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DIAGNOSIS AND MANAGEMENT OF PEPTIC ULCER DISEASE

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ABSTRACT

Globally, peptic ulcer disease is still a major cause of illness and mortality. About two-thirds of individuals with peptic ulcer disease do not exhibit any symptoms. Epigastric discomfort, which might be accompanied by dyspepsia, bloating, abdominal fullness, nausea, or early satiety, is the most typical sign of peptic ulcer disease in symptomatic patients. Nonsteroidal anti-inflammatory drug (NSAID) use, Helicobacter pylori infection, or both are linked to the majority of peptic ulcer disease cases. In this review, we address the role of proton pump inhibitors in the treatment of peptic ulcer disease, highlight the most recent recommendations regarding the diagnosis and treatment of H. pylori, and go over the most recent research on the treatment of peptic ulcer disease complications, such as endoscopic intervention for bleeding caused by peptic ulcers. In order to reduce related morbidity and mortality, prompt identification and treatment of peptic ulcer disease and its aftereffects are essential, as is prevention of peptic ulcer disease in people at high risk, such as those with H. pylori infection and NSAID users.

INTRODUCTION

In the past, it was widely believed that stress caused peptic ulcer disease by increasing the production of gastric acid. Antacids and anticholinergics were used to treat peptic ulcers in the US until the late 1970s, and ulcer disease frequently required surgery[1]. In 1976, histamine-2-receptor antagonists (H2RAs) were first made available. In gastrointestinal biopsies, Dr. J. Robin Warren found Helicobacter pylori, formerly known as Campylobacter pyloridis[2]. In 1982 Dr. Barry Marshall cultured H. pylori from patients with ulcers and gastritis[3]. However, it was not until 1994 that National Institutes of Health guidelines recommended the use of antibiotics in the treatment of patients with peptic ulcer disease

attributed to H. pylori[4]. By the end of the 20th century, a decrease in the occurrence of peptic ulcer disease was observed. The decrease in H. pylori infection rate in the population is assumed to have resulted from increasing hygiene standards[5].

EPIDEMIOLOGY

By the end of the 20th century, a decrease in the occurrence of peptic ulcer disease was observed. The decrease in H. pylori infection rate in the population is assumed to have resulted from increasing hygiene standards.Millions of individuals worldwide suffer from peptic ulcer disease, which is still one of the most prevalent gastrointestinal conditions. Because to overpopulation, inadequate sanitation, and low socioeconomic status, H. pylori infection is more common in underdeveloped nations. In general, duodenal ulcers are more frequent than stomach ulcers. Age and long-term NSAID use are significant risk factors for ulcer formation, particularly in older individuals[6].

DIAGNOSIS

Clinical characteristics and specific testing are typically used to diagnose peptic ulcer disease, however it's crucial to understand that particular symptoms and indicators are not always dependable.

Episodic gnawing or searing epigastric pain, discomfort that occurs two to five hours after meals or on an empty stomach, and noc-turnal pain that is eased by food intake, antacids, or antisecretory medications are typical signs of peptic ulcer disease. The most precise findings for a peptic ulcer are a history of episodic or epigastric pain, alleviation of discomfort after eating, and nightly awakenings due to pain with relief after eating. These findings aid in the diagnosis. Indigestion, appetite loss, intolerance to fatty meals, heartburn, and a favorable family history are less frequent symptoms. The physical examination is unreliable; in one study, the chance of an ulcer was decreased by discomfort on deep palpation[7].

EVALUATION TESTS

- Upper gastrointestinal endoscopy (gold standard)
- Urea breath test for H. pylori
- Stool antigen test
- Rapid urease test
- Barium contrast radiography
- Complete blood count to detect anaemia

SIGN AND SYMPTOMS

Duodenal ulcer

• Mid-epigastric pain

• Gnawing or burning, nonradiating, reoccurring, episodic pain

• Pain is typically relieved by food or antacids

Gastric ulcer

• Mid-epigastric pain

• Pain is typically aggravated by food and relieved by antacids

Nonspecific dyspeptic symptoms

• Indigestion

• Epigastric fullness

• Nausea and vomiting

• Loss of appetite

• Heartburn

Alarm symptoms

• Symptom onset after 55 years of age

• Progressive dysphagia

• Persistent of recurrent vomiting

• Severe abdominal pain

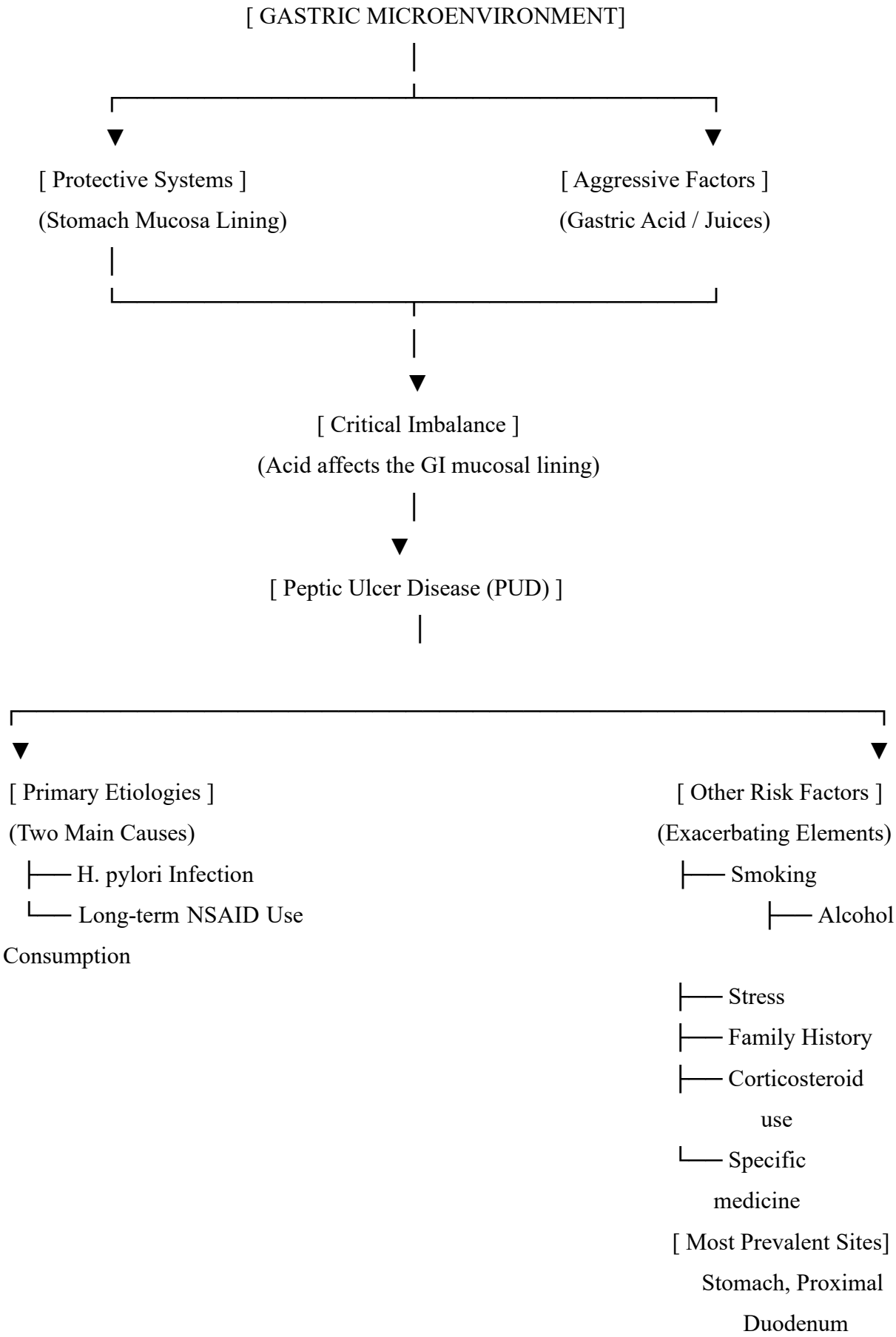
• Weight loss and/or anorexia

• Family history of gastric malignancy

• Blood in stool, melena, hematemesis, and/or anemia[8]

PATHOGENESIS

An imbalance between the protective systems of the stomach mucosa and aggressive factors including Helicobacter pylori infection, gastric acid, and NSAID use results in peptic ulcer disease (PUD). When acid affects the mucosal lining of the gastrointestinal tract, ulcers result. The two main causes of PUD are long-term NSAID use and H. pylori infection. Smoking, drinking alcohol, stress, a family history, using corticosteroids, and taking certain medications are other risk factors. The stomach and proximal duodenum are the most prevalent locations for peptic ulcers[9].


[GASTRIC MICROENVIRONMENT]
[Protective Systems]
(Stomach Mucosa Lining)
[Aggressive Factors]
(Gastric Acid / Juices)
[Critical Imbalance]
(Acid affects the GI mucosal lining)
[Peptic Ulcer Disease (PUD)]
[Primary Etiologies]
(Two Main Causes)
H. pylori Infection
Long-term NSAID Use
[Other Risk Factors]
(Exacerbating Elements)
Smoking
Alcohol Consumption
Stress
Family History
Corticosteroid use
Specific medicine
[Most Prevalent Sites]
Stomach, Proximal Duodenum

RISK FACTORS AND CAUSES

• H Pylori

• NSAIDs

• Gastric bypass surgery

• Cigarette smoking

• Selective serotonin reuptake inhibitors

• Zollinger-Ellison syndrome (uncommon, gastrin producing tumour usually located in the pancreas)

• Physiological stress associated with serious trauma and critical illness (eg, septicaemia)

• Gastric tumours mistaken for peptic ulcers

• Autoimmune diseases, eg, vasculitis, sarcoidosis, and Crohn's disease

• Infections, mainly in immunocompromised patients, eg, cytomegalovirus, tuberculosis, and syphilis.

• Psychological stress is not an established risk factor for peptic ulcer disease, although some research has suggested an association

• Consumption of alcohol or coffee does not seem to increase the risk of peptic ulcer disease[10].

TREATMENT AND THERAPEUTIC MANAGEMENT

Proton Pump inhibitors

The first-line treatment for ulcer healing is PPIs. They successfully lower stomach acid output by inhibiting the H+/K+ ATPase enzyme. In most cases, ulcers heal in 4–8 weeks[11].

Mechanism of action

Proton pump inhibitors (PPIs) act by irreversibly inhibiting the gastric proton pump, H+/K+ ATPase, in the parietal cells of the stomach. They are prodrugs activated in the acidic environment of the parietal cell canaliculi, where they bind covalently to the proton pump and block the final step of gastric acid secretion. Due to this irreversible inhibition, PPIs suppress gastric acid production very effectively, reducing acid secretion by up to 99%, and are more potent than H2 receptor antagonists. PPIs also aid in the eradication of Helicobacter pylori infection by increasing gastric pH and enhancing antibiotic effectiveness[12].

Examples of drugs with doses

Omeprazole- 20mg once daily

Pantoprazole- 40 mg once daily

Esomoprazole - 40 mg once daily

Lansoprazole- 30 mg once daily[13].

Helicobacter pylori Eradication treatment

A PPI combined with clarithromycin and amoxicillin/metronidazole for 14 days makes up standesoard triple therapy. Bismuth-based quadruple therapy is advised in cases of resistance[11].

Mechanism of action

Eradication therapy for Helicobacter pylori infection works by combining a proton pump inhibitor (PPI) with antibiotics to eliminate the bacterium and

promote ulcer healing. PPIs suppress gastric acid secretion by irreversibly inhibiting the H+/K+ ATPase proton pump in gastric parietal cells, thereby increasing gastric pH and enhancing the effectiveness of antibiotics. Antibiotics such as Amoxicillin inhibit bacterial cell wall synthesis, while Clarithromycin inhibits bacterial protein synthesis by binding to the 50S ribosomal subunit. Elimination of H. pylori reduces mucosal inflammation, promotes healing of peptic ulcers, and prevents ulcer recurrence[14].

H2-Receptors antagonists

Mechanism of action

H2 receptor antagonists act by competitively blocking histamine H2 receptors on the gastric parietal cells of the stomach. This inhibition reduces the stimulation of adenylate cyclase and decreases cyclic AMP (cAMP) production, leading to reduced secretion of gastric acid and pepsin. By lowering gastric acidity, these drugs promote healing of peptic ulcers and relieve ulcer-related pain. They are particularly effective in reducing nocturnal acid secretion[15].

Examples of drugs with doses

Standard triple therapy

Omeprazole /other PPI- 20mg twice daily.

Amoxicillin – 1g twice daily.

Clarithromycin- 500mg twice daily.

Alternative quadraple therapy

Pantoprazole/other PPI – 40 mg twice daily.

Bismuth sub salicylate – 524 mg four times daily.

Metronidazole-500mg three to four times daily.

Tetracycline-500mg four times daily.

While PPIs are more effective, H2 blockers can help with mild ulcer disease by reducing acid output[14].

Mucosal Defense Substances

Mechanism of action

Mucosal protective agents enhance the natural defense mechanisms of the gastric mucosa and protect ulcers from acid injury.

- Sucralfate forms a viscous, adhesive barrier at the ulcer site by binding to positively charged proteins in damaged mucosa, thereby protecting the ulcer from acid, pepsin, and bile salts.
- Misoprostol, a prostaglandin E1 analogue, increases secretion of mucus and bicarbonate, enhances mucosal blood flow, and decreases gastric acid secretion.
- Bismuth Subsalicylate and Colloidal Bismuth Subcitrate coat the ulcer base, stimulate prostaglandin and bicarbonate production, and also exhibit antibacterial activity against Helicobacter pylori infection[16].

Examples of drugs with doses

Sucralfate - 1 g four times daily before meals and at bedtime[17]

Misoprestol- 200 mcg four times daily

Bismuth subsalicylate- 524 mg four times daily,[18]

Colloidal bismuth subcitrate- 120 mg four times daily

Bismuth compounds have antibacterial effect against H. pylori, and sucralfate creates a barrier that protects the ulcer surface[19].

Lifestyle changes

It is recommended that patients abstain from alcohol, smoking, eating spicy meals, and taking unnecessary NSAIDs. Reducing stress and maintaining a balanced diet helps enhance recuperation[11.]

CONCLUSION

Peptic ulcer disease remains a significant gastrointestinal disorder worldwide. H. pylori infection and NSAID use are the leading causes. Advances in diagnostic techniques and the development of effective acid suppressants and eradication therapies have greatly improved treatment outcomes. Early diagnosis, appropriate pharmacological therapy, monitoring of adverse effects, and lifestyle modification are essential for successful management and prevention of complications[6,9].

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