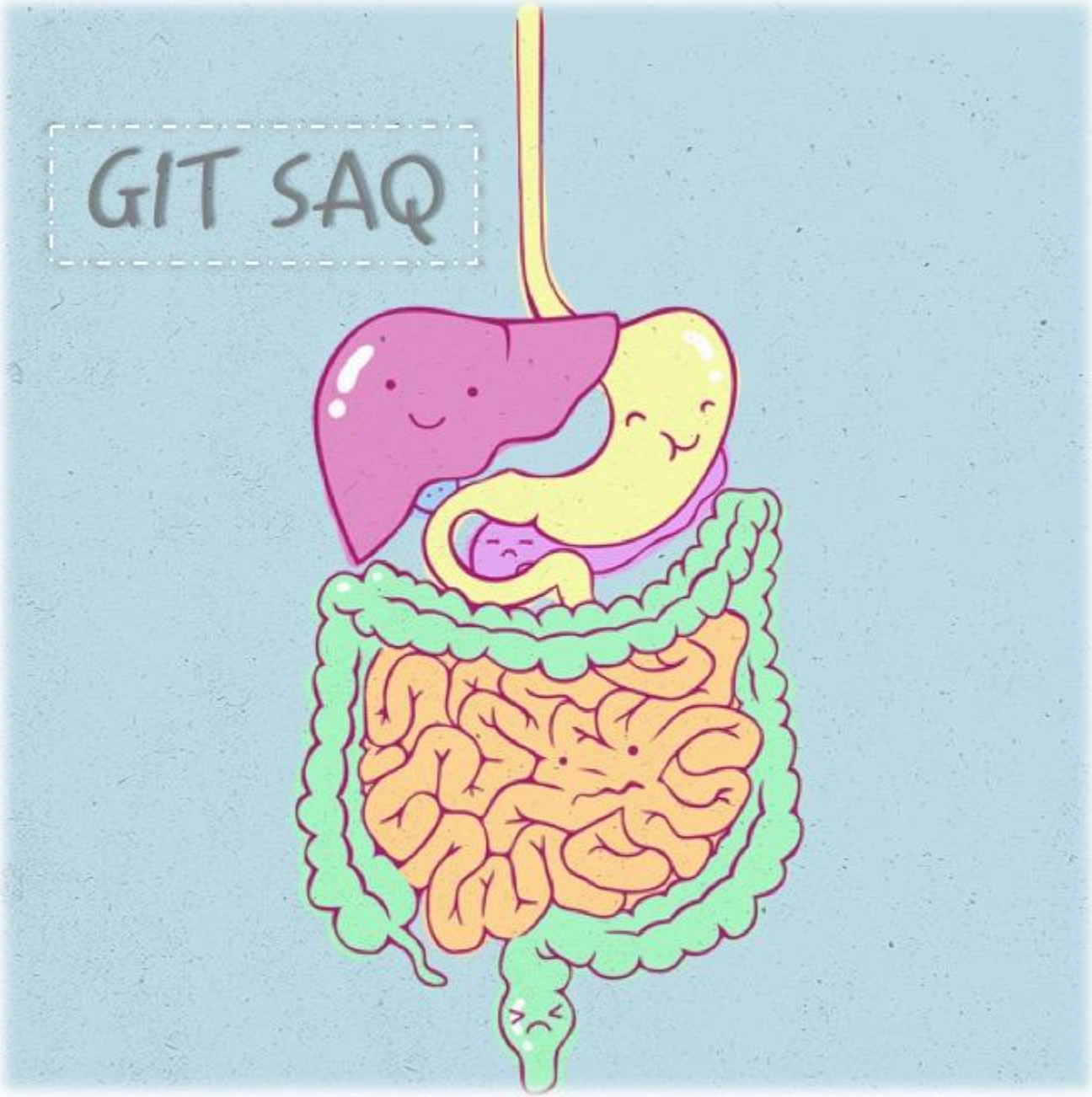
GIT SAQ



Pathology

Editing File

GERD

Define gastroesophageal reflux disease

Symptoms OR mucosal damage produced by the abnormal reflux of gastric contents into the esophagus

Pathophysiology (causes of GERD)

1-Abnormal lower esophageal sphincter (Hiatal hernia, functional, mechanical and others

2-increased abdominal pressure (obesity, increase gastric volume and pregnant.

Know clinical features of reflux esophagitis

1-Heartburn, Regurgitation

2- Atypical symptoms (coughing, chest pain, and wheezing)

Describe the pathological features of reflux esophagitis

1-Eosinophils and neutrophils

2-Elongation of lamina propria papillae

3-basal zone hyperplasia

Mention the complications of reflux esophagitis

1-Erosive esophagitis

2-Stricture

3-Barrett's esophagus → dysplasia → adenocarcinoma.

A patient came to the hospital complaining from a heartburn, Regurgitation, dysphagia with coughing and chest pain, how would you diagnose the patient?

using Esophagogastroduodenoscopy and PH monitoring.

Peptic ulcer

Define an Ulcer ?
a breach in the mucosa of the alimentary tract.

Mention the Types of Ulcer ?
1-Peptic ulcer (Acute or chronic)
2-Stress ulcers (acute gastric ulcers)

Write the Pathophysiology of GERD ?
imbalance between : 1- Aggressive factors 2- Defensive factors

Mention some examples of these factors ?
Aggressive: H.pylori, NSAIDs, pepsin, acid, bile salt.
Defensive: mucus, bicarbonate, BF, prostaglandins.

Acute peptic ulcer will find it in ?
1- acute gastriris (NSAIDs and alcohol)
2- severe stress response (burns,trauma, CVA)
3- extreme hyperacidity (zollinger-ellison syndroem)

What is the location of Chronic peptic ulcer:
98% in first part of the duodenum or stomach (4:1)

The two types of Chronic peptic ulcer are ?
A-Gastric ulcers: the mucosal defense against acid attack is :
1- mucus-bicarbonate barrier (broke by duodeno-gasrtic reflux)
2- surface epithelium (broke by H.pylori and NSAIDs)
In gastric ulcer, breakdown of mucosal defense is more important than hyperacidity.

B-Duodenal ulcers: hyperacidity is more impertant.
with infection, there will be increase acid secretion 2-6 times
H.pylori infection → gastric metaplasia (much more acid) → colonization (can't happen in intestinal type)

Discribe briefly what can we see in Gross picture & undermicroscop of Chronic peptic ulcer ?
- Gross: 20-100 mm, round to oval, sharply punched-out defect (raised and not sharp in cancer)
- **Micro (up down):** 1-necrosis 2-polymorph exudate 3-granulation tissue 4- fibrosis

What are the Clinical features that appear in chronic peptic ulcer ?
Epigastric pain (2-3 h after meal, relived by food)

Mention some Complication that follow chronic peptic ulcer ?
Iron deficiency anemia, frank hemorrhage, or perforation

Pancreatitis

What is the pathogenic mechanisms of pancreatitis?
Autodigestion of the pancreatic substance by inappropriately activated pancreatic enzymes.

Mention two factors could cause pancreatitis?

Ductal obstruction and long term alcohol abuse.

Inappropriate activation of pancreatic digestive enzymes happen due to what gene mutation?
genes encoding trypsinogen or trypsin inhibitors

Define acute pancreatitis?
group of reversible lesions characterized by inflammation of the pancreas,that ranges from focal edema and fat necrosis to widespread parenchymal necrosis and hemorrhage.

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Mention the clinical manifestation of acute pancreatitis ?
varies widely, from mild abdominal pain to rapidly fatal vascular collapse.

Define the chronic pancreatitis?
irreversible parenchymal damage and scar formation.

Clinical manifestation of chronic pancreatitis ?
1-calcifications of pancreas 2-Abdominal and back pain

Mention complication of chronic pancreatitis?
1- chronic malabsorption(due to pancreatic exocrine insufficiency).

2-diabetes mellitus (due to islet cell loss) .

Notes:

- In acute pancreatitis we find elevation of serum amylase levels in the first 24 hours- rising serum lipase level within 72 to 96 hours.

- In the chronic one we find mild elevation of serum amylase.

Malabsorption

What is the defination of Malabsorption Syndrome?

lnability of the intestine to absorb nutrients adequately into the bloodstream.

What is the main purpose of the gastrointestinal tract?

To digests and absorbs nutrients.

What are the mechanisms and causes of malabsorption syndrome?

1- primary mucosal abnormalities like (Celiac disease).
2- Inadequate small intestine. like (intestinal resection)
3- Inadequate digestion. (postgastroctomy)
4- Lymphatic obstruction.(melignant lymphoma)
5- Bile salts deficiency

Inadequate digestion happen in which organ?

1- stomach. 2- Pancrease. 3- Bile.

What are the causes of Small intestine abnormalities?

1- Mucosa. 2- Inadequate small intestine. 3- Lymphatic obstruction.

What are the clinical features of Malabsorption syndrome depend on deficient nutrient?

1- Protein.
2- B12, folic acid and iron deficiency.
3- Vitamin D, Calcium.
4- Vitamin K and other coagultion factor.

How to diagnosis Malabsorption Syndrome?

1- Fecal fat study. 2- Blood tests. 3- Stool studies. 4- Endoscopy.

What are the Complications of celiac disease?

1- Osteopenia, Osteoporosis. 2- infertility in women 3- Short stature, Delayed Puberty, anemia, Malignancies, intestinal t cell lymphoma.

Inherited lactase deficiency divided into: 1- Congenital lactase deficiency. 2- Childhood onset and adult onset lactase deficiency.

What kind of test do we use to diagnose Lactose intolerance?

Hydrogen breath test.

Diarrhea

Definition: 3 or more liquid or loose stools per day

Normally, fecal osmolality is equal to serum osmolality (290 mosm/kg)

Classification: **acute** (2 weeks), **persistent** (2-4 weeks), **chronic** (4 weeks)

Types:

A- **Secretory**: increase active secretion
1- high stool output, no benefit of fastig, normal osmo gab <100
2-causes: bacterial toxins (E.coli and cholera)&endocrine neoplasm(carcinoid)

B- **Osmotic**: poorly absorbed substanses pull water to lumen
1- normal stool output, benefit with fasting, high osmo gap >125
2- causes: malabsorption ans osmotic laxatives

C- **Exudative (inflammatory)**:outpouring protein blood or mucus form inflamed or ulcerated mucosa
1- presence of blood and/or pus in stools, no benefit with fasting
2- causes: inflammatory bowel disease and invading infections

D- Motility-related:rapid movement of food through intestine,irritible bowel syndrome

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Etiology:

Acute : mostly infections (viral;Rota), food poisoning, drugs(Antibiotic realated;C. difficle)

Chronic : infection(giardia),postinfection,malabsorption,IBD,IBS,colon cancer

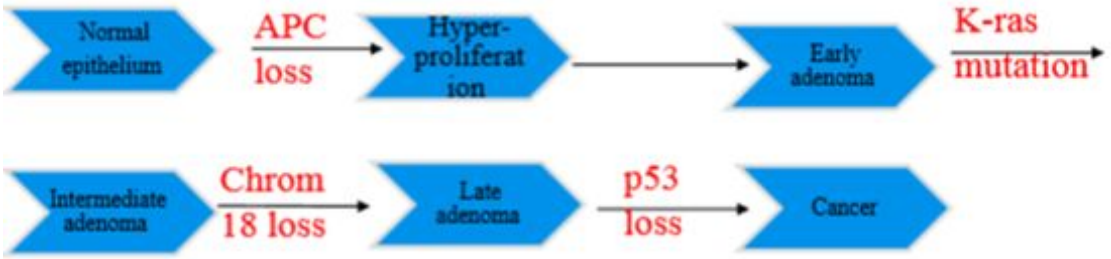
Complication: dehydration, electrolyte imbalance, metabolic acidosis, malnutrition.

Colonic polyps

A-*non* neoplastic polyps				
	Hyperplastic polyps	- hamartomatous polyps"juvenile". - retention in adult.	- hamartomatous polyps Peutz - Jeghers syndrome	Inflammatory polyps
Incidence +region +def	Rectosigmoid mostly adult .	Developmental malformation Rectum , mostly children<5 years.	Autosomal dominant.	long standing IBD
Features	1-sawtooth surface 2-starshaperd crypt 3-lined by goblet or absorptive cell.	1-Affect glands and lamina propria. *cronkhite-canada syndrome: non hereditary,polyps plus ectodermal abnormalities of nails.	1-large and pedunculated hamartomatous polyps. 2-Mucosal And Cutaneous Pigmentation	Pseudopolyps
Prognosis	no malignant potential		Carcinoma Of The Pancreas,breast,lung, ovary and uterus	-

B- neoplastic polyps			
	Tubular adenoma	Tubulovillous adenoma	Villous adenoma
Incidence +region +def	75% of all neoplastic polyps In sigmoid colon	20%-30% of all neoplastic polyps	Least common " serious " Age :60-65 Rectosigmoid
Features	Villous architecture less than 25%	Villous architecture between 25%-50	Villous architecture over 50% - Rectal bleeding - Anemia - Hypokalemia - hypoalbuminemia
Prognosis			Malignant.

The probability of a neoplastic polyp to become carcinoma is depends on lots of factors such as the villous features , size of polyps and dysplasia , the pathway:



Inflammatory bowle Disease

	Crohn's	Ulcerative Colitis
Site	Any part of the GIT	Colon only
Pattern	Skip areas of normal mucosa	Diffuse involvement of mucosa
Symptoms	Right lower quadrant pain (ileum)with non bloody diarrhea.	Left Lower quadrant (rectum) with bloody diarrhea.
Depth of the ulcer	Deep ulcers (fissure)	Superficial ulcers
Extent of inflammation	Transmural inflammation	Mucosal inflammation only
Inflammation	Lymphoid aggregates with granulomas	Crypts abscesses with neutrophils
Fistula Formation	Yes	No
Creeping mesenteric Fat		
Fibrous thickening wall		
Granulomas		
Dysplasia	rare	Common
Carcinoma	rare	More Common (10%)
Mucosal appearance	**Cobblestone**	**Pseudopolyps**
Bowel wall	Thickened wall narrow margin	Thin wall Dilated lumen
Complication	-Fistula formation -Bowel perforation -Stricture formation	-Haemorrhage -Toxic Megacolon -Systemic effects

LIVER Cirhosis

Characteristics	Fibrosis, nodules and disruption of the architecture of the **entire liver.**
Features	- Vascular architecture is reorganized by the parenchymal damage and scarring, with the formation of abnormal interconnections between **vascular inflow** and **hepatic vein outflow** channels.
Classifications	**1- Alcoholic liver disease. 2- Viral hepatitis. 3- Biliary diseases.**
Pathogenesis	Excess collagen due to: - Chronic inflammation, with production of inflammatory cytokines. - Cytokine production by activated endogenous cells (Kupffer cells, endothelial cells, hepatocytes, and bile duct epithelial cells).
Clinical Features	- Jaundice and even hepatic failure. - When symptomatic they lead to nonspecific clinical manifestations: anorexia, weight loss, weakness, osteoporosis, and, in advanced disease, frank debilitation.

Complications	Features
A- Portal Hypertension:	- Resistance to blood flow.
1- Ascites	- Is the accumulation of excess fluid in the peritoneal cavity: 85%.
2- Portosystemic venous shunts	- **Esophageal Varices:** Diseases that impede this flow cause portal hypertension and can lead to the development of esophageal varices, an important cause of esophageal bleeding. - **Hepatic Encephalopathy:** Appears to be associated with elevated blood ammonia levels, which impair neuronal function and promote generalized brain edema. - **—Hepatorenal Syndrome:** Appearance of **renal failure** in individuals with severe chronic liver disease.
3- Splenomegaly:	- Hematologic abnormalities.
B- Liver failure	- **—Jaundice & Cholestasis.**
C- Hepatocellular Carcinoma	-

Liver & Pancreas tumors

	Incidence	Etiology	Morphology	Prognosis
Hepatocellular Carcinomas	- Male. - More than 85% of cases of HCC → chronic HBV infection. - Western: cirrhosis 85-90% + chronic liver diseases	1. HBV & HCV. 2. Cirrhosis. 3. Chronic alcoholism. 4. Aflatoxins from aspergillus. 5. Tyrosinemia & hemochromatosis	- In well-differentiated tumors, cells that are recognizable as hepatocytic in origin. - Positive for alpha-fetoprotein - Satellite nodules & greenish cast	Death occurs from: 1. Cachexia. 2. Variceal bleeding. 3. Liver failure with hepatic coma. 4. Rupture of the tumor with hemorrhage
Fibrolamellar carcinoma	- A distinctive variant of hepatocellular carcinoma. - Young male and female adults (20 to 40 years of age).	No association with HBV or cirrhosis	well-differentiated polygonal cells growing in nests or cords and separated by parallel lamellae of dense collagen bundles	**Better prognosis**
Hepatic adenoma	-	**Oral contraceptive drugs.**	Solitary, sharply demarcated mass up to 40 cm.	30% peritoneal cavity bleeding → hypovolemic shock → emergency treatment.
Cholangio-carcinoma	malignancy of the biliary tree, arising from bile ducts	- Primary sclerosing cholangitis. - Congenital fibropolycystic - Exposure to Thorotrast. - In the Orient : Clonorchis sinensis	- Cholangiocarcinoma resemble adenocarcinomas. - Well to moderately differentiated. - Rarely bile stained	- **Late detection:** as obstruction to bile flow or as a symptomatic liver mass - Prognosis is poor - Alpha-fetoprorein is not elevated
Angiosarcoma	Malignant tumor of endothelial cells	- Cirrhosis 20-40% - exposure to vinyl chloride and thorotrast	-	-
pancreatic carcinoma	- Ductal epithelial cells. - Most common: ductal adenocarcinoma. - Less common: 1. Acinar cell carcinomas. 2. Adenosquamous carcinomas. 3. Undifferentiated carcinomas.	- 6^{th} to 8^{th} decade. - Blacks. - Males. - Diabetics. - Cigarette smoking. - K-RAS mutation.	60% arise in the head of the gland - Highly invasive. - Desmoplastic response	- One of the highest mortality rates of any cancer. - Jaundice, weight loss, pain ,massive metastasis to liver and migratory thrombophlebitis.

Cholecystitis

The Majority of gall stones are asymptomatic.. Their Types are :

1-Cholesterol stones (most common): containing crystalline cholesterol monohydrate : 5F : Female, fertile, forty, family history and Fat
2-Pigments stones: containing bilirubin calcium salts :A- Chronic hemolytic syndromes most common (Biliary infections)

Cholesterol stones	Pigments stones
Exclusively in gall bladder	Radiolucent
Yellow	Black
Hard external surface	Soft + multiple
Radiolucent	Radio-opaque

What is The most common complication of choliathsisis ? bile duct obstruction then Pencreatits.

Acute cholecystitis

- Acute calculous cholecystitis (within gallstones) -common-
- precipitated 90% of the time by obstruction of the neck or cystic duct.
- It is the primary complication of gallstones
- Empyema of the gallbladder
- Thickened, edematous and hyperemic (congested) gallbladder **right upper quadrant or epigastric pain (Colicky pain)**

Chronic cholecystitis

- Chronic cholecystitis may be a sequel to repeated bouts of acute cholecystitis But in most instances it develops without any history of acute cholecystitis
-It is associated with cholelithiasis in over 90% of cases **right upper quadrant or epigastric pain "colicky pain")**
- Patients often have intolerance to fatty food
- Fibrotic and atrophied of gallbladder
- porcelain gallbladder

- رند الحميضي
- حسين الكاف
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- ساره السلمان
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