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A Review Article on Peptic Ulcer

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ABSTRACT
Peptic ulcer disease (PUD) refers to the development of open sores or lesions on the lining of the stomach, duodenum, or esophagus, caused by an imbalance between aggressive factors (like gastric acid) and protective factors. The two most common types of peptic ulcers are gastric ulcers, which form in the stomach, and duodenal ulcers, which occur in the first part of the small intestine. The primary etiological factors for PUD include infection with Helicobacter pylori, long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs), and excessive alcohol consumption, though stress and smoking also contribute to its pathogenesis. The clinical presentation of PUD often includes epigastric pain, bloating, nausea, and in severe cases, bleeding or perforation. Diagnosis is confirmed through endoscopy or non-invasive tests for H. pylori. Treatment strategies involve the use of proton pump inhibitors (PPIs) to reduce gastric acid secretion, antibiotics to eradicate H. pylori, and lifestyle modifications. In cases of complications such as bleeding or perforation, surgical intervention may be required. Despite significant advancements in treatment, PUD remains a common gastrointestinal disorder, and management focus on eradication of the underlying causes, symptom control, and prevention of recurrence.

KeywordsHelicobacter pylori, stomach ulcers, NSAIDs, PPIs.

I. INTRODUCTION

Acid-induced digestive tract lesions known as peptic ulcers are usually found in the stomach or proximal duodenum. They are characterised by denuded mucosa, with the defect extending into the submucosa or muscularis propria. (1) An imbalance between the endogenous protective (acid and pepsin secretions) and aggressive (mucus and bicarbonate secretion, adequate blood flow, prostaglandin E2, nitric oxide, sulfhydryl compounds and antioxidant enzymes, and others) factors of the stomach mucosa leads to chronic peptic ulcer disease. Additionally, behavioural and environmental factors such as alcohol intake, smoking, Helicobacter pylori infection, poor diet, and use of non-steroidal anti-inflammatory drugs have been associated to the aetiology of stomach ulcers.(2) An exposed lesion on the skin or mucous membrane that is defined by the exfoliation of inflammatory, dead tissue is called a blister.Ulcers are lesions on the surface of the skin or a mucous membrane that show a superficial loss of tissue. Ulcers are most commonly found on the skin of the lower limbs and in the gastrointestinal tract, although they can occur almost anywhere. There are many different types of ulcers, such as oral, esophageal, peptic, and vaginal ulcers.(3) Acid-induced lesions in the stomach and duodenum known as peptic ulcers are characterized by mucosa that has been entirely damaged, with the defect extending into the muscularis propria or submucosa.(4).(PUD) is a prevalent ailment that continues to be a major source of morbidity and mortality associated with high healthcare expenses, even with a recent drop in incidence. The two primary risk factors are Helicobacter pylori infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs). The most prevalent and often defining symptom of PUD is dyspepsia, with most patients exhibiting no symptoms at all. Potential complications may include upper gastrointestinal bleeding, perforation, or stenosis. The upper gastrointestinal endoscopy is the recommended diagnostic technique. The three main pillars of treatment are stopping the use of NSAIDs, eliminating H. pylori, and using proton pump inhibitors (PPIs). However, prevention is the best course of action, and it entails treating H. pylori and offering a legitimate rationale for PPIs.(5) Due to its purported connection to stomach acid release, gastroduodenal ulcers, often known as "peptic ulcers," are still a prevalent condition. Before Helicobacter pylori was found to be a causative factor, PUD (peptic ulcer disease) was believed to be mostly caused by stress. Since the introduction of triple therapy with antibiotics and proton pump inhibitors (PPIs), the incidence of PUD patients has decreased, while the incidence of

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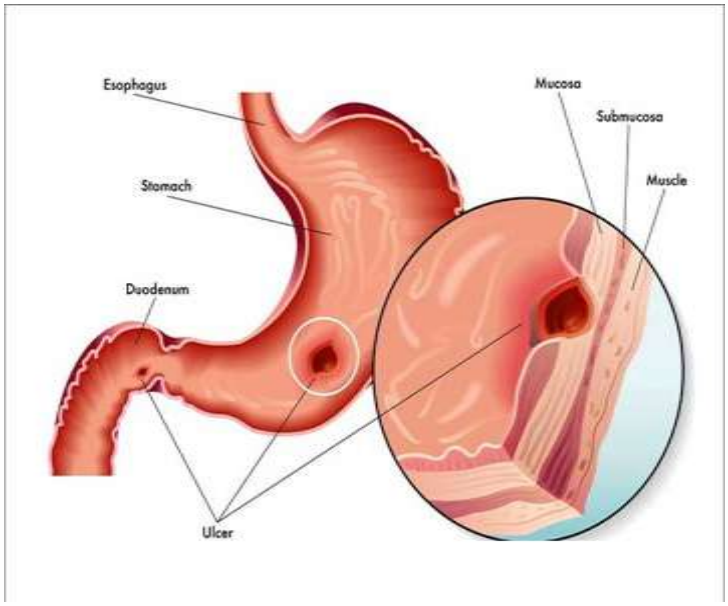
PPU patients has been constant.(6) A chronic condition that affects up to 10% of people worldwide is peptic ulcer disease. The existence of gastric juice pH and the lowering of mucosal defences are prerequisites for the development of peptic ulcers.Helicobacter pylori with non-steroidal anti-inflammatory medications (NSAIDs)infection are the two main factors influencing the mucosal resistance to injury. The disturbance of the GI tract's inner lining brought on by either pepsin or stomach acid secretion is the defining feature of Peptic Ulcer Disease (PUD). It penetrates the stomach epithelium's muscularis propria layer.(7) A skin or mucous membrane ulcer is an open sore that is characterised by the sloughing off of inflammatory, dead tissue.Ulcers are lesions that cause superficial tissue loss on the surface of the skin or a mucous membrane. Ulcers can appear almost anywhere, however they are most commonly found in the gastrointestinal tract and on the skin of the lower limbs.There are numerous varieties of ulcers, including vaginal, peptic, esophageal, and oral ulcers.(8) One of the most prevalent illnesses impacting people worldwide is ulcerative colitis.A person's health is negatively impacted by the dangerous side effects of allopathic ulcer treatment. It stops the organ of which that membrane is a component from performing its normal functions. Both inside and outside the human body, it can take many different forms.Different types of ulcers, including peptic ulcers, corneal ulcers, stomach ulcers, foot or leg ulcers, etc., are currently recognised in medicine.(9) A peptic ulcer is defined as the profound erosion of the mucosa and/or duodenum lining the stomach that extends past the muscularis mucosa and, in particular, to the muscle layer due to the production of gastric acid in the environment. The two most common etiological antecedents are the use of NSAIDs, which naturally involve the ASA, and a long-term Helicobacter pylori (Hp) infection. When considered collectively, there are unique, less common antecedents that can result in a PU and account for less than 5% of occurrences. Among these is Zollinger-Ellison syndrome (ZES), also known as gastronomy, an overactive and gastrin-secretory neuroendocrine tumour that is frequently seen in the duodenal wall or at the top of the pancreas.(10) It is asserted that ageing increases the risk of many gastroduodenal conditions, including PUD, ulcer bleeding, gastric cancer, and gastric atrophy with intestinal metaplasia.(11)



TYPES OF PEPTIC ULCER

The word "peptic ulcer" generally refers to digestive tract ulcers, such as duodenal and stomach ulcers. This type of ulcer was formerly believed to be brought on by stress and consuming spicy food. It has been shown by recent investigations that these are only the aggravating factors. The cause is either an H. pylori infection or a reaction to a particular medicine, like non-steroidal anti-inflammatory drugs. Weight loss, bloating, nausea, vomiting, and black stools are symptoms of internal bleeding in the

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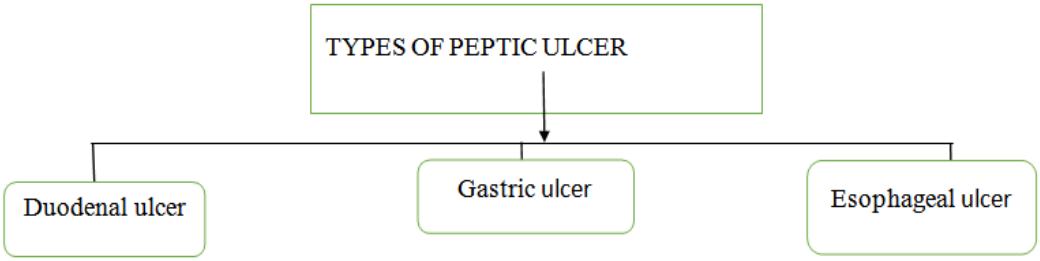
gastrointestinal system that can be caused by peptic ulcer.(12)Sores that develop on the inside lining of the mouth are called mouth ulcers. Mouth ulcers are frequent and typically result from trauma, including broken teeth, fillings, or loose, uncomfortable dentures.Anaemia, measles, viral infections, 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. It was found that compared to non-smokers, smokers had a lower incidence of aphthous ulcer.(13)



Duodenal ulcer:- Duodenal ulcers are included in the wide category of disorders known as peptic ulcer disease. The sickness condition and clinical manifestation resulting from a disruption in the mucosal membrane of the stomach or duodenum, the first part of the small intestine, are referred to as "peptic ulcer disease". Anatomically, the protective system on the surfaces of the stomach and duodenum is composed of pre-epithelial, epithelium, and subepithelial components. Ulceration is the outcome of damage to the mucosal surface that extends underneath the superficial layer. While dyspepsia is the most typical comorbidity of duodenal ulcers, there are other presenting forms as well.It can range in severity, including bleeding in the gastrointestinal tract, obstruction of the stomach outlet, puncture, or fistula formation. Consequently, the way a patient presents itself at the time of diagnosis or as the illness progresses has a big impact on the care they receive. If a patient presents with symptoms of upper abdomen discomfort or dyspepsia and reports using NSAIDs or has previously been diagnosed with Helicobacter pylori, it is worth investigating whether the patient has a duodenal or gastric ulcer. Particularly for patients with a diagnosis of duodenal ulcers, testing for H. pylori should be performed because it is a common cause of peptic ulcer disease.(14)

Gastric ulcer :- In the US, stomach ulcers are a common clinical presentation that often require millions of dollars to correct.They are an opening in the stomach lining's mucosal barrier that penetrates the muscularis mucosa and is larger than 5 mm in diameter. It's critical to understand that there is prevention and a cure for this illness process. Patients' course of treatment may vary depending on what caused their stomach ulcer. The body's inherent defences protect the stomach mucosa from the potentially harmful acidic environment of the gastric lumen. Variations in these defences could lead to alterations in the mucosa of the stomach, which could eventually cause erosion and ulcers. The stomach mucosa is shielded by prostaglandins, mucus, growth hormones, and an adequate blood supply.(15)

Esophagealulcer:- A distinct rupture in the esophageal mucosal membrane is known as an esophageal ulcer. This damage to the oesophagus mucosa is often caused by gastro-oesophageal reflux disease or severe, chronic oesophagitis from other sources. According to projections, esophageal ulcers will arise in 2% to 7% of cases of gastro-oesophageal reflux disease.The most prevalent cause of esophageal ulcers is gastro-oesophageal reflux disease, and most patients have some degree of hiatal hernia as revealed by endoscopic testing.Normally, the lower esophageal sphincter (LES) stops stomach contents from refluxing back up into the throat. However, when the LES weakens, this protective mechanism is weakened, which increases the risk of ulceration by exposing the esophageal mucosa to stomach acid. Additionally, as evidenced by bulimia nervosa sufferers, repeatedly causing vomiting exposes the stomach contents to the esophageal mucosa, which may cause an ulcer to form or worsen an existing ulcer.(16)

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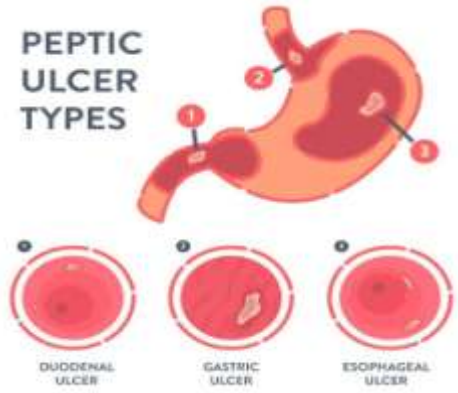

PATHOPHYSIOLOGY

Under normal circumstances, the mucus-bicarbonate barrier, neutral pH, and ongoing epithelial cell regeneration preserve the integrity of the duodenum and stomach mucosa. (17) PGE2 stimulates mucus production, cell division, and H3CO3 release, supporting a crucial function in mucosa preservation. An important distinguishing characteristic of gastric homeostasis is adequate blood flow. The stomach mucosa's proper perfusion is maintained by the NO and PGs, who also ensure that nutrients and oxygen are delivered to the area and that harmful metabolites are removed, preventing tissue damage. (18)

Helicobacter pylori:- The duodenal rather than the stomach side is better equipped with knowledge of the mechanisms by which the HP promotes the advancement of PU**.**(19)H. pylori causes epithelial cell deterioration and destruction as well as an inflammatory response including neutrophils, lymphocytes, plasma cells, and macrophages inside the mucosal layer Gastritis often worsens in the antrum in cases where the corpus is not inflamed. Testing for H. pylori should be performed on all patients who have developed peptic ulcers.(20) The type of peptic ulcer developed can be determined by sequencing the inflammation associated with H.pylori infection to either hypo- or hyperchlorhydria.(21, 22)

NSAIDs induced ulcer: There are two primary ways that NSAIDs damage the mucosa of the stomach and duodenum. On the one hand, these drugs act like weak, non-ionized acids that easily enter the mucous layer and the epithelial cells. One unique and absolutely necessary outcome is the cyclooxygenase inhibitory enzyme's capacity. Due to their intramucosal vasodilator effect, which preserves blood flow, they are important in preserving the integrity of the gastroduodenal mucosa function. They also stimulate local synthesis of mucus and H3CO3, which promotes cell turnover and epithelization.(23, 24) NSAIDs are extensively used for a number of scenarios to support to reducediscomfort and inflammation, however different consumers experience GI side effects over time.Over 90% of ulcers are caused by NSAIDs, and 25% of NSAID users will develop PUD**.** (25) Additionally, peptic ulcers are twice as likely to progress among aspirin users than in the general population. (26)

Stress and diet: Stress brought on by significant health issues, such as those requiring treatment in an intensive care unit, is widely recognised as a precursor to peptic ulcers, often known as stress ulcers **.**(27) Caffeine and coffee are widely believed to cause or worsen pain, yet they appear to have less negative effects.(28) Missing meals allows stomach acid to directly affect the lining of the stomach, creating irritation that ultimately leads to gastric ulcers. Abdominal pain is preceded by gastric ulcers and becomes worse with meals.(29)

Smoking and alcohol: Alcohol use and smoking are risk factors**.**Prolonged alcohol use disrupts stomach mucosal barriers by inhibiting COX 1 receptor enzymes, which reduce the release of prostaglandins that are cytoprotective. Smoking cigarettes causes the amount of circulating epidermal growth factor to decrease and increases the release of free radicals in the stomach mucosa**.** (30)

CAUSES OF NON-H. PYLORI, NON-NSAID PEPTIC ULCERS

- Gastric adenocarcinoma
- Gastric lymphoma
- Local drug irritation
- Irritation of a hiatus hernia (Cameron's ulcer) near the neck
- Idiopathic
- Anastomotic ulceration after previous gastric surgery
- After radiotherapy
- Multiple endocrine neoplasia
- Hyperparathyroidism without multiple endocrine neoplasia type-I
- Systemic mastcytosis
- Severe systemic illness stress ulcers
- Idiopathic eosinophilic and lymphocytic gastritis
- Duodenal Crohn’s disease
- Coeliac axis stenosis
- Hepatic artery chemotherapy (31)

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DIAGNOSIS OF PEPTIC ULCER DISEASE

Epigastric discomfort, postprandial and nocturnal pain, pain that can rouse a patient from sleep, and pain that is alleviated by food or antacids are common symptoms of peptic ulcer disease. Less frequent symptoms include anaemia from gastrointestinal blood loss, weight loss from decreased hunger brought on by pain tolerance, and vomiting linked to pyloric stenosis or a stomach ulcer.(32)But an ulcer is not defined by pain, and a diagnosis cannot be made if there is no discomfort, particularly in the elderly who may exhibit "silent" ulcer problems. Although there isn't a single symptom that distinguishes H. pylori-associated from NSAID-associated ulcers, a thorough medical history can reveal who is using NSAIDs without authorisation, and the right The H. pylori test can detect the infection.(33) For the precise diagnosis and differential diagnosis of peptic ulcer disease and ulcer complications, endoscopy is necessary (for example, a stomach ulcer can be biopsied to rule out cancer or to acquire tissue for an H. pylori diagnostic test). In clinical studies, endoscopic healing is the gold standard for assessing ulcer healing. Many people with dyspepsia symptoms may be evaluated endoscopically instead of being tested for and treated for an H. pylori infection in primary care in clinical practice. This method has been recommended by guidelines for young individuals with dyspepsia who do not exhibit warning signs.(34) Since in this patient population, The "test and treat" method for H. pylori is less expensive than endoscopy.Therefore, a tiny percentage of patients with dyspepsia will have their ulcer healed without receiving a formal diagnosis.(35)

Testing for H. Pylori Infection

Therearemany methods available now to check for H. pylori infection.Endoscopy is necessary for certain procedures such as the rapid urease test (RUT), the polymerase chain reaction (PCR) of stomach biopsy materials, and the culture and histologic inspection. Although PCR is highly sensitive, false-positive findings can occasionally be obtained.(36) The urea breath test (UBT), serology, or an Non-endoscopic testing include the H. pylori stool antigen test.The population's prevalence of H. pylori infection also affects the first test selection for H. pylori detection because the positive and negative predictive values of a single test differ based on the infection's prevalence.(37)

Tests can identify prior or current infections. The UBT is the most effective test for identifying a current infection.30 In actuality, though, a repeat test (RUT) may be conducted following an endoscopy on a patient with peptic ulcer disease if the first test yields a negative result.(38) To choose which H. pylori test is suitable for a given clinical environment, various factors (such as methodologic, technical, and therapy impact) must be taken into account for each test. For instance, anti-H. pylori antibody-based serology assays are unable to detect active infection.30 However, PPI or antibiotic treatment affects both the RUT and the UBT because they block urease activity, which directly affects test sensitivity.(39)

Confirming the eradication of infection should happen 4–8 weeks after therapy ends; non-invasive testing with the UBT is the recommended method.(40) Should the ulcer return following eradication therapy, a more meticulous investigation for reinfection or eradication failure needs to be conducted by determining whether active infection is present (e.g., through histologic inspection and culture in conjunction with an antibiotic-sensitivity test).(41) The decreased sensitivity of routine invasive tests limits the ability to diagnose H. pylori infection in patients with bleeding peptic ulcers; typically, endoscopy should be used to do both RUT and histologic testing, which should then be paired with the UBT test. Any test result that is positive should be interpreted as evidence of infection, although both the invasive tests and the breath test should come back negative.(42)

MANAGEMENT OF PEPTIC ULCER DISEASE

Patients with Helicobacter pylori infection should have this infection completely eradicated as part of their PUD treatment.(43)

Relief of ulcer pain, healing of the ulcer, prevention of ulcer recurrence, mitigation of ulcer-related problems, and elimination of H. pylori in H. pylori positive patients are the objectives of therapy.(44)

NON PHARMACOLOGIC TREATMENT:- Reduce or eliminate psychological stress; cut back on nonselective NSAIDs (including aspirin); use COX-2 selective inhibitors or acetaminophen as an alternative to aspirin; give up smoking; limit foods and beverages that cause dyspepsia or aggravate ulcer symptoms (such as alcohol, caffeine, spicy

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foods, and spicy foods); and consider emergency surgery for certain patients who have bleeding, perforation, or obstruction.(45)

PHARMACOLOGICAL TREATMENT :-The most effective classes of drugs were those that prevented the generation of stomach acid. H2-receptor antagonists transformed the treatment of peptic ulcers by curing and maintaining remission when used as a maintenance therapy.(46-47) The more potent class of acid-inhibitory drugs known as proton-pump inhibitors (PPIs), which went into effect in 1989, progressively replaced them. PPIs are specifically inhibiting the parietal cell's H+ K+ ATPase.(48) PPIs became the standard in ulcer treatment due to the fact that the rate at which ulcers heal is correlated with the degree of acid suppression. A second class of drugs aims to strengthen the mucosal stumbling block, which is why it is mostly used to defend against aspirin and NSAIDs. The most widely used prostaglandin analogue is misoprostol, although its usage is restricted by adverse effects in the abdomen, especially at larger dosages.(49) Bismuth salts and sucralfate both improve mucosal repair, which aids in ulcer healing. Sucralfate may also function in part by reducing the formation of acid and preventing H pylori infection.(50)Only in conjunction with antibiotics are bismuth salts that have some intrinsic anti-H pylori activity utilised in ulcer treatment. More potent medicines have rendered cytoprotective drugs obsolete. Current ulcer therapy includes PPI for mending and preventing peptic ulcers brought on by gastrointestinal drugs, as well as Helicobacter pylori eradication for H pylori-positive peptic ulcers. There is a tiny benefit to drugs that speed up mucosal resistance. (51)

CLINICAL MANIFESTATIONS AND DIAGNOSIS :-

Epigastric pain is the main symptom of simple peptic ulcer, and it may also be accompanied by other dyspeptic symptoms as bloating, fullness, nausea, and early satiety. Epigastric pain in patients with duodenal ulcers normally goes away when they eat or take acid-neutralizing medications. It commonly happens during the fasting state or even at night. Most of these individuals do not have erosive oesophagitis, and about one-third of them also have heartburn.(52)Asymptomatic chronic ulcers are possible.(53) This lack of symptoms is especially observed in ulcers caused by NSAIDs, when the first clinical sign of the illness may be upper gastrointestinal bleeding or perforation. Bleeding is the most common and serious complication of peptic ulcers, occurring in 50–170 cases per 100,000 cases, and is most common in adults over 60.(54)A perforation occurs approximately seven to ten times per 100,000, which is less common than bleeding.(55) Although it is fortunately uncommon, penetration of the retroperitoneal organs is defined by persistently excruciating pain.(56)Although it is uncommon, gastric outlet blockage brought on by ulcer-induced fibrosis may raise concerns about an underlying malignant illness.(57)A mucosal break covering five millimetres or more in diameter during an endoscopy is indicative of a peptic ulcer; a break less than five millimetres is referred to as an erosion. Although it is arbitrary, clinical trials employ the 5 mm criteria. It's uncertain how closely this criterion links to the pathological criterion of muscularis mucosa invasion. One or more peptic ulcers possible. The bulb, where the stomach's contents enter the small intestine, is where duodenal ulcers usually occur. Gastric ulcers can arise anywhere from the pylorus to the cardia, but they are most likely to form near the angulus of the lesser curvature.(58)On occasion, kissing ulcers are observed on the anterior and posterior walls of the duodenal bulb, facing each other.If ulceration is seen in the more distal duodenum, consideration should be given to underlying Crohn's disease, ischaemia, or the rare Zollinger-Ellison syndrome.Following an endoscopic diagnosis of peptic ulcer, biopsy samples from the antral, body, or fundus mucosa should be obtained in order to perform histological and fast urease tests to detect H pylori infection.(59)In many developed nations, patients up to 55 years old with ulcer-like symptoms are typically not evaluated with an endoscopic examination instead they are tested non-invasively for H pylori (using the 13C-urea breath test [UBT] and stool antigen test), and if found to be positive, they are treated with H pylori eradication.(60)This test-and-treat approach is justified by the observation that symptoms in some individuals will be caused by an underlying ulcer illness that can be healed with H pylori therapy. Furthermore, cancerous illness is uncommon in young individuals and in the absence of warning signs including nausea, vomiting, anaemia, and weight loss.(61)

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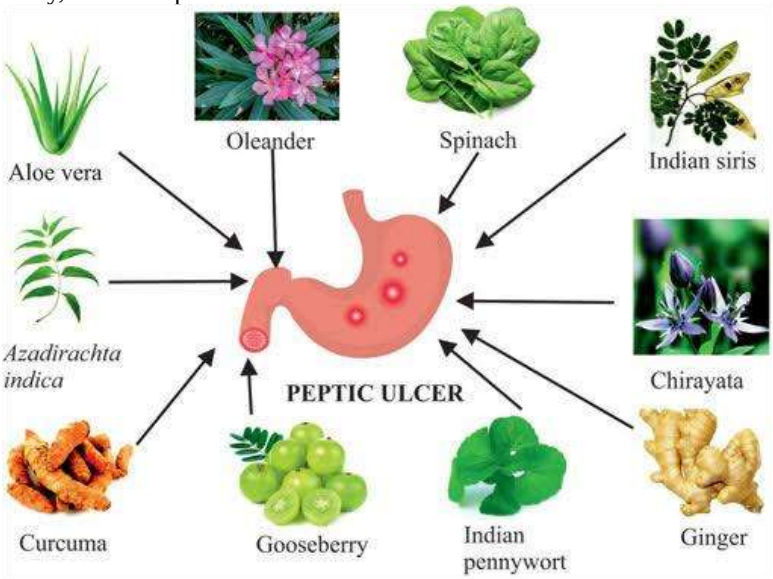
ALTERNATIVE THERAPY OF PEPTIC ULCER

Using medicinal plants to treat a range of ailments is known as phytotherapy, and it has existed for as long as humans.Additionally, interest in complementary therapies and the use of herbal products—particularly those made from medicinal plants—has grown in recent years.(62-63) Additionally, medicinal plants are thought to be the main source of possibly novel medications due to the emergence of different adverse effects from the use of traditional drugs for a wide range of disorders. The most important source of novel medications is plant extracts and their formulations, which have demonstrated encouraging benefits in the treatment of gastric ulcers as well.(64)Proton pump inhibitors, anticholinergics, antacids, antimicrobials, sucralfate, bismuth, and other pharmaceuticals are known to be ineffective in many cases and to have a variety of side effects, including gynaecomastia, impotence, arrhythmia, haematopoietic changes, and hypersensitivity.(65)As a result, studies into novel pharmacologically active compounds using various plant extract screening techniques produced safe, efficient medications with gastroprotective properties. In particular, plants that function primarily as antioxidants are utilised as a natural remedy for ulcer illness.(66)

The ability of medicinal plants to generate diverse and renewable secondary metabolites, sometimes referred to as phytochemical components, is what gives them their medicinal qualities. As a result, many plants have employed these phytochemicals as a defence strategy against infections.(67)Conversely, pharmaceutical companies have changed their approach to developing traditional antibiotics and created new antimicrobial medications derived from medicinal plants as a result of the emergence of resistant infections. However, as antimicrobial medications, synthetic antibiotics continue to be the most used.(68)In reality, during the past three decades, there has been an increase in the occurrence of infectious diseases, including new and distinct types of infections. Research has indicated that approximately 60% of these infections are zoonotic, meaning they can transmit to both humans and animals. Among the most common members of the group is H. pylori, which can lead to stomach cancer, peptic ulcer disease, and chronic gastritis.(69)Thus, highlighting certain medicinal plants with notable antioxidant and antibacterial action against H. pylori and peptic ulcer illness was one of the review's objectives. However, due to the rise of resistant strains, some plants become less effective against H. pylori. As a result, it is recommended to separate different components from the most potent plant extracts.(70)

It is crucial to stress that herbal products could have a variety of bioactive ingredients with both harmful and advantageous effects. Thus, further research on herbal therapy is required, as is legislation governing the quality of herbal products, particularly in light of the need for randomised studies to ascertain the efficacy and safety of several items in the treatment of digestive and other problems.(71)

Lastly, the combined knowledge of Ayurveda and modern medicine may lead to have fewer adverse effects.(72)



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TREATMENT

The increasing incidence of antibiotic resistance has made the treatment of related peptic ulcers and the avoidance of relapses more difficult, even if effective H. pylori eradication is essential.The first successful treatment was developed in the 1980s and involved two weeks of administration of a bismuth, tetracycline, and metronidazole combination.(73) A proton pump inhibitor (PPI) and two antibiotics, such as clarithromycin plus amoxicillin or metronidazole, administered for seven to fourteen days, constitute the conventional first-line therapy.(74) However, over the past ten to fifteen years, there has been a noticeable drop in the effectiveness of triple therapy due to the rising frequency of antibiotic resistance, particularly for clarithromycin. Tests for antibiotic susceptibility should be the foundation for eliminating H. pylori. Since susceptibility testing is frequently unavailable in clinical settings, the local prevalence of antibiotic resistance should be taken into consideration when selecting first-line treatments. In areas where the local rate of clarithromycin resistance is greater than 15%, clarithromycin-based regimens should be dis-continued.(75)By increasing the period to 14 days and using high-dose PPI, the rate of eradication can be accelerated.(76)

Either a 14-day concomitant therapy for patients intolerant to bismuth(PPI, clarithromycin, amoxicillin, and metronidazole)orabismuth-containing quadruple therapy (PPI, a bismuth salt, tetracycline, and metronidazole) is the recommended standard first-line therapy; both regimens yield eradication rates higher than 90%.(77)If a first-line treatment doesn't work, second-line therapy is recommended; metronidazole and clarithromycin shouldn't be part of it.(78)

With eradication rates ranging from 74% to 81%, levofloxacin triple therapy (PPI, amoxicillin, and levofloxacin) for 14 days appears to be an effective treatment.(79)Since H. pylori seldom develops amoxicillin resistance, a bismuth quadruple therapy with eradication rates of 77–93% or a high-dose dual-therapy regimen with amoxicillin and a PPI are recommended therapeutic options if a patient had first-line treatment with a clarithromycin-based regimen.(80)Five to ten percent of individuals still have infections after following established guidelines for selecting appropriate treatment plans. Suboptimal compliance or H. pylori's resistance to one or more antibiotics are the most frequent causes of treatment failure after two, in which case susceptibility testing is highly advised. One of the frequently suggested salvage regimens, with 66–70% eradication rates, is rifabutin-based triple therapy (PPI, rifabutin, and amoxicillin) for 10 days when at least three suggested approaches have failed.(81) But it's important to consider rifabutin's side effects, like myelotoxicity and crimson discharges.(82)

II. CONCLUSION

This article discusses the pathophysiology, clinical signs, and treatment options of peptic ulcers while also reviewing the data supporting a connection between long-term NSAID use and the condition. With this knowledge, doctors are better equipped to weigh the advantages and disadvantages of NSAIDs and provide substitutes with less adverse effects. For individuals without risk factors, toxicity can be prevented by prescribing the NSAIDs at the lowest effective dosage for the shortest amount of time. In conclusion, the largest risk factors for gastric ulcers are smoking, anticoagulant usage, drunkenness, a history of stomach ulcers, H. pylori infection, renal failure, etc. When compared to NSAID-induced toxicity, PPIs, misoprostol, sucralfate, and a variety of selective COX-2 inhibitors can be utilised as an alternative with more manageable side effects.

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