International Journal of Pharmaceutics and Drug Research
ISSN: 2347-6346
Available online at http://ijpdr.com

Review Article

REVIEW ON FOLK REMEDIES AND HERBAL APPROACHES IN THE TREATMENT OF GASTRIC ULCERS

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***Article History:**

Received: 16/07/2025
Revised: 10/08/2025
Accepted: 25/08/2025

ABSTRACT

Peptic ulcer disease is a prevalent gastrointestinal disorder characterized by mucosal erosion of the stomach or duodenum, primarily caused by *Helicobacter pylori* infection, prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs), stress, and lifestyle factors. Although conventional antiulcer therapies such as proton pump inhibitors, H_2-receptor antagonists, and antibiotics are effective, their long-term use is often associated with adverse effects, drug resistance, high cost, and ulcer recurrence. In recent years, increasing attention has been directed toward herbal medicines as alternative or complementary therapies for ulcer management due to their safety, affordability, and multi-targeted mechanisms of action. Medicinal plants are rich sources of bioactive phytochemicals, including alkaloids, flavonoids, terpenoids, tannins, saponins, and phenolic acids, which exhibit gastroprotective, antioxidant, anti-secretory, cytoprotective, and anti-*H. pylori* activities. Various experimental animal models, such as ethanol-, NSAID-, stress-, pylorus ligation-, and histamine-induced ulcer models, have been employed to evaluate the antiulcer potential of plant extracts and isolated compounds. This review provides a comprehensive overview of the pathophysiology, etiology, diagnosis, and treatment of peptic ulcers, along with a detailed discussion of screening models used for antiulcer activity. Special emphasis is placed on plant-derived chemical constituents and medicinal plants with proven antiulcer efficacy, highlighting their mechanisms of action and therapeutic relevance. The review underscores the potential of herbal medicines as promising candidates for the development of safer and more effective antiulcer agents and emphasizes the need for well-designed clinical trials and regulatory standardization to ensure their quality, efficacy, and safety.

Keywords: Peptic ulcer disease, Antiulcer activity, Medicinal plants, Phytochemicals, *Helicobacter pylori*, Experimental ulcer models, Herbal therapy, Gastroprotection.

INTRODUCTION

Ulceration is a crater-shaped lesion or wound that can form on the oral mucosa, large intestine (colon), and stomach mucosa. The formation of an ulcer may cause the region to bleed or discharge unpleasant materials, creating a painful sore that is susceptible to infection. Ulceration can be characterized by flaws in the epithelium, underlying connective tissue, or both. The diagnosis of ulcerative lesions may be difficult due to the wide range of potential causes and presenting characteristics of every organ. Despite the existence of various conventional and non-conventional treatment modalities for ulcer management, each approach is associated

with certain limitations, including toxicity, low efficacy, and high cost. It is crucial to highlight the fact that botanical products may contain a multitude of active constituents that possess both hazardous and advantageous effects. Thus, it is important to use herbal therapy and put in place regulations to ensure the quality of herbal products, especially when it comes to doing more randomized studies to find out how effective and safe different products are at treating ulcer disorders (Kuna *et al*., 2019).

Types of peptic ulcers:

Gastric Ulcers: The stomach lining is the site of these ulcers, which are frequently linked to excessive acid production and ongoing inflammation.

Duodenal Ulcers: An imbalance between defensive and aggressive components in the digestive system is often the origin of duodenal ulcers, which are found in the duodenum, the top portion of the small intestine (Lal and Singh, 2023).

Etiology of Peptic Ulcer:

Peptic ulcers are mostly caused by *Helicobacter pylori* (*H. pylori*) infection, the use of nonsteroidal anti-inflammatory medicines (NSAIDs), and, less commonly, stress-related mucosal injury.

Helicobacter pylori: One of the major causes of peptic ulcers, Helicobacter pylori, results in inflammation of neutrophils, lymphocytes, and macrophages as well as the degeneration and damage of epithelial cells. The symptoms of an H. pylori infection include decreased somatostatin levels and increased gastric juice output, which are caused by cytokines that inhibit parietal cell secretion. This causes the parietal cells to produce more histamine, which in turn causes them to secrete more stomach acid (Adebodun *et al*., 2023).

Non-steroidal Anti-Inflammatory Medicines (NSAIDs): Although NSAIDs harm the gastroduodenal mucosa in both systemic and local ways, the primary mechanism is thought to be the systemic suppression of prostaglandins generated from constitutively expressed cyclooxygenase 1 (COX-1). The maintenance of mucosal integrity depends on reduced mucosal prostaglandin levels, which are linked to low mucus and bicarbonate secretion, suppression of cell proliferation, and decreased mucosal blood flow (Lanas and Chan, 2017).

Stress-Related Mucosal Damage: People are more susceptible to stress-related mucosal damage and ulceration if they have experienced severe physiological stress, such as trauma, major surgery, burns, or a lifethreatening illness. Stress triggers the release of stress hormones, such as catecholamines and cortisol, which impair mucosal blood flow, reduce mucosal protective factors, and raise gastric acid secretion, all of which contribute to the development of ulcers (Singh *et al*., 2024).

Emotional factors and ulcer exacerbation: Psychological variables not only contribute to the development of peptic ulcers, but they can also affect the disease's progression. Emotional stressors and negative affective states have been linked to more ulcer symptoms, slower healing, and higher chances of ulcer recurrence. Understanding these processes is critical to creating comprehensive treatment options (Pramanik *et al*., 2024).

Diagnosis of ulcer:

Ulcer diagnosis is based on prior symptoms detected and the patient's history, such as

drunkenness, smoking, NSAID therapy, or any stress. Endoscopy (Gastroscopy) is the most reliable procedure for diagnosing ulcers since it allows for the observation of the ulcer's area and size. H. pylori is another cause of ulcers that can be detected using a variety of methods, including a urea breath test and the stool antigen test. Other supportive tests include a complete blood count to determine whether there is blood loss in situations of bleeding ulcers, a tissue culture test to detect any bacterial or fungal infections, and a stomach biopsy. Following a proper diagnosis, patients are given appropriate pharmacological therapy (antacids, antiseptics, or antibiotics) (Paricharak *et al*., 2021).

Treatments of peptic ulcer:

The purpose of peptic ulcer disease treatment is to alleviate symptoms, heal craters, prevent recurrences, and avoid complications. Medical therapy should comprise medication treatment and seek to achieve the following goals:

- Reduce gastric acidity by inhibiting or neutralizing acid release.
- Coat ulcer craters to prevent acid and pepsin from seeping into the ulcer base.
- Provide a prostaglandin analog.
- Eliminate environmental hazards including NSAIDs and smoking.
- Minimize emotional stress (for some patients) (Ithape *et al*., 2023).

Screening models for anti ulcer activity:

For the screening of the anti ulcer activity, various screening models are used that helps in the understanding of the etiology of ulcer and screening of anti ulcer agents. Animal models which were used in the screening of anti ulcer activity are as follows:

- **Ethanol induced ulcers:** Alcohol decrease mucosal resistance and causes secretion of gastric juice and due to which protein content of gastric juice is significantly increased. This could be leakage because of plasma protein in the gastric juice this leading to peptic ulcer with weakening of mucosal resistance barrier of gastric mucosa (Thamotharan *et al*., 2010).
- **Aspirin induced ulcers:** Aspirin inhibit the synthesis of prostaglandins which protects the gastric mucosa. Aspirin increases mucosal hydrogen peroxide and hydroxyl ions level to cause oxidative mucosal damage due to the inhibition of gastric peroxidise.
- **Water immersion stress induced model:** Stress can arise from tension, prolonged anxiety, emotion, burns and trauma, severe physical discomfort, surgical shock, haemorrhage thereby resulting in severe gastric ulceration (Goodman and Gilman, 1996).
- **Pylorus ligation induced ulcers:** In Pylorus ligation pylorus part of the stomach was ligated, it creates the acidic medium in stomach for longer time and produces the ulcer (Vinod *et al*., 2010).
- **Reserpine induced ulcers:** Reserpine induced ulceration has been attributed to the degranulation of gastric mast cells and subsequent liberation of histamine.
- **Serotonin induced ulcers:** Serotonin induced ulceration is arises from a

disturbance of gastric mucosal microcirculation.

- **Indomethacin induced ulcers:** Indomethacin is inhibits the synthesis of prostaglandins which are protective agents for gastric mucosa high doses of the NSAIDs causes ulceration (Rang, 2009).
- **Histamine induced ulcers:** Histamine causes ulceration by increasing the gastric acid secretion by directly acting on histamine receptors of parietal cells (Yahya *et al*., 1990).
- **Hydrochloric acid induced ulcers:** Hydrochloric acid causes ulceration by enhancing the acidity of the stomach contents.
- **Acetic acid induced ulcers:** Acetic acid induces the ulceration in stomach by increasing the acidity of stomach contents and it also causes gastric obstruction, which leads to the ulceration.

Herbal medicines

The utilization of medicinal plants for the treatment of various ailments has been practiced since ancient times and is commonly referred to as phytotherapy. The healing effects of medicinal plants come from the fact that they accommodate a wide range of secondary metabolites, called phytochemicals, which can be synthesized again and again in nature. The richest sources of active metabolites are contained in seeds, bark, roots, leaves, and fruits of plants. Therefore, a multitude of plants have employed these phytochemicals as a defense mechanism against pathogens.

Phytochemicals have been used since ancient times for the treatment of various disorders and are generally considered safer as compared to synthetic medicines. They are widely available in nature, and apart from therapeutic purposes, they are also consumed in the form of nutritional supplements. Several plant-derived chemical constituents have been reported to possess anti-ulcerative properties in various preclinical studies. These constituents belong to the class of alkaloids, tannins, flavonoids, terpenoids, glycosides, carotenoids, and saponins. Numerous botanical products have been confirmed to exhibit antiulcer activity although most studies focused on pharmacological action in animal models (Zhang *et al*., 2018).

Plant-derived chemical constituents

Alkaloids

Alkaloids represent a group of natural products that are nitrogen containing PSM, which display a considerable antiulcer activity. One important advantage of alkaloids compared to others PSM is that they have good solubility in acidic medium (stomach juice).

Epiisopiloturine hydrochloride, an imidazole alkaloid isolated from *Pilocarpus microphyllus* leaves, protects against naproxen-induced gastrointestinal damage in rats by reducing pro-inflammatory cytokines and oxidative stress and increasing gastric mucosal blood flow. Pretreatment with epiisopiloturine prevented naproxen-induced macro and microscopic gastric damages with maximal effects at 10 mg/kg. Cavidine, a major alkaloid compound isolated from C*orydalis impatiens* reduced gastric injuries in mice with ethanol-induced acute gastric ulcer at 10 mg/kg. Also, cavidine treatment resulted in increased mucosa GSH, SOD and

PGE2 levels, while decreased IL-6 and TNF-α levels. Alkaloids from *Mahonia bealei* possess anti-H^+/K^+-ATPase effects on pyloric ligation-induced gastric ulcer in rats at 18.6 mg/kg/day. Besides, 2-phenylquinoline effects were attributed to SOD and glutathione-*S*-transferase (GST) normalization activity and reduction in lipid peroxide (LPO) and TNF-α levels in the gastric mucosa from rats with gastric ulcer induced by 60% ethanol/0.03 M hydrochloric acid (HCl) and indomethacin. Chelerythrine reduced myeloperoxidase activity and nitric oxide concentration, pro-inflammatory IL-6 and TNF-α levels in ethanol-induced gastric ulcer mice at 1.5–10 mg/kg. Also, quinolone alkaloids from *Evodia rutaecarpa* have shown highly selective antibacterial activity against *H. pylori,* the minimum inhibitory concentration (MIC) found being 0.05 µg/mL. Still, an alkaloid-rich fraction extract from *Tylophora conpicua* was able to decrease histamine insulted gastric acid secretion in rats (Dey *et al*., 2017).

Terpenes and Terpenoids

Monoterpene β-myrcene isolated from *Citrus aurantium* decreased gastric and duodenal lesions, increased gastric mucus production and mucosal MDA levels, GPx and GR levels and decreased SOD activity in experimental ulcers models induced by ethanol, NSAIDs, stress, *H. pyroli*, ischemia reperfusion injury and cysteamine at 7.5 mg/kg. α-Pinene also confirmed antibacterial effects on metronidazole-resistant *H. pylori* at ethanol-induced gastric ulcer, with an EC_{50} value of 12.32 mg/kg. α-Pinene-rich essential oil (50.8%) increased gastric mucus production and induced PGE2 levels. The volatile oil of *Cedrus deodara* significantly reduced ulcers at a dose of 100 mg/kg, which justifies the traditional usage of this herb to treat peptic ulcers. Ulcer inhibition of 100 mg/kg *Cedrus deodara* and 20 mg/kg of rabeprazole was, respectively, 41.5% and 67.7%. α-Santalene rich essential oil of *Gallesia integrifolia* evidenced potent gastroprotective and curative effects in vivo and in vitro experimental models, which is probably due to its antioxidant, nitrergic, mucogenic, anti-secretory and anti-inflammatory effects. On the other hand, the essential oil of *Croton rhamnifolioides* with major components spathulenol (22.5%) and 1,8-cineole (18.3%) exhibited antiulcer activity by modulation of opioid receptors and nitric oxide. Triterpenoids 23-hydroxytormentic acid 28-*O*-glucoside isolated from *Rubus coreanus* increased SOD and GPx activities in rats with ulcer induced by combination of ethanol and sodium salicylate (Nam *et al*., 2006).

Flavonoids

Flavonoids are natural antioxidants present in different kinds of fruits and vegetables, possessing a characteristic C6-C3-C6 carbon skeleton structure. Recent studies indicated that flavonoid shows a wide range of pharmacological activities, including as antiallergic, anti-inflammatory, antimicrobial, anti-cancer, antidiarrheal and antiulcer. Due to the presence of a hydroxyl group(s) in their aromatic ring(s), they possess antioxidant activity. Quercetin, rutin and kaempferol are widespread in the plant kingdom. They inhibited the mucosal content of platelet-activating factor in rats with gastric damage produced by acidified ethanol. Rutin and quercetin isolated from *Piper umbellatum* L. showed antiulcer effect by exerting

antioxidant, anti-secretory, anti-inflammatory and mucosa regenerative activities. Hesperidin increased GSH and mucin levels and prevented oxidative cell injury in indomethacin and hypothermic restrain stress-induced ulceration models in rats. *Caryocar coriaceum* extract, with gallic acid, chlorogenic acid, caffeic acid, rutin and quercetin as major constituents, exhibited antiulcer activity through opioid and α_2-adrenergic receptors and primary afferent neurons sensitive to capsaicin in gastric ulcers induced by ethanol, acidified ethanol, acetic acid or indomethacin. Anthocyanins extracted from *Rubus coreanus* have shown antiulcer effect in association with the regulation of the matrix metalloproteinase-2 activity, preventing lipid peroxidation and even increasing CAT, SOD and GPx activities. Garcinol suppressed superoxide anion, hydroxyl radical and methyl radical in rats with acute ulceration stress induced by indomethacin and water immersion (Yamaguchi et al., 2000).

Saponins

Probably due to the presence of antioxidant saponins, the aqueous extract from *Bauhinia purpurea* leaf exhibited in vivo antiulcer activity, which confirm the traditional uses of *B. purpurea* in the treatment of ulcers (Paguigan *et al*., 2014).

Phenolic Acids

p-Coumaric acid elicited antioxidant activity by attenuating ulcers due to elevated MDA levels, reduced GSH levels and decreased SOD, CAT, GPx and GR activities. Gallic acid (1 mg/mL) showed high in vitro inhibitory effects against two *H. pylori* strains. Ellagic and gallic acids presented a prominent antiulcer action related to prostaglandins and nitric oxide/cyclic guanosine monophosphate pathway. A synergistic antiulcer activity using gallic acid and famotidine combination was observed against aspirin plus pyloric ligation induced ulcer in rats. Combination treatment resulted in increased levels of SOD, CAT, GR and glucose-6-phosphate dehydrogenase, while decreased lipid peroxidation and myeloperoxidase in gastric tissues (kumar *et al*., 2014).

Tannins

Tannins fraction of *Mouriri pusa* augmented cell proliferation, anti-inflammatory activity by reducing COX-2 levels, enhanced angiogenesis and increased mucus secretion. Ellagitannin-rich fraction increased GSH and SOD levels in rats with ethanol-induced gastric ulceration model. Hydroalcoholic extract from *Persea major* bark exerted antiulcer effects in rodents through empowering gastric protective factors. The main compounds found in the hydroalcoholic extract of *P. major* (HEPM) were polyphenols, such as condensed tannins, flavonoids heterosides derivatives from quercetin and kaempferol. HEPM (300 mg/kg) prevented gastric lesions induced by ethanol or indomethacin in rats by 58.98% and 97.48%, respectively, compared to the vehicle group (148 mm^2 and 12 mm^2, respectively) (Somensi *et al*., 2017).

CONCLUSION

Peptic ulcer disease is a common gastrointestinal disorder with limitations in conventional therapy due to side effects and recurrence. Herbal medicines offer a promising alternative as they contain multiple bioactive phytochemicals that exhibit antiulcer activity through antioxidant, anti-

secretory, anti-inflammatory, and mucosal protective mechanisms. Although many medicinal plants have shown significant efficacy in experimental models, further standardization, clinical validation, and regulatory control are essential to establish safe and effective herbal antiulcer therapies.

DECLARATION OF INTEREST

The authors declare no conflicts of interests. The authors alone are responsible for the content and writing of this article.

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Table 1: Medicinal plants with anti- ulcer activity

S. No.	Botanical name	Parts used	Model	Bibliography
1.	*Ficus arnottiana*	Leafs, fruits	Ethanol induced ulcer	Marslin *et al*., (2009)
2.	*Asparagus racemosus*	Roots	Indomethacin treated	Bhatnagar *et al*., (2006)
3.	*Azadirachta indica*	Fresh Juice	Indomethacin treated, ethanol and histamine	Chattopadhyay *et al*., (2004)
4.	*Butea foandosa*	Leaves	HCl induced ulcer	Ramesh and Ranirukmini, (2010)
5.	*Carica papaya*	Fruit and seeds	Aspirin induced ulcer	Kottaimuthu, (2009)
6.	*Careya arborea*	Bark	Ethanol induced ulcer, Pylorus ligation	Nadkarni's, (1976)
7.	*Euphorbia nerifolia*	leaf	Pylorus ligation	Azamthulla *et al*., (2009)
8.	*Hibiscus rosa sinensis*	Leaves	Pyloric ligation	Borra *et al*., (2011)
9.	*Lawsonia inermis*	Leaves	Ethanol induced ulcer	Kumar *et al*., (2013)
10.	*Momordica charantia*	Fruits	Acetic acid induced gastric ulcer, pyloric ligation induced ulcer	Alam *et al*., (2009)
11.	*Bauhinia variegata*	Leaves	Aspirin induced	Ramesh and Ranirukmini, (2010)
12.	*Aloe Vera*	Leaves	Indomethacin induced ulcer	Kumari *et al*., (2011)
13.	*Ficus religiosa*	Bark	Pyloric ligation	Nadkarni's, (1976)
14.	*Indigofera tinctoria*	Leaves	Aspirin plus pylorus ligated model	Nadkarni's, (1976)
15.	*Magnifera indica*	Leaves, flower	Aspirin-induced ulcer	Nadkarni's, (1976)
16.	*Mimosa pudica*	Leaves	Aspirin, Alcohol and pyloric ligation	Divya *et al*., (2012)
17.	*Moringa oleifera*	Leaves	Aspirin and ethanol induced	Srivastava *et al*., (2013)
18.	*Myrtus communis*	Berries	Ethanol, indomethacin and pyloric ligation induced models	Srivastava *et al*., (2013)

19.	*Ocimum sanctum*	Leaves	Methanol induced ulcer	Umarani *et al*., (2008)
20.	*Phyllanthus niruri*	Roots	Ethanol induced ulcer	Goli *et al*., (2011)
21.	*Psidium gugava*	Bark, leaves	Ethanol and pylorus ligature model	Neelapu *et al*., (2012)
22.	*Rhus coriaria*	Fruits	Ethanol induced	Neelima etal., (2012)
23.	*Sesbania grandiflora*	Leaves	Aspirin, ethanol and indomethacin induced ulcer	Vinothapooshan and Sundar, (2010)
24.	*Shorea robusta*	Resin	Ethanol and pyloric ligation	Vinothapooshan and Sundar, (2010)
25.	*Solanum nigrum*	Fruits	Cold restraint stress, ethanol and acetic acid induced ulcer	Alam *et al*., (2009)
26.	*Terminalia chebula*	Seed	ligation and ethanol induced ulcer	Rao *et al*., (2011)
27.	*Aframomum pruinosum*	Seeds	Methanol induced	Mabeku *et al*., (2017)
28.	*Bryophyllum pinnatum*	Leaf	Ethanol-induced ulcer model	Sharma *et al*., (2014)
29.	*Toona ciliata*	Wood	Ethanol induced ulcer	Malairajan *et al*., (2007)
30.	*Spondias mombin*	Leaves and flowers	Indomethacin-induced	Sabiu *et al*., (2015)
31.	*Anacardium humile*	Leaves and bark	Ethanol and piroxicam-induced	Ferreira *et al*., (2010)
32.	*Anacardium occidentale*	Leaf	Ethanol-induced	Morais *et al*., (2010)
33.	*Osyris quadripartita*	Leaf	Pylorus ligation-induced and ethanol-induced models	Abebaw *et al*., (2017)