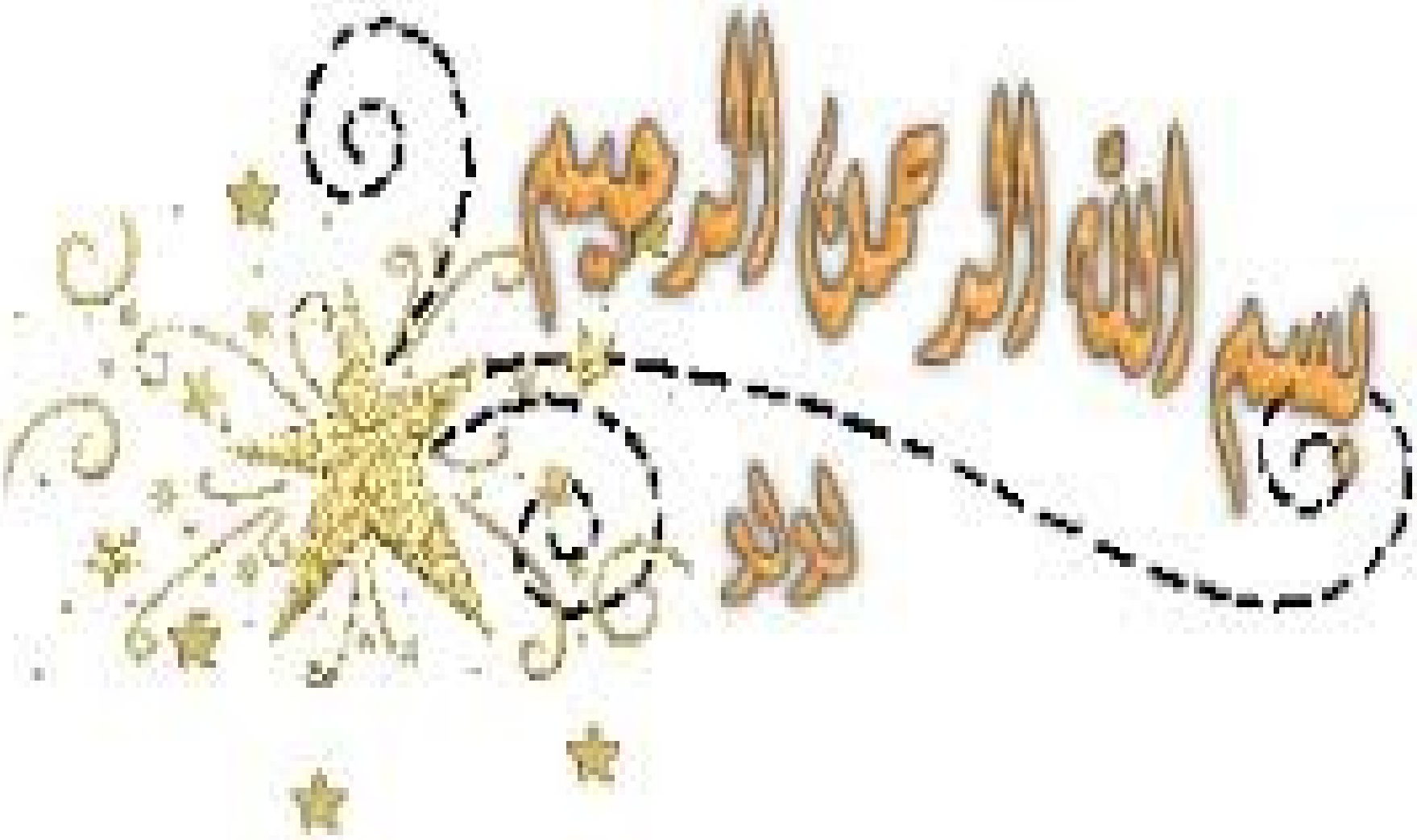
بسم الله الرحمن الرحيم

# UPPER GIT BLEEDING

# Learning objectives

- Common causes of upper GIT bleeding-1
- Clinical assessment of patient with upper-2
- GIT bleeding
- Management and assessment of-3 severity by Rockall Scoring System

Acute upper G.I hemorrhage is the most common G.I emergency, accounting for 50-170 admissions to hospital per 100,000 of the population each year in the UK.

The mortality of patient's admitted to hospital is about 10% but there is some evidence that outcome is better when patients are treated in specialized units.

Common causes of upper G.I bleeding:

1. Peptic ulcer (35-50%) due to e.g: H.pylori, NSIDs.
2. Gastric erosions (10-20%) due to e.g: Alcohol, NSAID.
3. Oesophagitis (10%) usually with hiatus hernia.
4. Varices (2-9%) due to liver disease as portal vein thrombosis.
5. Mallory-weiss tear (5%) due to retching.
6. Vascular malformation (5%).
7. Cancer of stomach or oesophagus (2%).
8. Aorto-duodenal fistula (0.2%) in aortic graft.

**Clinical assessment:**

1. Haematamesis is red with clots when bleeding is profuse, as black "coffe grounds" when less severe.
2. Syncope may occur.
3. Symptoms of anaemia suggest chronic bleeding.
4. Malaena is the passage of black, tarry stools containing altered blood, usually caused by bleeding from upper G.I tract, although occasionally arise from right side of the colon.
5. Severe acute upper G.I hemorrhage can some times cause maroon or bright red stool.

- **Management:**

A. Management of non-variceal bleeding:

1. Intravenous access (large bore canula).
2. Check full blood count, routine biochemistry and cross match blood.
3. Initial clinical assessment:

3.1:circulatory status (tachycardia, hypotension, oliquria, coldness, sweating, agitation).

3.2: Evidence of liver disease: jaundice, cutaneous stigmata, hepatosplenomegaly, and ascites.

3.3: identify comorbidity: cardiorespiratory, cerebrovascular or renal disease which may worsened by acute bleeding and because they increase the hazards of endoscopy and surgical operations.

Rockall scoring system used to predict severity (ranging from 0-11).

A score <3 is associated with a good prognosis, while a score >8 carries a high risk of mortality.

| Variable | Score 0 | Score1 | Score2 | Score 3 |
|---|---|---|---|---|
| Age | <60 | 60-79 | >80 | |
| Shock | No shock | pulse >100 | SBP <100 | |
| Comorbidity | Nil | CCF,IHD | Renal/liver | Metastatic cancer |
| Diagnosis | Normal or Mallory Weiss | All other | Malignancy | |
| Evidence of bleeding | non | Blood in stomach | Clot or visible vessel | |

4. Resuscitation: I.v crystalloid or colloid fluids,, and blood transfution in active bleeding "low blood pressure and tachycardia", broad spectrum antibiotics in suspected chronic liver disease, central venous pressure (CVP) in severe bleeding.
5. Oxygen: for all patients with shock .
6. Endoscopy: this should be carried out after adequate resuscitation, ideally within 24 hours. Recent bleeding can be treated endoscopically by thermal modality, injection of dilute adrenaline or by metallic clips.
8. Monitoring: Hourly pulse, blood pressure and urine out-put.
9. Surgery.

**B. Management of variceal bleeding:**

1. Restore the circulation with blood and plasma (to prevent further deterioration in liver function because of hypotension)

2. Endoscopy: even in patients with known varices, because about 20% of patients bleed from other cause.
3. Prophylactic broad spectrum antibiotics (oral ciprofloxacin or I.V cephalosporin) because sepsis is common and antibiotics has been shown to improve outcome.
4. The measures used to control acute variceal bleeding include endoscopic therapy (banding or sclerotherapy), balloon tamponade and oesophageal transection.

5. Vasopressor (terlipressin): reduces portal pressure, acute bleeding and risk of early re-bleeding.
6. Proton pump inhibitor (PPI): to prevent peptic ulcers.
7. Phosphate enema and/or lactulose to prevent hepatic encephalopathy.
8. Shunt surgery:

Transjugular intrahepatic portosystemic shunting (TIPSS). Portosystemic shunt surgery.

# Peptic ulcer disease

- Learning objectives
- Definition and pathology-1
- Epidemiological characteristic-2
- Understanding of pathophysiology-3
- Clinical presentation-4
- Investigations-5
- Management and complications-6
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Peptic ulcer:

The term peptic ulcer refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach, or rarely, in the ileum adjacent to a Meckel's diverticulum.

Ulcers in the stomach or duodenum may be acute or chronic; both penetrate the muscularis mucosae but the acute ulcer shows no evidence of fibrosis. Erosions do not penetrate the muscularis mucosae.

Gastric and duodenal ulcer:

1. The prevalence of peptic ulcer is decreasing in many western communities as a result of widespread use of H.pylori eradication therapy but it remains high in developing countries.
2. The male to female ration for duodenal ulcer varies from 5:1 to 2:1, while that for gastric ulcer is 2:1 or less.
3. Chronic gastric ulcer is usually single; 90% are situated on the lesser curve within the antrum or at the junction of body and antral mucosa.
4. Chronic duodenal ulcer usually occurs in the first part of the duodenum just distal to the junction of pyloric and duodenal mucosa; 50% are on the anterior wall.
5. Gastric and duodenal ulcers coexist in 10% of patients and more than one peptic ulcer is found in 10-15% of patients.

**Pathophysiology:**

**A**. H.pylori:

1. Peptic ulceration is strongly associated with H.pylori infection.
2. The prevalence in developed nations rises with age (in UK about 50% of people over 50 years are infected and up to 90% in developing world).
3. The infections are probably acquired in childhood.
4. The vast majority of colonized people remain healthy and asymptomatic and only a minority develop clinical disease.
5. Around 90% of duodenal ulcer patients and 70% of gastric ulcer patients are infected with H.pylori, the remaining 30% of gastric ulcers are due to NSAIDs.

6. H.pylori is Grain-negative and spiral, and her multiple flagella at one end making it motile, allowing it to barrow and live deep beneath the mucus layer closely adherent to the epithelial surface. Here the surface PH is close to neutral and any acidity is buffered by the organism's production of the enzyme urease. This produce ammonia from urea and raises the PH around the bacterium.

7. The bacteria spread by person to person contact

via gastric refluxate or vomit.

8. H.pylori exclusively colonises gastric type epithelium and only found in duodenum in association with patches of gastric metaplasia

- The bacterium stimulates chronic gastritis-9 by provoking a local inflammatory response in the underling epithelium. This depend on numerous factors , notably expression of bacterial cag A and vac A genes , lead to cell replication ,apoptosis , increased cell permeability . Host genetic polymorphisms are also important .

- In most people H.pylori causes antral gastritis associated with depletion of somatostatin (from D cells) and increased gastrin release from G-cells which stimulate parietal cell (Hcl). The role of H.pylori in the pathogenesis of gastric ulcer is less clear but it probably acts by .reducing gastric mucosal resistance

**B. NSAID:** impairment of mucosal defenses.

**C. Smoking:** increase risk of gastric cancer and to a lesser extent, duodenal ulcer

**Clinical features:**

1. Peptic ulcer is a chronic condition with a natural history of spontaneous relapse and remission lasting for decades, if not for life.
2. Duodenal and gastric ulcers share common symptoms which can be considered together.
3. The most common presentation is recurrent abdominal pain with three characteristics: localization to epigastrium, relationship to food and episodic occurrence.
4. Vomiting in about 40% of ulcer subjects; persistent daily vomiting suggest gastric out let obstruction.

5. In one-third of cases the history is less characteristic (especially in elderly subjects under treatment with NSAID). In these patient the pain is absent or slight as a vague sense.
6. Occasionally, the only symptoms are anorexia and nausea, or a sense of undue repletion after meals.
7. In some patients the ulcer is completely (silent), presenting for the first time with anaemia from chronic undetected blood loss, as an abrupt haematamesis or as acute perforation; or recurrent acute bleeding without ulcer pain between the attacks.
8. The diagnostic value of individual symptoms for peptic ulcer disease is poor; the history is therefore a poor predicator of the presence of an ulcer.

**Investigations:**

1. Endoscopy is the preferred investigation gastric ulcer may occasionally be malignant and therefore must always be biopsied and followed up to ensure healing.

2. Patient should also be screened for H.pylori infection by non-invasive tests which include serology (which is rapid, and good for population studies but lacks sensitivity and specificity, and cannot differentiate current from past infection), $^{13}C$- urea breath tests (high sensitivity and specificity but requires expensive mass spectrometer), faecal antigen test (cheap, specific ">95%", but poor acceptability), or invasive (antral biopsy) which include histology (sensitive and specific but it takes several days to process and false negative can occur), rapid urease tests (cheap, quick, specific (>95%) but the sensitivity (85%)), microbiological culture (Gold standard and defines antibiotics sensitivity but it slow and laborious and lacks sensitivity. Overall, breath tests are best because of their accuracy, simplicity and non-invasiveness.

- **Management:**

The aims of management are to relieve symptoms, induce healing and prevent recurrence. H.pylori eradication is the cornerstone of therapy for peptic ulcers, as this will successfully prevent relapse and eliminate the need for long-term therapy in the majority of patients.

**1. H.pylori eradication:**

All patients with proven acute or chronic duodenal ulcer disease and gastric ulcers who are H.pylori positive should be offered eradication as primary therapy.

- Treatment is based upon PPI taken simultaneously with two antibiotics (from amoxiciline, clarithromycin and metronidazole) for 7 days. Success is achieved in over 90% of patients side effects of H.pylori eradication therapy include (Diarrhoea, mild clostridium deficits associated colilis, flushing + vomiting when taken with alcohol "metronidazole", nausea + vomiting, Abd. Gramp, headache, rash) second-line therapy for patients who remain infected after initial therapy "after two treatments", the choice lies between a third attempt with quadruple therapy (bismuth, PPI and two antibiotics) or long term maintenance therapy with acid suppression.

Co-prescription of a PPI along with the NSAID is advised but is not always necessary for patients being given low dose aspirin in whom the risk of ulcer complication is lower.

:Other indication for H.pylori eradication include

1. **Definite**: peptic ulcer, H.pylori positive dyspepsia, MALToma.
2. **Uncertain**: family history of gastric cancer, non-ulcer dyspepsia, long-term NSAID users.
3. **Not indicated**: gastro-oesophageal reflux disease, Asymptomatic.

**2. General measures**: cigarette smoking, aspirin and NSAID, should be avoided alcohol in moderation is not harmful and no special dietary advice is required.

**3. Maintenance treatment**: not necessary after successful H.pylori eradication. For the minority who do require it, the lowest effective dose of PPI should be used.

**4. Surgical treatment**: indication include perforation, haemorrhage, complication, e.g: gastric outflow obstruction, recurrent ulcer following gast. Surgery

## Complications of gastric resection or vagotomy:

1. **Dumping: rapid gastric emptying** leads to distension of the proximal small intestine as the hypertonic contents draw fluid into the lumen leads to abdominal discomfort, diarrhea after eating, autonomic reflexes ( flushing, palpitations, sweating, tachycardia & hypotension). Patients should therefore avoid large meals with high carbohydrate.

2. **Bile reflux gastritis**: this is usually asymptomatic but dyspepsia can occur. Symptomatic treatment with aluminum – containing antiacid or sucralfate may be effective.

**3. Diarrhea and maldigestion**: usually occurs 1-2 hours after eating due to poor mixing of food in stomach, with rapid emptying, inadequate mixing with pancreatic secretions, reduced small intestinal transit times and bacterial over growth, may lead to mal-absorption.

Diarrhea often responds to dietary advice to eat small, dry meals with a reduced intake of refined carbohydrates, antidiarrheal drugs such as codeine phosphate (15-30mg) 4-6 times a day) or loperamide (2 mg after each loose stool) are helpful.

4. **Weight loss**: due to small gastric remnant, diarrhea and mild steatorhoea.

**5. Anaemia**: iron deficiency, (B12 & folic acid deficiency less common).

**6. Metabolic bone disease**: due to calcium & vitamin D malabsorption.

**7. Gastric cancer**: due to hypochlorhydria, duodenogastric reflux of bile, smoking and H.pylori infection. However, endoscopic surveillance is not indicated following gastric surgery.

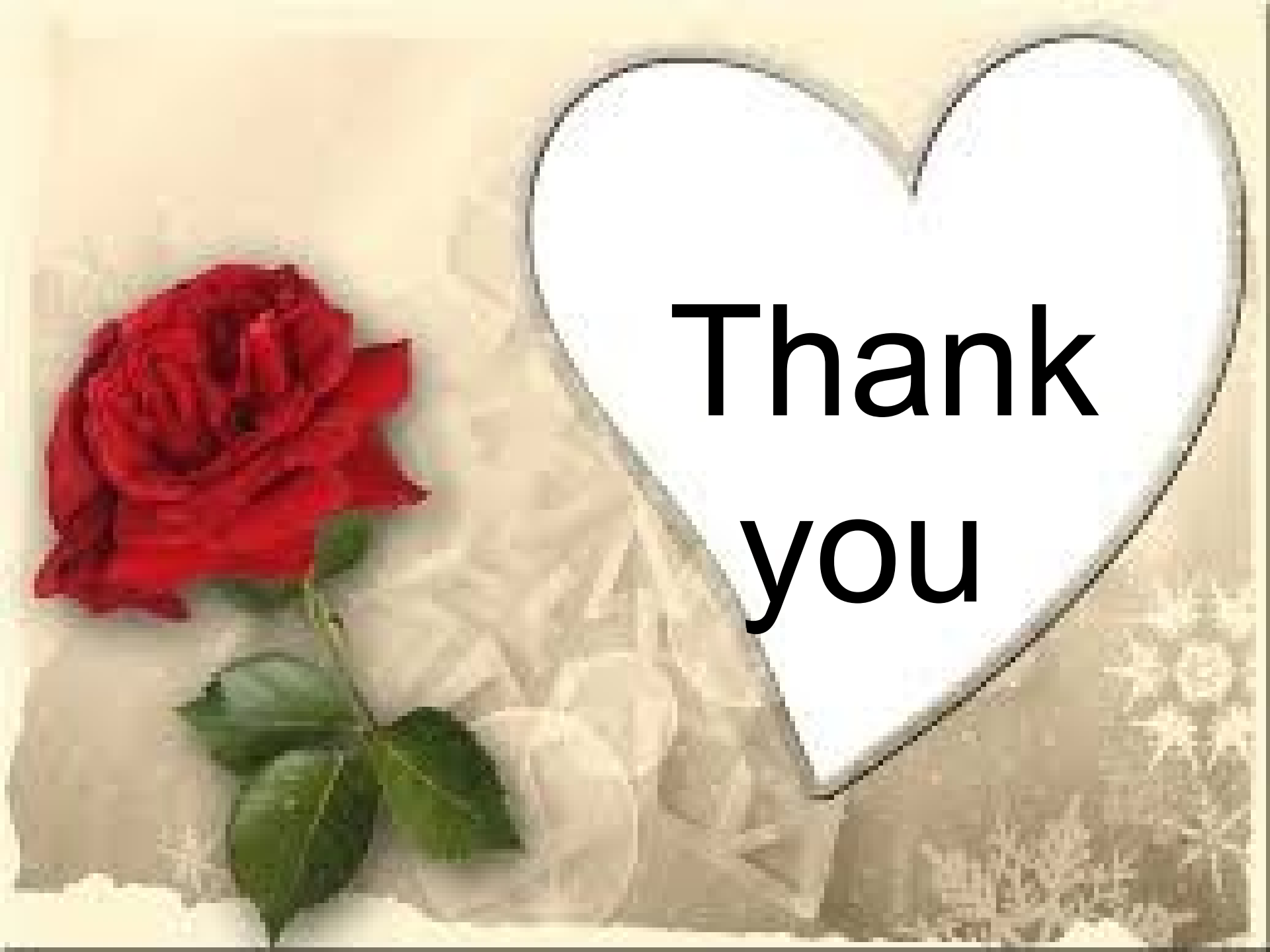
**Complications of peptic ulcer disease:**

**1. Perforation**: more common in duodenal than in gastric ulcers and on the anterior wall. Abdominal pain start in the upper abdomen and rapidly becomes generalized. Shoulder tip pain may occur but recurrent epigastric pain is uncommon. In at least 50% of cases an erect chest x-ray shows free air beneath the diaphragm.

**2. Gastric outlet obstruction**: may be caused by fibrotic stricture from duodenal ulcer, oedema from ulcer, carcinoma of antrum, adult hypertrophic pyloric stenosis lead to nausea, vomiting and abdominal distension. A succession splash may be elicited 4 hours or more after the last meal or drink.

Visible gastric peristalsis is diagnostic for gastric outlet obstruction. Loss of gastric contents leads to dehydration with low chloride and potassium and raised serum bicarbonate and urea. Nasogastric aspiration of at least 200 ml of fluid from the stomach after an overnight fast suggest the diagnosis.

**3. Bleeding.**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you