



Benign gastric and duodenal diseases

Objectives:

To know the following about the mentioned gastric and duodenal diseases:

- Definition
- Presentation
- Diagnosis
- Treatment

Resources:

- Davidson's.
- 436 doctors slides.
- Surgical recall.
- 435' team work

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COLOR INDEX:

NOTES , IMPORTANT , EXTRA , DAVIDSON'S

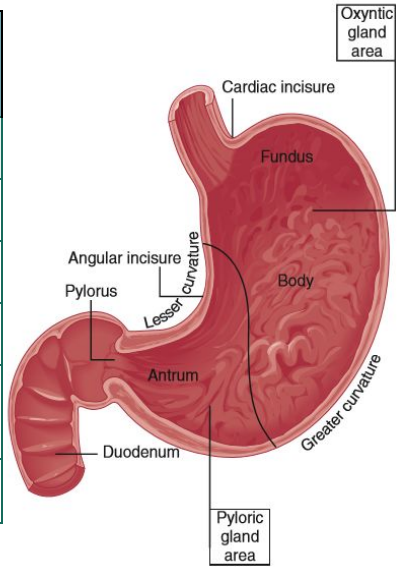
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Basic review(anatomy): First 3 slides are only recall

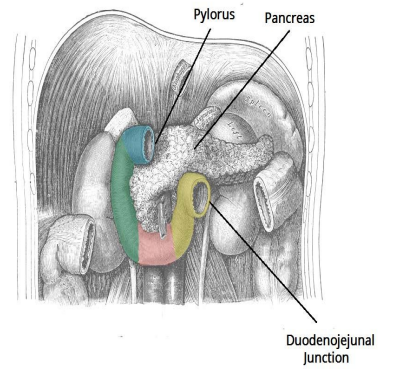
Stomach	
subdivided into 5 parts (each part has secretory and nonsecretory function)	
Part	Notes
Cardia	Site for Barrett's disease
Fundus	Secretory: parietal cell and Neurogenic: hunger feeling
Body	Secretory: parietal cell
Antrum	Site for surgical ulcer treatment: by cutdown the acid secretion (site for gastrin) Even if the ulcer in the duodenum.
Pylorus	Site for Dumping Syndrome (food goes immediately to SI)



Duodenum parts : 25 cm long and subdivided into 4 parts			
Part	Name	Level	Common site for
First	Upper	1st LV	Ulcer
Second	Vertical	2nd LV	Diverticulum
Third	Horizontal	3rd LV	Superior Mesenteric Artery Syndrome
Fourth	Ascending	3rd LV	-

Parts of the Duodenum

- Superior
- Descending
- Inferior
- Ascending

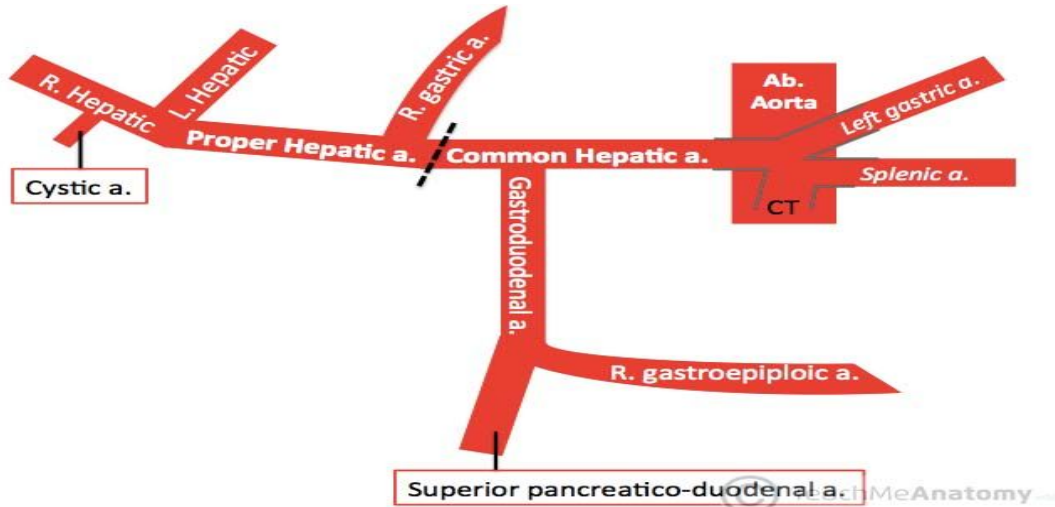


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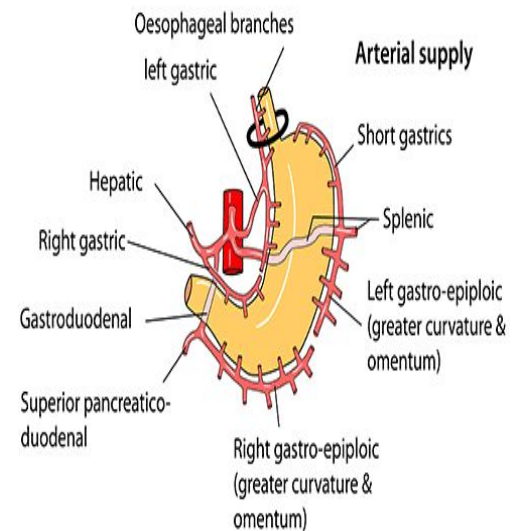
The 1st part of Duodenum: (Relations)

- Anteriorly: The quadrate lobe of the liver and the gallbladder .
- Posteriorly: The lesser sac (first inch only), the gastroduodenal artery (that's why Posterior ulcer bleed), the bile duct and portal vein, and the inferior vena.
- Superiorly: The entrance into the lesser sac (the epiploic foramen)
- Inferiorly: The head of the pancreas

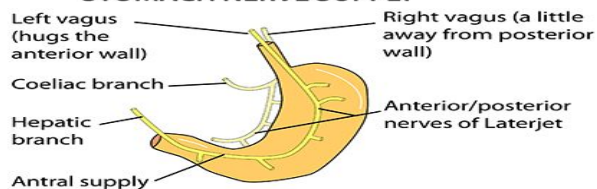
Basic review(anatomy):



Blood supplies		
Artery	Branch	Supply
Celiac trunk	Left gastric	Esophageal branches
Splenic a.	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
Common hepatic a. Gastroduodenal a.	Right gastroepiploic	Supplies the greater curvature of the stomach. Found between the layer of greater omentum, which is also supplies.
Gastroduodenal a.	Superiore pancreaticoduodenal	Divides into an anterior and posterior branch, which supplies the head of the pancreas.



STOMACH NERVE SUPPLY



Vagus nerves are 80% sensory. 20% motor for increasing motility, opening pylorus & initiating secretions

Sympathetics
Greater splanchnic nerves (T5-9) for decreasing motility, vasoconstriction, closing pylorus & sensation



Basic review(physiology of the stomach):

Source of this part is Davidson's

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 cholecystikinin, 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:

Classically, gastric secretion has been divided into three phases:

1. **Cephalic (neural) phase.** Signals arise in the central cortex or appetite centres, triggered by the sight, smell, taste and thought of food, and travel down the vagus nerves to the stomach.
2. **Gastric phase.** Food (in particular protein digestion products) causes the release of acid, this release controlled by a negative feedback mechanism dependent upon the pH of the stomach. The gastric phase accounts for the greatest part of daily secretion, approximately 1.5 litres
3. **Intestinal phase.** The presence of food in the duodenum triggers the release of a number of hormones, including duodenal gastrin. These exert a positive feedback effect on the stomach, causing a small increase in gastric secretion.

- **Mucus** is produced by all regions of the stomach. It is composed mainly of glycoproteins, water and electrolytes, and serves two important functions. It acts as a lubricant, and it protects 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.
- **The parietal cells in the stomach are responsible for the production of acid. Acid secretion by these cells is stimulated by two main factors: acetylcholine, released by the vagus nerve, gastrin from the antrum, and direct contact.** 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 inhibit acid secretion.
- **Pepsin** is a proteolytic enzyme produced in its precursor form, pepsinogen, by the peptic cells found in the body and fundus of the stomach. Pepsinogen production is stimulated by acetylcholine from the vagus nerve. The precursor is then converted to its active form, pepsin, by the acid contents of the stomach.



- **Intrinsic factor** is also produced by the parietal cells. 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

زمان اول ما اكتشفوا الالسر كان على بالهم ان السبب الوحيد له هو زيادة الاسديتي فقالوا خن نسمي الالسر الي يجي بالجى اي (بيبتك ألسر) بيببتك يعني اسيد! بس حديثا اكتشفوا انه مو بس الاسديتي العاليه تسبب ألسر بل حتى القاعدية العاليه ممكن تسببه! بس للحين ما زالوا يستخدمون التسمية القديمة (peptic ulcer) الي ممكن تكون misleading في بعض الأحيان!!! anything related to acid could be peptic ulcer: in esophagus, jejunum, Duodenum, stomach

General Considerations:

- Laceration Vs Ulcer: laceration is when you lose part of the superficial layer of the organ (e.g. endothelium in the GIT). ulcer when you lose the whole superficial layer. Perforation is when u lose all layers.
- **Sites of Peptic ulcer:**
 - Esophagus
 - **Stomach** (Will be covered in this lecture)
 - **Duodenum** (Will be covered in this lecture)
 - Jejunum (following a gastrojejunostomy)
 - Ileum (in relation to ectopic gastric mucosa in Meckel's diverticulum)

(Most common cause of epigastric pain related to the stomach and the duodenum)
- **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**

Clinical presentation:

Pain - epigastrium (well localized)	Bleeding - per-mouth or per-rectum	Vomiting - due to obstruction
perforation		obstruction



Duodenal Ulcer vs Gastric Ulcer:

	Duodenal Ulcer	Gastric Ulcer
Clinical features	<ol style="list-style-type: none"> Well localized epigastric pain (mid-day, noon and night), Daily cycle of the pain is often characteristic. Pain when hungry, and is relieved by food (increase in weight). If you see a patient with epigastric pain and you think it is ulcer, it is commonly Duodenal ulcer especially if the patient is young (<40 years) 	<ol style="list-style-type: none"> Epigastric pain The pain occur during eating (produce more acid) (decrease in weight) <p>VERY IMP: Gastric ulcer may develop into malignancy much more often than duodenal ulcers</p>
Age	Common in young and middle-aged males	Common in 40-60 years male
Site	95% occur in duodenum bulb (2 cm), (the 1st part of duodenum)	95% occur along lesser curve , (where is the incisura angularis) ¹ . See the 1st page for a pic :)
Note	<ul style="list-style-type: none"> Normal or increased acid secretion 90% caused by helicobacter pylori² very imp, very common exam question if it is on the anterior then it can perforate, but if it is on the posterior it can cause bleeding (because the gastroduodenal artery is on the posterior). 	<p>Types:</p> <ul style="list-style-type: none"> Type1: in incisura angularis & normal acid (the commonest) Type 2: ulcers are located close to the pylorus and occur in association with duodenal ulcers & high acid (most common) (acid related) Type3: in antrum, due to chronic use of NSAIDs Type4: At the gastroesophageal junction
Scenario	A patient comes to you complaining of epigastric pain. he is 25 years old , the pain is in the epigastric region, when he eats it relieved and when he doesn't eat it gets worse, and it's cyclic (when you wake up you are hungry so you will have the pain after breakfast it will go away, in afternoon you are hungry so you will have the pain after lunch it will goes away, in the evening same thing) so because the pain relieved by eating the patient might tell you he gained weight . This is Duodenal ulcer start to treat empirical for 4 weeks	Always biopsy a gastric ulcer – some will be malignant *Elderly comes complaining of epigastric pain which is worsen by food → most likely gastric ulcer. U should do EGD to know the site of ulcer, if it in the stomach u should take biopsy to exclude malignancy.

¹ The most common site of gastric ulcer is incisura angularis, the 2nd most common site is Pylorus.

² H.pylori is GNCB (gram negative, curve bacillus) aerophilic.



Diagnosis: (easy to catch from history, Then based on the history you give the treatment. After weeks some of them come back with the same pain this is when you need to investigate)
 Before doing all the test, you must first treat the patient if you suspect duodenal ulcer for at least 6 weeks, if he didn't recover or the ulcer recurred → scope.

- **EGD (Esophagogastroduodenoscopy)** the standard for diagnosing gastric and duodenal ulcer ³ / EGD with biopsy (Biopsy is important in gastric ulcer to exclude malignancy)
- Gastric analysis: can I measure the gastric acid? yes. Is it clinically important? no, nobody use it now, unless it is complicated cases (rare cases)
 - Basal vs maximal (basal: after waking up I put a tube (similar to NG tube) to the stomach and take fluid from the stomach, then I see how many mEq/h. Maximal: the same technique but here I stimulate by giving the patient gastrin)
- Gastrin serum level: Whe pt. Have unusual presentation of the pain.
 - Severe or refractory (done if Zollinger-Ellison syndrome is suspected or the treatment was not effective).
- Contrast meal:
 - Used when either endoscopy is contraindicated or complications of ulcer have occurred
 - 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

Treatment: First control the lifestyle: stop smoking, lose weight, no chocolates, no citrus food

Medical Treatment (80% in 6 weeks)	Surgical Treatment
<ul style="list-style-type: none"> ● H2 antagonist (eg.Zantac) - control acid secretion (Only neurogenic) ● Proton pump inhibitors PPI (eg.Omeprazole) (strong one)"PPI→block the ACTIVE secretion of hydrogen ions into the stomach so it can't combine with chloride ions in the stomach lumen to form gastric acid" ● Antibiotics (eg.Amoxicillin, Clarithro): for H. Pylori eradication 	<p>[it has been limited to patients in whom complications have occurred or to block hormonal stimulation]</p> <ul style="list-style-type: none"> ● Vagotomy (block the neuronal stimulation →which blocks hormonal stimulation) ● Antrectomy⁴ + vagotomy ● Subtotal gastrectomy

Complications Of Surgery For Peptic Ulcer

Early Complications: leakage, bleeding, gastric retention (poor gastric emptying),

Late Complications أهم أهم كومبليكيشن لازم تعرفونها الانيميا والدومينق

1. Recurrent ulcer (marginal ulcer, stomal ulcer ,anastomotic ulcer)
2. Gastrojejunocolic and gastrocolic fistula
3. **Dumping syndrome:** A condition where the ingested food bypasses the stomach too rapidly and enters the small intestine largely undigested. B/c there is no pylorus due to surgery → undigested food will go to

³ unless the pt has a **perforation, why? bc it will bring more acid and water which may cause sepsis or peritonitis**. So if a pt - known to have ulcer - came with severe epigastric pain → x-ray to make sure there's no perforation.

⁴ To take off G cells → decrease gastrin release.

small bowel directly, this food has an osmotic potential(it's hyperosmolar) thus it drags fluid,occurs 1-3 hours after a meal.

- **Early dumping:** It happens when the duodenum, expands too quickly due to the presence of hyperosmolar food from the stomach.leading to hypovolemia.(Syncope, loss of consciousness and lethargy) → takes 4 -6 mins.
- **Late dumping:** is due to hypoglycemia⁵ when the food arrives at the duodenum the pancreas will surprise and secretes a large amount of insulin → (Tachycardia, Flushing, Sweating, Colicky pain, hotness and diarrhea may lead to fainting)→ takes 15 mins.
- Advise the patient to eat less sugar or give him acarbose

4. Alkaline gastritis

5. Anemia (MCV : normal 80-100 fl)

Iron deficiency:

- MCV < 80 fl (microcytic) and low MCH (hypochromic)
- Due to decrease in acid production > decrease iron absorption.

B12 deficiency "megaloblastic or pernicious anemia":

- MCV > 100 fl (macrocytic) and hypochromic
- Due to loss of IF production from parietal cell in the fundus.

6. Postvagotomy diarrhea

7. Chronic gastroparesis

8. Pyloric obstruction/stenosis


Ulcer Complications:(Perforation,obstruction & Upper GI bleeding)

	Perforation ER	Obstruction
SYMPTOMS	<ul style="list-style-type: none"> - Acute onset of severe unremitting epigastric pain - Sudden, Severe, diffuse abdominal pain - Presents as ACUTE ABDOMEN CLINICAL SIGNS (RIGIDITY not GUARDING). <p>لا يجيك مريض بالطوارئ يقول عنده epigastric pain بدا من ساعة وألم شديد وتقولين هذا صغير ممكن عنده duodenal ulcer وتطولين تاخذين هيبستوري، بيطيح عليك المريض ميت! دائماً duodenal ulcer is chronic not acute</p>	<p>In stomach & duodenal obstruction:</p> <ul style="list-style-type: none"> - Dull epigastric pain & projectile vomiting of large volumes of undigested food matter - "this can happen also if the ulcer become scarred" - Vomiting, +/- weight loss, non bile stained vomiting.- No abdominal distension, gastric splash⁶
LOCATION	<ul style="list-style-type: none"> - Occurs in acute ulcers (duodenal mostly) - Gastric perforation is less common and has a strong association with NSAID use. - On the anterior wall of the duodenum (duodenal ulcer) or stomach - Anterior ulcers cause perforation, whereas posterior ulcers cause bleeding (due to the Gastroduodenal artery that lies behind the 1st part of the duodenum). 	<p>Could be due to stricture formation</p>

⁵ happens due to huge insulin release.

⁶ If gastric outlet obstruction is clinically suspected, the patient's abdomen may be shaken from side to side in an attempt to elicit a 'succussion splash": [video](#)



D A I G N O S I S	<ul style="list-style-type: none"> - Erect abdominal X-ray: will demonstrate free air under the diaphragm (85%) [which means air in the peritoneum indicating that there is perforation of the viscus] and fill 400 cc of air by the Nasogastric tube (NGT) (NEVER do EGD) - In comatose either elevate the bed or lateral (right up) because if left up and there is gas could stomach bubble 	<ul style="list-style-type: none"> - History: (smoking...) - abdominal X-ray you'll; see double bubble - EGD to locate area of obstruction - contrast swallow - Biopsy to rule out cancer
R X	<ul style="list-style-type: none"> - Initial management: ABC , then, NPO (nothing per mouth), IV Fluid, NGT(nasogastric tube), ABS (antibiotics) - Definitive one is surgical repair (Graham patch⁷) 	<ul style="list-style-type: none"> - Medical treatment (must make sure pt is taking their medication even if the pain stops) - Surgical treatment: <ol style="list-style-type: none"> 1. Resection (Remove) and anastomose 2. Bypass

كلمتك الممرضة تقولك المريض عنده **sever sudden epigastric pain, BP: 80/60 HR:120: a** على طول سوي **stabilization** بعدين إذا
 استقر المريض نسوي **CXR** وبنلاقي **air under diaphragm** وقتها بأبدا **management** اللي هم: **IV,NGT,ABS, pain killers, call Anastasia to go to OR and do graham patch**

Upper gastrointestinal hemorrhage

- **The commonest cause of upper GI bleeding is peptic ulcer disease**

The differential diagnosis of upper gastrointestinal bleeding:	
Common causes 95%	Uncommon causes 5%
<ul style="list-style-type: none"> ● Peptic ulcer 45% <ul style="list-style-type: none"> ○ Duodenal ulcer 25% ○ Gastric ulcer 20% ● Esophageal varices Portal hypertension 20% ● Gastritis 20% ● Mallory-Weiss syndrome 10% 	<ul style="list-style-type: none"> ● Gastric carcinoma ● Esophagitis ● Pancreatitis ● Hemobilia ● Duodenal diverticulum

- **Upper gastrointestinal bleeding presents with:**
 - ❖ **haematemesis** (vomiting fresh blood) /or
 - ❖ **Coffee-ground vomitus**(is due to vomiting of blood that has been in the stomach long enough for gastric acid to convert hemoglobin to methemoglobin,it's less severe than hematemesi)
 - ❖ **melaena** (the passage of black tarry stool that has a very characteristic smell).

⁷ Piece of omentum incorporated into the suture closure of perforation.

- ❖ **hematochezia** (the passage of bright-red blood from the rectum) always indicates lower GI bleeding except if there's huge massive peptic ulcer.



- **Management:**

1- Resuscitation (ABC) "always start with ABC"

رجاء رجاء بالاختبار اول شي تقولونه اي بي سي حتى لو المريض ستيبل حتى لو بس نقل دم سووا اي بي سي

A (airway): is the patient speaking? if no put him/her on tube

B (breath): O2 saturation, if it is <95% examine the chest to put chest tube

C (circulation): vital signs (BP + HR) if the patient hypotensive and tachycardia put 2 lines to take blood for type and screen and routine blood + 2L of IV fluid + NGT

2-Short History & Short Physical Examination (DIRECT).

3-COMMON DX

4-Investigations: Blood and EGD

Therapeutic options:

1- Detection and endoscopic treatment (If the cause is an ulcer we can put a clip on it, burn it, use a rubber band or injection of a sclerosing agent to form a clot and stop the bleeding)

2- Surgical management (Less than 10% of patients bleeding from a peptic ulcer require emerg surgery)

3- angiogram

1.Endoscopy 2. Angio 3. Surgery

Bleeding site in duodenal ulcers:

- Perforation occurs usually in the anterior walls ulcer.
- Bleeding more commonly occurs in the posterior ulcer, due to the Gastroduodenal artery that lies behind the 1st part of the duodenum, so when bleeding (hematemesis) is seen, we suspect the ulcer to be in the posterior wall of the 1st part of the duodenum.

Amal comes to the ER vomiting blood. How are you going to manage her?

1- stabilise the patient (ABC)

2- brief history (does she have ulcer? does she take steroids or NSAIDs? (to roll out bleeding ulcer) Does she has liver disease? jaundice? (to roll out portal hypertension cause Esophageal varices)

on clinical examination: look for jaundice, mass, signs of chronic diseases

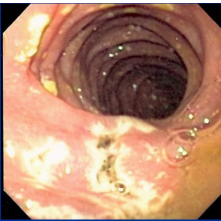
management: blood sample for crossmatch + endoscopy to stop the bleeding (by cauterizing, banding, clipping, inject adrenaline) if the bleeding didn't stop 1- if the patient stable do angio (go inside the vessel and block it) 2- if the patient is not stable go to OR

Other gastric and duodenal diseases

Abnormal presentations of ulcer

Zollinger-ellison syndrome (Gastrinoma)

Hints (persistence pain although taking medications, Multiple ulcerations, increase in gastrin)

<p>What is it:</p>	<p>Zollinger-Ellison syndrome is manifested by gastric acid hypersecretion caused by a gastrin producing tumor (gastrinoma). multiple ulcers, high gastrin The normal pancreas does not contain appreciable amounts of gastrin. Most gastrinomas occur in the submucosa of the duodenum; others are found in the pancreas</p> <ul style="list-style-type: none"> - Gastric hypersecretion + very high no. of ulcers + gastrinoma⁸. 	
<p>Types:</p>	<p>Malignant: If it exists alone “gastrinoma only (in the antrum or the head of the pancreas or gastrinoma triangle)” is usually malignant.</p> <p>Benign: If it exists in association with multiple endocrine neoplasms type 1 (MEN1) (MEN 1) is characterized by a family history of endocrinopathy and the presence of tumors in other glands, especially the parathyroid glands and pituitary G cells, pancreas Patients with MEN 1 usually have multiple gastrinomas. Pheochromocytoma is also cause of Zollinger-ellison syndrome</p>	
<p>Signs & symptoms</p>	<ul style="list-style-type: none"> - Symptoms associated with gastrinoma are principally a result of acid hypersecretion → Peptic ulcer disease (often severe) in 95% with Epigastric tenderness , Not recovering by medication and the pain becomes worse with eating, if you Do EGD, you will find a massive diffuse ulceration) - Ulcer symptoms are often refractory to large doses of antacids or standard doses of H2 blocking agents. - Hemorrhage, perforation, and obstruction are common complications - Some patients with gastrinoma have severe diarrhea from the large amounts of acid entering the duodenum, which can destroy pancreatic lipase and produce steatorrhea 	
<p>Dx:</p>	<p>Laboratory finding:</p> <ul style="list-style-type: none"> - Elevated serum gastrin MORE THAN 500 pg/ml then it is 100% gastrinoma, more than 200 is accepted as well (G cell in antrum secrete gastrin) <p>Image study:</p> <ul style="list-style-type: none"> - CT or MR scan often demonstrates the pancreatic tumors. - Somatostatin-receptor scintigraphy is extremely sensitive for detection of gastrinoma primary and metastatic sites (bc gastrinoma have somatostatin receptor) - EGD - Contrast swallow 	

⁸ G cells tumor.



Treatment:	<ul style="list-style-type: none"> - Medical Treatment : Acid control (massive dose of PPI) - the ideal & appropriate treatment is Surgery: Distal hemi-gastrectomy and ulcer excision <ul style="list-style-type: none"> - Find it and get it out , but If you can't find it (50%). Do gastrectomy
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Mallory-weiss syndrome

What is it:	<p>The lesion consists of a 1- to 4-cm longitudinal tear in the gastric mucosa near the esophagogastric junction; it usually follows a bout of forceful retching.</p> <ul style="list-style-type: none"> - Tear can be in esophageal-gastric junction(EGJ) (most common site), lower esophagus, Cardia and proximal stomach
presentation:	<p>Mallory-Weiss syndrome is responsible for about 10% of cases of acute upper gastrointestinal hemorrhage.</p> <p>Usually caused by severe retching, coughing, or forceful vomiting</p> <ul style="list-style-type: none"> - Typically, the patient first vomits food and gastric contents, This is followed by forceful retching until it cause tearing of oesophagus, proximal of the stomach, gastro-oesophageal junction then bloody vomitus.
Treatment:	<ul style="list-style-type: none"> ● First Manage by ABC then brief history (nothing significant on PE) ● 90% bleeding stop spontaneously by ice-water gastric lavage (cold gastric wash⁹). <ul style="list-style-type: none"> - If it doesn't stop, we perform EGD "to investigate and treat" - If the tear is small, we can burn it (cautery). If not, it will need surgical intervention to repair the tear. ● How control bleeding by scope (cauterize it, band it, clip it, inject or embolize) ● if the patient is not stable go to OR <ul style="list-style-type: none"> ○ Never Cauterize in varices

Recall:

What is mallory - weiss syndrome?
Post-retching, post emesis longitudinal tear (submucosa and mucosa) of the stomach near the GE junction; approximately three fourths are in the stomach

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

What is the "classic" history?
Alcoholic patient after binge drinking— First, vomit food and gastric contents, followed by forceful retching and bloody vomitus

⁹ To induce vasospasm to stop the bleeding.

What is the treatment?

Room temperature water lavage (90% of patients stop bleeding), electrocautery, arterial embolization, or surgery for refractory bleeding

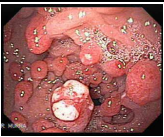
When is surgery indicated?

When medical/ endoscopic treatment fails (>6 u PRBCs infused)


Stress gastroduodenitis, stress, stress ulcer & acute hemorrhagic gastritis:

Stress ulcer:	Also called erosive gastritis it's ulcer due to shock or sepsis. May present as vehicle accident or any trauma couple of days ago that lead to shock > non suspected ulcer formation > Massive upper GI bleeding
Curling's ulcer:	ulcer due to burns acute gastric erosion results as a complication of severe burns, reduction in plasma volume leads to ischemia and cell necrosis (sloughing) of the gastric mucosa.
Cushing' ulcer:	due to the presence of a CNS tumor or injury (more to perforate, high acid production)
Acute Hemorrhagic Gastritis	This disorder may share some causative factors with the above conditions, but the natural history is different and the response to treatment considerably better. Most of these patients can be controlled medically.

Gastric Polyps

What is it:	Gastric polyps are single or multiple benign tumors that occur predominantly in the elderly. Those located in the distal stomach are more apt to cause symptoms. is incidental %90 يقولك الدكتور؟ what is the common presentation of polyp جاوب 90% is incidental finding	
Types:	<ul style="list-style-type: none">- Hyperplastic - treat with Omeprazole- Adenomatous (pre-malignant) - most serious you have to follow up the pt.- Inflammatory- Hamartomatous	
presentation:	mainly incidental finding. sometimes diarrhea and malnutrition rarely, Anemia	
Dx:	Perform EGD to Rule out malignancy (Whenever gastric polyps are discovered, gastric cancer must be ruled out)	
Treatment:	You have to resect the adenomatous type due to its malignant potential.	



Gastric leiomyomas	
What is it:	<p>Benign smooth muscle tumor</p> <p>Leiomyomas are common submucosal growths that are usually asymptomatic but may cause intestinal bleeding.</p> 
presentation:	90% asymptomatic, less than 1% present with massive bleeding
Dx:	<ul style="list-style-type: none"> - after PE refer the patient to EGD and CT scan. When seen typical -> send for CT to staging - Never take biopsy unless the shape of the protrusion is irregular
Management:	<ul style="list-style-type: none"> - by ABC "in case of bleeding" - Surgical wide excision

Diverticula	
<ul style="list-style-type: none"> - diverticula: any organ that has lumen is covered by "from in to out side (mucosa-muscle-serosa)". Bulging out or swelling from all the layers (called true diverticula) if mucosa only (it's called false diverticula) - stomach and duodenum diverticula most likely true diverticula . 	
Gastric diverticula:	Duodenal diverticula:
<ul style="list-style-type: none"> ● Uncommon ● Asymptomatic ● Weight loss, diarrhea ● It may cause anemia ● Diagnosis: EGD, X-Ray ● Rx:Surgery (take out this diverticulum) 	<ul style="list-style-type: none"> ● 20% OF POPULATION ● Asymptomatic - incidental finding ● 90% in the medial aspect of the duodenum ● Rare before 40 years of age ● Most are solitary and 2.5 cm peri-ampullary of vater ● It can cause obstruction, bleeding and inflammation ● If it's asymptomatic, we leave it. If there is superficial cancer, we excise it.

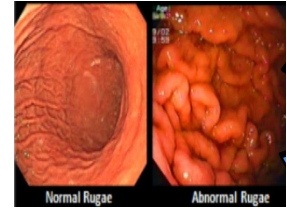


Menetrier's disease

What is it:

acquired, premalignant disease of the stomach characterized by massive gastric folds, excessive mucus production with resultant protein loss

Giant hypertrophy of the gastric rugae¹⁰.



presentation:

Present with hypoproteinemia, Edema, diarrhea, weight loss

Treatment:

- Atropine (to reduce the secretion)
- Omeprazole
- H.pylori eradication
- Rarely, gastrectomy

Prolapse of the gastric mucosa

What is it:

When the mucosa of antrum prolapse to duodenum

Occasionally accompanies small gastric ulcer

presentation:

Vomiting and abdominal pain

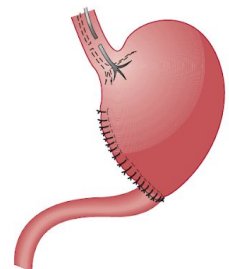
Dx:

X-ray : antral folds into duodenum (double ring on X-ray) [not well defined]

Treatment:

Antrectomy with Billroth 1

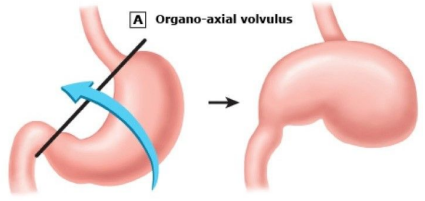
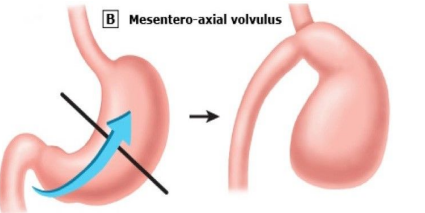
Antrectomy and vagotomy (Billroth I)



¹⁰ Stomach folds (it's 4 mm in width normally, but in menetrier's disease it's larger)



Gastric volvulus “emergency”

What is it:	defined as an abnormal rotation of the stomach of more than 180°, which creates a closed-loop obstruction that can results in ischemia Benign disease, but can be lethal.
types:	<p>organo-axial volvulus (longitudinal axis):¹¹</p> <ul style="list-style-type: none"> - Stomach is closed on two sides (more dangerous) When you introduce NGT it won't pass - More common - Associated with HH (hiatal hernia) <p>لأن المعدة صارت ملفوفة على نفسها وما صار يجيها blood supply فيالتالي ببصير فيه atrophy -</p>  <p>mesenteroaxial volvulus (Transverse):</p> <ul style="list-style-type: none"> - Line drawn from the mid lesser curvature to the mid greater curvature - Associated with vomiting (obstruction) - closed from one side (less dangerous) - the epigastric pain is less severe compare to organoaxial volvulus 
presentation:	Presents with: Severe abdominal (epigastric) pain and Borchardt's triad (Borchardt's triad= Vomiting followed by retching and then inability to vomit + Epigastric distention with severe sudden epigastric pain + Inability to pass a nasogastric tube)المعدة مسكره لاهو الي قادر بطرش ولا تقدر تدخل لمعدته نيوب The patient may present with hiatal hernia or GERD with no compliance with treatment for long time
Treatment	Exploratory laparotomy to untwist, and gastropexy (it is emergency case, go to OR immediately)

¹¹ Total rotation along the same axis → blind organ (most dangerous).



Superior mesenteric artery syndrome

What is it:	<ul style="list-style-type: none"> - Obstruction of the third portion of the duodenum which is compressed by superior mesenteric artery (SMA) (anteriorly) and Aorta (posteriorly)¹². - Normally the angle between SMA and Aorta is 50-60 degree and the distance between the - two vessels where the duodenum passes between them is 10–20 mm. In this syndrome angle is less than 45. - Appears after rapid weight loss following injury (happens due loss of mesenteric fat, typically in the history you will find sudden severe loss of weight (car accident, in ICU, CVA) and you are not feeding him/her right (instead of giving 1200 calories you give 1000)) - Fat is the only thing that lies between the duodenum and the SMA. So when a person is cachexic and chronically ill, the fat will diminish and this will bring the duodenum and SMA closer to each other, leading to the obstruction. 	
presentation:	Proximal bowel obstruction symptoms and signs (vomiting)	
Dx:	CT Scan to look at angle between Aorta & SMA	
Treatment:	Bypass surgery Chronic obstruction may require section of the suspensory ligament and mobilization of the duodenum, ora duodenojejunostomy to bypass the obstruction	

Bezoar

What is it:	scientific name of foreign body in the stomach Retained concretions ¹³ of indigestible foreign material in the stomach (Concretions formed in the stomach)	
Types:	<p>Trichobezoars: formed from hair غالبا مع المرضى النفسيين تجيهم حالة يقطعوا شعورهم ويبلعوها (, why? bc they are on medications that make the stomach lax (Stasis will happen) and cannot move</p>	
	<p>Phytobezoars: vegetable (Indigestible plant material)</p>	
presentation:	Obstruction (vomiting)	
Dx:	EGD, X-RAY	
Treatment:	SURGICAL REMOVAL	

¹² Abdominal aorta give 1st celiac branch then SM branch and go down



Regional enteritis of the stomach & duodenum Skipped by the doctor	
causes:	Food poisoning
presentation:	Abdominal pain and diarrhea
Dx:	Clinical
Treatment:	observation

Benign duodenal tumors ¹⁴ Skipped by the doctor	
Brunner's gland adenomas	-----
Carcinoid tumors:	<p>Neuroendocrine tumors of the duodenum are often endocrinologically active, producing gastrin, somatostatin, or serotonin.</p> <p>These are tumours of neuroendocrine origin that vary Enormously in their malignant potential. The majority encountered are benign, but occasionally malignant carcinoids can behave aggressively.</p>
Heterotopic gastric mucosa:	presenting as multiple small mucosal nodules, is an occasional endoscopic finding of no clinical significance.
Villous adenomas:	of duodenum may give rise to intestinal bleeding or may obstruct the papilla of Vater and cause jaundice

Gastric malignant tumors Skipped by the doctor	
carcinoma	-----
lymphoma	-----
Leiomyomas	-----
GIST:	GastroIntestinal Stromal Tumor
Metastases:	Melanoma, OTHERS

¹⁴ all of them are incidental. sometimes it comes with anemia or diarrhea. go for scope and take biopsy, if it is adenomatous you need to follow up, not adenomatous no need to follow



Gastric Carcinoma Skipped by the doctor	
incidence:	Old male, low vegetable and fruit intake, High starch
presentation:	Pain, Vomiting, Bleeding
Types:	<ul style="list-style-type: none">❖ Adenocarcinoma:<ol style="list-style-type: none">1. Ulcerating carcinoma (25%)2. Polypoid carcinoma (25%)3. Superficial Spreading carcinoma (15%)4. Linitis plastica (10%)5. Advanced carcinoma (35%)❖ Squamous cell tumor.<ul style="list-style-type: none">● Intestinal type vs Diffuse type
diagnosis:	<ul style="list-style-type: none">● Clinical presentation● EGD & BIOPSY● Staging : TNM●● 1- Clinical examination● 2- CT scan Chest, Abdomen, Pelvis● 3- Others (Alkaline pho, Bone scan, PET)● 4- TUMOR MARKER : CEA
treatment:	<ul style="list-style-type: none">➤ Chemotherapy➤ Surgery (Distal, Subtotal, Total)➤ Palliative



summaries:



SUMMARY BOX 13.3

Peptic ulcer disease

- *Helicobacter pylori* is the most important cause – eradicate it
- NSAID medication next commonest cause
- Surgery now only for complications (bleeding and perforation)
- Always biopsy a gastric ulcer – some will be malignant
- If an ulcer fails to heal with medical therapy look for rare causes (i.e. ZE).

DUODENAL ULCER



ESSENTIALS OF DIAGNOSIS

- ▶ Epigastric pain often relieved by food or antacids
- ▶ Epigastric tenderness
- ▶ Normal or increased gastric acid secretion
- ▶ Signs of ulcer disease on upper gastrointestinal x-rays or endoscopy
- ▶ Evidence of *H pylori* infection

ZOLLINGER-ELLISON SYNDROME (GASTRINOMA)



ESSENTIALS OF DIAGNOSIS

- ▶ Peptic ulcer disease (often severe) in 95%
- ▶ Gastric hypersecretion
- ▶ Elevated serum gastrin
- ▶ Non-B islet cell tumor of the pancreas or duodenum

GASTRIC ULCER



ESSENTIALS OF DIAGNOSIS

- ▶ Epigastric pain
- ▶ Ulcer demonstrated by x-ray
- ▶ Acid present on gastric analysis

Summary

Peptic Ulcer

Duodenal ulcer	Gastric ulcer
Epigastric pain	Epigastric pain
Relieved by food, weight gain	Increase by food, weight loss
Common in young – middle age male	Common in 40-60 years male
1 st part of duodenum	incisura angularis

Diagnosis:

- first is history, if history is clear no need for investigation and Start medical treatment for 4 to 6 weeks, if symptoms don't improve or history is unclear investigate.
- endoscope EGD (Best diagnostic test), unless ulcer is perforated If you suspect perforated ulcer do XRAY
- Contrast meal Contrast is done when there is no scope
- Gastrin serum level

Treatment

Empirical: Lifestyle changes: stop smoking chocolate high fat food

Medical Treatment: H2 blockers, Proton pump inhibitors, H. Pylori eradication

Surgical:

Don't do surgery gastrectomy before Increase dose of meds, if pain persists on maximal treatment check for Zollinger Ellison syndrome before surgery (Vagotomy, Antrectomy and vagotomy, Subtotal gastrectomy)

Surgical Complications:

Early Complications (leakage, bleeding, retention)

Late Complications: Dumping syndrome, Anemia (iron deficiency/ Megaloblastic)

Zollinger Ellison Syndrome	
Pathophysiology	Gastrinoma of G cells in antrum which leads to elevated gastrin levels → more acid production → multiple peptic ulcer disease (often severe)
Diagnosis	Epigastric pain on maximal treatment 1- EGD → multiple ulceration, 2- Serum GASTRIN LEVEL IS MORE THAN 500 pg./ml 3- CT antrum to localize gastrinoma 4- if not found on CT do portal vein blood sample.
Treatment	Medical Treatment Surgical Treatment I is best
SMA obstruction of the duodenum	
Cause	Obstruction of the third portion of the duodenum --compression SMA and Aorta. Appears after rapid weight loss following injury
Presentation	vomiting
Diagnosis	CT, angle between aorta and SMA is 10



Questions

1) The most common cause of upper GI bleeding is

- A. Esophageal varices
- B. Peptic ulcer disease
- C. Mallory Weiss syndrome
- D. Gastritis

2) Which ONE of the following statements is true about Mallory Weiss syndrome?

- A. It is caused by H. pylori organism infection
- B. It is a 1-4 cm longitudinal tear in gastric mucosa at EGJ
- C. It causes 80% of upper GI bleeding
- D. 5% of the bleeding stops spontaneously

3) A 24-year-old male presented to the ER with severe epigastric pain for 2 days, the pain was cyclical and relieved by food, what is most likely the diagnosis?

- A. Achalasia
- B. Gastric ulcer
- C. GERD
- D. Duodenal ulcer

4) Which one of these features is seen on X-ray in case of perforated duodenal ulcer?

- A. Air under the diaphragm.
- B. Increase mediastinal width.
- C. Bird's beak appearance.
- D. Flattened diaphragm.

5) All the following are true regarding Brochardt's Triad except:

- A. Patient will present with Vomiting followed by retching
- B. Inability to pass a nasogastric tube
- C. Constipation.
- D. Epigastric distention

6) Which ONE of these is seen in case of gastrinoma:

- A. Gastrin level is more than 500 pg/ml
- B. Gastrin level is more than 600 pg/ml
- C. Gastrin level is more than 300 pg/ml
- D. Gastrin level is more than 150 pg/ml

Answers:

1:B 2:B 3:D 4:A 5:C 6:A