GI PATHOLOGY
LECTURES 1-4 summary

DR H AWAD

Achalasia= لاارتخائية

- Caused by failure of the LES muscles to relax
- = Incomplete LES relaxation, increases LES tone and aperestalsis
- Can be Primary and secondary
- Primary: failure of the distal esophageal inhibitory neurons . Idiopathic
- Secondary= Chagas disease: Trypanosoma cruzi infection destroys myenteric plexus neurons
- characterized by difficulty in swallowing, regurgitation, and sometimes chest pain
- *ACHALASIA INCREASES RISK OF **SQUAMOUS** CELL CARCINOMA(squamous not adeno)*

Esophageal varices

- Esophageal veins are one of the sites where the splanchnic and systemic circulation can communicate is the esophagus .
- That’s why when portal hypertension increases, collateral vascular channels develop in the esophageal veins to allow blood to shunt from the portal to caval system (inferior vena cava)
- These collateral veins (varices) enlarge and can rupture.
- If rupture: cause bleeding

Causes of esophageal varices

- Any disease that causes increased portal hypertension will result in esophageal varices
- Liver cirrhosis is the most common cause worldwide, especially alcoholic liver disease
- Hepatic schistosomiasis is the second most common cause.

Esophageal lacerations vomiting

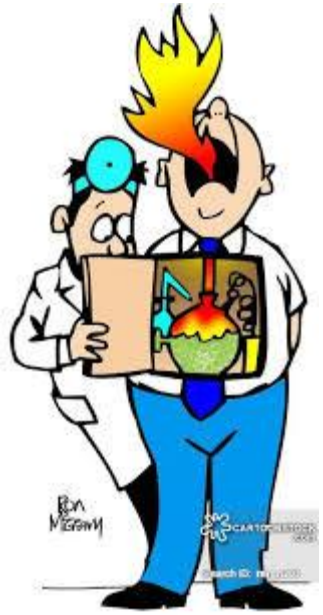
- Most common esophageal laceration: Mallory Weiss tears
- Associated with severe vomiting or with acute alcohol intoxication
- Normally there is a reflex relaxation of the gastroesophageal muscles before antiperistaltic contractile wave associated with vomiting
- This reflex fails during prolonged vomiting resulting in esophageal wall stretch and tear.
- Patients present with hematemesis (bloody vomit)
- The tears are longitudinal, superficial , cross the gastroesophageal junction and healing is usually rapid and complete… no surgical intervention is needed.

Reflux esophagitis

- Is inflammation of the lower esophagus due to reflux of gastric contents, which are acidic, from the stomach to the esophagus.

- It is the mot common cause of esophagitis

Also called: gastroesphageal reflux disease GERD



Pathogenesis of esophageal reflux

- Reflux of gastric juices into esophagus causes mucosal injury in the esophagus

Causes of this reflux

- 1. Decreased LES tone will cause reflux: alcohol, smoking
- 2. increased abdominal pressure: obesity, pregnancy,
- 3. delayed gastric emptying and increased gastric volume
- 4. in many cases, no cause is known !!

Esophageal adenocarcinoma

- Occurs in patients with Barrett mucosa
- Present as a mass in the lower esophagus, close to lower esophageal sphincter
- Prognosis: Depends on stage (extent of invasion and spread)
- 5 year survival 80% if the tumor is limited to the mucosa or submucosa
- 5 year survival is 25% if the disease is advanced.

Esophageal Squamous cell carcinoma

- Affects adults older than 45
- Risk factors: alcohol, smoking, poverty, caustic injury, **achalasia,** frequent consumption of very hot drinks, previous radiotherapy.
- Present as mass. Usually in the middle third of the esophagus
- They spread to adjacent structures and to lymph nodes early in their development because of the rich lymphatic supply
- Overall 5 year survival is 9%, because the majority of cases are discovered at a late stage

Acute peptic ulcer

- = focal loss of the mucosa.

<u>Causes:</u>

1. NSAIDS
2. Stress ulcer (physiologic stress in critically ill patients)
3. Curling ulcer, in burns and trauma
4. Cushing ulcer, occur in association with increased intracranial pressure.

Chronic gastritis

- H Pylori associated chronic gastritis.. Type B gastritis.. majority of cases..
- Autoimmune gastritis.. Type A, 10% of cases

H pylori

- Gram negative bacilli
- Found in : duodenal ulcer, gastric ulcer, chronic gastritis
- Also associated with gastric carcinoma and lymphoma

Autoimmune gastritis

- Less than 10% of chronic gastritis
- Usually spares the antrum
- Induces hypergastrinemia

Characteristics of autoimmune gastritis

- Antibodies to parietal cells and intrinsic factor
- Reduced pepsinogen 1 level
- Antral endocrine cell hyperplasia
- Vitain B 12 def
- achlorohydria

	H pylori gastritis	Autoimmune gastritis
site	Mainly antrum	Body.. Spares antrum
Acid production	Usually increased	decreased
gastrin	Normal or decreased	increased
serology	H pylori antibodies	Antibodies to parietal cells and intrinsic factor
complications	Peptic ulcer, adenocarcinoma, lymphoma	Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor
associations	Poverty, low socioeconomic class	Other autoimmune diseases: thyroiditis, diabetes,

Peptic ulcer disease (PUD)

Causes

- H pylori
- NSAIDS

- In USA ulcer due to NSAIDS is commoner nowadays than those due to H pylori due to 1. decreased pylori infection and 2. increased aspirin use in the aging population (as a protection of thrombosis)

epidemiology

- 10% of males and 4% of females develop peptic ulcer in their lifetime

- Sites: PUD can occur in any site exposed to gastric acids ; antrum and first part of duodenum are the most common sites.
- It can also occur in the esophagus as a complication of reflux
- Ectopic gastric mucosa can also be affected.

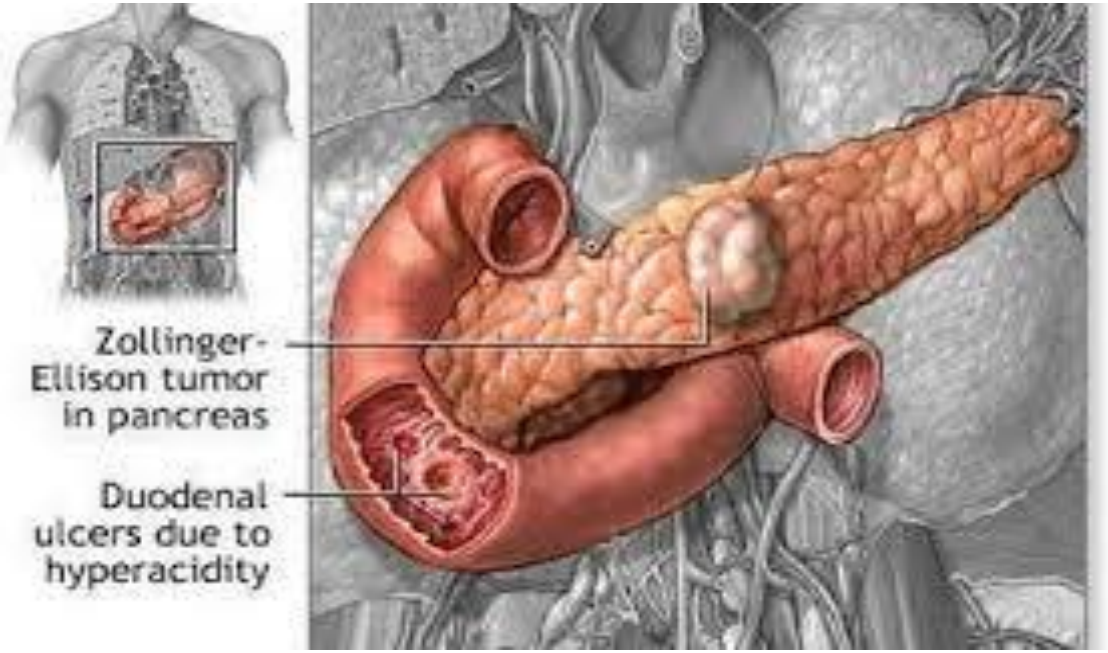
pathogenesis

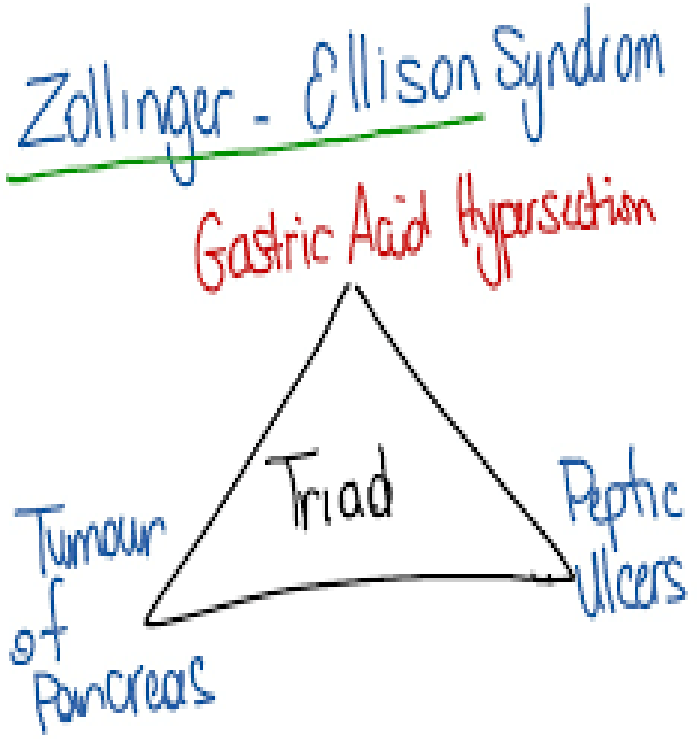
- Hyperacidity: essential.. Caused by: H pylori, parietal cell hyperplasia, or increased gastrin like in Zollinger Ellison syndrome (see next slide)
- NSAIDS
- Smoking
- Hypercalcemia: increases gastrin
- Psychologic stress can increase acid secretion

Zollinger Ellison syndrome

- Multiple gastric ulcers in the stomach, duodenum and even jejunum .. Due to uncontrolled gastrin secretion from a tumor … this results in massive acid secretion.

Z-E syndrome


Zollinger-
Ellison tumor
in pancreas
Duodenal
ulcers due to
hyperacidity


Zollinger - Ellison Syndrom
Gastric Acid Hypersection
Triad
Tumour of Pancreas
Peptic Ulcers

Gastric adenocarcinoma

- 90% of gastric tumors are adenocarcinomas
- Symptoms: nausea, vomiting and epigastric pain.. All are non specific which delays diagnosis

Gastric adenocarcinoma

Intestinal type:

- occurs mainly in high risk areas
- Develops from dysplasia or adenoma
- Mean age 55
- Male : female= 2:1

Diffuse type

- Incidence is uniform across countries
- No known pre-cancer lesion
- Male: female 1:1

outcome

- 5 year survival for early lesions: 90% even if there is lymph node metastasis
- 5 year survival for advanced disease: 20%
- Overall 5 year survival 30% .. Because of late detection