

REVIEW ON PEPTIC ULCER DISEASE

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ABSTRACT

Peptic ulcer disease (PUD) is a prevalent medical condition globally. Although there are several risk factors for the development of peptic ulcer disease, nonsteroidal anti-inflammatory medications (NSAIDs) and *Helicobacter pylori* infection are the two most significant ones. Patients typically arrive with bleeding from a stomach ulcer or dyspepsia. The three mainstays of treatment for peptic ulcer disease are the avoidance of nonsteroidal anti-inflammatory medicines, acid suppressant therapy, and *H. pylori* eradication. Life-threatening bleeding from a stomach ulcer is possible. Endoscopic hemostasis, intravenous proton pump inhibitor treatment, and adequate supportive care are used to manage it. If endoscopic therapy is unsuccessful, transarterial embolization (TAE) and surgery are rarely necessary. Stomach discomfort is typically the most prevalent sign of a peptic ulcer. Peptic ulcers are open sores that form on the inside lining of your stomach and the upper region of your small intestine. Up until recently, it was thought that eating spicy food and stress were two of the main causes of peptic ulcers. Nevertheless, it turns out that the majority of stomach and duodenal ulcers are really caused by bacterial infections and the use of specific medications rather than stress or nutrition. Conversely, stomach acid reflux, or gastroesophageal reflux disease, is the cause of esophageal ulcers when stomach acid backs up into the esophagus. Although peptic ulcers are among the most prevalent illnesses worldwide, there is good news: treatment for peptic ulcers is currently available.

1. INTRODUCTION

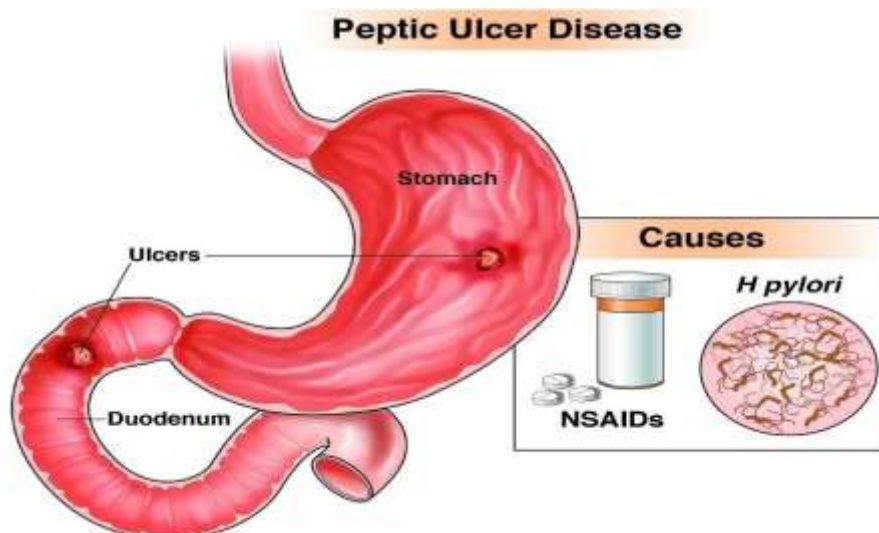


fig. no.1

The hallmark of peptic ulcer disease (PUD) is the breakdown of the gastrointestinal tract's inner lining due to either pepsin or gastric acid release. It penetrates the stomach epithelium's muscularis propria layer. Usually, it affects the proximal duodenum and stomach. The jejunum, distal duodenum, or lower esophagus could be affected. Patients often experience epigastric pain 15 to 30 minutes after eating. In contrast, the discomfort associated with a duodenal ulcer usually manifests two to three hours following a meal in cases of gastric ulcers. These days, it is advised that all patients with peptic ulcer disease undergo testing for *Helicobacter pylori*. Some individuals may need an endoscopy to confirm the diagnosis, particularly if they are exhibiting concerning symptoms. Nowadays, triple-drug therapies based on proton pump inhibitors (PPIs) can handle the majority of patients. [1] Around the world, a sizable percentage of patients seeking surgical advice have peptic ulcer disease, which encompasses both stomach and duodenal ulcers.

A lesion on the lining of the stomach or duodenum is called a peptic ulcer. The two most common types of peptic ulcers are "duodenal ulcers" and "gastric ulcers." Hydrochloric acid (HCL), pepsin, refluxed bile, leukotrienes (LTs), reactive oxygen species (ROS), and mucus-bicarbonate barrier, prostaglandins (PGs), mucosal blood flow, cell renewal

and migration, nonenzymatic and other defensive factors are forces that work together to cause peptic ulcers. *H. pylori* infection and nonsteroidal anti-inflammatory drug use are the two most frequent causes of peptic ulcer disease (NSAIDs). Additionally, a number of factors, including bacterial infections (*Helicobacter pylori*), certain medications (NSAIDs), chemicals (HCl/ethanol), and stomach cancer, are linked to the pathophysiology of gastric ulcers. Minor factors also include stress, smoking, spicy food, and inadequate nutrition. The purpose of treating ulcers is to lessen the amount of acid your stomach produces, balance that acid, and shield the injured area so it can heal. This review study's main goal was to provide an overview of the ulcerogenic Mechanisms of several mediators connected to peptic ulcer disease.[2].

A chronic illness known as peptic ulcer arises from an imbalance between the stomach mucosa's endogenous protective factors (acid and pepsin secretions) and aggressive factors (prostaglandin E2, nitric oxide, mucus and bicarbonate secretion, adequate blood flow, sulfhydryl compounds and antioxidant enzymes, and others). The genesis of stomach ulcers has also been linked to behavioral and environmental factors, including smoking, eating poorly, consuming alcohol and non-steroidal anti-inflammatory drug intake, and having an infection caused by *Helicobacter pylori*. [3].

TYPES OF ULCER

Peptic Ulcer : Stomach and duodenal ulcers are among the digestive tract ulcers that fall under the general term "peptic ulcer." Previously, it was believed that eating spicy food and stress were the causes of this type of ulcer, but more recent research has shown that these are only exacerbating factors. The causative agents, on the other hand, are the bacterium *Helicobacter pylori* or an adverse reaction to certain medications, like non-steroidal anti-inflammatory drugs. Symptoms of peptic ulcers can include vomiting, bloating, nausea, low appetite, weight loss, and black stools, which are indicators of gastrointestinal bleeding.

Aphthous Ulcers : Sores that develop on the inside of the mouth lining are called mouth ulcers. Trauma, such as damaged teeth, fillings, or loose or painful dentures, is a common cause of mouth ulcers. Anemia, measles, viral infections, oral candidiasis, chronic infections, throat cancer, mouth cancer, and vitamin B deficiency are some typical causes of mouth ulcers or sores. Aphthous minor is one of the most common types of oral ulceration disorders, affecting an estimated 15- 20% of the global population. It is most common in North America, where incidences as high as 50–66% have been documented. Research has shown that smokers were less likely than non-smokers to develop aphthous ulcers.

Chronic ulcers:

Some ulcers persist for long periods of time without healing; this occurs due to Several reasons, such as:

1. Not complying with the doctor's instructions and the specified doses of Treatment.
2. Some types of bacteria are resistant to antibiotics.
3. Continuous smoking during treatment, which slows down the healing Process of ulcers.
4. Continued use of NSAIDs.

Other rare causes of chronic ulcers include:

1. Zollinger-Ellison syndrome, which causes a steady increase in the Secretion of stomach acid due to excessive secretion of certain Hormones.
2. Other infections in the stomach or digestive system.
3. Other digestive diseases, including cancers.



Fig.no.2

2. PATHOPHYSIOLOGY OF PEPTIC ULCER DISEASE

In the past, the pathophysiology of peptic ulcer disease was primarily understood to be related to anomalies in the secretion of pepsin and gastric acid, as well as the suppression of acid as a therapeutic approach. Today, gastric hypersecretion associated with gastrinoma in Zollinger–Ellison Syndrome, antral G-cell hyperplasia, an increase in parietal-cell mass, and a physiological imbalance between the antagonistic gastric hormones Gastrin and somatostatin—is still an important issue in peptic ulcer disease. Furthermore, it is recognized that pepsin stimulation—which is frequently disregarded as a cofactor in the development of erosive injury to the stomach mucosa—and cholinergic hypersensitivity and parasympathetic dominance are associated to the stimulation of hydrochloric acid.

Peptic ulcer disease has been linked to a number of factors, including immunosuppressive drugs, alcohol consumption, smoking, psychological stress, and NSAID use, which includes aspirin, potassium chloride, oral bisphosphonates, and prostaglandin decline with age.[5]. However, the discovery that *H. pylori* is the primary cause of peptic ulcer disease and its isolation prompted researchers to investigate the function of inflammation and the cytokine cascade it triggers in the production of gastric acid. By using a number of strategies, *H. pylori* avoids the host immune system's assault and results in persistent, indolent inflammation.

Throughout all phases of the infection, *H. pylori* can harm the mucosal defense system by weakening the mucus gel layer, decreasing mucosal blood flow, and interacting with the gastric epithelium. A *H. pylori* infection can also enhance the production of gastric acid. This is because *H. pylori* causes inflammation by the production of several antigens, virulence factors, and soluble mediators. This increases the bulk of parietal cells, which in turn increases the ability to release acid. Another significant function of the *H. pylori* cytotoxin associated gene CagA is its interference with gastric epithelial cell-signaling pathways, which controls cellular responses and may aid in the formation of the apical junction. Disturbance of the barrier, release of interleukin-8, and alterations in the phenotype of stomach epithelial cells.[7].

Peptic ulcer disease pathophysiology is somewhat of a dead end, with major differences in the causes of injury between duodenal and stomach ulcers. Duodenal Ulcer is fundamentally an *H. pylori*-related condition and is caused mostly by a rise in acid and pepsin load, and gastric metaplasia in the duodenal cap.[7]. The most common cause of gastric ulcers, at least in Western countries, is the consumption of NSAIDs, however *H. pylori* infection may also be present.[8]. Even normal acid levels can be linked to mucosal ulceration in people with gastric ulcers; chronic, superficial, and atrophic gastritis prevail.[9]. Ulcers are linked to an unbalance between aggressive and defensive forces in both illnesses, and inflammation is a major contributor to this unbalance.

H pylori-positive ulcer :

The final evidence that *H. pylori* is the primary cause of ulcer disease was the elimination of the infection, which permanently cured peptic ulcers.[10,11] Over half of the world's population has a chronic *H. pylori* infection of the gastric mucosa, but only 5–10% of those infected develop ulcers. The factors that determine whether an infection will result in disease include the pattern of histological gastritis induced by the infection, changes in gastric hormone homeostasis and acid secretion, gastric metaplasia in the duodenum, *H. pylori*'s interaction with the mucosal barrier and immunopathogenesis, ulcerogenic strains, and genetic factors.

The entire gastric epithelium, from the prepyloric antrum to the cardia, is colonized by *H. pylori*. Clinical outcomes depend on the pattern of chronic mucosal inflammation induced.[12,13] In patients with duodenal ulcers, the acid-secreting body mucosa is spared, and the distal antral region has the highest infection density and severity. After *H. pylori* eradication, gastric mucosal changes are typically fully reversible. In gastric ulcers, inflammation affects both the body and the subcutaneous mucosa to a similar extent, though it varies depending on the location of the ulcer.[14] Unlike in duodenal ulcers, acid secretion in gastric ulcers can be decreased due to the more severe involvement of acid-secreting body mucosa. However, a critical amount of acid production is always conserved.

Both basal and stimulated gastric acid output are increased in antrum-predominant non-atrophic *H. pylori* gastritis. This effect is most noticeable in patients with duodenal ulcers. Patients with duodenal ulcers and *H. pylori* infection produce more acid in response to the same stimulation with gastrin than do infected people without ulcers. This finding is attributable to an impaired acid response to gastrin in infected people without ulcers, which is likely caused by the intense inflammation of their mucosal cells that secrete acid.[16] Patients with duodenal ulcers also have more parietal cells that secrete acid than do people without ulcers.

The premorbid acid-secreting status may have a major role in establishing the development pattern of *H. pylori* gastritis and, consequently, the risk of duodenal ulcer development. This suggestion is based on data showing that in patients with *Helicobacter pylori* infection, the reduction of gastric acid secretion with proton-pump inhibitor (PPI)

therapy increases the degree of inflammation of the acid-secreting mucosa.[17].Therefore, a high constitutive acid secretory capability may favor the development of body-sparing antral-predominant gastritis and consequent duodenal ulceration.

CAUSES OF NON-H. PYLORI, NON-NSAID PEPTIC ULCERS [18]

- Gastric adenocarcinoma
- Gastric lymphoma
- Local drug irritation
- Irritation at the neck of a hiatus hernia (Cameron's ulcer)
- Idiopathic
- Anastomotic ulceration after previous gastric surgery
- After radiotherapy
- Zollinger-Ellison syndrome (gastrinoma) e particularly for duodenal ulcers
- Multiple endocrine neoplasia type-I
- Hyperparathyroidism without multiple endocrine neoplasia type-I
- Systemic mastocytosis
- Severe systemic illness stress ulcers (Cushing's ulcer)
- Idiopathic eosinophilic and lymphocytic gastritis
- Duodenal Crohn's disease
- Coeliac axis stenosis
- Hepatic artery chemotherapy.

Peptic ulcer disease (PUD) has various causes; However, Helicobacter pylori-associated PUD and NSAID Associated PUD account for the majority of the disease Ethology [19].

Common

1. H. pylori infection
2. NSAIDs
3. Medications

Rare :

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

In most cases, the main causes of ulcers include:

1. The presence of a bacterium called Helicobacter pylori.
2. Smoking has been found to increase the secretion and concentration of Gastric acid, thus increasing the risk of ulcers. Smoking also slows the Healing process of sores during treatment.
3. In case the Helicobacter pylori tests come back negative, then the other Most likely factor for peptic ulcers is the use of painkillers such as NSAIDs, which the patient should stop using.
4. If the patient is proven not to use these drugs, then the cause is often Acid reflux.

Signs and symptoms : Signs and symptoms of a peptic ulcer can include one or More of the following :

1. abdominal pain, classically epigastric, strongly Correlated with mealtimes. In case of duodenal Ulcers, the pain appears about three hours after Taking a meal and wakes the person from sleep;
2. bloating and abdominal fullness;
3. waterbrash (a rush of saliva after an episode of Regurgitation to dilute the acid in esophagus, Although this is

more associated With gastroesophageal reflux disease);

4. nausea and copious vomiting;
5. loss of appetite and weight loss, in gastric ulcer;
6. weight gain, in duodenal ulcer, as the pain is Relieved by eating;
7. hematemesis (vomiting of blood); this can occur Due to bleeding directly from a gastric ulcer or from Damage to the esophagus from severe/continuing Vomiting.
8. melena (tarry, foul-smelling feces due to presence Of oxidized iron from hemoglobin);
9. Rarely, an ulcer can lead to a gastric or duodenal Perforation, which leads to acute peritonitis and Extreme, stabbing pain, requires immediate Surgery. [18].

The most common peptic ulcer symptom is burning stomach pain, and the Cause of this pain is ulceration in the mucous membranes themselves. The Contact of stomach acids with the ulcer itself is what makes the pain worse. Ulcer pain is characterized by the following:

1. It is located in the upper abdomen area just below rib cage and above The navel.
2. The pain may last between minutes to several hours.
3. The pain may get worse between meals when the stomach is empty.
4. Occasionally, patients have been known to wake up in the middle of the Night due to nagging pain.
5. The pain can often be relieved by eating certain foods that buffer Stomach acid or by taking an acid-reducing medication.
6. Symptoms usually disappear for several days to several weeks only to Reappear again after a while in a recurring cycle of pain.

Symptoms and signs of severe peptic ulcers include:

1. Vomiting blood which may appear red or black due to bleeding ulcers.
2. Dark blood in stools, or stools that are black or tarry.
3. Nausea or vomiting.
4. Loss of appetite
5. Unexplained weight loss.

Complications: Failure to treat peptic ulcers may put the patient at risk of internal bleeding and Open sores may continue to form on the lining of the stomach or small intestine Until it is punctured after which the patient is at risk of peritonitis. Peptic ulcers May also result in a blockage in the digestive tract due to the swelling of the area Affected by the ulcer, which causes a feeling of early satiety accompanied by Frequent vomiting and weight loss. PUD can cause bleeding, perforation, penetration, obstruction of the stomach outlet, persistent symptoms, and Gastric cancer (adenocarcinoma and MALT lymphoma).

15% to 20% of people have bleeding, which is the most common result. PUD is responsible for a considerable portion (about 40–60%) of acute upper GI bleeding[20]. Prolonged inflammation and scarring caused by ulcers can constrict the duodenum and potentially block the gastric exit. Additionally, it sets off a proliferative inflammatory response and an overactive immune system, both of which contribute to the development of cancer. Despite the fact that 50% of people have H. pylori infection, less than 2% of people globally will ever develop stomach cancer[21].

Diagnosis: Radiologic and/or endoscopic techniques are usually required to document the existence of ulcers. Because endoscopic testing is invasive and expensive, it is only advised for patients with new-onset dyspepsia who are 60 years of age or older. Those with dyspepsia who are younger than 60 years of age should still be checked for H. pylori using noninvasive methods and treated if found positive. If the patient tests negative for H. pylori, they should be offered an acid-suppressive medication trial or continue with endoscopy. If dyspepsia persists after an acid-suppressive medication trial, upper endoscopic assessment is required. Standard laboratory testing is not useful for diagnosing PUD.

To identify bleeding, stool guaiac testing, hemoglobin, and hematocrit testing are performed. Endoscopic and nonendoscopic diagnostic tests are available to identify the presence of H. pylori. In order to do an endoscopic diagnosis, stomach tissue samples must be extracted and then analyzed for H. pylori. H. pylori can be detected nonendoscopically by the stool antigen assay, serologic testing, and urea breath test. These diagnostics are less costly and intrusive than endoscopies. Because of its high sensitivity, specificity, and quick turnaround time, the urea breath test is typically used as the first line of testing. False-negative results may arise from concurrent use of antibiotics or acid suppressants. Confirmation of the H. pylori infection's eradication can also be achieved using the

urea breath test.

Serologic testing gives a rapid (within 15 minutes) Office-based assessment of exposure to H. pylori, but it Cannot identify active infection from previously Treated infection; patients can stay seropositive for Years after eradication. Serologic testing is advised in patients with recent Or present antibiotic or acid-suppressive medication. For an initial diagnosis or to confirm the eradication of H. pylori, stool antigen testing can be helpful. Their sensitivity and specificity are good, and concurrent medication use has less of an impact on them.

Treatment: The goal of therapy for peptic ulcer disease is to relieve Symptoms, heal craters, prevent recurrences, and Prevent complications. Medical therapy should include Treatment with drugs, and attempt to accomplish the Following:

1. Reduce gastric acidity by mechanisms that inhibit or Neutralize acid secretion,
2. Coat ulcer craters to prevent acid and pepsin from Penetrating to the ulcer base,
3. Provide a prostaglandin analogy,
4. Remove environmental factors such as NSAIDs and Smoking, and
5. Reduce emotional stress (in a subset of patients).

Treatment of H. Pylori-Associated Ulcers :

1. Uncomplicated ulcer – PPIs (such as omeprazole 20 mg twice daily) given for 14 days in conjunction with an antibiotic program to treat H. pylori are typically sufficient to induce healing in people with uncomplicated ulcers.

2. Complicated ulcer – Acid suppression treatment with an intravenous PPI should be given to all patients with complicated peptic ulcers (ulcers with bleeding, perforation, penetration, or obstruction of the gastric outlet). To improve healing, patients should be shifted to an oral PPI at a high dose twice daily once they are able to take oral medications (eg, Omeprazole 40 mg twice daily). After four weeks, the dosage should typically be lowered to once daily. However, a lower oral dose (such as 20 mg of omeprazole once daily) can be used instead of an intravenous PPI in individuals who are bleeding. After the endoscopy, 72 hours The existence of penicillin allergy and risk factors for macrolide resistance should influence the first antibiotic regimen chosen to treat Helicobacter pylori.

We recommend triple medication (a proton pump inhibitor [PPI], amoxicillin (1 g twice daily), and clarithromycin (500 mg twice daily) for 14 days (Grade 2B) as the first line of treatment for patients lacking risk factors for macrolide resistance. Only in penicillin-allergic individuals do we recommend substituting metronidazole for amoxicillin, as metronidazole resistance is prevalent and can decrease treatment efficacy. For patients who have macrolide resistance risk factors, we recommend bismuth quadruple therapy as an initial course of treatment. In quadruple treatment, Bismuth is a PPI.

NSAID-related peptic ulcer :

- Any Patients with PUD should eliminate or Reduce use of NSAIDs (including aspirin). If Possible, alternative agents such as Acetaminophen or a nonacetylated salicylate (eg, Salsalate) should be used for pain relief.
- Patients with NSAID-associated ulcers should be Treated with a PPI (eg, omeprazole 20 to 40 mg Daily) for four to eight to weeks based on the size Of the ulcer. In patients with peptic ulcers who Need to remain on NSAIDs or aspirin, Maintenance antisecretory therapy with a PPI (eg, omeprazole 20 mg daily) can reduce the risk Of ulcer complications or recurrence.

Treatments and medications:

Many peptic ulcers are caused by a Helicobacter pylori infection, therefore, Treatment is usually aimed at achieving the following two goals:

1. Eliminating the bacteria.
2. Reducing acid production in the digestive tract by relieving pain and Accelerating the healing of peptic ulcers. To achieve these two goals, several types of medications are usually used, such As:

1. Antibiotics: To treat an H. pylori infection, doctors combine many antibiotics because treating the infection with just one antibiotic is insufficient to eradicate this kind of bacteria. Additionally, for therapy to be effective, patients must take their medication as directed by their doctor. Some pharmaceutical companies may produce combinations of medications to treat the H. pylori bacteria, such as antibiotics plus proton-pump inhibitors, which lower the amount of acid in the stomach by preventing the stomach's lining cells from producing acid.

2. Acid blockers : These medications, often known as antihistamines, lessen the quantity of stomach acid that is secreted into the digestive tract, lessening ulcer discomfort and hastening healing. Prescription and over-the-counter

versions of these medications can be found in pharmacies.

3. Antacids : An antacid neutralizes stomach acid that has already been secreted and can provide quick pain relief; as such, the doctor may add it to the list of medications provided to the patient in addition to or instead of an acid blocker.

4. Proton pump inhibitors : This technique, which depends on the closure of proton pumps in gastric acid-secreting cells, lowers the quantity of gastric acid secreted. Only prescriptions are accepted for this class of medications.

Management and prevention of NSAID-Associated peptic ulcer :

In patients who continue to take NSAIDs, NSAID-associated duodenal ulcers heal after 4 weeks' treatment with a PPI, and gastric ulcers After 6–8 weeks.⁴⁸ Co- therapy with a PPI reduces The risk of developing a peptic ulcer in both acute (OR 0.70; 95% CI 0.24–2.04) and chronic (OR 0.32; 95% CI 0.15–0.67) elderly users of NSAIDs Or aspirin.⁴⁹ Thus, it is advisable to give a PPI To symptomatic, elderly patients who need an NSAID and/or aspirin. The best ulcer-preventive strategy for Patients who need to continue NSAID use Is still debated. Current strategies to reduce Ulcer complications are not considered cost Effective in patients without risk factors, but All are cost-effective in patients with a history Of ulcer bleeding.⁵⁰ Misoprostol, a mucosa Protective analog of prostaglandin E2, reduces The risk of ulcer complications, but only at the Recommended dose of 800 µg/day. Lower doses Of misoprostol are not effective.⁵¹ In a multi Center trial of 535 H. pylori-uninfected patients With a history of gastric ulcers who required Chronic NSAID treatment, misoprostol 200 µg Four times daily or lansoprazole 15mg or 30mg Once daily all significantly reduced gastric-ulcer Recurrence (4%, 7% and 0%, respectively) Compared with placebo (65%) at 12 weeks.⁵² Adverse effects were, however, higher in the Misoprostol group.

3. FUTURE DIRECTIONS

Although H. pylori infection can be successfully controlled with currently available pharmacologic approaches, there is still a serious need for novel eradication monotherapies that will simplify treatment regimens while improving eradication rates. Although molecular techniques will continue to aid in the identification of genetic factors that predict the development of idiopathic ulcers, peptic ulcer disease research has historically been dominated by H. pylori infection and the use of NSAIDs.

In the future, genetic markers linked to peptic ulcer disease may be identified due to host-related differences in the physiology of acid secretion. The discovery of an H. pylori gene that promotes the development of duodenal ulcers has led to the identification of a novel marker that can identify patients at increased risk of duodenal ulcer development and reduced risk for gastric atrophy and cancer.⁷⁴ The array of predisposing factors is, however, predominantly host-oriented; that is, based on the genetic characteristics of the patient.

Evaluation: Should the patient's first clinical presentation indicate that peptic ulcer disease is the diagnosis, they should be assessed for warning signs.

Hematemesis, melena, anemia, or heme Anorexia or weight loss signifies cancer; positive stool indicates bleeding; acute, spreading upper abdominal pain suggests perforation; persistent upper abdominal pain radiating to the back shows penetrations; vomiting suggests obstruction. Individuals who have alarm symptoms and are over 55 years old have to be referred right away for an upper endoscopy. Compared to upper gastrointestinal barium studies, esophagogastroduodenoscopy (EGD) is more sensitive and selective for peptic ulcer disease and enables gastric lesion biopsies.[22].

Individuals under 55 who do not exhibit alarm symptoms ought to have an H. Pylori infection tested, and they should be encouraged to stop NSAID use, alcohol use, smoking, and illicit drug use. A stool antigen test, endoscopic biopsy, urea breath test, or serum enzyme-linked immunosorbent assay (ELISA) can all be used to confirm the presence of H. pylori. The least reliable test is serum ELISA, which should only be used to identify the first infection. Like the urea breath test, the stool antigen test is less convenient but quite accurate in confirming the removal of H. pylori.[22]

4. CONCLUSIONS

H pylori eradication and/or antisecretory therapies are the mainstay of today's treatment strategies. In the future, it is anticipated that advances in the fields of molecular biology and genetic engineering will assist in the management of Peptic Ulcer disease.

As the prevalence of Peptic Ulcer disease increases with advancing age, it is expected that this common disease will continue to have a significant global impact on health-care delivery, health economics, and the quality of life of patients. A search for H. pylori infection, the overt or covert use of NSAIDs, and the possibility of an acid hypersecretory state are important considerations in the diagnosis of peptic ulcer and determine the therapeutic approach.

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