



**Round :** ..... Internal medicine

**Group no :** ..... A4

**Done By :** ..... Batool ALzubaidi



# STOMACH DISORDERS

## I. PATHOPHYSIOLOGY

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- B. PEPTIC ULCER DISEASE (PUD)

## II. CLASSIC FINDINGS OF STOMACH DISORDERS

- A. GASTRITIS
- B. PUD

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- A. GASTRITIS
- B. PUD

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- B. ASSESS FOR EVIDENCE OF ALARM FEATURES
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## V. TREATMENT OF PANCREATIC DISORDERS

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- B. ERADICATE H. PYLORI
- C. MANAGEMENT OF COMPLICATIONS

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## I. PATHOPHYSIOLOGY

### A. GASTRITIS

#### 1. Pathophysiology:

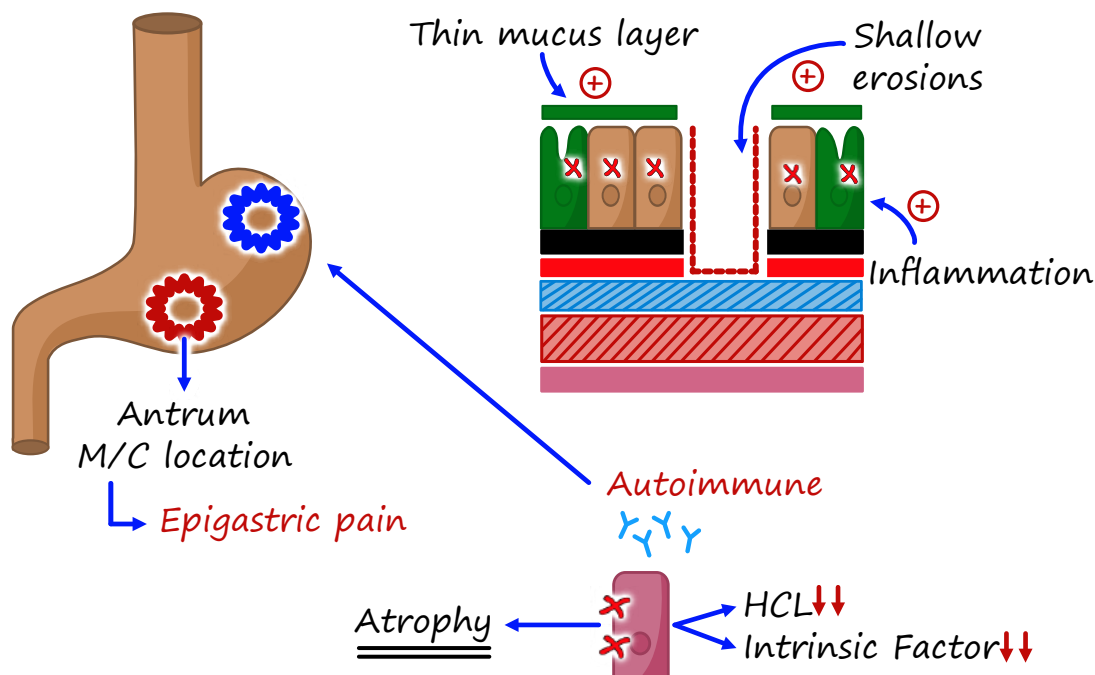
##### a) Acute Gastritis

- **↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection)** in the stomach → ↑Mucosal injury of stomach lining → ↑Inflammation of stomach lining → Small mucosal erosions may develop
  - The most common location for **acute gastritis** is in the **antrum of the stomach**

##### b) Chronic Gastritis

- **An autoimmune attack against the parietal cells** in the stomach lining → ↓HCL production and intrinsic factor → Atrophy and inflammation of the stomach lining
  - The most common location for **chronic gastritis** is the **fundus of the stomach**

## Stomach involvement



## 2. Causes of Acute Gastritis

### a) NSAIDs and Ethanol Use

- **↓Cyclo-oxygenase enzyme** activity in the stomach lining →  
↓Prostaglandin production →  
↑HCL acid production and  
↓Alkaline mucus production

### b) *H. pylori* Infection

- **↑Gastrin production** →  
↑HCL acid production
- **↑Urease** →  
↑NH<sub>3</sub> production →  
*H. pylori* survival → ↑Direct cytotoxicity (via toxin release)

### c) Stress Ulcers (Curling's and Cushing's)

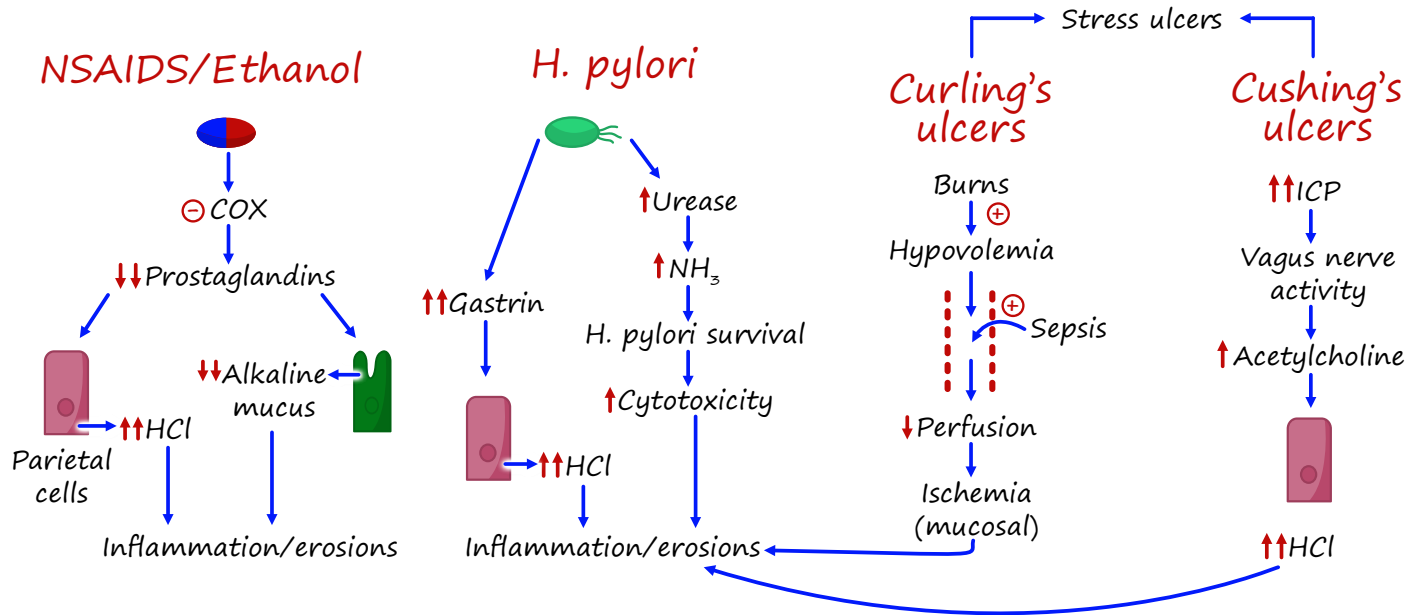
#### ○ Curling's Ulcer

(Occur via a different mechanism vs the rest)

- **Burns** → Hypovolemia → ↓Perfusion → Gastric ischemia → Mucosal lesions
- **Sepsis** → Hypotension → ↓Perfusion → Gastric ischemia → mucosal lesions

#### ○ Cushing's Ulcers:

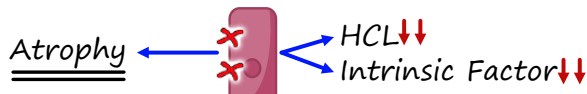
- **↑ICP** → ↑Vagus nerve activity → ↑Acetylcholine →  
↑HCL acid production



## 3. Causes of Chronic Gastritis

### Atrophic Gastritis

- This may be secondary to **HLA-B8 and HLA-DR3 mutations** → Trigger autoimmune reaction against parietal cells
- This may be due to **Chronic *H. pylori* infections** → Trigger ↑HCL acid production and ↑Direct cytotoxicity

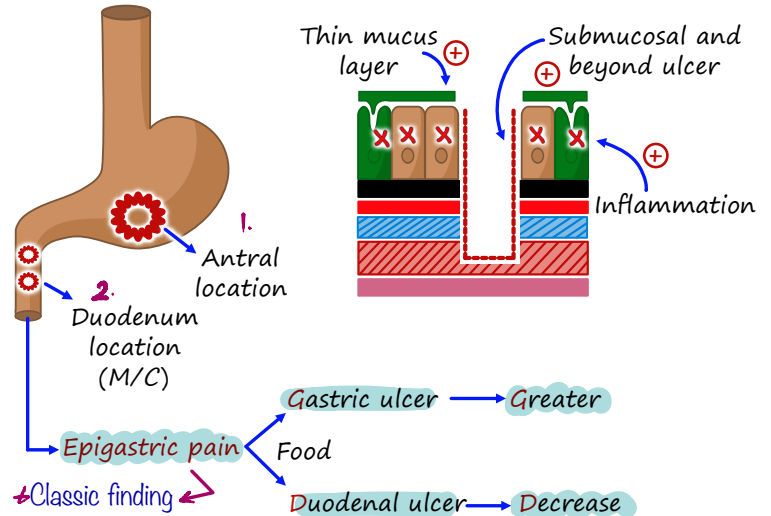


## B. PEPTIC ULCER DISEASE (PUD)

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### 1. Pathophysiology:

- **↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection)** in the stomach →
- ↑mucosal and submucosal **injury** of the stomach lining →
- ↑**inflammation** of the stomach lining →
- Deep ulcers extending past the submucosa may occur
  - The most common locations for **PUD** are the **duodenum** and the **antrum of the stomach**



### 2. Causes of Peptic Ulcer Disease (PUD):

#### a) NSAIDs and Ethanol Use

- **↓Cyclo-oxygenase enzyme activity in the stomach lining** →
- ↓Prostaglandin production →
- ↑HCL acid production and ↓Alkaline mucus production
- Rich of bicarbonate » to neutralize the hydrochloric acid and protect the cells
- NSAIDS/Ethanol**
- Causes the inflammation and injury to all the mucosa lining which can lead to erosion
- By parietal cells: ↑↑HCL, ↓↓Mucus → Ulcers/inflammation

#### b) H. pylori Infection

- **↑Gastrin production** →
- ↑HCL acid production
- **↑Urease** → **↑NH<sub>3</sub> production** →
- H.pylori **survival** →
- ↑Direct **cytotoxicity** (via toxin release)
- Direct **H. pylori (M/C)** stimulates effect on parietal cells → ↑Gastrin → ↑HCL → Ulcers/inflammation
- It breaks down urea to ammonia → ↑Urease → ↑NH<sub>3</sub> → It makes the PH of the stomach more alkaline → ↑H. pylori survival → ↑Cytotoxicity → Ulcers/inflammation

#### c) Zollinger Ellison

- **Pancreatic tumor (Gastrinoma)** →
- ↑Gastrin production →
- ↑HCL acid production
- Commonly precipitate **refractory duodenal ulcers**
- Gastrinoma**: Pancreas → ↑↑Gastrin → ↑↑HCL → Refractory duodenal ulcers/inflammation
- Zollinger-Ellison syndrome (ZES)**

It has particular types of toxins that affect the permeability and directly injure the actual cells of mucosa

## II. CLASSIC FINDINGS OF STOMACH DISORDERS

### A. GASTRITIS

#### 1. Epigastric Abdominal Pain

- Inflammation of the stomach → Known as gastritis, typically causes pain in the epigastric region, which may also radiate to the back

### B. PUD

#### 1. Epigastric Abdominal Pain

- Inflammation of the stomach → Known as gastritis, typically causes pain in the epigastric region, which may also radiate to the back
- **This pain changes with meals**
  - **Gastric ulcers are Worse with Meals**
    - Food → ↑HCL production in the stomach
  - **Duodenal ulcers are Better with Meals**
    - Food → ↑HCL production in the stomach, and the acidic chyme has not reached the duodenum yet



### III. COMPLICATIONS OF STOMACH DISORDERS

#### A. GASTRITIS

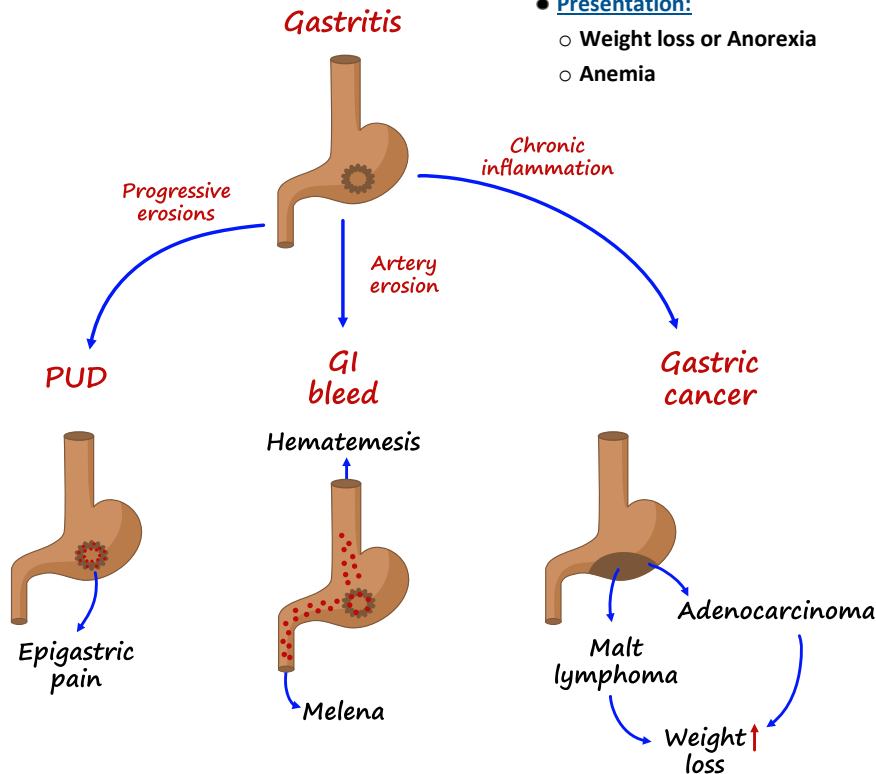
##### 1. Upper GI Bleed

###### ● Pathophysiology:

- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach →
- ↑Mucosal injury of stomach lining → ↑Inflammation of stomach lining → Small mucosal erosions may develop → These erosions may ulcerate into a small mucosal/submucosal artery → **Blood leaks into the stomach lumen**

###### ● Presentation:

- Hematemesis
- Melena
- Anemia



##### 2. Gastric Cancer

###### ● Pathophysiology:

- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach →
- ↑Mucosal injury of stomach lining →
- ↑Repeated inflammation of stomach lining →
- **↑Risk of Dysplasia**
  - High risk in Atrophic gastritis and H.pylori infections

###### ● Presentation:

- Weight loss or Anorexia
- Anemia

##### 3. Anemia

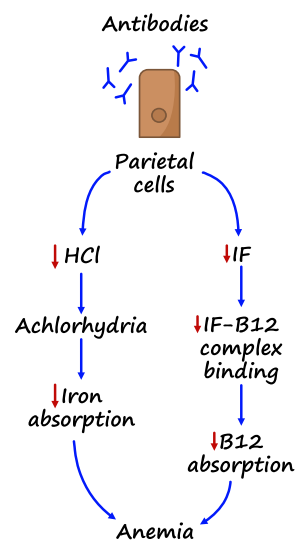
###### ● Pathophysiology:

- **Autoantibodies** attack parietal cells →
- ↓HCL acid production and ↓Intrinsic factor (IF) production →
- ↓Chloride in the intestinal lumen leads to ↓Iron absorption, and ↓IF leads to ↓B<sub>12</sub> absorption →
- **↓Iron and B<sub>12</sub>** present in the bone marrow for RBC production

###### ● Presentation:

- ↓Hgb and Hematocrit → **Supports Anemia**
  - The presence of ↓B<sub>12</sub>, Iron, and IF/Parietal cell Autoantibodies → Supports **Autoimmune (Atrophic Gastritis)** as the cause

###### Autoimmune gastritis



## B. PUB

### 1. Upper GI Bleed

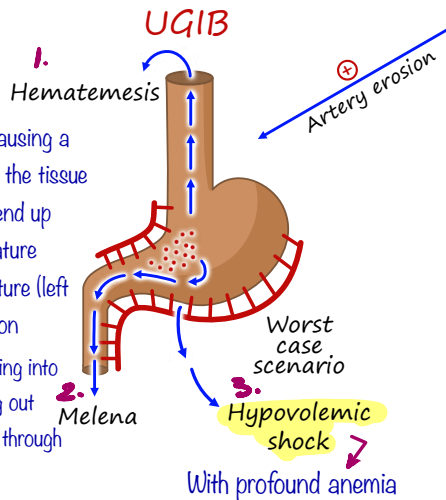
#### • Pathophysiology:

- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach → ↑Mucosal injury of stomach lining → ↑Inflammation of stomach lining → Small mucosal erosions may develop → These erosions may **ulcerate into a small mucosal/submucosal artery** → **Blood leaks into the stomach lumen**

#### • Presentation:

- Hematemesis
- Melena
- Anemia

As patients start to develop the ulcer causing a lot of inflammation and eroding through the tissue wall especially the submucosa » it can end up causing artery erosion on greater curvature (gastrooduodenal artery) or lesser curvature (left gastric artery) which is the most common. When erosion happens they will start bleeding into the stomach » it can either cause vomiting out blood or it can get oxidized then move out through anus causing darker stool (black)



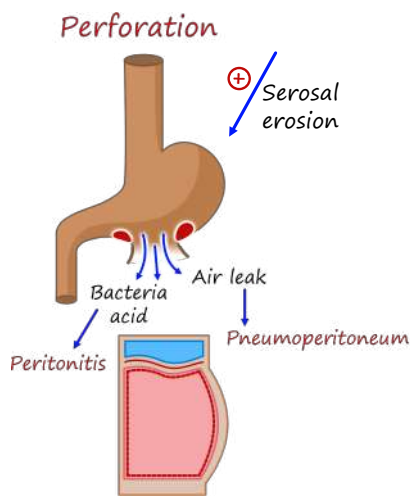
### 2. Gastrointestinal Perforation

#### • Pathophysiology:

- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach → ↑mucosal and submucosal injury of stomach lining → ↑inflammation of stomach lining → **Deep ulcers extending past the submucosa to the serosa** → **Most outer layer** → **Air and gastric contents leak into the peritoneal cavity**

#### • Presentation:

- Severe abdominal pain and distention
- Peritoneal signs with concerns of Peritonitis
  - Guarding, rigidity, rebound tenderness
- ↑Risk of peritonitis and Sepsis
  - Perforation → Air and gastric contents leak into the peritoneal cavity → Inflammation of the peritoneal lining → **↑WBC, ↑Fever**



**Supports Peritonitis** (along with peritoneal signs) + **Hypotension** may also support **Septic Shock**

**SIRS**

1. Temperature >38°C or <36°C
2. Heart rate >90 beats per minute
3. Respiratory rate >20 breaths per minute or PaCO<sub>2</sub> <32 mmHg
4. White blood cell count >12,000/mm<sup>3</sup> or <4,000/mm<sup>3</sup> or >10% Immature (band) forms

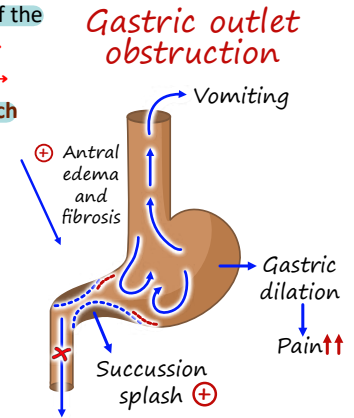
### 3. Gastric Outlet Obstruction

#### • Pathophysiology:

- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach → ↑Mucosal and submucosal injury of stomach lining → ↑Inflammation of stomach lining → Repair response leads to **fibrosis of the inflamed tissue near the antrum** → **Narrows the antrum and pylorus** → **Impedes the movement of stomach contents into the duodenum**

#### • Presentation:

- Nausea and vomiting
- Abdominal distension
- Weight loss secondary to food aversion secondary to N/V and abdominal pain
- **Succussion Splash**
  - The succussion splash sound, indicative of retained gastric contents, is heard when the **patient rolls from side to side**, suggesting an **obstruction** preventing chyme from passing into the duodenum



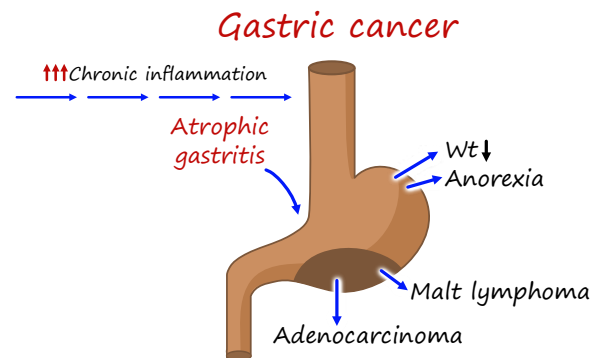
### 4. Gastric Cancer

#### • Pathophysiology:

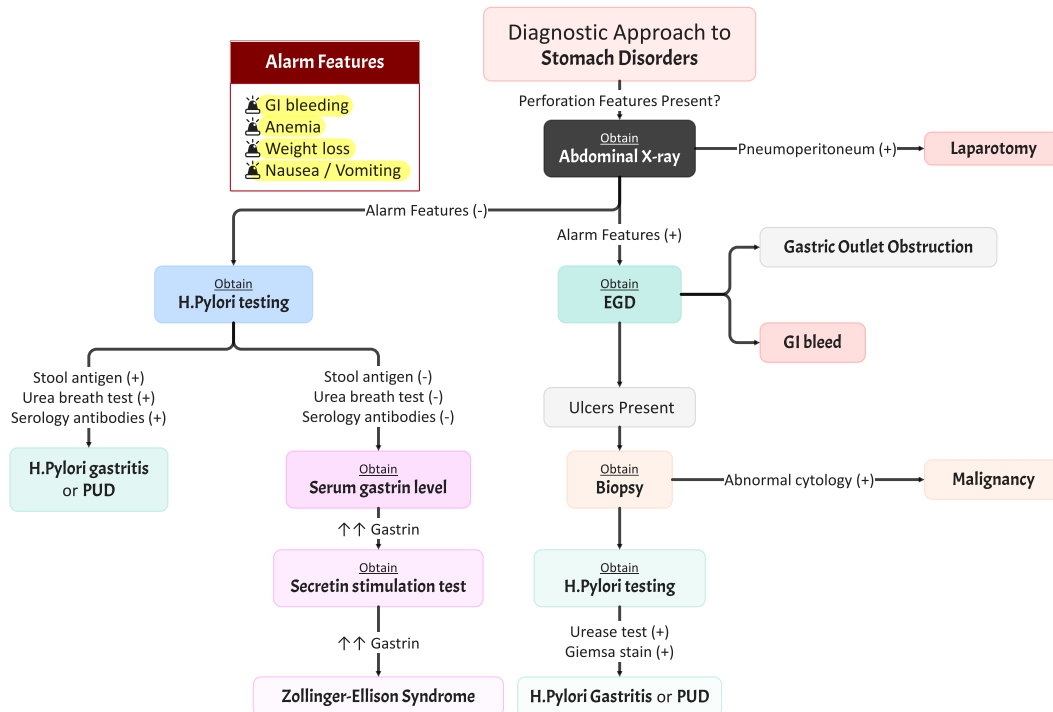
- ↑HCL acid production (erosive) and ↓Alkaline mucus production (less protection) in the stomach → ↑Mucosal injury of stomach lining → ↑Repeated inflammation of stomach lining → **↑Risk of Dysplasia**
  - **Very high risk for H.pylori infections**

#### • Presentation:

- Weight loss or Anorexia
- Anemia



## IV. DIAGNOSTIC APPROACH TO STOMACH DISORDERS



### A. Assess for Evidence of GASTROINTESTINAL PERFORATION

#### Obtain Abdominal X-Ray

- **Indications:**
  - **History of PUD** with features such as severe abdominal pain, distention, guarding, rigidity, and rebound tenderness suggesting perforation
- **Abnormal Findings:**
  - The presence of **"air underneath the diaphragm"** suggests a pneumoperitoneum
    - **Pneumoperitoneum along with HD instability** should be further evaluated via an **exploratory laparotomy** to provide an intraoperative diagnosis of PUD-related perforation as well as allow simultaneous treatment

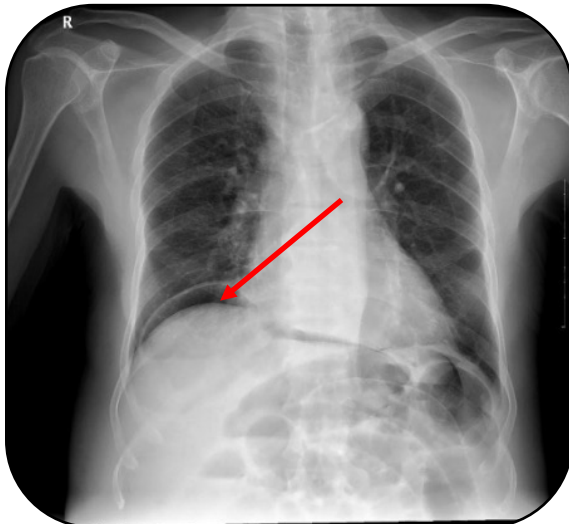


FIGURE 1. XRAY INDICATING PNEUMOPERITONEUM

### B. Assess for Evidence of ALARM FEATURES

#### Obtain History to Assess for Alarm Features

#### a) GI Bleeding

- **History of gastritis or PUD** along with **melena** or **hematemesis** suggests the presence of a **UGIB** and should be further evaluated via an EGD

#### b) Anemia

- **History of gastritis or PUD** along with **anemia** suggests the possibility of **UGIB** (especially if associated with melena or hematemesis) or **Gastric Cancer** (especially if associated with weight loss or anorexia) and should be further evaluated via an EGD

#### c) Weight Loss

- **History of gastritis or PUD** along with **weight loss** suggests the possibility of **Gastric Cancer** (especially if associated with anemia) or **Gastric outlet obstruction** (especially if associated with N/V and **Succussion splash**) and should be further evaluated via an EGD

#### d) Nausea and Vomiting

- **History of gastritis or PUD** along with persistent **nausea** and **vomiting** suggests the possibility of **Gastric outlet obstruction** (especially if associated with **succussion splash**) and should be further evaluated via an EGD



## C. ASSESS FOR CAUSES OF STOMACH DISORDERS IF THERE ARE ALARM FEATURES PRESENT

### 1. Obtain EGD

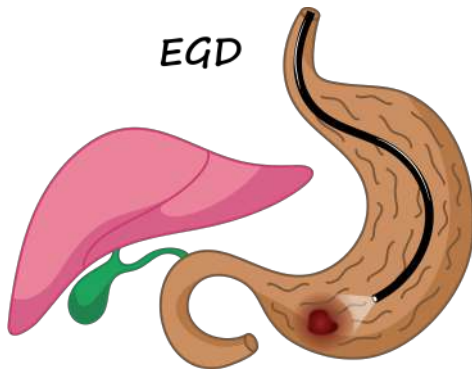
#### • Purpose of EGD:

##### i) Visually Identify Complications such as:

- Gastric Outlet Obstruction
- Upper GI bleed
- Gastric or duodenal ulcers

##### ii) Biopsy of Ulcers [Page 8](#)

- Identify H. Pylori infection-related ulcer vs Gastric cancer-related ulcer
  - Obtain invasive H.pylori testing via biopsy of the ulcer
  - Perform cytology examination via biopsy of the ulcer



#### • Abnormal Findings:

##### i) Gastric Outlet Obstruction

- Narrowed lumen near the **antrum and pylorus**

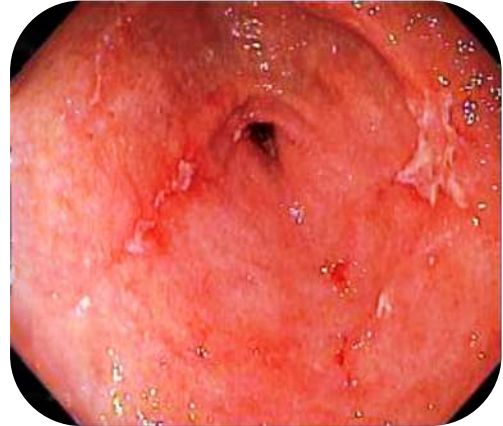


FIGURE 2. EGD INDICATING GASTRIC OUTLET OBSTRUCTION

##### ii) Upper GI Bleed

- Presence of bleeding from a duodenal or gastric ulcer

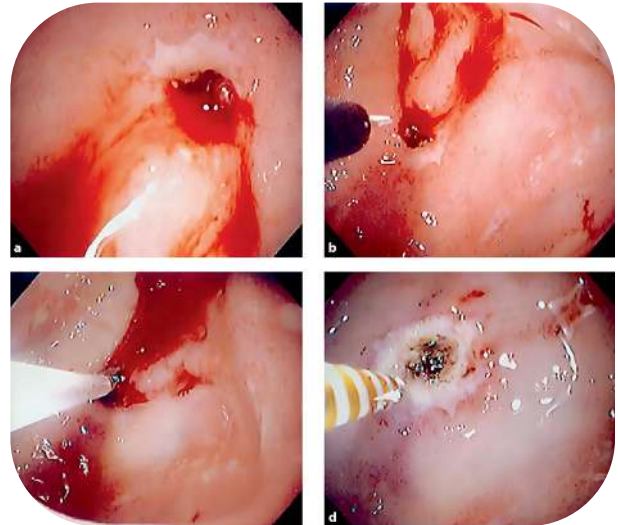


FIGURE 3. EGD INDICATING GI BLEED

##### iii) Gastric or Duodenal Ulcers

- H. pylori infection-related ulcer



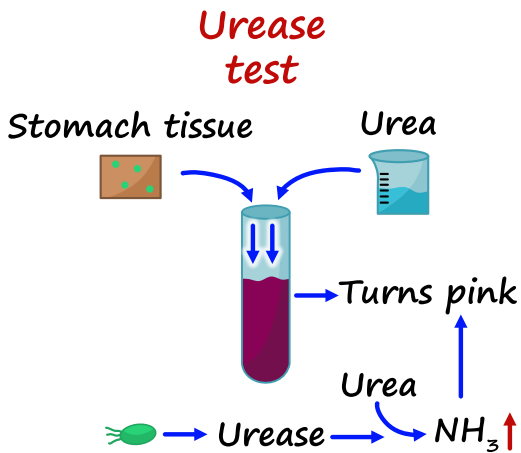
FIGURE 4. EGD REVEALING AN ULCER



## 2. Obtain a Biopsy for the Ulcers

### a) Urease Test on Biopsy Sample

- Tissue containing H.pylori is placed into a urea-containing solution → If H.pylori is present, it should produce Urease & break down urea → Trigger the production of  $\text{NH}_3$  →  $\uparrow \text{pH}$  which would turn the solution color pink if a pH indicator is present in the solution
  - High specificity → This is a quick test allowing real-time identification of H. pylori



### b) Giemsa Stain on the Biopsy Sample

- This is a stain added to the tissue sample, and under microscopy, it should aid in visualizing the presence of the curved and rod-shaped bacteria (such as H.pylori) under the microscope
  - High specificity → Requires time for a pathologist to review
    - However, this is the gold standard

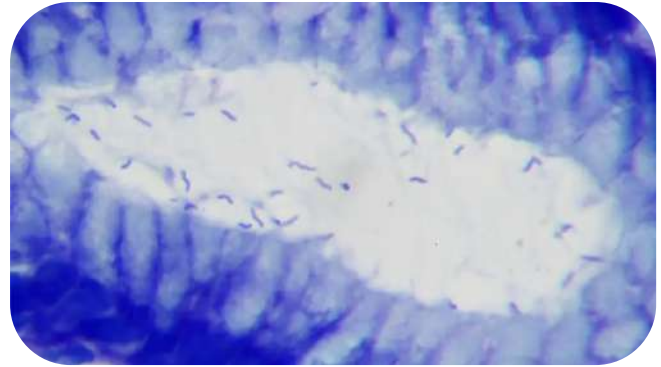


FIGURE 5. H.PYLORI GIEMSA STAIN

### c) Gastric Cancer-Related Ulcers

#### (Irregular Appearing Ulcers)

- A biopsy to confirm if cytology indicates Adenocarcinoma-Related cells in ulcer



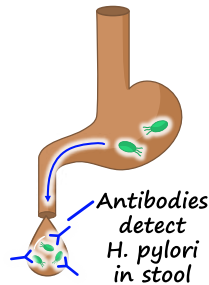
## D. ASSESS FOR CAUSES OF STOMACH DISORDERS IF THERE ARE NO ALARM FEATURES PRESENT

### 1. Obtain Non-invasive H. Pylori testing

- **Types of testing:**
  - Stool antigen test
  - Urea breath test
  - Serology antibodies
- **Indications:**
  - Suspicion of H.pylori-related Gastritis or PUD
- **Abnormal Findings:**

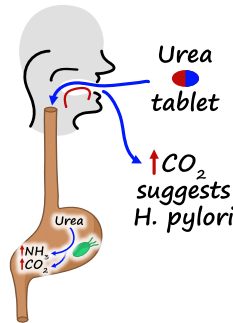
#### a) Stool Antigen Test

- Uses enzyme immunoassay to **detect H.pylori** antigens passed in the stool
  - Effective for initial diagnosis, but most often utilized to **confirm eradication post-treatment**



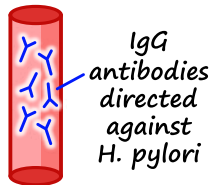
#### b) Urea Breath Test

- **The patient ingests Radiolabeled Urea tablet** →  
If H.pylori is present it will release urease which will **breakdown urea into CO<sub>2</sub>** (which is radiolabeled) → CO<sub>2</sub> is often times released from the stomach and can be exhaled and detected, thus a **higher than normal CO<sub>2</sub> level suggests H.pylori infection**
  - **Higher sensitivity and specificity** than Stool antigen test and serology antibodies. It is highly effective for initial diagnosis and to confirm eradication post-treatment



#### c) Serology Antibodies

- **Detects the presence of IgG antibodies directed against H.pylori in the blood**
  - Decent sensitivity, but **very poor specificity**; → This test struggles with being able to identify an active vs past infection (given antibodies can be elevated years after treatment)  
→ **This test should be avoided as there are better options**



### 2. Obtain Gastrin level

- **Indications:**
  - Negative H.pylori testing (e.g. urea breath and stool antigen test) with concern of **Zollinger-Ellison Syndrome** given refractory PUD
- **Abnormal Findings:**

#### ↑↑ Serum Gastrin Level

- This suggests the presence of a Gastrinoma
  - This should be confirmed with a Secretin stimulation test
    - **Secretin is administered** which should ↓gastrin production normally so the presence of persistent **↑↑serum gastrin despite secretin administration suggests a Gastrinoma** and as such imaging should be done to identify the Gastrinoma



## V. TREATMENT OF STOMACH DISORDERS

| Goal of Treatment          | Treatment                                                                                     |
|----------------------------|-----------------------------------------------------------------------------------------------|
| Acid Suppression therapy   | - PPI                                                                                         |
| Cytoprotection therapy     | - Sucralfate or Misoprostol                                                                   |
| Eradicate <i>H. pylori</i> | - Clarithromycin + Amoxicillin (metronidazole if PCN allergy)<br>- Monitor stool antigen test |
| Complication management    | - EGD (GI bleed)<br>- Surgery (Perforation)<br>- Dilation (GOO)                               |

### A. ACID SUPPRESSION & CYTOPROTECTION THERAPY

- **Treatment:**
  - **Acid Suppression Therapy**
    - Proton Pump Inhibitor (PPI)
  - **Cytoprotection Therapy**
    - Sucralfate or Misoprostol
- **Indications:**
  - Epigastric pain secondary to PUD or Gastritis
  - UGIB secondary to PUD or Gastritis
- **Purpose:**
  - PPI → ↓ HCL production → ↓ Progression of erosions and ulcers
  - Sucralfate or Misoprostol → ↑ Prostaglandin production → ↑ Alkaline mucus production → Protects mucosal lining from the further progression of erosions and ulcers
- **Monitoring:**
  - Improvement/Resolution of epigastric pain
  - Improvement in UGIB features (improvement in anemia, hematemesis, or melena)

### B. ERADICATE *H. PYLORI*

- **Treatment:**
  - **Antibiotics** (e.g. Clarithromycin + Amoxicillin)
- **Indications:**
  - Diagnosed *H. pylori* infection via non-invasive (e.g. urea breath or stool antigen test) or Invasive testing (e.g. urease test or Giemsa stain)
- **Purpose:**
  - Eradicate *H. pylori* → ↓ HCL production and ↓ Direct cytotoxic effects → ↓ Progression of inflammation, erosions, and ulcers
- **Monitoring:**
  - Eradication of *H. pylori* infection via:
    - Absence of *H. pylori* detection via Stool antigen tests or ↓ ↓ CO<sub>2</sub> detection after urea breath test

### C. MANAGEMENT OF COMPLICATIONS

#### 1. Upper GI Bleed

- **Treatment:**
  - **Medical management**
    - Transfusion of PRBCs for Hgb > 7 or > 8 if active myocardial ischemia
    - IV PPI
  - **Surgical management**
    - EGD guided hemostasis via cauterization, clipping, or epinephrine injection
- **Indications:**
  - Presence of UGIB such as hematemesis, melena, or anemia with no obvious source other than GI bleed
- **Purpose:**
  - To achieve hemostasis
- **Monitoring:**
  - Monitoring Hgb/Hematocrit for improvement in anemia
  - Improvement in hematemesis, or melena
  - Improvement in Hemodynamics if was previously HD unstable



## 2. Gastrointestinal Perforation

- **Treatment:**

- **Medical therapy**

- Nothing by Mouth "NPO"
- IVF
- Antibiotics (e.g. Cefepime, Piperacillin-tazobactam, meropenem +/- metronidazole)
  - Especially if peritoneal signs, fever, leukocytosis, or new HD instability

- **Surgical Therapy**

- Exploratory Laparotomy or Laparoscopy
  - **Graham Patch**
    - Use of the omentum to close off the perforation in the stomach or duodenum

- **Indications:**

- Pneumoperitoneum present on imaging
- Peritoneal signs, fever, leukocytosis, and **HD instability** with suggestion of bowel perforation progressing to peritonitis/sepsis

- **Purpose:**

- To patch the perforation in the stomach or duodenum

- **Monitoring:**

- Monitor WBC, Temperature, and BP to assess for progression to septic shock
- Monitor Intrabdominal pressures postoperatively to assess for abdominal compartment syndrome

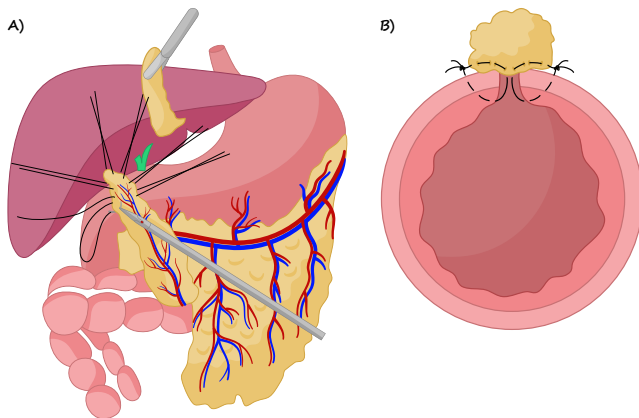


FIGURE 6. GRAHAM PATCH FOR PERFORATION

## 3. Gastric Outlet Obstruction

- **Treatment:**

- **Medical Management**

- NPO and NGT decompression if severe nausea or vomiting
- IVF while NPO to maintain fluid balance

- **Surgical Management**

- Endoscopic Dilation of gastric outlet obstruction
  - If refractory to dilation can consider Gastrojejunostomy to divert contents into the jejunum rather than the duodenum

- **Indications:**

- Diagnosis of gastric outlet obstruction with persistent vomiting and weight loss

- **Purpose:**

- To open up the antrum/pylorus lumen to ensure easy movement of chyme from the stomach into the duodenum

- **Monitoring:**

- Improvement in nausea and vomiting and appropriate appetite

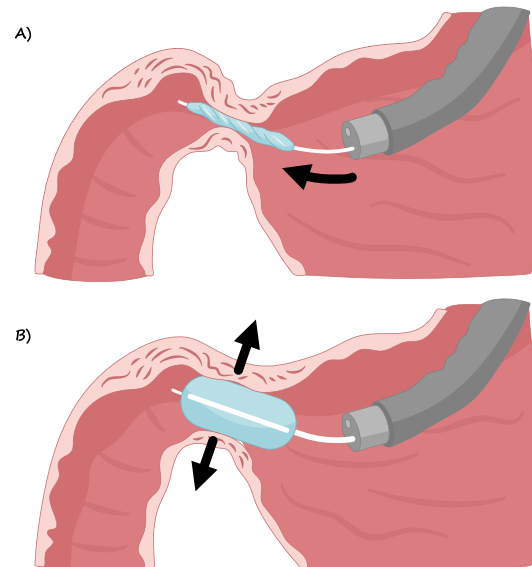


FIGURE 7. GASTRIC OUTLET OBSTRUCTION DILATATION

