

Peptic Ulcer and Role of a Polyherbal Formulation in Pathogenesis of Ulcer in Wistar Rats

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ABSTRACT

Peptic ulcers are characterized by gastrointestinal tract mucosal erosions that may extend to the muscular layer. Their complex aetiology is brought on by an imbalance between the mucosa's offensive and defensive components. Peptic ulcers, which afflict millions of people globally and have high rates of recurrence, are a major public health concern. The use of non-steroidal anti-inflammatory medicines (NSAIDs) and *Helicobacter pylori* infection are two of the most significant risk factors for the development of peptic ulcers. Up until the later decades of the 20th century, when epidemiological patterns started to indicate to an astonishing decline in its prevalence, peptic ulcer disease had a significant impact on morbidity and death. As a result, novel complementary treatment strategies are required to stop the onset of ulcers and their recurrence. In experimental models of peptic ulcers, natural products including medicinal plants and their extracted components have been frequently employed. The Sarangdhar Samhita, a work of Ayurvedic literature, emphasised the idea of polyherbalism as a means of enhancing medicinal efficacy. Individual plants' active phytochemical components are inadequate to produce the desired therapeutic effects. The medicinal effects and toxicity are improved when several herbs are combined in a certain ratio. The importance of polyherbalism and its therapeutic value are primarily the subject of this review.

Keywords: Peptic Ulcer; Polyherbal; *Helicobacter Pylori*; Clinical; Indomethacin; Ibuprofen; Peptic Ulcer Disease

Introduction

Peptic ulcer disease (PUD) is a medical term used to describe ulcerative conditions affecting the lower oesophagus, upper duodenum, and lower stomach. PUD affects over 4.5 million people annually in the United States alone, with a lifetime frequency of 10% [1]. PUD is more common as people age, with most ulcers developing between the ages of 25 and 64 [2]. The majority of individuals with uncomplicated PUD can be treated successfully, if adequately evaluated. Through clinical examination, those who are PUD-at-risk or who already have it can be identified earlier, reducing the risk of consequences. Globally, peptic ulcer disease is a major cause of illness and mortality. Consequences can include every aspect from gastrointestinal bleeding and abdominal pain to perforation and occlusion of the gastric outlet. Peptic ulcers, which afflict millions of

people globally and have high rates of recurrence, are a major public health concern. One of the key risk factors for the onset of peptic ulcers is the co-infection of *Helicobacter pylori* and the use of non-steroidal anti-inflammatory medicines (NSAIDs).

Pathophysiology

A physiological balance between stomach acid secretion and gastroduodenal defence operates under normal circumstances. Peptic ulcers are caused by damage to the mucosa and develop when the balance between aggressive and protective elements is upset [2]. Acid erosion of the GI tract lining leads to the development of peptic ulcers. The gastric mucosa breaks down as a result of this injury, resulting in ulcerations [3]. The most likely cause of PUD in the past was thought to be a hypersecretory, acidic environment combined with dietary

variables and/or stress. However, new perceptions of PUD aetiology have emerged as a result of the identification of *Helicobacter pylori* (*H. pylori*) infection and the growing popularity of non-steroidal anti-inflammatory medicines (NSAIDs). The two main causes of peptic ulcers using NSAIDs and having *H. pylori* are uncommonly combined to cause PUD. This suggests that individual susceptibility to bacterial virulence and drug toxicity may be crucial to the beginning of mucosal damage [4].

H. pylori Induced Ulcer

A very substantial correlation between *H. pylori* infection and duodenal and stomach ulcers was found by epidemiological investigations. The permanent treatment of peptic ulcers by the eradication of the infection [5,6] served as the conclusive evidence of *H. pylori* as the primary cause of ulcer disease. The entire stomach epithelium, from the prepyloric antrum to the cardia, is colonised by *H. pylori* (Figure 1). Gram negative bacteria produces various heat shock proteins like cytokines, histamine, liposaccharides (urease, protease, fructosidase etc.) and enzymes (phospholipase).

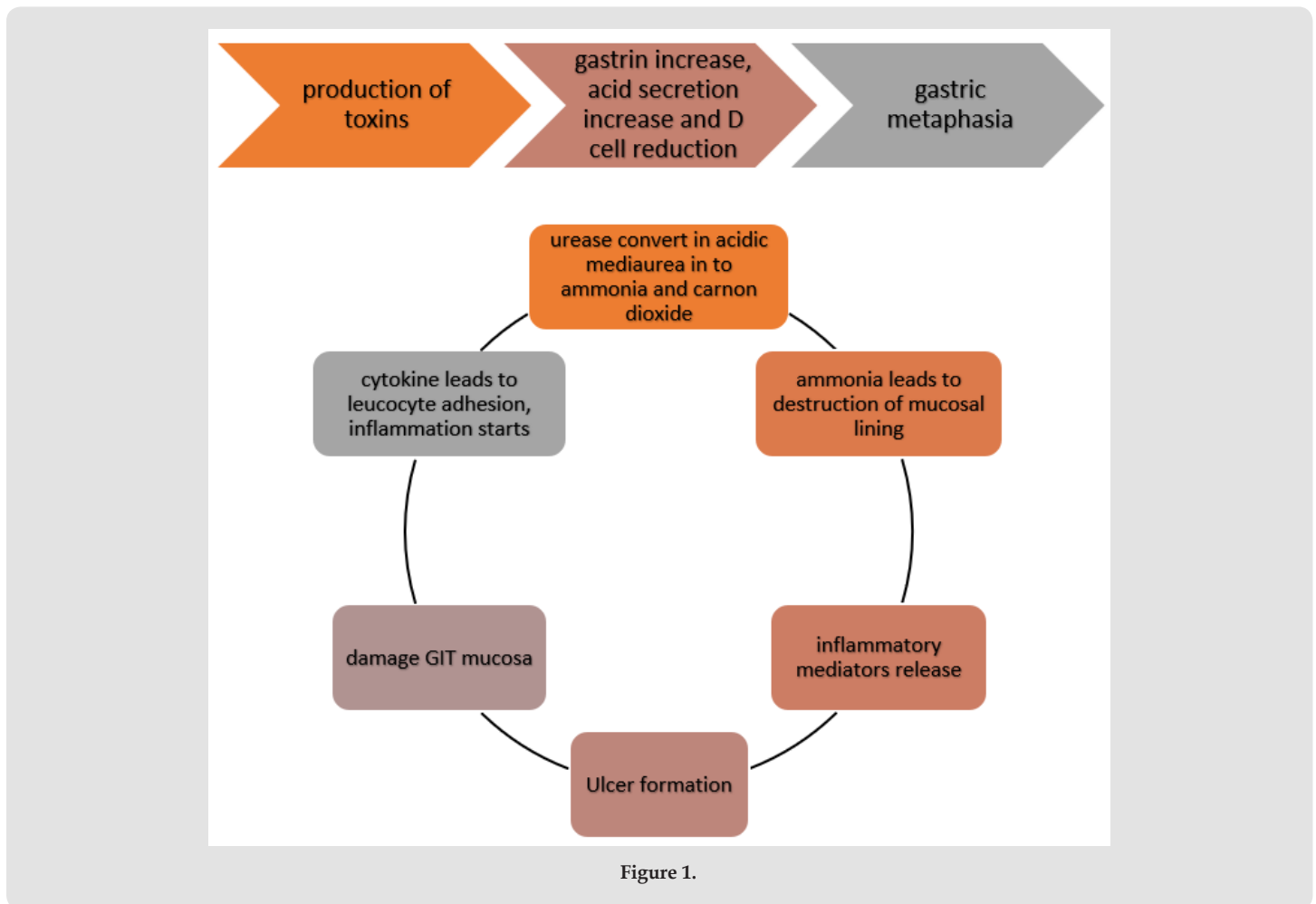


Figure 1.

NSAIDs Induced Peptic Ulcer

It was originally believed that one of the key mechanisms of NSAID-induced stomach damage was topical irritation caused by ion trapping [7] and loss of mucus gel hydrophobicity [8]. Later research revealed that NSAIDs primarily harm the stomach by inhibiting gastric prostaglandin formation. It was originally believed that an important mechanism of NSAID-induced stomach damage involved topical

irritation caused by ion trapping and a decrease in the hydrophobicity of mucus gel. Later research revealed that NSAIDs primarily harm the stomach by inhibiting gastric prostaglandin production [9]. By generating proteases, releasing oxygen free radicals, and blocking capillary blood flow, neutrophil adhesion harms the mucosa. In animal models, inhibiting neutrophil adhesion reduces the harm caused by NSAIDs. These drugs, like aspirin, indomethacin, ibuprofen, etc., are

known to cause ulcers when misused (Figure 2). The suppression of the prostaglandin synthetase 5 enzyme in the COX pathway is thought to be the cause of these ulcers. Prostaglandins are found in a variety of

tissues, including the stomach, where they serve a protective purpose by causing the release of bicarbonates and mucous membranes, which increases the blood flow through the mucous membranes.

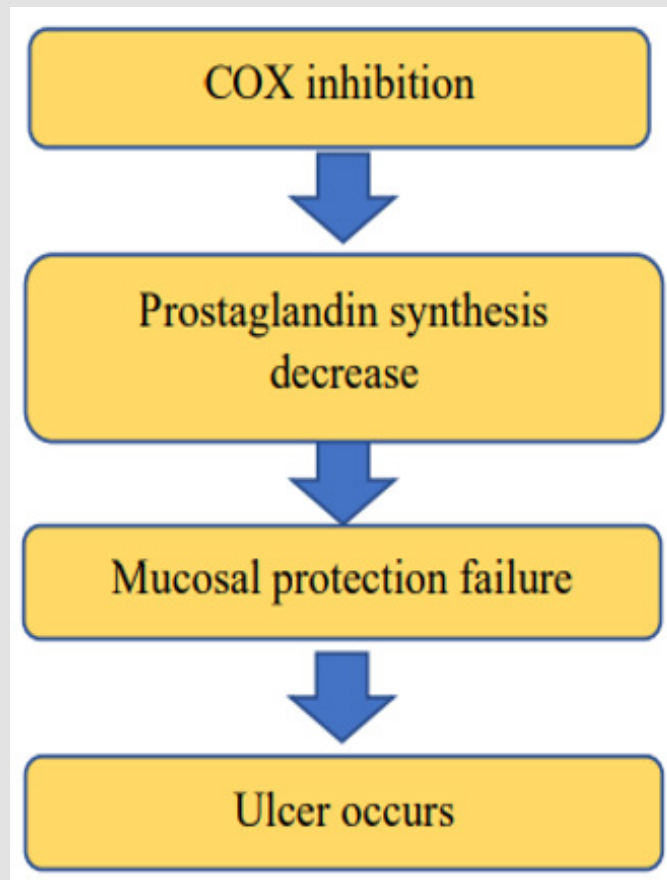


Figure 2.

Stress Induced Peptic Ulcer

Stress-related apparent ulcers have a single or numerous mucosal abnormalities. The physiopathology of stress-induced ulcers is

complicated; ulcers are brought on by the release of histamine, which also induces the secretion of acid. Increased stress has also been linked to decreased stomach mucus volume and consistency (Figure 3).

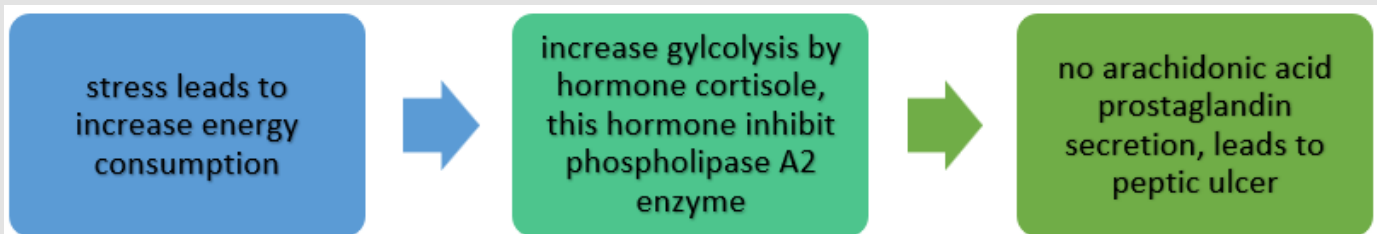


Figure 3.

Clinical Manifestation and Diagnosis

Epigastric discomfort, which might also include additional dyspeptic symptoms as fullness, bloating, early satiety, and nausea, is the main sign of an uncomplicated peptic ulcer. Patients with duodenal ulcers frequently have epigastric pain when fasting or even at night, which is typically eased by eating or using acid-neutralizing medications. Heartburn is also experienced by around a third of these individuals, usually without erosive oesophagitis [10]. Asymptomatic chronic ulcers are possible [11]. Asymptomatic chronic ulcers are possible. This lack of symptoms is particularly noticeable in NSAID-induced ulcers, where upper gastrointestinal bleeding or perforation may be the initial clinical sign of the condition. The most common and serious complication of peptic ulcers is bleeding, which affects 50–170 persons out of every 100,000, with people over 60 having the highest risk [12-14]. With an incidence of seven to ten per 100 000, perforation is less common than bleeding [15,16]. Although it is fortunately uncommon, retroperitoneal organ penetration is defined by persistent, excruciating discomfort [17]. A mucosal break that is at least 5 mm in diameter and covered in fibrin is a peptic ulcer; any mucosal break that is smaller than 5 mm is referred to as an erosion. Despite being arbitrary, the 5 mm threshold is applied in clinical trials. Uncertainty exists regarding the relationship between this criterion and the pathological criterion of muscularis mucosa penetration. One or more peptic ulcers are possible.

The bulb, where the contents of the stomach enter the small intestine, is the usual site of the duodenal ulcer. Although they can develop everywhere from the pylorus to the cardia, gastric ulcers are most common in the angulus of the lesser curvature. Considerations for underlying Crohn's disease, ischaemia, or the uncommon Zollinger-Ellison syndrome should be made if ulceration is found in the more distal duodenum. Following endoscopic peptic ulcer diagnosis, biopsy samples of the antral, body, or fundus mucosa should be obtained for fast urease and histological testing to identify H pylori infection. Patients up to 55 years old with ulcer-like symptoms are typically not examined endoscopically in many industrialised nations, but rather non-invasively for H pylori (13C-urea breath test [UBT], stool antigen test), and if positive, treated with H pylori eradication [18]. The theory behind this test-and-treat approach is that a part of patients' symptoms will be caused by an ulcer illness that can be healed by treating H pylori. Malignant illness is also uncommon in young people and in the absence of warning signs such as nausea, vomiting, anaemia, or weight loss.

Management

The development of medical remedies has focused on gastric acid secretion and mucosal defence mechanisms since Karl Schwarz's maxim, "No acid, no ulcer." Although several medications have been used to treat ulcers, not all of the early therapies were successful. The medication classes that prevented stomach acid secretion were

the most effective. When used as maintenance therapy, H₂-receptor antagonists have transformed the treatment of peptic ulcers by curing ulcers and maintaining them in remission [19,20]. The PPIs, a more potent family of acid-inhibitory medications that became available in 1989, progressively took their position. PPIs specifically inhibit the parietal cell's H⁺ K⁺ ATPase [21]. PPIs became the standard in ulcer management because of the theory that the degree of acid suppression is related to how quickly an ulcer heals. However, following the healing phase ulcers were frequently seen to return, and for years until the ground-breaking development of H pylori eradication therapy, standard practise was to maintain patients on maintenance acid suppression. A second class of medications works to strengthen the mucosal barrier and finds its main use in NSAID and aspirin resistance. The most popular prostaglandin analogue, misoprostol, has been used but its usage has been constrained by abdominal side-effects, particularly at larger doses [22]. By enhancing mucosal repair, sucralfate and bismuth salts also aid in the healing of ulcers. Sucralfate may also have some effect by lowering acid production and stifling H pylori infection [23].

Only when combined with antibiotics are bismuth salts, which have modest inherent anti-H pylori action, utilised in ulcer therapy. More efficient treatments have rendered cytoprotective medicines obsolete. In order to treat and prevent peptic ulcers brought on by gastro-toxic medications, H pylori must be eradicated in cases when the patient has a peptic ulcer that is H pylori-positive. In India, peptic ulcer disease (PUD) has become a widespread illness. Around 15000 people each year pass away from the peptic ulcer disease (PUD). Antacids and antiulcer medications account for 6.2 billion rupees and 4.3% of the Indian pharmaceutical market. Three methods are typically employed by licenced medical professionals to treat or cure ulcers. The first one focuses on reducing the formation of stomach acid, the second one on neutralising gastric acid, and the third and last one on reinforcing gastrointestinal mucosal protection. The availability of drugs that impact the mucosal barrier, histamine H₂ receptor blockers, proton pump blockers or inhibitors, and prostaglandin analogues has made these methods possible. Studies and clinical assessments of these medications reveal drug tolerance and recurrent ulcers. The effectiveness of these medications is called into question by these negative effects. The development of novel antiulcer medications, some of which contain herbal sources, was motivated by these negative effects. Peptic ulcer diseases (PUD) are among the many conditions that can be treated with the use of Indian medicinal plants and herbs. The general tendency has recently changed, and people are turning more and more to herbal remedies instead of synthetic ones. So, we might refer to this as "returning to nature." In this sense, India has a distinct and well-established identity in the globe, where several recognised native and indigenous medical systems, including Ayurveda, Siddha, Unani, Homoeopathy, Yoga, and naturopathy, are employed to treat the populace's health [24,25].

Medicinal Plants in the Treatment

Because they have less adverse effects than allopathic therapy, medicinal plants are now employed for the treatment and prevention of illnesses. They are reasonably priced and conveniently accessible. Because of this, medicinal plants are becoming more and more important. Modern pharmaceutical substances were built on the use of plant parts, extracts, and their separated pure components [26]. Since ancient times, medicinal plants have been utilised to heal various illnesses. In order to cure, prevent, or promote mental and physical health, traditional medicine is described as the body of knowledge, skills, and practises based on ideas, beliefs, and experiences that are native to many cultures. Ancient Hindu writings have extensive documentation of knowledge of therapeutic herbs. Approximately 70% of the Indian population today still relies on the conventional medical system. Historically, man's most effective defence against viruses and illnesses has been plants. The majority of the people from every sector of the community uses medicinal herbs. Many medications that doctors recommend either come in intentionally altered forms of natural products or are extracted from plants. In order to fight sickness, medicinal plants are essential to human society [27]. Due to the existence of multiple chemical components with various chemical structures, known as secondary plant metabolites, medicinal plants have therapeutic properties. One more area of the plant contains these secondary plant metabolites.

These secondary plant metabolites are classified as alkaloids, glycosides, corticosteroids, flavonoids, essential oils, etc. Based on their chemical makeup and structure. Because whole plant extracts or part extracts contain multiple chemical constituents that interact to produce therapeutic effects and have a lower risk of side effects, traditional medical practitioners prefer using them over extracting plant components. Since more plants contain more chemical components and are more likely to be effective at curing diseases and reducing the dose and preparation, herbalists and herbal medical professionals typically favour employing polyherbal preparations for the treatment of ailments. The demand for herbal remedies and preparations is currently skyrocketing throughout the globe, and pharmaceutical companies are getting involved in studies on plant material for its potential and efficacy as a medication [28]. Due to their less adverse effects, medications with plant origins are becoming more popular nowadays. There are several historically used plant-based products on the market that have been shown effective in treating peptic ulcers. Some plants which have ulcer treating property janglibathua (*Chenopodium ambrosioides*), banana (*Musa paradisiaca*), bhang (*Cannabis sativa*), dhatura (*Datura stramonium*), kher (*Acacia catechu*), *Asparagus racemosus*, *Glycerrhiza glabra*, *Malva Sylvestris*, *Nerium indicum*, *Rawvolfia serpentine*, *Tamerindus indica*, *Abutilon indicum*, *Andrographis paniculate*, *Asystasia gangatica*.

Many ayurvedic products that are used to treat peptic ulcers are offered in India. The herbal teas of chamomile, calendula, and *Malva sylvestris* are among them, as are *Avipattikar churna*, *Kamdudha ras*, *Sutshekhar ras*, *Amlapittantak lauh*, *Leelavilas rasa*, *Alsarax tab*, *aloe vera gel*, *Nirgundi taila*, and *Shankha Bhasma*. In herbal practise, the practitioner may prescribe some plants in their raw form or in any ayurvedic preparation, such as liquorice decoction, cabbage and potato juice, tulsi leaves and the dried and candied fruits of *Embillica officinalis* [29-31]. Previously, the antiulcer properties of several medicinal herbs were studied. *Ficus arnottiana* (leaf extract in methanol), *Desmodium gangeticum* (plant extract in ethanol), *Asparagus racemosus* (root extract), and *Terminalia pallida* (plant extract) are a few examples. Flavonoids and tannins in *Ficus arnottiana* are what give it its antiulcer properties [32-34]. Garlic (*Allium sativum*) bulb juice, *Aegle marmelos* fruit, *Glycerrhiza glabra* roots, *Panax japonica* and *Panax binnatifidus* rhizomes, *Aesculus hippocastanum* seeds, and *Callindra portoticensis* leaves all have antiulcer efficacy since saponins are present in them [35-37]. Plants that contain tannins, such as *Callindra portoticensis* leaves, *Ficus arnottiana* leaves, *Linderae umbellata* stems, and *Mallotus japonica* bark, also have antiulcer properties. In many plants, mucilage and gum are also responsible for the antiulcer action. *Aloe*, *Abelmoschus esculentus*, *Cyamopsis tetragonolobus*, *Malva sylvestris*, and *Commiphora molmol* are a few of them [38,39].

Single Herbal versus Polyherbal Formulation

Ayurvedic Medication Formulation is Based on Two Principles

using more than one drug (PHF) and using more than one drug as a single drug. This important traditional therapeutic herbal approach, also known as polypharmacy or polyherbalism, makes use of the interaction between many therapeutic plants to increase therapeutic efficacy. Historically, the Ayurvedic literature "Sarangdhar Samhita" dated centuries ago in 1300 A. D. has highlighted the concept of polyherbalism in this ancient medicinal system [40]. In the traditional system of Indian medicine, plant formulations and combined extracts of plants are chosen rather than individual ones. It is known that Ayurvedic herbals are prepared in a number of dosage forms, in which mostly all of them are PHF [41,42]. Even while the active phytochemical components of specific plants have a well-established history, they are typically only found in trace amounts and are seldom enough to provide the desired therapeutic effects. Because of this, research has shown that combining these different plants with differing potencies might conceivably result in a better outcome than using them separately or adding up their unique effects. Synergism is the term used to describe this beneficial herb-herb interaction phenomena. Certain pharmacological effects of herbal products' active ingredients are notable only when amplified by those of other plants; they are not noticeable when taken alone.

Role of Polyherbal Preparation

Research was carried out in Rajiv Academy for pharmacy, Mathura by Himani Gupta. In which a polyherbal preparation was prepared using three different plants named as *Catharanthus roseus*, *Azadirachta indica* and *Ocimum sanctum*. In the study, Ethanol extract of polyherbal preparation was used for the evaluation of anti-ulcer activity by the means of two different animal model i.e., Pylorus ligation induced ulcer and Ethanol induced ulcer. Ethanol extract of polyherbal preparation at dose of 200 and 400mg/kg showed significant antiulcer activity by decreasing the gastric volume in pylorus ligation method and also reducing the formation of ulcer in pylorus ligation and ethanol induced model in test group while compared to control group animal. The extract reduced the ulcer index and protected animal from the ulcers. This ethanolic extract also reduced the gastric secretion in pylorus ligation model test animals. Also, the pH of gastric content drastically increased denoting the decreased acidity in the stomach in same model, even total acidity and free acidity was also reduced [43].

Conclusion

From the result of the previous study, it can be concluded that the ethanolic extract of polyherbal preparation have antisecretory and cytoprotective activity which may be due to presence of various phytochemical like tannins, alkaloids, phenols, sterols etc. As literature reveals that the tannins affect the secretion of mechanism of pepsin. Flavonoids increase prostaglandin synthesis and decrease histamine-induced gastric acid secretion. Alkaloids stimulates mucin secretion. They can potentially be used as preventive and complementary drugs or as dietary supplements to prevent the development of peptic ulcer and its episodes of recurrence and/or assist in the traditional treatment of ulcerative lesions. Products of natural origin, especially composed of plant foods and plants, often referred to as complementary and alternative medicines, such as nutraceuticals and herbal medicines, respectively, have stood out for their therapeutic potential, which can assist in the management of many diseases.

References

- Dunlap JJ, Patterson S (2019) Peptic ulcer disease. *Gastroenterology Nursing* 42(5): 45-454.
- Anand BS, O Katz (2018) Peptic ulcer disease.
- Lamont JT (2022) Patient education: Peptic ulcer disease (Beyond the Basics).
- Lanas A, Chan FK (2017) Peptic ulcer disease. *The Lancet* 390(10094): 613-624.
- Rauws EA, Tytgat GN (1990) Cure of duodenal ulcer associated with eradication of *Helicobacter pylori*. *The Lancet* 335(8700): 1233-1235.
- Malfertheiner P, Leodolter A, Peitz U (2000) Cure of *Helicobacter pylori*-associated ulcer disease through eradication. *Best Practice & Research Clinical Gastroenterology* 14(1): 119-132.
- Davenport HW (1969) Gastric mucosal hemorrhage in dogs: Effects of acid, aspirin, and alcohol. *Gastroenterology* 56(3): 439-449.
- Lichtenberger LM, Wang ZM, Romero JJ, Ulloa C, Perez JC, et al. (1995) Non-steroidal anti-inflammatory drugs (NSAIDs) associate with zwitterionic phospholipids: Insight into the mechanism and reversal of NSAID-induced gastrointestinal injury. *Nature medicine* 1(2): 154-158.
- Wallace JL (2008) Prostaglandins, NSAIDs, and gastric mucosal protection: why doesn't the stomach digest itself?. *Physiological reviews* 88(4): 1547-1565.
- Malfertheiner P, Dent J, Zeijlon L, Sipponen P, Veldhuyzen Van Zanten, et al. (2002) Impact of *Helicobacter pylori* eradication on heartburn in patients with gastric or duodenal ulcer disease—results from a randomized trial programme. *Alimentary pharmacology & therapeutics* 16(8): 1431-1442.
- Dew MJ (1987) Asymptomatic peptic ulcer disease. *British Medical Journal (Clinical research ed.)* 295(6595): 401.
- Blatchford O, Davidson LA, Murray WR, Blatchford M, Pell J, et al. (1997) Acute upper gastrointestinal haemorrhage in west of Scotland: case ascertainment study. *Bmj* 315(7107): 510-514.
- Rockall TA, Logan RFA, Devlin HB, Northfield TC (1995) Incidence of and mortality from acute upper gastrointestinal haemorrhage in the United Kingdom. *Bmj* 311(6999): 222-226.
- Longstreth GF (1995) Epidemiology of Hospitalization for Acute Upper Gastrointestinal Hemorrhage: A Population--Based Study. *American Journal of Gastroenterology (Springer Nature)* 90(2): 206-10.
- Gisbert JP, Pajares JM (2003) *Helicobacter pylori* infection and perforated peptic ulcer prevalence of the infection and role of antimicrobial treatment. *Helicobacter* 8(3): 159-167.
- Malfertheiner P, Chan FK, McColl KE (2009) Peptic ulcer disease. *The lancet* 374(9699): 1449-1461.
- McColl K (2000) Should Non-Invasive *Helicobacter pylori* Testing Replace Endoscopy in Investigation of Dyspepsia?. *Helicobacter* 5(S1): 11-15.
- McColl KEL, Murray LS, Gillen D, Walker A, Wirz A, et al. (2002) Randomised trial of endoscopy with testing for *Helicobacter pylori* compared with non-invasive H pylori testing alone in the management of dyspepsia. *Bmj* 324(7344): 999-002.
- Collen MJ (1991) Idiopathic gastric acid hypersecretion. *Pharmacology of Peptic Ulcer Disease*, pp. 325-348.
- Bianchi Porro G, Lazzaroni M (1992) Peptic ulcer—medical treatment. The stomach. Edinburgh, London, Madrid, Melbourne, New York, Tokyo. Churchill Livingstone, pp. 246-265.
- Fellenius E, Berglindh T, Sachs G, Olbe L, Elander B, et al. (1981) Substituted benzimidazoles inhibit gastric acid secretion by blocking (H⁺⁺ K⁺) ATPase. *Nature* 290(5802): 159-161.
- Silverstein FE, Graham DY, Senior JR, Davies HW, Struthers BJ, et al. (1995) Misoprostol reduces serious gastrointestinal complications in patients with rheumatoid arthritis receiving nonsteroidal anti-inflammatory drugs: A randomized, double-blind, placebo-controlled trial. *Annals of Internal Medicine* 123(4): 241-249.
- Banerjee SUBHAS, El Omar EMAD, Mowat ANNETTE, Ardill JE, Park RH, et al. (1996) Sucralfate suppresses *Helicobacter pylori* infection and reduces gastric acid secretion by 50% in patients with duodenal ulcer. *Gastroenterology* 110(3): 717-724.
- Koehn FE, Carter GT (2005) The evolving role of natural products in drug discovery. *Nature reviews Drug discovery* 4(3): 206-220.

25. Manonmani S, Vishwanathan V P, Subramanian S, Govindasamy S (1995) Biochemical studies on the antiulcerogenic activity of cauvery 100, an ayurvedic formulation in experimental ulcers. *Indian journal of Pharmacology* 27(2): 101-105.
26. Kar A (2003) *Pharmacognosy and pharmacobiotechnology*. New Age International.
27. Uniyal B, Shiva V (2005) Traditional knowledge on medicinal plants among rural women of the Garhwal Himalaya, Uttaranchal.
28. Pandey MM, Rastogi S, Rawat AK (2008) Indian herbal drug for general healthcare: an overview. *The internet journal of alternative medicine* 6(1): 3.
29. Pandit S, Sur TK, Jana U, Bhattacharyya D, Debnath PK, et al. (2000) Anti-ulcer effect of Shankha bhasma in rats: A preliminary study. *Indian Journal of Pharmacology* 32(6): 378-380.
30. Babu Dr SS (2005) *A Text book of A Treatise on Home Remedies*. Pustak Mahal, New Delhi.
31. Bakhru HK (1992) *Herbs that heal: Natural remedies for good health*. Orient paperbacks.
32. Aguwa CN, Lawal AM (1988) Pharmacologic studies on the active principles of *Calliandra portoricensis* leaf extracts. *Journal of ethnopharmacology* 22(1): 63-71.
33. John TA, Onabanjo AO (1990) Gastroprotective effects of an aqueous extract of *Entandrophragma utile* bark in experimental ethanol-induced peptic ulceration in mice and rats. *Journal of ethnopharmacology* 29(1): 87-93.
34. Ezaki N, Kato M, Takizawa N, Morimoto S, Nonaka G I, et al. (1985) Pharmacological Studies on *Linderae umbellatae* Ramus, IV*. Effects of Condensed Tannin Related Compounds on Peptic Activity and Stress-Induced Gastric Lesions in Mice. *Planta medica* 51(01): 34-38.
35. Iatsyno AI, Belova LF, Lipkina GS, Sokolov SI, Trutneva EA, et al. (1978) Pharmacology of calendulose B, a new triterpene glycoside from the roots of *Calendula officinalis*. *Farmakologiya i toksikologiya* 41(5): 556-560.
36. Aguwa CN, Lawal AM (1988) Pharmacologic studies on the active principles of *Calliandra portoricensis* leaf extracts. *Journal of ethnopharmacology* 22(1): 63-71.
37. Yamahara J, Kubomura Y, Miki K, Fujimura H (1987) Anti-ulcer action of *Panax japonicus* rhizome. *Journal of ethnopharmacology* 19(1): 95-101.
38. Harju E (1984) Guar gum benefits duodenal ulcer patients by decreasing gastric acidity and rate of emptying of gastric contents 60 to 120 minutes postprandially. *The American Surgeon* 50(12): 668-672.
39. Al Harbi MM, Qureshi S, Raza M, Ahmed MM, Afzal M, et al. (1997) Gastric antiulcer and cytoprotective effect of *Commiphora molmol* in rats. *Journal of Ethnopharmacology* 55(2): 141-150.
40. Srivastava S, Lal VK, Pant KK (2012) Polyherbal formulations based on Indian medicinal plants as antidiabetic phytotherapeutics. *Phytopharmacology* 2(1): 1-15.
41. Parasuraman S, Thing GS, Dhanaraj SA (2014) Polyherbal formulation: Concept of ayurveda. *Pharmacognosy reviews* 8(16): 73-80.
42. Parasuraman S, Kumar EP, Kumar A, Emerson SF (2010) Anti-hyperlipidemic effect of triglize, a polyherbal formulation. *Int J Pharm Pharm Sci* 2(3): 118-122.
43. Gupta H, Shakya MK, Sharma GK (2022) Evaluation of Anti-ulcer activity of Polyherbal preparation in Ulcerogenic Wistar rats. *Research Journal of Pharmacy and Technology* 15(8): 3471-3474.

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