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A review on antacids therapy for peptic ulcer disease

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Abstract:

Antacids appear to be effective ulcer healing agents with efficacies resembling those of other antiulcer drugs. In peptic ulcer disease, antacids present a therapeutic effect by neutralizing gastric acid and reducing acid delivery to the duodenum. Clinical impression strongly suggests that antacids relieve pain in peptic ulcer but objective confirmation is lacking. A low calorie, low fat, synthetic dietary product is suggested as a substitute for milk products in the treatment of certain patients with peptic ulcer. The effect of antacids on the stomach is due to partial neutralisation of gastric hydrochloric acid and inhibition of the proteolytic enzyme, pepsin. Antacids are commonly used self-prescribed medications. They consist of calcium carbonate and magnesium and aluminum salts in various compounds or combinations.

Keywords: Antacids, peptic ulcer disease, gastric acid, aluminium hydroxide,calcium carbonate.

1. Introduction:

Peptic ulcer is a sore in the gastrointestinal lining resulting in the breakdown of the mucosal and submucosal layers [1]. Peptic ulcers come about due to the peptic acid injury of the gut, leading to the breakdown of the digestive system's mucosal layer, with injury greater than 3-5 mm. This injury can occur along the oesophagus (oesophageal ulcer), stomach walls (gastric ulcer), and the duodenum (duodenal ulcer). It occurs due to rupturing of the mucosal layer's protective barrier for these three digestive system components. Individual susceptibility to Non-Steroidal Anti-inflammatory Drugs (NSAID) toxicity and H. pylori virulence determines the degree of damage to the mucosa layer. The mucosa layer has the unique ability to resist injury resulting from high peptic acid concentration, influx of bile, and pepsin [2]. The breakdown of this layer is due to the imbalance between the protective and aggressive factors of the mucosal layer Today, testing for Helicobacter pylori is recommended in all patients with peptic ulcer disease. Endoscopy may be required in some patients to confirm the diagnosis, especially in those patients with sinister symptoms. Today, most patients can be managed with a proton pump inhibitor (PPI).

Pathophysiology of peptic ulcer disease:

Peptic ulcer disease develops when gastric/duodenal mucosal defenses are overwhelmed by aggressive factors, leading to mucosal erosion that extends through the muscularis mucosa.

1. **Imbalance Between Aggressive and Defensive Factors**

A. Aggressive Factor

1. Helicobacter pylori (most common cause)

H. pylori contributes to ulcer formation through mucosal injury + increased acid secretion:

Urease production → ammonia buffers acid → allows bacterial survival

CagA and VacA toxins → epithelial cell injury and apoptosis

Chronic active gastritis → inflammatory cytokine release → mucosal damage.

Increased gastrin release + reduced somatostatin → ↑ acid production (especially in duodenal ulcers).

Gastric metaplasia in the duodenum → creates a niche for H. pylori → duodenal ulceration.

Net effect: damaged mucosal barrier + increased acid load → ulcer formation.

2. Gastric Acid & Pepsin

Acid is the final common aggressor.

Ulcers occur when:

Acid secretion is excessive (e.g., duodenal ulcers, Zollinger–Ellison syndrome), or

Mucosal resistance is impaired (gastric ulcers).

Acid + pepsin digest the mucosal lining → ulcer crater.

3. NSAIDs

NSAIDs are the second most common cause.

Mechanisms:

COX-1 inhibition → ↓ prostaglandins, which normally:

Increase mucus and bicarbonate

Promote mucosal blood flow

Support epithelial repair

Topical irritation from acidic NSAIDs → epithelial injury

Loss of prostaglandins makes the mucosa vulnerable even to normal acid levels.

4. Other Aggressive Factors

Smoking → reduces mucosal blood flow and bicarbonate

Alcohol → direct mucosal toxin

Bile reflux → disrupts protective mucus

Physiologic stress (ICU, burns, trauma) → mucosal ischemia → "stress ulcers"

B. Defensive Factors (When Impaired → Ulcers)

1. Mucus–bicarbonate barrier

H. pylori, NSAIDs, and bile salts disrupt the barrier → acid penetrates deeper.

2. Prostaglandins

Key for cytoprotection. Loss (via NSAIDs) → impaired defense and repair.

3. Mucosal Blood Flow

Reduced perfusion (shock, smoking, NSAIDs) → ischemia → impaired healing.

4. Epithelial Cell Turnover & Tight Junctions

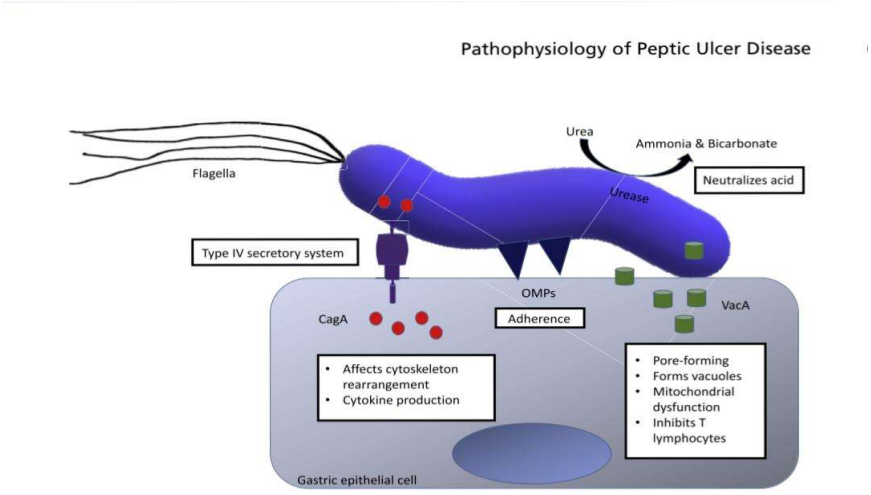
Inflammation, toxins, and ischemia weaken restitution → ulcer formation.

2. Final Common Pathway

Regardless of the trigger:

Aggressive factors (acid, pepsin, H. pylori, NSAIDs) exceed mucosal defenses → mucosal necrosis → ulcer.

Complications: bleeding, perforation, gastric outlet obstruction.



Fig;1 pathophysiology of peptic ulcer disease

Role of H.pylori infection:

a. Disruption of the Mucosal Barrier

H. pylori produces urease, which converts urea to ammonia, creating a more alkaline microenvironment. This allows the bacterium to survive in gastric acid but also damages epithelial cells and weakens the mucous barrier, making the mucosa more susceptible to acid injury.

b. Induction of Inflammation

The bacterium triggers a strong inflammatory response:

Recruitment of neutrophils and lymphocytes
Release of pro-inflammatory cytokines (IL-1β, IL-8, TNF-α)
Chronic inflammation leads to mucosal injury and ulcer formation.

c. Increased Gastric Acid Secretion

Especially in duodenal ulcer disease, H. pylori infection of the gastric antrum:
Reduces somatostatin (inhibits acid secretion)
Increases gastrin levels
→ This results in hyperchlorhydria, contributing to duodenal ulcer formation.

Classification of antacids:

Antacids are **weak bases** that neutralize gastric hydrochloric acid, providing fast relief from heartburn and dyspepsia.
They can be classified into **systemic** and **non-systemic** antacids.

1)Systemic Antacid

These antacids are absorbed into the systemic circulation and may cause **metabolic alkalosis** when used in excess.

a) Sodium Bicarbonate ($NaHCO_3$)

- Rapid onset, short duration
- Produces CO_2 → may cause gastric distension and belching
- Risk of systemic alkalosis and sodium overload
- **Example:** Baking soda

2)Non-Systemic Antacids

These are **poorly absorbed** from the GI tract and have **minimal systemic effects.**

a) Calcium-containing Antacids

Calcium Carbonate ($CaCO_3$)

- Rapid and potent neutralization
- Produces CO_2 → belching
- Hypercalcemia possible with excessive use

Examples: Tums, Caltrate**b) Magnesium-containing Antacids**

b)Magnesium Hydroxide ($Mg(OH)_2$)

- Fast onset
- Causes **osmotic**
- **diarrhea**

Often combined with aluminum salts to counteract bowel effects.

Example: Milk of Magnesia

Magnesium Trisilicate

- Slow reaction
- Has protective colloidal silica gel
- May cause diarrhea

c) Aluminum-containing Antacids

Aluminum Hydroxide ($Al(OH)_3$)

- Slow onset
- Causes **constipation**
- Binds phosphate → risk of hypophosphatemia with long use
- Often combined with magnesium hydroxide.

d) Combination Antacids
Used to balance bowel effects and enhance efficacy.
Examples:
- Aluminum hydroxide + magnesium hydroxide
- Magnesium hydroxide + simethicone
- Calcium carbonate + magnesium carbonate

3. Antacids with Added Agents
a) Antacid + Simethicone
- Relieves
- gas/bloating
  Examples: Mylanta, Maalox

b) Antacid + Alginic Acid
- Forms a "foam raft" over gastric contents
  Example: Gaviscon

Summary Table

Class	Example Agents	Key Features
Systemic	Sodium bicarbonate	Rapid, risk of alkalosis
Calcium salts	Calcium carbonate	Potent, CO_2 gas, hypercalcemia
Magnesium salts	Magnesium hydroxide, Mg trisilicate	Rapid, diarrhea
Aluminum salts	Aluminum hydroxide	Slow, constipation
Combinations	$Mg(OH)_2 + Al(OH)_3$	Balanced bowel effects
Additives	Simethicone, alginic acid	Anti-gas / reflux barrier

2. Mechanism of antacids

1. Neutralization of Gastric Acid (Primary Mechanism)

Antacids are **weak bases** that react chemically with **gastric hydrochloric acid (HCl)** to form **salt + water (and sometimes CO_2),** thereby **increasing gastric pH.**

Example reactions:

- **Calciumcarbonate**
  $CaCO_3 + 2HCl \rightarrow CaCl_2 + H_2O + CO_2\uparrow$
- **Magnesiumhydroxide**
  $Mg(OH)_2 + 2HCl \rightarrow MgCl_2 + 2H_2O$
- **Aluminumhydroxide**
  $Al(OH)_3 + 3HCl \rightarrow AlCl_3 + 3H_2O$

Raising gastric pH reduces acidity and relieves symptoms of dyspepsia and GERD.

2. Reduction of Pepsin Activity
Pepsin becomes inactive when PH rises above PH 3.4

By increasing gastric pH, antacids **decrease peptic activity**, reducing mucosal damage.

3. Mucosal Protection (Mainly Aluminum-containing Antacids)
Aluminum hydroxide can:
- stimulate **prostaglandin** production

- increase **mucus** and **bicarbonate** secretion

→ enhancing gastric mucosal protection.

4. Increased Lower Esophageal Sphincter (LES) Tone (Calcium-containing Antacids)
Calcium ions mildly **increase LES tone,** reducing reflux episodes.
5. Binding of Bile Acids (Aluminum-based Antacids)
Aluminum salts can bind and inactivate bile acids, reducing bile reflux gastritis.
Formulation of antacids in PUD:
Antacids used in PUD are **basic inorganic salts** of aluminum, magnesium, calcium, or sodium. They are formulated alone or in combinations to maximize acid neutralization and minimize adverse effects.
1) Aluminum-Based Antacids
Common Formulations

- **Aluminum hydroxide gel**
- **Aluminum oxide**
- **Aluminum phosphate**

Properties

- Slow acting
- Long duration of action
- Causes constipation
- Provides mild mucosal protection (↑ prostaglandins, mucus)

Formulation examples

- **Aluminum hydroxide suspension**
- **Aluminum hydroxide tablets** (e.g., 300–600 mg)

2)Magnesium-Based Antacids
Common Formulations

- **Magnesium hydroxide** ("milk of magnesia")
- **Magnesium oxide**
- **Magnesium trisilicate**

Properties

- Rapid onset
- Laxative effect (can cause diarrhea)

3)Calcium-Based Antacids
Common Formulation

- **Calcium carbonate ($CaCO_3$)**

Properties

- Potent, long-lasting neutralization
- Produces CO_2 → belching
- May cause rebound acid secretion
- Can lead to hypercalcemia with overuse
- **Formulation examples**

Calcium carbonate chewable tablets (e.g., 500 mg) **xamples**

- **Magnesium hydroxide oral suspension** (usually 400–800 mg per dose)
- **Magnesium trisilicate tablets**

4)Sodium-Based Antacids
Common Formulation

- **Sodium bicarbonate**

Properties

- Very rapid, short acting
- Produces CO_2 (belching)
- Risk of metabolic alkalosis

- High sodium load → **not preferred in PUD**

Formulation examples

- Effervescent tablets
- Powder solutions

5) Combination Antacid Formulations (Most Common in PUD Therapy**)**

Combination formulations are preferred to:

- Balance bowel effects (Mg causes diarrhea, Al causes constipation)
- Improve acid-neutralizing capacity
- Reduce overall side effects

Common Combinations

- **Aluminumhydroxide+Magnesiumhydroxide**
  (e.g., Maalox®, Mylanta®)
- **Aluminum hydroxide + Magnesium trisilicate**
- **Calcium carbonate + Magnesium hydroxide**

Properties

- Better tolerated
- More effective acid neutralization
- Less constipation/diarrhea due to balanced effects

6) Specialized Antacid Formulations

Gaviscon® (Alginic Acid Formulation

Contains:

- **Aluminum hydroxide + Magnesium carbonate + Alginic acid**
- Alginic acid forms a **protective raft** over gastric contents → useful in reflux-associated PUD.

Role of antacids in peptic ulcer disease:

1.Neutralize gastric acid (Immediate symptom relief)

Antacids are alkaline compounds (e.g., aluminum hydroxide, magnesium hydroxide, calcium carbonate) that directly neutralize stomach acid.

This provides rapid relief from epigastric pain, heartburn, and dyspepsi

2. Reduce pepsin activity

Pepsin is most active in an acidic environment.

By raising gastric pH, antacids decrease pepsin activity, reducing mucosal injury.

3. Promote mucosal protection

Certain antacids enhance mucosal defense:

Aluminum-containing antacids stimulate prostaglandin synthesis, increasing mucus and bicarbonate secretion.

Magnesium salts may increase gastric motility and improve emptying.

4. Adjunctive, not curative

Antacids do not eradicate H. pylori.

They do not promote ulcer healing as effectively as PPIs or H2 blockers.

Used mainly for symptomatic relief during treatment.

Advantages and limitations of antacids

1.Rapid relief of symptoms

Antacids neutralize existing stomach acid, giving immediate relief from heartburn, sour stomach, and dyspepsia.

2.Useful for mild, occasional symptoms

Effective for intermittent or non-severe GERD or indigestion.

3.Widely available and inexpensive

Over-the-counter; affordable and easy for patients to access without a prescription.

4.Generally safe for short-term use

When taken appropriately, most formulations have a favorable safety profile.

Calcium and magnesium antacids are often considered safe in pregnancy (in recommended doses).
5.Some formulations offer additional benefits
Alginate-containing antacids form a raft barrier that reduces reflux episodes.

Limitations:

1.Short duration of action (1–2 hours).
2.Multiple daily doses required.
3.Interfere with absorption of drugs (e.g., tetracycline, fluoroquinolones, iron).
4.Magnesium can cause diarrhea; aluminum can cause constipation.

3. Conclusion

Antacid therapy provides rapid and effective short-term relief of pain and dyspeptic symptoms in peptic ulcer disease by neutralizing existing gastric acid. However, while useful for symptomatic control, antacids do not promote long-term ulcer healing, do not eradicate Helicobacter pylori, and are less effective than proton pump inhibitors and H2-receptor antagonists in reducing acid secretion. Their frequent dosing requirements, drug–drug interactions, and salt-specific side effects further limit their role. Therefore, antacids are best regarded as adjunctive therapy for temporary symptom relief rather than definitive treatment for peptic ulcer disease.

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