



Benign Gastric & Duodend



Disedses

<u>Objectives:</u>

- Definition
- Presentation
- Diagnosis
- Treatment

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Different from 436's lecture

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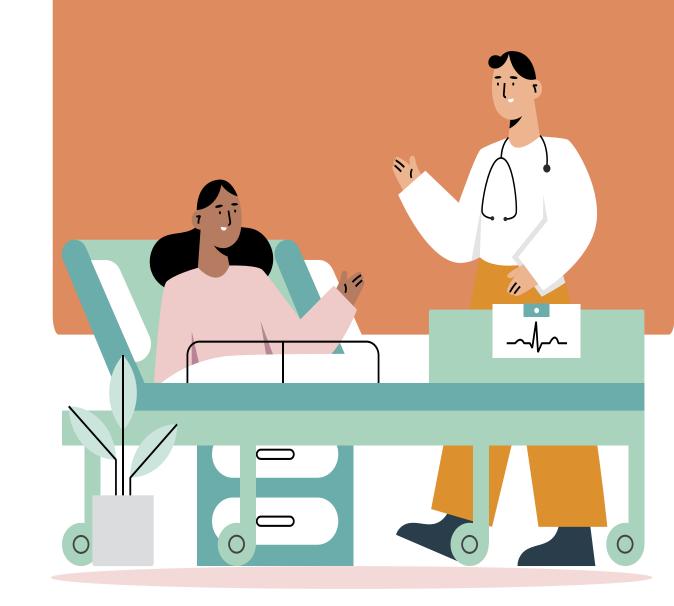
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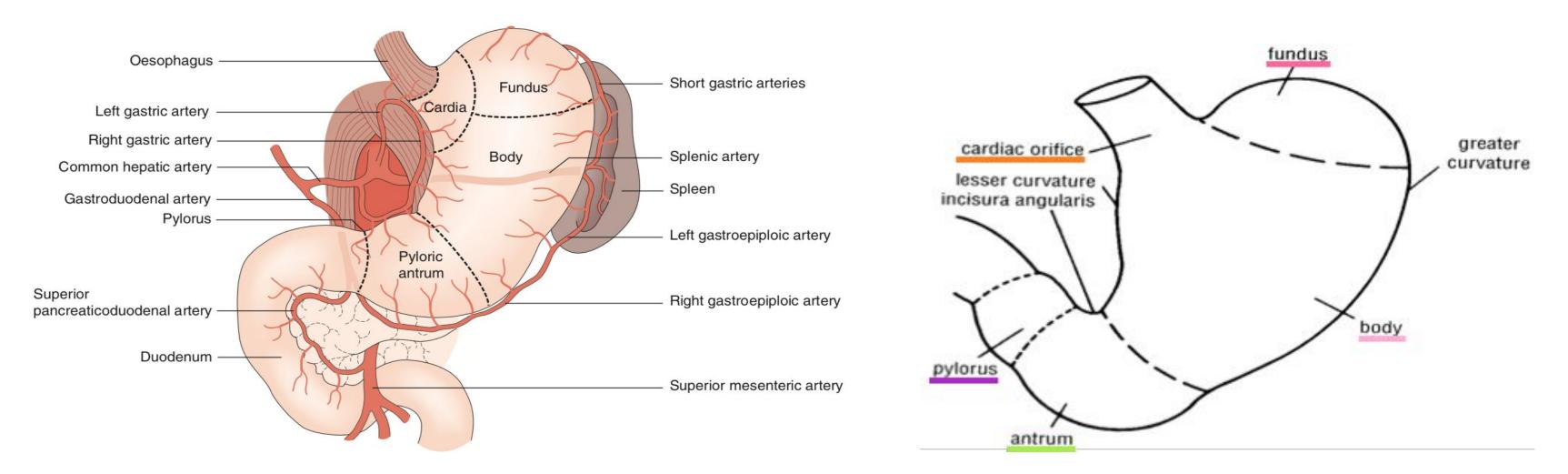






Basic Review

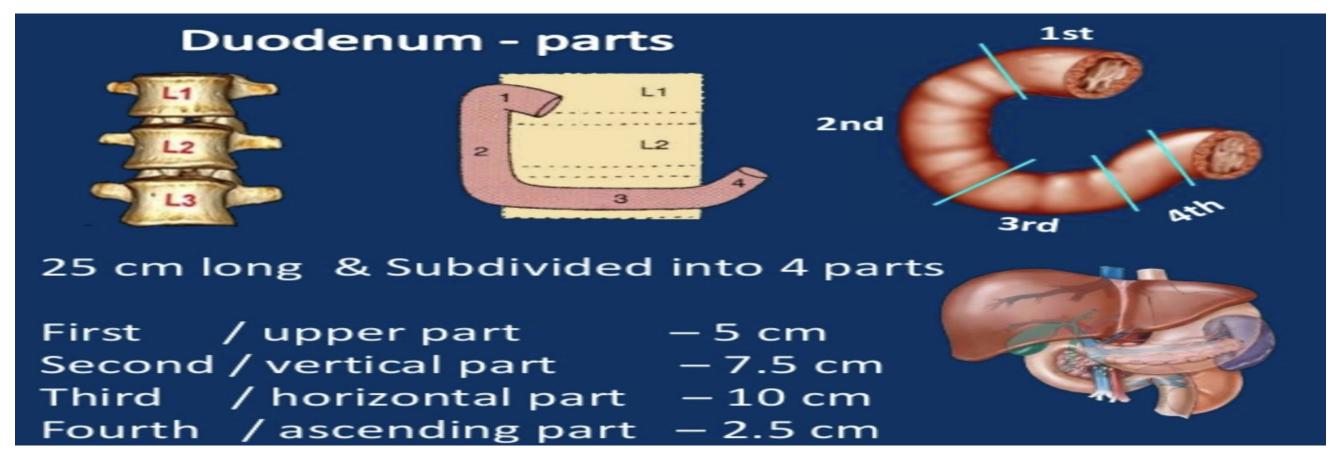
The doctor said it's important to know the **anatomy** and **physiology** of the stomach and duodenum and it will come on the exam.



Stomach

subdivided into 5 parts (each part has secretory and nonsecretory function)

Cardiac orifice	Site for Barrett's disease
Fundus	Secretory: parietal cell and Neurogenic: hunger feeling - The fundus has an important role in vit B12 absorption by secreting intrinsic factor.
Body	Secretory: parietal cell
Antrum	Site for surgical ulcer treatment: by cutdown the acid secretion (site for gastrin) Even if the ulcer in the duodenum.
Pylorus	Site for Dumping Syndrome (food goes immediately to SI)

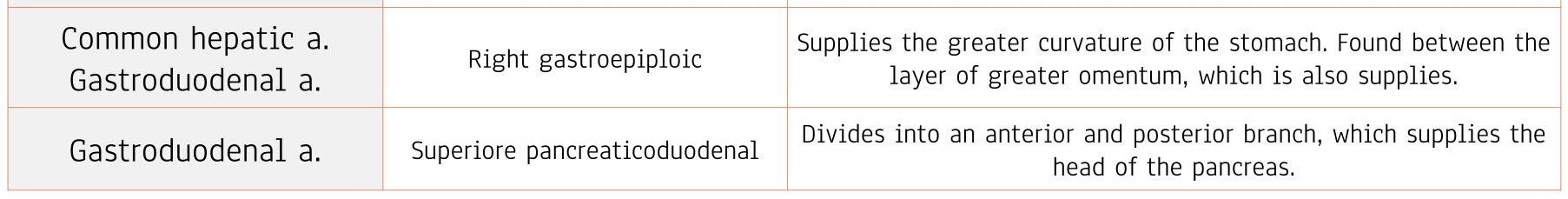


The first part is a common site of **ulcer**

The second part is a common site of **diverticulum**

The third part is a common site of Superior Mesenteric Artery Syndrome. will be explained later

Blood Supply		
Artery	Branch	Supply
Celiac trunk	Left gastric	Esophageal branches
Splenic artery	Left gastroepiploic	Supply the greater curvature of the stomach. Anastomosis with the right gastroepiploic artery.
	Short gastric arteries	5-7 small branches supplying the fundus of the stomach.
	Pancreatic branches	Supply the body and tail of pancreas
Common hepatic a. Proper hepatic a.	Right gastric	Supplies the pylorus and lesser curvature of the stomach



Basic Review

Gastric motility

- Food is passed from the oesophagus into the stomach, where it is stored, ground and partially digested. As food enters the stomach:
 - 1. The muscles in the stomach walls relax and intragastric pressure rises only slightly. This effect is known as **receptive relaxation**, and is mediated by the **vagus nerve**.
 - 2. It is followed by muscular contractions that increase in amplitude and frequency, starting in the fundus and moving down towards the body and antrum. In the **antrum**, the main role is the grinding of food and propulsion of small amounts (now called chyme) into the duodenum when the pyloric sphincter relaxes.
- Gastric emptying is controlled by two mechanisms: **hormonal feedback** and a **neural reflex called the** enterogastric reflex:
 - 1. In the former, fat in the chyme is the main stimulus for the production of a number of hormones, the most powerful being cholecystokinin, which exerts a negative feedback effect on the stomach, decreasing its motility.
 - 2. The enterogastric reflex is initiated in the duodenal wall, and this further slows stomach emptying and secretion.

Gastric Secretions

- Gastric secretion is divided into three phases: **cephalic** (neural), **gastric** and **intestinal**.
- **Mucus** is produced by all regions of the stomach. It is composed mainly of glycoproteins, water and electrolytes, and acts as a lubricant protecting the surface of the stomach against the powerful digestive properties of acid and pepsin. Bicarbonate ions are secreted into the mucus gel layer and this creates a protective buffer zone against the effects of the low pH secretions. Alkaline mucus is produced in the duodenum and small intestine, where it has a similar function of mucosal protection.
- **Parietal cells** in the stomach are responsible for the production of acid. Acid secretion by these cells is stimulated by two main factors: <u>acetylcholine</u>, released by the vagus nerve, and <u>gastrin</u> from the antrum. Acetylcholine and gastrin act on neuroendocrine cells located close to the parietal cells. On stimulation, these cells release histamine, which has a paracrine action on the parietal cell, stimulating acid production and secretion. Parietal cells secrete acid via an active transport mechanism, the proton pump. Somatostatin, gastric inhibitory peptide and vasoactive intestinal peptide (VIP) inhibit acid secretion.
- **Pepsin** is a <u>proteolytic</u> enzyme produced in its precursor form, pepsinogen, by the <u>peptic cells found</u> in the body and fundus of the stomach. Pepsinogen production is stimulated by <u>acetylcholine</u> from the vagus nerve. The precursor is then converted to its active form, pepsin, by the <u>acid</u> contents of the stomach.

Intrinsic factor is also produced by the <u>parietal cells</u>. It is a glycoprotein that binds to vitamin B12

present in the diet and carries it to the terminal ileum. Here specific receptors for intrinsic factor

exist and the complex is taken up by the mucosa. Intrinsic factor is broken down and vitamin B12 is

then absorbed into the bloodstream.

Peptic Ulcer

Presentation:

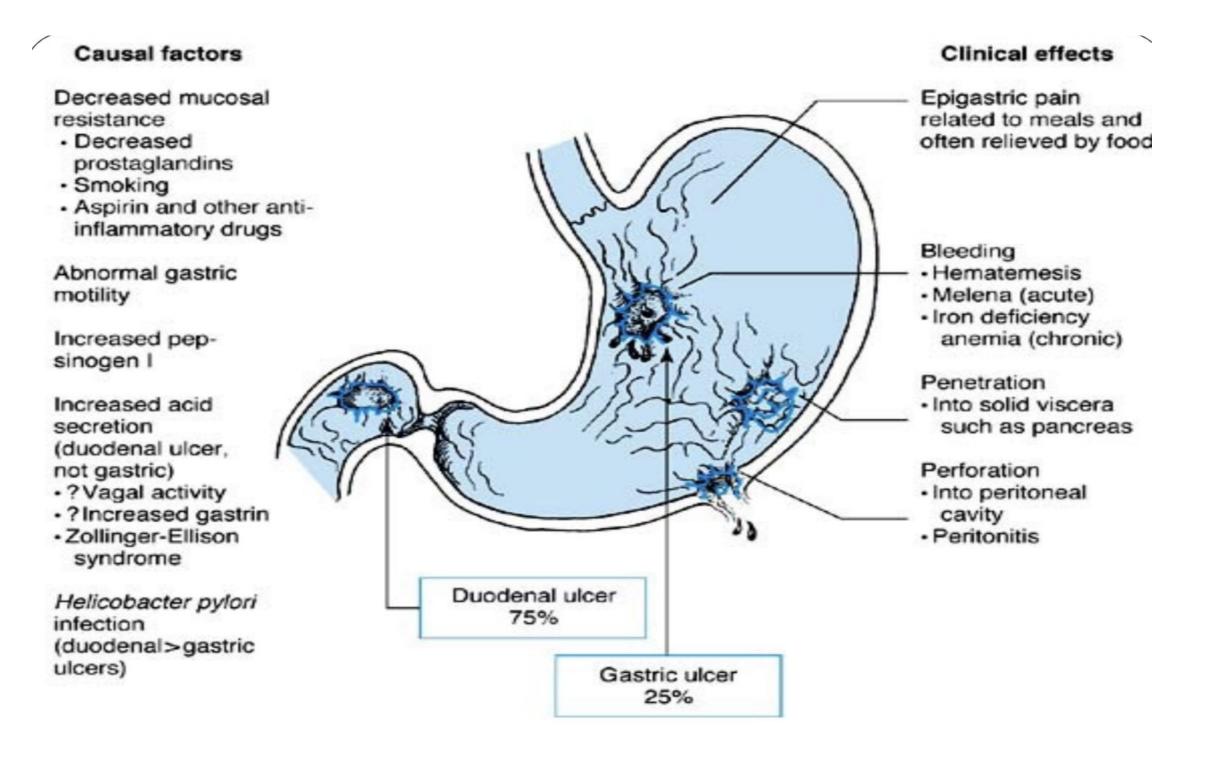
- Pain stabbing in nature:
 - \circ Ulcer disease
 - Perforation
- Bleeding: per-mouth or per-rectum
 - Five causes
- Vomiting:
 - \circ Obstruction

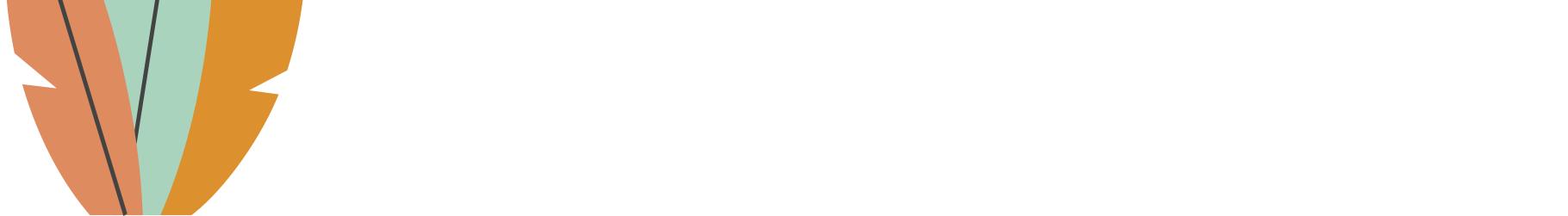
Sites:

- When we say (peptic ulcer) this is related to the acid not to the site bc PU can occurs anywhere in the GI tract
- Esophagus
- Duodenum
- Stomach
- Jejunum after surgical construction of a gastrojejunostomy
- Ileum in relation to ectopic gastric mucosa in Meckel's diverticulum

Definitions (histopathology):

- Abrasions **partial** loss of the superficial layer
- Ulcer <u>complete</u> loss of the superficial layer.
- Perforation loss of <u>all</u> the layers
- Men are affected three times as often as women
- Duodenal ulcers are ten times more common than gastric ulcers in young patients
- In the older age groups the frequency is about equal





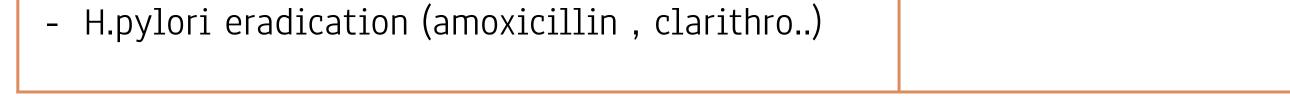
Peptic Ulcer

Duodenal Ulcer	Gastric Ulcer	
Epigastric area, mid-day, noon,night (frequent and persists)	Epigastric area pain <mark>Not persists</mark>	
Relieved by food, so they most likely gain weight	Increased by food, they most likely lose weight	
Common in young – middle age male	Common in 40-60 years male (biopsy to exclude tumor)	
95% in duodenal bulb (2cm) 1st part of the duodenum	95% along lesser curve (where is the Incisura)	
Normal or increased acid secretion 90% principle cause is H pylori (GNCB aeroph)	 the doctor said: "سلها علاقة بكم بس" در الله الله علاقة بكم بس" Types: "اذا واحد سألكم تعرفون ان فيه أنواع لها Type 1 : in incisura angularis & normal acid Type 2: prepyloric, DU & high acid Type 3: antrum due to NSAID Type 4: at GE] 	

Diagnosis:

- 1. Epigastric area pain and tenderness (taking a history further investigation ¹:
- 2. EGD is the best for diagnosing any ulcer (But we don't do it immediately for any patients there are some indication as shown below) its contraindicated in the **perforated** ulcer we do erect abdominal x-ray instead.
- 3. Gastric analysis (basal vs maximal) Used just in certain cases if the pain is persistent and not relieved by treatment and there are multiple ulcers, so we do this test bc it might be another disease like gastrinoma, zollinger-ellison syndrome.
- 4. Gastrin serum level (severe or refractory)
- 5. Contrast meal (show complication) If the hospital doesn't have EGD.
- Contrast swallow: visualizes esophagus only.
- Contrast meal visualizes stomach and esophagus.
- Contrast follow through visualized stomach, esophagus and small bowel
- ★ in elderly patients (above 60) ALWAYS take a biopsy from the gastric ulcer because the old age is an alarming symptom and it could be a malignancy.

Medical Treatment	Surgical Treatment
<pre>Strat with a behavior therapy: smoking cessation coffee, 80% in 6 weeks - H2 antagonists (zantac) - Proton pump inhibitors (omeprazole)</pre>	 Vagotomy Antrectomy and vagotomy Subtotal gastrectomy



¹ Indications for further investigations: (EGD)

1.Persistent pain after 6 weeks of treatment.

2.Recurrent pain after 2 months. .

Complications of Surgery for Peptic Ulcer

- Early Complications (leakage, bleeding, retention)
- Late Complications
- Recurrent ulcer (marginal ulcer, stomal ulcer, anastomotic ulcer) 1.
- Gastrojejunocolic and gastrocolic fistula 2.
- 3. Dumping syndrome. when food moves from the stomach into the small intestine too quickly.
 - a. Early dumping: The rapid emptying of hyperosmolar (mainly carbohydrate) gastric contents into the small intestine leads to the influx of fluid down an osmotic gradient into the bowel lumen leading to hypovolemia \rightarrow (Syncope, loss of consciousness).
 - b. Late dumping: It is a reactive hypoglycemia caused by more rapid absorption of glucose from the upper small intestine. This causes hyperglycemia, which in turn leads to increased insulin production and rebound hypoglycemia \rightarrow (Tachycardia, Flushing, Sweating, Colicky pain, diarrhea)
- 4. Alkaline gastritis
- 5. Anemia.
 - a. Iron deficiency: MCV < 80 fl (microcytic)
 - i. Due to decrease in acid production > decrease iron absorption.
 - **b.** B12 deficiency "megaloblastic or pernicious anemia": MCV > 100 fl (macrocytic)
 - i. Due to loss of IF production from parietal cell in the fundus.
- 6. Postvagotomy diarrhea
- Chronic gastroparesis 7.

Zollinger-Ellison Syndrome (Gastrinoma)

What is it?	Peptic ulcer disease (often severe) in 95% with gastric hypersecretion (there will be Massive ulceration in EGD)
Types	 Single one is malignant if it's only gastrinoma and no other endocrine tumors Multiple is benign (MEN 1) if it's part of multi-endocrine tumor
Diagnosis	 Elevated gastrin serum level: diagnostic it it's more than 500 pg/ml If it's less than 500 pg/ml then there will be other criteria Epigastric tenderness Not responding to medication like H2 blockers and the pain becomes worse with eating EGD Contrast swallow CT scan Somatostatin scan Portal vein blood sample



Ulcer Complications

1. Perforation

- High risk : female, old age, gastric one —
- Anterior ulcers cause perforation, whereas posterior ulcers cause bleeding (due to the Gastroduodenal artery)
- Located anteriorly common in the 1st part of the duodenum. -
- Acute presentation
- Sudden, persistent, Severe, Steroid-related, diffuse abdominal pain
- Presents as ACUTE ABDOMEN CLINICAL SIGNS (RIGIDITY VS GUARDING) —
- **Diagnosis:** erect abdominal X-ray (NEVER DO EGD) X-ray: free air under the diaphragm (85%) & fill 400 cc air by NGT
- **Management**: ABC, then, NPO, IVF, NGT, FC, ABS, pain killer — -Definitive one is surgical repair (Graham patch)
- Factors that causes the perforation:
 - 1. Untreated ulcer.
 - 2. Medication: NSAID, steroids,...

2. Obstruction

- **Symptom** is **Vomiting**, +/- weight loss, not bilish-color —
- **Clinically**: no abdominal distension, gastric splash
- **Diagnosis** : ch. History, non-cooperative pt, smoker
- **Investigation** : abdominal X-ray, EGD, contrast swallow
- **Treatment**: R/O¹ malignancy, possible resection vs bypass -
- How to differentiate between small bowel and large bowel obstruction? —
 - Colon: Obstipation (absence of stool and flatus) then distention then vomiting.
 - Small bowel: vomiting and distention
 - Stomach and duodenum: vomiting alone

1-Rule out.

Surgical Recall:

- What is PUD? Gastric and duodenal ulcers
- What are the possible consequences of PUD?
- Pain, hemorrhage, perforation, obstruction
- What percentage of patients with PUD develops bleeding from the ulcer? $\approx 20\%$
- What is the treatment for H. pylori? Treat H. pylori with MOC or ACO 2-week antibiotic regimens: MOC: Metronidazole, Omeprazole, Clarithromycin (Think: MOCk) or ACO: Ampicillin, Clarithromycin, Omeprazole
- What is the name of the sign with RLQ pain/peritonitis as a result of succus collecting from a perforated peptic ulcer?
 Valentino's sign
- Which is more common overall: gastric or duodenal ulcers? Duodenal ulcers are more than twice as common as gastric ulcers (Think:
 Duodenal = Double rate)

When is surgery indicated? The acronym "I HOP":
 Intractability, Hemorrhage (massive or relentless), Obstruction (gastric

PUD

	outlet obstruction), Perforation
DU	 What is the classic pain response to food intake? Food classically relieves duodenal ulcer pain (Think: Duodenum = Decreased with food) What is the cause? Increased production of gastric acid What are the differential diagnosis? Acute abdomen, pancreatitis, cholecystitis, all causes of UGI bleeding, ZES, gastritis, MI, gastric ulcer, reflux What artery is involved with bleeding duodenal ulcers? Gastroduodenal artery
GU	 What is the classic pain response to food intake? Food classically increases duodenal ulcer pain. What is the cause? Decreased cytoprotection or gastric protection (i.e.decreased bicarbonate/mucous production)
Perforated PU	 What are the symptoms? Acute onset of upper abdominal pain. Which diagnostic tests are indicated? X-ray: free air under diaphragm or in lesser sac in an upright CXR What is a Graham patch? Piece of omentum incorporated into the suture closure of perforation What type of perforated ulcer may present just like acute pancreatitis? Posterior perforated duodenal ulcer into the pancreas

Upper Gastrointestinal Hemorrhage

Presentation:

- ★ Hematemesis (vomiting fresh blood)
- ★ Melana (passage of black stool)
- ★ Hematochezia (passage of fresh blood per anus 'rectum')

Causes of Massive Upper Gastrointestinal Hemorrhage:

	Causes	Relative Incidence
	Peptic Ulcer 1. Duodenal ulcer 2. Gastric ulcer	45% 1. 25% 2. 20%
Common causes	Esophageal varices	20%
	Gastritis	20%
	Mallory-Weiss Syndrome	10%
	Gastric carcinoma	
	Esophagitis	
Uncommon causes	Pancreatitis	5%
	Hemobilia	

Upper gastrointestinal bleeding causes: **in order**

- 1. peptic ulcer
- 2. Portal hypertension.
- 3. Mallory-Weiss syndrome. "Forceful frequent vomiting> tearing> bleeding"
- 4. Malignancy

Management: **ABC**, EGD, surgery.

Mallory-Weiss Syndrome

- Accounting for 10% of UGIB (Upper Gastrointestinal Bleeding)
- 1-4 cm longitudinal tear in gastric mucosa at EG]
- Presentation: forceful vomiting followed by severe retching and bloody vomitus
- **Diagnosis:** EGD (esophagogastroduodenoscopy)
- Management: 90% bleeding stops spontaneously by cold gastric wash, EGD-cautery & surgery

Stress Ulcer	Due to shock & sepsis also called erosive esophagitis may be presented with history of trauma couple days ago that lead to shock and ulcer
Curling's ulcers	Due to burns
Cushing's Ulcer	Due to CNS tumor, injury (more to perforates & high acid production)
Acute Hemorrhagic	This disorder may share some causative factors with the above

Acute Hemormagic

Gastritis

conditions, but the natural history is different and the response to treatment considerably better. Most of these patients can be controlled medically.

Same treatment as before, ABC \rightarrow history & examination \rightarrow EGD \rightarrow a. Bandage & ligate the bleeding or b. Angiogram

Surgical Recall:

- What is mallory weiss syndrome? Post-retching, post emesis longitudinal tear (submucosa and mucosa) of the stomach near the GE junction.
- What are the causes of a tear? Increased gastric pressure, often aggravated by hiatal hernia
- What are the risk factors? Retching, alcoholism (50%), >50% of patients have hiatal hernia
- What are the symptoms? Epigastric pain, thoracic substernal pain, emesis, hematemesis
- What percentage of patients will have hematemesis? 85%
- How is the diagnosis made? EGD

Gastric Polyps

- \star Presentation is mainly Incidental finding , rarely by anemia
- **Types** : (Histopathology)
 - Hyperplastic (benign)
 - Adenomatous (premalignant) most serious you have to follow up with patient
 - Inflammatory (benign)
 - Hamartomatous (benign)
- ★ Affecting distal stomach
- **Diagnosis:** EGD (Esophagogastroduodenoscopy)
- Rule out malignancy

Gastric Leiomyomas (GIST)

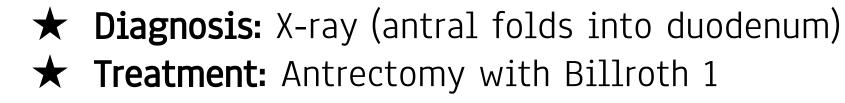
- \star GIST (GastroIntestinal Stromal Tumor) any tumor related to the fibrous tissue of the organ
- ★ Common submucosal growth (normal mucosa, single, isolated.)
- **Presentation:** Asymptomatic & massive bleeding (it's found incidentally or it might \star cause massive bleeding.)
- **Diagnosis:** EGD & CT Scan (Never take a biopsy unless the shape of the protrusion is irregular, or multiple leiomyomas, or you found multi spread in the CT scan)
- **Treatment:** Surgical wide excision

Menetrier's disease

- Giant hypertrophy of the gastric folds (gastric rugae) \star
- Present with hypoproteinemia \star
- **Symptoms:** Edema, diarrhea, weight loss \star
- **Treatment:** atropine, omeprazole, H,pylori eradication, rarely gastrectomy is done \star

Prolapse of the Gastric Mucosa

- \star Occasionally accompanies small gastric ulcer
- **Presentation:** Vomiting and abdominal pain



Gastric Volvulus

Its longitudinal axis (organo-axial volvulus):

- More common
- The patient can't breath
- cause: laxity of the ligaments i.e murphan syndrome
- Associated with HH (hiatal hernia)

Line drawn from the mid lesser to the mid greater curvature (mesenteroaxial volvulus) **Present with:**

- 1. Severe abdominal pain
- 2. **Brochardt"s triad:** Vomiting followed by retching and then **inability to vomit** + Epigastric distention + Inability to pass a nasogastric tube

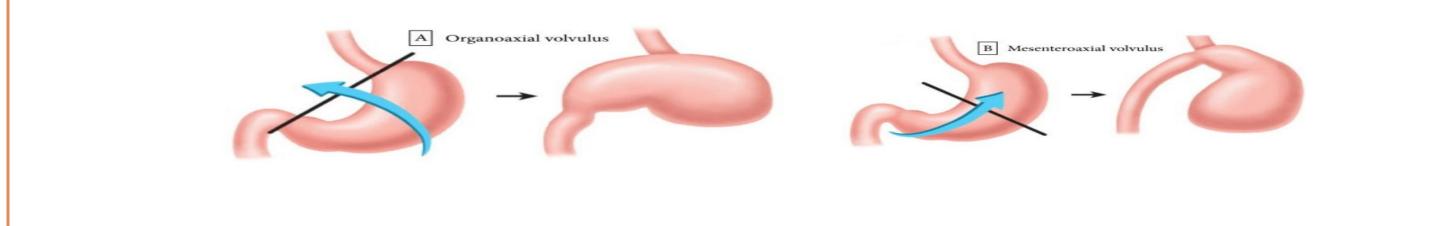
Types of Gastric Volvulus

Mesenteroaxial volvulus

(line drawn from the mid lesser curvature to the mid greater curvature)

Organoaxial volvulus

The stomach closed on two sides



Diverticula

Outpouching of: 1.all the layers (true diverticula). 2.mucosa only (false diverticula)

Gastric Diverticula	Duodenal Diverticula
 Uncommon Asymptomatic Weight loss, diarrhea Diagnosis/ EGD, X-ray 	 20% OF POPULATION Asymptomatic 90% medial aspect of the duodenum Rare before 40 years of age
 Rx:Surgery Found anywhere but mainly in the 	 Most are solitary and 2.5 cm periampullary of vater





Bezoar

 \blacktriangleright It is a Concretions formed in the stomach

★ Types:

- \circ Trichobezoars: formed from hair
- Phytobezoars: vegetable
- ★ Presentation: Obstruction and vomiting
- **Diagnosis:** EGD (esophagogastroduodenoscopy) & X-RAY
- ★ Management: Surgical removal

Benign Duodenal Tumors

- ★ Brunner's gland adenomas
- \star Carcinoid tumors
- ★ Heterotopic gastric mucosa
- ★ Villous adenomas

Regional Enteritis of the Stomach & Duodenum

- ★ Food poisoning
- ★ Pain & diarrhea
- \star Clinical diagnosis
- \star Observation

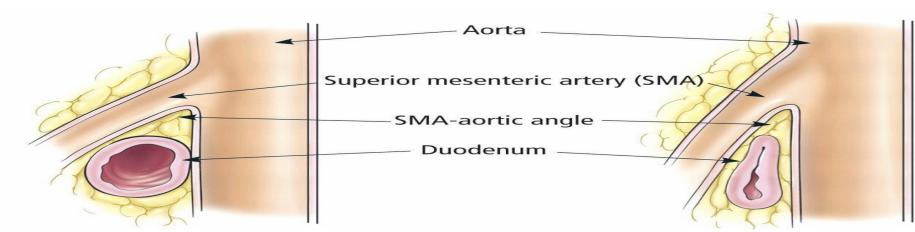
Superior Mesenteric Artery Obstruction of the Duodenum

Characteristics

Diagnosis

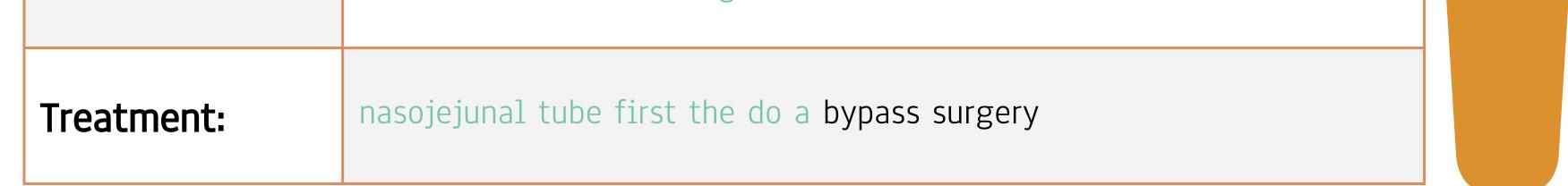
• Obstruction of the <u>third portion</u> of the duodenum (mcq)

- Compression SMA and Aorta
- Appears after rapid weight loss following injury
- Distance between two vessels is 10-20 mm, the angle between them in this syndrome is less than 45 degree.
- History you will find sudden severe loss of wight (car accident, in ICU, CVA) and they feed him/her less than the requirement



Presentation: Proximal bowel obstruction symptoms and signs

- clinical: by history
 - X-ray
 - CT-Scan to look at angle between Aorta & SMA



Summary

Peptic Ulcer

	Duodenal Ulcer	Gastric Ulcer
Symptoms	Epigastric pain Relieved by food, weight gain Common in young - middle age male 1st part of duodenum	Epigastric pain Increase by food, weight loss Common in old males incisura angularis
Diagnosis	 from history start PPI treatment directly for 6w. if symptoms persist or relapse or hx of atypical symptoms > EGD (best diagnostic test) - the only exception is Perforation > X-ray if scope found massive ulcerations > Gastrin level test 	
Treatment	Empirical: Lifestyle changes: stop smoking chocolate high fat food <u>Medical</u> : H2 blockers, Proton pump inhibitors, H. Pylori eradication <u>Surgical</u> : Vagotomy, Antrectomy and vagotomy, Subtotal gastrectomy You never do surgical treatment due its complication. Mainly Dumping syndrome (abdominal cramping after eating, hypoglycemia and tachycardia) and i ron deficiency Anemia	
Complications	Perforation Symptoms: Sudden, persistent, Severe, Steroid-related, diffuse abdominal pain Examination: rigidity & Guarding Diagnosis: erect X-ray (air under diaphragm) Management: ABC, > NPO, IVF, NGT, FC, Painkiller > Graham patch	Obstruction <u>Symptoms</u> : Vomiting, +/- weight loss, not bilish color hx of non-cooperative, smoker <u>Examination</u> : gastric splash <u>Diagnosis</u> : x-ray, EGD <u>Management</u> : malignancy, possible resection vs bypass

Summary

Zollinger Ellison Syndrome (Gastrinoma of G cells)

Presentation	Persistent pain not responding to medication,
Diagnosis	 1- EGD → multiple ulceration, 2- Serum GASTRIN LEVEL (MORE THAN 500 mg/ml) 3- CT antrum to localize gastrinoma
Notes	Gastrinoma only is Malignant, Multiple endocrine Neoplasms is benign (MEN 1)

Gastric Volvulus

(the stomach closes on one or both sides)

Presentation

Severe abdominal pain with

Notes	Gastric Volvulus is an Emergency. If > 6h it may lead to ischemia
	Vomiting followed by retching and then inability to vomit + Epigastric distention + Inability to pass a nasogastric tube
	Brochardt''s triad:

Superior mesenteric artery obstruction of the duodenum

Definition	Obstruction of the third portion of the duodenum (mcq)
Notes	Appears with hx of sudden rapid weight loss



Quiz

- 1. Which one of the following is the most common cause of epigastric pain in young male patients?
 - a. Gastric ulcer
 - b. Duodenal ulcer
 - c. Gallstones
 - d. Bezoar
- 2. A 60y old male presented with localized epigastric pain. Endoscopy was done and we found a gastric ulcer, What's the next step in managing this patient?
 - a. PPI
 - b. Gastrin serum level
 - c. X-ray
 - d. Biopsy
- 3. A 40y old patient who had chronic epigastric pain for the last 4 weeks, presented to the emergency department with a sudden severe diffuse abdominal pain. Examination found rigidity on the abdomen. What's the next step in managing this patient after ABC?
 - a. EGD
 - b. X-rayc. Graham patch
 - d. PPI
- 4. Superior mesenteric artery obstruction of the duodenum affect which part?
 - a. First part
 - b. Second part
 - c. Third pard
 - d. Fourth part
- 5. Which one of the following describe Dumping syndrome?
 - a. A complication of untreated peptic ulcer
 - b. A complication of surgeries of peptic ulcer
 - c. Related to gastric volvulus
 - d. Happens after rapid weight loss with superior mesenteric artery obstruction of the duodenum
- 6. Which one of the following is the best investigation for Gastric Leiomyomas?
 - a. EGD & Biopsy
 - b. CT scan & Biopsy
 - c. EGD & CT scan
 - d. Only biopsy

Answers: 1. B 2. D

3. B 4. C 5. B 6. C