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Peptic Ulcer:Mechanism, Pathogenesis & Treatment

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Abstract: *Peptic ulcer disease, defined as the breakdown of the stomach and/or duodenumsepithelial. The mucosal barrier is still a leading source of morbidity and mortality. One of the main etiological. The factoris H. Pylori. Using non-steroidal anti-inflammatory medicines while infected with H. Pylori(naiads). Epigastric Patients frequently experience discomfort, heartburn, reflux symptoms, and Nausea. Stomach lining inflammation is a symptom of peptic ulcer disease. Most of the time,thediagnosis is made after the endoscopy of the upper gastrointestinal tract. Ulcer is prevalent Conditionthat affects people all over the world. Allopathic ulcer treatment has negative health Consequencesdue to unpleasant side effects. Numerous herbal plants and secondary metabolites Arenow used to treat ulcers. The two most frequent causes of peptic ulcers are infection with Helicobacter pylori or taking non-steroidal anti-inflammatory drugs (nsaids), such aspirin. Nadis'rebecoming a more common cause of ulceration, notably ulcers worsened by gastrointestinal (GI)Bleeding, as H. Pylori infection becomes less common in affluent nations. Only around 15% of Peopleinfected with H. Pylori develop an ulcer in their lifetime, with the risk being dictated by the Virulenceof the H. Pylori strain, host genetics, and environmental factors (particularly smoking). TheInhibition of the gastroprotective cyclooxygenase (COX)-1 enzyme is a major cause of NSAID induced Ulcers.*

Keywords: *Peptic ulcer disease*

I. INTRODUCTION

"Usually occurring in the stomach or proximal duodenum, peptic ulcers are acid-induced lesions of the digestive tract that are characterised by denuded mucosa with the defect extending into the submucosa or muscularis propria"[1] In the general population, peptic ulcer disease is thought to affect 5–10% of people ,[2] However, recent epidemiological studies[3] have revealed a decline in the incidence, hospital admission rates, and mortality linked to peptic ulcers. The advent of new treatments and enh"Usually occurring in the stomach or proximal duodenum, peptic ulcers are acid-induced lesions of anced hygiene practises, which led to a decrease in Helicobacter pylori (H. Pylori) infections, are most likely secondary causes of this.Traditionally, it has been thought that mucosal disruption in people with acid peptic disorder results from a hypersecretory acidic
environment combined with dietary factors or stress. H. Pylori contamination, alcohol and cigarette usage, use of non-steroidal anti-inflammatory.
Medicines (NSAIDs), and Zollinger-Ellison syndrome are all risk factors for developing peptic ulcers[4] . pylori contamination and NSAID use are the key risk factors for both gastric and duodenal ulcers[5] . However, only a small proportion of people who have H. pylori or use NSAIDs develop peptic ulcers. disorder, which means that person susceptibility is vital withinside the starting of mucosal damage. Functional polymorphisms in distinct cytokine genes are related to peptic ulcers. For example, polymorphisms of interleukin 1 beta (IL 1B) have an effect on mucosal interleukin 1 beta production, inflicting H. pylori – related gastroduodenal diseases[6].
"Aspirin and NSAID users, on the other hand, had twice the risk and four times the risk, respectively, of stomach ulcer complications"[7] "Upper gastrointestinal hemorrhage is more likely when NSAIDs or aspirin are taken in conjunction[8] with anticoagulants, corticosteroids, and selective serotonin reuptake inhibitors"[9]."Although H. pylori infections are common in those who take NSAIDs or aspirin, there is debate over the role these medications play in the

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development of gastric ulcer disease. The use of independently increases the risk of developing stomach ulcer disease, according to a meta-analysis of observational studies"[10]

"Idiopathic stomach ulcer illnesses, which are categorized as H. pylori-negative, NSAIDnegative, and aspirin-negative ulcers, can be identified in roughly one-fifth of cases"[11]. "The pathogenic mechanism behind the formation of Idiopathic gastric ulcer is yet unknown but it is caused by an imbalance between elements that contribute to mucosal integrity and aggressive stroke"[6]. "Danish study has shown that psychological stress can increase the incidence of gastric ulcers[12]."Aside from ischemia, medicines (steroids, chemotherapeutic agents), radiation therapy, viruses, histamine, eosinophil infiltration, gastric bypass surgery, and metabolic problems are further causes"[13].

The most common causes of peptic ulcers are infection with Helicobacter pylori bacteria and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin or ibuprofen. Lifestyle factors like smoking, alcohol consumption, and stress can worsen the condition.Clinically, peptic ulcers present with burning epigastric pain, indigestion, nausea, and in severe cases, gastrointestinal bleeding or perforation.[7] If left untreated, they may lead to life-threatening complications. Early detection and management with acid-suppressing drugs, antibiotics for H. pylori, and lifestyle modifications play a crucial role in ulcer healing and prevention of recurrence.

Peptic ulcers are classified mainly into two types:

Gastric ulcers – occurring in the stomach.

Duodenal ulcers – occurring in the duodenum.

Typical clinical features include epigastric pain that worsens with an empty stomach, bloating, nausea, and in some cases vomiting or bleeding. If not treated properly, complications like perforation, hemorrhage, and gastric outlet obstruction may occur. Management involves eradicating H. pylori, reducing acid secretion with proton pump inhibitors or H2 blockers, and adopting lifestyle changes to promote healing and prevent recurrence.

Peptic ulcer disease is not only a common medical condition but also one with serious complications if left untreated. With the availability of diagnostic tools like endoscopy and effective treatment strategies such as proton pump inhibitors, H2 blockers, and H. pylori eradication therapy, the management of peptic ulcers has improved significantly. However, awareness regarding prevention, early diagnosis, and lifestyle modification remains essential for reducing the burden of this condition.

II. TYPES OF ULCERS

2.1 Peptic Ulcer

Any area of the digestive tract exposed to the aggressive action of acid-peptic fluids can develop persistent, most frequently solitary lesions called gastric ulcers. At least 98 percent of gastric ulcers can be found in the stomach or the first section of the duodenum, roughly in a 4:1 ratio[14] . "The contamination because of the micro-organism H. pylori and acid pepsin secretion are broadly speaking accountable for technology of peptic ulcer even as non- steroidalanti-inflammatory drugs (NSAIDs), shock, intense trauma, septicemia, intracranial lesions, irritated like alcohol, smoking and spices meals additionally accountable for production of peptic ulcers"[15] . "Symptoms of peptic ulcers consist of stomach discomfort, pain, weight loss, terrible appetite, bloating, nausea and vomiting normally and blood in stool and vomit rarely"[16].

"According to where they develop, gastric ulcers are separated into gastric ulcers and duodenal ulcers, and they happen in the stomach and duodenum, respectively. On the basis of severity, it can also be separated into acute and chronic ulcers. Acute gastric ulcers appear in the form of single or multiple lesions with submucosal tissue in all parts of the stomach and depths up to the first centimeter of the duodenum. Chronic gastric ulcers develop by themselves in the pyloric antrum of the stomach and duodenum and can penetrate the epithelial and muscular layers of the stomach or duodenum to spread to the nearby pancreas or liver"[17] . They can cause complications such as obstruction, bleeding, perforation and malignant transformation[18]

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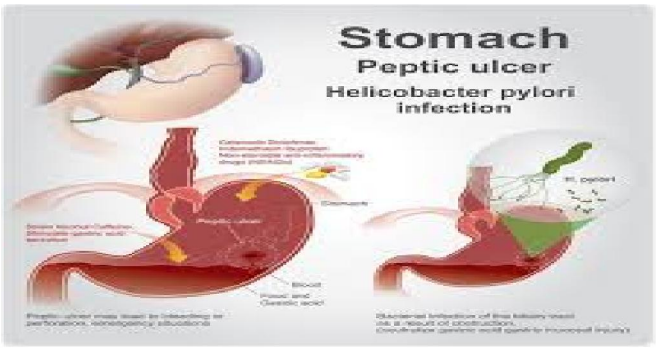

Fig:1 Peptic Ulcer

2 Esophageal Ulcer

A lesion that develops in the esophagus is called an esophageal ulcer (the food pipe). These are most frequently developed at the esophageal margin and can cause pain directly below the sternum, where heartburn symptoms are also felt. Esophageal ulcers are associated with gastroesophageal reflux disease or long- term use of drugs such GERD, NSAIDs, and smoking [19]

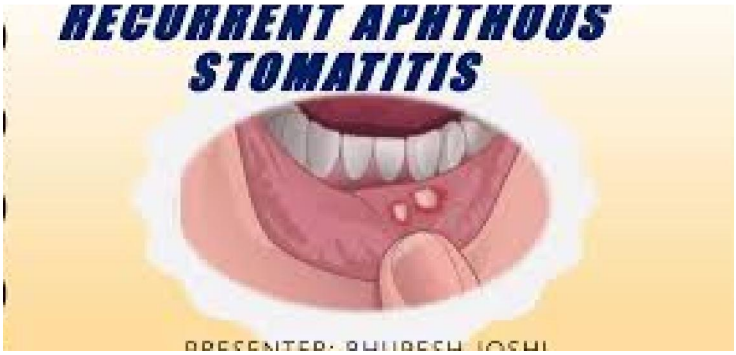

Fig 2: Esophageal Ulcer

2.3 Aphthous Ulcer

"Aphthous ulcers are typically recurrent round or oval sores or ulcers inside the mouth on areas where the skin is not tightly bound to the underlying bone, such as on the inside of the lips and cheeks or underneath the tongue. These sores or ulcers have a yellow greyish pseudo membrane surrounded by raised margins and an erythematous hole. They are sometimes referred to as canker sores, mouth ulcers, aphthosis, and aphthous stomatitis"[20] . Mouth ulcers occur in families (up to 40%) and are usually traumatic (due to improper placement of teeth, damage to teeth and fillings), anemia, ulcers, viral infections, oral candidiasis, chronic infections, and laryngeal cancer, mouth cancer and vitamin B deficiency. According to estimates, 50–66% of people in North America and 15-20% of the world's population experience mouth ulcers[21] . "They are herpetiform ulcers (less than 5 mm in size, 10-14 percent duration, and 5-10 percent prevalence), big ulcers (above 10 mm in size, more than 2-week duration), and moderate ulcers (size 510 mm, duration 10-14 days, prevalence

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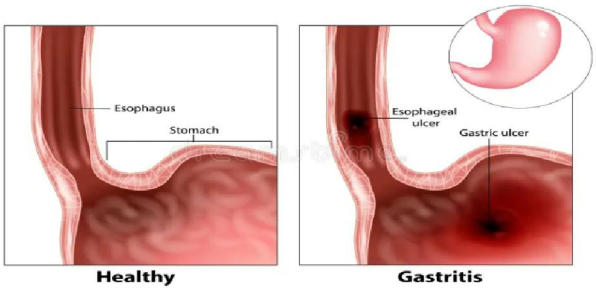

Fig 3: Aphthous Ulcer

III. TYPES OF PEPTIC ULCER

3.1 Gastric Ulcer

A gastric ulcer is a type of peptic ulcer disease (PUD) characterized by the development of an open sore or lesion on the inner lining of the stomach. It occurs when the normal balance between the protective mechanisms of the gastric mucosa (mucus, bicarbonate secretion, prostaglandins, and adequate blood flow) and the aggressive factors (gastric acid, pepsin, Helicobacter pylori infection, and irritant drugs such as NSAIDs) is disrupted. This imbalance leads to erosion of the stomach's mucosal barrier, exposing the underlying tissues to acid and digestive enzymes, which cause pain, inflammation, and potential complications.

Gastric ulcers are a significant global health concern, affecting millions of people worldwide[10]. They can present with burning epigastric pain, nausea, vomiting, bloating, and, in severe cases, gastrointestinal bleeding. Unlike duodenal ulcers, which usually improve after eating, gastric ulcer pain often worsens with food intake, leading to loss of appetite and weight loss[19].The condition is most associated with H. pylori infection and prolonged use of NSAIDs, but lifestyle factors such as smoking, alcohol consumption, and stress may aggravate the disease. If untreated, gastric ulcers can result in serious complications such as bleeding, perforation, gastric outlet obstruction, or even malignant transformation.

Because of these risks, early diagnosis and management are essential. With the availability of advanced diagnostic tools such as endoscopy and effective therapies like proton pump inhibitors and H. pylori eradication regimens, most gastric ulcers can be treated successfully, leading to significant improvement in patient outcomes.

Duodenal Ulcer A duodenal ulcer is a type of peptic ulcer disease (PUD) that occurs in the duodenum, the first part of the small intestine immediately beyond the stomach. It is one of the most common forms of peptic ulcers, affecting millions of people worldwide. The ulcer represents a localized erosion or sore in the mucosal lining of the duodenum caused by an imbalance between the aggressive factors (gastricacid, pepsin, Helicobacter pylori infection, and NSAID use) and the protective factors (such as mucus secretion, bicarbonate, and mucosal blood flow]

Duodenal ulcers are usually chronic and recurrent in nature, often presenting characteristic symptoms like burning epigastric pain that tends to improve after meals but worsens a few hours later or at night[9]. Unlike gastric ulcers, duodenal ulcers are more common in younger individuals (especially men aged 20–40 years). The condition is most strongly associated with Helicobacter pylori infection, which leads to chronic inflammation of the duodenal mucosa, and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs). Lifestyle factors such as smoking, alcohol intake, and stress can worsen the disease. untreated, duodenal ulcers can result in serious complications such as bleeding, perforation, gastric outlet obstruction, or penetration into adjacent organs, making early diagnosis and proper management essential. With advances in endoscopy, diagnostic tests for H. pylori, and effective pharmacological therapies (like proton pump inhibitors and antibiotics), the prognosis of duodenal ulcers has significantly improved, reducing recurrence and complications[4].

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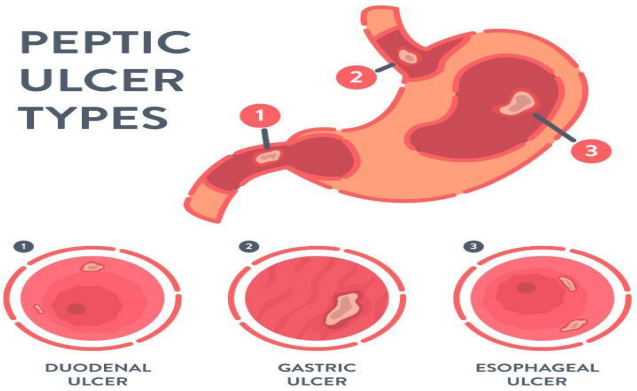

Fig 4: TYPES OF ULCER

VI. ETIOLOGY AND RISK FACTOR

The etiology of peptic ulcer disease (PUD) refers to the various underlying causes and mechanisms that lead to the breakdown of the gastrointestinal mucosa, resulting in ulcer formation. Peptic ulcers develop when the protective factors of the mucosa are overwhelmed by aggressive factors such as gastric acid, pepsin, bacterial toxins, and chemical injury.

Historically, stress and diet were considered the primary causes, but the discovery of Helicobacter pylori (H. pylori) infection in the 1980s transformed the understanding of peptic ulcer etiology[20]. Today, H. pylori infection and nonsteroidal anti-inflammatory drugs (NSAIDs) are recognized as the two most important causes worldwide, with additional contributions from acid hypersecretion, lifestyle habits, genetic predisposition, and systemic diseases. The etiology is multifactorial, meaning that more than one cause may interact with the same patient. For example, a person with H. pylori infection who also takes NSAIDs, and smokes has a much higher risk of ulcer formation than someone with just one of these factors.

Main Etiological Factors in Detail

Helicobacter pylori Infection

Most common cause of duodenal and gastric ulcers. The bacteria colonize the gastric mucosa, producing urease, which neutralizes acid locally and allows survival. Release of cytotoxins and induction of chronic gastritis damage the mucosal barrier. Leads to increased gastric acid secretion (especially in duodenal ulcers).

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

The second most important cause of ulcers, especially gastric ulcers.

NSAIDs inhibit COX-1 enzyme, leading to ↓ prostaglandins → ↓ mucus, bicarbonate, and mucosal blood flow. Directly irritate gastric mucosa. Common in elderly patients using aspirin/NSAIDs for cardiovascular disease or arthritis.

Gastric Acid and Pepsin Overactivity

Hyperacidity itself may not always cause ulcers but contributes to mucosal damagewhen defenses are impaired. Seen in Zollinger–Ellison syndrome (gastronome) – causes severe, multiple, refractory ulcers.Acid and pepsin act synergistically to digest and erode mucosa.

Lifestyle and Environmental Factors

Smoking: impairs mucosal blood flow, reduces healing, and increases recurrence. Alcohol: irritates gastric mucosa, causes superficial erosions, increases bleeding risk. Caffeine and spicy foods: stimulate acid secretion (not direct cause but aggravating). Psychological stress: previously considered primary cause; now recognized to exacerbate but not initiate ulcers.

.Genetic and Familial Predisposition

Family history increases risk, showing genetic susceptibility. The blood group of individuals have a higher incidence of duodenal ulcers. Genetic variations may affect mucosal defense or immune response to H. pylori.

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Systemic Diseases and Other Causes

Cirrhosis, chronic kidney disease, COPD – increase ulcer risk due to impaired mucosal circulation. Corticosteroids: weak cause alone, but when curling's ulcer – after severe burns.

- Cushing's ulcer – associated with head trauma or brain surgery.
- Chemotherapy and radiotherapy: can damage gastric mucosa. combined with NSAIDs strongly increase ulcer risk. Critical illness/physiological stress.

RISK FACTOR

1.Infection-related Risk Factors

Helicobacter pylori infection:

Most important worldwide risk factor.

Prevalence is higher in developing countries. It leads to chronic gastritis, impaired mucosal defense, and acid hypersecretion.

2. Medication-related Risk Factors

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs):

Long-term use of aspirin, ibuprofen, naproxen etc.

Especially dangerous in elderly patients and those with comorbidities.

Corticosteroids: Alone, they have minimal risk, but in combination with NSAIDs the ulcer risk increases dramatically.

Anticoagulants and antiplatelets (e.g., warfarin, clopidogrel): Increase the severity of bleeding in existing ulcers.

3. Lifestyle Risk Factors

Smoking:

Increases gastric acid secretion.

Impairs mucosal blood flow and healing → higher recurrence rate.

Alcohol:

Direct irritating to gastric mucosa.

Increases the risk of bleeding and delays healing.

Caffeine and Diet:

Coffee and caffeine stimulate gastric acid secretion.

Spicy, oily, or irregular meals aggravate symptoms but are not direct causes.

Stress:

Physical or psychological stress increases susceptibility.

Severe stress (trauma, burns, ICU stay) leads to "stress ulcers."

4. Genetic and Familial Risk Factors

Family history: Increases overall susceptibility.

Blood group O: Higher incidence of duodenal ulcers due to possible differences in mucosal glycoproteins.

Twin studies: Show higher concordance in monozygotic twins, indicating a genetic component.

5. Demographic Risk Factors

Age:

Duodenal ulcers → common in younger adults (20–40 years).

Gastric ulcers → common in elderly (50–70 years), partly due to NSAID use.

Gender:

Duodenal ulcers more common in men.

Gastric ulcers slightly more common in women, especially postmenopausal.

6. Medical Conditions as Risk Factors

Chronic diseases:

Cirrhosis of liver.

Chronic kidney disease.

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COPD (chronic obstructive pulmonary disease).
Endocrine conditions:Zollinger–Ellison syndrome (gastrinoma → hypersecretion of acid).
Critical illness:
Curling's ulcer → after severe burns.
Cushing's ulcer → associated with head trauma or brain surgery.
7. Socioeconomic and Environmental Risk Factors
Low socioeconomic status: Higher prevalence of H. pylori due to poor sanitation and overcrowding.
Geographical variations: Developing countries have higher infection and ulcer rates.

Aggressive Factor	Protractive Factor
Gastric acid (HCL)	Muscus seceration
Pepsin	Bicaaarbonate secreation
H. pylori infection	Prosatglandins
NSAIDs, Alcohol, Smoking.	Adequate blood flow
Stress, Corticosteroids	Rpaid epithelial cell renewal

V. PREVALENCE

"Modern times have seen a decline in the prevalence of peptic ulcer disease (PUD), in part due to the widespread use of proton pump inhibitors (PPIs) and in part due to the early detection and treatment of H. pylori infections. An infection of stomach epithelium by the gram-negative spiral bacillus H. pylori Leadbeater development of gastric ulcers. Infection with Helicobacter pylori continues to be the most frequent cause of PUD, accounting for 95% of duodenal ulcers and 70% of stomach ulcers"[22] .Increased use on NSAIDs further increases the prevalence of PUD, which accounts for the majority of the remaining 30% of stomach ulcer disease (21). A higher risk of hemorrhagic PUD complications is also linked to the use of NSAIDs[23]."Predisposition to gastric ulcer disease includes the use of corticosteroids, physiological stress, inflammatory bowel disease, and Zollinger-Ellison syndrome"[24] ."In addition, smoking and drinking lead to the development of more severe PUD and delayed healing of the treated disease" [25].
Peptic ulcer disease is a global health problem, but its prevalence has declined over the last few decades due to better hygiene, improved detection and treatment of Helicobacter pylori (H. pylori), and widespread use of proton pump inhibitors (PPIs). However, in certain populations, it remains common and a major cause of morbidity.

Global Prevalence

- Worldwide prevalence of peptic ulcer is estimated at 5–10% of the population at some point in their lifetime.
- H. pylori infection is the leading cause and its prevalence parallels that of ulcers:
- In developing countries → H. pylori infection rates may reach 70–90%, making ulcers more common.
- In developed countries → lower prevalence (20–40%) due to better sanitation and effective eradication therapy.

Lifetime risk of developing an ulcer:
10–12% in men
8–10% in women

Prevalence in India

- In India, the prevalence of PUD remains relatively high due to widespread H. pylori infection, socioeconomic factors, and high use of NSAIDs for pain relief.
- Community-based studies in India show 8–10% prevalence in adults.
- Duodenal ulcers are more common in India than gastric ulcers, especially in younger populations.

Changing Trends

- Over the past 30 years, the overall prevalence has declined due to:

Better understanding and eradication of H. pylori.

- Widespread use of PPIs.

Improved sanitation and hygiene.

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However, NSAID-related ulcers and complications (like bleeding in elderly patients) are increasing.

VI. PATHOGENS

Lesions, defenses, and repairs of the gastric mucosa that are continuously exposed to the harmed environment play a role in the development of gastric ulcers[25] . These harmful environments disagree between harmful factors (e.g., Pepsin, acid, H. pylori infection) and protective factors (e.g., Prostaglandins, mucins, nitrogen monoxide, bicarbonate, and growth factors). It is due to. The main risk factors for stomach ulcers are frequent use of NSAIDs, heavy alcohol consumption, a poor diet, smoking habits, and psychological stress[26] . In the etiology of peptic ulcer, NSAID overdose and H. pylori play the most significant roles (Fig. 1).

6.1 Helicobacter Pylori and Its Role to Induce Ulcer

The primary contributing factor to duodenal and stomach ulcers has been identified as H. pylori (Fig. 1). Most pathogens populating the human gastric antral mucosa are H. pylori"[27] , "which is also responsible for around 95% of gastric and 70% of duodenal ulcerations in humans"[28]." The epithelium is harmed as a result of an inflammatory reaction inside the mucosa brought on by the local irritation brought on by H. pylori infection. The most hazardous virulence genes linked to peptic ulcer illness are vacuolating cytotoxin (VacA) and cytotoxin associated gene A (CagA) in H. Pylori, which contains a variety of genes"[29]."The CagA gene is in charge of translocating a carcinogenic material into the host gastric cells. The type IV secretion system (T4SS), encoded by the cag pathogenicity island (cag-PAI), is used for the translocation"[30]. "A group of 31 genes known as the pathogenicity island have been identified in specific bacterial strains"[31] . According to earlier publications, some of the cag-PAI genes contained T4ss[32] , "which formed a syringe-like pilus structure that extended from the bacterial surface into host cells for the delivery of the virulence factors, primarily CagA"[33] . "After injection, CagA is tyrosinephosphorylated, causing changes in the intracellular signaling system, which virtually leads to infection. T4SS is a solid indicator of severe gastric ulcer infection. Therefore, advanced research to investigate the role of T4SS in CagA transmission is very important. In addition, Cagl is also involved in Cagan secretion and is capable of binding to integrin membrane β1 as an additional T4SS component"[34] . "It is believed that the host's genetic makeup has a significant role in determining how duodenal or gastric ulcers develop in terms of their pattern of gastric inflammatory development". "The only means to ensure their survival is by the creation of the urease enzyme by H. pylori, which converts urea to ammonia and carbon dioxide. As the urease enzyme is produced, stomach acid is subsequently buffered, allowing bacteria to defend themselves against the acidic gastric environment. [35] "It is known that the environment's alkalinity release prevents the stimulating release of somatostatin from antral D cells. The less stimulation of somatostatin inhibits the control of gastrin secretion by antral G cells" [30]. "Results to hypergastrinemia, excessive gastric acid secretion and parietal cell hyperplasia due to the uncontrolled production of gastrin. H. pylori infectionis also interfered with the neural pathways of the brain in terms of regulation of the secretion of gastric acid and impede the inhibitory reflex that down regulates the release of acid. Moreover, an impaired signal from the neural pathway results in hypersecretion of stomach acid thus diminishing the pH inside the duodenum. The duodenal bulb contains this. The optimal environment for H. pylori colonization, which ultimately leads to ulceration, is metaplasia".

6.2 Dignosis of Peptic Ulcer

The most typical method of diagnosing ulcers is by symptoms, which also consider the patient's age and the location of the ulcer. A stomach ulcer is a painful condition that typically starts on an empty stomach, is treated with antacids and food, but is also brought on by alcohol and caffeine. However, stomach ulcer disease is more likely to cause weight loss and gastrointestinal bleeding. Duodenal ulcers typically cause more persistent pain in the morning, which is only reduced by eating and sleeping for a short while at night. Signs of bleeding, repeated vomiting, or abdominal pain are important in diagnosing duodenal ulcer. [12]

"Peptic ulcer can be diagnosed specifically by direct visualization by endoscopy or radiology and by detection of H. pylori by various endoscopic and non-endoscopic tests. Endoscopic tests involve Histology, Culture of Biopsy,

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Rapid urease detection with ammonia, while non-endoscopic tests consist of detection of antibodies to H. pylori in serum, Urea breathe test (H. pylori urease breaks down ingested labelled C urea, patient exhales labelled CO2) and stool antigen test (presence of antigen against H. pylori in stoolchanges its color which can be detected visually or by spectrophotometer)" [30] Fig. 2.

6.3 Anatomy and Location of Peptic Ulcer Disease

"Both the duodenum and the stomach can develop peptic ulcers. The gastric cardia, fundus, body, antrum, and pylorus are the different parts of the stomach. Visceral peritoneum lines every side of the stomach. The incisura angularis is an acute angle indentation along the lesser curvature of the stomach wall and marks the division of the stomach body and the antrum [Fig. 1] Gastric ulcers typically occur at the gastric antrum, with the lesser curvature being the most common location" [17]. "With the use of NSAIDs, peptic ulcers can also develop along the larger curvature of the stomach" [22]. There is no mesentery in the duodenum, and the peritoneum only partially encloses the organ. [5] "There are four sections in the duodenum. The duodenal bulb, which rises from the pylorus, is the first portion of the duodenum. Only the bulb of the duodenum has visceral peritoneum covering its whole surface. The duodenum's remaining sections are retroperitoneal. The pancreatic duct and common bile duct are located in the second section of the duodenum, which descends. At the level of the second lumbar vertebral body, the third (horizontal) portion crosses the retroperitoneal median. From the aorta to the Treitz ligaments is the fourth segment of the duodenum" [30] Fig. 3. 95 percent of duodenal ulcers occur in the duodenal bulb, making it the most frequent place [19]. Ulcers distal to the duodenal bulb, known as post-bulbar ulcers, are uncommon, occurring in only 3-5 percent of patients. Rare etiologies like Zollinger-Ellison Syndrome or Crohn's disease should be taken into consideration when post-bulbar ulcers are observed. [14]. Additionally, compared to bulbar ulcers, post-bulbar ulcers are more likely to bleed because of their closeness to the gastroduodenal artery [10].

"The majority of posterior duodenal ulcers develop on the duodenum's anterior wall, close to the ampulla. Due to edema, reactive tissue, and spasm at the ulcer disease site, duodenal ulcers might show an inward bowing of the opposite all that can conceal an outpouching characteristic of an ulcer crater. As a result, this inflammatory process may make it difficult to spot ulcer craters, which are a particular feature that can be seen on fluoroscopic imaging" [13].

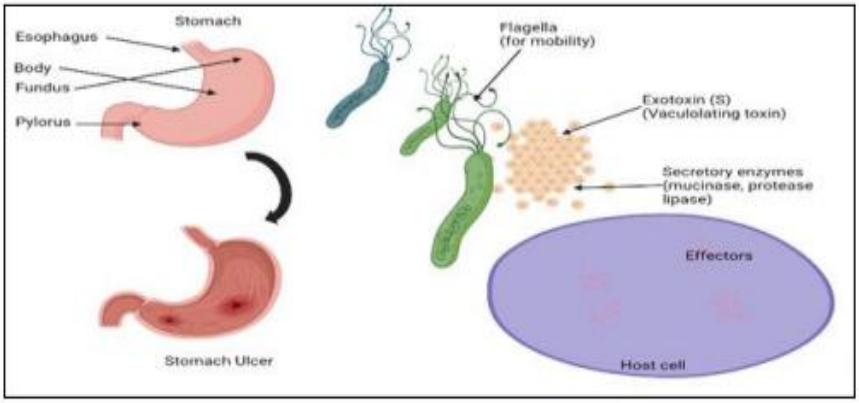

Fig. 1. Pathogenesis of peptic ulcer disease. The secretion during the pathogenesis is shows The normal stomach and the formation of peptic ulcer are also displayed

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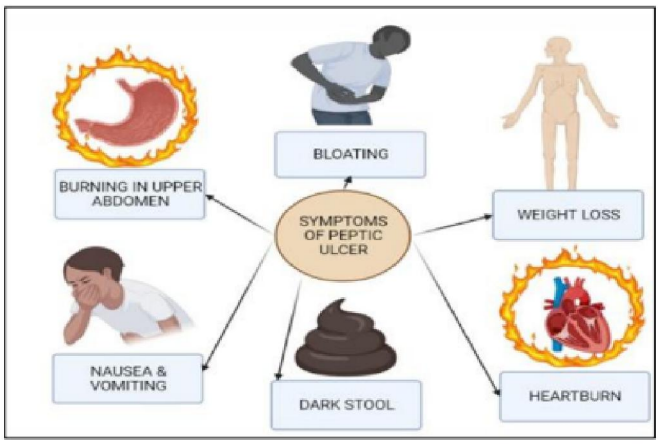

Fig. 2. Symptoms of peptic ulcer

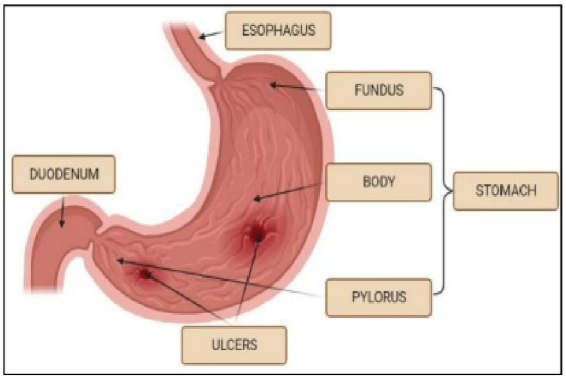

Fig. 3. Cross sectional view of duodenum

VII. MECHANISM OF PEPTIC ULCER

Peptic ulcer disease (PUD) develops due to a disruption of the equilibrium between mucosal defensive mechanisms and aggressive factors (acid, pepsin, H. pylori, and NSAIDs).[33]

The mechanism involves three sequential phases:

1. Initiation of mucosal injury
2. Progression to ulceration
3. Failure of mucosal healing

1. Initiation Phase: Disruption of the Mucosal Barrier

Under normal conditions, the stomach and duodenum are protected by a mucus-bicarbonate-phospholipid barrier, tight epithelial junctions, and adequate mucosal blood flow.

A. Aggressive Factors Acting on the Barrier

a.Gastric Acid (HCl) : Secreted by parietal cells under the stimulation of, Gastrin (from G-cell) Acetylcholine (via vagal stimulation), Histamine (from enterochromaffin-like cells), Acid diffuses through the mucus layer if the barrier is compromised, directly injuring epithelial cells.

b. pepsin:

Formed by activation of pepsinogen (from chief cells) in acidic pH (<3.5). It digests mucosal proteins and damages the epithelium, exposing deeper layers.

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c.Helicobacter pylori Infection

H. pylori colonize the gastric antrum and secretes urease, which converts urea → ammonia (NH_3) and CO2 Ammonia neutralizes acid locally but damages epithelial cells.

The bacterium also produces.

CagA (cytotoxin-associated gene A): Disrupts cell signaling and tight junctions.

VacA (vacuolating cytotoxin A): Causes vacuolation, mitochondrial dysfunction, and apoptosis.

Inflammation due to IL-1β, IL-8, and TNF-α recruit's neutrophils and macrophages, which release reactive oxygen species (ROS) and proteases, damaging the mucosa.

d.NSAIDs

Systemic effect: Inhibition of COX-1 and COX-2 enzymes → ↓ prostaglandins (PGE_2, PGI_2) → ↓ mucus, ↓ bicarbonate, and ↓ blood flow.

e. Bile Reflux and Smoking

Bile salts disrupt the phospholipid layer and increase H^+ back-diffusion.Smoking impairs healing by reducing mucosal blood flow and prostaglandin synthesis.

2. Propagation Phase: Cellular and Molecular Events

Once the mucosal barrier is disrupted, acid and pepsin penetrate deeper into the mucosa, initiating cellular injury and inflammation.

A. Back-Diffusion of Hydrogen Ions

Acid (H^+) enters epithelial cells → intracellular acidosis → mitochondrial dysfunction → ATP depletion.

This causes loss of cell membrane integrity and necrosis.

B. Inflammatory Cascade Activation

Damaged cells release cytokines (IL-1β, TNF-α, IL-8) and arachidonic acid metabolites.

Neutrophil infiltration increases → release of ROS and proteolytic enzymes.

Endothelial damage → vasoconstriction → decreased mucosal blood flow → ischemia.

Ischemia further aggravates the injury and slows healing.

C. Role of Histamine

Tissue injury stimulates mast cell degranulation → histamine release.

Histamine increases vascular permeability and stimulates parietal cells → more acid secretion → vicious cycle of injury.

D. Apoptosis and Cellular Death

H. pylori VacA and ROS activate mitochondrial pathways → caspase-mediated apoptosis.

CagA disrupts β-catenin signaling and increases epithelial permeability.

Loss of epithelial cells expose the lamina propria → inflammatory exudate forms an ulcer crater.

3. Ulcer Formation Phase

When the injury extends through the muscularis mucosae, an ulcer crater is formed.

Pathological Sequence:

1.Erosion: Superficial necrosis confined to mucosa.

2.Acute Ulcer: Necrosis extends into submucosa and muscularis mucosae.

3. Chronic Ulcer: Persistent inflammation → fibrosis, granulation tissue, and thickened base.

Microscopic Features:

1.Surface zone of necrotic debris.

2.Underlying layer of nonspecific inflammatory cells (neutrophils, macrophages).

3.Granulation tissue beneath the inflammatory zone.

4.Fibrotic scar at the base in chronic lesions.

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4. Failure of Healing and Chronicity

1.Several factors interfere with healing:

2.Persistent H. pylori infection

3.Continuous NSAID therapy

4.Smoking and alcohol use

5.Reduced mucosal blood flow (ischemia)

Nutritional deficiency (e.g., Vitamin C deficiency impairs collagen synthesis)

Ulcers heal by fibrosis and epithelial regeneration, but when aggressive factors persist, healing is incomplete, and the ulcer recurs.

5. Molecular Mechanisms Summary

Mechanism	Key Mediator	Effect
H. Pylori infection	Urease, CagA, VacA,IL-8	Inflammation, cell injury.
Acide pepsin injury	Gastric, histamine,	Chemical corrosion of mucosa
Ischemia	DecreesNO, and blood flow	Hypoxia and tissue necrosis
NSAIDs	COX inhibition peroxide	Bicarbonate secreation

6. Vicious Cycle of Ulcer Formation

(1) Mucosal damage

(2) H^+ ion back-diffusion

(3) Inflammation and histamine release

(4) Increased acid secretion

(5) Further mucosal damage

(6) Ulceration

(7) Impaired healing due to continued exposure.

7.Healing Mechanism (If Aggressive Factors Removed)

Epithelial restitution: Migration of healthy cells to cover the ulcer base.

Angiogenesis: Granulation tissue formation and capillary growth restore perfusion.

Prostaglandins and growth factors (EGF, TGF-β) stimulate regeneration.

Healing is rapid if acid secretion is suppressed (e.g., by PPIs) and H. pylori eradicated.

VIII. TREATMENT

Peptic ulcer is a condition characterized by sores or ulcers in the lining of the stomach (gastric ulcer) or the first part of the small intestine (duodenal ulcer). Treatment aims to relieve symptoms, heal the ulcer, prevent complications, and reduce recurrence.[21]

1. Lifestyle and Dietary Measures

a. Avoid NSAIDs (e.g., ibuprofen, aspirin) if possible, as they can worsen ulcers.

b. Limit alcohol intake and stop smoking.

c.Eat small, frequent meals; avoid spicy, acidic, or irritating foods.

d.Manage stress through relaxation techniques, as stress can exacerbate symptoms.

2. Medical Treatment

a) Eradication of Helicobacter pylori (H. pylori)

Most peptic ulcers are caused by H. pylori infection. Eradication is crucial to prevent recurrence.

- Standard Triple Therapy (usually 14 days):

  Proton Pump Inhibitor (PPI): Omeprazole, Pantoprazole, or Lansoprazole

  Clarithromycin.

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Amoxicillin (or Metronidazole if allergic to penicillin).

- Quadruple Therapy (if resistance or failure of triple therapy):

PPI
Bismuth subsalicylate
Tetracycline
Metronidazole

b) Acid Suppression Therapy

- To promote healing and reduce pain:

Proton Pump Inhibitors (PPIs) – Most effective for ulcer healing:
Omeprazole, Esomeprazole, Pantoprazole, Lansoprazole
Usually taken once daily before meals
Duration: 4–8 weeks for gastric ulcers, 2–4 weeks for duodenal ulcers

- H2-Receptor Antagonists (H2 blockers) – Reduce acid secretion:

Ranitidine (less used now due to recalls), Famotidine, Nizatidine
Used if PPIs are not tolerated

- Antacids and Alginates – Provide symptomatic relief by neutralizing stomach acid:

Example: Magnesium hydroxide, Aluminum hydroxide, Sodium alginate

c) Cytoprotective Agents
Sucralfate: Forms a protective barrier over ulcer site.
Misoprostol: Prostaglandin analogue; useful especially for NSAID-induced ulcers.
3. Management of Complications
Complicated ulcers may require hospitalization and sometimes surgery:
Bleeding ulcer: Endoscopic therapy, proton pump inhibitors, or surgery if severe.
Perforation: Emergency surgery (e.g., Graham patch repair).
Obstruction: Balloon dilation or surgery if there is pyloric stenosis.
4. Follow-Up
Re-evaluate with endoscopy if the ulcer is large, gastric ulcer is present (to rule out malignancy), or if symptoms persist after treatment.
Long-term acid suppression may be needed in high-risk patients (e.g., elderly, chronic NSAID users).
Overview of conventional antiulcer treatment options is summarized in Table 1 and Table 2 [30].

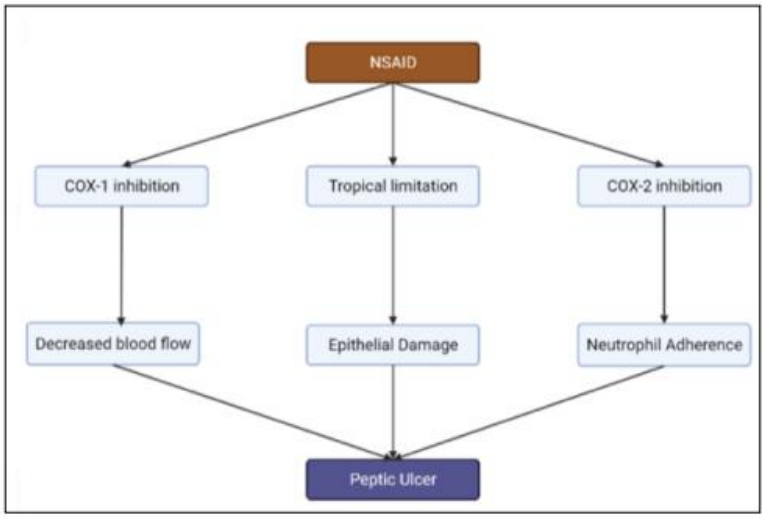

Fig: Diagnosis of gastric ulcer and the number of associated complications

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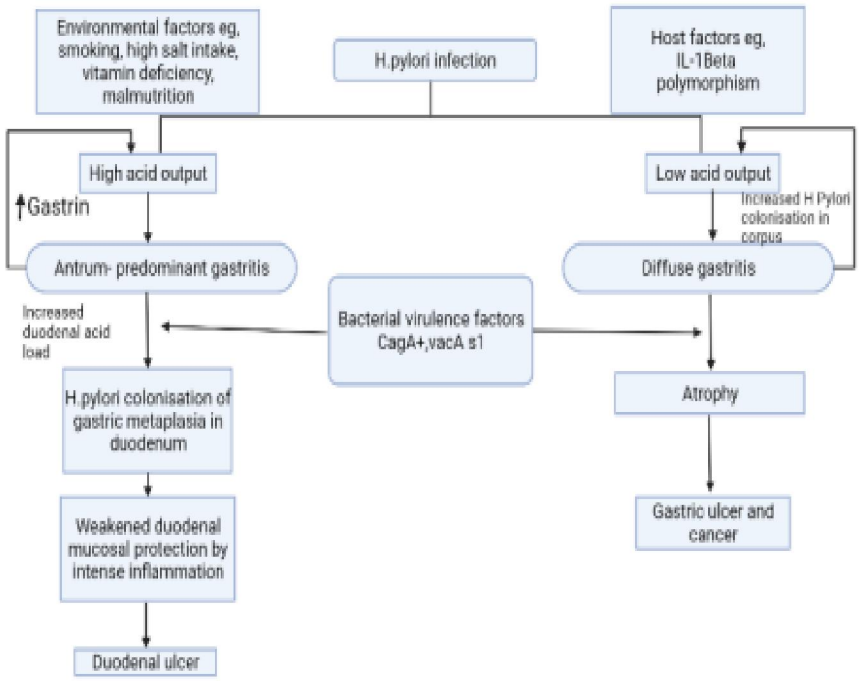

Fig: Non- aspirin NSAID

Table 1. Mechanisms of action and adverse effects of the most commonly used antiulcer treatment options

Medicine	Mechanism of action	Adverse Effects	References
H2 Receptor Blockers Cimetidine Famotidine Nizatidine Ranitidine	Blocking the activity of histamine on parietal cells' histamine H2 receptors.	Headache, Dizziness, Depression, Anxiety, Cardiovascular, Events, Thrombocytopenia.	[57]
Proton pump inhibitors(PPIs) Omeprazole Pantoprazole Lansoprazole Rabeprazole Esomeprazole	Inhibition of the gastric H/K-ATPase (proton pump) enzyme system	Headache Abdominal pain Nausea Vomiting Constipation Vitamin B12 Deficiency Osteoporosis Flatulence	[58,59]
Antacids Aluminium Hydroxide Magnesium Hydroxide	Increases gastric pH to greater than four, and inhibits the proteolytic activity of pepsin Causes osmotic retention of fluid	Frequency not defined: Nausea Vomiting Hypophosphatemia Chalky taste Constipation Abdominal Cramping Diarrhoea Electrolyte imbalance	[60]
Potassium-competitive acid blocker Vonoprazan	Inhibits H+, K+-ATPase in gastric parietal cells at the final stage of the acid secretory pathway	Contusion Nasopharyngitis Fall Upper respiratory Tract inflammation Eczema Diarrhoea Constipation	[61,62,63,64,65]

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Table 2. Types and efficiency of Helicobacter(H-pylori) eradication options [56]

Type	Duration	Efficiency	References
First line PPI+two antibiotics (clarithromycin + metronidazole or amoxicillin)	7-14 days	70-85%	[66]
Second line	14 days	77-93%	[67,68]
Bismuth-containing quadruple therapy: PPI + bismuth salt + tetracycline + metronidazole			
Non- bismuth based concomitant therapy: PPI + clarithromycin + amoxicillin + metronidazole	14 days	75-90%	
Levofloxacin triple therapy: PPI+ amoxicillin + levofloxacin	14 days	74-90%	
Salvage regimens	10 days	66-70%	[69]
Rifabutin- based triple therapy: PPI + rifabutin + amoxicillin			

IX. PREVENTION OF PEPTIC ULCER DISEASE

NSAIDs increase the risk of rebleeding in patients with a history of gastric ulcer. The 2012 ACG Guidelines for the Management of Hemorrhagic Ulcer Patients Carefully Note the Need for Continued Use of NSAIDs in Patients with a History of Gastric Ulcer Disease and, if Possible, permanent Discontinuation of NSAIDs. Recommended to evaluate[14] . It is advised to use PPIs with NSAIDs that are selective for cyclooxygenase (COX) 2 at the lowest effective dose. Combining proton pump inhibitors with COX2 selective NSAIDs lowers the risk of rebleeding. This is believed to be caused by COX1's diminished impact on the gastrointestinal mucosa [29].

In addition, when taking NSAIDs and having H. pylori infection, the risk of bleeding is higher than when taking NSAIDs by themselves [23] Eradication lowers the likelihood of rebleeding in people with H. pylori infection who are unable to quit taking NSAIDs. Long-term PPI medication paired with H. pylori infection.For patients with gastric ulcer due to 57 H. pylori alone. These patients do not require long-term PPI therapy because the H. pylori infection can be treated effectively [15]. Aspirin is also significantly associated with gastric ulcer and bleeding. In one study, the relative risk of bleeding from low-dose aspirin was 1.80 (95% confidence interval 1.59 to 2.03) compared to placebo [85].However, the mortality rate of those who restarted aspirin immediately after discharge is much lower in patients taking aspirin for secondary prevention of cardiovascular and cerebrovascular illness. In one study, patients with comorbid cardiovascular disease who discontinued aspirin after admission due to gastric ulcer bleeding died and had cardiovascular events within the first 6 months compared to patients who resumed aspirin at discharge. We found that the risk of bleeding was significantly increased (31% vs. 8%) [14]. In a controlled experiment conducted in 2011, patients taking low doses of aspirin and receiving endoscopic hemostatic therapy for gastric ulcer bleeding were treated with continuous aspirin therapy for gastric ulcer bleeding. We have found that the risk of recurrence is increased, but the mortality rate may be reduced. For this reason, ACG guidelines may recommend it to patients.

It is less known how to treat idiopathic ulcers (H. pylori negative, not caused by NSAIDs or aspirin, for example). Compared to controls who had pylori-positive ulcers, H. pylori patients with idiopathic ulcers had a greater relative risk of rebleeding and mortality [22]. The ACG Guideline conditionally recommend daily PPI therapy for these patients, but the data are very limited [32].

X. CONCLUSION

The clinical burden of PUD is decreasing as a result of a decline in H. pylori infections, improved accessibility to anti-secretory drugs, and more cautious NSAID use. However, because of its high lifetime frequency and diverse clinical presentation, early detection and treatment are essential.PUD care is essential for avoiding and minimizing serious consequences.The review could be valuable in supplementing knowledge on identifying symptoms, diagnosing them,

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treating them, determining their prevalence, managing them, and using allopathic medications to treat ulcers. This article encourages scientists and helps to identify ulcer illness.

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