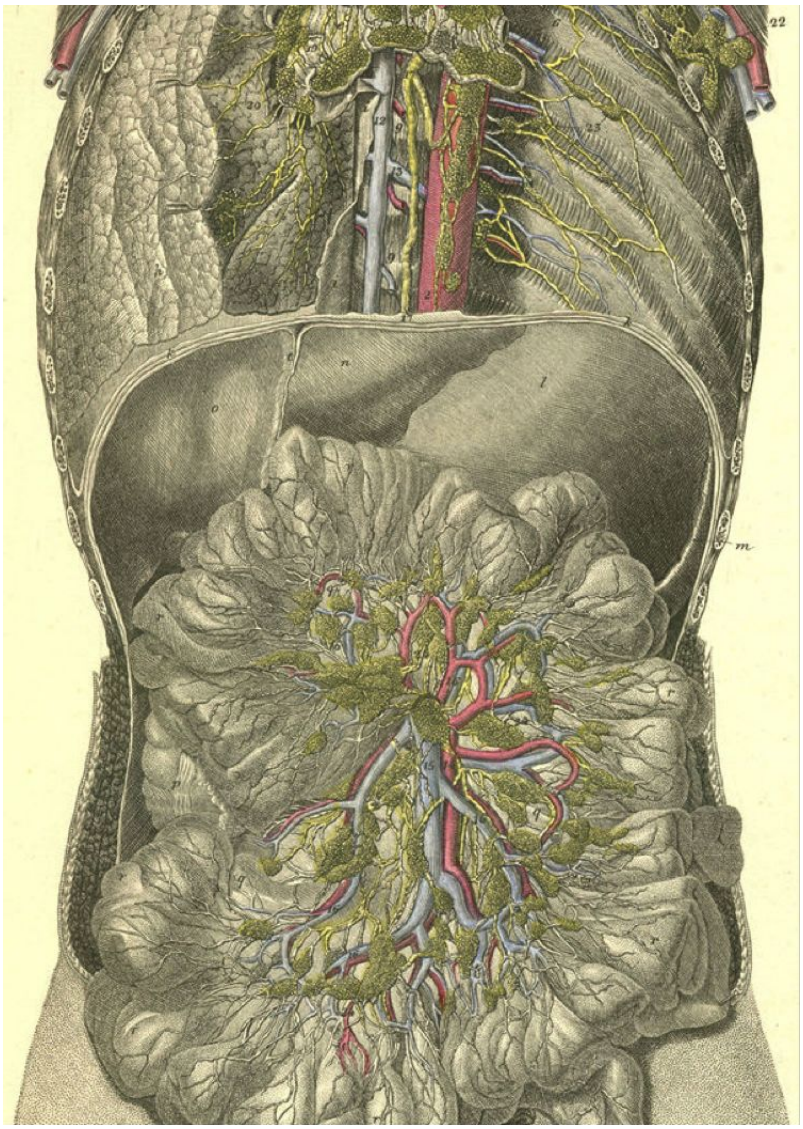
GIT BLock midterm REVISION



Important note: **Please check out this link before viewing the file to know if there are any additions or changes. The same link will be used for all of our work: Pathology Edit.**

1- GastroEsophageal Reflux Disease (GERD)

Difference between Physiologic and Pathologic GER:

Physiologic GER	Pathologic GERD
Asymptomatic, Postprandial (after meal). Short lived (for a period of time). No nocturnal symptoms.	Symptoms, Mucosal injury Nocturnal symptoms (problems while sleeping at night)

GERD: Occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury.

This is caused by two mechanisms:

1. Decrease in LES tone.
2. Increase abdominal pressure

Risk factors: alcohol , tobacco, obesity, pregnancy, medications, caffeine and hiatal hernia.

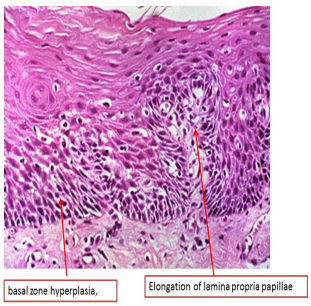
Clinical Manifestations: Heartburn, Regurgitation ,Dysphagia.

Diagnostic Evaluation:

1- Esophagogastroduodenoscopy[1]: Allows the detection, and management of esophageal injury or complications of GERD. * absence of endoscopic features does not exclude a GERD diagnosis.

2- pH: 24-hour pH monitoring.

Complications:

Erosive esophagitis	Esophageal stricture	Barrett's Esophagus (main risk factors)
– Responsible for 40-60% of GERD symptoms  **Microscopic shows:** 1. **elongation of lamina propria papilia.** 2. **basal zone hyperplasia.** 3. **eosinophils and neutrophils.**	Result of healing of erosive esophagitis. Caused by inflammation → fibrosis → esophagus becomes narrow → increase risk of choking with food.	Acid damages lining of esophagus → metaplasia of the lower esophageal mucosa **from stratified squamous epithelium to nonciliated columnar epithelium with goblet cells** (intestinal metaplasia). - may progress to dysplasia and adenocarcinoma **Endoscopic surveillance** is recommended for all patients with Barrett's esophagus.

[1] Endoscopic examination of the upper alimentary tract using a video instrument.

2- Peptic Ulcer Disease

It is a breach in the lining of the stomach or the duodenum .

Clinical Features:

- Epigastric pain (most common symptom)
- Gnawing or burning sensation.
- Some present with complications such as IDA , frank hemorrhage or perforation.

Acute Peptic Ulcer (In stomach)	Chronic Peptic ulcer	
	Gastric Ulcer (In stomach)	**Duodenal Ulcers (In duodenum)**
-As a part of an acute gastritis: Acute response to an irritant 'chemical' injury by drugs(NSAIDs) or alcohol. **-As a result of extreme hyperacidity** Zollinger-Ellison syndrome **-As a complication of a severe stress response:** Severe burns (Curling's ulcer) Major trauma (Stress ulcer) Cerebrovascular accident (Cushing ulcer)	**Breakdown of mucosal defence** by: - **H. pylori infection.** - NSAIDs. - Duodenogastric reflux (bile reflux). Pain **worsens with meals.**	**Increased production of Acid** by: - **H.pylori infection.** - Zollinger-Ellison syndrome. Pain **relives with meals.**

Important notes:

- Epithelium produce prostaglandins as defensive mechanism, NSAIDs inhibit their synthesis.
- Not all individuals with H.pylori infection develop peptic ulcer.
- Malignant transformation doesn't occur in duodenal ulcers and extremely rare in gastric ulcers. (most rare complication)
- chronic peptic ulcer can develop in esophagus as a result of GERD.
- H.pylori doesn't cause acute ulcer and is always chronic.
- 95% of duodenal ulcer and 75 % of gastric ulcer are caused by H.pylori.
- Increase in aggressive factors (H.pylori , NSAIDs , Acid ...) or decrease in defensive factors (Prostaglandins, Blood Flow, Mucus …) → Peptic ulcer.

Gastrin (produced by G cells which are in antrum and duodenum) regulates acid production from parietal cells (which are present in Body of stomach).

- H.pylori → binds to the epithelial cells → produce urease enzyme → converts urea to ammonia → ammonia is a base that lowers the acidity → G cells sense the lowered acidity → initiate more Gastrin production → increases the production of acid.

That is why **duodenal ulcer** is caused by increased production of acid.

3- Pancreatitis

	Acute Pancreatitis	Chronic Pancreatitis
Characteristic	Ranging in severity from focal edema and **fat necrosis** to parenchymal necrosis with **severe hemorrhage.**	Destruction of exocrine parenchyma, **fibrosis**, and, in the late stages the destruction of endocrine parenchyma .
Type of lesion	Reversible.	Irreversible.
Etiology	Gallstone, alcohol, biliary tract disease. - hereditary. - metabolically: hyperlipidemia and hypercalcemia.	Most commonly due to **alcohol abuse**, idiopathic.
Pathogenesis	Premature activation of trypsinogen → inappropriately activated pancreatic enzymes → autodigestion of the pancreatic substance.	- Activation of trypsinogen. - Fibrotic destruction.
Morphology	- Hemorrhage. - Fat necrosis: **enzymatic destruction of fat cells** → release fatty acids → combine with **calcium** → form insoluble salts that precipitate in situ.	- Fibrosis. - Acinar atrophy. - **Grossly:** **Gland is hard (because of extensive fibrosis),** sometimes with extremely dilated ducts and **visible calcification**.
Clinical features	Acute abdominal pain that radiates to the back.	Chronic abdominal pain. • **Silent** or recurrent attacks of abdominal pain, or persistent abdominal and back pain. •Mild **fever** • **Calcificationscan within the pancreas.**
Investigations	- Elevated **serum amylase.** ★ Elevated **serum lipase** level (more specific).	**mild-to-moderate elevations of serum amylase.**
Complications	- Shock. - Pancreatic abscess. - Acute respiratory distress syndrome. - Pancreatic pseudocyst.	- Irreversible impairment in pancreatic function. - Chronic malabsorption. - Diabetes mellitus (due to destruction of islets of Langerhans،endocrine part). - Pancreatic pseudocyst.

• The chief distinction between acute and chronic pancreatitis is the irreversible impairmentin pancreatic function that is characteristic of chronic pancreatitis.

PSEUDOCYSTS OF PANCREAS

- localized collections of necrotic-hemorrhagic material rich in pancreatic enzymes such cysts **lack an epithelial lining**, instead it's lined by a fibrotic wall.

• Pseudocysts usually arise after an episode of **acute pancreatitis**(usually present after 4-6 weeks of pancreatitis)**, or of chronic pancreatitis.**

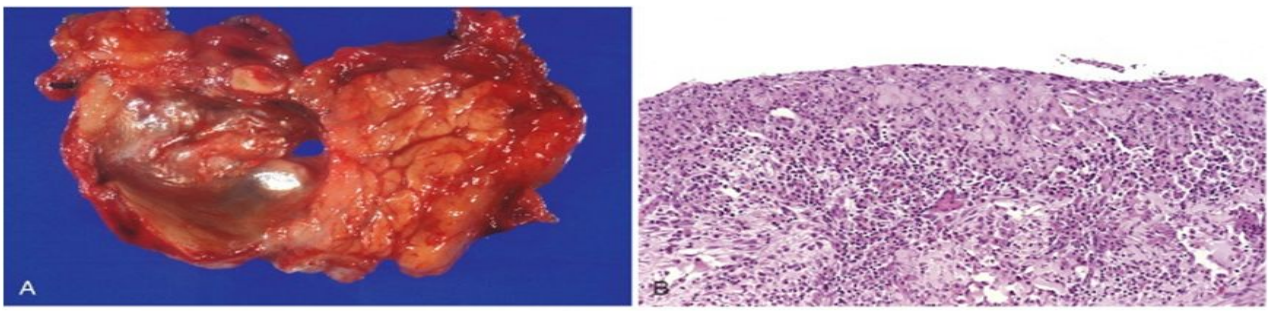
Complications:

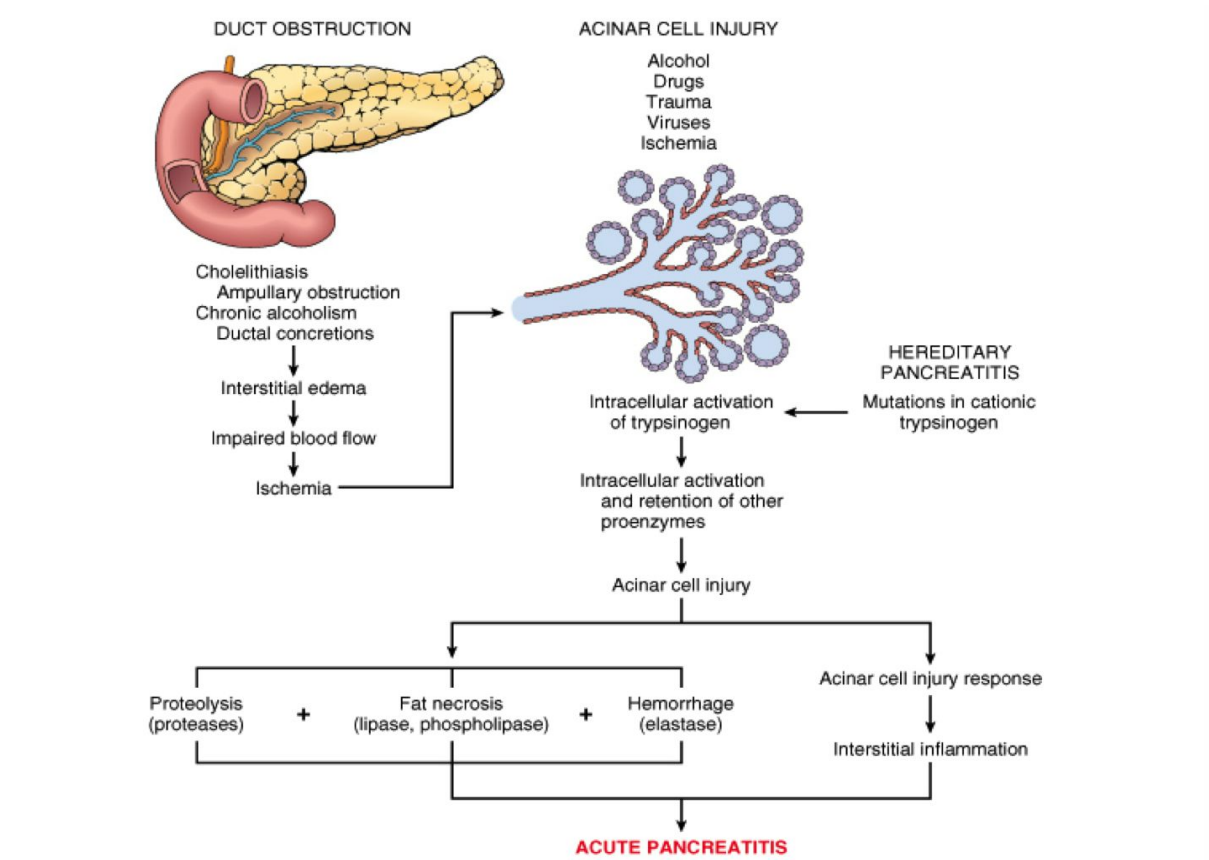
Infection, intraperitoneal hemorrhage **and peritonitis**

Morphology:

• Solitary.

• Pseudocysts form by walling of **areas of hemorrhagic fat necrosis.**





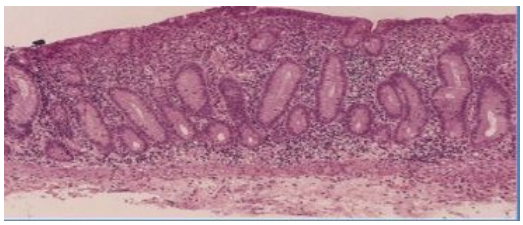
4- Malabsorption

Malabsorption: abnormal digestion or small intestinal mucosal abnormalities.

- Presents most commonly as **chronic diarrhea.**
- **Causes:** 1-Inadequate digestion. 2- deficient bile salt. 3-Primary mucosal abnormality
  4- Inadequate small intestine. 5- Lymphatic obstruction
- **Clinical features:** 1-General - abnormal stool → steatorrhea
  - Failure to thrive or poor growth.
  2- Depend on deficient nutrients.
- **Diagnose:** Stool studies, Blood test, Endoscopy.

Celiac disease

- An immune reaction to gliadin fraction of the wheat protein gluten.
- Patients have raised antibodies to gluten auto-antibodies.
- **Diagnosis :**
  1. Steatorrhea.
  2. Serology + IgA → to tissue transglutaminase or IgG → to deamidated gliadin or anti-endomysial antibodies.
  3. Biopsy of small intestinal → villous atrophy
  4. Improvement of symptoms and mucosal histology on gluten withdrawal from diet.
- Highly specific association withclass **II HLA DQ2** (95% of cases) and to a lesser extent DQ8.
- **Pathogenesis:** Person drink milk → Gluten → by digestive enzymes → gliadin peptide that is resistant to degradation →deamidated by tissue transglutaminase → Antigen presenting cells→ MHC class2 → CD4 T cells produce → cytokines that release matrix proteases → cell death and degradation in the epithelial cells → resulting in the loss of the villous surface in the small intestine.
- **Histology Findings** :
  1. Mucosa flattened with villous atrophy.
  2. Increased intraepithelial lymphocyte.
  3. Crypt elongation
- **Complication:** T-cell Lymphoma and GI tract Carcinoma.
- **Differential diagnose:** Tropical Sprue.

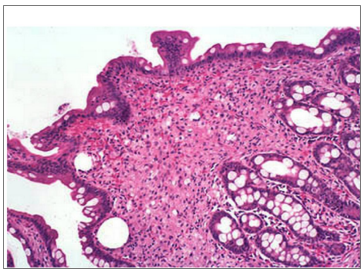


Lactose Intolerance

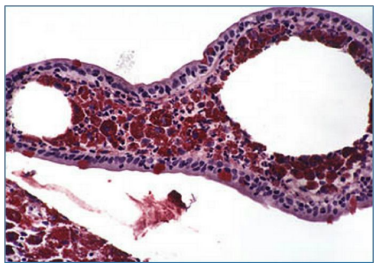
- Absent activity of the enzyme **lactase.**
- Bloating, abdominal discomfort, and flatulence, explosive diarrhea.
- 1 hour to a few hours after ingestion of milk products.
- It can Inherited or Acquired lactase deficiency.
- **Pathogenesis:** unabsorbed lactose withdraws water + in colon lactose is metabolised by bacteria → organic acid, Co2 , H2 → irritant and cause osmotic effect.
- **Diagnosis:** 1- Lactose free diet → resolution of symptoms 2- Hydrogen breath test

Small bowl whipple disease

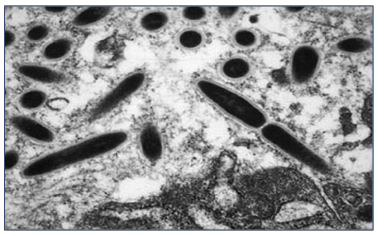
Caused by agram-positive bacterium **Tropheryma whippelii.**



Numerous macrophages are seen throughout the lamina propria.(of small intestine)



A PAS stain highlights the cytoplasmic inclusions (staining them a deep red).



An electron micrograph reveals that these inclusions are rod-shaped bacilli.

Intestinal obstruction

- Herniation , Adhesion, volvulus, intussusception.
- **Clinical presentation:** abdominal pain, distention, vomiting, constipation.
- **Treatment:** Surgical intervention.

5- Diarrhea

Abnormally high fluid content of stool 200-300 gm/day.

Complications: Dehydration, Electrolytes imbalance, Metabolic acidosis

Classification → based on duration

1.Acute (less than 2 weeks)

Aetiology

- **Infections** : 80% of acute diarrhea **-Rotavirus most common**
- **Food poisoning**
- **Drugs**

Antibiotic-Associated Diarrheas

- Caused ***Clostridium difficile due broad spectrum*** **Antibiotic**
- **Complication :pseudomembranous colitis.**

2.Persistent (2 to 4 weeks)

3.Chronic (over 4 weeks)

Aetiology

- Infection such as *Giardia lamblia* , Cryptosporidiosis , AIDS
- Post-infectious Following acute viral, bacterial or parasitic infections (ex:tropical sprue)
- Malabsorption (ex: Celiac disease)
- Inflammatory bowel disease (IBD)
- Endocrine diseases ex: Carcinoid
- Irritable bowel syndrome

Classification → based on pathophysiology

Osmotic	Secretory
•**Fasting improve the condition** •**Stool osmotic gap is high**, > **125 mOsm**/kg (**loss of hypotonic fluid)** **Causes :** ● **Malabsorption** in which the nutrients are left in the lumen to pull in water e.g. **lactose intolerance،celiac disease** ● **osmotic laxatives** ● **Hexitols** (poorly absorbed): sorbitol, mannitol, xylitol).	•**Lack of response to fasting** •**Stool osmotic gap < 100 mOsm/kg (isotonic)** **Causes :** ● ***The most common cause of this type of diarrhea is a bacterial toxin (E. coli , cholera) that stimulates the secretion of anions.*** • **Other causes:** **–Enteropathogenic virus e.g**. rotavirus and norwalk virus **– Neuroendocrine tumours** (carcinoid tumor, gastrinomas) **–Rectal villous adenoma**
Exudative (inflammatory)	**Motility-related**
•**Presence of blood and pus in the stool**. •**Persists on fasting** **Causes :** ● **Inflammatory bowel diseases such as** Crohn's disease or ulcerative colitis. ● **Invasive infections** e.g. ***E. coli***, ***Clostridium difficile*** **and *Shigella.*** *(not only their toxins)* • **Some bacterial infections** cause damage by invasion of the mucosa. → diarrhea with blood and pus in the stool caused by → **bacterial dysentery** **The main organisms are:** ❑ •***Campylobacter*** invades mucosa in the jejunum, ileum and colon, causing ulceration and acute inflammation. ❑ •***Salmonella typhi, S. paratyphi*** *A*, *B*, and *C* ❑ •***Shigella*** **infections** are mainly seen in young children. ❑ •**Enteroinvasive and enterohemorrhagic *E. coli*.**	-Rapid movement of food. -No inflammation in bowel mucosa. **Causes** ● **Irritable bowel syndrome (IBS)** – a motor disorder that causes abdominal pain and altered bowel habits with diarrhea predominating. ● **Carcinoid Syndrome** → Increased serotonin -It increases bowel motility.

For any suggestions or questions please don't hesitate to contact us on: Pathology434@gmail.com
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