Gastrointestinal diseases

- Define atresia congenitally interrupted hollow organ
- Define fistula abnormal connection between two hollow structures
- Describe esophageal atresia
 - most common congenital abnormality of the esophagus
 - Unknown pathophysiology
 - Is a congenital interruption of the esophagus with or without a tracheoesophageal fistula
 - o Incidence 1/2500-4500 live births
 - Survival is improving with improving neonatal care
- Classification of esophageal atresia and tracheoesophageal fistula

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- Type A, isolated esophageal atresia.
- Type B, blind-ending lower esophageal pouch with a fistula between the trachea (proximal tracheoesophageal fistula) and the upper esophageal pouch (distal atresia).
- Type C, esophageal atresia with a blind proximal esophageal pouch and a distal tracheoesophageal fistula.
- Type D, esophageal atresia with two fistulas between the trachea and the lower and upper esophageal pouches (proximal and distal fistulas).
- Type E, fistula between the esophagus and the trachea without atresia (H-type).
- F Congenital esophageal stenosis.
- What are the swallowing disorders?

Achalasia

GERD

Ectopia

Esophageal stenosis

. . . .

Describe esophageal varices

Venous blood from the gastrointestinal tract (GI) is transported to the liver via the portal vein before reaching the inferior vena cava. his route allows the material absorbed from the intestine to be transported to the liver before entering systemic circulation. Diseases like cirrhosis may obstruct this flow and cause **portal hypertension**. **Sub-epithelial longitudinal veins** might enlarge to form esophageal varices. With increasing size and with rising intravascular pressure, they are more likely to rupture resulting in esophageal bleeding

• describe gastritis in general

- Gastritis is an inflammation of the gastric mucosa.
- It can be acute or chronic.
- Etiology is multifactorial
- Symptoms are more severe in patients with the acute form than in patients with the chronic one where it could be totally asymptomatic.
 - They vary from mild dyspepsia to massive hemorrhage.
 - nausea, vomit
 - heart burn
 - halitosis
 - frequent belching

• main characteristics of acute gastritis

- o it is an acute inflammation of the stomach mucosa
- o complications: necrosis and hemorrhage
- possible causes and risk factors:

NSAIDs (main)

- inhibit prostaglandin production, reducing the resistance of the mucosa to acid environment
- aspirin promotes accumulation of protons (H+) in the lumen and H+ stimulate histamine release which in turn stimulates HCl acid secretion
- smoking
- alcohol
- chemotherapy (reduce ability to heal from erosions)
- ingestion of corrosive substances (aliphatic acids)
- bile (?)
- H. pylori infection (which tends to turn into chronic)

main characteristics of chronic gastritis

- Chronic inflammation of the gastric mucosa
- often associated with small intestine inflammation
- Classification based on the histopathological wall features: localization and depth of lesions - type of infiltrating inflammatory cells:
 - Specific Bacterial infection, e.g. Helicobacter spp. (Helicobacter felis, H. heilmannii, H. pylori)
 - Non-specific types of chronic gastritis
 - Lymphoplasmacytic gastritis (diffuse infiltration of lymphocytes and plasma cells into the lamina propria of the stomach resulting in diffuse mucosal inflammation)
 - Eosinophilic gastritis (a rare disorder, usually in children or young adults, characterized by eosinophilic infiltration, which also involves esophagus or duodenum)
 - Granulomatous gastritis (granuloma surrounded by dense

infiltrates of inflammatory cells; more than half are associated with Crohn's disease)

- describe peptic ulcers
 - PU is an acid-induced lesion that affects the mucosa, the submucosa and sometimes muscularis mucosae. It can be gastric or duodenal.
 - There is tissue necrosis accompanied by inflammation, formation of granulation tissue with presence of fibrous reaction.
 - Main risk factors are those reducing or altering mucosal cell defensive barrier (H. pylori infection, alcohol and tobacco consumption, NSAIDs).
 - They are at risk for neoplastic transformation
- about H. pylori
 - H. pylori; colonises the gastric mucosa of 50% of the human population
 - o main cause of chronic gastritis and peptic ulcer disease
 - two major virulence factors are:

cytotoxin-associated gene A (CagA)

- inducing host cell morphological changes by activating tyrosine kinases and subsequent pro-inflammatory and mitogenic responses.
- ⋄ Bacterial strains with the cagA gene are associated with the ability to cause ulcers, MALT lymphomas, and gastric cancer

vacuolizing cytotoxin-associated protein A (VacA)

- inducing vacuolization with formation of membrane channels, cytochrome c release from mitochondria with apoptosis activation. It is associated with precancerous gastric lesions and progression to malignant phenotype.
- H.Pylori produces urease that hydrolyzes urea into CO2 and ammonia, thus creating a basic environment around the bacterial colony, but increasing the acidic pH of the stomach.
- Diagnosis:

Urea Breath test (UBT)

- 1. Patient swallows test capsules containing urea tagged with radioactive carbon13 (13C-urea).
- 2. If the patient is infected with H. pylori, 13C-urea will be cleaved, and the produced 13CO 2 is quickly absorbed moving from the blood to the lungs.
- 3. 13CO 2 is exhaled in the breath.
 - 1. The test is positive if the analysis of the patient's breath reveals 13CO 2 presence (breathed 13CO 2 peaks within 15-20 minutes and declines thereafter).
- Serologic tests: IgG and IgA

- H. pylori stool antigen test
- what can cause malabsorption syndromes?- defect
- what are the general causes (concept) of malabsorption syndromes?
 - reduction of the absorption area
 - o alteration of transport mechanisms through the intestinal barrier
 - defective digestion of food
- Increased undigested or badly-digested substances can cause:
 - Intestinal water influx due to osmosis
 - enzymatic attack by bacteria (flora) → inflammation
- bowel obstruction (open)
 - Types

can happen in the small or large intestine. They can be functional or mechanical

They can also be classified in partial, complete or closed loop. the causes can be intraluminal, intramural or external (in both LBO and SBO)

Symptoms

Obstruction frequently causes abdominal pain, nausea, vomit, alternate constipation/diarrhea. This, in turn, prevents the normal movement of digested products.

Most common types

Most common of all are the small bowel obstructions (**SBO**) Most common causes of SBO are **external**, in particular:

- post-surgical adhesions: fibrous tissue that forms between organs and/or the peritoneal membrane. Can lead to necrosis.
- cancers
- hernias: weakness of abdominal wall, a piece of the intestine "escapes" through
- Most common causes of intraluminal obstruction of the small bowel are bezoars and gallstones.

Most common cause of **intramural** obstruction, both for SBO and LBO is **Chron's disease**

Most common causes of LBO are ****

- adenocarcinoma
- diverticulitis
- volvulus: a loop of intestine twists around itself
- what is intussuception
 Intussusception (Invaginazione): due to an abnormal peristaltic
 wave due to the presence of tumor masses or wide lesions.
- 0 ?
- what is IBS?

It is a **functional** GI disorder, (disorder of gut-brain interaction), that causes chronic intermittent abdominal pain associated with diarrhea,

constipation, or both. it is characterized by

- Altered gut immune activation
- Increased intestinal permeability
- Intestinal microbiota changes
- Visceral hypersensitivity
- Altered brain-gut interaction associated with alterations in autonomic nervous system output causing emotional changes (alteration of hypothalamic pituitary adrenal axis activity)
 - associated to multiple comorbidities such as anxiety, depression, and somatization.
- Inflammatory bowel diseases (USE THE BOOK)
 These are the questions from general surgery
 - Crohn's disease: what is it? signs and symptoms?
 - Crohn's disease is a granulomatous inflammation, it affects any part of the small or large intestine, with ulcerations in the wall and formation of fistulae and abscesses.
 - Signs and symptoms:
 - mucous diarrhea
 - hematochezia
 - nutrient malabsorption
 - dehydration
 - abdominal pain
 - diarrhea
 - Ulcerative colitis: what is it? signs and symptoms?
 - involves the terminal tract of the intestine: mainly rectum and colon, it is mostly inflammatory and interests only mucosa and submucosa layers of the intestine.
 - Signs and symptoms:
 - mucous diarrhea
 - hematochezia (passage of fresh blood per anus)
 - nutrient malabsorption.
 - main difference between ulcerative colitis and Crohn's disease

•	• ULCERATIVE COLITIS	CHRON'SDISEASE
• LOCATION	 Starts at the rectum and spreads to the colon 	 can affect any part of GIT, from mouth to the anus
DEPTH OF INFLAMMATION	• mucosa	 can involve the mucosa, submucosa, and even the deeper layers.
• DISTRIBUTION	continuous area of the colon and rectum	 patches of inflammation with normal areas of tissue in between

 SYMPTOMS in common: abdominal pain, diarrhea, rectal bleeding, and weight loss 	• the diarrhea is often more frequent and may contain blood and mucus.	 the diarrhea is associated with abdominal pain, especially in the right lower quadrant, due to involvement of the small intestine.
• COMPLICATIONS	increased risk of colon cancer	• STRICTURE. Also more likely to cause complications beyond the digestive tract, such as joint pain, skin problems, eye inflammation, and liver involvement.