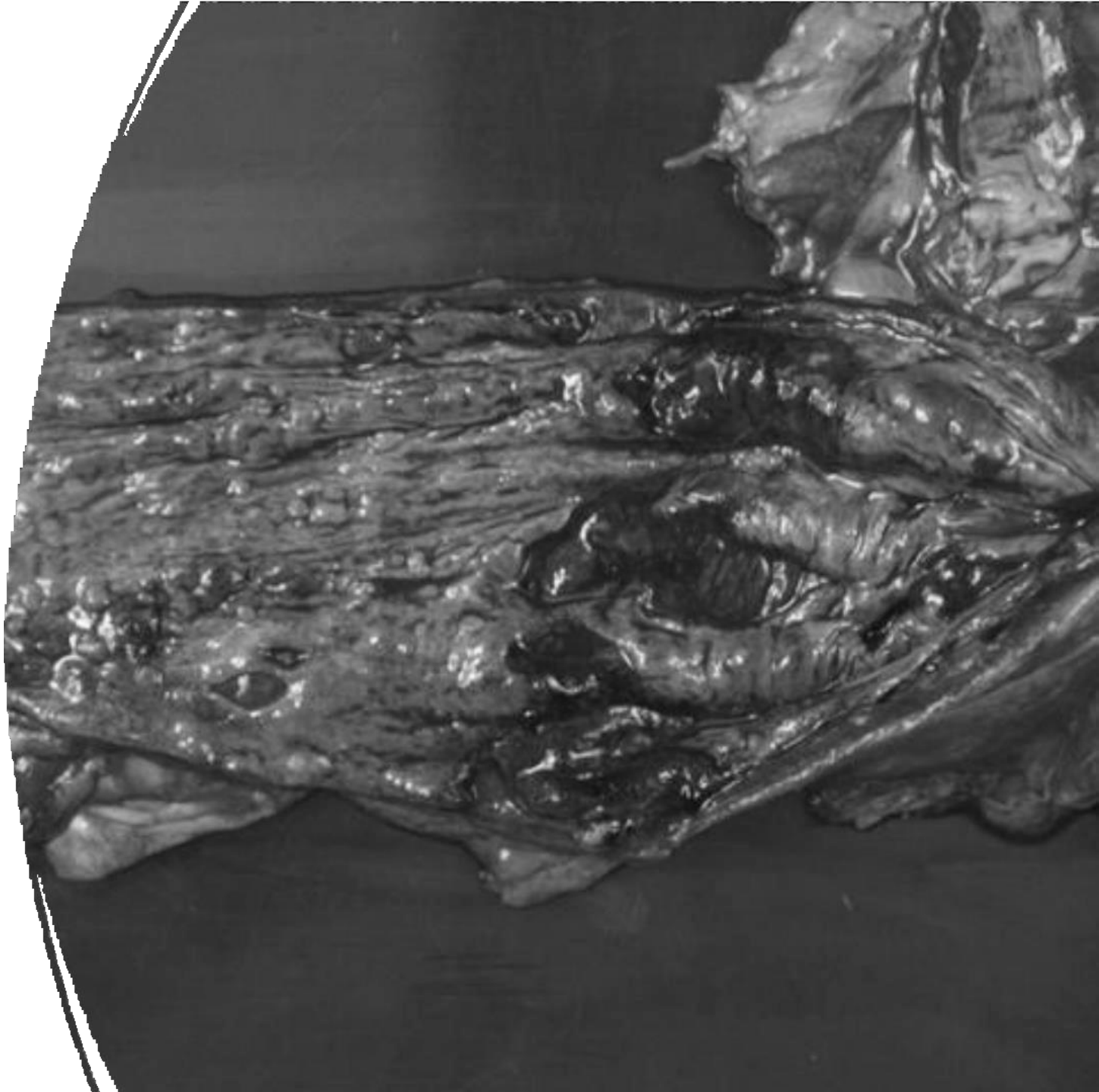
Shortly after birth: regurgitation during feeding

* Needs prompt surgical correction (rejoin).
* Complications if w/ fistula: Aspiration Suffocation- Pneumonia- Severe fluid and electrolyte imbalances.

Esophageal varices

Past paper question

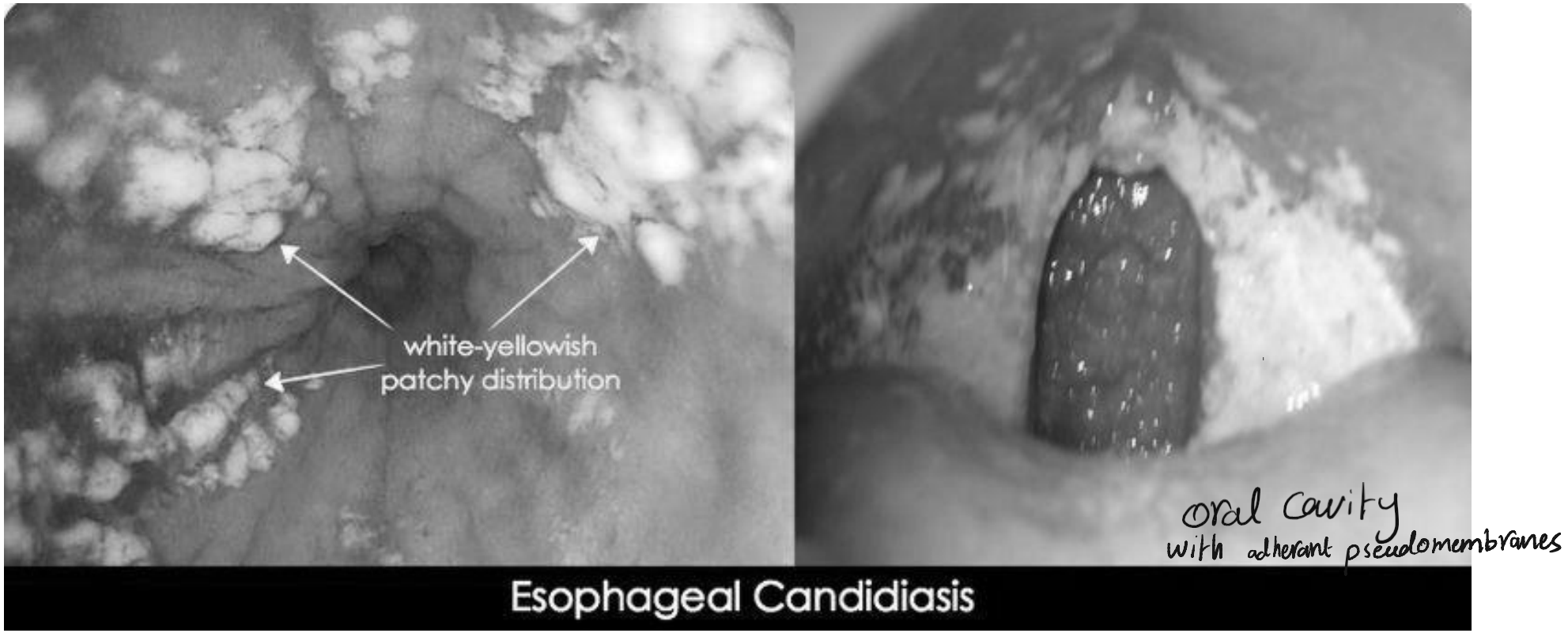
* Tortuous dilated veins within the submucosa of the distal esophagus and proximal stomach.
* Diagnosis by endoscopy or angiography.



Medpics - UCSD School of Medicine •

Esophageal candidiasis

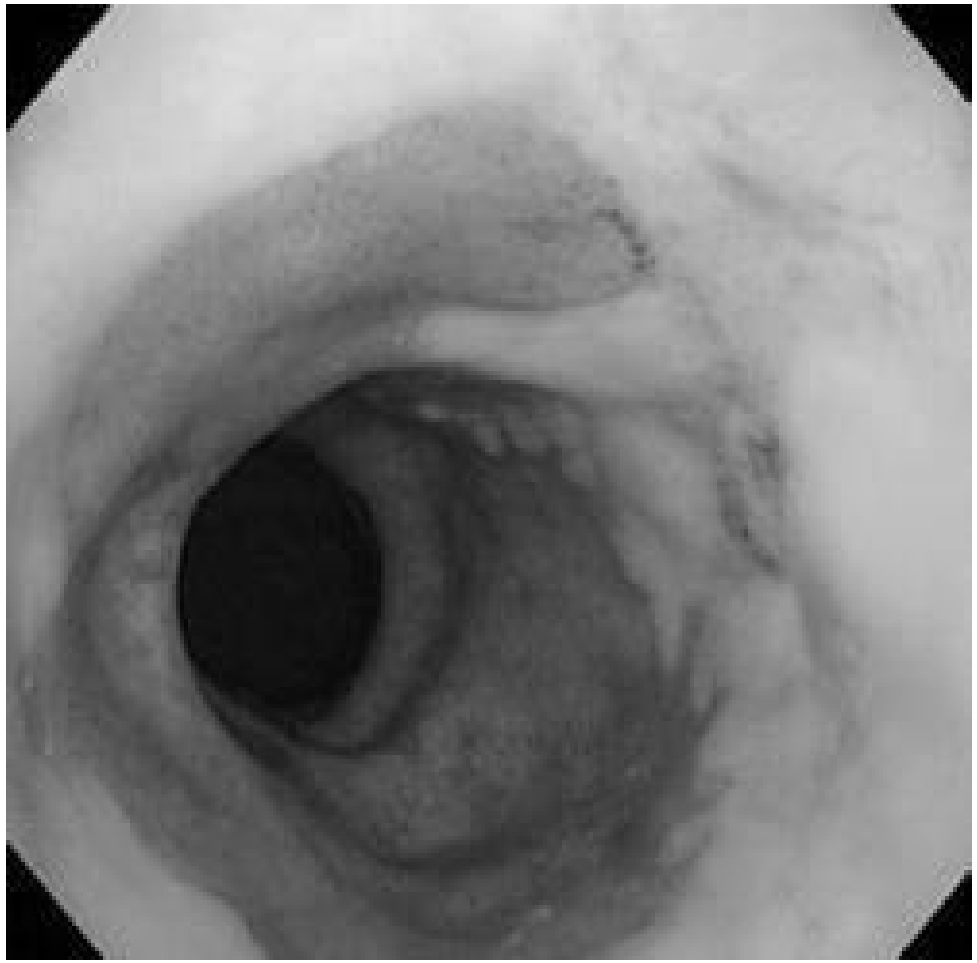
Whitish, grayish adherent pseudomembranes



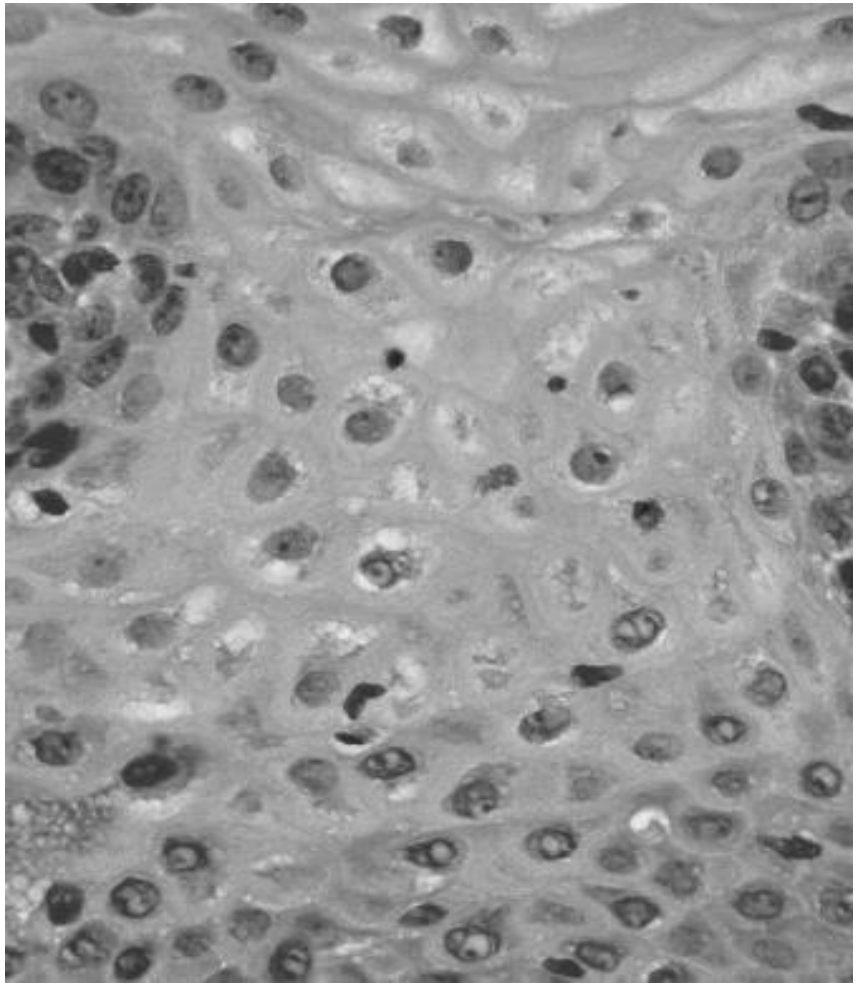
https://www.pinterest.com/pin/374291419013418659/

* Adherent
* Gray-white pseudo membranes , Composed of matted fungal hyphae and inflammatory cells

Reflux esophagilis it may cause severe chest pain (mistaken for heart disease similar to myocardial infarction).



Reflux esophagitis



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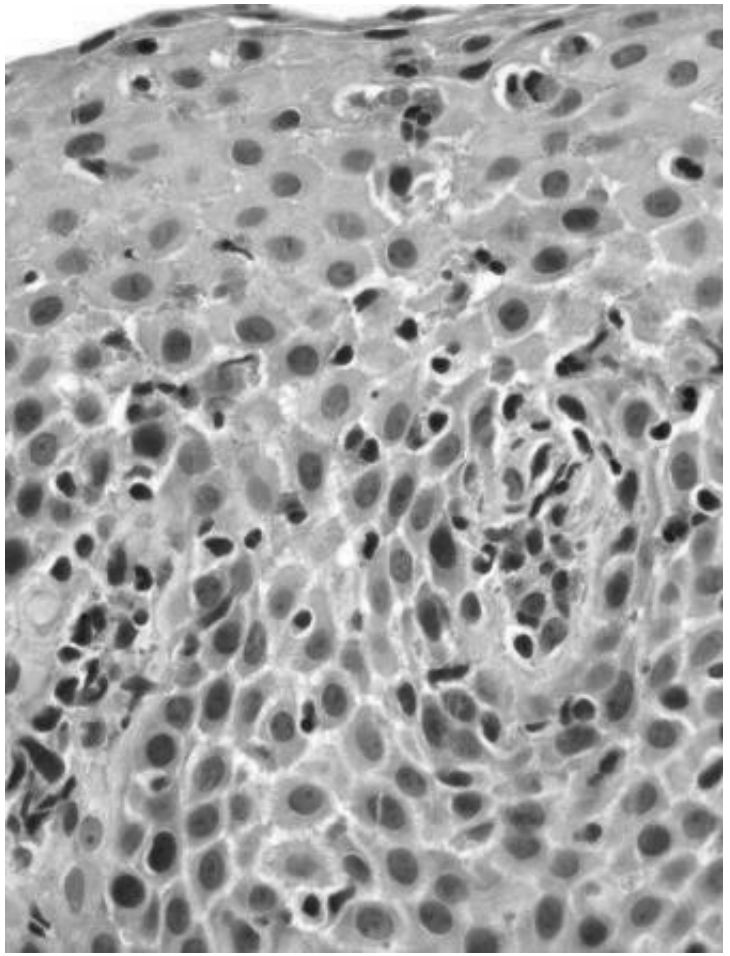
Rings in eosinophilic esophagitis

Chronic, immune mediated disorder

* The rings appear in the upper , middle part of esophagous.



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Robbins

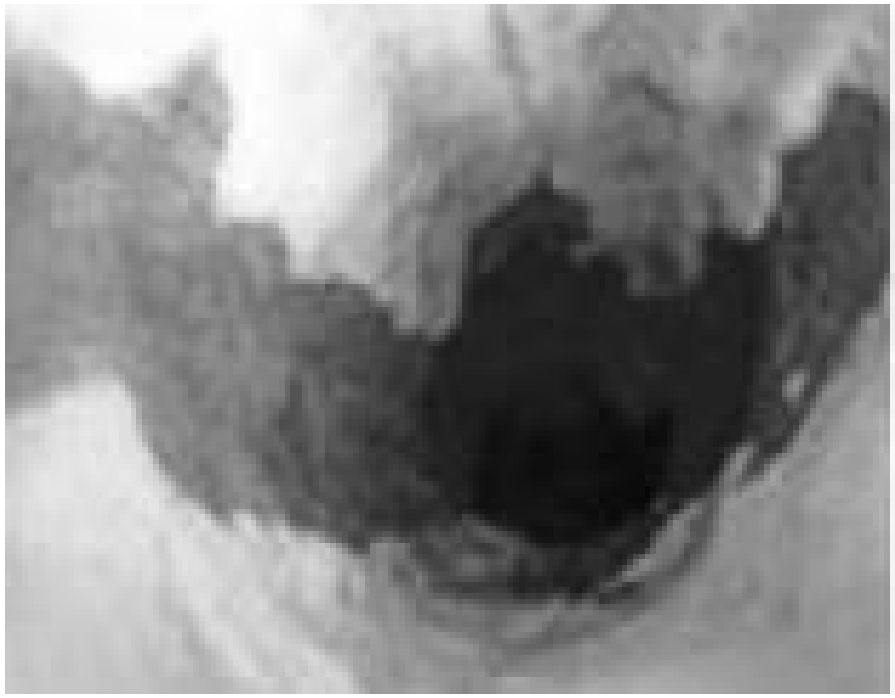
Case

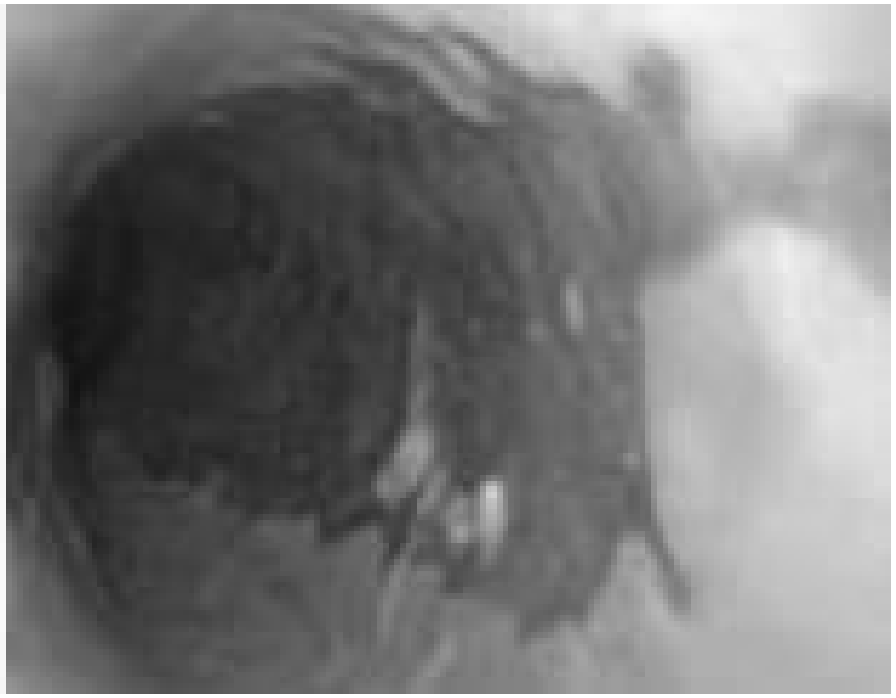
* Numerous eosinophils within epithelium the treatment of this is corticosteroids No milk or soy products intake
Most pts suffer from dermatitis, allergic rhinitis, asthma

- ***Eosinophilic Esophagitis***
- Chronic immune mediated disorder
- Symptoms: Food impaction and dysphagia in adults , Feeding intolerance or GERD-like symptoms in children
- Morphology: Rings in the upper and mid esophagus.
- Numerous eosinophils in epithelium , Far from the GEJ.

Tongues in Barrett esophagus

Direct causation of adenocarcinoma





A complication from GERD
Metaplasia (from squamous epithelium to columnar epithelium

Barrett Esophagus

Complication of chronic GERD 1-Intestinal metaplasia.

2- 10% of individuals with symptomatic GERD

3- Males>>females, 40-60 yrs Direct precursor of esophageal adenocarcinoma

4- 0.2-1% /year develop dysplasia (precursor of adenocarcinoma)MORPHOLOGY

5- Endoscopy: Red tongues extending upward from the GEJ.

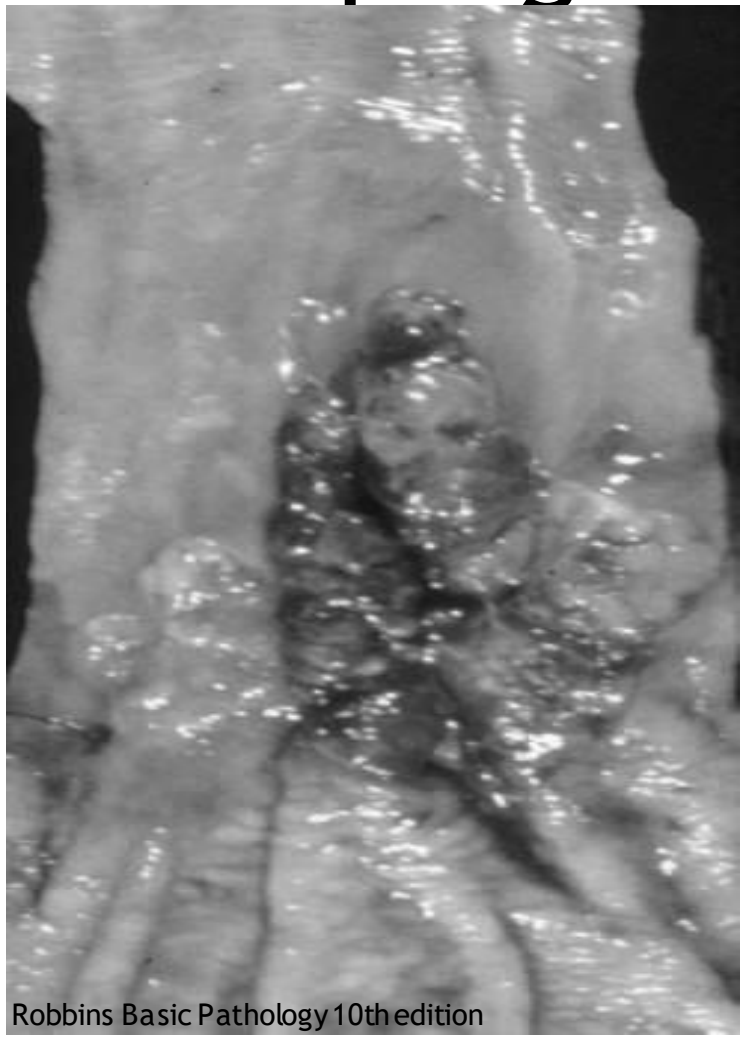
6- Histology: Intestinal metaplasia (defined by Presence of goblet cells) +-Dysplasia : low-grade or high-grade

7- Intramucosal carcinoma: invasion into the lamina propria.

Arises from background of Barret, long standing GERD

Esophageal adenocarcinoma

Mutation in TP53 gene and chromosomal abnormalities



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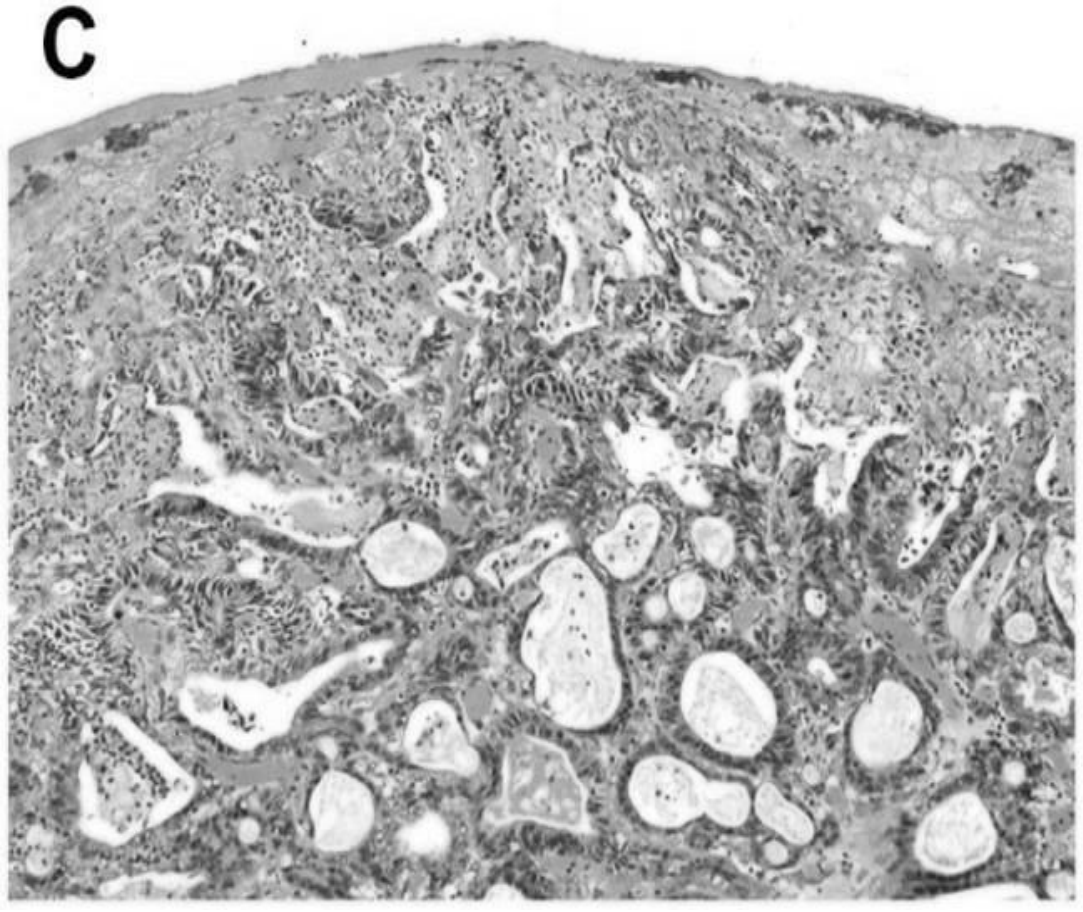


- Distal third.
- Early: flat or raised patches
- Later: exophytic infiltrative masses
- Microscopy: Forms glands and mucin.

Mid esophagus

- Esophageal squamous cell carcinoma

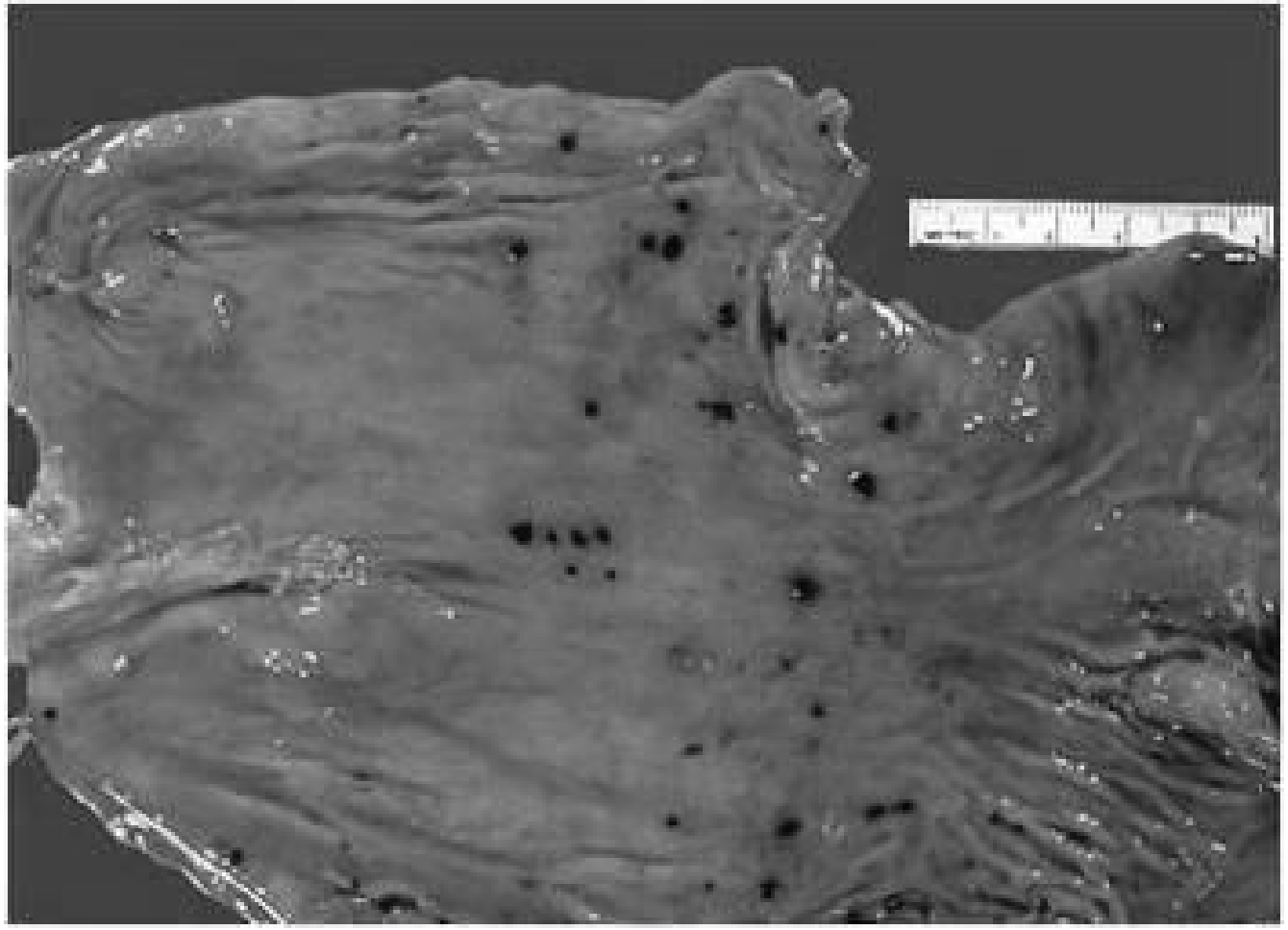
- Male : female (4:1)

More in rural, low resource countries.
Risk factors: Alcohol Squamous Cell Carcinoma ,Tobacco use , Poverty , Caustic injury , Achalasia .
Plummer-Vinson syndrome (iron deff.anemia, dysphagia,webs)
Frequent consumption of very hot beverages
Previous radiation Tx .
In western : alcohol and tobacco use. Pathogenesis Other areas: nutritional deficiency, polycyclic hydrocarbons, nitrosamines, fungus-contaminated foods
HPV infection implemented in high-risk regions.
MORPHOLOGY
Middle third (50% of cases)

- Polypoid, ulcerated, or infiltrative.
- Wall thickening, lumen narrowing
- Invade surrounding structures (bronchi, mediastinum, pericardium, aorta).

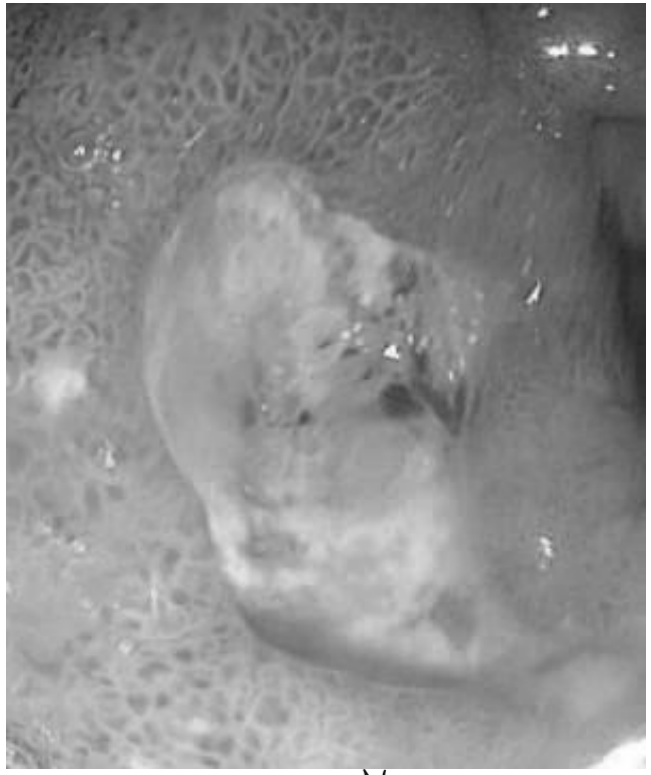
Stress gastric ulcers

Past paper question !!

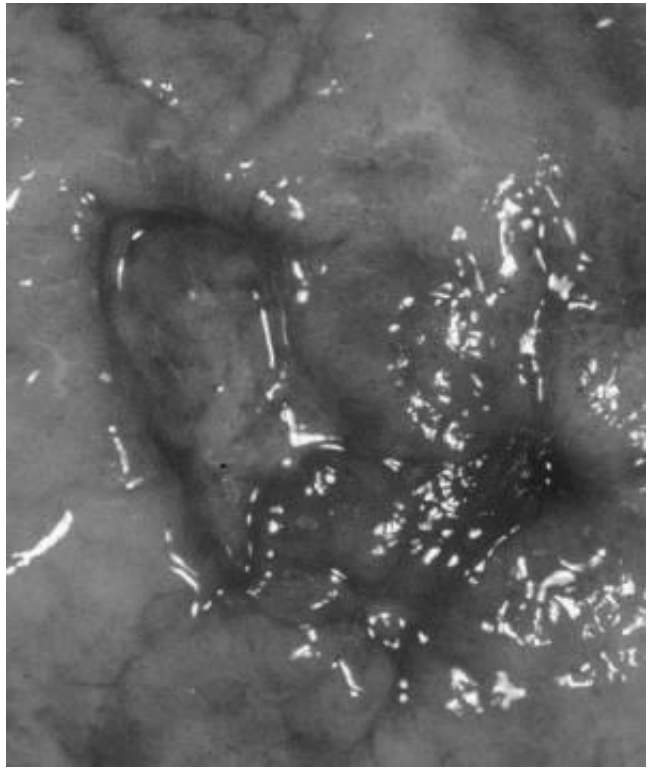


- ***Stress related injury:***
- Mostly due to Local ischemia caused by.
- Systemic hypotension.
- Decreased blood flow (Splanchnic vasoconstriction)
- Systemic acidosis (lower intracellular PH).
- COX2 expression is protective.
- CNS injury and Cushing ulcers: Direct vagal stimulation, acid hypersecretion.

- ***Clinical features***
- Nausea, vomiting, Melena ,Coffee -ground hematemesis , Perforation complication.
  Prophylaxis with proton pump inhibitors
  Outcome depends on severity of underlying cause



punched out, white clean oval background ulcers.



- new blood vessels formation
- The center of epithelium is lost

Chronic gastric ulcers

main causes

- H. pylori
- NSAIDs

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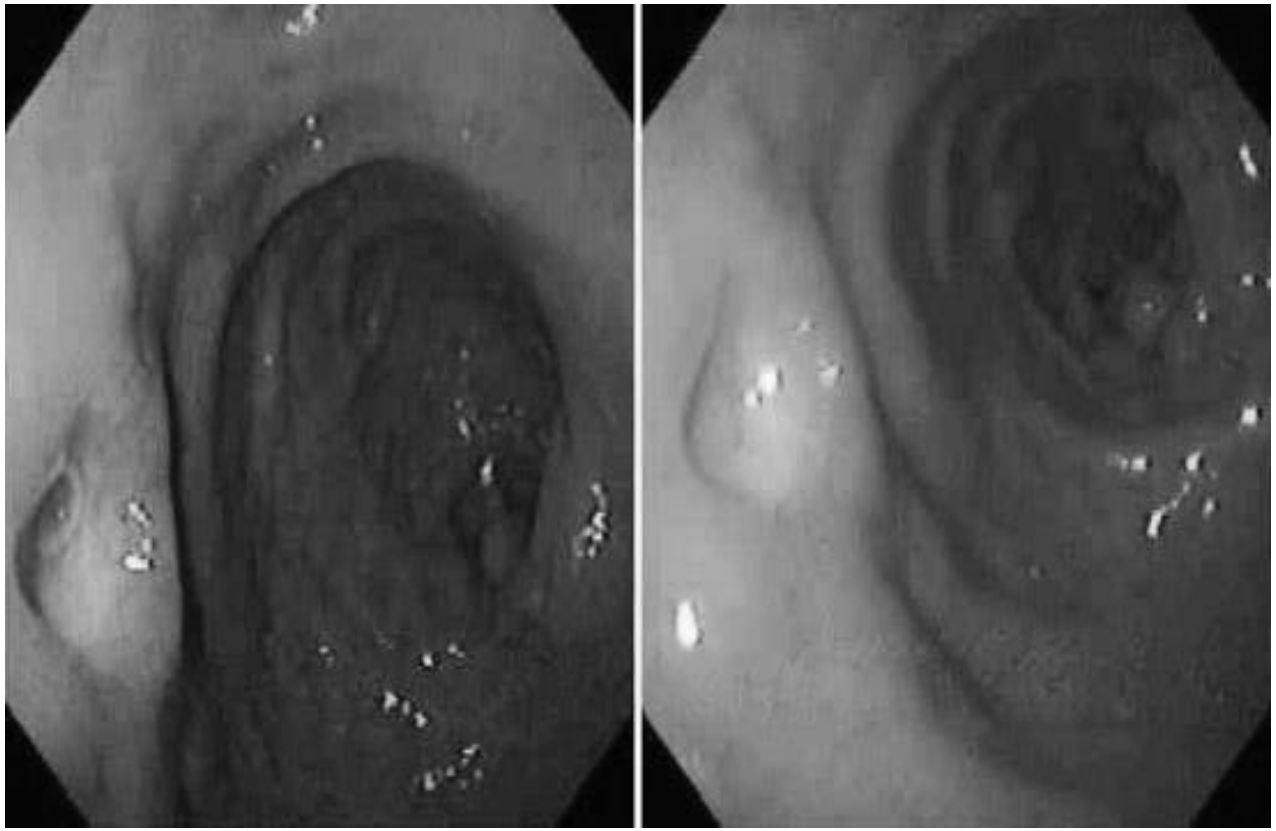
Duodenal ulcer well circumscribed ulcer in the wall of doudenum.

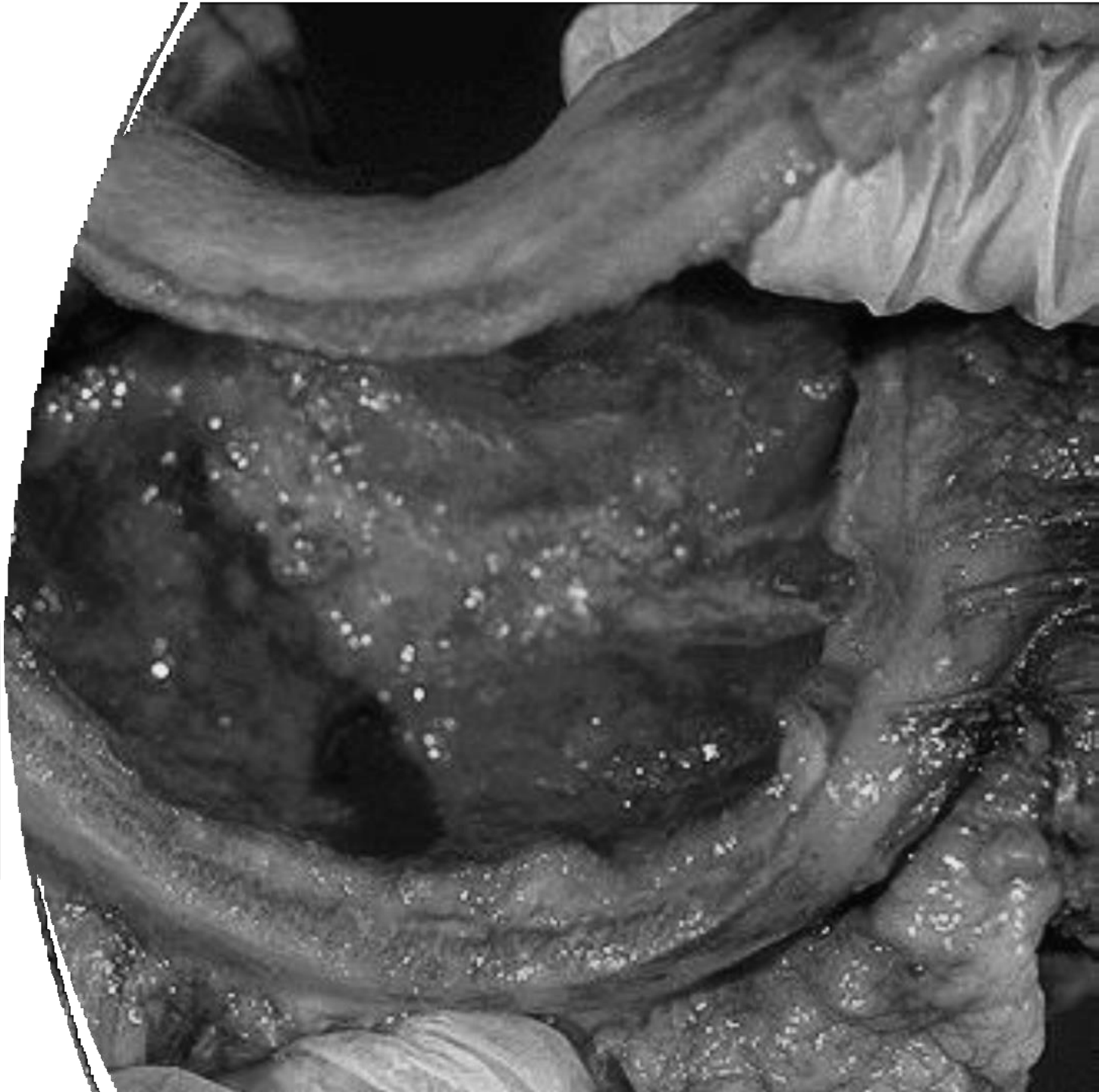


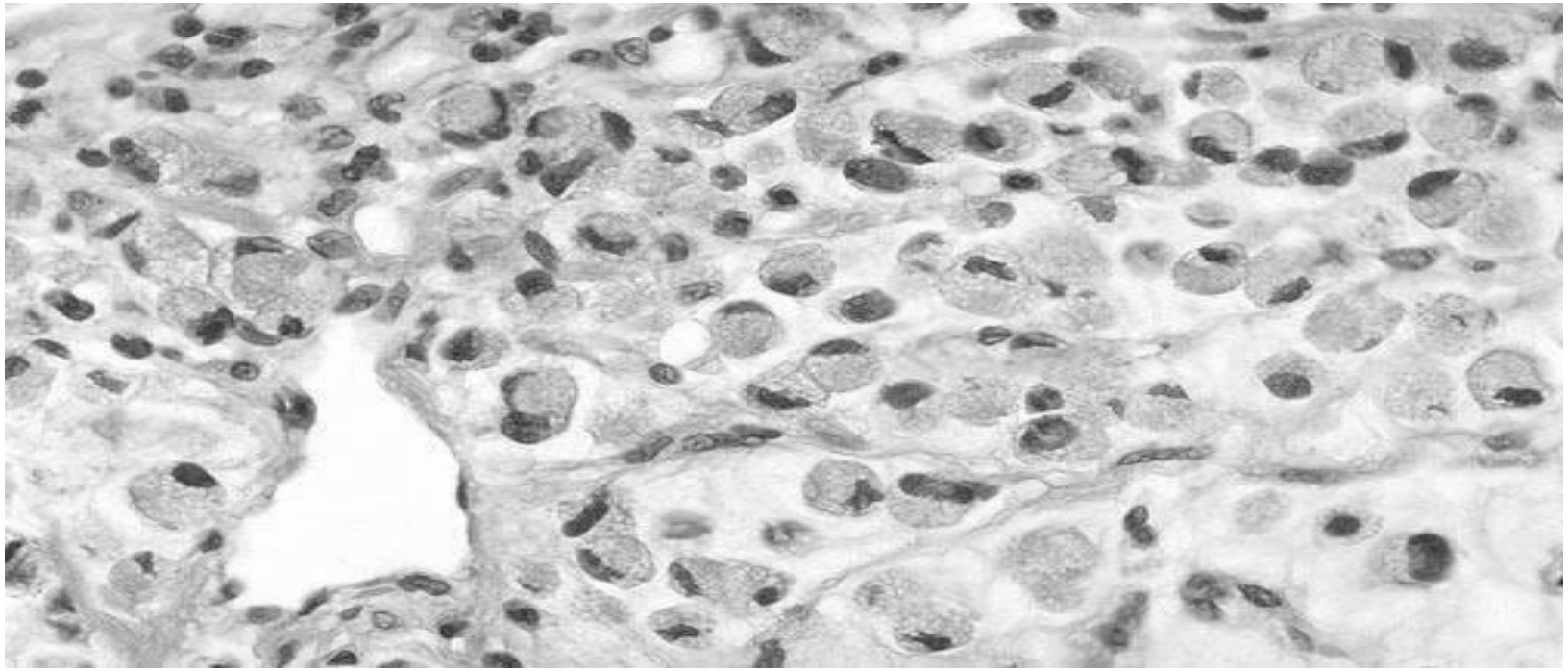
- ***Clinical Features***
- Epigastric burning or aching pain , Complication: Iron deficiency anemia, frank hemorrhage, or perforation.
- Pain 1 to 3 hours after meals at daytime
- Worse at night, relieved by alkali or food
- Nausea, vomiting, bloating, bletching.
- Current therapies are aimed at H.pylori eradication.
- Surgery reserved for complications.

Diffuse type gastric cancer (linitis plastica)

- ***Diffuse type:***
- Infiltrative growth pattern
- Discohesive cells (signet ring cells)
- Desmoplastic reaction (stiffens wall, flat ruge, linitis plastic). Mutation in CDH 1 gene

↳ E-cadherin

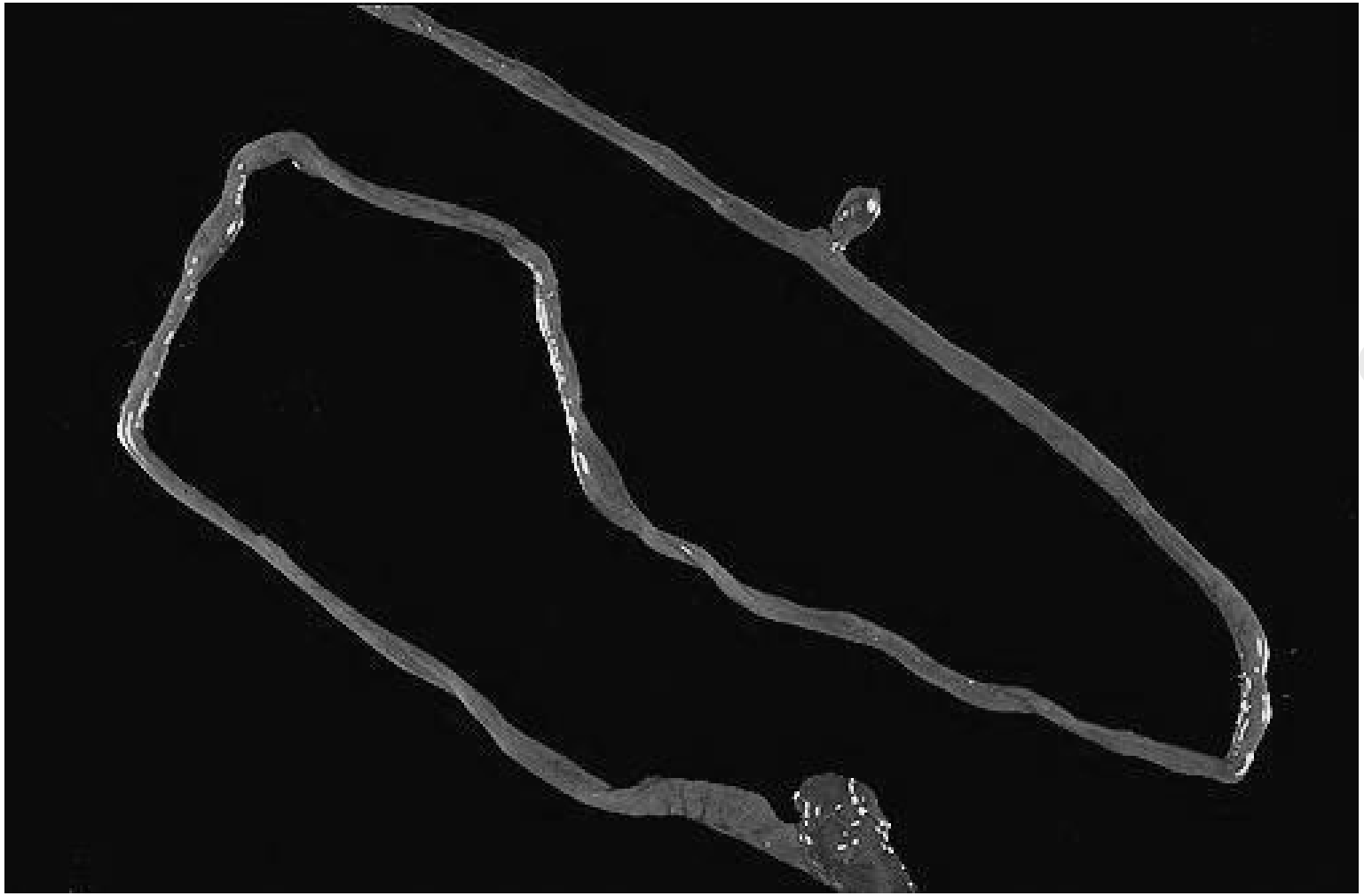




Diffuse type, signet ring cells

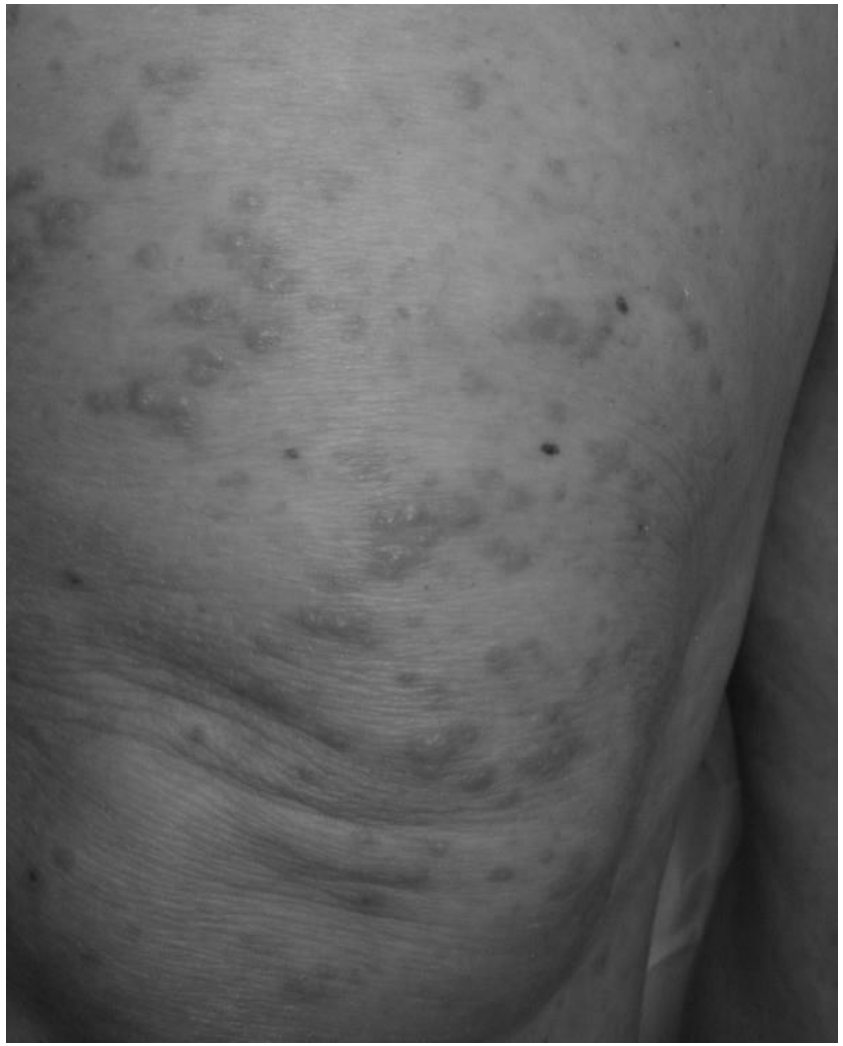
Signet ring cells: large mucin vacuoles that expand the cytoplasm and push the nucleus to the periphery,
Mutation in CDH 1 gene

Meckel's diverticulum



- The most common congenital anomaly of the GI tract
- Incomplete obliteration of omphalomesenteric duct
- True diverticulum.
- Remember (rule of 2):
- About 2% of people have them;
- Located 2 feet from the ileocecal valve.
- 2 inches in length.
- 2 types of heterotopic mucosa (gastric or pancreatic).
- Most common cause of lower GI bleeding before age of 2.

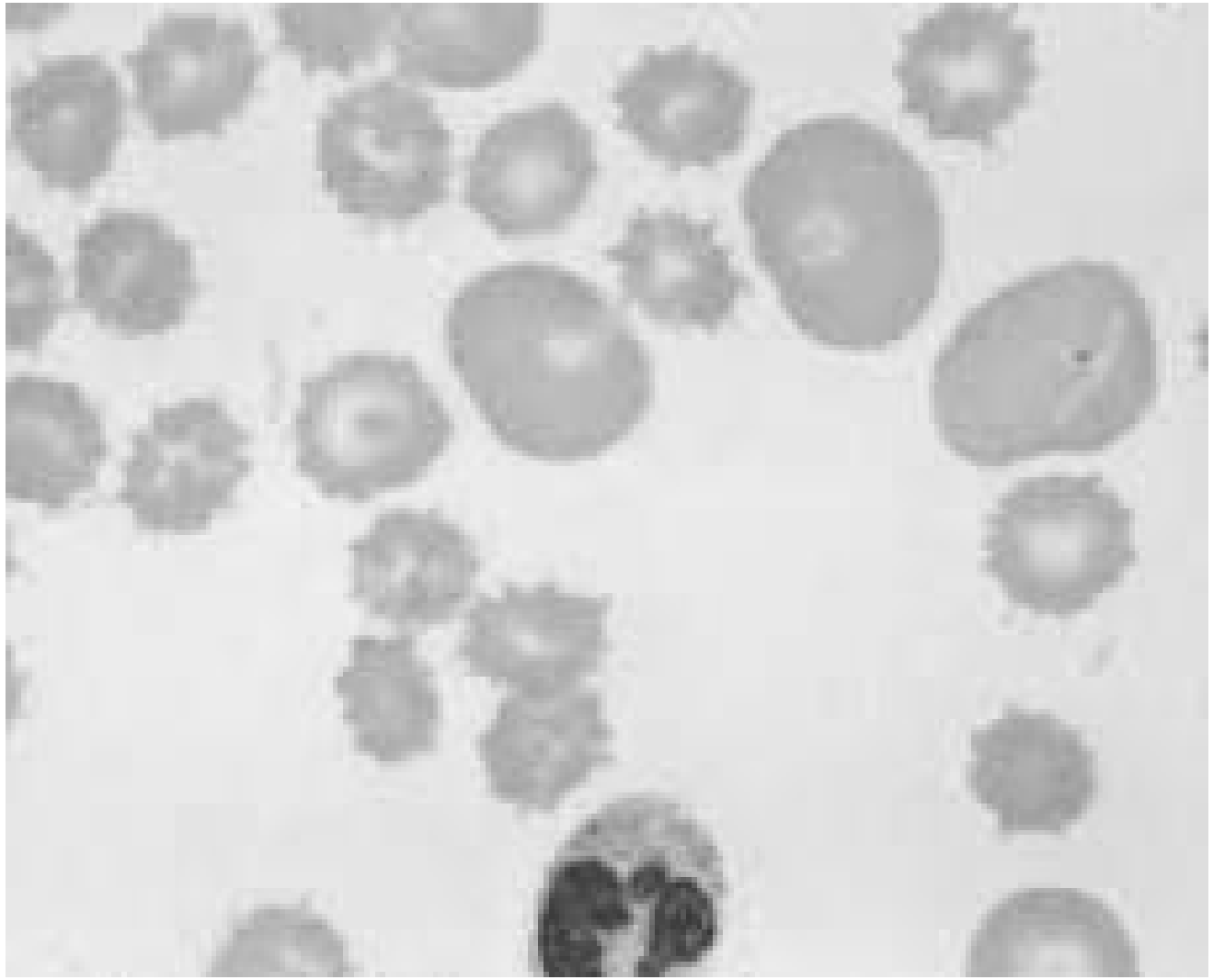
Dermatitis herpetiformis with celiac disease.

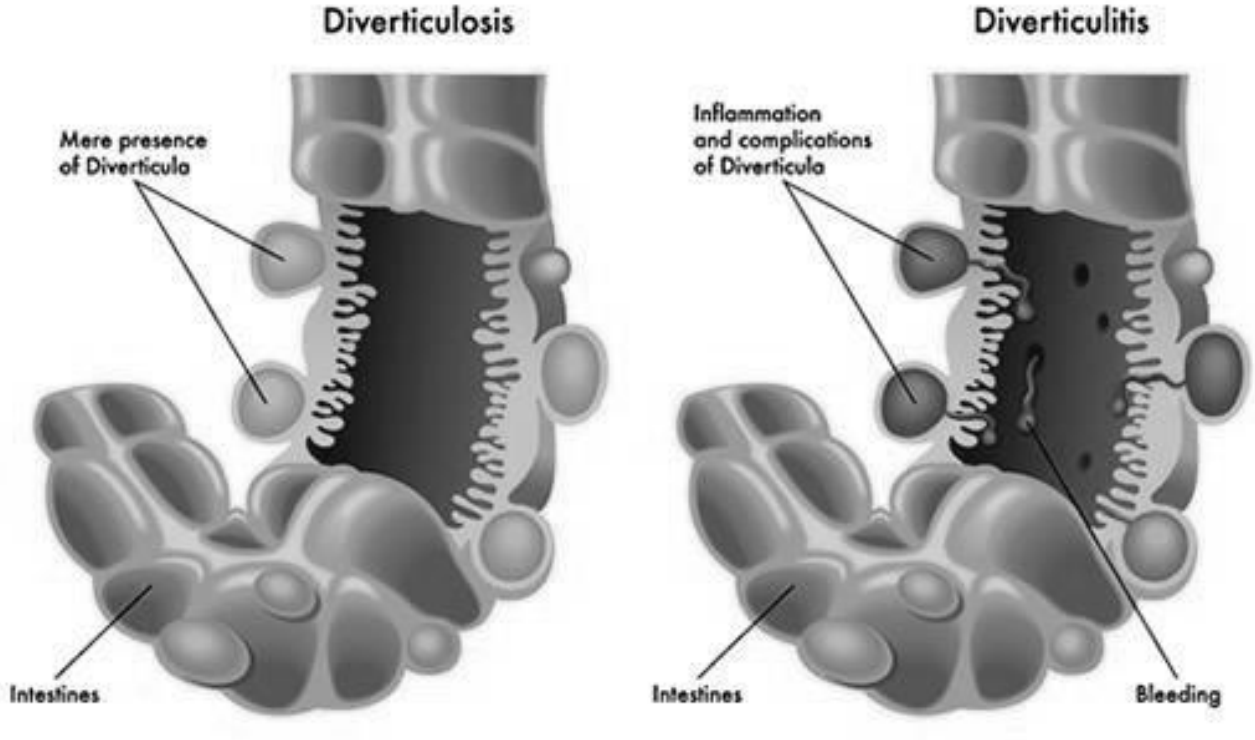


- Adults (30-60 years)
- Anemia: iron deficiency
- B12 and folate deficiency: less common.
- Diarrhea , bloating, and fatigue.
- Missed diagnosis: Silent celiac (positive serology and biopsy but asymptomatic).
- Increased risk of enteropathy associated T cell lymphoma & Small intestinal adenocarcinoma.

- Spur cells in abetalipoproteinemia

- ***Abetalipoproteinemia***
- Autosomal recessive, rare.
- Inability of enterocytes to secrete triglyceride-rich chylomicrons.
- Lack of absorption (Transepithelial transport defect of lipoproteins, FAs and fat-soluble vitamins).
- Infants' w/ failure to thrive, diarrhea, and steatorrhea
- Vitamin K deficiency, skeletal CNS and retinal abnormalities.
- Spur cells in peripheral blood.
- Monoglycerides and triglycerides accumulate in epithelial cells.





Pathogenesis: Elevated intraluminal pressure. Unique location (discontinuous muscle layer at points of nerve and vessels entry). Longitudinal muscle layer is discontinuous in colon (taeniae coli) Area of weakness: outward herniation of mucosa and submucosa . Most common in sigmoid (narrowest part) Exaggerated peristaltic contractions. Low fiber diet, constipation, sedentary lifestyle, obesity, and smoking.

MORPHOLOGY

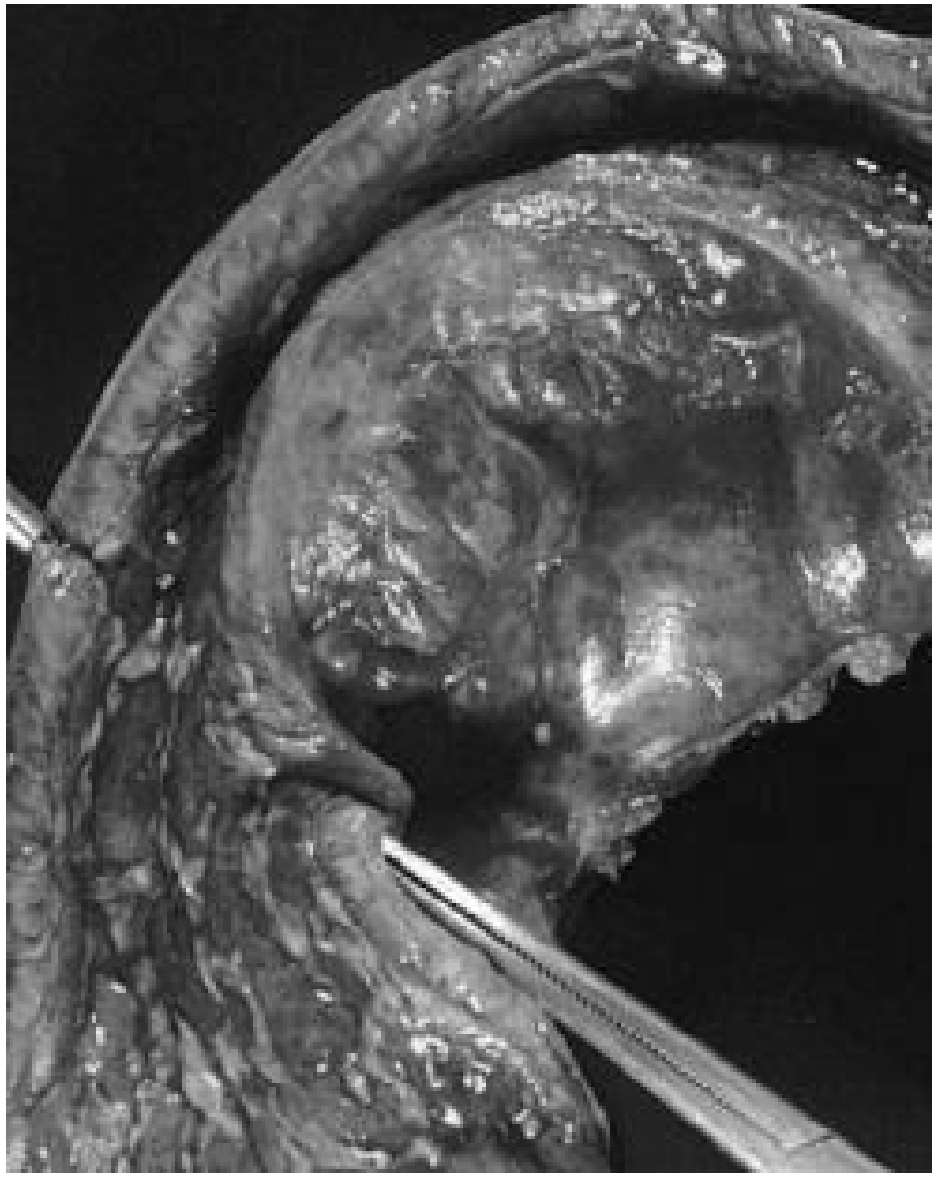
Flasklike outpouchings Between taeniae coli. Thin wall (atrophic mucosa, compressed submucosa) Attenuated or absent muscularis propria. Obstruction leads to diverticulitis. Risk of perforation. Recurrent diverticulitis leads to fibrosis (strictures).

Small bowel stricture

Crohn disease

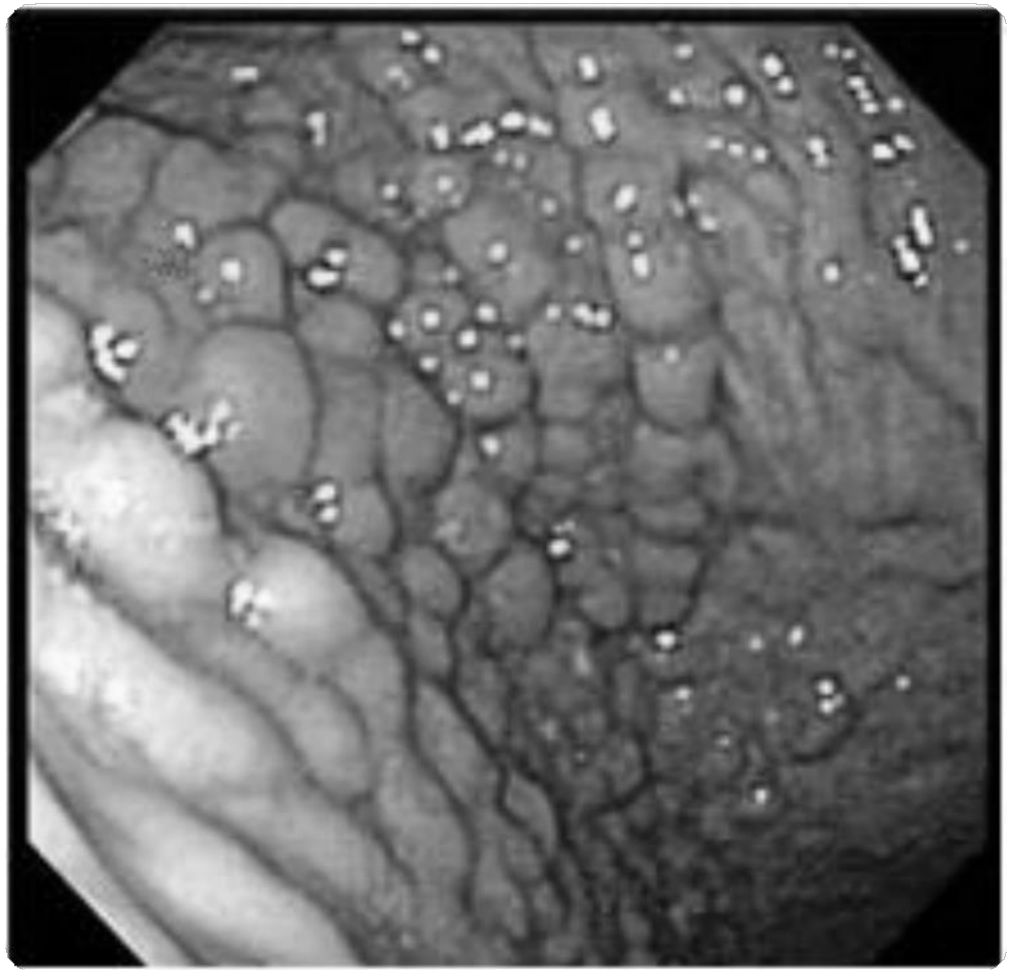
Earliest lesion: aphthous ulcer

- Elongated, serpentine ulcers.
- Edema , loss of bowel folds.
- Cobblestone appearance
- Toxic megacolon (before fibrosis)
- Fissures (fistulas, perforations).
- Thick bowel wall (transmural inflammation, edema, fibrosis, hypertrophic MP) >>strictures.
- Creeping fat



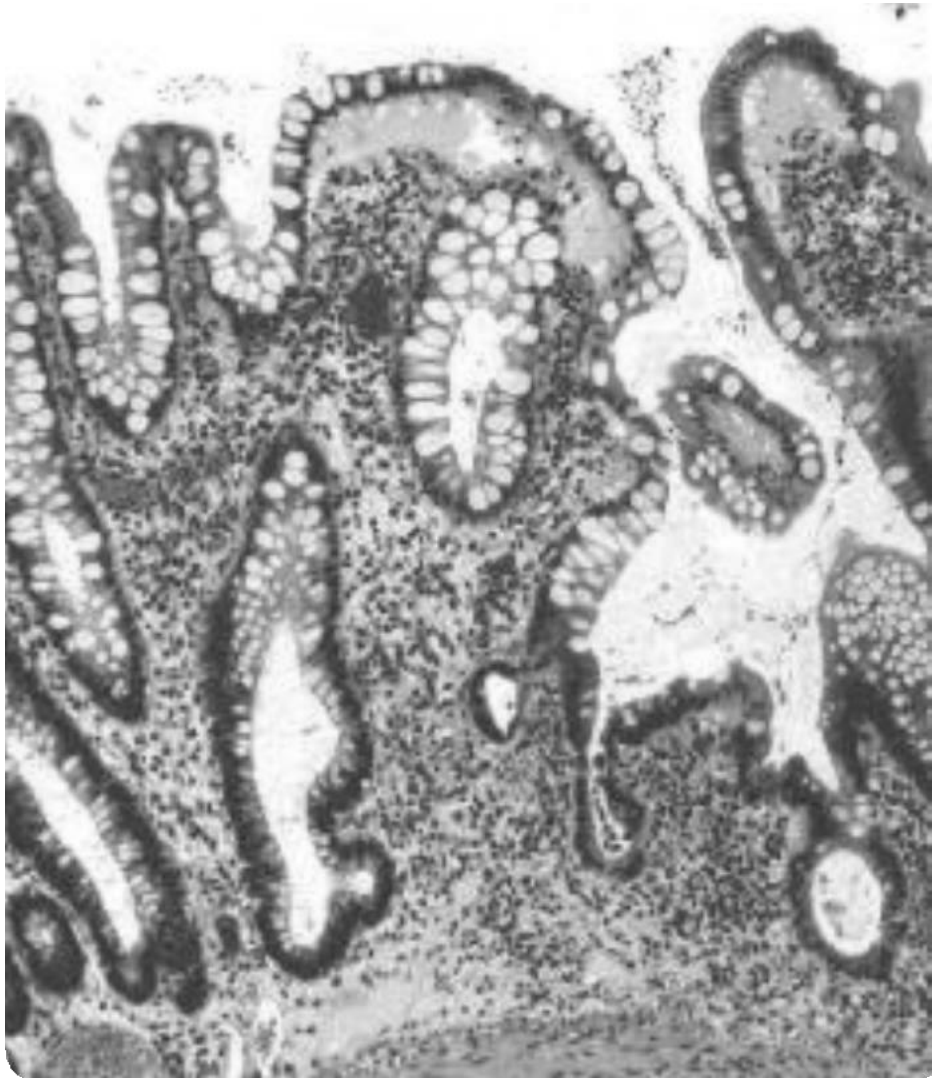
Cobblestone appearance

Cobblestone appearance, Crohn disease

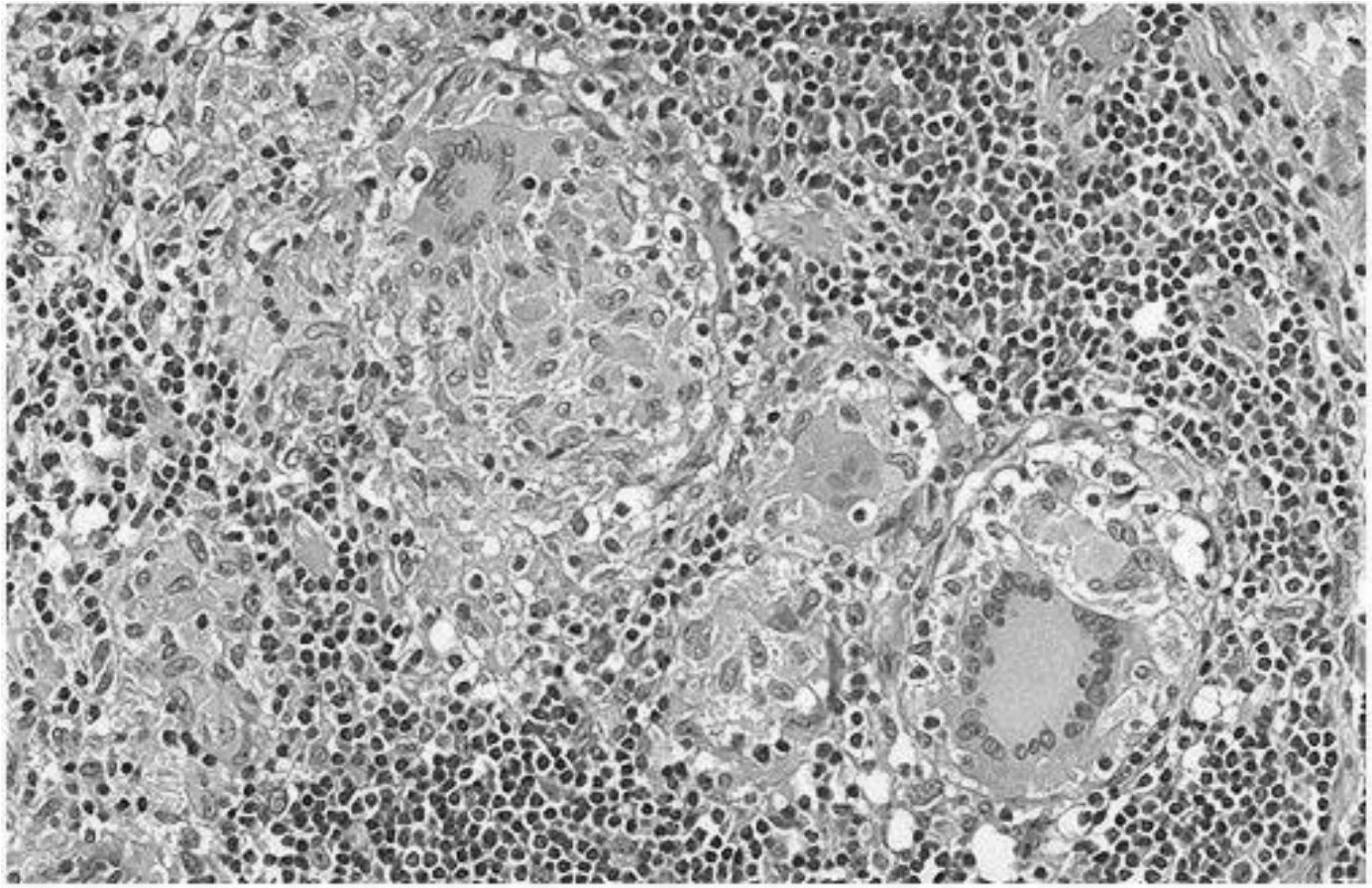


ResearchGate

Haphazardly arranged crypts, C IBD



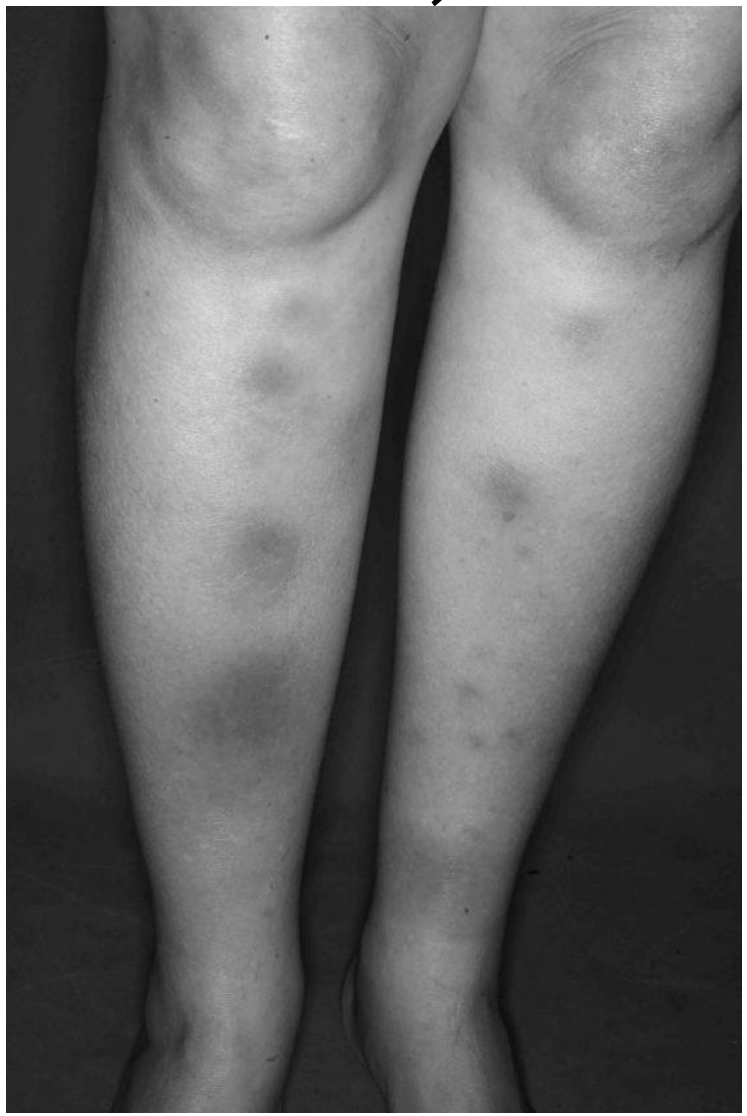
Non-caseating granuloma, Crohn disease.



Clinical Features

- Intermittent attacks of mild diarrhea, fever, and abdominal pain.
- Acute right lower-quadrant pain and fever (20%)
  - Bloody diarrhea and abdominal pain (colonic disease)
- Asymptomatic intervals (weeks to months)
- Triggers: physical or emotional stress, specific dietary items, NSAID use, and cigarette smoking.
- Complications: Colonic: Iron-deficiency anemia
- Small bowel: Hypoproteinemia and hypoalbuminemia, malabsorption of nutrients, vitamin B12 and bile salts, Fistulas, peritoneal abscesses, strictures ,Risk of colonic and small intestinal adenocarcinoma ……

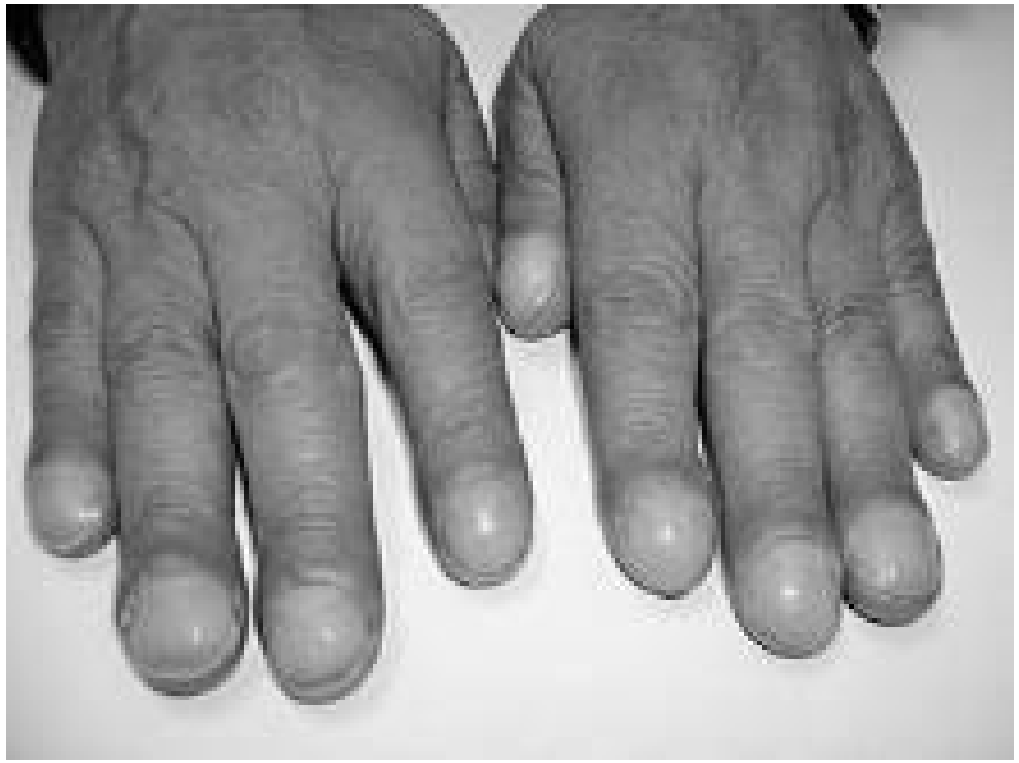
Erythema nodosum, Crohn disease



Neurology Advisor

- Red, elevated lesions that appear mainly in the lower limb.

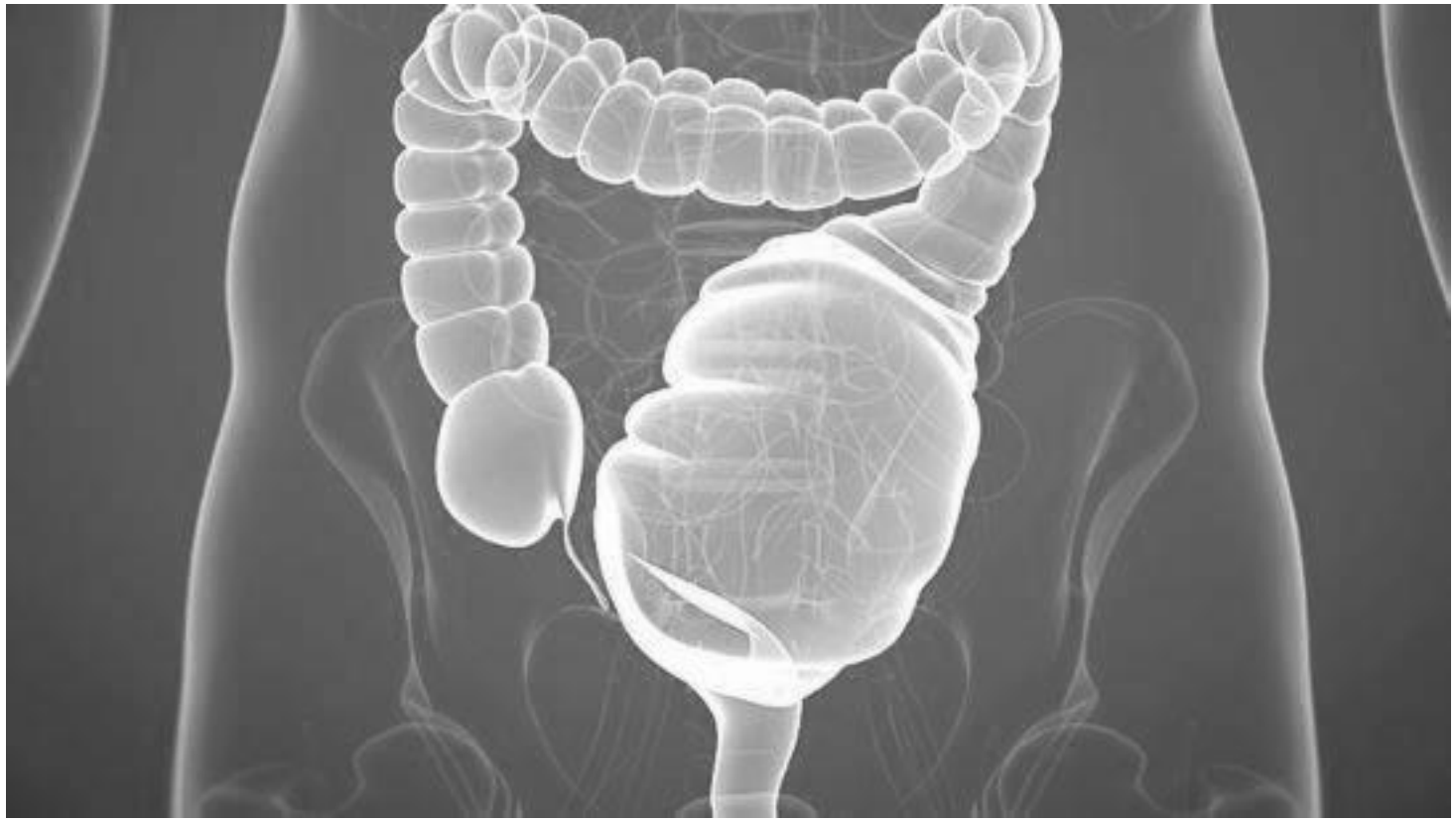
Clubbing



- appears in longstanding crohn's disease.
- Non specific sign

Wikipedia

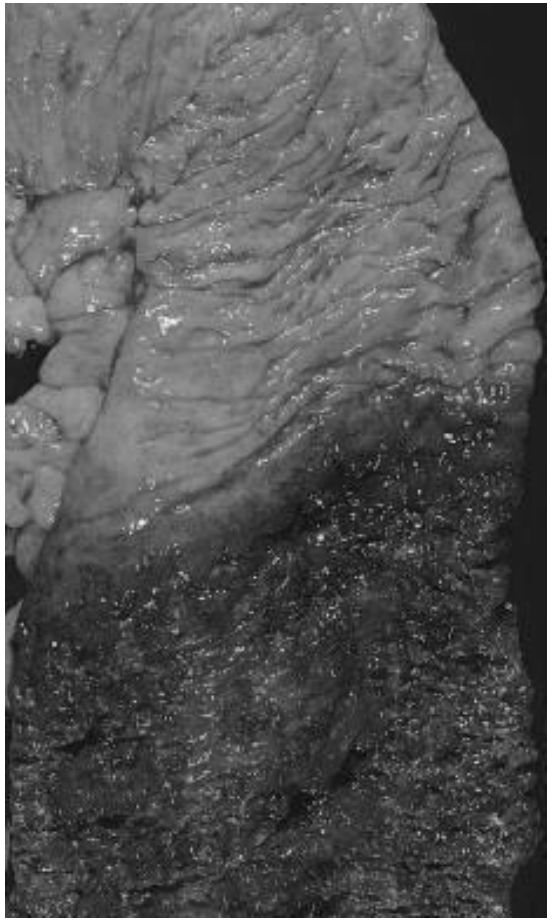
Toxic megacolon → has the risk of rupture, sepsis and perforation



Pancolitis.

- The whole colon is involved
- Most severe form of UC.

Abrupt transition b/w normal and disease segment, ulcerative colitis.

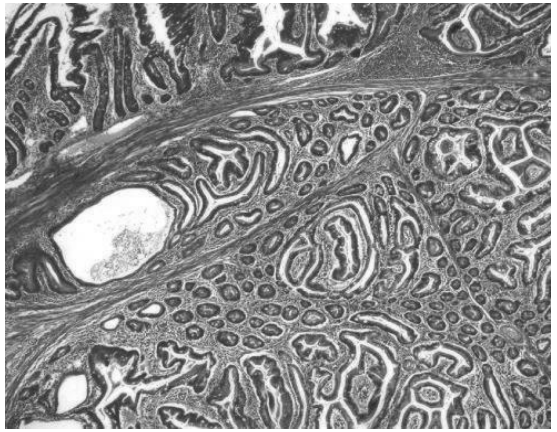


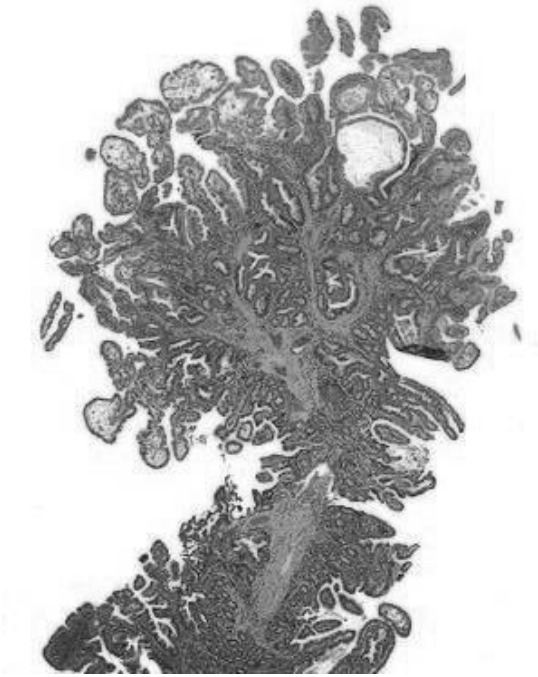
Clinical Features

Relapsing remitting disorder
Attacks of bloody mucoid diarrhea +lower abdominal cramps
Temporarily relieved by defecation
Attacks last for days, weeks, or months.
Asymptomatic intervals.
Infectious enteritis may trigger disease onset, or cessation of smoking.
Colectomy cures intestinal disease only
Anti-inflammatory and biologic agents.

Peutz-Jeghers polyp

- Christmas tree pattern.

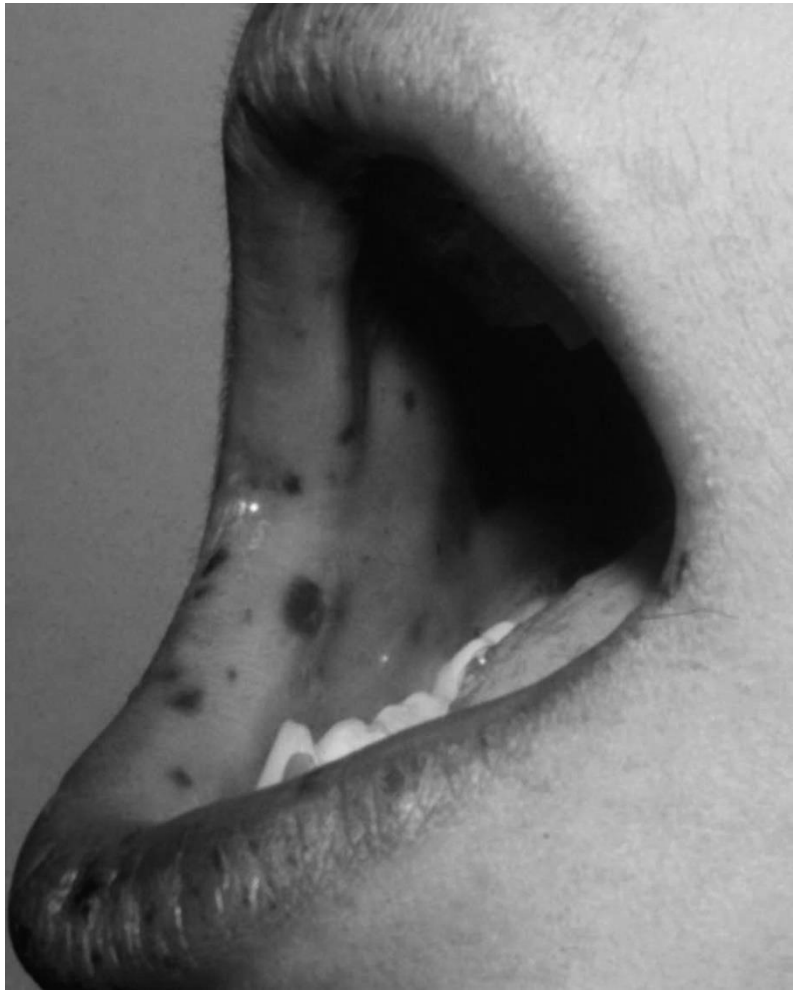


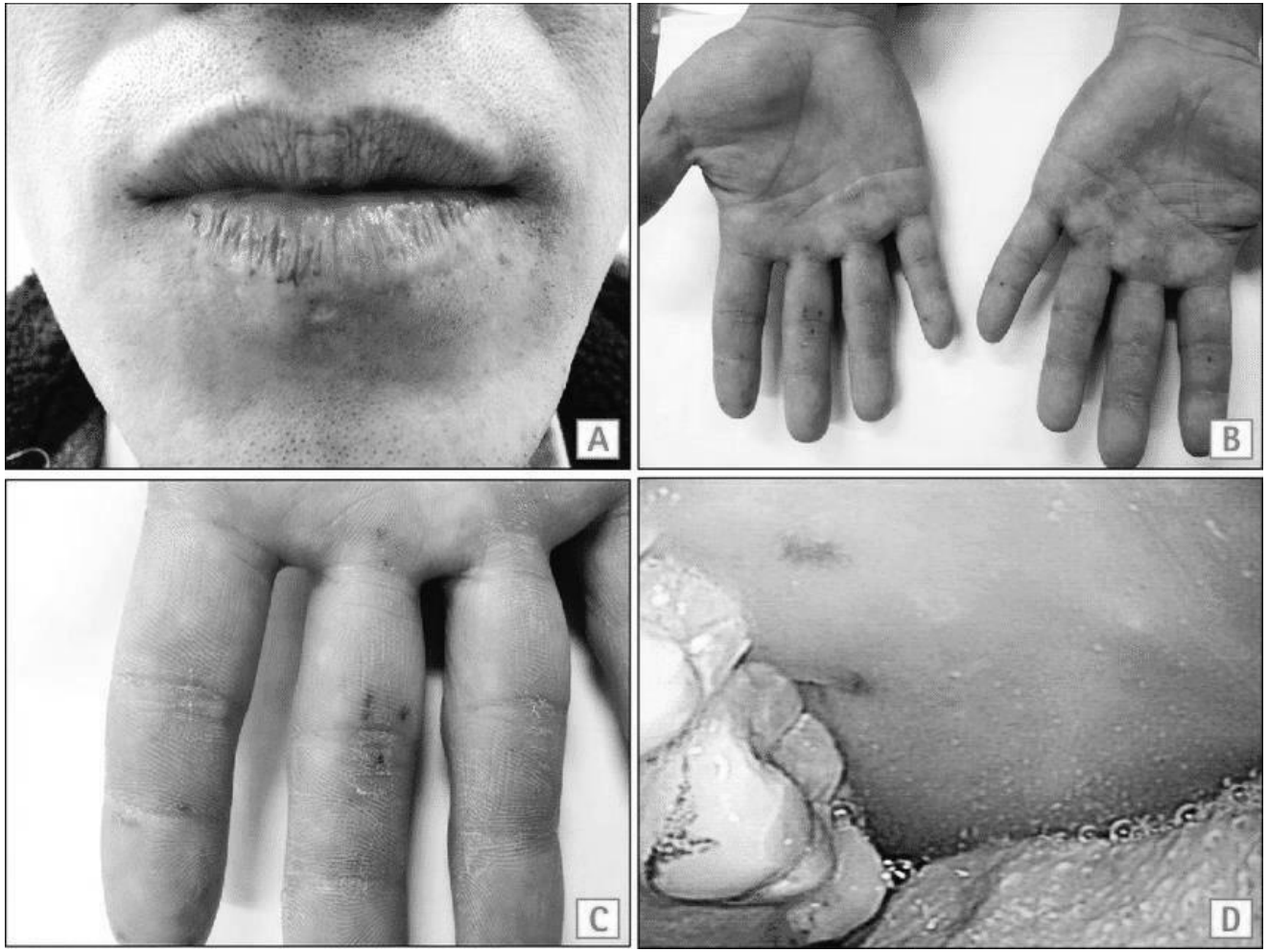


Mostly in small intestine.

1- Large, pedunculated, lobulated.
Arborizing network of connective tissue, smooth muscle
2- lamina propria and glands
3- Normal-appearing intestinal epithelium
Christmas tree pattern.

Mucocutaneous pigmentation, Peutz Jegher syndrome

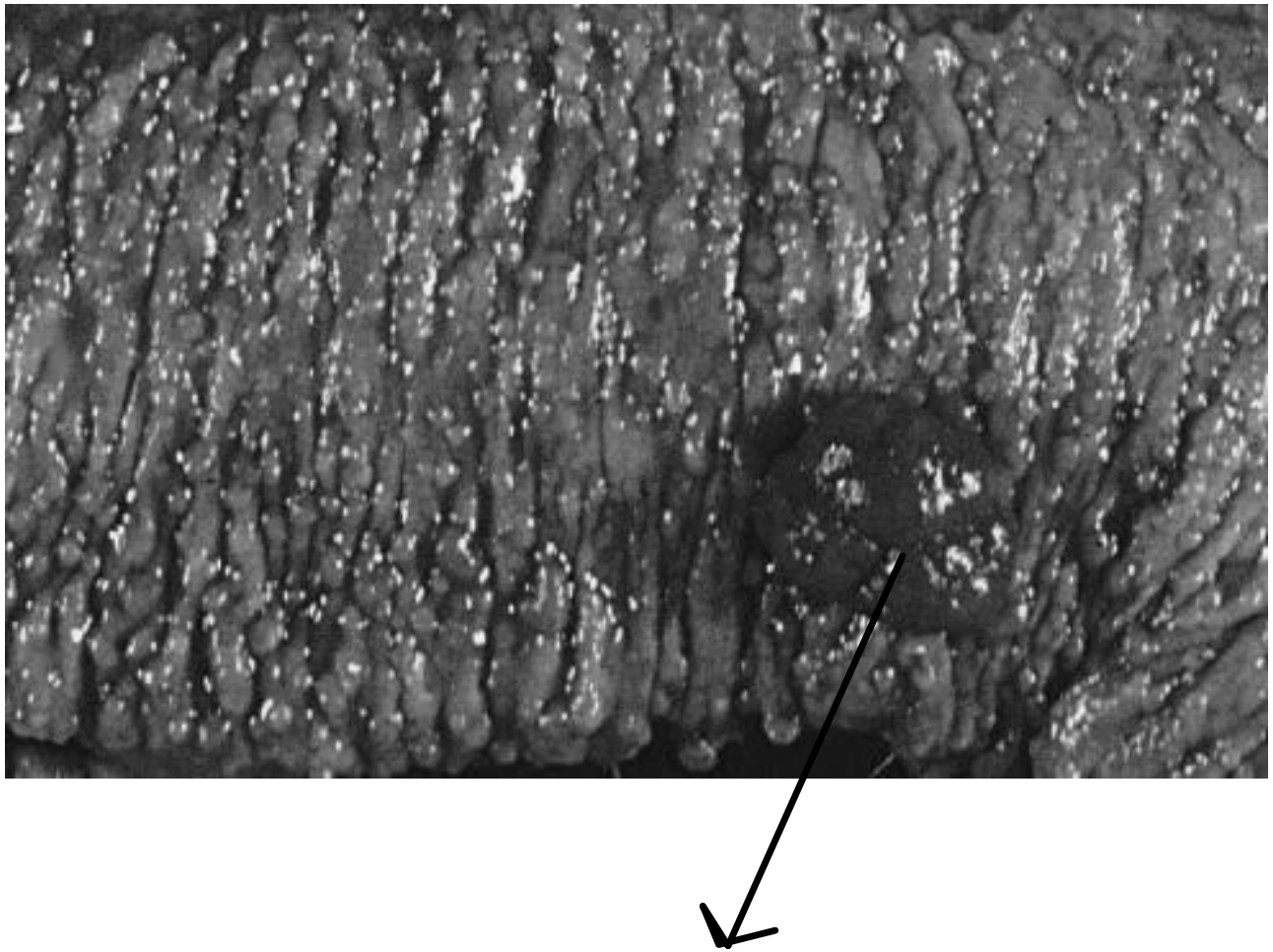




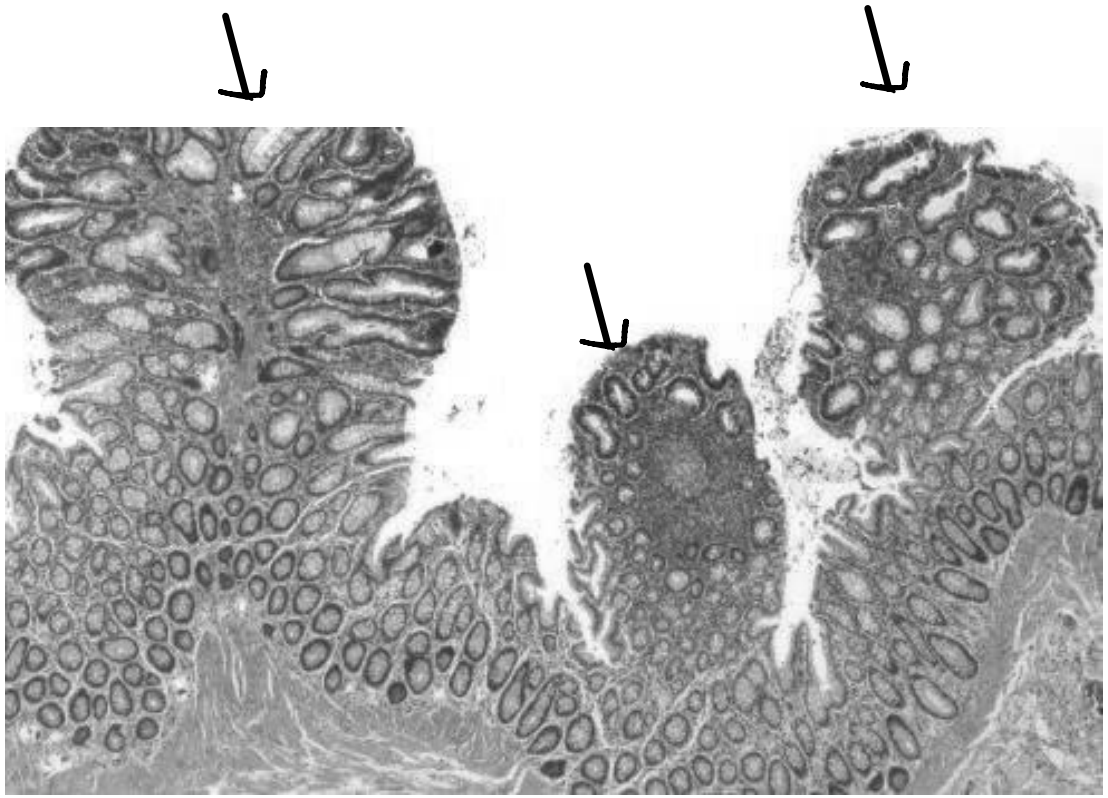
Familial Syndromes

- Syndromes associated with colonic polyps and increased rates of colon cancer
- Genetic basis.
- Familial Adenomatous Polyposis (FAP)
- Hereditary Nonpolyposis Colorectal Cancer (HNPCC)Familial adenomatous polyposis FAP
- Autosomal dominant.
- Numerous colorectal adenomas: teenage years.
- Mutation in APC gene.
- At least 100 polyps are necessary for a diagnosis of classic FAP.
- Morphologically similar to sporadic adenomas
- 100% of patients develop colorectal carcinoma, IF UNTREATED, often before age of 30.
- Standard therapy: prophylactic colectomy.
- Risk for extraintestinal manifestations
- Specific APC mutations. Variants of FAP:Gardner syndrome: intestinal polyps + osteomas (mandible, skull, and long bones); epidermal cysts; desmoid and thyroid tumors; and dental abnormalities.
- Turcot syndrome: intestinal adenomas and CNS tumors (medulloblastomas >> glioblastomas)

- Familial adenomatous polyposis



This mass maybe adenocarcinoma

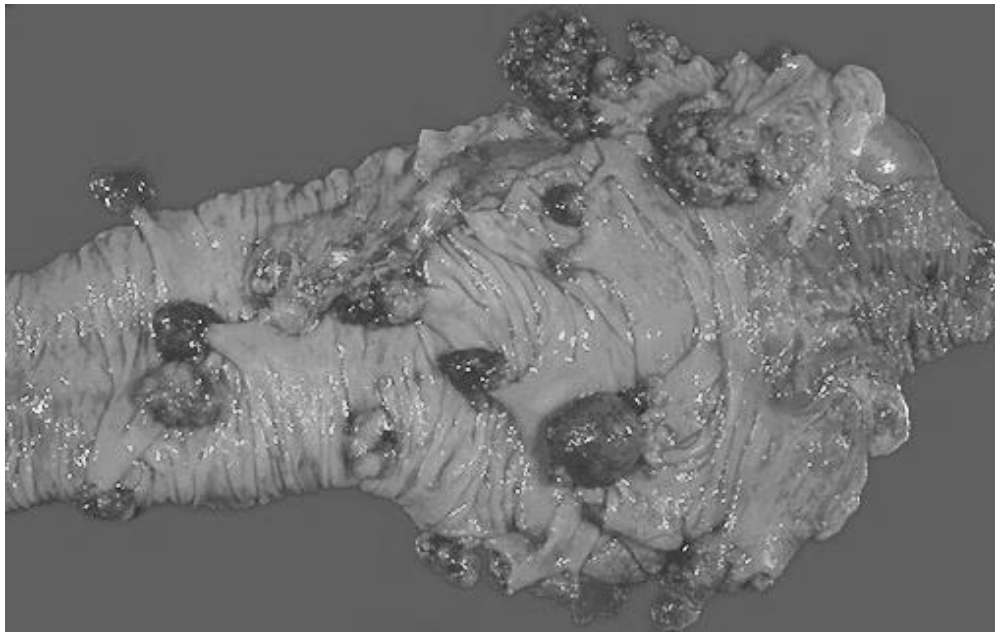


FAP

Three Tubular adenomas are present

Cecal polyps in HNPCC.

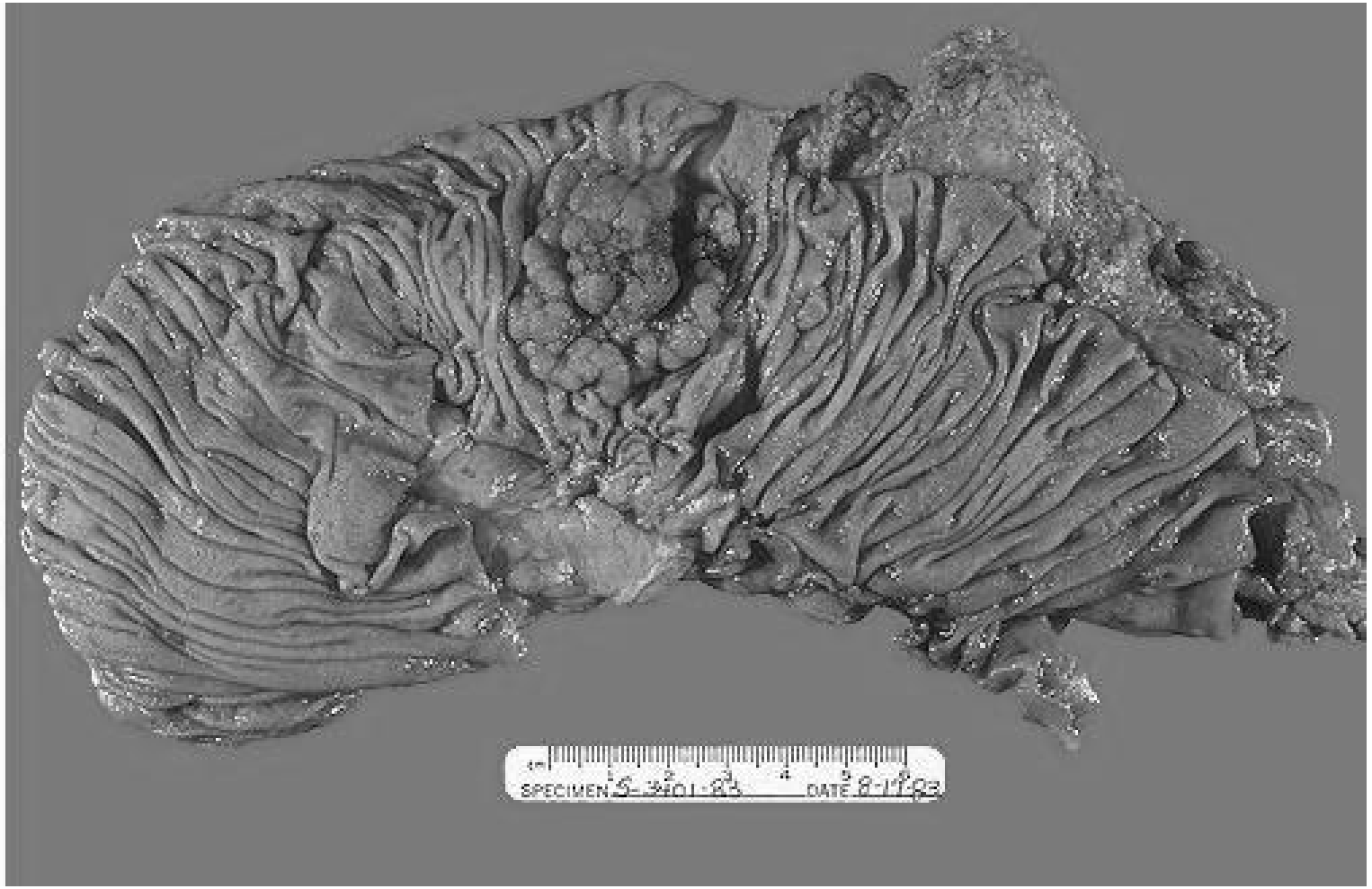
Hereditary Nonpolyposis Colorectal Cancer: HNPCC, Lynch syndrome

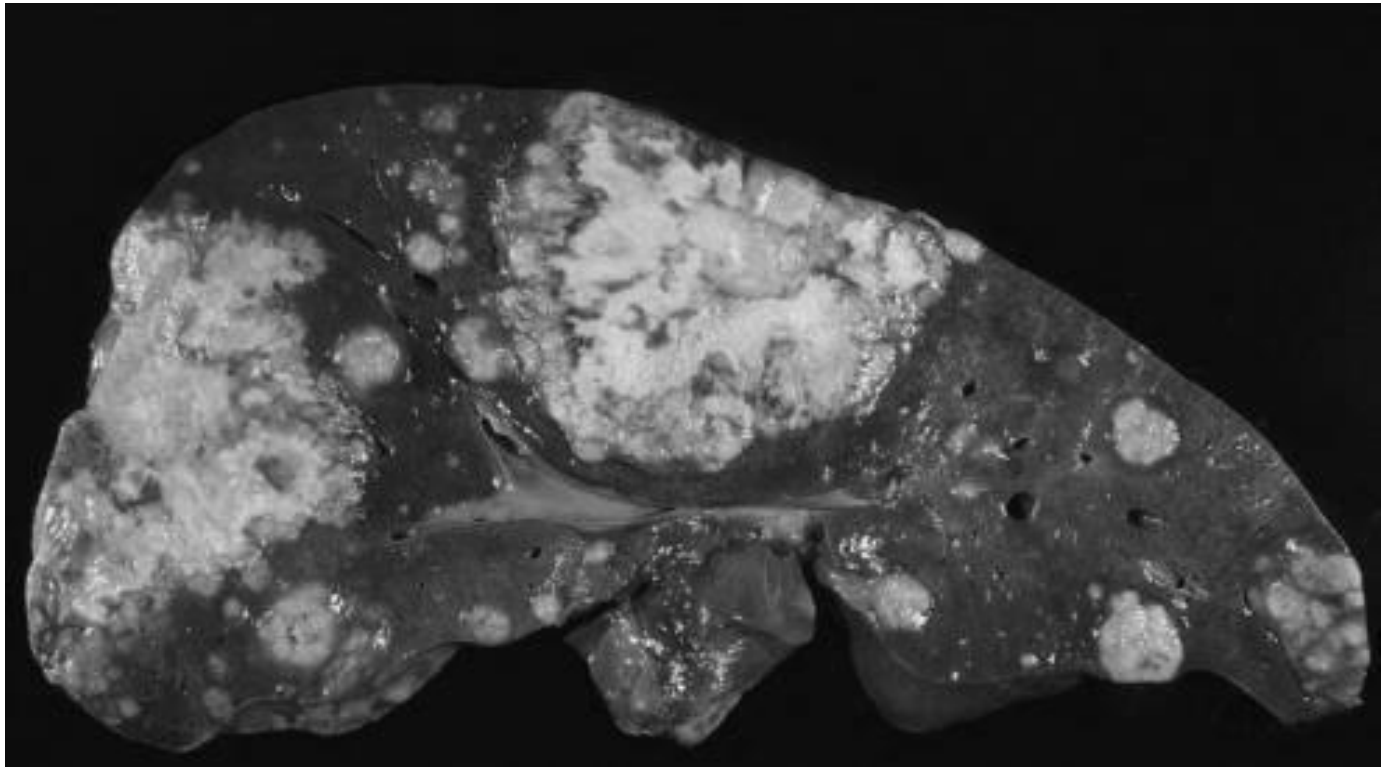


Hereditary Nonpolyposis Colorectal Cancer: HNPCC, Lynch syndrome

- Autosomal dominant. Inherited germ-line mutations in DNA mismatch repair genes (detection, resection and repair of errors in DNA replication).
- Increased risk of: Colorectum, endometrium, stomach, ovary, ureters, brain, small bowel, hepatobiliary tract, and skin cancers.
- Colon cancer at younger age than sporadic cancers
- Right colon, abundant mucin.
- Only few adenomatous precursors (typically sessile serrated adenomas).HNPCC, cont
- Accumulation of mutations at 1000x higher rates in microsatellite DNA (short repeating sequences)
- Resulting in microsatellite instability.
- 5 genes identified but Majority of cases involve either MSH2 or MLH1.

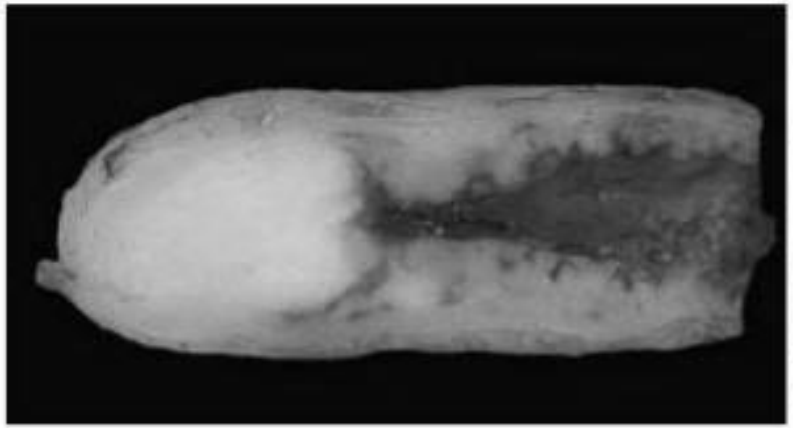
Exophytic adenocarcinoma, colon



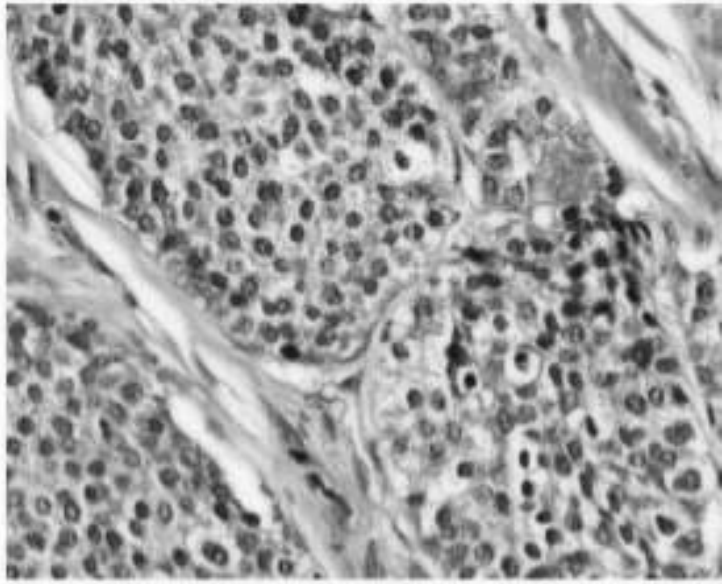


Liver metastasis from colon cancer.

Carcinoid tumor



Gross



Microscopic

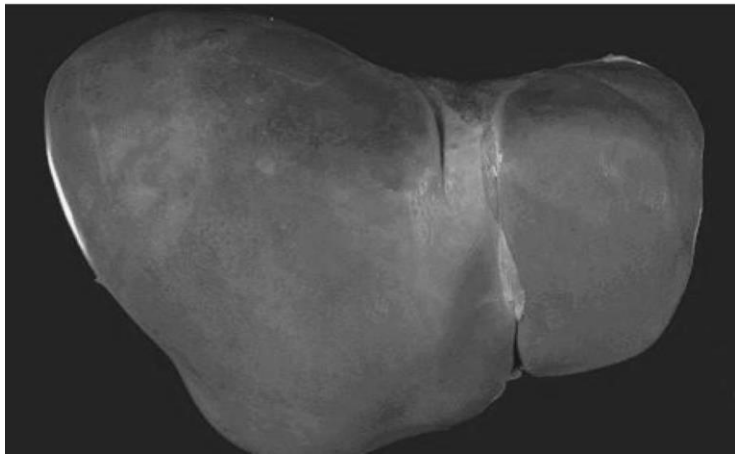
The most common tumor: carcinoid (neuroendocrine tumor)

- Incidentally found during surgery or on examination of a resected appendix
- Distal tip of the appendix
- Nodal metastases & distant spread are rare.

Appendix

انتهت جزئية الميد

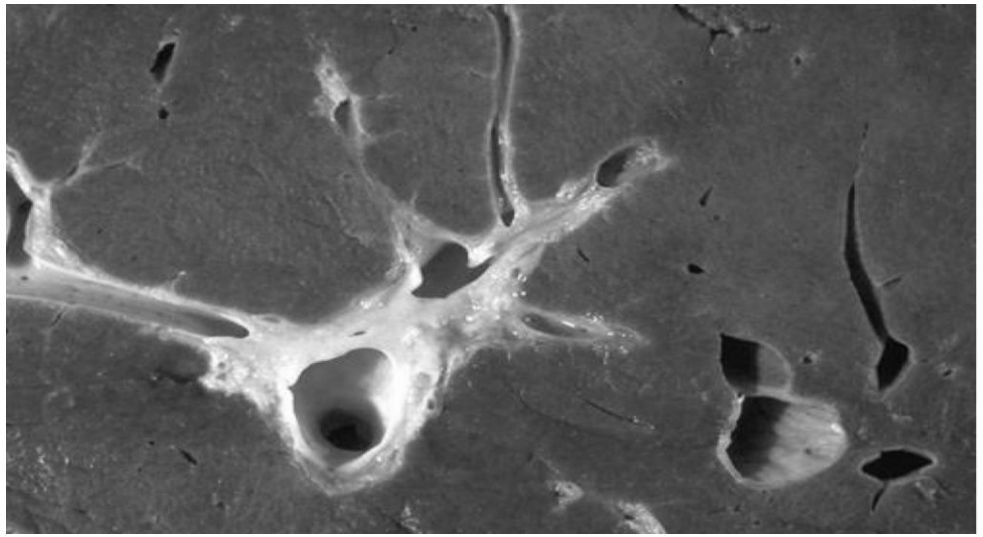
Final material



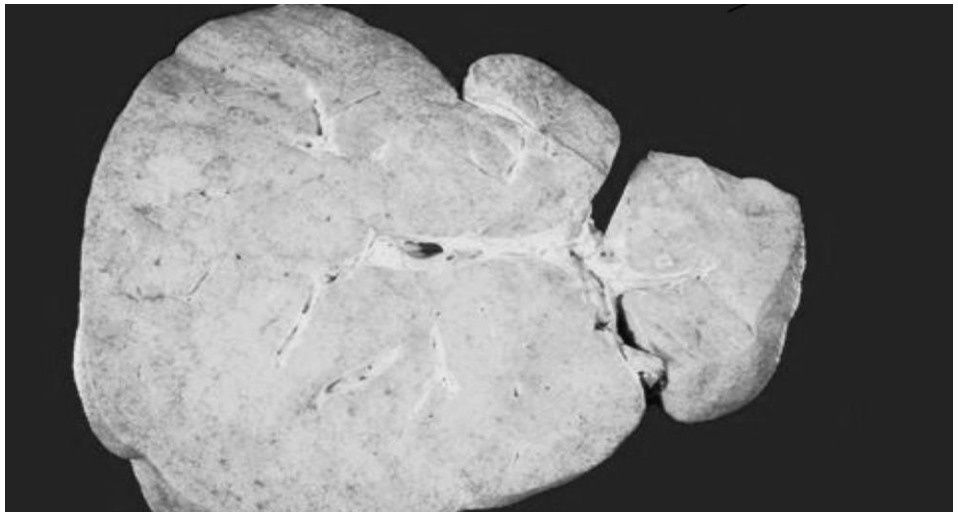
A normal liver: look at its brownish color smooth surface with no nodules or fatty changes.

What Does the Liver Look Like? A healthy liver is dark reddish-brown in color, and is shaped like a wedge.

↳ Extra information, not mentioned in slides



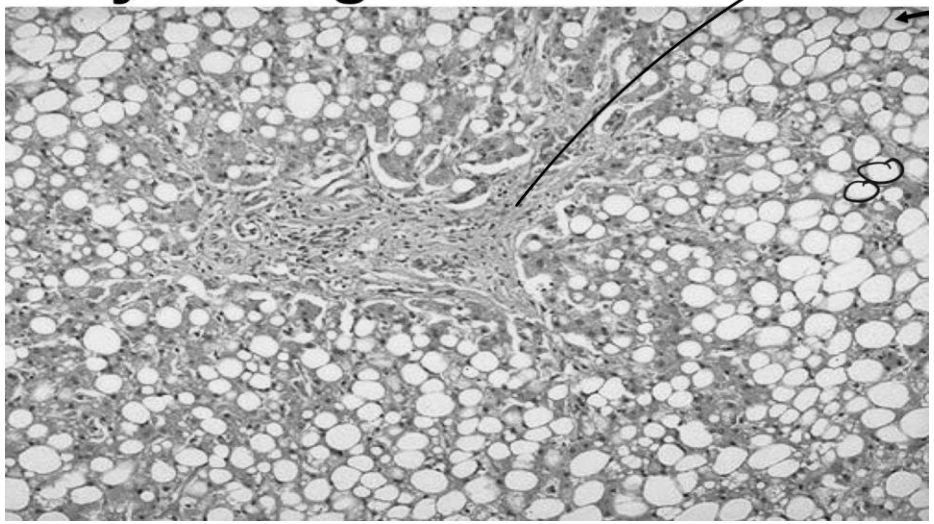
This is a cross section through the liver, notice the homogenous brown color.



accumulation of triglycerides (TG) as fat droplets within the cytoplasm of hepatocytes, yellow fatty areas.

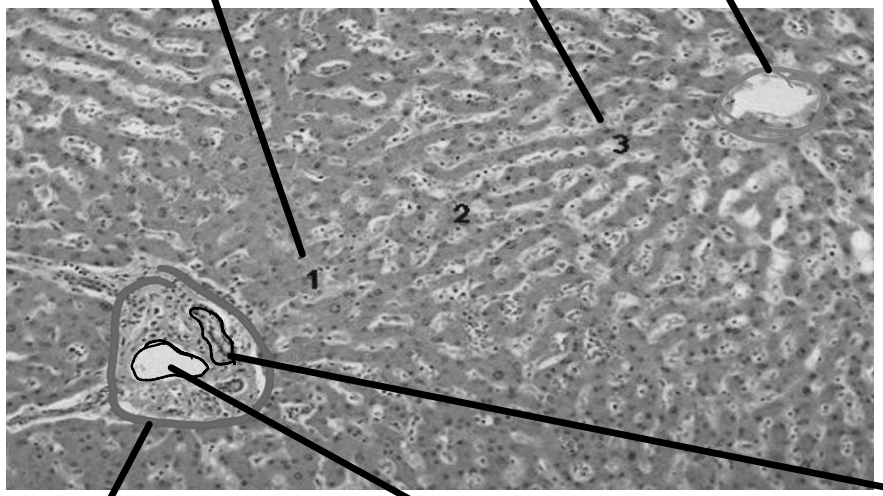
Fatty, greasy, yellow not normal appearance of the liver

fatty change

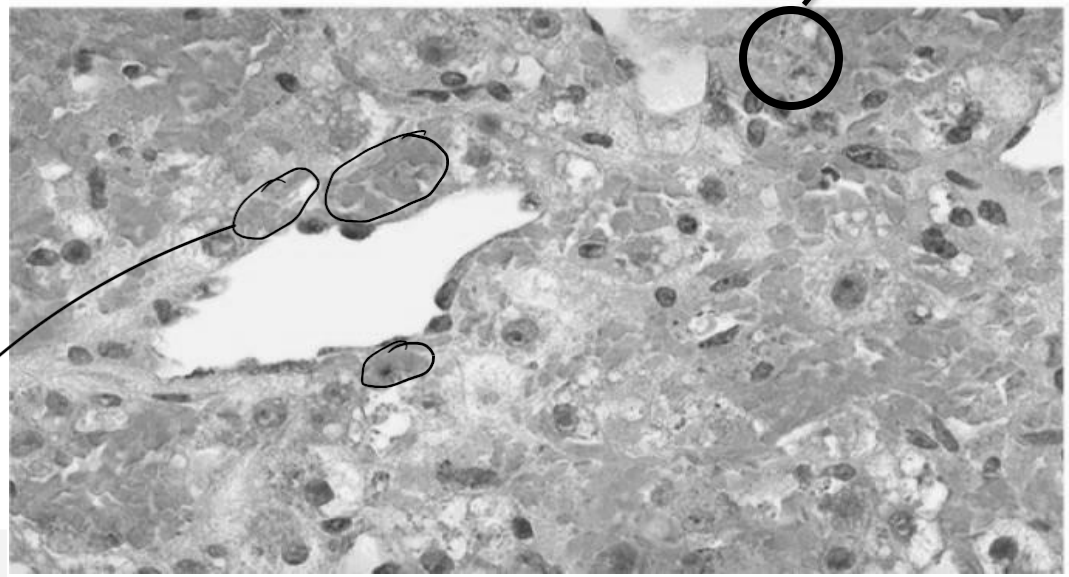


Macrovesicular , severe degree of fatty change

The center , This is the portal area, with a background of fibrous tissue.


Periportal area
Pericentral area
Central vein
1
2
3
bile duct
portal vein branch
Portal area

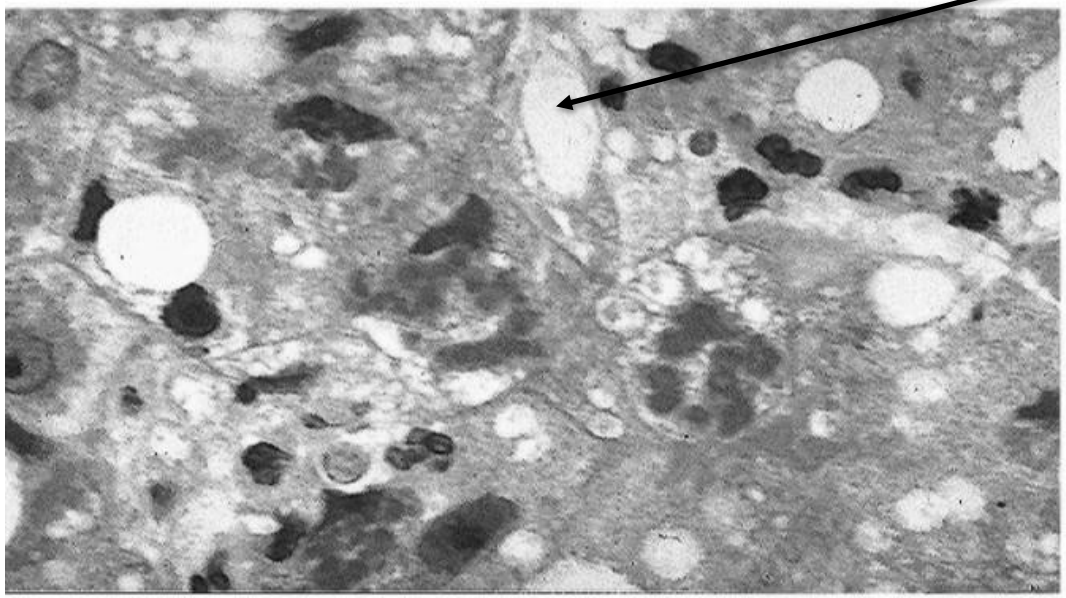
Necrosis of liver



This pigment could be bile, indicating intracellular cholestasis
Or iron: we can differentiate between the two by using special stains

Necrotic hepatocytes, no nuclei around central vein

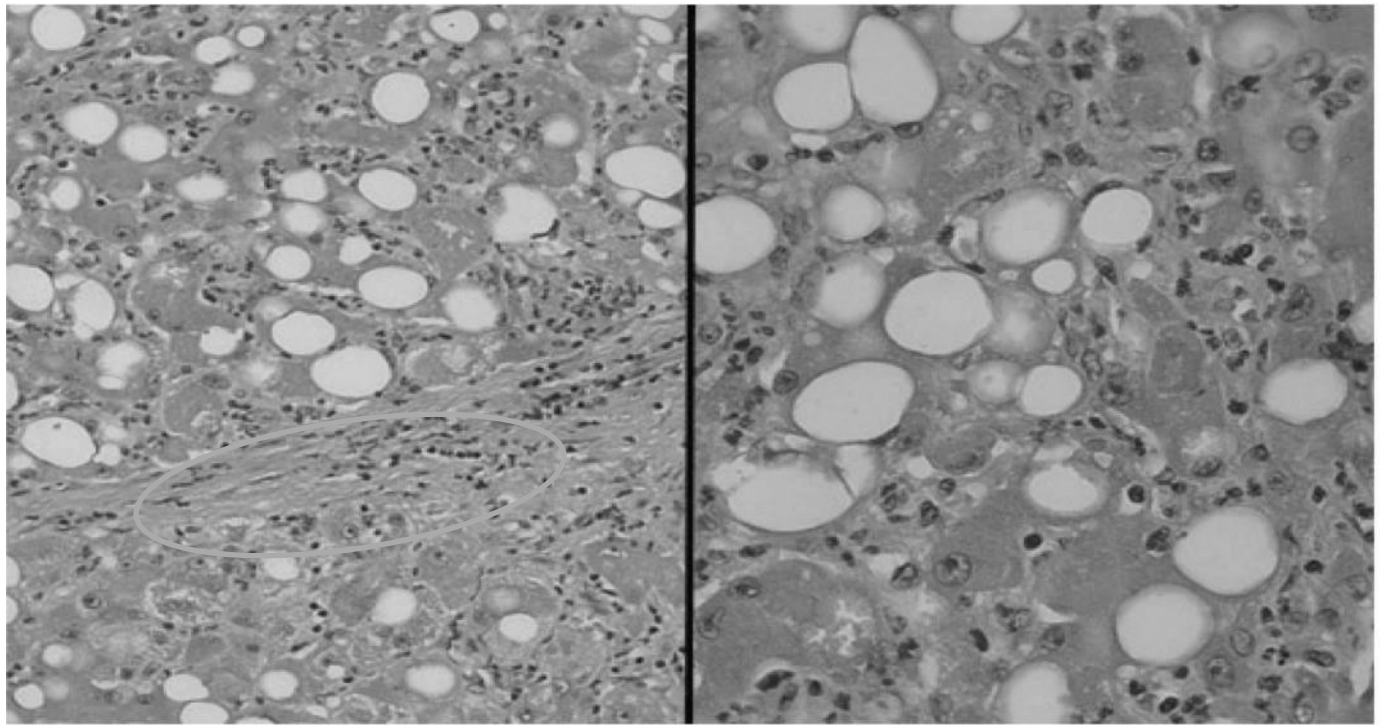
Mallory-hayline bodies



Fat droplet

Relatively large, deeply eosinophilic (characteristic of Mallory hyaline bodies)

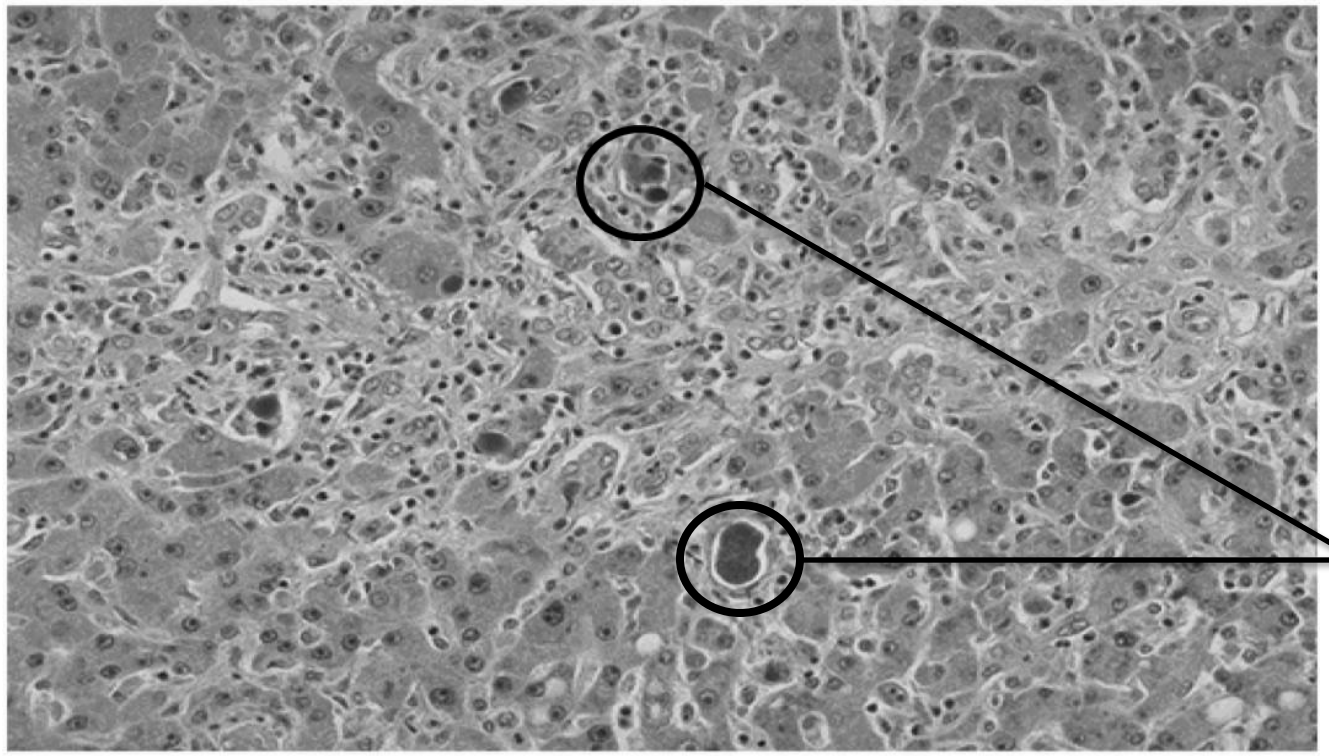
Alcoholic hepatitis



* Black dots indicate inflammatory cells

* We can see bridging fibrosis which is not normal in the liver

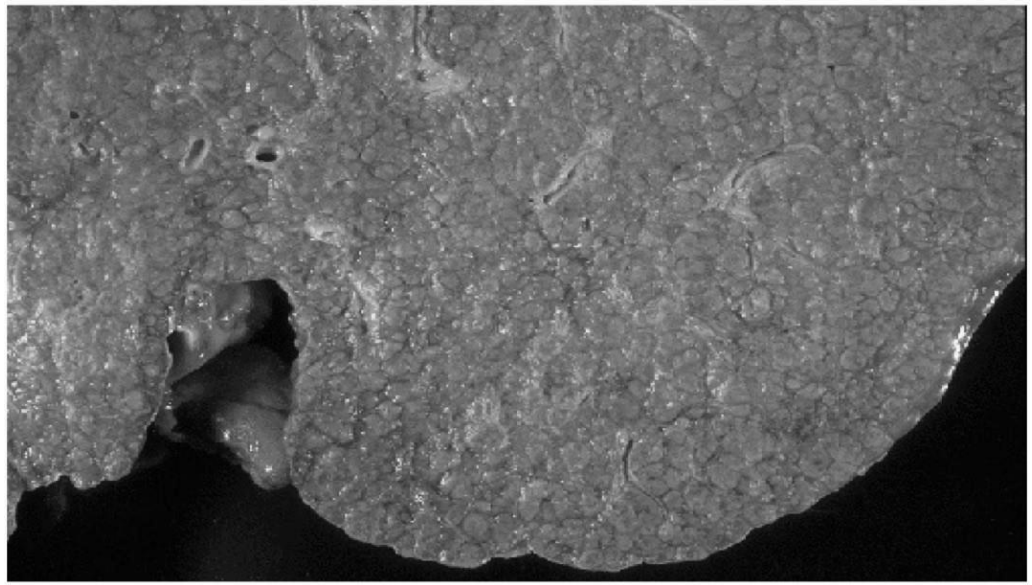
Cholestasis



Cholestasis starts intracellularly, when it affects the biliary system it's considered severe

Bile stagnant within the biliary system, indicating severe cholestasis

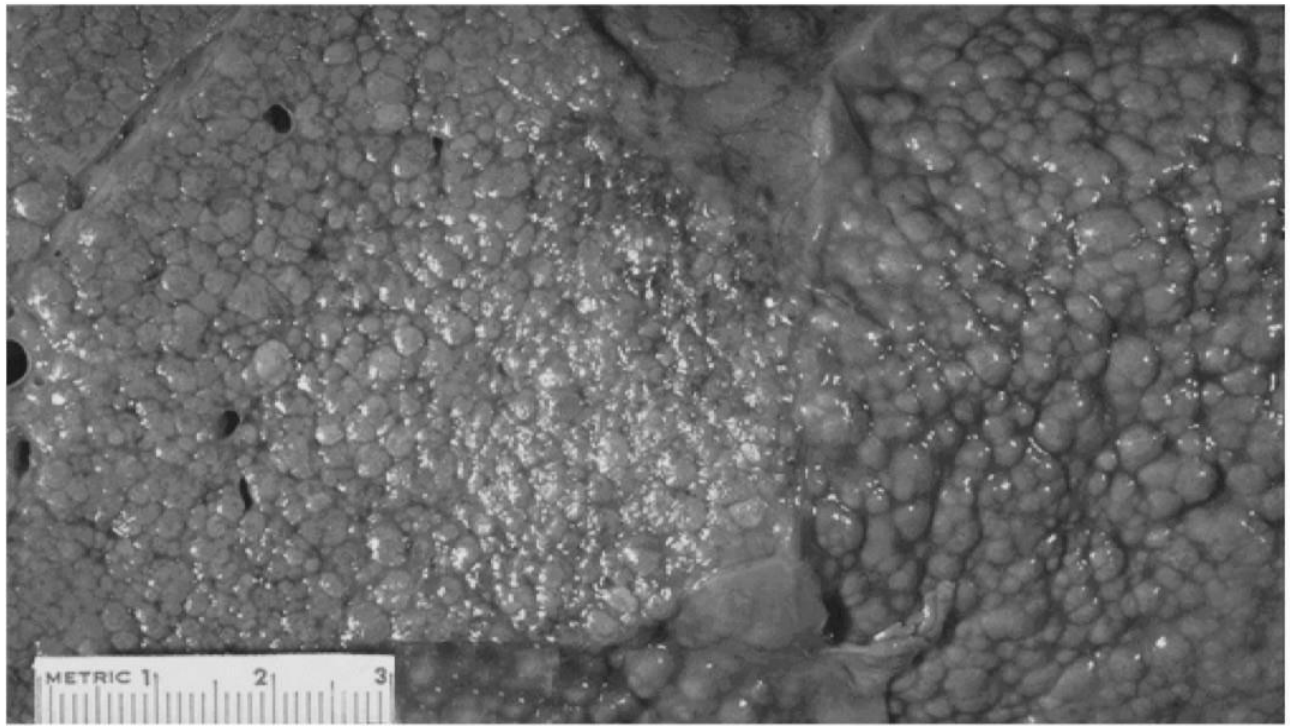
Liver cirrhosis



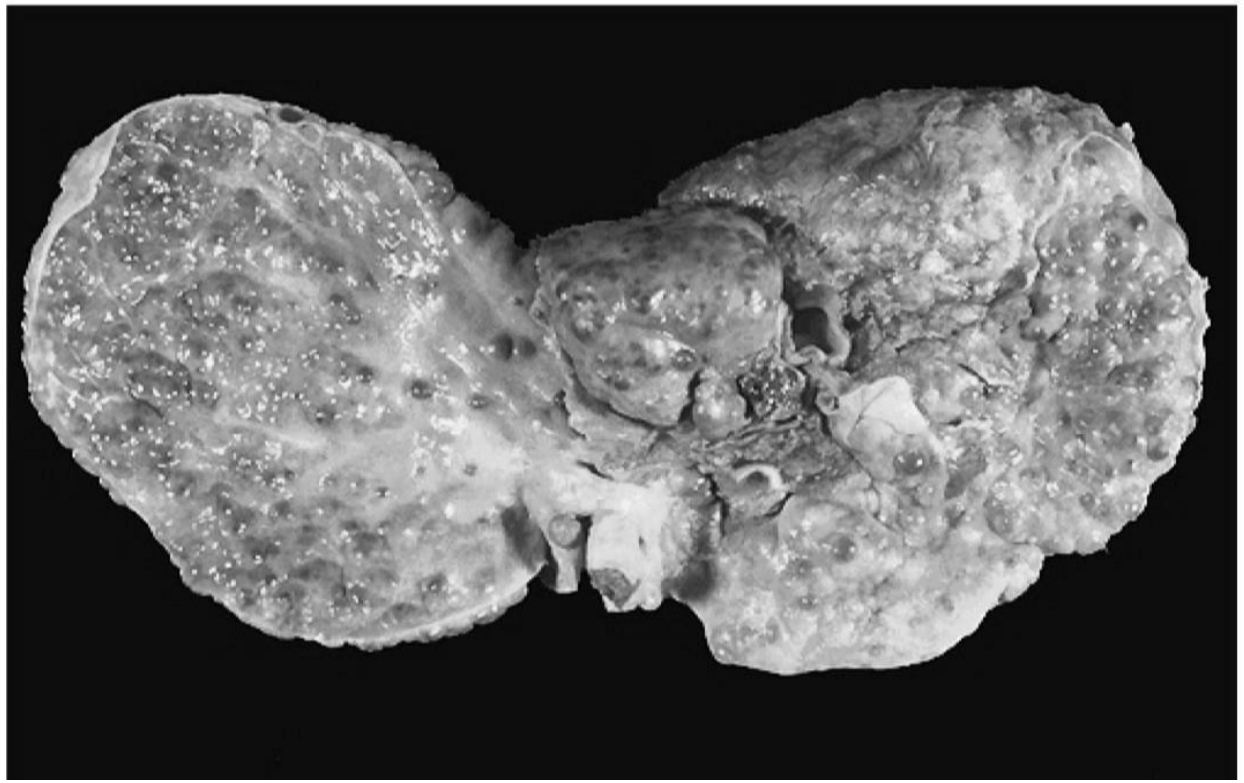
Micronodular cirrhosis following chronic alcoholism, fibrotic liver is seen (nodules < 3 mm)

* Remember that cirrhosis is a diffuse process, so the whole liver is included.

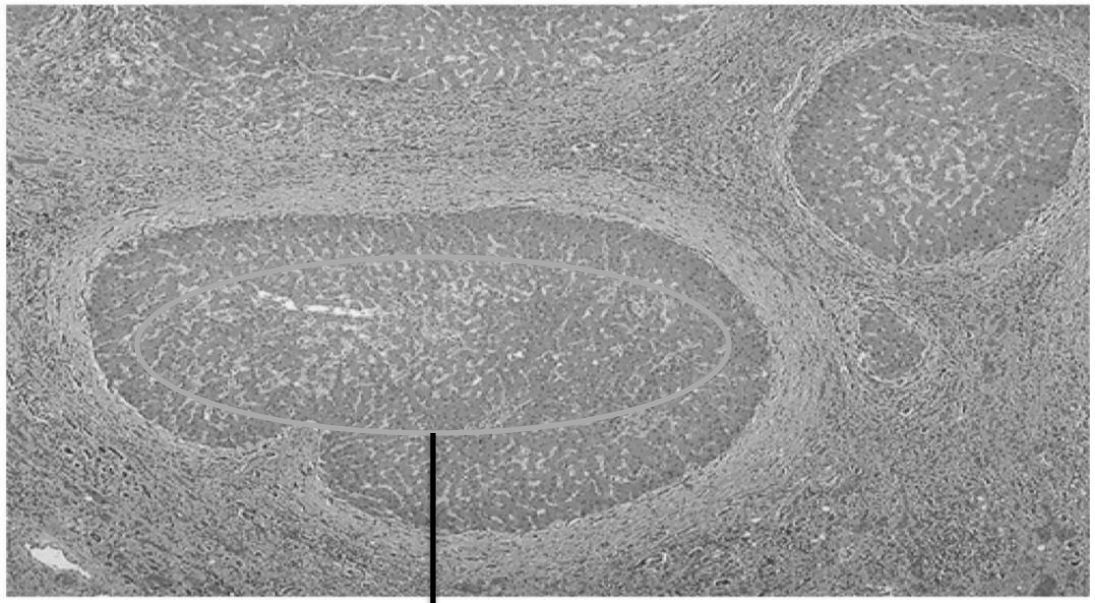
Micronodular cirrhosis



Macronodular cirrhosis

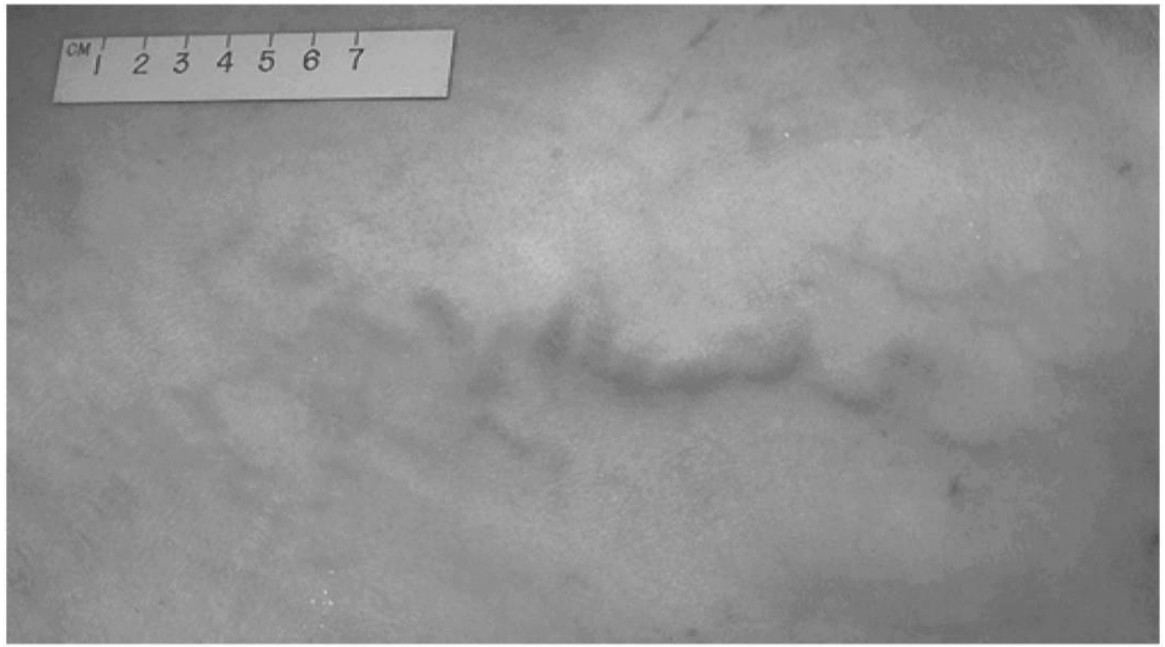


liver is distorted, this is caused by fibrosis, it destroys the whole architecture causing disfiguration.



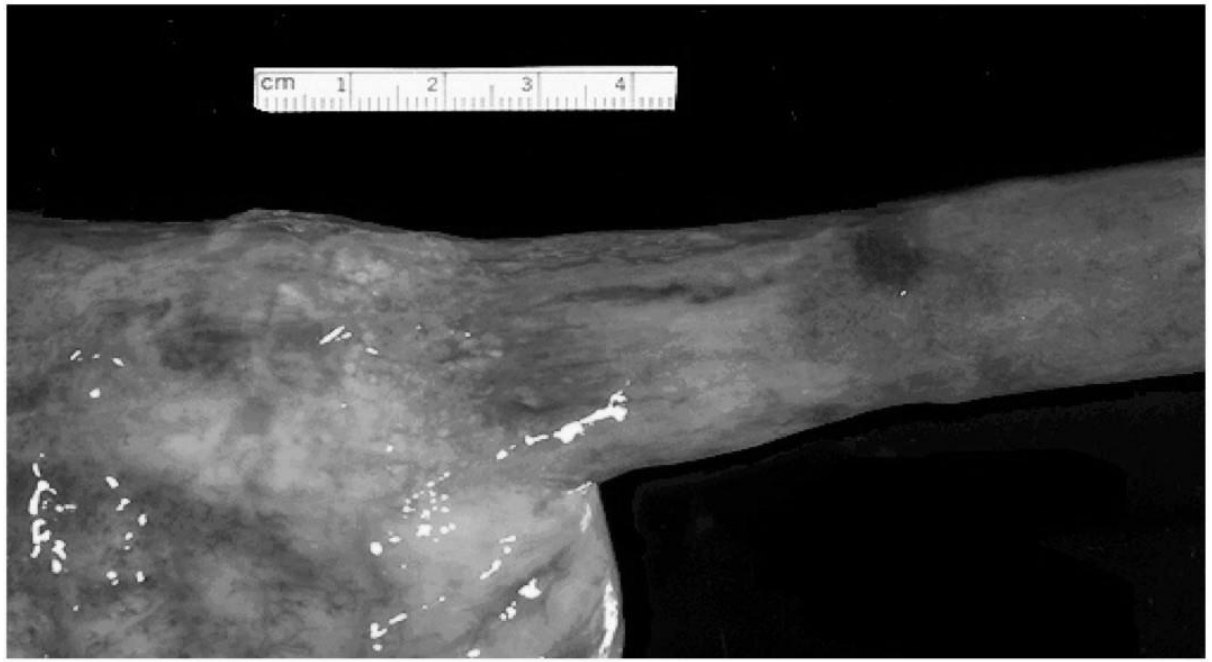
Hepatocytes are surrounded by fibrosis (the pale band or area)

Caput medusae-abdominal skin



Dilated blood vessels radiate from the umbilicus giving a Medusa head appearance.

Esophageal varicies

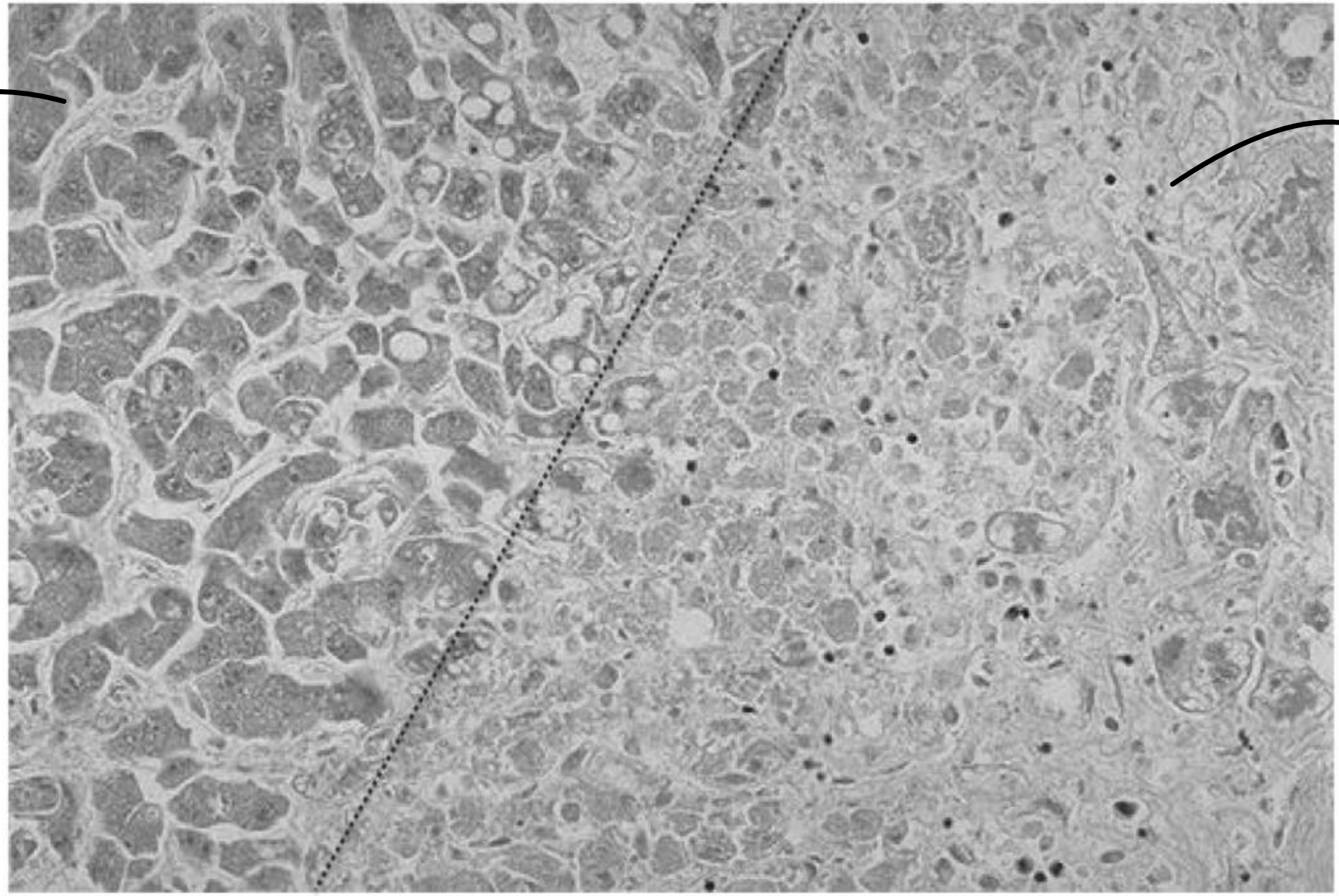


very serious condition because

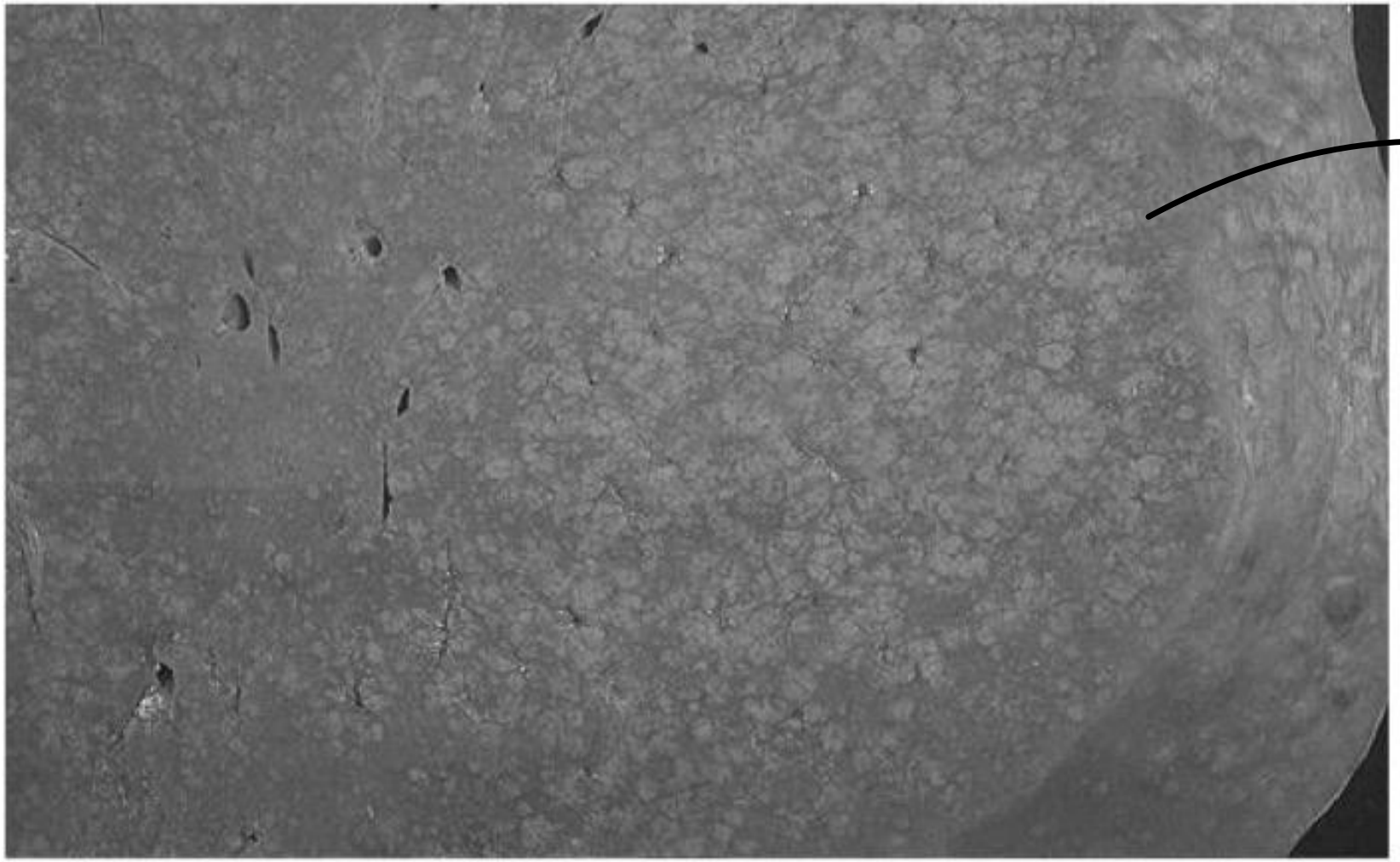
➢ Upper GI bleeding can lead to death in the first attack, and if treated recurrence is common.

➢ It can cause liver failure.

the left side presents preserved Hepatocytes without necrosis

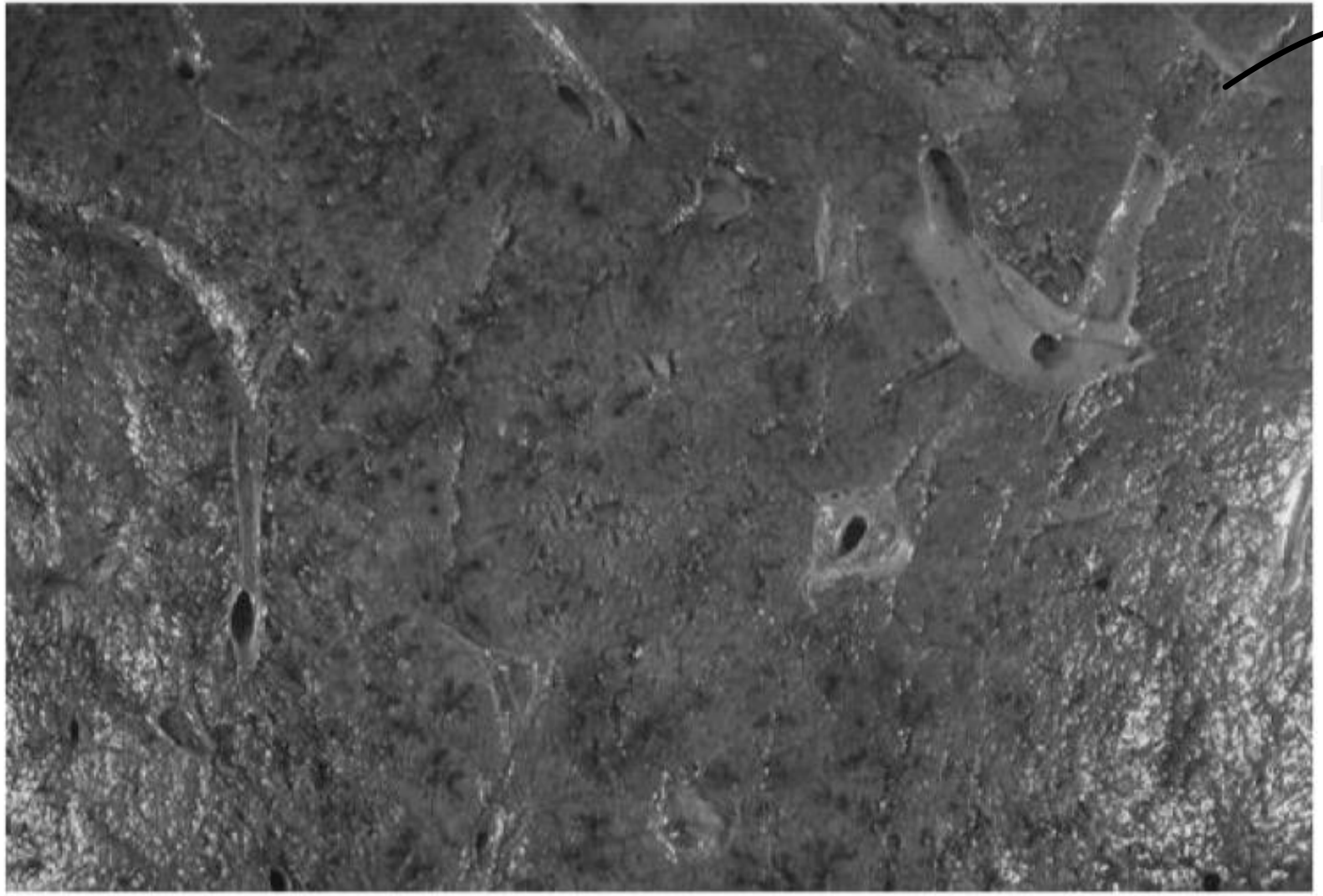


Right Side: shows necrosis,Pale, nuclei loss



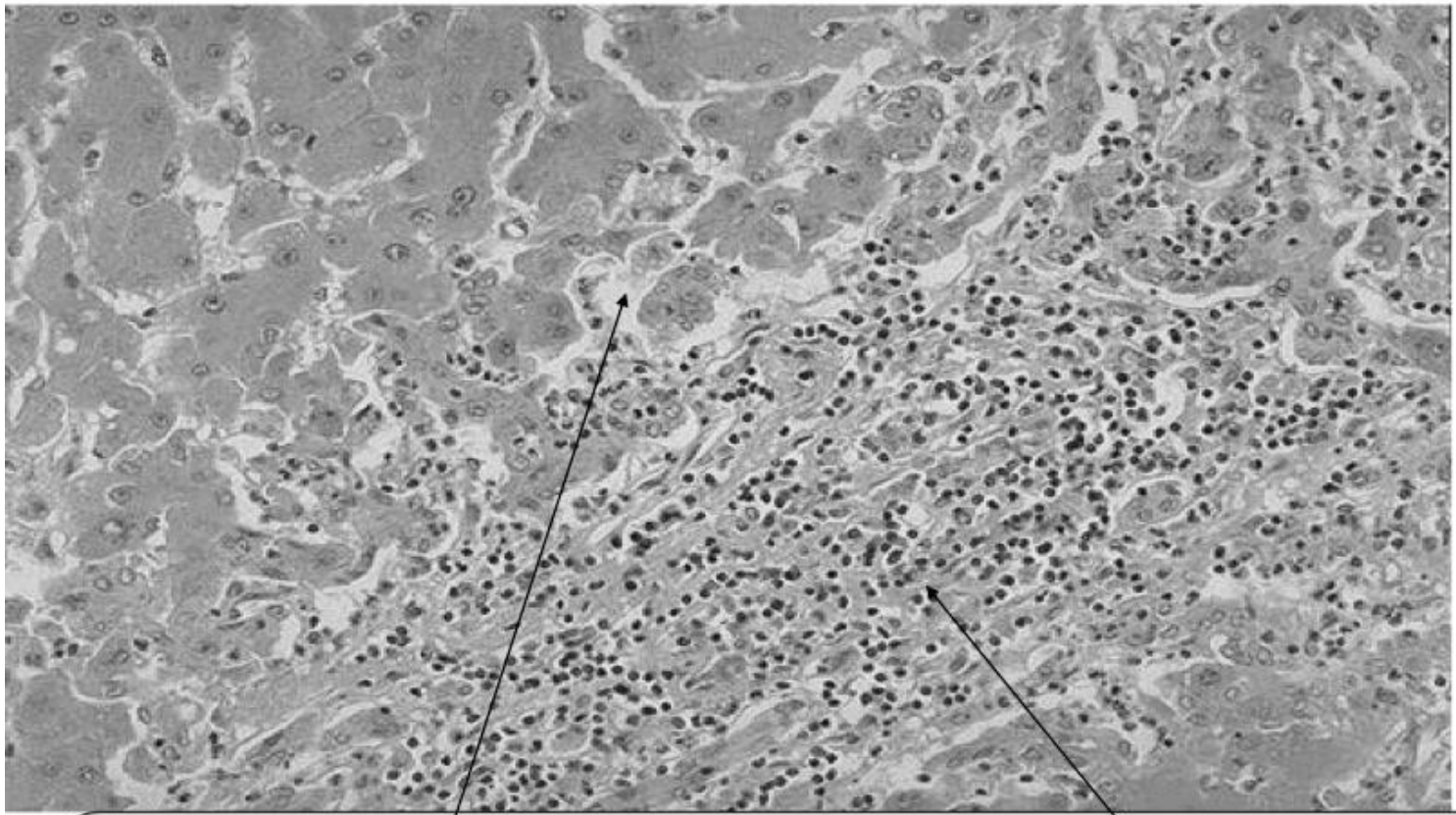
Fulminant hepatitis

this is the appearance of the liver with necrosis, the pale areas are the necrotic areas . the degree of necrosis is variable; there are also loss of homogenicity , all these are indications of necrosis



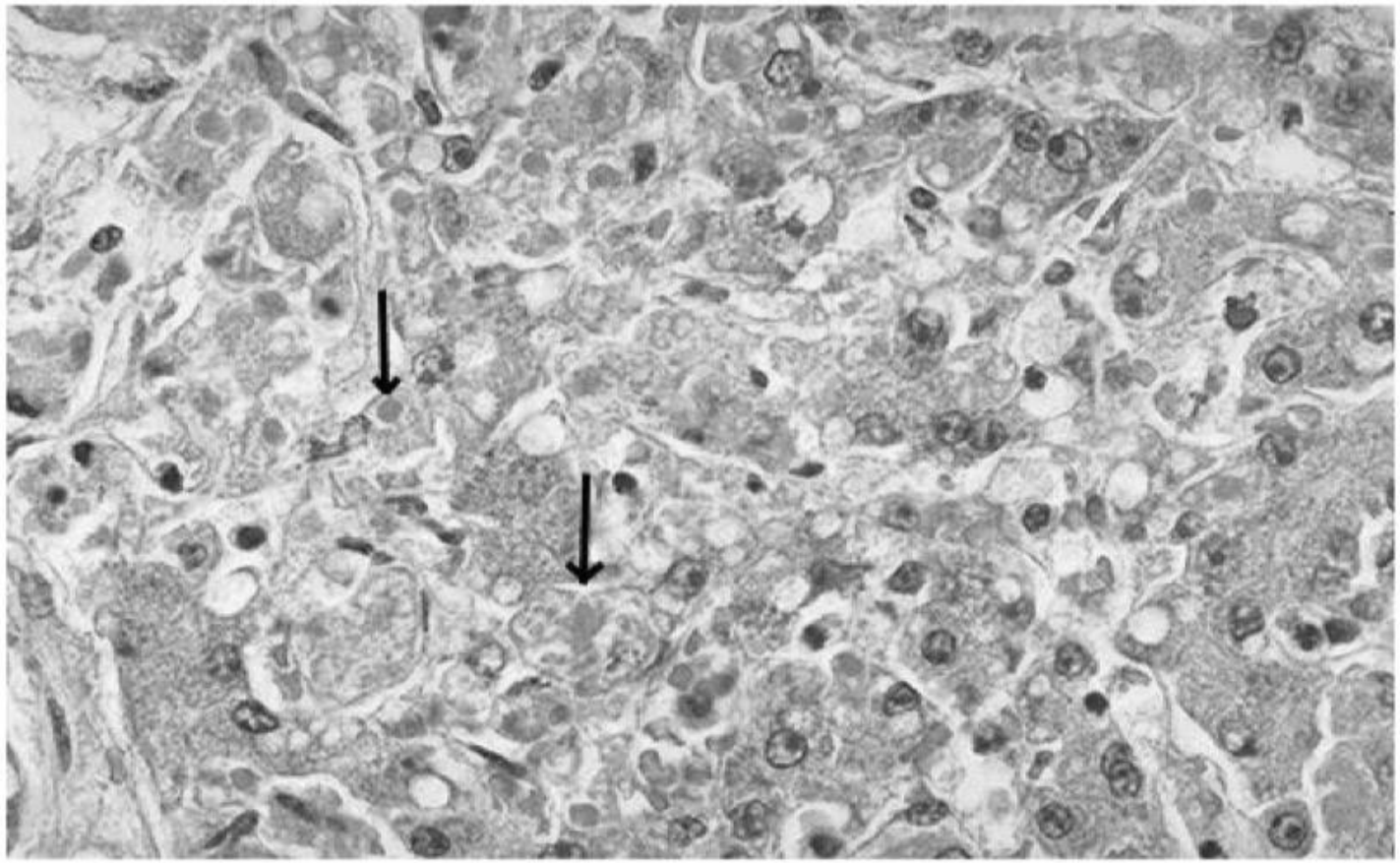
Morphology of chronic hepatitis

There is loss of homogeneity as seen by color changes, pale areas and surrounding is darker. This due to presence of fibrosis. There are some nodule formation

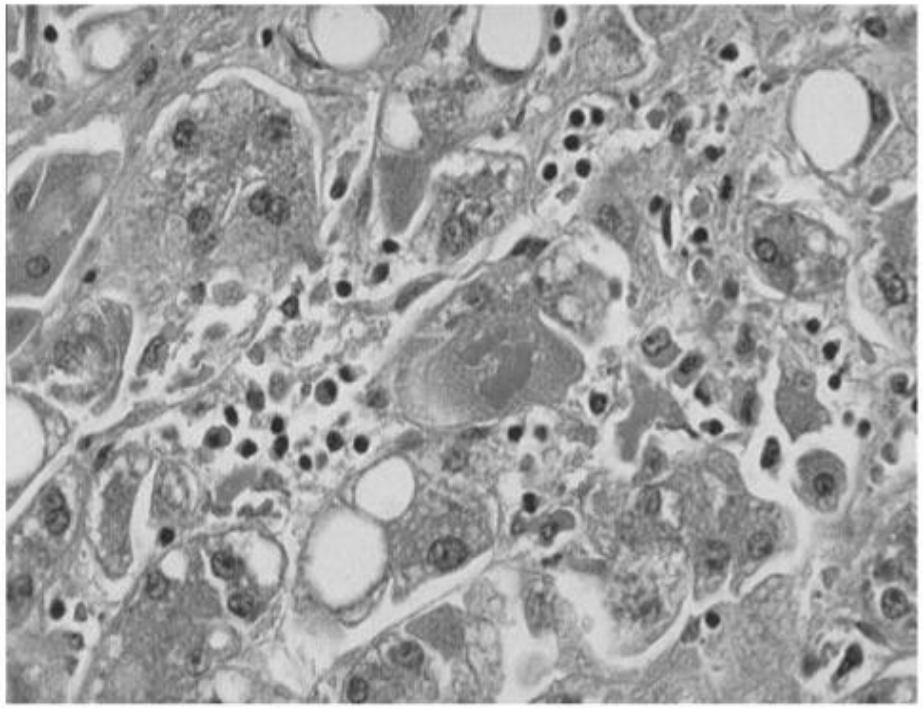


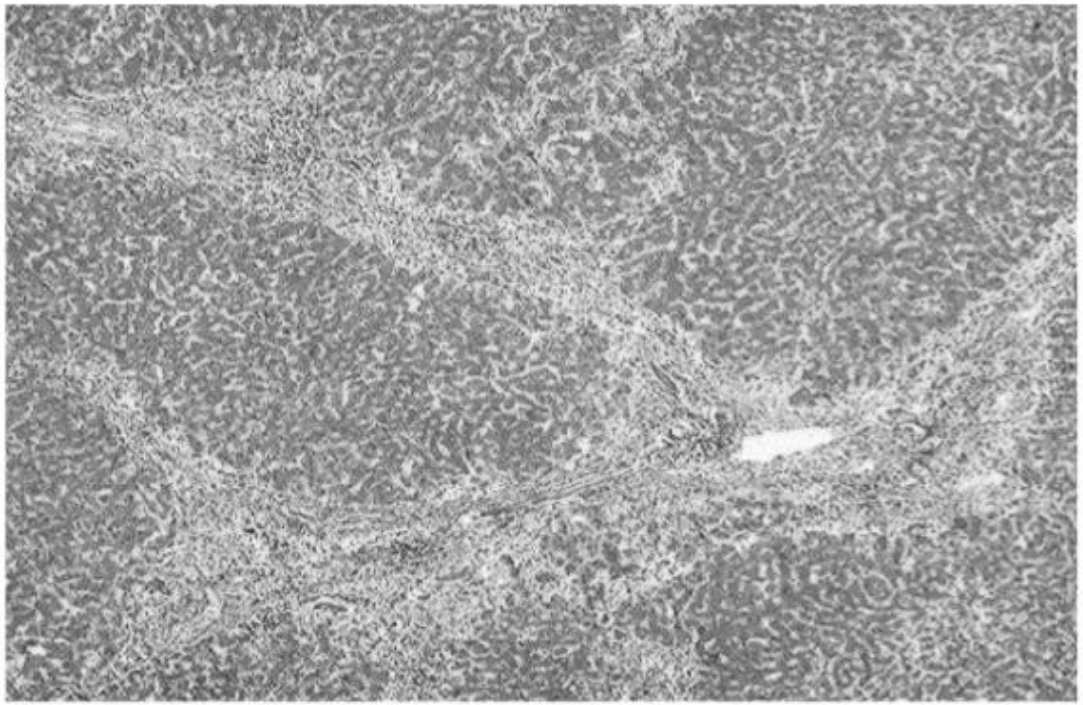
It is microscopic appearance of severe form of chronic hepatitis: there are some bridging fibrosis – follow the arrow -
(indication of cirrhosis development) and extensive lymphocytes--> النقاط السوداء

Necrosis of hepatocytes-councilman bodies (arrows)
Pic shows fibrosis and chronic hepatitis



External: hepatitis B can result in a fulminant hepatitis with extensive necrosis. A large pink cell undergoing "ballooning degeneration" is seen below the right arrow. At a later stage, a dying hepatocyte is seen shrinking down to form an eosinophilic "councilman body" below the arrow on the left.



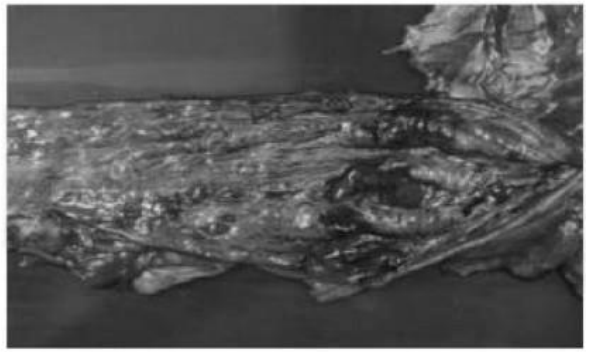


we can see loss of hepatocytes architecture and the collapse of the liver parenchyma with viral hepatitis + fibrous tissue on it

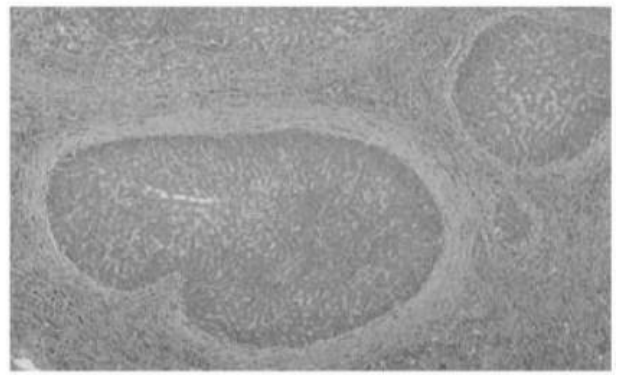
Past Paper Questions *from JU medicine*

1- This is a section from the esophagus from a 60-year-old patient with liver cirrhosis who developed massive hematemesis, what is the most likely cause of this bleeding based on the picture.

A. Esophagitis
B. Gastric ulcer
C. Gastric cancer
D. Esophageal cancer
E. Esophageal varices



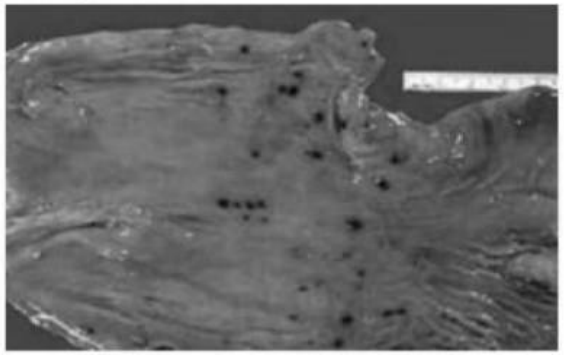
2- This represents a microscopic appearance of a condition that can result of all of the following EXCEPT one:



A. Wilson disease
B. Viral hepatitis
C. Hemochromatosis
D. Biliary diseases
E. Reye syndrome

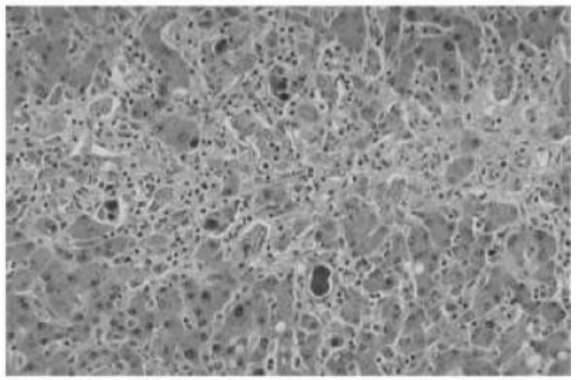
3- A 50-year-old man in the intensive care unit (ICU) after a major surgery, and suddenly developed hematemesis, based upon the picture given above from the stomach, the most likely diagnosis is:

A. Gastric carcinoma
B. Autoimmune gastritis
C. Viral gastritis
D. Stress ulcers
E. Chronic H pylori gastritis



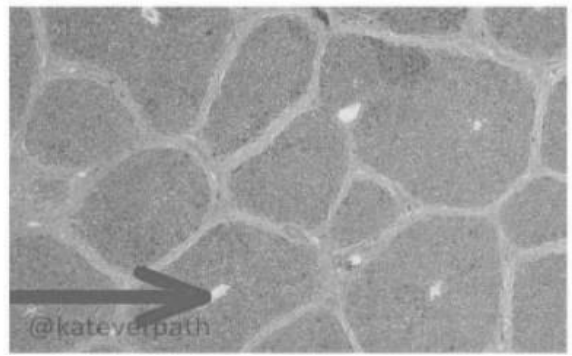
4- The intra canalicular and intracellular accumulation of this brown pigment in Liver represents:

A. Hemochromatosis
B. Steatosis
C. Wilson disease
D. Cholestasis
E. Drug toxicity



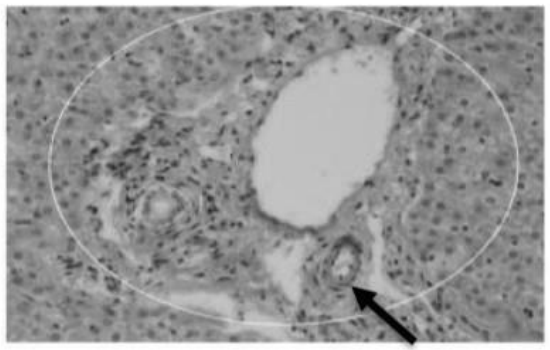
5- Identify the pointed structure in this section:

A. Branch from portal vein
B. Bile duct
C. Central vein
D. Branch from hepatic artery
E. Blood sinusoids



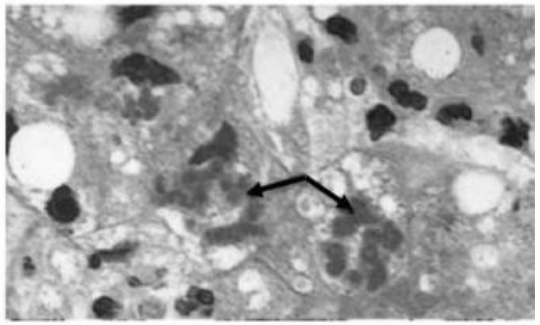
6- The pointed structure represents:

A. Portal triad
B. Portal vein
C. Bile duct
D. Hepatic artery



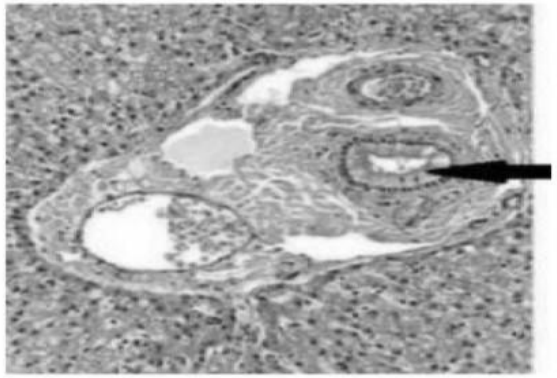
7- the following deposited things are:

A. Fat
B. Cytoskeleton
C. Iron
D. Copper



8- Identify the pointed structure:

A. Portal Vein
B. Hepatic Artery
C. Porta hepatis
D. Blood Sinusoids
E. Bile Duct



Your evaluation of the previous file, I hope you to answer these questions, for developmental purposes 🙏🖤

https://forms.gle/crevD5uLq8cWY8TJA

Click here

Good luck in the exam