A Comprehensive Review on Peptic Ulcer

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Abstract

Peptic ulcers are lesions resulting from damage to the stomach's mucosal lining or the upper portion of the duodenum. Disruption of the equilibrium between harmful elements such as gastric acid and the body's inherent defensive mechanisms within the gastrointestinal lining can lead to development. A significant cause of peptic ulcers is the use of nonsteroidal anti-inflammatory drugs (NSAIDs) especially in people without helicobacter pylori infection. Nonsteroidal anti-inflammatory drugs (NSAIDs) impair mucosal defence mechanisms by inhibiting cyclooxygenase enzymes, leading to decreased prostaglandin synthesis.

This reduction leads to decreased mucosal blood supply and lower secretion of protective mucus and bicarbonate. This review discusses the underlying mechanisms, clinical features, diagnostic methods, and therapeutic management of NSAID-induced peptic ulcers. Preventive measures such as patient risk assessment, use of proton pump inhibitors (PPIs), and selection of safer alternatives for pain relief are also emphasized.

Index Terms

Aggressive factors, Cyclooxygenase, Helicobacter Pylori, NSAID's, Peptic Ulcer Disease, Proton Pump Inhibitors.

I. INTRODUCTION

A peptic ulcer is typically a lesion within the digestive tract caused by acid, usually located in the stomach or the beginning part of the duodenum, and can be identified by exposed mucosa, with the affected area extending into the submucosa and muscularis propria.[1] Peptic ulcers are generally defects or sores in the gastroduodenal mucosa. Common symptoms of ulcers include stomach pain, which in certain instances can also result in additional gastrointestinal symptoms that may lead to bleeding. This disorder impacts the lower esophagus, the upper segment of

the duodenum, and the lower segment of the stomach.[2]

Types-

1. Gastric Ulcer
2. Stomach Ulcer
3. Duodenal Ulcer [3]

II. PATHOPHYSIOLOGY

Under normal physiological circumstances, a protective mucus-bicarbonate layer, a near-neutral surface pH, and the continuous regeneration of epithelial cells help maintain the integrity of the gastric and duodenal mucosa.[4][5] In most cases, Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are the main risk factors for peptic ulcer disease and gastritis. Contributing factors that are less common include alcohol consumption, smoking, cocaine use, severe systemic illness, autoimmune disorders, radiation therapy, and Crohn's disease, among other specific conditions.[6]

III. SIGNS AND SYMPTOMS

1. Bloating
2. Abdominal Pain
3. Nausea
4. Vomiting
5. Loss of appetite [7]

IV. CAUSES

There are various causes of Peptic ulcer but Helicobacter pylori and NSAID's are the common causes of it.[1]

Other causes are

- Zollinger-Ellison syndrome
- Malignancy (gastric/lung cancer, lymphomas)
- Stress (Acute illness, burns, head injury)
- Viral infection
- Vascular insufficiency
- Radiation therapy
- Crohn disease

V. DIAGNOSIS AND TESTS

Endoscopy plays a key role in accurately diagnosing peptic ulcers and differentiating them from associated complications. Through endoscopy, gastric ulcers can be biopsied to exclude malignancy or to collect specimens for Helicobacter pylori testing. In research settings, endoscopic evidence of ulcer healing is considered the gold standard. However, in everyday clinical practice, many patients with dyspepsia are managed using a "test-and-treat" strategy for H. pylori without undergoing endoscopy. Current guidelines endorse this approach, particularly for

patients younger than 30 who have no alarm features[8]

The urea breath test operates based on the fact that Helicobacter pylori, when it is in the stomach, produces the enzyme urease that breaks down orally given ^{13}C- or ^{14}C-labeled urea into carbon dioxide and ammonia. Carbon dioxide labeled with $^{13}CO_2$ or $^{14}CO_2$ is absorbed into the bloodstream, transported to the lungs, and then released in exhaled breath, where it can be quantitatively measured.[9]

VI. SURGERY

Surgical treatments for peptic ulcer disease have evolved through various procedures, including vagotomy, which is aimed at reducing gastric acid production. A truncal vagotomy, commonly performed in conjunction with a drainage procedure, is frequently employed due to its effectiveness but is associated with delayed gastric emptying [1]. The standard emergency surgical procedure for closure of perforated ulcers remains the application of an omental (Graham) patch [11]. A vagotomy paired with an antrectomy led to a low rate of ulcer recurrence, but it was associated with postoperative complications such as dumping syndrome [12]. In selective vagotomy, the vagal branches outside the stomach are preserved, however research has not demonstrated a noticeable improvement over the traditional method [13]. A highly selective (parietal cell) vagotomy selectively disables the area responsible for acid secretion while preserving antral function, which requires a significant amount of surgical expertise [14][15].

VII. TREATMENT

Treatment for peptic ulcer disease typically involves medications that either reduce gastric acid production or boost the mucous membrane's natural protective functions. The primary drug categories encompass proton pump inhibitors, H_2-receptor antagonists, antacids, potassium-competitive acid blockers, and cytoprotective agents.

Drug classification and mode of action

1. Proton Pump Inhibitors (PPIs)

   PPIs such as omeprazole, esomeprazole, and pantoprazole irreversibly inhibit the gastric H^+/K^+-ATPase enzyme in parietal cells, resulting in profound suppression of acid secretion [16][17]

2. H_2-Receptor Antagonists

   Drugs such as cimetidine and ranitidine reduce acid secretion by blocking histamine H_2 receptors on gastric parietal cells [18].

3. Antacids

   Aluminium and magnesium salts neutralize gastric acid, thereby providing rapid symptomatic relief by raising the stomach's pH level [17].

4. Potassium-Competitive Acid Blockers (P-CABs)

   Agents like vonoprazan block gastric H^+/K^+-ATPase competitively for potassium and suppress acid secretion quickly and efficiently [19].

5. Cytoprotective Agents

   Sucralfate and misoprostol boost mucosal protection by elevating mucus and bicarbonate production and enhancing mucosal blood circulation [17][18].

VIII. CONCLUSION

Peptic ulcer disease is generally associated with Helicobacter pylori infections and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). Treatment strategies involve acid-suppressive therapy, eradication regimens, and the avoidance of factors that trigger problems, with invasive interventions typically only being recommended for complications. Pharmacological treatments have experienced substantial progress, leading to improved recovery results and reduced instances of recurrence.

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