Gastritis: Causes, Classification, Diagnosis and Treatment

Gastritis is defined as inflammation of the gastric mucosa—the mucous membrane lining the inner surface of the stomach. Under normal circumstances, the stomach is well protected against its own acidic environment by a mucosal barrier. However, various endogenous and exogenous factors can disrupt this protective layer, leading to mucosal injury and subsequent inflammation.

Etiology of Gastritis

Gastritis has multifactorial causes, including:

- **Infectious Agents**
  - *Helicobacter pylori* infection (most common cause of chronic non-erosive gastritis)
  - Viral infections (e.g., Cytomegalovirus in immunocompromised hosts)
- **Medications**
  - *Nonsteroidal anti-inflammatory drugs (NSAIDs)* – inhibit prostaglandin synthesis, reducing mucosal protection
- **Alcohol and Dietary Factors**
  - Excessive alcohol intake and consumption of spicy or contaminated foods
- **Stress-Related Mucosal Disease**
  - Acute stress (e.g., major surgery, burns, sepsis) can result in ischemia-induced acute erosive gastritis
- **Autoimmune Conditions**
  - Autoimmune gastritis leads to chronic atrophic gastritis with potential vitamin B12 deficiency
- **Parasitic and Allergic Causes**
  - Eosinophilic gastritis can result from parasitic infections like roundworm and food allergens

Predisposing Risk Factors

- Alcohol abuse
- Rapid or excessive food intake
- Radiation therapy
- Uremia
- Poorly preserved or contaminated food
- Highly seasoned or spicy food

Classification of Gastritis

1. **Based on Mucosal Injury:**
   - **Erosive Gastritis:** Involves superficial mucosal erosions, often with bleeding
   - **Non-Erosive Gastritis:** Involves mucosal inflammation without erosion (e.g., *H. pylori*-associated gastritis)
2. **Based on Anatomical Location:**

    - **Cardia**
    - **Body (Corpus)**
    - **Antrum**
3. **Based on Histopathology:**
    - **Acute Gastritis:** Predominantly neutrophilic (PMN) infiltration
    - **Chronic Gastritis:** Lymphoplasmacytic infiltration, with glandular atrophy or intestinal metaplasia

Pathophysiology

Gastritis involves disruption of mucosal integrity by damaging agents:

- **Initial Response:**
    - Edema and hyperemia of the mucosa
    - Superficial erosions or ulceration
- **Progression:**
    - Inflammatory infiltration
    - Mucosal atrophy and loss of secretory function

In chronic forms, the destruction of **G cells** (antral gastritis) decreases **gastrin** secretion, while destruction of **oxyntic glands** (corpus gastritis) impairs production of **acid, pepsin,** and **intrinsic factor**—contributing to vitamin B12 deficiency.

Erosive Gastritis

Definition: Mucosal erosions caused by breakdown of protective barriers.

Etiologies:

- NSAIDs
- Alcohol
- Stress (e.g., ICU patients)
- Radiation
- Viral infections (e.g., CMV)
- Mechanical trauma (e.g., nasogastric tubes)

Clinical Presentation:

- Often asymptomatic
- Possible symptoms: nausea, vomiting, dyspepsia
- Signs of upper GI bleeding: hematemesis, melena, or positive NG aspirate
- Lesions may occur as early as 12 hours after the insult

Acute Stress Gastritis

A subset of erosive gastritis occurring in **critically ill patients** (approx. 5% incidence).

Risk factors include:

- Mechanical ventilation > 48 hours
- CNS injury or burns (Cushing and Curling ulcers)
- Coagulopathy
- Shock, sepsis, or multi-organ failure

Pathophysiology:

- Splanchnic hypoperfusion leads to impaired mucosal defense and ulceration
- Increased acid secretion contributes in some cases

Diagnosis

Gold Standard: Esophagogastroduodenoscopy (EGD)

- Reveals mucosal inflammation, erosions, and bleeding
- Biopsy may help identify *H. pylori*, CMV, or histologic features

Management

For Acute Gastritis:

- **Initial Care:**
  - *NPO* (nil per os) until symptoms subside
  - IV fluids for hydration
  - Bland diet upon resumption of oral intake
  - **If Bleeding is Present:**
  - **Endoscopic Hemostasis** (e.g., clips, cautery)
  - **Acid suppression therapy**:
  - *Proton pump inhibitors (PPIs)* – first-line (e.g., pantoprazole IV)
  - *H2 receptor blockers* as alternatives
  - **Severe bleeding:** IV fluids, blood transfusion if needed
- **Surgical intervention** (e.g., gastrectomy) as last resort
  - **Angiographic embolization** rarely effective due to rich collateral supply

For Chronic Cases:

- Remove offending agent (e.g., NSAIDs)
- Treat *H. pylori* if present (triple or quadruple therapy)
- Long-term acid suppression if indicated

Prevention of Stress Gastritis

Candidates for Prophylaxis:

- ICU patients with:
  - Mechanical ventilation > 48 h
  - Coagulopathy
  - Sepsis, shock, burns, trauma
  - Hepatic or renal failure

- History of peptic ulcer disease or GI bleeding

Prophylactic Measures:

- IV **PPIs** or **H2 blockers**
- Maintain **intragastric pH > 4.0**