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A Review on Gastroesophageal Reflux Disease

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Abstract: Gastroesophageal reflux disease (GERD) is a chronic gastrointestinal disorder caused by the abnormal reflux of gastric contents into the esophagus, resulting in symptoms and potential mucosal injury. Its pathogenesis involves lower esophageal sphincter dysfunction, impaired esophageal clearance, delayed gastric emptying, and exposure to acidic and non-acidic refluxate. GERD presents as erosive or non-erosive disease, with varying risks of complications such as Barrett's esophagus. Management includes lifestyle modifications and pharmacological therapy, with proton pump inhibitors as first-line agents. Emerging therapies, including potassium-competitive acid blockers, offer alternative approaches. This review highlights the epidemiology, pathophysiology, clinical features, and current treatment strategies for GERD.

Keywords: *Gastroesophageal Reflux Disease; Risk Factors; Proton Pump Inhibitors; Potassium-Competitive Acid Blockers.*

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I. INTRODUCTION

Gastroesophageal reflux disease (GERD) refers to symptoms or mucosal damage resulting from the abnormal retrograde movement of gastric contents from the stomach into the esophagus. When the esophagus is repeatedly exposed to refluxed material for prolonged periods, inflammation of the esophagus (reflux esophagitis) can occur and in some cases it progresses to erosion of the squamous epithelium (erosive esophagitis). Severe reflux symptoms associated with normal endoscopic findings are referred to as "symptomatic GERD," nonerosive reflux disease, or endoscopy-negative reflux disease.[1]

II. EPIDEMIOLOGY

GERD is a highly prevalent gastrointestinal disorder, affecting about 20% of adults in Western countries. In the United States, Prevalence estimates range from 18% to 28%, though the true burden may be underestimated due to self-medication with over-the-counter acid-suppressing drugs. Gender differences exist in disease presentation, with women more commonly experiencing non-erosive reflux disease, while men are more prone to erosive esophagitis and Barrett's esophagus.[2]

III. PATHOPHYSIOLOGY

Gastroesophageal reflux disease (GERD) develops primarily due to abnormal reflux of gastric contents into the esophagus. This condition is commonly associated with impaired lower esophageal sphincter (LES) function, including reduced LES pressure caused by transient LES relaxations, increased intra-abdominal pressure (Table 1.1).

Additionally, alterations in normal mucosal defense mechanisms- such as prolonged esophageal acid clearance, delayed gastric emptying, and reduced mucosal resistance- play a significant role in disease pathogenesis.

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Esophageal mucosal injury is promoted by aggressive components of the refluxate, including gastric acid, pepsin, bile acids, and pancreatic enzymes. The extent of damage depends largely on the composition and volume of the refluxate, as well as the duration of esophageal exposure.[1]

Table 1:- Foods and Medications That May Worsen Gastroesophageal Reflux Disease Symptoms

Decreased lower esophageal sphincter pressure	
Foods	
Fatty meal	Garlic
Carminatives (peppermint, spearmint)	Onions
Chocolate	Chili peppers
Coffee, cola, tea	
Medications	
Anticholinergics	Ethanol
Barbiturates	Nicotine (smoking)
Caffeine	Nitrates
Dihydropyridine calcium channel blockers	Progesterone
Dopamine	Tetracycline
Estrogen	Theophyline
Direct irritants to the esophageal mucosa	
Foods	
Spicy foods	Tomato juice
Orange juice	Coffee
Medications	
Aspirin	Iron
Bisphosphonates (e.g., alendronate)	Quinidine
Nonsteroidal anti-inflammatory drugs	Potassium chloride

IV. CAUSES

Gastroesophageal reflux disease (GERD) results from recurrent reflux of acidic or non-acidic gastric contents into the esophagus. Under normal physiological conditions, the lower esophageal sphincter (LES) relaxes transiently to allow passage of food into the stomach and then maintains closure to prevent reflux. Impaired relaxation or reduced tone of the LES permits retrograde movement of gastric contents into the esophagus. Persistent exposure to refluxate leads to irritation and inflammation of the esophageal mucosa, contributing to the development of GERD.[3]

V. RISK FACTORS

Conditions that can increase the risk of GERD include:[3]

- Obesity.
- Bulging of the top of the stomach up above the diaphragm, known as a hiatal hernia.
- Pregnancy.
- Connective tissue disorders, such as scleroderma.
- Delayed stomach emptying.

Factors that can aggravate acid reflux include:[3]

- Smoking.
- Eating large meals or eating late at night.
- Eating certain foods, such as fatty or fried foods.
- Drinking certain beverages, such as alcohol or coffee.
- Taking certain medicines, such as aspirin.

VI. SYMPTOMS

Gastroesophageal reflux disease commonly presents with heartburn, characterized by a retrosternal burning sensation, and acid regurgitation, in which gastric contents reflux into the throat. Patients with these classic symptoms are frequently treated empirically without extensive diagnostic evaluation. Persistent or chronic GERD may result in esophageal inflammation and, over time, can cause structural and cellular alterations of the esophageal lining, which are associated with an increased risk of esophageal malignancy.[4]

VII. TREATMENT

A. Pharmacological Therapy

➢ *Proton Pump Inhibitors*

Proton pump inhibitors (PPIs) are a group of drugs widely used to manage disorders associated with excessive gastric acid secretion. They are indicated in various acid-related conditions and work by effectively suppressing stomach acid production. This class of medications plays an important role in the treatment and prevention of acid-mediated gastrointestinal diseases, with established indications and specific contraindications guiding their appropriate clinical use.[5]

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The FDA has approved the following PPIs as of 2015:[5]

- Omeprazole
- Esomeprazole
- Lansoprazole
- Dexlansoprazole
- Pantoprazole
- Rabeprazole

➢ *Potassium-Competitive Acid Blockers*

Vonoprazan is a potassium-competitive acid blocker (PCAB) used for the management of acid-related disorders and as part of Helicobacter pylori eradication therapy. It suppresses gastric acid secretion by reversibly inhibiting the H^+, K^+-ATPase enzyme at the parietal cell surface through competition with potassium ions, effectively reducing both basal and stimulated acid output. Unlike proton-pump inhibitors (PPIs), which irreversibly inactivate the enzyme via covalent binding to cysteine residues, PCABs act by blocking potassium binding, resulting in a distinct and targeted mechanism of acid suppression.[6]

➢ *H2 Receptor Antagonists*

H2-receptor antagonists (H2RAs) reduce gastric acid secretion by competitively blocking histamine receptors on parietal cells. They promote healing of esophagitis and provide symptomatic relief of heartburn, although their efficacy is lower than that of PPIs. H2RAs may be used as step-down therapy in patients with uncomplicated GERD who achieve symptom control with PPIs, but they are generally not recommended in cases of erosive esophagitis or Barrett's esophagus.[7]

➢ *Reflux-Reducing Agents*

Reflux-reducing agents, such as baclofen, decrease transient lower esophageal sphincter relaxations and reflux episodes. Despite demonstrated physiological benefits, their clinical use is limited due to central nervous system adverse effects and inconsistent long-term symptom improvement.[7]

➢ *Adjunct Medications*

Adjunctive therapies play a supportive role in GERD management. Antacids provide rapid but short-term relief of intermittent symptoms without promoting mucosal healing or preventing complications. Alginates form a protective barrier over gastric contents and, when combined with antacids, offer superior symptom control compared to antacids alone. Prokinetic agents may improve reflux symptoms by enhancing esophageal clearance and gastric emptying; however, their additional benefit when combined with PPIs appears modest.[7]

B. Nonpharmacological Management

Non-pharmacological management focuses on lifestyle and dietary modifications to reduce reflux symptoms and improve esophageal clearance. Elevating the head of the bed during sleep helps limit nocturnal reflux. Dietary measures include avoiding foods that lower lower-esophageal sphincter (LES) tone or irritate the esophageal mucosa, while encouraging protein-rich meals that enhance LES pressure. Eating smaller, more frequent meals and avoiding late-night meals reduces gastric volume and reflux episodes. Weight reduction and smoking cessation are recommended due to their strong association with symptom improvement. Alcohol intake and tight-fitting clothing should be avoided as they exacerbate reflux. Whenever possible, medications known to promote reflux should be discontinued, and drugs that can irritate the esophageal mucosa should be taken with adequate fluids to minimize mucosal injury.[1]

VIII. CONCLUSION

GERD is a prevalent disorder with a broad clinical spectrum. Early diagnosis and a combination of lifestyle interventions and acid-suppressive therapy are essential to achieve symptom control and prevent complications. Individualized treatment remains central to effective long-term management.

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