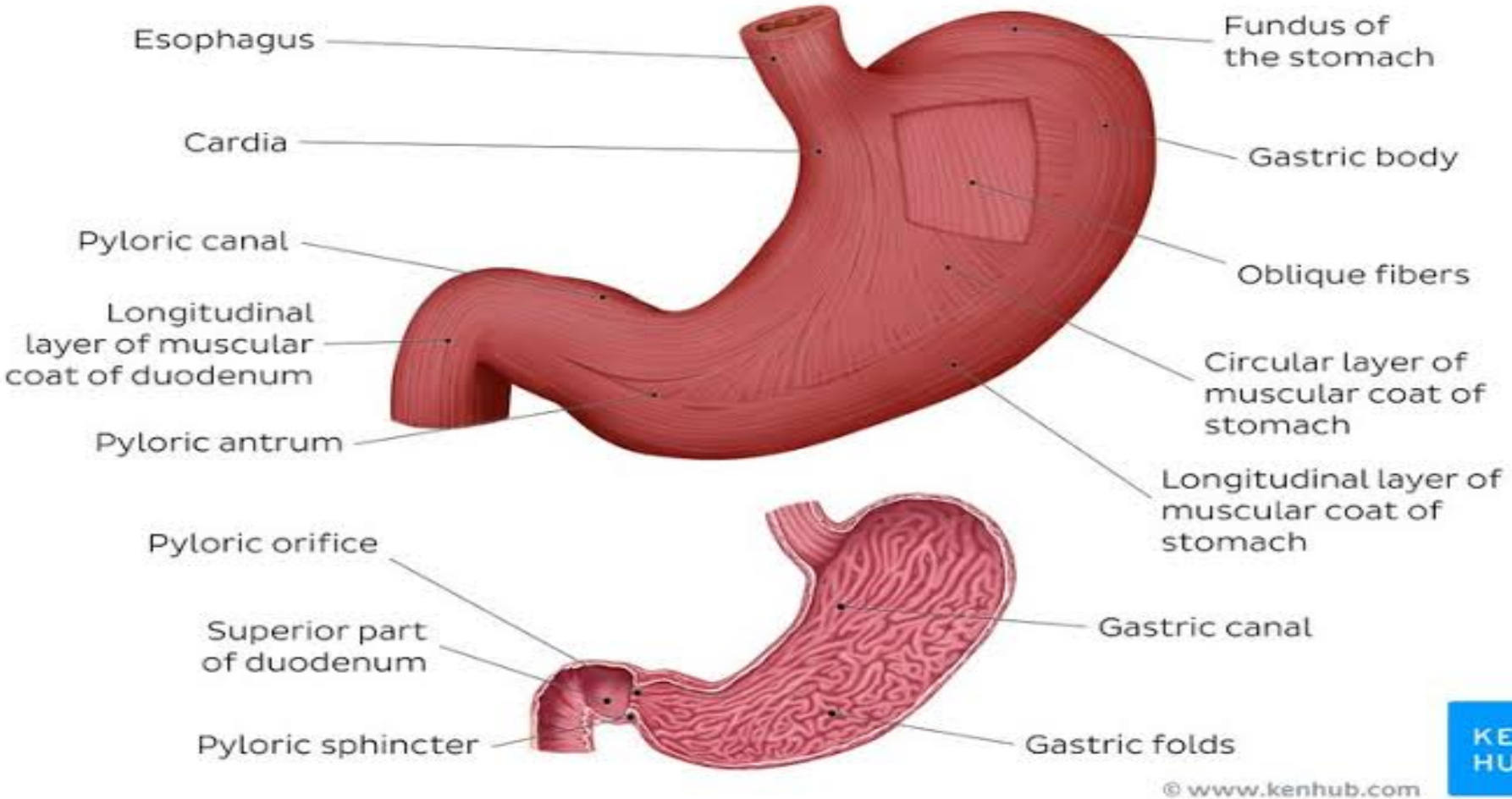
PEPTIC ULCER

vishalraghuvanshi636@gmail.com


Esophagus
Cardia
Pyloric canal
Longitudinal layer of muscular coat of duodenum
Pyloric antrum
Fundus of the stomach
Gastric body
Oblique fibers
Circular layer of muscular coat of stomach
Longitudinal layer of muscular coat of stomach
Pyloric orifice
Superior part of duodenum
Pyloric sphincter
Gastric canal
Gastric folds
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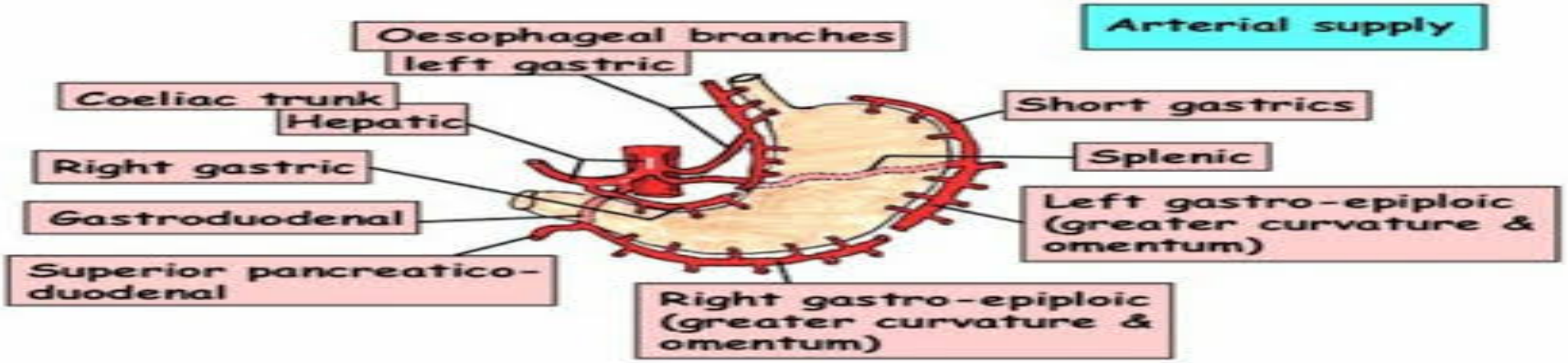
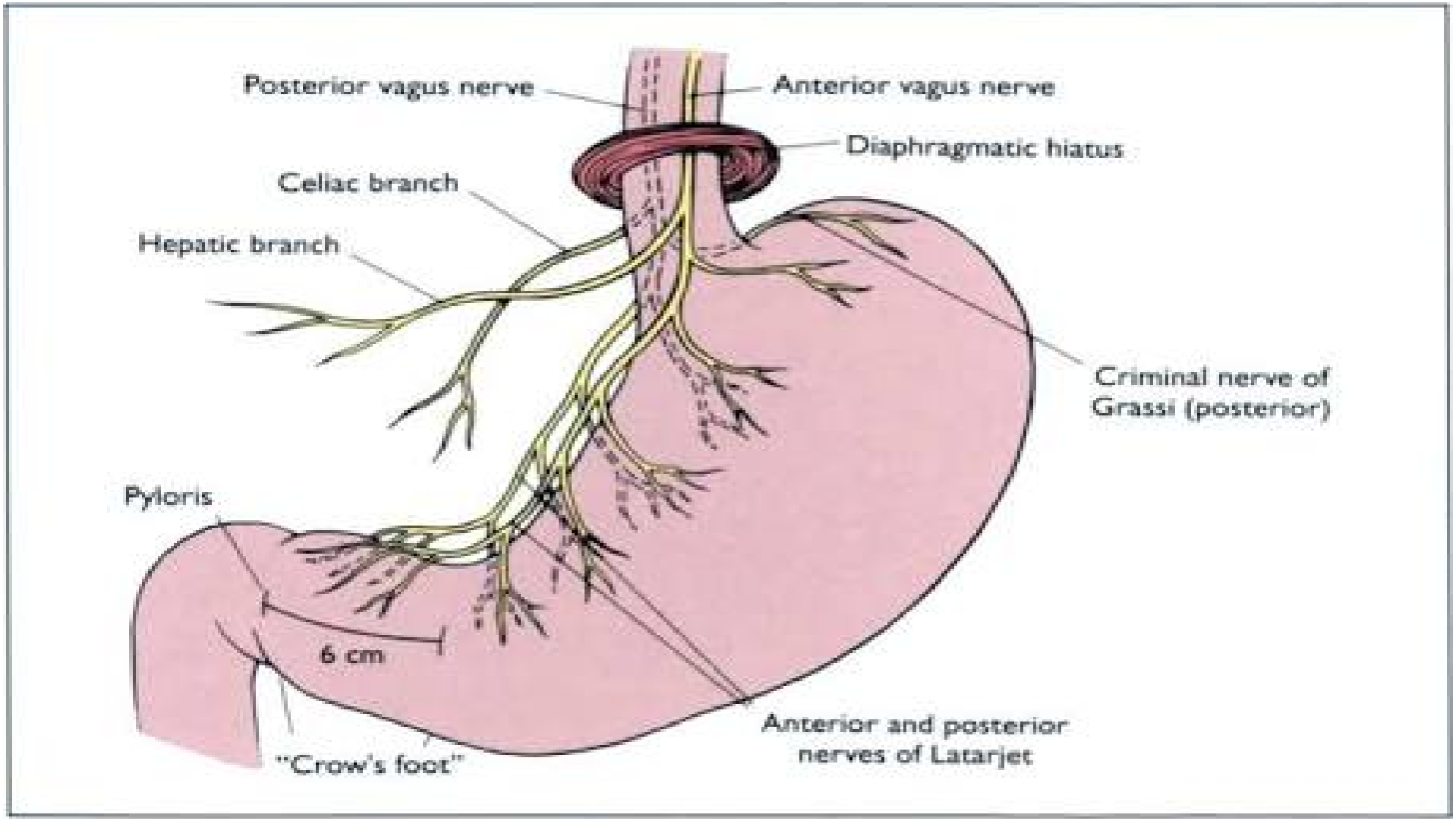
STOMACH - BLOOD SUPPLY & VENOUS DRAINAGE

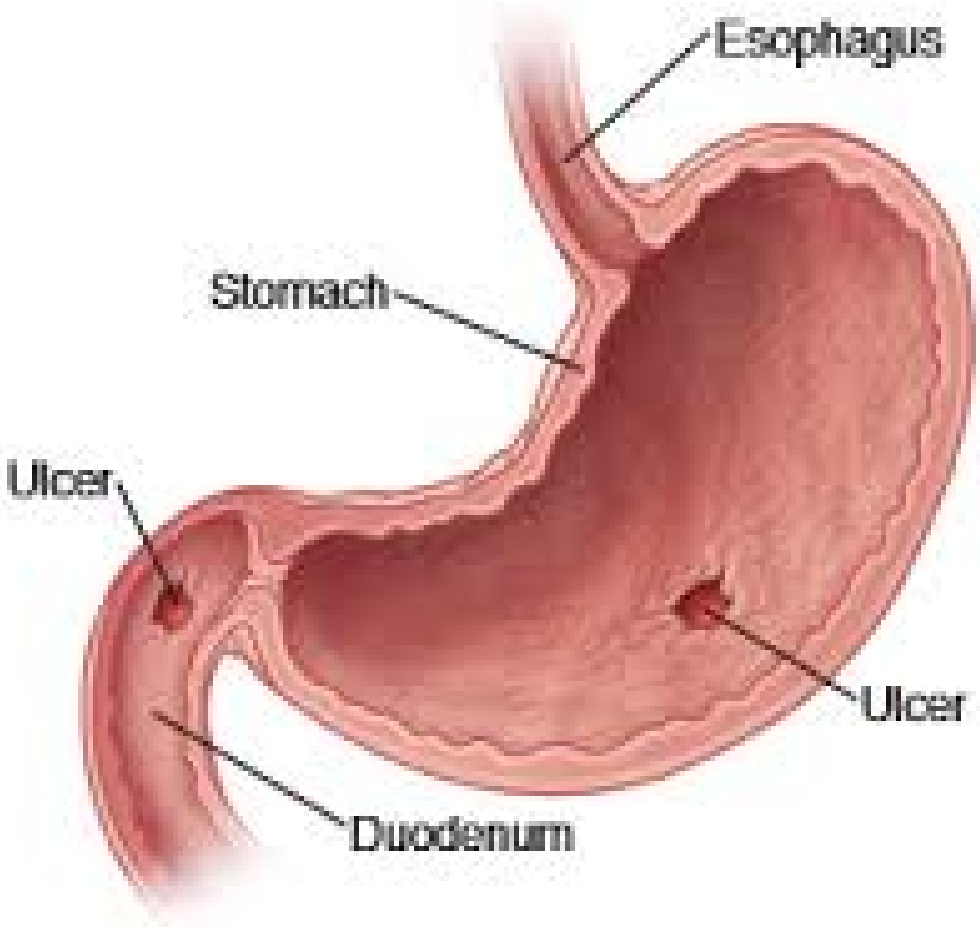


Note : Rt. Gastroepiploic "Vein" drains into "Superior Mesentric"
[In contrast to Rt Gastroepiploic "Artery" which is a branch of "Gastroduodenal" artery]


Posterior vagus nerve
Anterior vagus nerve
Diaphragmatic hiatus
Celiac branch
Hepatic branch
Criminal nerve of Grassi (posterior)
Pyloris
6 cm
"Crow's foot"
Anterior and posterior nerves of Latarjet

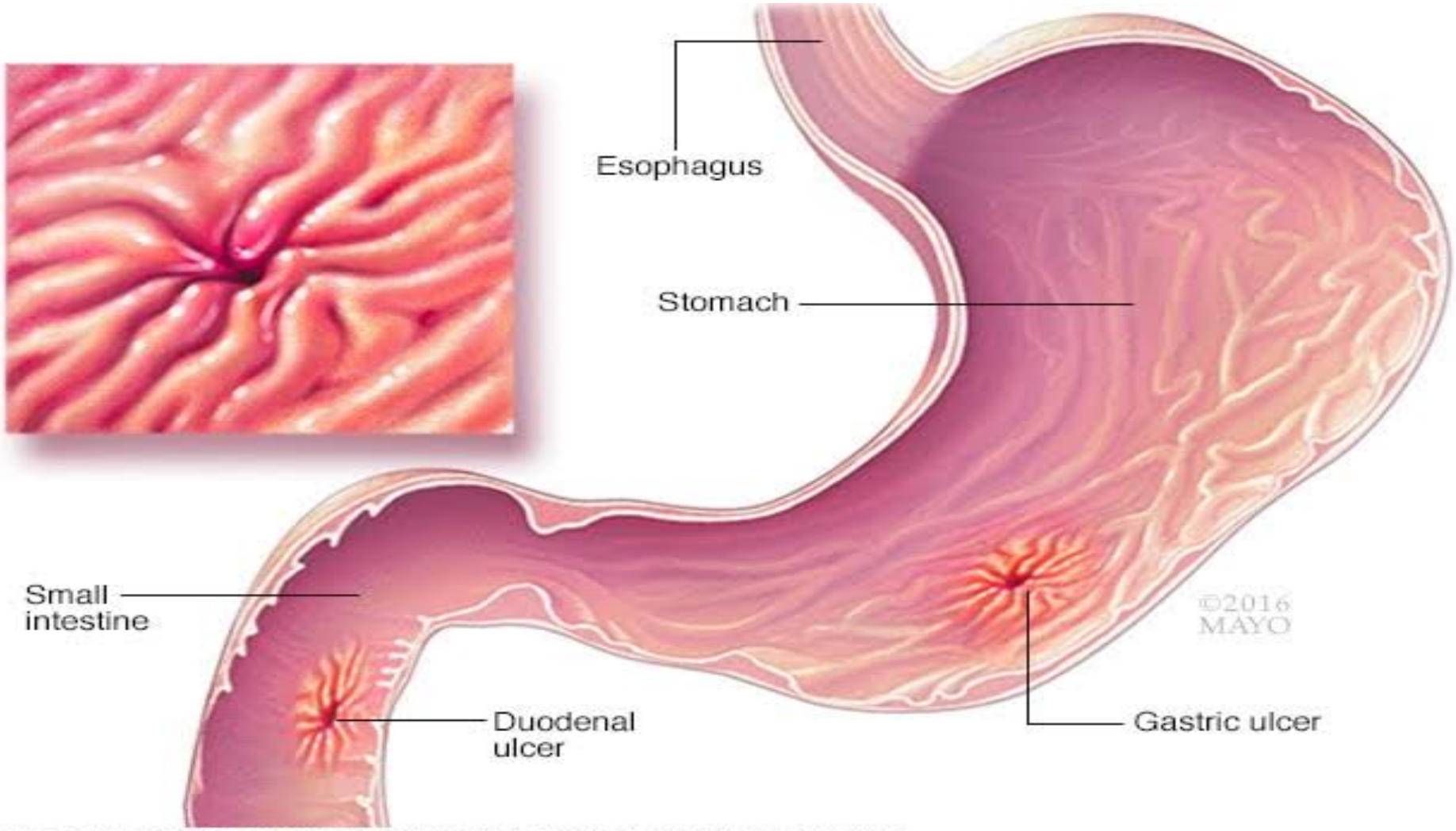
PEPTIC ULCER

DEFINITION:- A lesion in the lining (mucosa) of the digestive tract, typically in the **stomach** or duodenum, caused by the digestive action of pepsin and **stomach** acid.



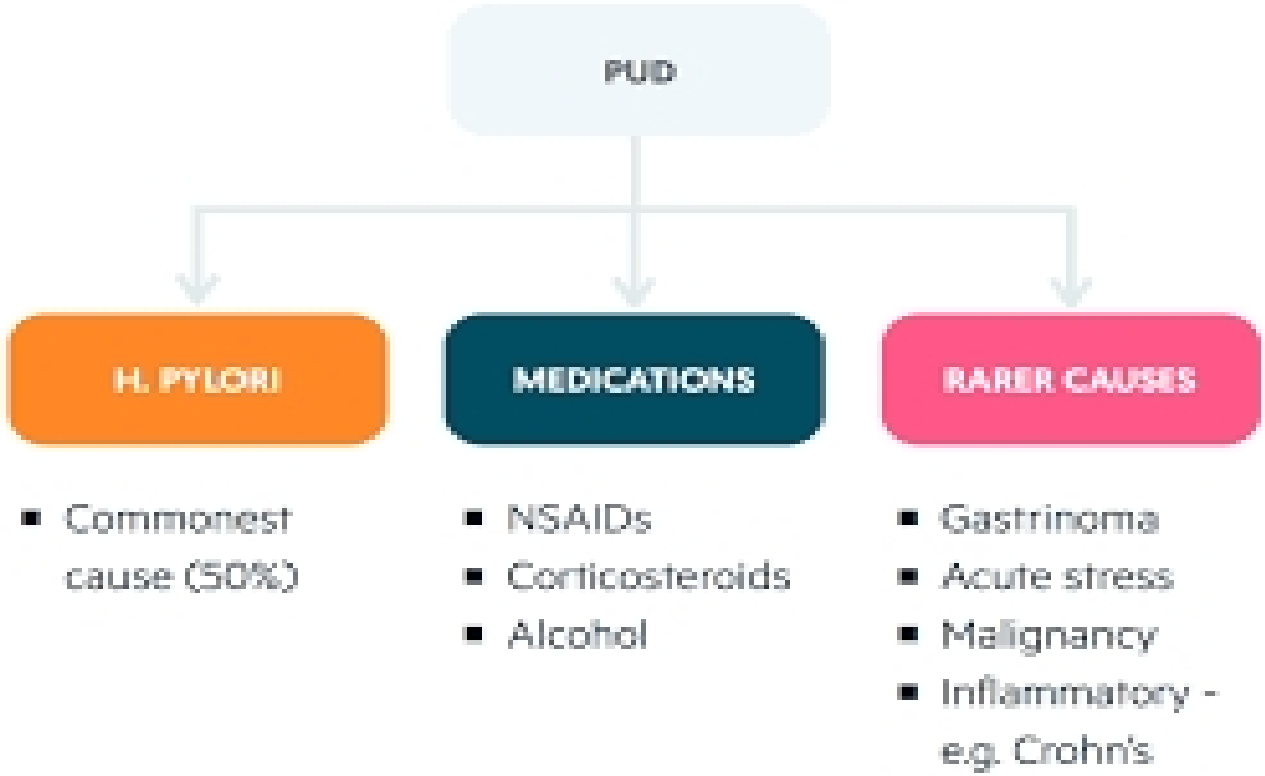
Types

- There are two main sites of peptic ulcer — **gastric** and **duodenal**.
- Rare peptic ulcers may be seen in the
- (i) cardiac end of oesophagus;
- (ii) Meckel's diverticulum (due to presence of ectopic gastric
- mucosa);
- (iii) In any segment of bowel (anastomotic ulcer) which has been surgically anastomosed to the gastric fundus.
- Peptic ulcers may be acute ulcers, which are shallow and multiple and chronic ulcers, which are single, deep and scirrhous



ACUTE PEPTIC ULCER (DUODENAL OR GASTRIC ULCER)

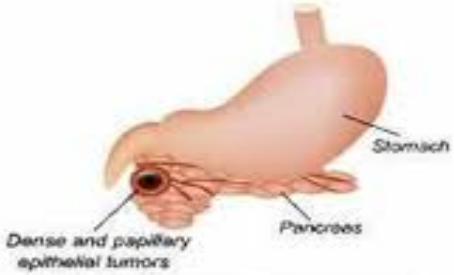
- They are usually multiple erosions due to disruption of the mucosal barrier.
- **Causes**
- Stress,
- Drugs like analgesics,
- Steroids,
- Surgeries.



COMMON CAUSES THAT LEAD TO PEPTIC ULCERS

HELICOBACTER PYLORI BACTERIAL INFECTION

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS



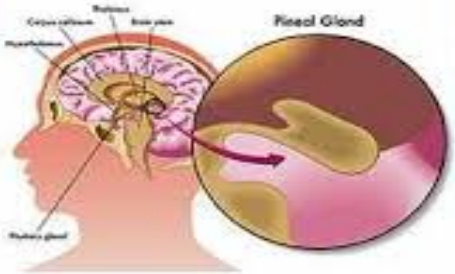
ZOLLINGER-ELLISON SYNDROME

ALCOHOLIC BEVERAGES

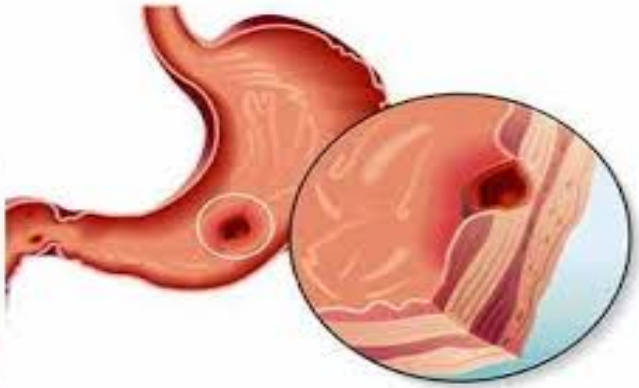
HYPERCALCEMIA

FAMILY HISTORY

EXCESS STRESS



LOW LEVELS OF MELATONIN



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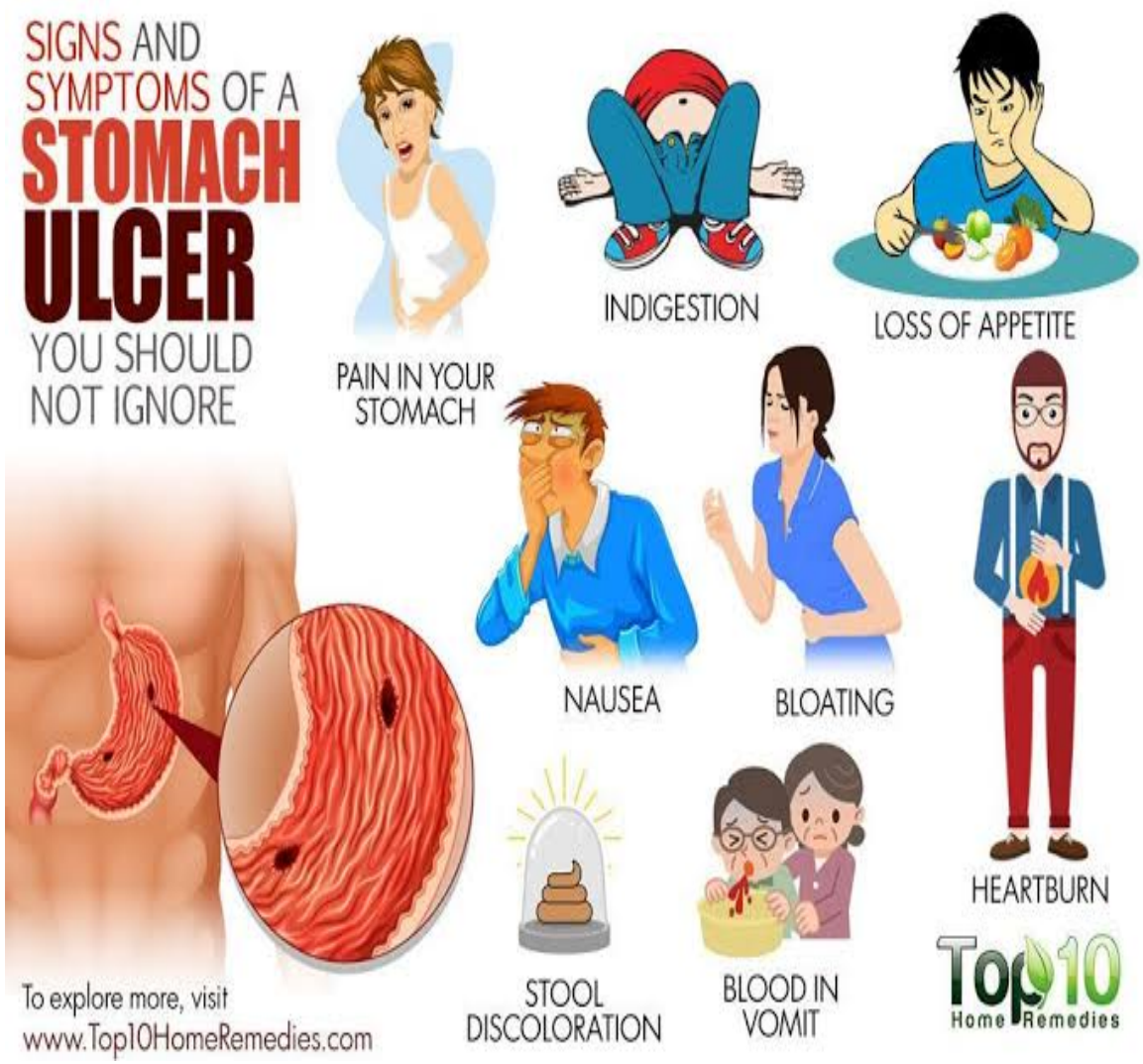


Clinical Features

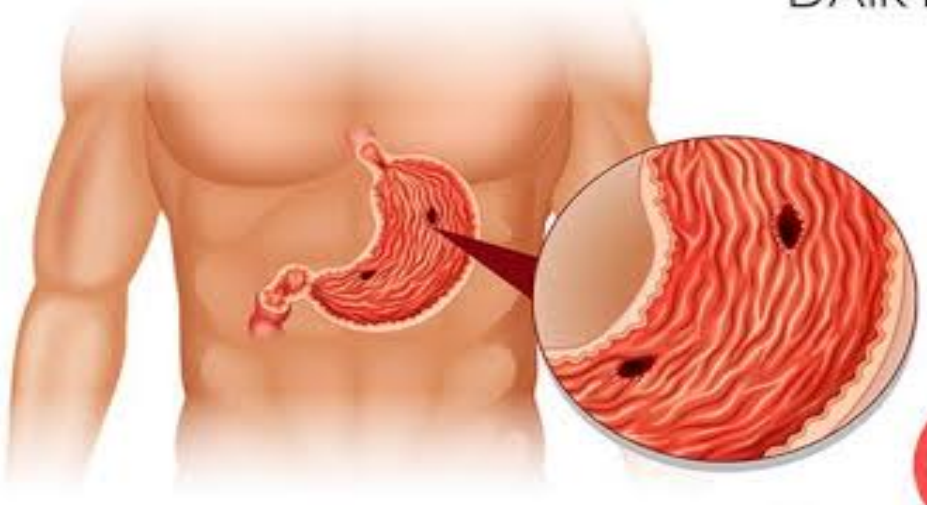
- Sudden onset of acute pain and tenderness in epigastric region.
- Vomiting with or without haematemesis.
- Often acute peptic ulcers can lead to perforations.
- Acute ulcers after cerebral trauma or neurosurgeries are called as Cushing's ulcers.
- Acute ulcers after major burns are called as Curling's ulcers.
- Diagnosis is by gastroscopy.

FOODS TO AVOID IF YOU HAVE A STOMACH ULCER

ALCOHOLIC BEVERAGES

COFFEE

CARBONATED DRINKS



MILK AND OTHER DAIRY PRODUCTS

RED MEAT

SALT AND SALTY FOODS

Top 10 Home Remedies

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Treatment

- Intravenous ranitidine 50 mg, 8th hourly.
- IV fluids.
- Blood transfusions if there is bleeding.
- Most of the time surgery is not required for acute ulcers. During follow-up patients are advised to take antiulcer drugs for 4-6 weeks—ranitidine, omeprazole or lansoprazole.

- **Curling's ulcers**
- They are acute ulcers which develop after major burns, presenting as pain in epigastric region, vomiting or haematemesis.
- Treatment is conservative- IV ranitidine.
- IV pantoprazole 80 mg in 100 ml DNS—slow, later 40 mg IV maintenance.
- Note: Curling's ulcer occurs when burn injury is more than 35%.
- It is observed in the body and fundus not in antrum and duodenum.

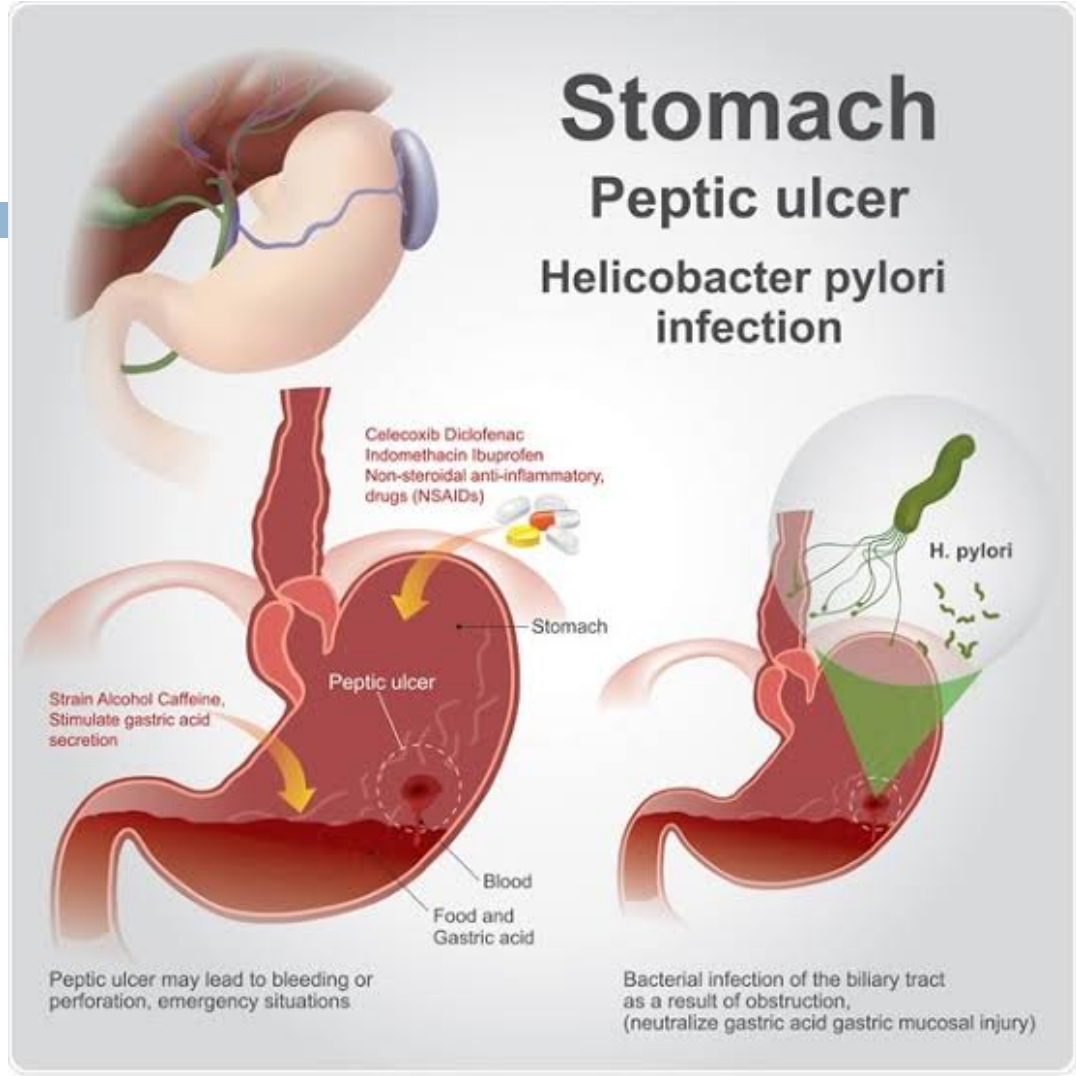
- **Cushing's ulcers**
- They are acute ulcers which develop after cerebral trauma or after neurosurgical operations.
- Treatment is conservative by IV Ranitidine.

GASTRIC ULCER

Gastric ulcer patients secrete either low normal or below normal amounts tiset of acid . Only 5 % of patients may demonstrate acid hypersecreation.

AETIOLOGY

1. DIMINISHED MUCOSAL RESISTANCE
2. PYZORODUODENAL REFLUX
3. DEFICIENT MUCOUS BARRIER
4. MUCOSAL TRAUMA
5. LOCAL ISCHAEMIA
6. ANTRAL STASIS
7. NONSTEROIDAL ANTIINFLAMMATORY DRUGS (NSAIDs)
8. HELICOBACTER PYLORI
9. ATROPHIC GASTRITIS .
10. SMOKING , ALCOHOL
11. LOVER SOCIOECONOMIC GROUP

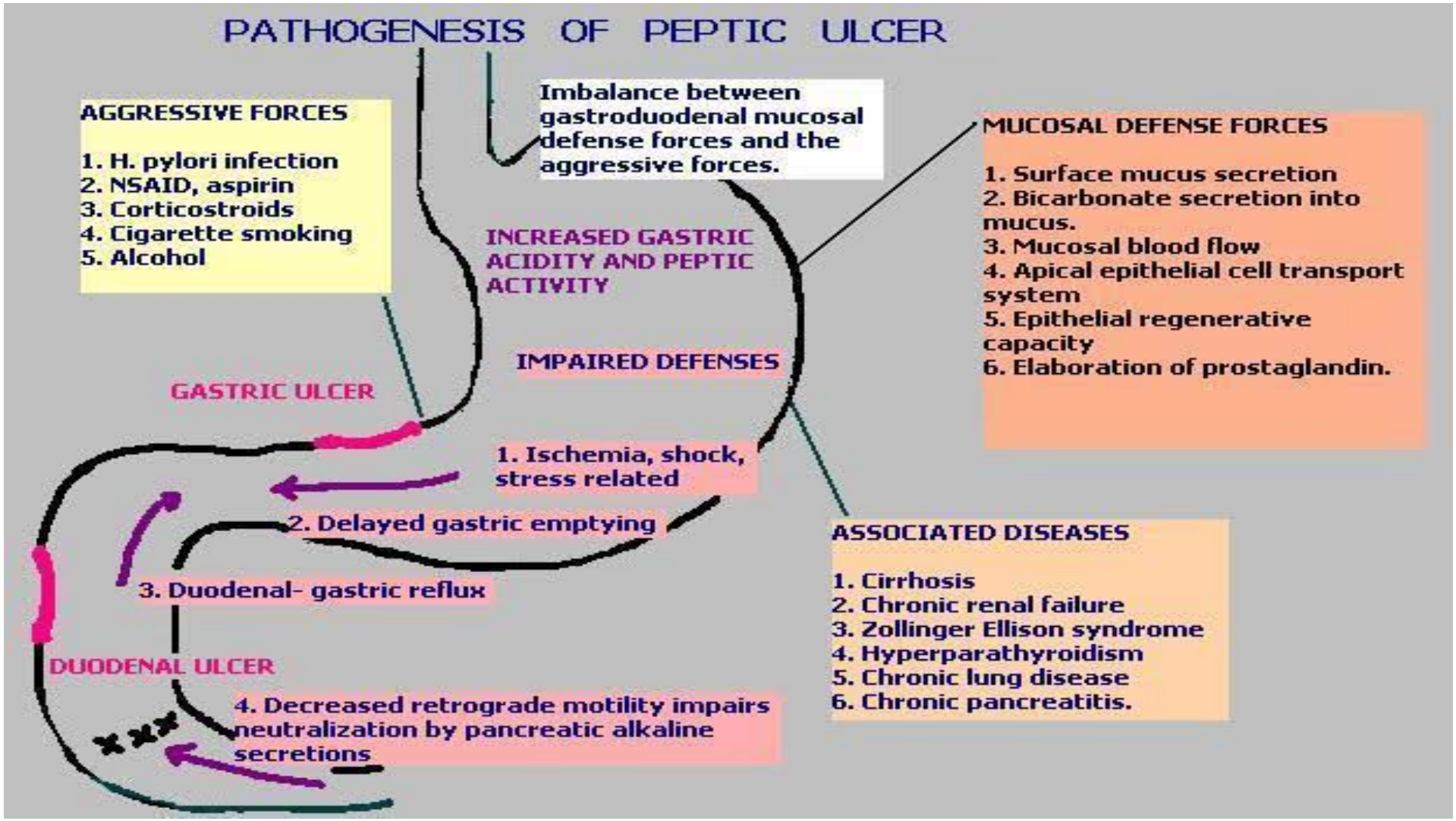


Factors Involved in Gastric Ulcer Formation

- Duodeno gastric reflux – reflux containing bile salts and lysolecithin break the mucosal barrier making it more vulnerable for injury, action of drugs and pepsin injury.
- Gastric stasis.
- Ischaemia of the gastric mucosa.

PATHOLOGY

- Gastric ulcer is large in size, usually lies in the lesser curvature, its floor being formed by the muscular layer.
- Posteriorly it may penetrate into the pancreas; it may cause torrential bleeding by eroding left gastric (commonly) vessles or splenic vessels or vessels in the gastric ulcer wall.
- Anteriorly it may perforate or penetrate into the liver. It may lead into hour glass contracture, or tea-pot deformity.
- Microscopically, it shows ulcer crater with chronic inflammatory cells and granulation tissue, end arteritis obliterans and epithelial proliferation. (Ulcer to the right of the incisura is malignant unless proved otherwise).
- Gastric ulcer > 3 cm is called as giant gastric ulcer. It has got 6-23% chances to turn in to malignancy.
- Grossly, margin of the benign gastric ulcer is clear; deep; near lesser curve; edge is not everted with gastric mucosal folds converging towards the base of the ulcer.
- 95% of benign gastric ulcer occurs towards lesser curve, as it takes more burden of passage of food and so more of wear and tear. Benign gastric ulcer is rare in greater curvature, fundus and cardia.


PATHOGENESIS OF PEPTIC ULCER
AGGRESSIVE FORCES
1. H. pylori infection
2. NSAID, aspirin
3. Corticostroids
4. Cigarette smoking
5. Alcohol
Imbalance between gastroduodenal mucosal defense forces and the aggressive forces.
MUCOSAL DEFENSE FORCES
1. Surface mucus secretion
2. Bicarbonate secretion into mucus.
3. Mucosal blood flow
4. Apical epithelial cell transport system
5. Epithelial regenerative capacity
6. Elaboration of prostaglandin.
INCREASED GASTRIC ACIDITY AND PEPTIC ACTIVITY
IMPAIRED DEFENSES
GASTRIC ULCER
1. Ischemia, shock, stress related
2. Delayed gastric emptying
3. Duodenal- gastric reflux
DUODENAL ULCER
4. Decreased retrograde motility impairs neutralization by pancreatic alkaline secretions
ASSOCIATED DISEASES
1. Cirrhosis
2. Chronic renal failure
3. Zollinger Ellison syndrome
4. Hyperparathyroidism
5. Chronic lung disease
6. Chronic pancreatitis.

Types of Gastric Ulcer (Daintree Johnson)

Type	*Location*	*Incidence*	*Acid level*
Type I	In the antrum, near the lesser curve	55%	Normal
Type II	Combined gastric ulcer (in the body) with duodenal ulcer	25%	High
Type III	Prepyloric ulcer	15%	High
Type IV	Gastric ulcer in the proximal stomach or cardia	5%	Normal

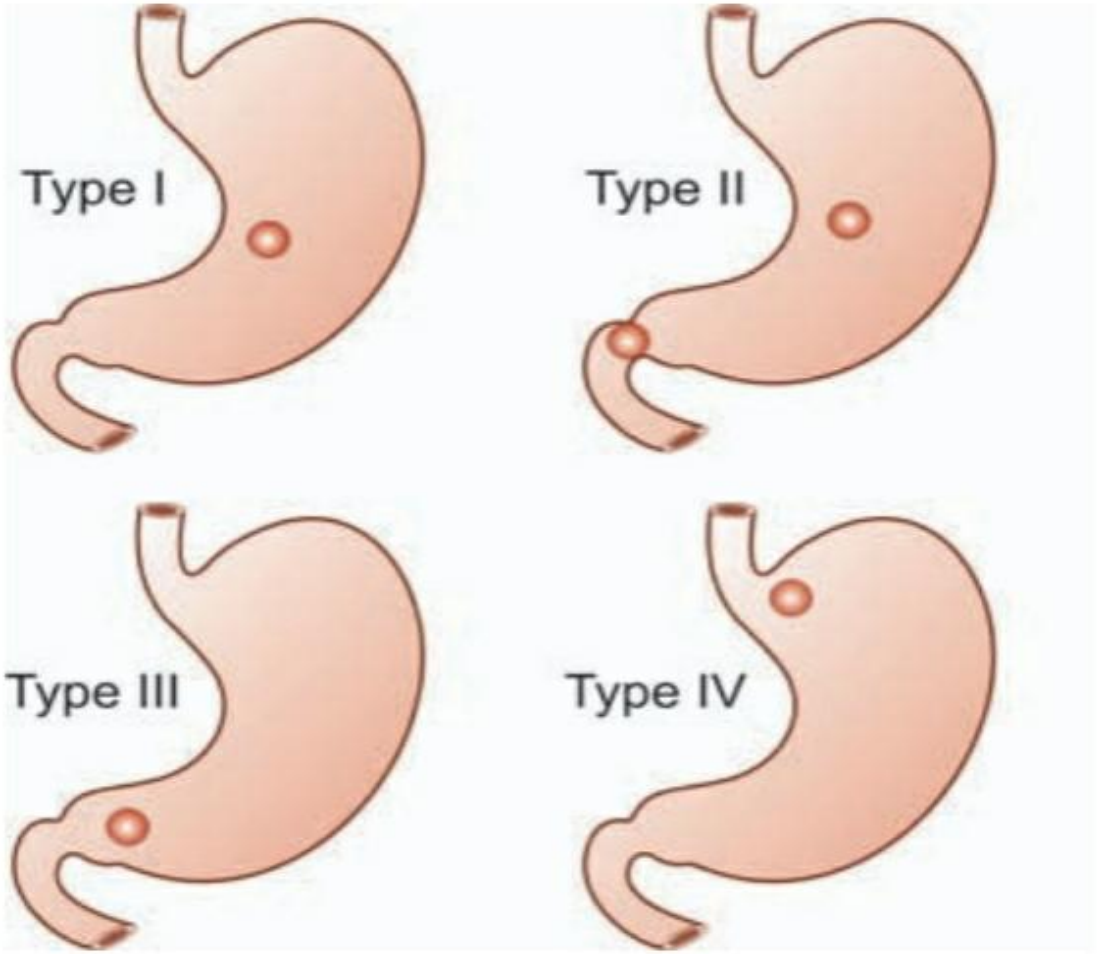


Fig. 20.16: Types of gastric ulcer.

Clinical Features

1. Age - The patients are usually middle
2. Sex - More common in males than females
3. Constitution . The patients are usually thin and anaemic with J - shaped hypotonic
4. Periodicity is less marked . The attacks last several weeks followed by intervals of freedom from symptoms for 2 to 6 months stomach .
5. Pain . It is strictly epigastric . Pain is boring or pricking in nature . When the ulcer penetrates , pain may radiate to the back .
6. Vomiting.- In more than half the cases symptom . It often occurs after food . It relieves the pain and may be self - induced .
7. Appetite - is good . But the patient is afraid to eat as this initiates pain . So the patient actually complains of ' does not feel to eat ‘ .

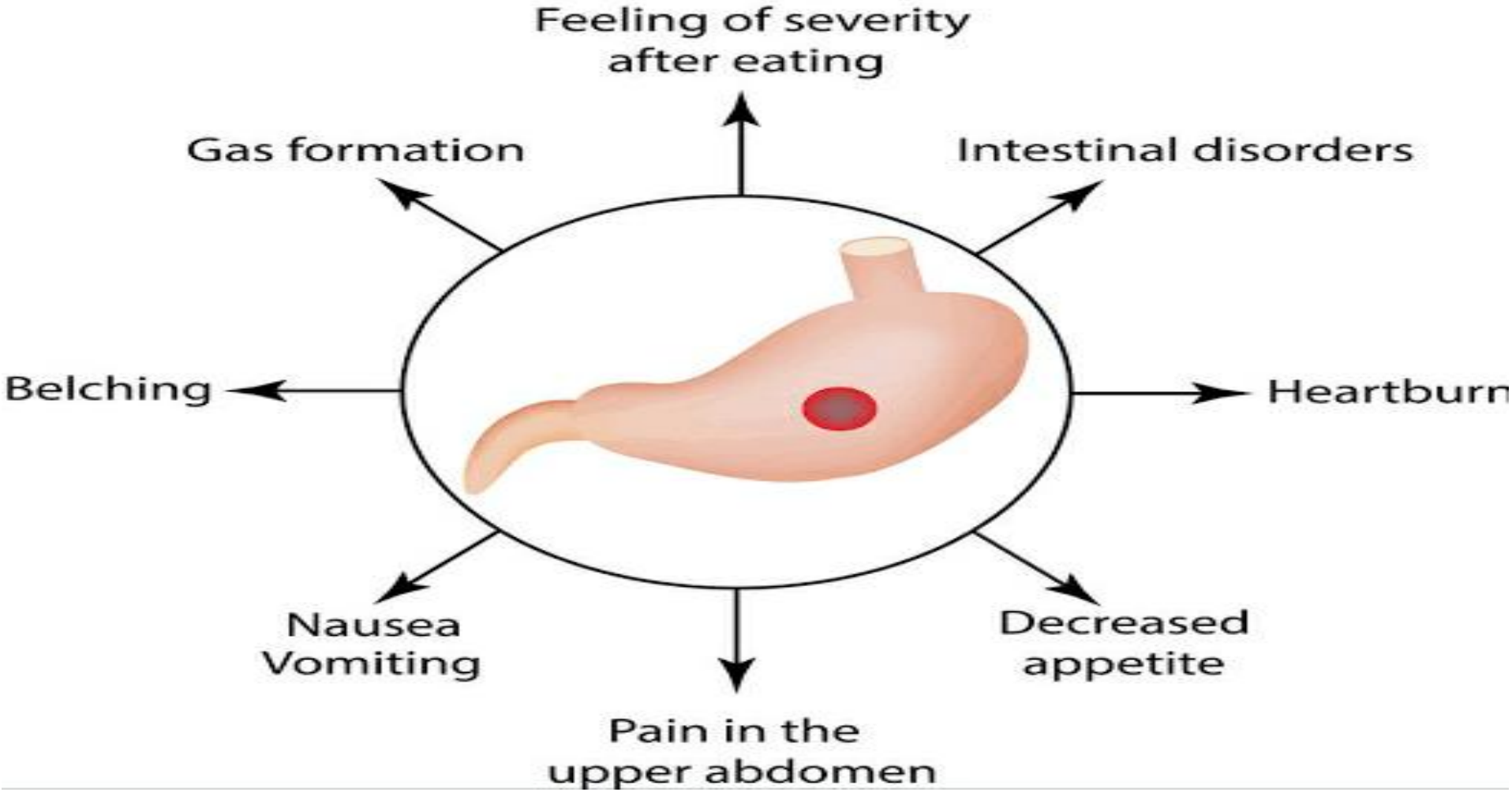
Cont.....

8. Diet . These patients usually learn to avoid fried and spicy foods since this initiates pain immediately .

9. Weight.- Some loss of weight is usually present

10. Haemorrhage is less common than duodenal ulcer (approximately 30 %) . Haematemesis is more common than melaena .

11. On examination -- tenderness can be elicited in the mid - epigastrium or slightly to the left of it .


Feeling of severity
after eating
Gas formation
Intestinal disorders
Belching
Heartburn
Nausea
Vomiting
Decreased
appetite
Pain in the
upper abdomen

	Benign gastric ulcer	*Malignant gastric ulcer*
Mucosal folds	Converging mucosal folds upto the margin	Effacing mucosal folds
Site	95% lesser curve	Greater curvature
Margin	Regular margin	Irregular margin
Floor	Granulation tissue in floor	Necrotic slough in floor
Edge	Not everted; punched or sloping	Everted edge
Surrounding area	Surrounding area and rugae are normal	Surrounding area shows nodules, ulcers and irregulaties
Size and extent	Small, deep up to part of muscle layer	Large and deep

Differential Diagnosis

- Hiatus hernia.
- Cholecystitis.
- Chronic pancreatitis.
- Chronic gastritis.
- Dyspepsia.
- Carcinoma stomach.

Special investigation :-

1.Examination of blood.
2. Examination of stool.
3. gastric function tests.
i. Night fasting section.
ii. Basal section.
iii. Maximum section or peak outputiiii. pentagastrin test.
iv. key's augmented histemin testv. Hollander 's insulin test
4.Radiological investigation
5.endoscopy

Treatment

- Diets should be regulated. Diets must be taken in time. Spicy foods should be avoided Bland diet is advised. In case of duodenal ulcer meals should be taken at 2 hours interval is widely used in the treatment of ulcer disease, but there are few data to support its effice Calcium in milk is known to stimulate release of gastric acid secretion.
- Now that a host of medications with proven efficacy are available, majority of patients do not require surgery. It is only required in less than 5% of patients.
- The management of upper gut ulceration varies for duodenal and gastric ulcers.
- H - receptor antagonist and proton pump inhibitors peptic ulceration. Most gastric ulcers and duodenal ulcers can be healed by a few weeks treatment with these drugs provided they are taken in time and absorbed. But a few patients may be relatively refractory to conventional doses of H.-receptor antagonists. In these cases proton pump inhibitors can be used and majority of ulcers heal within 2 weeks. Relief of symptoms is quite impressive. Both these drugs are quite safe and with little serious side-effects The only problem is the cessation of therapy.

Cont.....

- Drugs like H2 blockers, proton pump inhibitors, carbenexolone (Biogastrone, Sucralfate, prostaglandins which coats the ulcer and so creates a mucosal barrier) helps in reducing or eliminating the symptoms.
- But asymptomatic ulcer may exist silently and may turn into malignancy.
- So surgery is the preferred line of treatment. Partial gastrectomy and Billroth I gastroduodenal anastomosis is done.
- Type IV proximal gastric ulcer is difficult to manage. It is treated by subtotal gastrectomy. Often distal gastrectomy with selective sleeve like extension cut along the lesser curve to remove the ulcer is done by Pauchet's procedure.

Eating Tips to Reduce Peptic Ulcer Symptoms

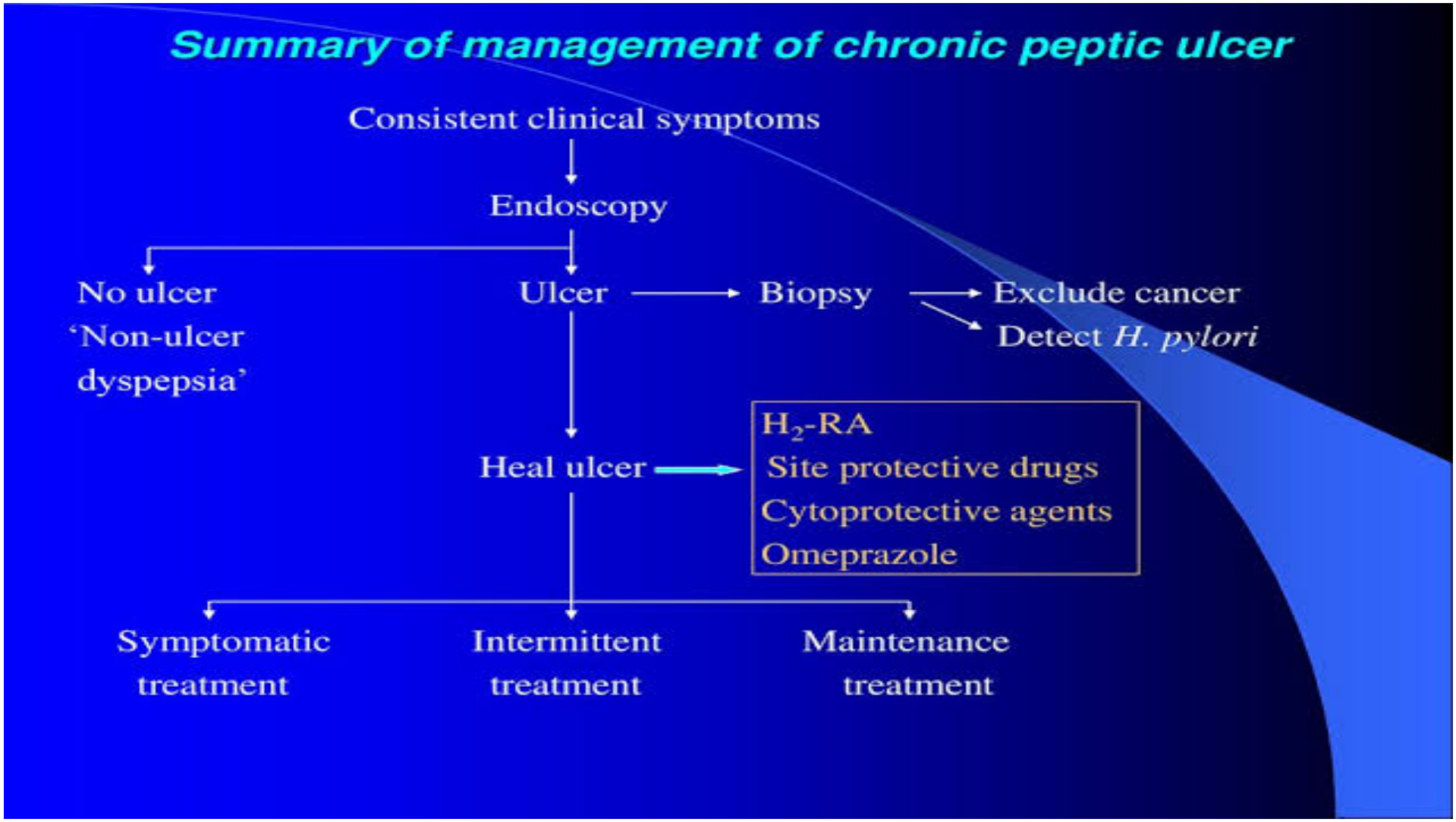
Enjoy five or six
small meals per day

Chew each bite
thoroughly

Always sit upright
in a chair while eating

Rest a few minutes
before and after each meal

Enjoy your last meal or snack
at least 3 hours before bedtime


Summary of management of chronic peptic ulcer
Consistent clinical symptoms
Endoscopy
No ulcer
'Non-ulcer dyspepsia'
Ulcer
Biopsy
Exclude cancer
Detect H. pylori
Heal ulcer
H2-RA
Site protective drugs
Cytoprotective agents
Omeprazole
Symptomatic treatment
Intermittent treatment
Maintenance treatment

COMPLICATION

1. Hour glass contracture: It occurs exclusively in women, is due to cicatricial contracture of lesser curve ulcer.

– Here stomach is divided into two compartmen

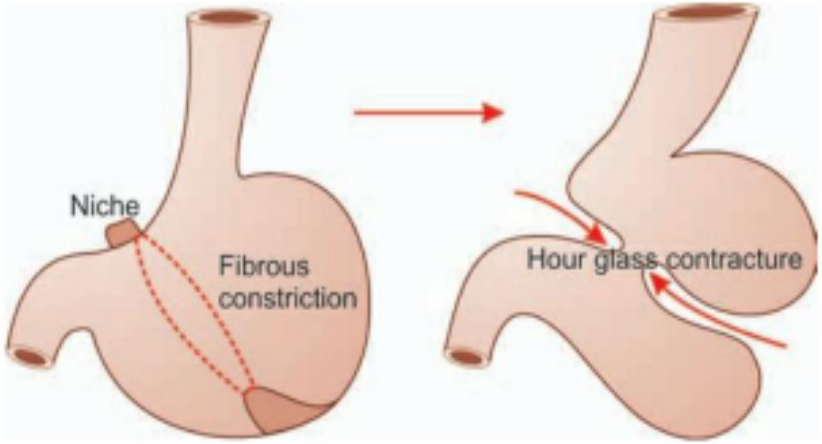


Fig. 20.24: Hour glass contracture.

• **Clinical features**

– Loss of periodicity.

– Persistent pain.

– Vomiting.

– Loss of appetite and weight.

• **Diagnosis**

– Barium meal: It shows filling only in the proximal stomach or double pouched stomach.

– Gastroscopy.

• **Treatment:** Partial astrectomy and Billroth-1 anastomosis.

2. Tea-pot deformity: (Hand-Bag stomach) is due to cicatrisation and shortening of the lesser curvature.

– They present with features of pyloric stenosis.

– Treatment is partial gastrectomy with Billroth-I anastomosis.

3. Perforation.

4. Bleeding by erosion into the left gastric and rarely splenic vessels or to vessels in the wall of ulcer.

5. Penetration posteriorly into pancreas, anteriorly into liver.

6. Malignant transformation usually into adenocarcinoma of stomach (2-5%).

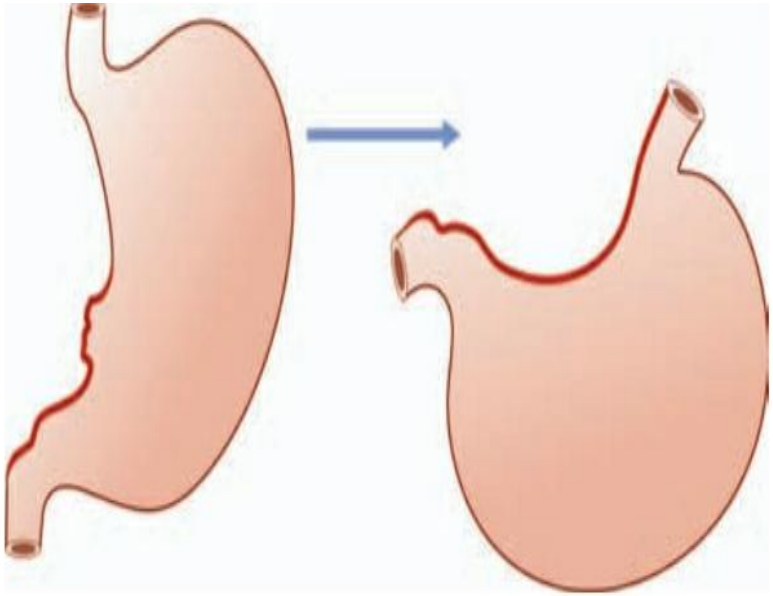


Fig. 20.25: Tea-pot deformity.

Duodenal Ulcer

Aetiology

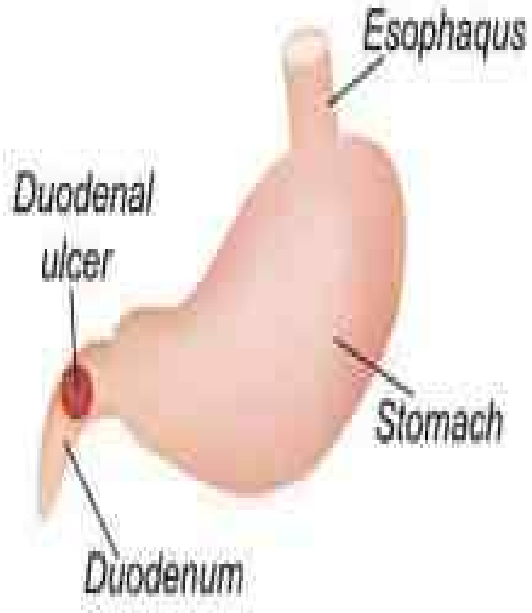
- Common in people with blood group O +ve.
- Stress, anxiety—'hurry, worry, curry'.
- Helicobacter pylori infection is an important aetiology

for duodenal ulcer (90%).

- NSAIDs, steroids.
- Endocrine causes: Zollinger–Ellison syndrome, MEN

syndrome, hyperparathyroidism.

- Other causes: Alcohol, smoking, vitamin deficiency.



#PatientsAwareness

KAIZEN HOSPITAL
Institute of Gastroenterology & Research Centre
Care - Compassion - Cure

Causes of Duodenal Ulcers

H Pylori Bacteria

Alcohol

Tobacco

NSAIDs

Radiation Therapy

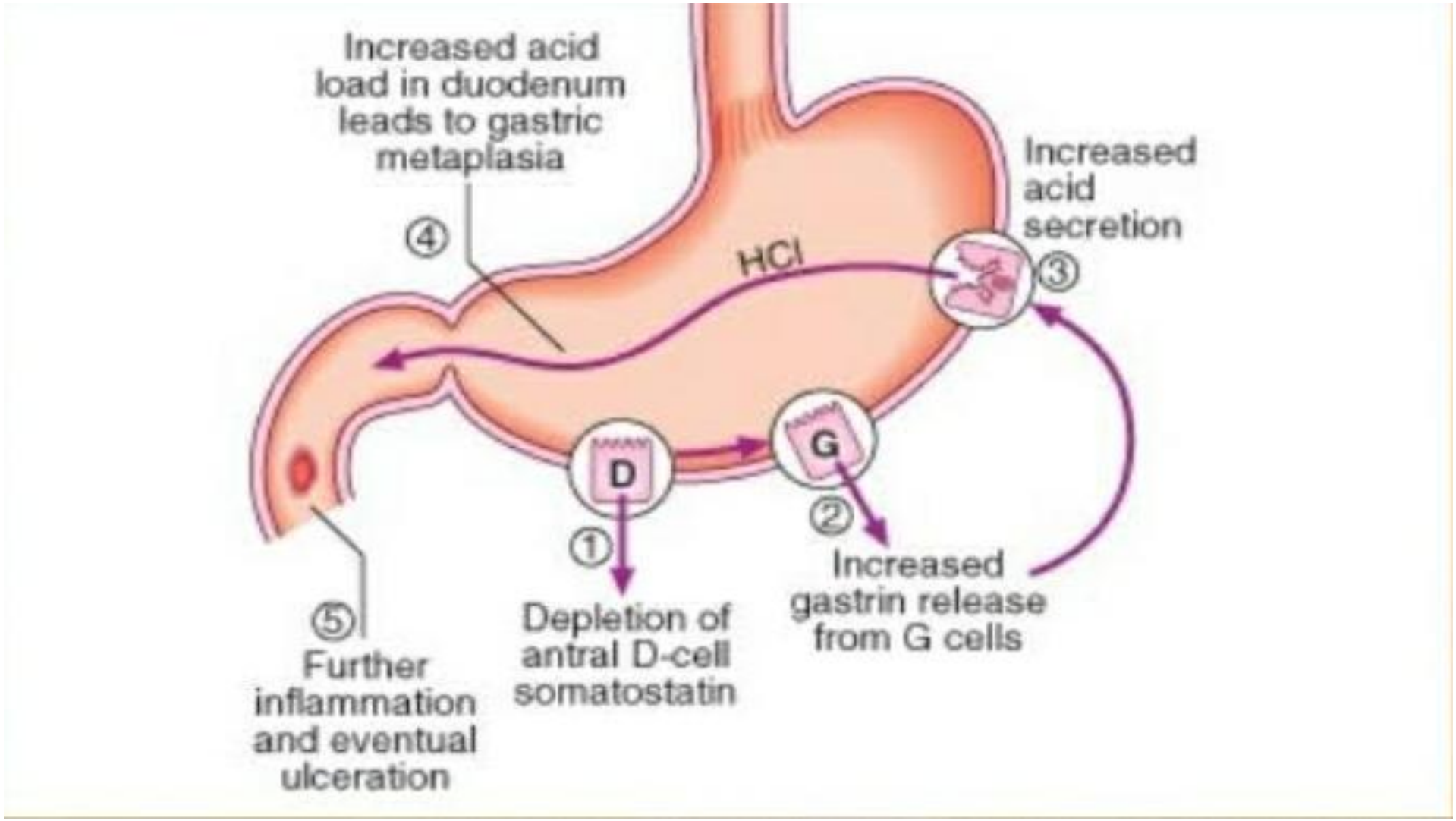
Stress, severe illness

PATHOLOGY

- Ulcer occurs in the first part of duodenum, usually with in the first inch,involving the muscular layer.
- Sites:
  a. In the bulb (bulbar)—95%.
  b. Post bulbar (5%).
- Eventually it shows cicatrisation causing pyloric stenosis. Serosa overlying the site of duodenal ulcer shows petechial haemorrhages with speckled red dots, appearing like sprinkled **cayenne pepper.**

Conti.....

- Microscopically, ulcer with chronic inflammation with granulation tissue, gastric metaplasia of duodenal mucosa, endarteritis obliterans are visualised.
- Sometimes two opposing ulcers, i.e. over anterior and posterior surfaces of duodenum are present and are called as **kissing ulcers**.
- An anterior ulcer perforates commonly, posterior ulcer bleeds or penetrates commonly.


Increased acid load in duodenum leads to gastric metaplasia
④
HCl
Increased acid secretion
③
D
G
①
②
Increased gastrin release from G cells
⑤
Further inflammation and eventual ulceration
Depletion of antral D-cell somatostatin

Clinical Features

1. Age.— The patients are usually young adult or mid-adult (25 to 40 years).

2. Sex.— Males dominate though not as much as gastric ulcer.

3. Constitution.— The patients are healthy males with steer-horn stomach which is high in position.

4. Periodicity is well marked. The attacks also last for several weeks with intervals of freedom from 2 to 6 months. Attacks usually appear in the spring and autumn.

5. Pain.— Pain is more severe and spasmodic in character.

(i) Site.— In duodenal ulcer patient complains of pain on the transpyloric plane about 1 inch to the right of the midline.

(ii) Relation with food.— Pain usually starts 2Vi to 3 hours after food when the stomach

gradually pushes the chyme into duodenum and irritates ulcer.

(iii) Pain is very much **felt in empty** stomach, which is called 'hunger-pain'. Excess acid is not neutralised by food and irritates ulcer.

(iv) **Food relieves pain** as this dilutes acid.

(v) **Pain at dead of night** is very characteristic. The patient gets up with pain, he takes biscuits and milk. He is relieved of pain and goes to sleep.

6. Vomiting is rare, unless pyloric stenosis complicates the procedure. More common is regurgitation of acidic fluid into the mouth or pain behind the sternum due to reflux oesophagitis (‘heart-bum’).

7. Appetite is quite good and he eats frequently to avoid pain.

8. Diet. — As any particular food does not initiate pain immediately, he usually does not avoid anything. Only a few intelligent patients may avoid fried and spicy foods.

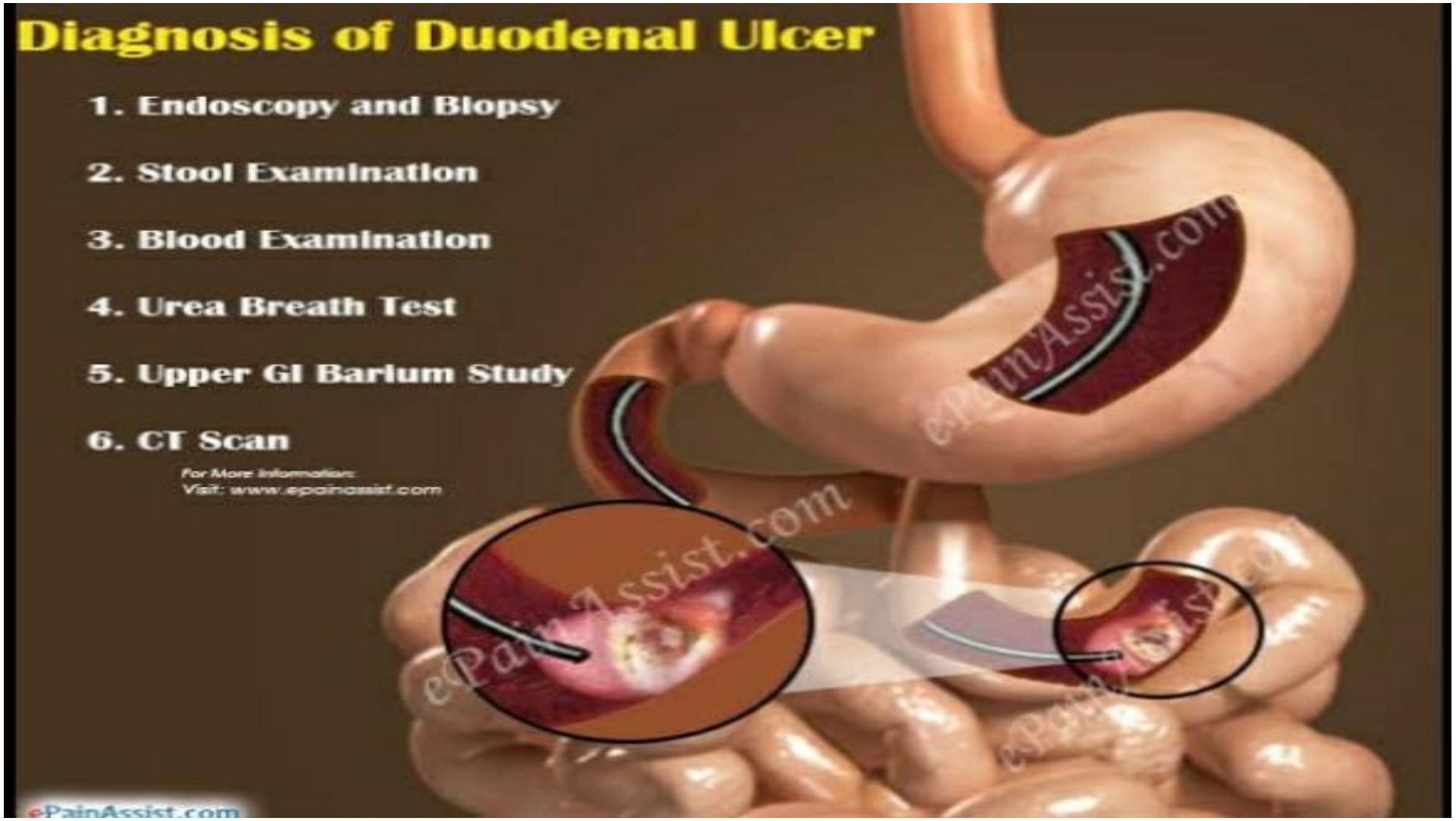
9. Weight.— Some gain of weight is expected, as the patient learns to take food .frequently.

10. Haemorrhage is more common than in gastric ulcer. Melaena is more common than haematemesis. Haematemesis is only possible when massive haemorrage forces open the pylorus.

11 .On examination — tenderness can be elicited at the ‘duodenal point’ which is situated on the transpyloric plane 1 inch right to the midline.

INVESTIGATION

- Barium meal X-ray shows deformed or absence of duodenal cap (because of spasm). Appearance of ‘trifoliate’ duodenum is due to secondary duodenal diverticula which occurs as a result of scarring of ulcer.
- Gastroscopy reveals the type, location of ulcer, narrowing if any. Biopsy also can be taken to look for the presence of Helicobacter pylori. Usually biopsies are taken from duodenum, pylorus, antrum, body, fundus, and confirmed by rapid urease test or C13 or C14 breath tests.
- Estimation of serum gastrin level, serum calcium level.


Diagnosis of Duodenal Ulcer
1. Endoscopy and Biopsy
2. Stool Examination
3. Blood Examination
4. Urea Breath Test
5. Upper GI Barium Study
6. CT Scan
For More Information:
Visit: www.epainassist.com
ePainAssist.com

Differential diagnosis

- Carcinoma stomach (pylorus)
- Dyspepsia due to other causes
  - Hiatus hernia
  - Oesophagitis
  - Cholecystitis
  - Chronic pancreatitis

Treatment

- **I. General measures:** Avoid alcohol, NSAIDs, smoking, spicy foods. Have more frequent food.
- **II. Specific measures:**
- **Drugs**
- **1. H2 Blockers:** Promotes ulcer healing in 4-8 weeks, by reducing acid secretion.

• Tab cimetidine.

• Tab ranitidine (300 mg HS or 150 mg BID), (IV preparation is available).

• Tab famotidine (IV is available) Most potent H2 blocker. Dose is 20-40 mg/day.

• Tab roxatidine.

• Tab nizatidine.

- **2. Proton pump inhibitors**: Inhibit parietal cell H+, K+ ATPase enzyme responsible for acid secretion. They are used for 6-12 weeks. They stop acid secretion completely.

• Omeprazole 20 mg OD 1 hour before food IV preparation is available.

• Esomeprazole 40 mg.

• Lansoprazole 30 mg.

• Pantoprazole 40 mg and Rabeprazole 20 mg—IV preparation available.

Cont.....

- Antacids
- Sucralfate
- Anti-Helicobacter pylori regime
- Colloid bismuth sulphate
- Misoprostol
- Note: Antacids and H2 blockers should not be used along with PPI as these drugs will reduce the action of PPIs by creating alkaline media.

Anti-Helicobacter regime (Triple regime)		
Clarithromycin 500 mg BD OR Amoxycillin 500-750 mg BD OR Tetracyclines, or Bismuth, etc.	Metronidazole 400 mg BD OR Tinidazole 600 mg BD	Omeprazole 20 mg BD OR Lansoprazole 30 mg BD OR Pantoprazole 40 mg BD

Surgical Treatment

- **Indications for surgical intervention for chronic DU (Uncomplicated DU):**
- 1. Uncomplicated DU, not responding to drug therapy of 8-12 weeks-intractable duodenal ulcer
- 2. Repeated recurrences -Presently most of the uncomplicated DU does not require surgery

Surgery for Uncomplicated DU

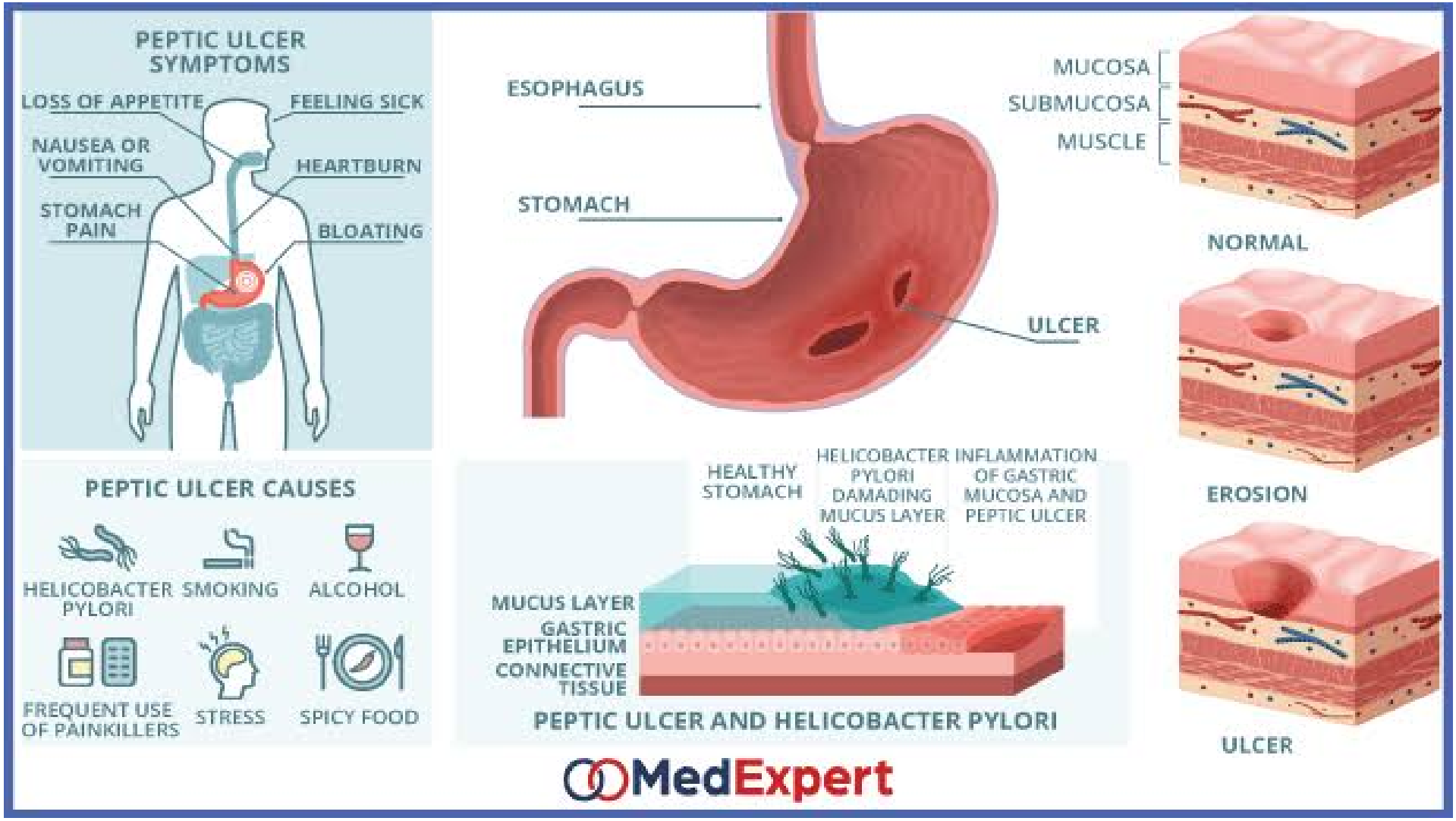
- Highly Selective Vagotomy (HSV).
- Selective vagotomy with pyloroplasty (SV + P).
- Truncal vagotomy with gastrojejunostomy (TV + GJ).
- Posterior truncal vagotomy with anterior seromyotomy—Taylor's operation. It can be done through laparoscopy.

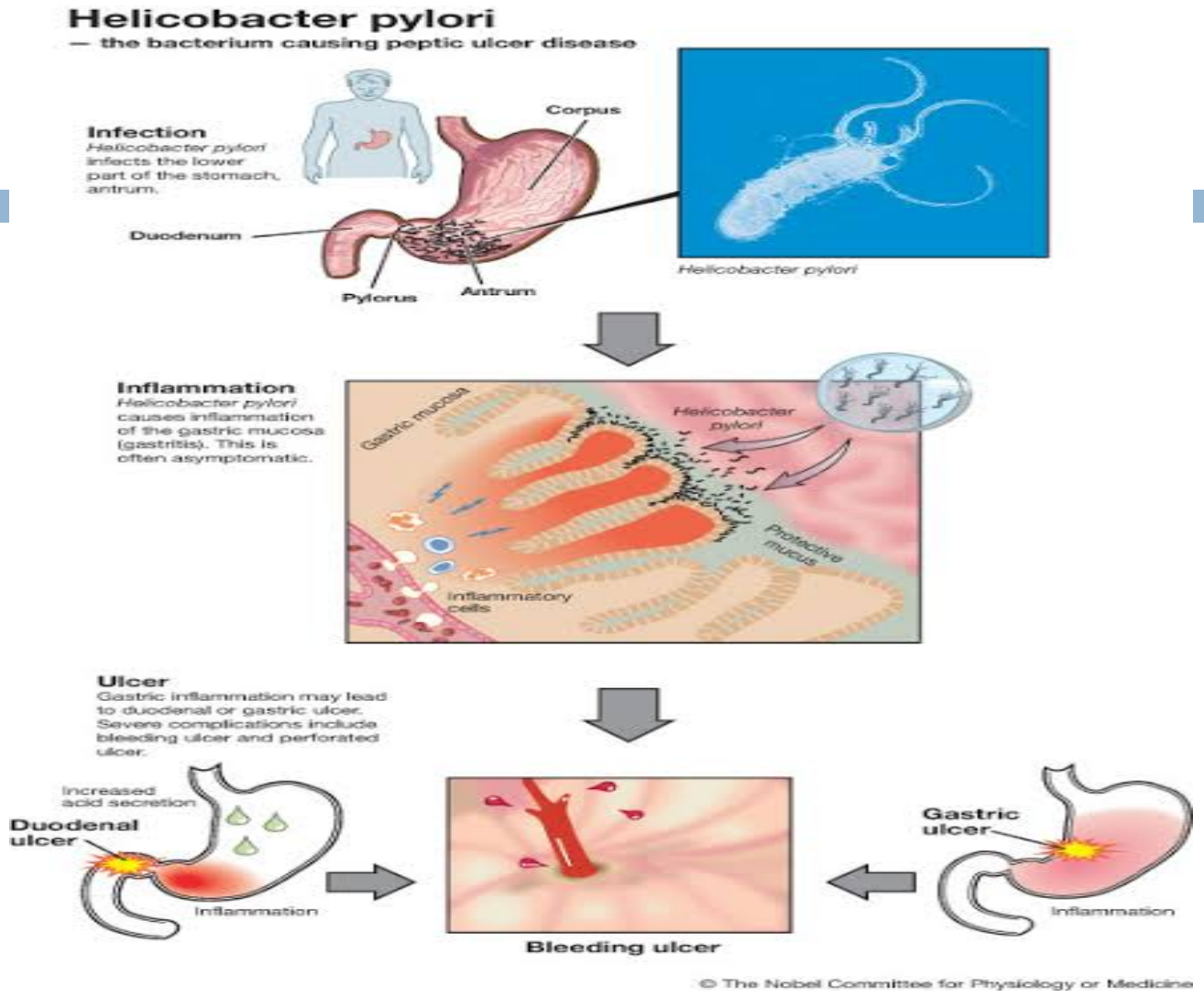
Note: Presently, there is no role of gastrectomy or gastrojejunostomy (Just GJ) for uncomplicated DU.

Difference between gastric and duodenal ulcer

	Duodenal Ulcer	Gastric ulcer
Age	Any age specially 30-40	middle age 50-60
Sex	More in male	More in male
Occupation	Stress job eg. Manager	Same
Pain	Epigastric , discomfort	Epigastric. Can radiate to back
Onset	2-3 hours after eating & midnight	Immediately after eating
Agg.by	Hunger	Eating

	Duodenal Ulcer	Gastric ulcer
Relived by	Eating	Lying down or vomiting
Duration	1-2 months	Few weeks
Vomiting	Uncommon	Common(to relieve the pain)
Appetite	Good	Patient is afraid to eat
Diet	Good , eat to relieve the pain	Avoid fried food
Weight	No wt. loss because patient eats to avoid pain.	wt. Loss because patient is afraid to eat.
Hematemesis	40%	60%
Melena	60%	40%


PEPTIC ULCER SYMPTOMS
LOSS OF APPETITE
FEELING SICK
NAUSEA OR VOMITING
HEARTBURN
STOMACH PAIN
BLOATING
PEPTIC ULCER CAUSES
HELICOBACTER PYLORI
SMOKING
ALCOHOL
FREQUENT USE OF PAINKILLERS
STRESS
SPICY FOOD
ESOPHAGUS
STOMACH
ULCER
HEALTHY STOMACH
HELICOBACTER PYLORI DAMADING MUCUS LAYER
INFLAMMATION OF GASTRIC MUCOSA AND PEPTIC ULCER
MUCUS LAYER
GASTRIC EPITHELIUM
CONNECTIVE TISSUE
PEPTIC ULCER AND HELICOBACTER PYLORI
MUCOSA
SUBMUCOSA
MUSCLE
NORMAL
EROSION
ULCER
MedExpert


Helicobacter pylori
— the bacterium causing peptic ulcer disease
Infection
Helicobacter pylori infects the lower part of the stomach, antrum.
Corpus
Duodenum
Pylorus
Antrum
Helicobacter pylori
Inflammation
Helicobacter pylori causes inflammation of the gastric mucosa (gastritis). This is often asymptomatic.
Gastric mucosa
Helicobacter pylori
Protective mucus
Inflammatory cells
Ulcer
Gastric inflammation may lead to duodenal or gastric ulcer. Severe complications include bleeding ulcer and perforated ulcer.
Increased acid secretion
Duodenal ulcer
Inflammation
Bleeding ulcer
Gastric ulcer
Inflammation
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THANK YOU