A review on conventional and herbal drug approach to peptic ulcer

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Abstract
Peptic ulcer, a common digestive ailment, has been considered as an inflammatory response and necrotic lesions of the gastric mucosa. Peptic ulcer reaches intensely to the mucosal muscle layer in the stomach and duodenum. Various factors have been documented to be involved in the pathogenesis of peptic ulcers like Helicobacter pylori, nonsteroidal anti-inflammatory drugs (NSAIDs), acid and pepsin, genetics, and smoking. The conventional use of drugs like proton pump inhibitors (PPIs), histamine (H2) receptor antagonists, antacids, potassium competitive acid blockers, and antibiotics has shown antulcer effects. However, various researches have shown that herbal drugs can successfully treat peptic ulcers in preclinical and clinical models by different mechanisms. Many herbal drugs and their extracts from different parts like root, stem, leaf, flower, and seed showed potent ulcerprotective effects in the experimental setup. This review critically discusses the factors involved in the pathogenesis of peptic ulcers. In addition, the potential of herbal drug extracts has been highlighted in the present review.

Keywords: peptic ulcer; ulcerprotective
Introduction

Peptic ulcer has been regarded as a widespread health problem whose prevalence has amplified during recent decades [1]. According to recent reports, the anticipated incidence of peptic ulcers has been estimated to be 5–10 % in the general population [2,3]. Peptic ulcers cause sores to form in the inner lining of the stomach, the lower esophagus, and the duodenum [4]. The development of gastric and intestinal sores has been primarily attributed to the Helicobacter pylori (H. pylori) mediated inflammation or gastric acid induced gastric erosion [5]. H. pylori and NSAIDs are just two of the many elements that have been identified to play a role in the etiology of peptic ulcer [5,6]. In addition, complex acid-pepsin imbalance, genetics and smoking are the other factors that have been demonstrated to be involved in the development and progression of peptic ulcer [6,7].

According to reports, aspirin and NSAID patients risk of developing peptic ulcer problems has increased by a factor of many times in recent decades. Ischemia, chemotherapy, radiotherapy, gastric bypass surgery and metabolic disturbances have also been demonstrated to play a significant charge in the pathogenesis and progression of peptic ulcers [8–10]. PPIs, H₂ receptor antagonists and antacids present as the chief conventional therapy for patients presented with peptic ulcer [11]. Additionally, potassium competitive acid blockers and antibiotics have well been accepted as potential antulcer therapies [11,12]. Herbal drugs and their extracts have been used since ages for the treatment of patients presenting with fatal diseases [13]. Consequently, the use of herbal drug products has shown intensifying significance in the past few decades. Animal experiments using herbal medicine extracts from various plant components have yielded encouraging results [14]. These extracts include root, stem, leaf, flower and seed extracts, which showed potent ulcerprotective effects in the experimental animals [15–17]. The present review will discuss the risk factors involved in the pathogenesis of peptic ulcers and the potential use of herbal drug extracts in the management of peptic ulcers.

Pathogenesis of peptic ulcer

Numerous factors, including H. pylori, NSAIDs, acid and pepsin, heredity, and smoking, contribute to the pathogenesis of peptic ulcers (Figure 1). The infection caused by H. pylori represents one of the major factors for the pathogenesis and development of peptic ulcers. H. pylori has been known to induce significant epithelial cell degeneration and injury due to the inflammatory response with neutrophils, lymphocytes, plasma cells, and macrophages [16]. Cytokines have been considered as chief mediators of H. pylori infection which results in parietal cell secretion. Also, the H⁺/K⁺ ATPase-subunit gets directly affected by H. pylori. Additionally, H. pylori have been reported to inhibit gastrin production [16,17].

Further, the cyclooxygenase-1 (COX-1) enzyme has been known to cause prostaglandin synthesis. Moreover, it has been discovered that COX-1 inhibits cell growth, mucosal blood flow, and bicarbonate secretion [18]. The systemic inhibition of COX-1 enzyme expression presents the most important route of NSAID-associated damage. The enzyme is inhibited reversibly in a concentration-dependent manner by NSAIDs. The mucosal damage gets initiated by the uncoupling of mitochondrial oxidative phosphorylation, which is a result of mucus phospholipids disruption by NSAIDs [17]. When NSAIDs are exposed to acidic gastric juice (pH 2), they become protonated. This further causes them to penetrate lipid membranes and enter epithelial cells (pH 7.4), where H⁺ ions are released. Furthermore, in that state, NSAIDs are unable to pass the lipid membrane and become trapped in epithelial cells. This ultimately results in the uncoupling of oxidative phosphorylation, increased cellular permeability, decreased mitochondrial energy production, and reduced cellular integrity [17–19].

This has been well documented that peptic ulcers occur more commonly in smokers when compared to non-smokers [20]. Smoking-induced peptic ulcers are caused by a number of factors, including an increase in acid secretion and changes in blood flow. Additionally, it has been suggested that factors including bile reflux induction and a decrease in prostaglandin production can result in peptic ulcers in smokers [20,21]. According to reports, peptic ulcer development and progression are significantly influenced by genetics [22]. This has been shown that autosomal dominant inheritance of hyperpepsinogenemia I is seen in patients presenting with duodenal ulcers. Additionally, a variety of uncommon genetic abnormalities, including familial amyloidosis, gastrocutaneous syndrome, stiff man syndrome, and tremor nystagmus ulcer syndrome, have been linked to peptic ulcer disease [22,23].

Conventional therapy for peptic ulcer

Several conventional treatments, such as PPIs, H₂ receptor antagonists, potassium competitive acid blockers, antacids, and antibiotics, have been documented for the treatment of patients presented with peptic ulcers [24,25]. The PPIs have been documented to block the gastric hydrogen potassium (H⁺/K⁺) ATPase, an enzyme that resides on the luminal surface of the parietal cell membrane [11]. The ulcer then heals as a result of the reduction in gastric lining irritation and the inhibition of gastric acid output in the stomach and intestine [26]. Surprisingly, PPIs have also been reported to treat H. pylori infection when used along with antibiotics. In addition, PPIs have also been used to prevent ulcers in the patients exposed to long-term use of NSAIDs [27]. The main PPIs are omeprazole, lansoprazole, rabeprazole, esomeprazole and pantoprazole. H₂ receptor antagonists, which include cimetidine, famotidine, ranitidine, and nizatidine, are a different class of medications that are frequently used to treat peptic ulcers [28]. It is known that the histamine type-2 receptors on the basolateral surface of stomach parietal cells bind to H₂ receptor antagonists. This in turn inhibits the binding and activity of histamine, thereby interfering with the gastric acid production pathway, ultimately leading to the inhibition of gastric acid secretion [29]. This inhibition of gastric acid secretion further reduces irritation to the gastric lining, ultimately helping in the healing of an ulcer. Antacids like aluminum hydroxide, sodium bicarbonate, magnesium hydroxide and calcium carbonate have been known to act by neutralizing the gastric acid in the stomach and intestine [30]. Antacids have been known to increase the pH inside gastric and intestinal cells, thereby reducing the acid delivery to these sites. In addition, the antacids have been shown to restrain pepsin, a proteolytic enzyme inside gastric and intestinal cells, thus producing potent therapeutic effects [31]. Anotherclass of drugsthat have a place in conventional therapy for the treatment of peptic ulcer is potassium-competitive acid blockers like vonoprazan and revaprazan [22]. These new medications have been discovered to reversibly bind to K⁺ ions, inhibit H⁺/K⁺ ATPase enzyme in gastric parietal cells, and ultimately halt the generation of stomach acid [33]. In addition, this class of drugs possesses dose-dependent effects on gastric acid production, and is known to comprise of fast onset of action [34].

Figure 1 Factors involved in the pathogenesis of peptic ulcer

COX-1 inhibits cell growth, mucosal blood flow, and bicarbonate secretion [18]. The systemic inhibition of COX-1 enzyme expression presents the most important route of NSAID-associated damage. The enzyme is inhibited reversibly in a concentration-dependent manner by NSAIDs. The mucosal damage gets initiated by the uncoupling of mitochondrial oxidative phosphorylation, which is a result of mucus phospholipids disruption by NSAIDs [17]. When NSAIDs are exposed to acidic gastric juice (pH 2), they become protonated. This further causes them to penetrate lipid membranes and enter epithelial cells (pH 7.4), where H⁺ ions are released. Furthermore, in that state, NSAIDs are unable to pass the lipid membrane and become trapped in epithelial cells. This ultimately results in the uncoupling of oxidative phosphorylation, increased cellular permeability, decreased mitochondrial energy production, and reduced cellular integrity [17–19].

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Antulcer and antioxidative effects of ethanolic root extract of Aerva persica were investigated in wistar rats. Administration of root extract at 200 mg/kg afforded a significant reduction in the ulcer index in rats when compared to the control group. Also, histopathological studies showed a complete reduction in ethanol-induced hemorrhagic necrosis in rats after pretreatment with the ethanolic root extract, which confirmed the extracts' anticancer potential [41]. In another study, the ulcer protective potential of ethanolic root extract of Potentilla fulgens was investigated on experimental rats. When root extract was administered to rats under stress conditions caused by pyloric ligation, ethanol, and cold restraint, the study demonstrated the root extract's antulcer potential [43]. Cochlospermum planchonii, a common medicinal plant, was investigated for its anti-ulcerogenic activity in experimental rats. A number of models, including ethanol, acetic acid, cold restraint stress, pyloric ligation, and histamine-induced ulcers, were used to test the anti-ulcer activity of Cochlospermum planchonii methanolic root extract. By significantly lowering the ulcer index in rats at doses of 250 mg/kg, 500 mg/kg, and 1000 mg/kg, methanolic root extracts significantly improved the ulcer protective index. The powerful ulcer-protective abilities of the methanolic root extract were attributed to the extract's cytotoxic, antioxidant, and anti-secretory characteristics [44]. In another study, the effect of ethanol root extract of Memra nodosa on the indomethacin-induced gastric ulcers in mice was investigated. The ethanolic extract when administered at doses 100mg/kg, 300mg/kg and 1000 mg/kg significantly reduced ulcer lesions in mice. The ability of the root extract to prevent ulcers was further demonstrated by the observation of a considerable increase in adherent stomach mucus in mice when compared to the control group with lesions [45]. In ethanol-induced peptic ulcer in rats, Raesi et al. looked at the gastroprotective effects of hydro-methanolic root extract of Biebersteinia multifida. Nitric oxide levels and total antioxidant capacity in the rats' stomach mucosa were considerably increased after pretreatment with hydro-methanolic root extract at 150mg/kg and 300 mg/kg, confirming the extract's ulcer-protective properties [46].

**Stem bark extracts**

The methanolic extract of Laoeomis pacari was investigated to elucidate the probable mechanism for its antulcer potential in rats. The methanolic extract showed significant gastroprotection in ethanol, idomethacin, and cold stress-induced ulcers in rats. Moreover, the extract afforded significant ulcer healing potential in acetic acid-induced chronic ulcers, which confirmed the extract's antulcer potential. The mechanism of gastroprotection was attributed to anti-oxidant and anti-secretory properties exhibited by the extract along with pro-inflammatory cytokines inhibition [47]. Additionally, the evaluation of the antulcer potential of Synclisia scabrida ethanolic and hot aqueous stem extracts was conducted using experimentally produced ulcer models. The results demonstrated the potent antulcer potential of ethanol and hot water extracts when investigated in indomethacin, histamine- and stress-induced ulcers in albino mice [48]. Another study examined the ulceric effects of a methanol stem extract of Chasmanthra dependens in male wistar rats. Indomethacin-induced stomach ulcers were pretreated with the methanol extract at doses of 200 mg/kg, 400 mg/kg, and 800 mg/kg, and this greatly reduced ulcer formation. The outcomes demonstrated the methanolic extract's strong ulcer-protective properties, which were supported by enhanced antioxidant defenses, decreased acid output and lipid peroxidation, and therefore better stomach mucosal architecture following administration of the extract to rats with ulcers [49]. The hydroethanolic stem bark extract of Virola elongate was investigated for gastric antulcer properties by de Almeida et al. By decreasing the gastric output and acidity in ulcerated rats, the stem bark hydroethanolic extract revealed strong ulcerprotective potential. The presence of phenolic chemicals in the hydroethanolic extract of the stem bark, which provided protection due to antioxidative properties, was attributed to the mechanism of gastroprotection [50]. The study conducted by Pirbalouti et al. evaluated the potential of...
hydro-alcoholic extract from the stems of Ephedra pachyclada against ethanol-induced gastric ulcers in wistar rats. The outcomes confirmed the plant’s stem extract’s strong antiulcerogenic potential by showing that the hydro-alcoholic stem at a dose of 1000 mg/kg decreased the ulcer index and prevented stomach mucosal damage in ulcerated rats [51]. The ulcerprotective potential of aqueous stem bark extract of Ziziphus jujuba was investigated against ethanol-induced gastric ulcers in rats. Rats exhibited the stem extract at doses of 100mg, 200mg, and 400 mg demonstrated dose-dependent gastroprotection against stomach mucosal injury, confirming the plant extract’s antiulcer potential [52]. The aqueous stem bark extract of Balanites aegyptiaca was assessed for evaluation of ulcerprotective activity in Wistar rats. In the ethanol and indomethacin-induced ulcer models, administration of stem bark extract at doses of 125 mg/kg, 250 mg/kg, and 500 mg/kg resulted in a notable decrease in mean ulcer indices. The outcomes showed that the aqueous stem bark extract has strong ulcer-healing and gastroprotective effects [53]. Dietroanthenus benthamianus stem bark melanic extract’s cytoprotective and antisecretory effects on acute gastric ulcer in rats were examined by Marthe et al. The results demonstrated that the extract at 125 mg/kg, 250 mg/kg, and 500 mg/kg showed a significant reduction in ulcer index in all experimental models like ethanol-, indomethacin-, pylorus ligation-, histamine-pylorus ligation- and carbachol-pylorus ligation-induced experimental models in rats. According to reports, the melanic extract works through cholinergic and histaminergic pathways to offer an antisecretory effect [54]. The antulcer efficacy of the hydro-methanol extract of Ficus thorniiingi stem bark in rats was examined. At doses of 100 mg/kg, 200 mg/kg, and 400 mg/kg, the stem extract showed a dose-dependent, substantial reduction in total acidity. Additionally, the extract significantly decreased the ulcer index, supporting the antiulcer efficacy of the plant extract in the mouse model [55].

Leaf extracts

In order to assess the pharmacological potential of Bauhinia purpurea, the ulcerprotective activity of lipid-soluble chlorform leaf extract was investigated in rats. The results demonstrated that potent dose-dependent antulcer activity was exhibited by the plant extract in ethanol-induced ulcerations in rats. Moreover, the leaf extract has shown a significant decrease in total gastric substance volume and total gastric acidity pylorus ligation assay. The results confirmed the ulcerprotective activity as the extract showed a significant increase in gastric wall mucus production and gastric pH in ulcerated rats [56]. In albino rats, the effects of Guiera senegalensis aqueous leaf extract on stomach mucosal injury were assessed utilizing the ethanol-, water immersion-, and aspirin-induced ulcer models. The results showed that the leaf extract dose-dependently reduced the ulcer index, which confirmed the ulcerprotective potential exhibited by the plant extract [57]. Gregory et al. investigated the antulcer potential of ethanolic leaf extract of Ficus religiosa in a stress-induced rat ulcer model. This was demonstrated that administration of leaf extract at 2000 mg/kg showed dose depended on prevention in ulcer area and gastric secretion, confirming the antulcer potential exhibited by the plant extract [58]. In rat models of ethanol and pylorus ligation, the antulcer potential of the melanic leaf extract of Oxyris quadripilare was assessed. In pylorus, ligation-induced and ethanol-induced ulcer models, administration of the leaf extract at a concentration of 400 mg/kg showed a significant reduction in stomach ulcer index. The antulcer ability of the extract was further supported by pre-treatment with leaf extract, which significantly inhibited ulcers in both pylorus ligation-induced and ethanol-induced models [59]. The hydromelanic leaf extract of Urtica simensis was evaluated for ulcerprotective and ulcer healing activity in rats. The study demonstrated a dose-dependent inhibition of ulcer risk in rats, the effect which was attributed to the antisecretory activity exhibited by the leaf extract. Moreover, the leaf extract showed significant ulcer protection in a dose-dependent manner, which confirmed the antulcerogenic activity of the leaf extract [60]. Mahmoud et al. investigated the ulcerprotective potential of leaf extract of Syzygium samarangense on indomethacin-induced gastric ulcers in rats. The study’s findings showed a significant reduction in ulcer index, inflammatory cell infiltration, and inflammatory markers following pretreatment with the leaf extract. Additionally, rats with ulcers had much higher amounts of endogenous antioxidants and mucus, which supported the leaf extract’s antulcer properties [61]. Rats were used in the evaluation of the hydroalcoholic extract of Utara baccifera leaves for its gastroprotective properties. The leaf extract’s antioxidant potential, which was later discovered to be the source of the plant’s highly effective gastroprotective potential, was confirmed by the extract’s significant increases in GSH and superoxide dismutase levels and corresponding decreases in malondialdehyde peroxide levels [62]. The hydroalcoholic leaf extract of Ziziphus joazeiro was evaluated for gastroprotective activity in ulcerated rats. The hydroalcoholic leaf extract showed substantial ulcerprotective efficacy by significantly reducing indomethacin-induced stomach damage in rats when administered at a dose of 400 mg/kg [63].

Flower and seed extracts

The antioxidant and antulcer potential of Opuntia ficus indica metanolic flower extract was evaluated in ulcerated rats. The results demonstrated that the flower extract’s potent antioxidant effect was responsible for its ulcer-protective properties, which were supported by the dose-dependent inhibition of lipid peroxidation and the maintenance of normal antioxidant enzyme activities in rats with ethanol-induced gastric ulcers. In addition, the antulcer potential of the flower extract was supported by pretreatment with methanolic extract at doses of 250mg/kg, 500mg/kg, and 1000 mg/kg, which dramatically reduced deep necrotic lesions of the stomach epithelium in ulcerated rats [64]. Silva et al. conducted a study for the evaluation of the ulcerprotective outcome of hydroalcoholic extract of flowers of Chresta martii in indomethacin-induced gastric lesions in mice. The administration of flower extract significantly inhibited the indomethacin-induced gastric lesions, accounting for its potent ulcerprotective potential [65]. The metanolic extract of Tabernaemontana divaricata flowers was investigated for antulcerogenic effect in rats. The levels of antioxidant enzymes like catalase and superoxide dismutase were considerably increased after the administration of floral extract at doses of 125, 250, and 500 mg/kg. Also, the results showed a significant reduction in ulcer index, and total protein levels along with malondialdehyde levels, which further confirmed the antulcer effect exhibited by the extract [66]. In another study, the Loranthus acaciae flower extract was investigated for gastroprotective activity in ethanol-induced ulcers in rats. The flower extract at 250 mg/kg and 500 mg/kg showed reduced mucosal hemorrhage and submucosal edema in treated rats. Moreover, the flower extract dose-dependently enhanced the glutathione levels in ulcerated rats, which further confirmed the antulcer potential of the flower extract [67]. Zhang et al. investigated the gastroprotective effect of the extract of Jasminum grandiflorum flower in ethanol-induced ulcers in mice. According to the findings, the floral extracts significantly reduced the risk of gastrointestinal mucosal ulcers, perhaps by boosting the activity of antioxidant enzymes. Additionally, the floral extract prevented the release of pro-inflammatory cytokines, lipid peroxidation, and reactive oxygen species in mice with ulcers [68]. The antulcer and antioxidant potential of Myrtle berry seed aqueous extract was investigated in ethanol-induced peptic ulcers in male wistar rats. Treatment with the Myrtle berry seed extract dose-dependently guarded against ethanol-induced changes in the histology and macroscopic structure of the duodenum and stomach. The extract further demonstrated the antulcer and antioxidant capabilities of the seed by significantly preserving baseline antioxidant enzyme activities and nonenzymatic antioxidant levels [69]. Rats were used to test the antulcer effects of aqueous lplantacca floribunda seed extract. According to the study, pretreatment with the aqueous extract dramatically decreased rat stomach ulcers and gastric pH. In addition, the extract demonstrated potent antioxidant activity, which helped in affording gastric protection in ulcerated rats [70]. The study aimed at assessing the
potential of selenium and grape seed extract on indomethacin-induced gastric ulcers in rats. The research showed that the extract considerably reduced the rise in stomach ulcer index caused by indomethacin. Additionally, pretreatment with the seed extract increased the levels of catalase, glutathione peroxidase, and superoxide dismutase in rats with ulcers, further demonstrating the extract's antiulcer properties [71]. The antiulcer activity of the hydromethanolic seed extract of Cordia africana was investigated in pylorus-ligated rats. Administration of extract showed a significant reduction in secretion volume and gastric acidity along with a decrease in ulcer score in rats [72]. Male albino rats that had peptic ulcers caused by indomethacin were given an aqueous seed extract of Carica papaya to test for antiulcer properties. In rats with ulcers, the seed extract dramatically raised stomach pH and the percentage of ulcer inhibition. The antiulcer potential of the plant extract was further supported by the seed extract, which significantly decreased stomach acidity, gastric acid production, gastric peptic secretion, ulcer index, and gastric secretion volume in ulcerated rats [73].

Based on the above literature, herbal drugs can be classified according to the mechanism through which the ulcerprotective effect is afforded. The first classification is attributed to the significant reduction of ulcer index in experimental animals [39,53,54,55,57,59,66]. Secondly, the herbal drugs can be categorized according to the mechanism involving an increase in the activity of gastric antioxidant enzymes [40,46,49,62,66,69,70,71]. Thirdly, the herbal drugs may be classified by a mechanism attributed to the reduction in gastric acidity and consequent increase in gastric pH by the herbal drugs [41,42,44]. Fourthly, herbal drugs can be categorized on the basis of the prevention of gastric mucosal damage by increasing gastric wall mucus production [51,52,56,68,69,73]; and reducing gastric secretion in ulcerated animals [50,58,72]. Lastly, the herbal drugs may be classified on the basis of a mechanism involving a reduction in deep necrotic lesions [4,65,67] and inflammatory markers in ulcerated animals [47,60,61].

Future perspectives

The herbal drugs have managed to offer potent antiulcerative effects in experimental and clinical setups. The narrow adverse effect profile and contracted recurrence rates make herbal drugs the choice of drugs for the management of ulcers. Additionally, a synergistic effect of herbal medicines and traditional antiulcer medications has been demonstrated against peptic ulcers. This can be said that herbal drugs alone or in combination with conventional drugs could serve as a potential therapy for gastric ulcers alongwith prevention of recurrence. Our laboratory is in the process of elucidating the antiulcer potential of a few herbal drugs in experimental animals. The likely outcomes would be used to enhance the prognosis for patients who have peptic ulcers in the future. Moreover, licensing is necessary to enhance the safety and quality of herbal medicine items used for therapeutic purposes so that they can be employed as a possible target for the management of peptic ulcers.

Conclusion

Peptic ulcer has been a recurrent clinical predicament that is affecting people of all ages. In the past, it has been widely accepted to treat peptic ulcers with medications such as PPIs, H2 receptor antagonists, antacids, and antibiotics. However many synthetic drugs have been reported to treat peptic ulcer patients but herbal drug extracts have shown great potential as ulcerprotective agents in the last decade. Traditionally, several herbal drugs have been used as the remedy for peptic ulcers, still scientific information regarding their potential in in-vivo, in vitro and clinical studies remain insufficient. Hence, further research is warranted in order to explicate precise mechanisms of ulcerprotection by herbal plants and their extracts. It would be of great interest to know the probable mechanisms of ulcerprotection of herbal plants and their extracts so that they may present a promising potential therapy in the clinical setup as well.

References

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