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A REVIEW ON: PEPTIC ULCER DISEASE CONVENTIONAL THERAPIES AND THE EMERGING ROLE OF HERBAL MEDICINE

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ABSTRACT

Peptic ulcer disease, including gastric and duodenal ulcers, affects millions worldwide. It occurs when aggressive factors like hydrochloric acid (HCl), pepsin, bile reflux, leukotrienes (LTs), and reactive oxygen species (ROS) overpower defensive mechanisms such as the mucus-bicarbonate barrier, prostaglandins (PGs), mucosal blood flow, and antioxidants. The primary causes are Helicobacter pylori (H. pylori) infection and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) like aspirin and ibuprofen. Additional risk factors include chemical agents (HCl/ethanol), stomach cancer, stress, smoking, spicy foods, and nutritional deficiencies. While stress and diet do not directly cause ulcers, they can worsen symptoms. Up to 70–90% of ulcers are linked to H. pylori, which thrives in the stomach's acidic environment.Treatment aims to reduce acid production, neutralize existing acid, and protect the ulcer to promote healing. In recent years, herbal medicine has gained global significance for its potential anti-ulcer benefits. However, concerns remain regarding its quality, safety, and efficacy. Research on medicinal plants shows they prevent ulcers in a dose-dependent manner, with histological studies confirming their lack of acute toxicity. Phytochemical screening has identified beneficial secondary metabolites like flavonoids and tannins, which contribute to their protective effects.Extensive pharmacological research has explored the anti-ulcer properties of various compounds. This review highlights the ulcerogenic mechanisms involved in peptic ulcer disease and examines the gastroprotective potential of medicinal plants, emphasizing their active constituents as promising alternative treatments.

KEYWORDS: Peptic ulcer, H.pylori, NSAIDs, Acid production, Gastroprotection, Herbal medicine, Flavonoids, Phytochemicals.

INTRODUCTION[1,2,3,4,5,6]

Peptic ulcer is a chronic condition caused by an imbalance between protective factors of the gastric mucosa (such as mucus and bicarbonate secretion, adequate blood flow, prostaglandin E2, nitric oxide, sulfhydryl compounds, and antioxidant enzymes) and aggressive factors (including acid and pepsin secretions). Various behavioral and environmental factors, including smoking, poor diet, alcohol consumption, non-steroidal anti-inflammatory drugs (NSAIDs), and Helicobacter pylori infection, contribute to the development of gastric ulcers.Peptic ulcer disease is identified as a mucosal break exceeding 3–5 mm in the stomach or duodenum with a visible depth, making it an endoscopic diagnosis, unlike dyspepsia, which is diagnosed clinically based on symptoms. The condition arises due to an imbalance between protective and damaging factors affecting the stomach and duodenum. Patients with gastric and duodenal ulcers often experience similar symptoms, including epigastric or retrosternal pain, early satiety, nausea, bloating, belching, and postprandial discomfort. However, these symptoms are non-specific and may be challenging to distinguish from functional dyspepsia.Ulcers are characterized by open sores on the skin or mucous membranes, marked by the shedding of inflamed dead tissue. They can develop on the skin, particularly on the lower extremities, as well as in the gastrointestinal tract and other areas of the body. Several types of ulcers exist, including mouth ulcers, esophageal ulcers, peptic ulcers, and genital ulcers. Ulcers are widespread and affect populations globally. The conventional allopathic treatments for ulcers often have adverse effects on health, sometimes impairing the function of affected organs.Currently, medical science recognizes various forms of ulcers, such as peptic ulcers, corneal ulcers, stomach ulcers, and foot or leg ulcers. Helicobacter pylori, a Gram-negative bacterium, resides between the mucous layer and the gastric epithelium, enabling it to survive in the stomach's harsh environment. Initially, H. pylori colonizes the antrum but gradually migrates toward the upper stomach regions. Peptic ulcer disease is a prevalent gastrointestinal disorder, affecting approximately 10% of the global population. Among peptic ulcers, about 95% are duodenal ulcers. It is estimated that 15,000 deaths occur annually due to complications from peptic ulcers. The annual incidence rates for peptic ulcer hemorrhage and perforation range from 19.4–57 and 3.8–14 per 100,000 individuals, respectively. The recurrence rate of hemorrhage within seven days is approximately 13.9%, while the long-term recurrence of perforation is about 12.2%.Ulcer

severity is scored as follows: no ulcer = 0, superficial ulcers = 1, deep ulcers = 2, and perforation = 3. The mean ulcer score for each subject is calculated as the ulcer index. The percentage of ulcer protection is determined using the formula:

% Protection = [(Control mean ulcer index - Test mean ulcer index) / Control mean ulcer index] × 100

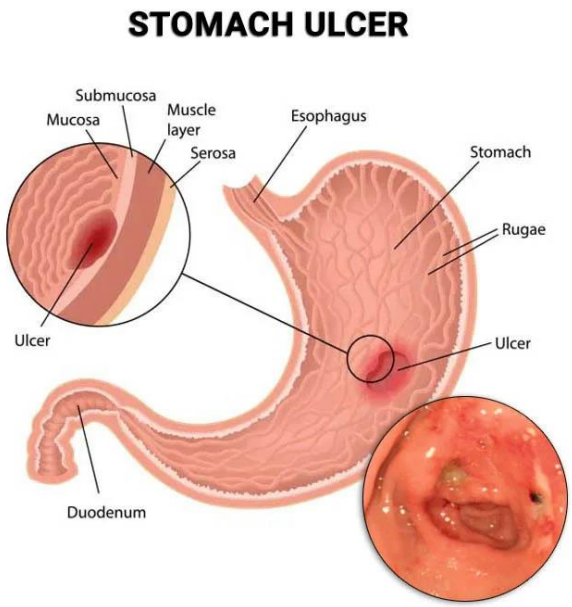


Fig. No. 01: Stomach ulcer.

TYPES OF ULCER

1) Peptic Ulcer[7]

Peptic ulcer is a general term referring to ulcers that develop in the digestive tract, specifically in the stomach or duodenum. Previously, it was thought that stress and spicy food were the primary causes of these ulcers. However, recent research has revealed that these factors merely worsen the condition rather than cause it. The primary causes of peptic ulcers are infections caused by the bacterium *Helicobacter pylori* or adverse reactions to certain medications, such as non-steroidal anti-inflammatory drugs (NSAIDs). Common symptoms of peptic ulcers include weight loss, loss of appetite, bloating, nausea, vomiting, and black stools, which may indicate gastrointestinal bleeding.

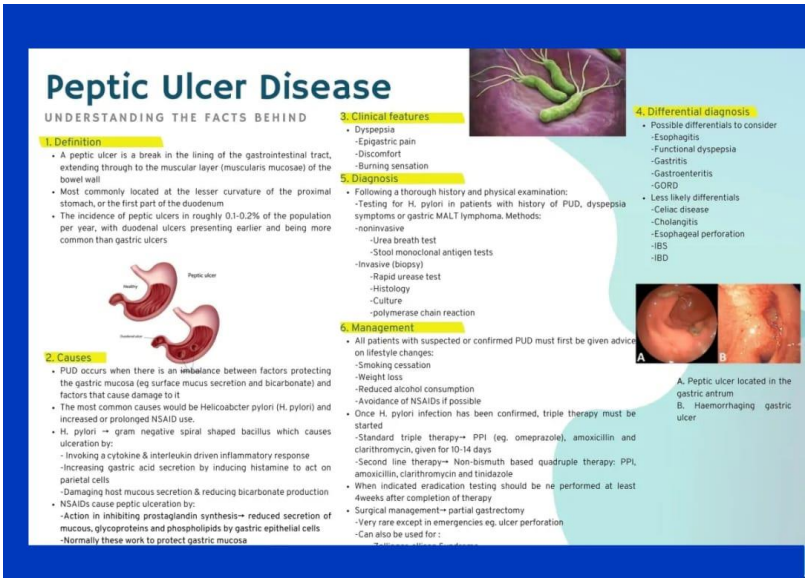


Fig. No. 02: Peptic ulcer Disease.

2) Aphthous Ulcers[7]

Mouth ulcers are sores that form on the inner lining of the mouth. They are a common condition, often resulting from trauma caused by ill-fitting dentures, fractured teeth, or dental fillings. Other potential causes include anemia, measles, viral infections, oral candidiasis, chronic infections, throat cancer, mouth cancer, and vitamin B deficiency.Aphthous minor is one of the most prevalent types of oral ulcerative diseases, affecting approximately 15–20% of the global population. In some regions, the prevalence has been reported to be as high as 50–66%, with a particularly high occurrence in North America. Interestingly, research indicates that the incidence of aphthous ulcers is lower among smokers compared to non-smokers.

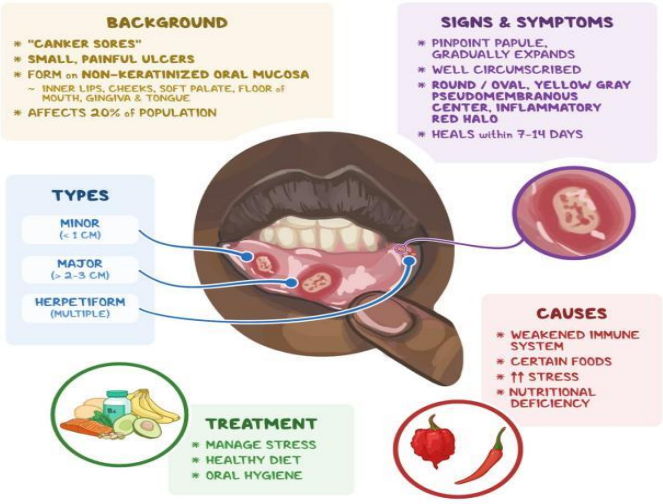


Fig. No.03: Aphthous Ulcers.

ETIOLOGY AND PATHOGENESIS OF ULCER H. PYLORI[6-7]

Helicobacter pylori is the primary cause of stomach ulcers and was first discovered by two Australian scientists in 1982. This bacterium exhibits pathogenic activity by encoding the effector protein cytotoxin-associated gene A (*cagA*). Once translocated into the host cell, *cagA* alters cell shape, enhances cell motility, disrupts cell junction activity, and contributes to the development of gastric ulcers and gastric carcinomas.

H. pylori infection leads to an increased expression of cytokines, including tumor necrosis factor-alpha (*TNF-α*), in gastritis. Additionally, interleukin-1 beta (*IL-1β*) is also overexpressed in *H. pylori*-induced gastritis. The infected gastric mucosa exhibits infiltration of polymorphonuclear leukocytes, lymphocytes, monocytes, and plasma cells within the lamina propria, along with severe neutrophil infiltration within the epithelium. Proper treatment leads to the complete resolution of mucosal inflammation and significantly reduces the likelihood of ulcer recurrence.

Gastric acid secretions[8,9,10,11]

Gastric acid is a key factor in gastric ulcer disease, with about 50% of patients experiencing excess acid and pepsin secretion. However, it also serves as a defense by preventing bacterial colonization. Acid secretion is primarily stimulated by histamine, acetylcholine, and gastrin, which act on parietal cell receptors to regulate production.Peptic ulcer disease (PUD) occurs when the protective mucosal mechanisms are overpowered by gastric acid and pepsin. Ulcers mainly develop in the stomach (gastric ulcers) or duodenum (duodenal ulcers). While *H. pylori* infection was initially seen as the primary cause, gastric ulcers are now more often linked to NSAID and ASA use. Duodenal ulcers are associated with smoking and aging, though studies suggest lower prevalence in men, Black, and Hispanic populations compared to non-Hispanic Whites. Pressure ulcers, linked to reduced mobility, require multivariable risk assessment models for better prevention. A systematic review for the NIHR Programme Grant aimed to identify independent risk factors to enhance early detection and resource allocation.

USE OF FOOD FIBERS IN PEPTIC ULCER TREATMENT[12]

The physicochemical properties of different fiber types lead to various physiological effects in the body. Soluble fibers, present in foods like apples, oatmeal, and pears, increase the viscosity of intestinal contents. In contrast, insoluble fibers, found in whole grains, granola, and flaxseeds, add bulk to stools, shorten transit time in the large intestine, and facilitate

easier and faster elimination. Fibers play a crucial role in regulating bowel function, making them essential for overall health and an important component in the dietary management of various medical conditions. Recommended daily diat preferred.

PEPTIC ULCER DIAGNOSIS[13,14]

Participants were asked about peptic ulcer diagnoses during an 11-year period, with confirmation obtained from the National Danish Hospital Discharge Registry (NDHDR) using WHO ICD-8 codes. Only cases verified by endoscopy, barium meal, or surgery were considered true ulcers.Peptic ulcer disease (PUD) is primarily diagnosed via upper gastrointestinal endoscopy (OGD). Most cases are treated medically, while complications like bleeding or perforation may require interventional procedures. Ulcers typically form on the stomach's lesser curvature or the duodenum.The rapid urease test (CLO test) is the most common *H. pylori* detection method during endoscopy, offering 97% sensitivity and 100% specificity. Non-invasive alternatives include the urea breath test (95% sensitivity, 100% specificity) and the *H. pylori* stool antigen test (95% accuracy). Serological testing is not recommended due to potential false positives.

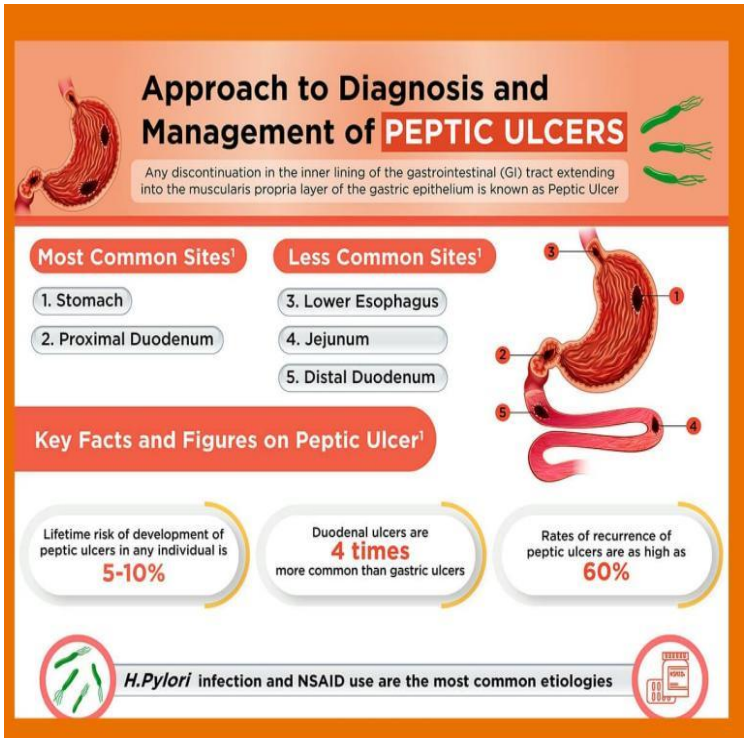


Fig. No. 04: Peptic ulcer diagnosis.

SYMPTOMS OF ULCER

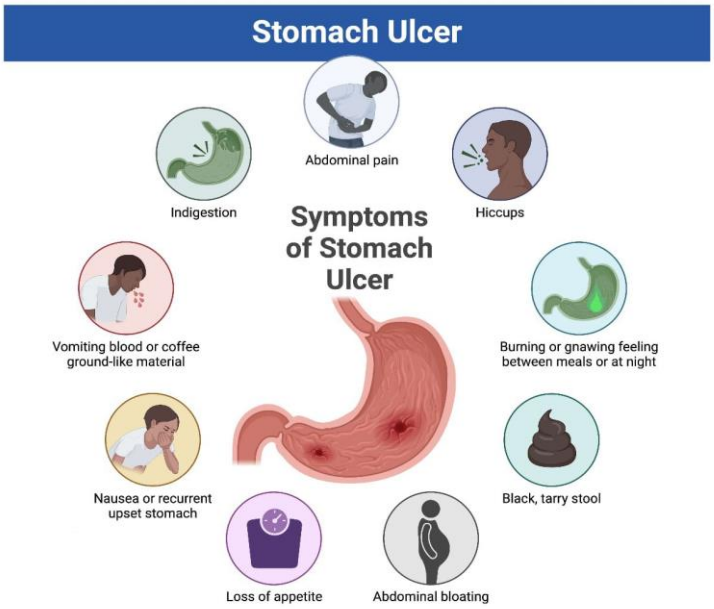


Fig. No.05: Symptoms of stomach ulcer.

Radiology[15]

An erect chest X-ray is commonly performed in patients with acute upper abdominal pain suspected of perforat ion. CT scans can rule out other acute abdominal conditions such as acute cholecystitis, acute pancreatitis, acute appendicitis and acute mesenteric ischemia to name but a few. Some of these acute abdominal conditions may not require surgical intervention at least in the initial phase. In resource-poor healthcare facilities, an erect chest X-ray is extremely useful in detecting free air under the diaphragm confirming visceral perforation. Peptic ulcer perforation can also generate detectable sonographic signs, such as pneumoperitoneum; free intraperitoneal air tends to accumulate around the liver, duodenum, and stomach, local thickening of the gastroduodenal wall containing an echogenic focus or line, the presence of localized extraluminal gas and fluid.

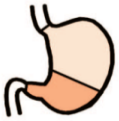
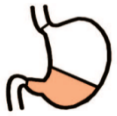
PUD mortality risk factor analysis: correlation[15]

The link mortality rates and the chosen factors are shown in the scatterplots. As demonstrated, stomach ulcers had a positive correlation with mortality ($r=0.23$, $p=0.036$), while duodenal ulcers had a negative correlation ($r=-0.19$, $p=0.07$). Mortality was positively correlated with both bleeding and perforated ulcers, however the latter was more strongly correlated ($r=0.41$, $p<0.0001$). We looked into the relationship between mortality and publication year in order to test the theory that survival rates have been rising recently.

Perioperative death rates and publication year were positively correlated (r=0.22, p=0.032); similarly, the mortality rate rose as surgical age increased (r=0.23, p=0.036).

PUD mortality risk factor analysis: meta-regression[16-24]

To explore the sources of heterogeneity, a meta-regression analysis was conducted using covariates such as study-level mean or median age at surgery, publication year, sub-region, and study quality score. Significant heterogeneity in perioperative mortality rates was observed ($p < 0.0001$). The multivariable meta-regression model indicated that for every 10-year increase in publication year, mortality rates rose by 1%. Regional differences were not significantly linked to mortality rates, though age showed a marginally significant association, with a 2% increase in mortality risk per decade ($p = 0.07$).The management of acid-related diseases, including peptic ulcer disease (PUD) and gastroesophageal reflux disease (GERD), has undergone significant advancements over the past two decades. While pharmacological inhibition of gastric acid secretion promotes ulcer healing, recurrence is common upon treatment cessation. The discovery of *Helicobacter pylori* as a key factor in peptic disease marked a breakthrough, demonstrating that eradication of the bacterium prevents ulcer relapse. Less than 30 years ago, Robin Warren and Barry Marshall successfully cultured *H. pylori* from gastric biopsy specimens, confirming its role in peptic ulcer disease. Recognized as a type I carcinogen in 1994, *H. pylori* is now considered a leading cause of infection-related cancers, accounting for 5.5% of global cancer cases. In 2005, Warren and Marshall received the Nobel Prize in Medicine for their discovery.*H. pylori* is a Gram-negative bacterium that specifically colonizes the gastric epithelium. It is urease, catalase, and oxidase positive, spiral-shaped, and equipped with 3 to 5 polar flagella for motility. The bacterium employs virulence factors that interfere with host cell signaling and has adapted to the stomach's acidic environment by converting urea into ammonia via urease, creating a localized neutral zone for survival.

Pattern of gastritis	*Gastric histology*	*Duodenal histology*	*Acid secretion*	*Clinical condition*
 Pan-gastritis	• Chronic inflammation • Atrophy • Intestinal metaplasia	• Normal	• Reduced	• Gastric ulcer • Gastric cancer
 Antral-predominant	• Chronic inflammation • Polymorph activity	• Gastric metaplasia • Active chronic inflammation	• Increased	• Duodenal ulcer

This study found that individuals with lower education levels face a higher risk of asymptomatic peptic ulcer disease (PUD), likely due to increased *Helicobacter pylori* (*H. pylori*) infection associated with poor hygiene, psychological stress, and physically demanding lifestyles. *H. pylori* virulence factors, such as the East Asian CagA genotype, and environmental influences contribute to epithelial damage. Some dietary elements like chili peppers and garlic may offer protection against *H. pylori*, and studies suggest helminth and parasite infections may also play a protective role.*H. pylori* is a spiral-shaped, gram-negative, microaerophilic bacterium that persistently colonizes human gastric mucosa. The lifetime risk of peptic ulcer disease is estimated at 20%, with a 1–2% risk of gastric cancer. Early epidemiological studies focused on symptomatic patients undergoing endoscopy, leaving limited data on *H. pylori* prevalence in the general population. This study aims to expand knowledge on its global impact on upper gastrointestinal diseases.

Study Objectives

1. Estimate *H. pylori* prevalence in asymptomatic individuals.
2. Assess associations between *H. pylori* infection and risk factors such as age, gender, education, housing conditions, water source, bed-sharing, and animal ownership.

H. *pylori*-associated PUD was defined by documented eradication therapy, involving a 7–14-day regimen of clarithromycin or metronidazole with amoxicillin or tetracycline, alongside proton pump inhibitors (PPIs) or H2 blockers.Gastric acid secretion relies on H+, K+-ATPase, which exchanges intracellular protons for luminal potassium, reducing stomach pH. PPIs like omeprazole suppress acid secretion but have slow onset and instability in acidic environments, limiting efficacy. A newer class of acid suppressors, potassium-competitive acid blockers (P-CABs), offers faster and longer-lasting acid inhibition, improving treatment outcomes for acid-related diseases.

CAUSES OF NON-H. PYLORI, NON-NSAID PEPTIC ULCERS[25]

- Gastric adenocarcinoma
- Gastric lymphoma
- Localized drug-induced irritation
- Irritation at the neck of a hiatal hernia, also known as Cameron's ulcer.
- Unexplained (idiopathic) causes
- Anastomotic ulceration following previous gastric surgery
- Post-radiotherapy effects

- Zollinger-Ellison syndrome (gastrinoma), especially in duodenal ulcers
- Multiple endocrine neoplasia type I
- Hyperparathyroidism without multiple endocrine neoplasia type I
- Systemic mastocytosis
- Severe systemic illness leading to stress ulcers (Cushing's ulcer)
- Idiopathic eosinophilic and lymphocytic gastritis
- Duodenal Crohn's disease
- Coeliac axis stenosis
- Hepatic artery chemotherapy

ROLE OF H+-ATPASE IN MAINTAINING THE MEMBRANE POTENTIAL AND ACIDIC PH AT THE PLASMA MEMBRANE SURFACE[26]

Plant cell growth is greatly influenced by environmental conditions and inherent developmental processes. Plasma membrane H+-ATPases (H+-pumps) act as key active transporters, pumping protons out of the cell to generate electrical and chemical gradients that drive solute movement. This enzyme also helps maintain an acidic cell wall environment, promoting cell expansion. In contrast, animals utilize Na+/K+ ATPase for similar purposes, creating membrane potential and a sodium gradient that facilitates sodium-dependent transporters and ion channels. Apart from the difference in cation selection, plants generally have a higher membrane potential, likely to enable the uptake of potassium to high levels, even in nutrient-poor soils, ensuring the turgor pressure needed for structural integrity and growth.

Assay of H+-K+ ATPase activity[27-28]

Proton-potassium ATPase was extracted from goat mucosal scrapings using a modified version of Cheon's method. The stomach from freshly slaughtered goats was rinsed with tap water, and the fundic mucosal layer was carefully scraped and homogenized in ice-cold phosphate buffer (pH 7.4). The homogenate underwent centrifugation at 18,000 rpm for 20 minutes, followed by a second centrifugation at 100,000 rpm for 60 minutes. The obtained pellet was reconstituted in the homogenization buffer. H+-K+ ATPase was then isolated through Ficoll-sucrose discontinuous density gradient centrifugation, and protein concentration was measured using the Lowry method. Various extract concentrations (10–50 µg/ml) were incubated in a reaction mixture containing 40 mM Tris-HCl buffer (pH 7.4), 2 mM $MgCl_2$, and 10 µg membrane protein in a total volume of 1 ml. The reaction was

initiated with 2 mM ATP-Tris salt and incubated at 37°C for 20 minutes. To halt the reaction, 1 ml of ice-cold 10% trichloroacetic acid (v/v) was added. H+-K+ ATPase activity was analyzed in the presence and absence of varying extract concentrations and omeprazole. The amount of inorganic phosphate released from ATP hydrolysis was measured spectrophotometrically at 400 nm.Gastric cancer remains one of the leading causes of cancer-related deaths worldwide. Strategies to prevent this disease focus on eliminating *Helicobacter pylori* (*H. pylori*), as early eradication before significant gastric damage can reduce the risk of cancer. *H. pylori*-associated gastric cancer is categorized as an inflammation-related malignancy. Standard eradication therapy typically involves a proton pump inhibitor or bismuth compounds combined with antibiotics such as amoxicillin, tetracycline, metronidazole, levofloxacin, clarithromycin, and bismuth. However, the increasing antibiotic resistance of *H. pylori* has led to rising treatment failure rates, exceeding 40% globally.

Functional Domains of Gastric HK ATPase[29]

Glycopeptidase F (N-glycopeptidase) breaks down a binding. There may be membrane-inserted portions of the putative cytosolic domain of the pump protein if the consensus beginning at asn 493 is, in fact, N-glycosylated from the extracellular face. Prediction based on hydrophobicity would not be possible since the membrane insertion would occur between the peptide chains rather than in the bilayer's phospholipid. However, it is not certain that this orientation is preserved in the mature protein after glycosylation, nor is it true that all of the protein gets glycosylated, even though N-glycosylation from the cytosolic face is rare.

Peptic ulcer treatment and helicobacter pylori eradication[30]

Recent research has shown that liver cirrhosis is associated with an increased risk of peptic ulcers, with a higher prevalence observed in decompensated cirrhosis compared to compensated cirrhosis. Currently, proton pump inhibitors (PPIs) are the primary treatment for peptic ulcers in the general population. Additionally, cirrhotic patients exhibit a high incidence of *Helicobacter pylori* (*H. pylori*) infection. *H. pylori* facilitates the conversion of urea into ammonia, which then enters the systemic circulation, contributing to hyperammonemia and subsequent hepatic encephalopathy (HE) episodes in cirrhotic individuals.The eradication of *H. pylori* has been enhanced through PPI-based triple therapy, which has been shown to significantly reduce blood ammonia levels in cirrhotic patients. Given that PPI-based triple therapy effectively treats *H. pylori* infection—a known factor in

increasing HE risk—it is plausible that PPIs may also help lower the likelihood of HE in patients with *H. pylori* infection.

Risk factors for the presence of symptoms in patients with PUD[31]

Individuals with symptomatic peptic ulcer disease (PUD) exhibited a higher alcohol intake compared to those with asymptomatic PUD (p=0.025). Additionally, active-stage ulcers were more frequently observed in symptomatic PUD cases than in asymptomatic ones (p=0.003). However, there were no significant differences between the two groups concerning age, sex, BMI, smoking status, *Helicobacter pylori* infection, NSAID usage, ulcer location, or ulcer count. Multivariate analysis identified heavy alcohol consumption (OR: 2.515; 95% CI: 1.315–4.812; p=0.005) and active-stage ulcers (OR: 2.143; 95% CI: 1.323–3.472; p=0.002) as independent risk factors for the presence of symptoms in PUD.

PATHOGENESIS[32,33,34]

The intestinal mucosa consists of three layers: epithelium, lamina propria, and muscularis mucosa. Gastritis involves inflammatory cells, while gastropathy refers to mucosal damage without inflammation. Peptic ulcers develop when inflammation compromises the muscularis mucosa. Gastric acid, produced by parietal cells via proton pumps (H+/K+ ATPase), becomes harmful when acid production increases or protective mechanisms fail. These defenses include a mucus layer, a pH-neutral buffer zone, epithelial tight junctions, and a rich blood supply that neutralizes excess protons. Peptic ulcers occur when damaging factors overpower these protections.Hypothermic stress ulcers significantly reduce GSH levels ($p < 0.01$) but do not affect SOD or catalase, alongside a decline in mucin and protein ($p < 0.01$-0.001). Hesperidin at 450 mg/kg notably increased GSH, protein ($p < 0.05$), and mucin ($p < 0.001$), while lower doses were ineffective. Gastric mucosa protects against injury by forming a mucous barrier. Normal gastric tissue has a structured cellular arrangement, while indomethacin induces redness, spots, and hemorrhagic ulcers.Diabetes mellitus is a prevalent chronic disease linked to lifestyle changes, reduced physical activity, and rising obesity. In 2013, 415 million people worldwide had diabetes, projected to reach 592 million by 2035. Diabetic foot ulcers (DFUs) are among its severe complications, leading to high medical costs, decreased quality of life, and an amputation risk exceeding 85%. The International Diabetes Federation prioritizes DFUs due to their significant social, medical, and economic impact.

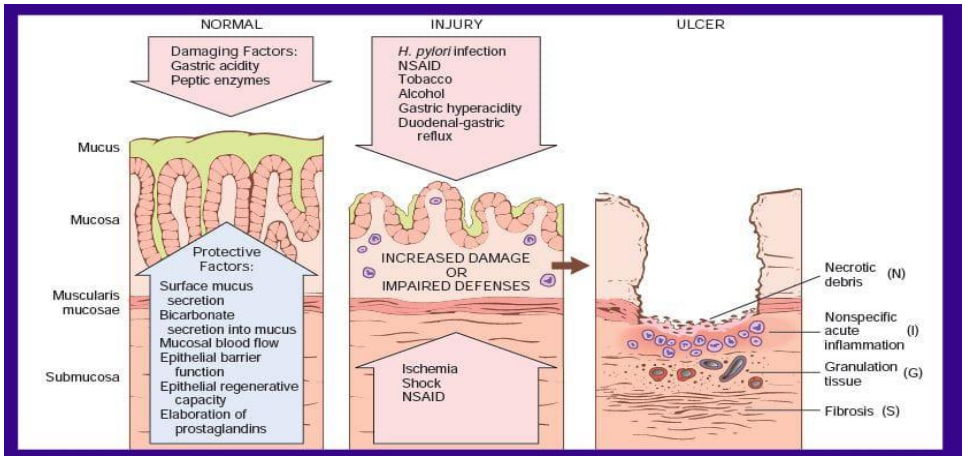


Fig. No. 06: Pathogenesis.

ULCER PREVENTION[35]

In recent years, it has become evident that selective COX-2 inhibitors are not as gastrointestinal-safe as initially promoted, prompting research into alternative methods to prevent NSAID-induced gastric ulceration. Proton pump inhibitors (PPIs) have been widely recognized for reducing serious gastrointestinal complications associated with NSAID use. Studies have shown that PPIs significantly lower the risk of ulcers in both acute and chronic NSAID users, with a low "number needed to treat"—only three elderly patients require PPI treatment to prevent one ulcer.Chan et al. investigated PPI efficacy in preventing recurrent ulcer bleeding in patients using aspirin for vascular disease prevention. After ulcer healing, patients were randomized to receive either aspirin plus esomeprazole or clopidogrel, an ADP receptor antagonist that inhibits platelet aggregation. Clopidogrel is often recommended for individuals with severe gastrointestinal intolerance to aspirin.

USE OF ANTIOXIDANTS TO ERADICATE HELICOBACTER PYLORI[18,36,39]

Research indicates that eradicating *H. pylori* is the most effective treatment for peptic ulcers. Antioxidants like vitamin C have shown promise, with lower doses (up to 500 mg/day for three months) proving more effective than higher doses. Capsaicin, found in peppers, has demonstrated gastroprotective effects in aspirin-induced ulcers but may irritate the gastric mucosa in some individuals. Depression, a mood disorder linked to HPA axis dysregulation, is associated with various gastrointestinal conditions, though its connection to PUD remains understudied. While stress is a known risk factor for PUD, previous research has mainly explored its link to anxiety and schizophrenia, not depression. Additionally, distinguishing between unipolar depression and bipolar disorder (BD) can be complex, as BD often presents initially with depressive episodes. Given the established link between BD and PUD, this

study aims to clarify whether depression alone increases PUD risk, which could have important clinical implications.

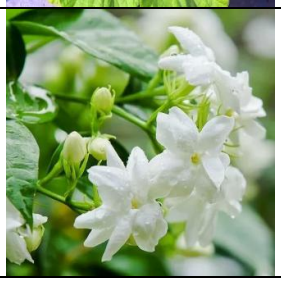
Dietery Foods for peptic ulcer

Fig. No.07: Dietery Foods for peptic ulcer.

Some Herbal plants.[40-47]

S.No	IMAGE	PLANT DISCRIPTION	PARTS	Solvent & Extraction	USES
1.		**Name:** Alchornea Castaneaefolia **Common name:** Ipururo, Ipurosa, Macochihua Nian do, Pajaro. **Family:**Euphorbiaceae	Bark,Leaves and Roots	Alchorneine, Alchorneinone, Alkaloids, Anthranilic acid, Gentisinic acid Yohimbine, flavonoids, glycosides	Antioxidant, antifungal, antiinflammatory, antibacterial, antiulcer.
2.		**Name:** Psidium guajava **Common name:** Common guava **Family:** Myrtacea	Roots, bark and leave	Methanol, acetone and N, N-dimethyl formamide (DMF) fractions	Hepatoprotective, anti-diarrheal, antihypertensive, hepatoprotective, antioxidant, antimicrobial, hypoglycemic and antimutagenic activities

3.		**Name:** Prunus amygdalus **Common name:** Almond **Family:** Rosaceae	Seed and Fruits	Methanolic, Phenolic, Ethanolic and Water	Antiulcer, anti-HIV, anti-inflammatory and in vitro antiproliferative activities, antidiabetic
4.		**Name:** Polyalthia longifolia **Common name:** False Ashoka tree **Family:** Annonaceae	Whole plant	Ethanolic, aqueous, methanol	Antiulcer, hepatoprotective, antiinflammatory, antibacterial oil used as emollient, moisturizer
5.		**Name:** Oxystelma esculentum **Common name:** Dudhia Lata **Family:**Asclepiadaceae	Leaves, petiole, stem, root and rhizome	Petroleum Ether, Methanol, Chloroform, Water	This is used in gleet, jaundice,gonorrhoea, pain in the muscles, cough and also anti-hementics antiseptic, galactagogue properties.
6.		**Name**: Nerium indicum **Common name**: Kaner **Family**: Apocynaceae	Root, leaves, whole plant	Methanolic	ardiotonic, diaphoretic, diuretic, emetic, treatment of scabies and to reduce swellings. The oil prepared from the root bark is used in the treatment of leprosy and skin diseases of scaly, nature,anti-cancer activity.
7.		**Name:** Musaparadisiacal **Common name**: Banana **Family**: Musaceae	Root, leaves, trunk	Aqueous extract, methanolic	Haemostatic, Coccidiostat, Diuretic effect, diarrhoea, dysentery, intestinal lesions in ulcerative colitis, diabetes, sprue, uremia, nephritis, gout, hypertension and cardiac disease.

8.		**Name:** Lagenaria siceraria **Common name:** Bottle gourd **Family:** Cucurbitaceae	Fruit	Methanolic	Antioxidant, antihyperlipedemic, antihyperglycemic, cardiotonic, hepatoprotective, immunomodulatory.
9.		**Name:** Jasminum grandiflorum **Common name**: Chamel **Family**: Oleaceae	Leaves, flower	Hydro-alcoholic, methanolic, ethanolic, aqueous	Ulcerative stomatitis, skin diseases, ulcers, wounds healing, antibacterial, antioxidant
10.		**Name:** Ficus religiosa **Common name**:Peepal **Family**: Moraceae	Stem, bark, Leaves, Tender Shoots,Latex, Seeds, Fruits	Ethanolic , methanolic	antidiabetic, cognitive enhancer, wound healing, anticonvulsant, anti-inantiasthmatic, parasympathetic modulatory,esterogenic, antitumor, antiulcer, inflammatory,
11.		**Name:** Bauhinia variegata **Common name:** Orchid-tree, poorman's orchid, mountainebony **Family:** Fabaceae (Leguminosae)	Root, bark, leaves	Ethanolic, methanolic and aqueous	Antibacterial, antifungal, antiulcer, hepato protective, antioxidant, anti-hyperlipidemic, bronchitis, leprosy, tumors.
12.		**Name**: Anogeissus latifolia **Common name**: Dhawa, Ghatti, Gum Ghatti, Indian Gum Tree, Indian Sumac **Family:** Combretacea	Roots, bark, leaves, fruits	Methanolic	Wounds and ulcers, inflammations, diabetes, haemorrhages, haemoptysis, diarrhoea, dysentery, haemorrhoids, skin diseases, liver diseases, and general debility.
13.		**Name:** Alchornea castaneaefolia **Common name**: Iporuru, Iporoni, Iporuro, Ipururo, Ipurosa, Macochihua, Niando, Pajaro **Family**: Euphorbiaceae	Leaves, bark , root	ethenolic, hydroethanolic	Antioxidant, antifungal, anti-inflammatory, antibacterial, antiulcer.

14.		**Name:** Achyranthes aspera **Common name:** Prickly Chaff Flower, Devil's Horsewhip **Family:** Amaranthaseae	Whole plant, root, seeds	Ethanolic	Obstetrics and gynaecology, antiulcer. For snake bites, the ground root is given with water until the patient vomits and regains consciousness, Crushed leaves rubbed on aching back to cure strained back

CONCLUSION

Peptic ulcer disease (PUD) remains a significant clinical concern, affecting individuals of all ages and posing challenges for healthcare systems, economics, and patient quality of life. As its prevalence increases with age, diagnosing PUD in patients with dyspepsia remains essential. Understanding gastric acid secretion and the role of *H. pylori*, a key risk factor, is crucial in determining disease prognosis and potential complications like stomach cancer.Medicinal plants play a vital role in managing various diseases, including PUD. Numerous herbal extracts have demonstrated antiulcer activity in animal models, exhibiting mucoprotective and gastric anti-secretory properties. These extracts are generally non-toxic, with effects often being dose-dependent. Bioactive compounds like flavonoids and tannins contribute to their therapeutic potential. Findings suggest that certain plant-based extracts could serve as effective treatments for PUD, offering promising alternatives for disease management.

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