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Natural Products in the Prevention and Management of Gastric Ulcers

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Abstract

Gastric ulcer Many of the most common diseases are prevalent in gastrointestinal diseases, peptic ulcer is a chronic inflammatory condition defined by ulceration in the upper gastrointestinal tract regions where parietal cells are located and secrete hydrochloric acid and pepsin. The stomach and duodenum are the two anatomical areas where ulcers often form, resulting in ulceration of the stomach and duodenum, respectively. Ulcer physiopathology is caused by a conflict between aggressive factors like acid, pepsin, *Helicobacter pylori*, and non-steroidal anti-inflammatory drugs, as well as locally mucosal protection factors, including mucus bicarbonate, blood flow, and prostaglandins. To prevent or cure gastro-duodenal ulcers, medications are commonly utilized. H2-receptor antagonists, proton pump inhibitors, and cytoprotectants are among them. Because of the complications connected with recurrence after therapy, there is a need to look for other options.

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1. Introduction

Gastric ulcer is one of the most common gastrointestinal disorders, affecting approximately 5–10% of the global population. It is characterized by damage to the protective mucosal lining of the stomach, leading to inflammation, erosion, and ulcer formation. Peptic ulcers develop when the balance between aggressive factors, such as gastric acid and pepsin, and the protective mechanisms of the gastric mucosa is disrupted. Common risk factors include *Helicobacter pylori* infection, tobacco smoking, excessive alcohol consumption, psychological stress, genetic predisposition, and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin and ibuprofen [1].

Several conventional therapies have been developed for the management of gastric ulcers. These include histamine H2-receptor antagonists, proton pump inhibitors, cytoprotective agents, antacids, and prostaglandin analogs. Although these medications are generally effective, their long-term use may be

associated with adverse effects, high treatment costs, drug interactions, and recurrence of ulcers after discontinuation [2].

Medicinal plants have been used for centuries as traditional remedies for gastrointestinal disorders, including peptic ulcers. Herbal medicines are widely accepted because of their natural origin, cultural significance, relatively low toxicity, and reduced incidence of adverse effects compared with synthetic drugs. Numerous medicinal plants have demonstrated significant gastroprotective and antiulcer activities in experimental and clinical studies [3].

Plants reported to possess antiulcer potential include *Allophylus serratus*, *Ocimum sanctum*, *Cissus quadrangularis*, *Butea frondosa*, *Moringa oleifera*, *Zingiber officinale*, *Glycyrrhiza glabra*, *Solanum nigrum*, *Terminalia chebula*, *Cynodon dactylon*, *Azadirachta indica*, *Swietenia mahagoni*, *Bauhinia*

purpurea, *Ficus religiosa*, *Melastoma malabathricum*, *Spondias mombin*, *Eruca sativa*, and *Osyris quadripartita*. These plants have exhibited antiulcer activity in various experimental ulcer models through mechanisms such as antioxidant activity, enhancement of mucosal defense, reduction of gastric acid secretion, anti-inflammatory effects, and promotion of ulcer healing [4].

In Ghana, herbal formulations containing combinations of medicinal plants are routinely used for the management of peptic ulcer disease. Traditional herbal therapies, including Enterica, Dyspepsia decoctions, and Natural Pain Killer (NPK 500), are employed in clinical practice. Several Ghanaian medicinal plants, including *Carapa procera*, *Trichilia monadelpha*, *Persea americana*, *Trema orientalis*, *Momordica charantia*, *Vernonia amygdalina*, *Cassia sieberiana*, *Citrus aurantifolia*, *Bidens pilosa*, *Morinda lucida*, *Maytenus senegalensis*, *Psidium guajava*, *Cnestis ferruginea*, *Chromolaena odorata*, *Blighia sapida*, *Cyperus rotundus*, *Mangifera indica*, *Alchornea cordifolia*, *Calotropis procera*, *Hoslundia opposita*, *Kigelia africana*, *Spathodea campanulata*, *Strophanthus hispidus*, *Zingiber officinale*, and *Paullinia pinnata* have also demonstrated promising antiulcer properties. Their widespread availability, traditional use, and pharmacological efficacy make them valuable candidates for the development of safer and more effective antiulcer therapies [5].

1.1 Etiology

Gastric cancer is a complex and heterogeneous malignancy with multifactorial etiologies involving both genetic and environmental factors. The development of gastric cancer is influenced by several risk factors, including *Helicobacter pylori* infection, inherited genetic susceptibility, dietary habits, and lifestyle-related factors. Among these, *H. pylori* infection is considered one of the strongest risk factors and plays a significant role in gastric carcinogenesis [6].

Although hereditary gastric cancer accounts for only a small proportion of all cases, genetic alterations are frequently observed in sporadic gastric cancers. Individuals with a family history of gastric cancer are at increased risk, particularly when two or more first- or second-degree relatives are affected, or when at least one affected family member is diagnosed before the age of 50 years. Genetic susceptibility is believed to influence inflammatory and immune responses to *H. pylori* infection, thereby increasing the likelihood of malignant transformation. However, only a limited number of high-penetrance genes associated with

gastric cancer have been identified [7].

Among the genetic factors implicated in gastric cancer, polymorphisms in the interleukin-1 (IL-1) gene cluster and the interleukin-1 receptor antagonist (IL-1RN) gene have been associated with an increased risk of disease. IL-1 plays a crucial role in initiating and amplifying inflammatory responses, and genetic variations in these genes may enhance chronic gastric inflammation, promoting carcinogenesis. Genome-wide association studies have further identified several susceptibility loci associated with gastric cancer risk, including polymorphisms in the MUC1 (mucin 1), PSCA (prostate stem cell antigen), and PLCE1 (phospholipase C epsilon 1) genes [8].

These genetic variants have shown consistent associations with specific subtypes of gastric cancer, particularly in Chinese, Korean, and Japanese populations. Although these findings have improved the understanding of genetic susceptibility to gastric cancer, the precise molecular mechanisms through which these polymorphisms contribute to disease development remain incompletely understood [9].

1.2 Epidemiology

Peptic ulcer disease, including gastric ulcers, has a lifetime prevalence of approximately 5–10% of the population, although this may be underestimated because many individuals remain asymptomatic. The prevalence of gastric ulcers increases with advancing age and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs). Smoking is another significant risk factor, with smokers having nearly twice the risk of developing gastric ulcers compared to non-smokers. The incidence of gastric ulcers is generally similar in both men and women. In the United States, approximately half of the population is infected with *Helicobacter pylori* by the age of 60 years. Additionally, it is estimated that nearly 25% of individuals receiving long-term NSAID therapy develop gastric ulcers, highlighting the importance of appropriate preventive strategies and careful monitoring in high-risk patients [10].

2. Ulcer in the Stomach

2.1 Signs and Symptoms

The clinical manifestations of gastric ulcers vary depending on the severity and duration of the disease. The most common symptom is epigastric abdominal pain, which is typically described as a burning or gnawing sensation and may worsen or improve after meals. Many patients also experience abdominal bloating and a persistent feeling of fullness, even after consuming small amounts of food. Nausea and occasional vomiting are frequently

reported, particularly in individuals with more advanced disease. Changes in body weight, including either unintended weight loss or weight gain due to altered eating habits, may also occur. In severe cases, gastric ulcers can lead to upper gastrointestinal bleeding, presenting as hematemesis (vomiting of blood), which requires immediate medical attention [11].

2.2 Gastric Ulcer Diagnosis

The diagnosis of gastric ulcer begins with a detailed medical history and physical examination, particularly in patients who are at increased risk of ulcer complications such as perforation, bleeding, or gastric malignancy. Although clinical assessment and evaluation of risk factors provide important information, symptoms alone cannot reliably distinguish organic gastric disease from functional dyspepsia. Therefore, further diagnostic investigations are often required to establish an accurate diagnosis. For patients presenting with dyspepsia without alarm features, a "test-and-treat" strategy for *Helicobacter pylori* infection is recommended. Testing for *H. pylori* is particularly indicated in individuals with active or previous peptic ulcer disease, dyspeptic symptoms, or gastric mucosa-associated lymphoid tissue (MALT) lymphoma. Identification and eradication of *H. pylori* infection significantly reduce ulcer recurrence and improve long-term clinical outcomes [12].

Patients younger than 55 years of age who present with dyspepsia but do not exhibit alarm symptoms—including unexplained weight loss, progressive dysphagia, persistent vomiting, gastrointestinal bleeding, iron-deficiency anemia, abdominal mass, jaundice, or a family history of gastric cancer—are generally managed using the non-invasive *H. pylori* test-and-treat approach. In contrast, patients aged 55 years or older or those presenting with alarm features should undergo upper gastrointestinal endoscopy to exclude gastric ulcer complications or malignancy. Several diagnostic methods, including the urea breath test, stool antigen test, rapid urease test, histological examination, and bacterial culture, are available for detecting *H. pylori* infection, each with high diagnostic accuracy when used appropriately [13].

Clinical suspicion of gastric ulcer is raised in patients presenting with epigastric pain, burning sensation, abdominal discomfort, early satiety, postprandial fullness, nausea, or vomiting. Gastric ulcer pain typically occurs shortly after meals and may be associated with loss of appetite and unintended weight loss. In contrast, duodenal ulcer pain commonly develops several hours after eating or

during the night and is often relieved by food intake. Elderly patients may present with few or no symptoms, making diagnosis more challenging. Patients with chronic or uncontrolled peptic ulcer disease frequently experience recurrent episodes of symptom exacerbation and remission, largely due to persistent risk factors such as continued nonsteroidal anti-inflammatory drug (NSAID) use or persistent *H. pylori* infection. In patients with typical ulcer symptoms but without alarm features, empirical acid-suppressive therapy may be initiated while further evaluation is performed. However, persistent symptoms, treatment failure, recurrent ulcers, or suspicion of complications warrant endoscopic evaluation to confirm the diagnosis, assess ulcer healing, and exclude malignancy [14].

2.3.1 Urea Breath Test

The urea breath test is one of the most accurate non-invasive methods for detecting active *Helicobacter pylori* infection. The test involves the oral administration of urea labeled with either carbon-13 (^{13}C) or carbon-14 (^{14}C). If *H. pylori* is present, the bacterial urease enzyme hydrolyzes the labeled urea, releasing labeled carbon dioxide that is subsequently detected in the patient's exhaled breath. The test demonstrates excellent sensitivity and specificity, approaching 100% under optimal conditions. It is also widely used to confirm successful eradication of *H. pylori* and is generally performed 4–6 weeks after completion of eradication therapy. To ensure accurate results, proton pump inhibitors (PPIs) should be discontinued for at least two weeks before testing. The principal limitations of the urea breath test are its relatively higher cost and the need for specialized equipment [15].

2.3.2 Stool Antigen Test

The stool antigen test is another reliable non-invasive diagnostic method for detecting active *H. pylori* infection. Laboratory-based monoclonal antibody assays provide diagnostic accuracy comparable to the urea breath test while being less expensive and requiring simpler laboratory facilities. Like the urea breath test, stool antigen testing detects only active infection and is suitable for confirming eradication following treatment. Patients are advised to discontinue PPIs for at least two weeks before testing to improve diagnostic accuracy, although stool antigen tests are generally less affected by PPI therapy than urea breath tests [16].

2.3.3 Serological Tests

Serological testing detects *H. pylori*-specific immunoglobulin G (IgG) antibodies in the patient's blood. Although these tests are simple and widely available, they cannot differentiate between active

infection and previous exposure because antibodies may remain detectable long after eradication. Consequently, serological testing is not recommended for confirming treatment success. However, it may be useful in epidemiological studies and in patients who cannot discontinue proton pump inhibitors or antibiotics, such as those with acute gastrointestinal bleeding or individuals requiring continuous NSAID therapy [17].

2.3.4 Endoscopy and Biopsy

Upper gastrointestinal endoscopy with biopsy is recommended for patients aged 55 years or older and for individuals presenting with alarm features such as gastrointestinal bleeding, unexplained weight loss, persistent vomiting, dysphagia, iron-deficiency anemia, or suspected gastric malignancy. Endoscopy allows direct visualization of the gastric mucosa, assessment of ulcer characteristics, tissue sampling, and exclusion of gastric cancer [18].

Biopsy specimens obtained during endoscopy can be analyzed using the rapid urease test, histopathological examination, bacterial culture, or molecular techniques such as polymerase chain reaction (PCR). The rapid urease test is a rapid, accurate, and cost-effective method for detecting *H. pylori* infection, provided the patient has not taken PPIs within the previous 1–2 weeks or antibiotics and bismuth compounds within the previous four weeks. In patients who have recently received these medications, histopathological examination is generally preferred because drug therapy may reduce bacterial load and decrease the sensitivity of urease-based tests. Although bacterial culture and PCR permit antimicrobial susceptibility testing and molecular characterization of *H. pylori*, their use is largely limited to specialized laboratories and research settings [19].

3. Gastric Ulcer Mechanism and Management

The management of gastric ulcers is primarily aimed at promoting ulcer healing by reducing gastric acid secretion, increasing gastric pH, and restoring the integrity of the gastric mucosal barrier. Proton pump inhibitors (PPIs), such as pantoprazole, are the cornerstone of therapy because they effectively suppress gastric acid secretion, creating a favorable environment for mucosal repair and ulcer healing. Patients presenting with alarm features should undergo upper gastrointestinal endoscopy (esophagogastroduodenoscopy, EGD) to determine the underlying cause of the ulcer and to exclude malignancy. Alarm symptoms include unintentional weight loss, gastrointestinal bleeding, persistent nausea or vomiting, dysphagia, anemia, and age above 50 years. During endoscopy, biopsy specimens

should be obtained from the ulcer margin and surrounding gastric mucosa to evaluate for *Helicobacter pylori* infection, gastritis, dysplasia, or gastric cancer [20].

Patients diagnosed with gastric ulcers are generally treated with proton pump inhibitors for approximately 8 weeks, after which repeat endoscopy may be performed to confirm ulcer healing, particularly in cases of large, complicated, or suspicious ulcers. If the ulcer is associated with the use of nonsteroidal anti-inflammatory drugs (NSAIDs), these medications should be discontinued whenever possible to prevent further mucosal injury. When *H. pylori* infection is identified, appropriate eradication therapy consisting of proton pump inhibitors combined with antibiotics should be initiated, and successful eradication should be confirmed using validated non-invasive tests after completion of treatment. In patients with bleeding gastric ulcers, endoscopic hemostatic therapy is the preferred first-line treatment. Several endoscopic techniques are available, including epinephrine injection, thermal coagulation, and the placement of metallic or absorbable clips to achieve hemostasis. The choice of therapy depends on the severity of bleeding and the Forrest classification of the ulcer [21].

Surgical intervention is reserved for patients in whom endoscopic therapy is unsuccessful or contraindicated. Surgery may also be indicated in cases of perforated ulcers, uncontrolled or recurrent bleeding, gastric outlet obstruction, or ulcers that fail to heal despite adequate medical treatment. Timely surgical management helps prevent life-threatening complications and improves overall patient outcomes [22].

4. Gastric Ulcer Natural Herbs

Medicinal herbs have long been used in traditional systems of medicine for the prevention and treatment of gastric ulcers. These herbs promote ulcer healing through multiple mechanisms, including protection of the gastric mucosa, reduction of gastric acid secretion, enhancement of mucus production, antioxidant activity, anti-inflammatory effects, and inhibition of *Helicobacter pylori*. Based on their pharmacological properties, antiulcer herbs can be classified into the following categories [23].

4.1 Demulcent Herbs

Demulcent herbs contain mucilage that forms a protective coating over the gastric mucosa, shielding it from acid and digestive enzymes while promoting tissue repair. These herbs soothe irritation, reduce inflammation, and accelerate ulcer healing. Common

examples include *Glycyrrhiza glabra* (licorice), *Ulmus rubra* (slippery elm), *Althaea officinalis* (marshmallow), and *Aloe vera* [24].

4.2 Astringent Herbs

Astringent herbs are rich in tannins, which precipitate proteins on the ulcer surface to form a protective layer over damaged tissues. This action helps reduce inflammation, control minor bleeding, and promote regeneration of the gastric mucosa. Examples include *Terminalia chebula*, *Terminalia bellirica*, *Emblica officinalis*, *Psidium guajava*, and *Hamamelis virginiana* [25].

4.3 Antimicrobial Herbs

Antimicrobial herbs inhibit the growth of pathogenic microorganisms, particularly *Helicobacter pylori*, which is a major causative agent of gastric ulcers. In addition to their antibacterial activity, many of these herbs possess antioxidant and anti-inflammatory properties that further contribute to ulcer healing. Common examples include *Allium sativum* (garlic), *Curcuma longa* (turmeric), *Zingiber officinale* (ginger), *Azadirachta indica* (neem), and *Ocimum sanctum* (holy basil) [26].

4.4 Bitter Herbs

Bitter herbs contain phytochemicals that stimulate digestive secretions, improve gastrointestinal function, and enhance mucosal defense mechanisms [27]. Many also exhibit antioxidant and anti-inflammatory activities that help protect the gastric lining against ulceration. Representative bitter herbs include *Andrographis paniculata*, *Swertia chirata*, *Gentiana lutea*, and *Artemisia absinthium* [28].

4.5 Vulnerary Herbs

Vulnerary herbs promote wound healing by stimulating tissue regeneration, collagen synthesis, angiogenesis, and epithelial repair. These herbs facilitate the healing of damaged gastric mucosa and help restore normal tissue architecture. Examples include *Centella asiatica*, *Calendula officinalis*, *Plantago major*, *Aloe vera*, and *Curcuma longa* [29].

5. Natural Herbs Used in the Treatment of Gastric Ulcers

Medicinal plants have been widely employed in traditional medicine for the prevention and treatment of gastric ulcers because of their remarkable gastroprotective, antioxidant, anti-inflammatory, antimicrobial, and wound-healing properties. Many phytochemicals, including flavonoids, tannins, saponins, alkaloids, terpenoids, polysaccharides, and phenolic compounds, contribute to their antiulcer effects by enhancing mucus secretion, reducing gastric acid production,

scavenging reactive oxygen species, promoting mucosal regeneration, and inhibiting *Helicobacter pylori*. The major medicinal herbs reported to possess antiulcer activity are discussed below [30].

5.1 Cabbage (*Brassica oleracea*)

Cabbage has long been used as a natural remedy for gastric ulcers because of its high content of glutamine, vitamin C, and mucilage. These constituents help protect the gastric mucosa, promote epithelial regeneration, and accelerate ulcer healing. Fresh cabbage juice has traditionally been recommended for the treatment of peptic ulcers, gastritis, and gastric inflammation [31].

5.2 Coconut (*Cocos nucifera*)

Coconut possesses antimicrobial and anti-inflammatory properties that help improve digestive health. Coconut oil promotes the absorption of fat-soluble nutrients while inhibiting the growth of harmful microorganisms. Its antioxidant constituents protect the gastric mucosa from oxidative damage and contribute to ulcer healing [32].

5.3 Banana (*Musa paradisiaca*)

Bananas contain natural flavonoids and antibacterial compounds that inhibit the growth of *Helicobacter pylori*. They stimulate mucus production, strengthen the gastric mucosal barrier, reduce gastric acidity, and protect the stomach lining against ulcer formation [33].

5.4 Fenugreek (*Trigonella foenum-graecum*)

Fenugreek seeds are rich in mucilage, flavonoids, saponins, and phenolic compounds. The mucilage forms a protective coating over the gastric mucosa, while the antioxidant constituents reduce inflammation and oxidative stress. Experimental studies have demonstrated significant gastroprotective and antiulcer activities of fenugreek extracts [34].

5.5 Licorice (*Glycyrrhiza glabra*)

Licorice is among the oldest medicinal plants used for peptic ulcer treatment. It contains glycyrrhizin, flavonoids, coumarins, and polysaccharides that stimulate mucus secretion, suppress gastric acid production, and promote regeneration of the gastric epithelium. Deglycyrrhizinated licorice (DGL) is commonly used because it minimizes adverse effects while retaining antiulcer activity [35].

5.6 Cayenne Pepper (*Capsicum annum*)

Although traditionally considered irritating, capsaicin present in cayenne pepper has been shown to enhance gastric mucosal blood flow, stimulate mucus and bicarbonate secretion, and reduce gastric acid-

induced injury. Moderate consumption may therefore provide gastroprotective benefits [36].

5.7 Honey

Honey possesses potent antioxidant, antimicrobial, and anti-inflammatory properties. It enhances mucus production, stimulates tissue regeneration, reduces oxidative stress, and accelerates ulcer healing. Manuka honey, in particular, has demonstrated remarkable gastroprotective effects in experimental ulcer models.[37]

5.8 Garlic (*Allium sativum*)

Garlic contains numerous organosulfur compounds, including diallyl disulfide and allicin, which exhibit strong antioxidant and anti-inflammatory activities. These compounds inhibit oxidative damage, reduce inflammatory cytokine production, suppress *H. pylori* growth, and protect the gastric mucosa from ulceration [38].

5.9 Slippery Elm (*Ulmus rubra*)

Slippery elm bark is rich in mucilage that forms a soothing protective layer over the gastric mucosa. It reduces irritation, decreases inflammation, and facilitates healing of damaged gastric tissues, making it useful in gastric ulcer management [39].

5.10 Wood Apple (*Limonia acidissima*)

Wood apple contains flavonoids, tannins, polyphenols, coumarins, saponins, and essential minerals that exhibit antioxidant and gastroprotective properties. These phytochemicals protect the gastric lining from oxidative stress while supporting tissue repair [40].

5.11 Aloe vera

Aloe vera gel contains polysaccharides, vitamins, enzymes, and phenolic compounds that possess anti-inflammatory, antioxidant, and wound-healing activities. It promotes gastric mucosal regeneration, reduces inflammation, and accelerates healing of gastric ulcers [41].

5.12 Myrtus communis

Myrtus communis is traditionally used to manage gastrointestinal disorders. The plant exhibits anti-inflammatory, astringent, antioxidant, analgesic, and antimicrobial activities that contribute to protection and healing of the gastric mucosa [42].

5.13 Acacia (*Acacia arabica*)

The bark and gum of *Acacia arabica* contain high concentrations of tannins that exhibit astringent and wound-healing properties. These compounds help protect ulcerated tissues, reduce inflammation, and facilitate mucosal repair [43].

5.14 Bael (*Aegle marmelos*)

Bael is rich in flavonoids, tannins, and saponins that demonstrate significant antioxidant and anti-inflammatory activities. Experimental studies have shown that bael extracts protect the gastric mucosa by reducing acid secretion and enhancing mucus production [44].

5.15 Beetroot (*Beta vulgaris*)

Beetroot contains betalains, polyphenols, vitamins, and minerals that possess antioxidant properties. These bioactive constituents reduce oxidative stress and promote healing of gastric lesions while improving mucosal defense mechanisms [45].

5.16 Papaya (*Carica papaya*)

Papaya contains papain, chymopapain, pectin, carotenoids, and flavonoids that improve digestion and protect gastric tissues. Experimental studies have demonstrated that papaya extracts reduce gastric acidity, decrease ulcer formation, and enhance mucosal healing [46].

5.17 Sacred Fig (*Ficus religiosa*)

Ficus religiosa contains flavonoids, tannins, saponins, steroids, and phenolic compounds. These phytochemicals exhibit antioxidant, anti-inflammatory, antibacterial, and gastroprotective properties that contribute to ulcer prevention and healing [47].

5.18 Custard Apple (*Annona squamosa*)

Leaves of *Annona squamosa* are rich in alkaloids, flavonoids, tannins, and saponins. Experimental studies have shown that aqueous leaf extracts protect against ethanol-induced gastric ulcers through antioxidant and cytoprotective mechanisms [48].

5.19 Purple Tephrosia (*Tephrosia purpurea*)

Tephrosia purpurea contains flavonoids, rutin, and phenolic compounds with antioxidant and anti-inflammatory properties. Traditionally, its roots have been used in combination with honey for treating ulcers and promoting wound healing [49].

5.20 Baobab (*Adansonia digitata*)

Baobab is rich in mucilage, tannins, polysaccharides, vitamins, and minerals. Its leaves and fruit pulp possess antioxidant and anti-inflammatory properties that soothe the gastric mucosa, promote tissue regeneration, and facilitate ulcer healing. Overall, medicinal herbs represent a valuable source of bioactive compounds with significant gastroprotective potential. Their diverse pharmacological activities—including antioxidant, anti-inflammatory, antimicrobial, cytoprotective, and mucosal regenerative effects—support their use as

complementary or alternative therapies for gastric ulcer management. Further preclinical and clinical studies are needed to standardize herbal formulations, establish safety profiles, and validate their therapeutic efficacy in human patients [50].

6. Anti-Gastrointestinal Properties of Natural Plants

Natural medicinal plants possess significant anti-gastrointestinal properties due to their antioxidant, anti-inflammatory, antimicrobial, and gastroprotective activities. They help protect the gastric mucosa, reduce gastric acid secretion, enhance mucus production, promote tissue regeneration, and inhibit the growth of *Helicobacter pylori*. Many medicinal plants also improve digestion, relieve abdominal pain, reduce bloating, and support the healing of gastric ulcers and other gastrointestinal disorders. Their multitarget therapeutic effects make them promising

complementary agents for maintaining gastrointestinal health and preventing gastric mucosal damage [51].

Table 1 summarizes the medicinal plants traditionally used for the management of gastric ulcers and related gastrointestinal disorders. The table highlights their botanical names, common names, plant parts used, major bioactive constituents, and therapeutic applications. Most of these plants are rich in flavonoids, phenolic compounds, tannins, saponins, terpenoids, and alkaloids, which exhibit antioxidant, anti-inflammatory, antimicrobial, and gastroprotective activities. These phytochemicals promote ulcer healing by reducing gastric acid secretion, enhancing mucus production, protecting the gastric mucosa, and accelerating tissue repair. The information emphasizes the potential of natural plants as safe and effective complementary therapies for gastric ulcer treatment [52].

Table 1. Medicinal Plants with Reported Antiulcer Activity

S. No.	Botanical Name (Family)	Common Name	Part(s) Used	Major Active Constituents	Medicinal Uses	References
1.	<i>Aloe vera</i> (Liliaceae)	Gritkumari	Leaves	Barbaloin, isobarbaloin, saponins	Antiulcer, wound healing, skin burns, laxative	[53]
2.	<i>Azadirachta indica</i> (Meliaceae)	Neem	Leaves	Phenolic compounds, flavonoids, saponins	Antiulcer, anti-inflammatory, wound healing	[54]
3.	<i>Curcuma longa</i> (Zingiberaceae)	Turmeric (Haladi)	Rhizome	Curcuminoids, phenolic compounds, flavonoids, tannins	Antiulcer, anti-inflammatory, antioxidant, wound healing	[41]
4.	<i>Terminalia chebula</i> (Combretaceae)	Haritaki (Harad)	Fruits	Tannins, gallic acid, chebulinic acid, sorbitol	Antiulcer, Triphala constituent, anti-inflammatory	[54]
5.	<i>Terminalia bellirica</i> (Combretaceae)	Bibhitaki (Baheda)	Fruits, bark	Tannins, gallic acid, ellagic acid	Antiulcer, cough, vomiting, Triphala constituent	[55]
6.	<i>Vetiveria zizanioides</i> (Poaceae)	Vetiver	Roots	Phenolic compounds, flavonoids, saponins	Antiulcer, burning sensation, vomiting	[56]
7.	<i>Napoleonaea vogelii</i> (Lecythidaceae)	Wedi Quah	Leaves	Tannins, terpenoids, alkaloids, flavonoids, steroids	Antiulcer, gastroprotective, cough, asthma	[34]
8.	<i>Ficus arnottiana</i> (Moraceae)	Paras Peepal	Leaves	β -Sitosterol, sterols, alkaloids, tannins, phenolics	Antiulcer, anti-inflammatory, antidiarrheal	[37]

9.	<i>Nigella sativa</i> (Ranunculaceae)	Kalonji	Seeds	Nigellicine, nigellidine, quinazoline alkaloids, tannins	Antiulcer, antioxidant, antidiabetic	[57]
10.	<i>Jatropha curcas</i> (Euphorbiaceae)	Ratanjot	Leaves	Phenolic compounds, flavonoids, saponins	Wound healing, antiulcer, antimicrobial	[58]
11.	<i>Manilkara hexandra</i> (Sapotaceae)	Milk Tree	Bark	Phenolic compounds, flavonoids, saponins	Antiulcer, skin disorders	[59]
12.	<i>Panax ginseng</i> (Araliaceae)	Ginseng	Roots, stems	Ginsenosides, polysaccharide s, flavonoids, peptides	Antiulcer, antioxidant, adaptogenic	[60]
13.	<i>Falcaria vulgaris</i> (Apiaceae)	Ghazzyaghi	Seeds	Tannins, saponins	Antiulcer, gastrointestinal disorders	[57]
14.	<i>Basella rubra</i> (Basellaceae)	Malabar Spinach	Leaves	Flavonoids, saponins, proteins	Antiulcer, antioxidant	[61]
15.	<i>Nerium indicum</i> (Apocynaceae)	Kaner	Leaves, roots	Phenolic compounds, flavonoids	Traditional use in ulcers and skin disorders*	[62]
16.	<i>Acacia nilotica</i> (Fabaceae)	Kikar	Bark, leaves, pods	Tannins, flavonoids, phenolic compounds	Antiulcer, antidiarrheal, anti- inflammatory	[63]
17.	<i>Albizia lebbeck</i> (Fabaceae)	Indian Siris	Bark, leaves	Phenolic compounds, flavonoids, saponins	Anti-inflammatory, wound healing	[64]
18.	<i>Ageratum conyzoides</i> (Asteraceae)	Billy Goat Weed	Leaves	Flavonoids	Antiulcer, wound healing, anti- inflammatory	[65]
19.	<i>Glycyrrhiza glabra</i> (Fabaceae)	Licorice	Roots, rhizomes	Glycyrrhizin, triterpenoid saponins	Antiulcer, gastroprotective	[66]
20.	<i>Capsicum annuum</i> (Solanaceae)	Chilli/Papri ka	Fruits	Capsaicin, triterpenoids	Antiulcer, digestive stimulant	[67]
21.	<i>Spathodea campanulata</i> (Bignoniaceae)	African Tulip Tree	Leaves	Terpenoids, steroids	Antiulcer, antipyretic, anti- inflammatory	[55]
22.	<i>Cassia migrans</i> (Fabaceae)	Emigrant Cassia	Leaves	Flavonoids	Anti-inflammatory, antioxidant	[64]
23.	<i>Calendula officinalis</i> (Asteraceae)	Pot Marigold	Flowers	Triterpene glycosides, saponins, sesquiterpenes	Wound healing, anti- inflammatory, antiulcer	[68]
24.	<i>Bauhinia racemosa</i> (Fabaceae)	Beedi Leaf Tree	Flower buds	Phenolic compounds, flavonoids	Antiulcer, anti- inflammatory	[69]
25.	<i>Swertia chirata</i>	Chirata	Whole plant	Tannins,	Gastroprotective,	[57]

	(Gentianaceae)			flavonoids, secoiridoids	digestive tonic	
26.	<i>Kochia scoparia</i> (Amaranthaceae)	Kochia	Fruits	Phenolic compounds, flavonoids, saponins	Antiulcer, anti-inflammatory	[70]
27.	<i>Panax japonicus</i> (Araliaceae)	Japanese Ginseng	Rhizomes	Ginsenosides, flavonoids, saponins	Antiulcer, anti-inflammatory, tonic	[71]
28.	<i>Panax bipinnatifidus</i> (Araliaceae)	Asian Ginseng	Rhizomes	Phenolic compounds, flavonoids, proteins	Antiulcer, anti-inflammatory, antioxidant	[72]

7. Current Challenges

Despite the promising therapeutic potential of medicinal plants for gastric ulcer management, several challenges limit their clinical application. The major concerns include variability in phytochemical composition due to geographical location, harvesting season, and extraction methods, resulting in inconsistent therapeutic efficacy. Most herbal studies are limited to in vitro and animal models, while high-quality randomized clinical trials remain scarce [73]. In addition, the lack of standardized dosages, quality control measures, pharmacokinetic data, and long-term safety evaluations restricts their widespread clinical use. Herb drug interactions, poor bioavailability of certain phytochemicals, and insufficient regulatory guidelines further hinder the development of evidence-based herbal antiulcer therapies [74].

8. Future Directions

Future research should focus on the standardization of herbal formulations, identification of active phytoconstituents, and elucidation of their molecular mechanisms of action. Well-designed multicenter clinical trials are required to establish the efficacy and safety of medicinal plants in gastric ulcer management. Modern drug delivery approaches, including nanoparticles, liposomes, phytosomes, and hydrogel-based delivery systems, may enhance the bioavailability and targeted delivery of plant-derived compounds. Furthermore, integrating phytochemical profiling, metabolomics, artificial intelligence-assisted drug discovery, and systems pharmacology could facilitate the development of novel plant-based antiulcer therapeutics with improved clinical outcomes [75].

Conclusion

It is obvious from this research that medicinal plants have a vital role in treating a wide variety of diseases of ailments. In animal models, certain herbal plants and plant extracts have strong antiulcer efficacy. When compared to reference medications, it has

neuroprotective and stomach anti-secretory properties. Even in relatively high quantities, the extract is non-toxic. The presence of flavonoids in all of these plants is most likely responsible for the antiulcer activity. Our findings revealed that the medicinal herbs indicated above could prevent ulcers in rats in a dose-dependent way. Antiulcer activity has been observed in several botanical products; however, compounds with antiulcer action, such as flavonoids and tannins, are of special therapeutic value. This research's findings suggest that leaf and plant extracts from a variety of medicinal plants have good promise for treating peptic ulcer disease. These medicinal plants could prevent ulcers in rats in a dose-dependent way, according to our findings. The goal of this study was to look into medicinal plants' mechanisms of action in the management of experimentally produced stomach ulcers. In rats, the bark extracts were evaluated against aspirin- and ethanol-induced gastric ulcer models, as well as histamine-induced stomach acid secretion. According to the findings of the study, and investigation, the medication has antiulcer action in various gastric ulcer models. The drug's antiulcer efficacy is due to its ability to scavenge free radicals, reduce acid secretory parameters, and improve the stomach mucosal barrier.

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PA: Conceptualization; **DSL:** Literature review; **MP:** Data curation; **PG:** Supervision.

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AI Declaration

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