

Herbal Potential for Treatment of Peptic Ulcer by Coconut

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Abstract - Gastric ulcer is a common disorder of the digestive system. Current therapeutic regimens largely rely on Western medicine. However, numerous studies have demonstrated that herbal medicines can effectively treat gastric ulcer in humans and various animal models via divergent mechanisms.

This review updates the efficacy and safety of herbal medicines in treating gastric ulcer, and the mechanisms of their action in humans and animal models. Studies have demonstrated that the efficacy of herbal medicines is comparable or superior to that of drugs such as omeprazole or cimetidine in humans and animal models, and herbal medicines display fewer adverse effects.

The mechanisms by which herbal medicines benefit gastric ulcer include stimulation of mucous cell proliferation, anti-oxidation, and inhibition of gastric acid secretion and H(+)/K(+)-ATPase activity. Some herbal medicines also exhibit antimicrobial properties. Utilization of herbal medicines could be a valuable alternative to treat gastric ulcer in humans effectively, with few adverse effects. Gastric ulcer is a common digestive disorder. Herbal medicines can effectively treat gastric ulcers in humans and various animal models. The efficacy of herbal medicines is comparable or superior to drugs such as omeprazole or cimetidine, and herbal medicines display fewer adverse effects.

The mechanisms by which herbal medicines benefit gastric ulcer include stimulation of mucous cell proliferation, anti-oxidation, and inhibition of gastric acid secretion as well as H(+)/K(+)-ATPase activity.

Peptic ulcer is a gastrointestinal disorder due to an imbalance between the aggressive factors like acid, pepsin, Helicobacter pylori and defensive factors like bicarbonate secretion, prostaglandins, gastric mucus, innate resistance of the mucosal cell factors. Normally peptic ulcer develops when aggressive factors overcome the defensive factors. Peptic ulcer can be categorized on the basis of location and on the severity of disease. Basically, word "peptic" is derived from Greek term "peptikos" whose meaning is related to digestion.

Various reports indicate that old age group patients are more prone to gastric ulcer.

Younger individuals have a higher risk of duodenal ulcers. A number of drugs including proton pump inhibitors and H₂ receptor antagonists are available for the treatment of peptic ulcer, but clinical evaluation of these drugs has shown incidence of relapses, side effects, and drug interactions. But therapeutic uses of plants are safe, economical & effective as their ease of availability. Different plants are rich sources of medicines. Currently, increasing health concern urged the researchers to revitalize the natural products and to alleviate the diseases without harming the body.

Ulcer is a common gastrointestinal disorder which is seen among many people. It is basically an inflamed break in the skin or mucous membrane lining the elementary tract. Ulceration occurs when there is a disturbance of normal equilibrium caused by either enhanced aggression or diminished mucosal resistance. It may occur due to the regular usage of drugs, irregular food habits, stress and so forth. Peptic ulcers are a broad term that includes ulcers of the digestive tract in the stomach or the duodenum.

INTRODUCTION

The most common cause of stomach ulcer is a bacterium called Helicobacter pylori. Similarly, ulcers may be caused by overuse of pain killers, such as aspirin and non-steroidal anti-inflammatory drugs such as ibuprofen, naproxen etc.

A peptic ulcer is essentially a wound that affects the mucous membrane of the digestive tract. Different names are given to ulcers depending on where they are located (gastric ulcers are located in the lining of the stomach, duodenal ulcers are located in the duodenum). Many times, in clinical practice we see a lack of digestive enzymes to be the root cause of the ulcer. A lack of enzymes can create an imbalanced

environment leading to a growth of *H.pylori* which can infect the mucous membrane.

The pathophysiology of peptic ulcer disease involves an imbalance between offensive (acid, pepsin, *Helicobacter pylori*) and defensive factors (mucin, prostaglandin), bicarbonate, nitric oxide and growth factors.

Peptic ulcers are once believed to be caused by spicy food and stress; these have been found merely to be aggravating factors and the real cause have been found by research to include bacterial infection (*Helicobacter pylori*) . The gram-negative bacterium *Helicobacter pylori* remains present between the gastric epithelium and mucous layer and is strategically designed to live within the aggressive environment of the stomach.

Peptic ulcers are broad term that includes ulcers of digestive tract in the stomach or the duodenum. The formation of peptic ulcers depends on the presence of acid and peptic activity in gastric juice plus a breakdown of mucosal defenses. A number of synthetic drugs are available to treat ulcers.

But these drugs are expensive and are likely to produce more side effects when in compared to herbal medicines. The ideal aims of treatment of peptic ulcer disease should be to relieve pain, heal the ulcers and delay ulcer recurrence. In this review attempts have been made to know about some common medicinal plants

proximal segments of the stomach. Peptic ulcer is one of the world's major gastrointestinal disorders and affecting 10% Of the world population. About 19 out of 20 peptic ulcers are duodenal.

In the Indian pharmaceutical industry, antacids and antiulcer drugs r of the market in this modern area also of the world population still used herbal medicine mainly in developing countries, for primary health care because of better compatibility with the human body, and lesser side effects.

Preliminary photochemical screening of this medicinal plant identified in the presence of important secondary metabolites like flavonoids and tannins which are the active principle of anti-ulcer activity.

Materia medica provides lots of information about ethno medicinal herbs, which are valuable as antiulcer agents and their use experimentally evaluated and proved by many researchers for its suggested that medicinal plant those are evidently reported for its antiulcer activity. The pain is described to be sharp

growing and burning. Depending on the where the ulcer is located the symptoms of ulcer can vary.

In case of bleeding ulcer accompanied by vomiting or dark to black stool.

Typically, a peptic ulcer is worse during the night or when a person lays down.

PATHOGENESIS OF PEPTIC ULCER

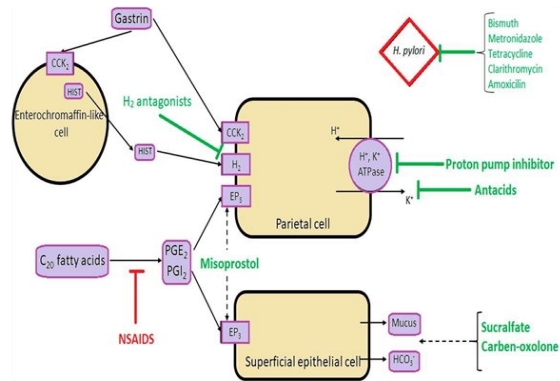
H. pylori, which remains one of the most common causes of peptic ulcer disease. *H. pylori* causes epithelial cell degeneration and injury, which is usually more severe in the antrum, by the inflammatory response with neutrophils, lymphocytes, plasma cells, and macrophages. The mechanism by which *H. pylori* induces the development of different types of lesions in the gastroduodenal mucosa is not fully explained. *H. pylori* infection can result in either hypochlorhydria or hyperchlorhydria. thus determining the type of peptic ulcer.

The main mediators of *H. pylori* infection are cytokines that inhibit parietal cell secretion, but *H. pylori* can directly affect the H^+/K^+ ATP ase α -subunit, activate calcitonin gene-related peptide (CGRP) sensory neurons linked to somatostatin, or inhibit the production of gastrin . Although the formation of gastric ulcers is associated with hyposecretion, 10–15% of patients with *H. pylori* infection have increased gastric secretion caused by hypergastrinemia and reduced antral somatostatin content . This leads to increased histamine secretion, and subsequently the increased secretion of acid or pepsin from parietal and gastric cells. Additionally, the eradication of *H. pylori* leads to a decrease in gastrin mRNA expression and an increase in somatostatin mRNA expression.

In the remaining majority of patients, gastric ulcers are associated with hypochlorhydria and mucosal atrophy. The main mechanism of NSAID-associated damage of the gastroduodenal mucosa is the systemic inhibition of constitutively expressed cyclooxygenase-1 (COX-1), which is responsible for prostaglandin synthesis, and is associated with decreased mucosal blood flow, low mucus and bicarbonate secretion, and the inhibition of cell proliferation. NSAIDs inhibit the enzyme reversibly in a concentration-dependent manner. The co-administration of exogenous prostaglandins and cyclooxygenase-2 (COX-2)-selective NSAIDs use reduces mucosal damage and

the risk of ulcers . However, the different physicochemical properties of NSAIDs cause differences in their toxicity . NSAIDs disrupt mucus phospholipids and lead to the uncoupling of mitochondrial oxidative phosphorylation, thus initiating mucosal damage. When exposed to acidic gastric juice (pH 2), NSAIDs become protonated and cross lipid membranes to enter epithelial cells (pH 7.4), where they ionize and release H⁺ . In that form, NSAIDs cannot cross the lipid membrane, and are trapped in epithelial cells, leading to the uncoupling of oxidative phosphorylation, decreased mitochondrial energy production, increased cellular permeability, and reduced cellular integrity. Patients who have a history of peptic ulcers or hemorrhage, are over the age of 65, also use steroids or anticoagulants, and take high doses or combinations of NSAIDs are at the highest risk for acquiring NSAID-induced ulcers.

Many strategies are available for the prevention of NSAID and aspirin-associated gastroduodenal ulcers and their complications, such as the co-therapy of NSAIDs with a PPI, H₂ receptor antagonist, or misoprostol; the use of COX-2-selective NSAIDs.



pathophysiological mechanisms involved in the development of peptic ulcer disease, and the sites of action of the most commonly used pharmacological options in the treatment of peptic ulcer disease. CCK2 = Cholecystinin Receptor; PGE2 = Prostaglandin E2; PGI2 = Prostaglandin I2; EP3 = Prostaglandin E receptor 3; HIST = Histamine.

H. pylori induces the development of different types of lesions in the gastroduodenal mucosa is not fully explained. H. pylori infection can result in either hypochlorhydria or hyperchlorhydria, thus determining the type of peptic ulcer. The main mediators of H. pylori infection are cytokines that inhibit parietal cell secretion, but H. pylori can directly

affect the H⁺/K⁺ ATPase α -subunit, activate calcitonin gene-related peptide (CGRP) sensory neurons linked to somatostatin, or inhibit the production of gastrin]. Although the formation of gastric ulcers is associated with hyposecretion, 10–15% of patients with H. pylori infection have increased gastric secretion caused by hypergastrinemia and reduced antral somatostatin content.

This leads to increased histamine secretion, and subsequently the increased secretion of acid or pepsin from parietal and gastric cells. Additionally, the eradication of H. pylori leads to a decrease in gastrin mRNA expression and an increase in somatostatin mRNA expression [18]. In the remaining majority of patients, the main mechanism of NSAID-associated damage of the gastroduodenal mucosa is the systemic inhibition of constitutively expressed cyclooxygenase-1 (COX-1), which is responsible for prostaglandin synthesis, and is associated with decreased mucosal blood flow, low mucus and bicarbonate secretion, and the inhibition of cell proliferation. NSAIDs inhibit the enzyme reversibly in a concentration-dependent manner.

The co-administration of exogenous prostaglandins and cyclooxygenase-2 (COX-2)-selective NSAIDs use reduces mucosal damage and the risk of ulcers [19]. However, the different physicochemical properties of NSAIDs cause differences in their toxicity [20]. NSAIDs disrupt mucus phospholipids and lead to the uncoupling of mitochondrial oxidative phosphorylation, thus initiating mucosal damage.

TRETMENT

The most common cause of stomach ulcer is a bacterium called Helicobacter pylori. Similarly, ulcer may cause by overuse of pain killers, such as aspirin and non-steroidal anti-inflammatory such as ibuprofen, naproxen. peptic ulcer is essentially a wound that affects the mucous membrane of the digestive tract. Different names are given to ulcers depending on where they are located (gastric ulcers are located in the lining of the stomach , duodenal ulcers are located in the duodenal). Many times, in clinical practice we see a lack of digestive enzymes to be the root cause of the ulcer a lack of enzymes can create an imbalanced environment leading to a growth of H.pylori which can infect the mucous membrane. The

patho physiology of peptic ulcer disease involves an imbalance between offensive (acid, pepsin, Helicobacter pylori) and defensive factors (mucin, prostaglandin), bicarbonate, nitric oxide and growth factors. Peptic ulcers are once believed to be caused by spicy food and stress; these have been found merely to be aggravating factors and the real cause have been found by research to include bacterial infection (Helicobacter pylori). The gram-negative bacterium Helicobacter pylori remains present between the gastric epithelium and mucous layer and is strategically designed to live within the aggressive environment of the stomach

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For prevention you will want to consume foods that increase digestive function bitter greens as a salad and warming spices such as turmeric and garlic can be helpful, if there is systematic inflammation and heat, then focusing on eliminating and draining heat systemically will be a strong focus. Cooling foods, berries, and healthy fats should be emphasized while heating foods such as alcohol and excessive hot peppers should be eliminated.

THE NATURAL HERBS USED IN NATURAL TREATMENT OF ULCERS

1.CABBAGE

Cabbage is a best remedy for a stomach ulcer, being a lactic acid food; cabbage helps to produce secretion of amino acid that stimulates blood flow to the stomach lining. This in turn helps strengthen the stomach lining and heal the ulcer. Cabbage contains a good amount of vitamin-C. Which has been found to be particularly beneficial for patient with H.pylori infection. Also experiments indicate that fresh carrot juice contains an anti-peptic ulcer factor (vitamin). Method of uses; Cut one half of a raw head of cabbage and two carrots into small pieces and put them in a blender to extract the juice. Drink one half of this juice

before each meal and at bedtime. Repeat daily for a few weeks. Be sure to use fresh juice each time.

2.BANANAS

For stomach ulcer treatment both ripe and unripe bananas are very effective.

There are certain antibacterial compounds in banana that inhibit the growth of ulcer causing H.pylori. Banana also protects the system by wiping out the acidity of gastric juice. This helps reduce inflammation and also strengthens the stomach lining. Method of uses: To treat an ulcer eat at least three ripe bananas a day. If you do not eat banana, you can make banana milk shakes. Alternatively, peel two or three bananas and cut them into thin slices. Put the slices in the dried banana pieces in the sun until they become dried. Grind the dried banana pieces into a fine powder. Mix together two-teaspoon of honey. Take this mixture three times a day for about a week.

3.HONEY

Raw honey has potent healing properties that help a lot in the treatment of stomach ulcer. An enzyme called Glucose oxidase in honey produces hydrogen peroxide, which in turn kills harmful bacteria that cause ulcer. Plus it soothes and reduces the inflammation of stomach lining. Method of use: Take two tablespoons of raw honey daily early in the morning on an empty stomach. It will help clean the bowl strengthen the stomach lining and also treat the stomach ulcer. Taking a tablespoon of honey in the morning and at night to calm a fiery belly. Spread it on toast or a cracker to keep it in the stomach longer. Because H. pylori is slow growing, be sure to keep up your honey regimen until ulcer symptoms are long gone

4.COCONUT

Coconut is very good for people, suffering from stomach ulcer because of its anti-bacterial qualities. It kills the bacteria that cause ulcer. Coconut milk and coconut water have anti-ulcer properties. Coconut milk and water are both said to have antibacterial qualities that help kill ulcer-causing bacteria. Drink a cup of fresh coconut milk. Coconut is very good and essential for who are suffering from peptic ulcers because of its antibacterial qualities. It kills the bacteria that cause ulcers. Moreover, coconut milk and coconut water have anti-ulcer properties.

Drink a few cups of fresh coconut milk or tender coconut water daily. Also, eat the kernel of the tender coconut. Follow this treatment for at least up to one week to get positive results.

Alternatively, take one tablespoon of coconut oil in the morning and another at night for one week. As coconut oil is mainly composed of the medium-chain fatty acids, it can be easily digested.

COCONUT OIL USED IN PEPTIC ULCER

COCONUT OIL NAD HOW IT KILLS BACTERIA

Coconut oil is an excellent anti-biotic. It kills bacteria and fungi on contact. Coconut oil is made of saturated fatty acids of the medium length variety. Its major saturated acid is called LAURIC ACID. The lauric acid invades the cell wall of the bacteria to destroy them. Coconut oil helps treat and prevent ulcers, without doing any harm to the body.

Ulcers can occur anywhere along your digestive tract] and in or near your mount. Unlike before, it ‘now known that a bacterium called helicobacter pylori causes pylori causes peptic or stomach ulcers. Gastric ulcers are brought about by bacteria burrowing into the stomach’s wall antibiotics are sometimes prescribed to deal with ulcer. Problem is antibiotics can lead to health problems by killing even bacteria. Coconut oil provides a naturally effective and totally harmless approach. H. pylori, herpes and streptococcus are associated with various types of ulcers and are all killed by medium chain fatty Acids generally found in coconut oil.

Coconut oil possesses cancer-fighting properties as the immune system in seeking out and destroying cancerous cells and H. pylori bacteria.

Coconut oil is an excellent “antibiotic”. It can kill and destroy bacteria and fungi through contact. Because coconut oil is made of saturated fatty acids, it invades the cell wall of the bacteria to destroy them. Most of all, the stomach ulcers are caused by the infection of the bacteria

H. pylori. Therefore, coconut oil can be the answer to kill this bacteria that causes stomach ulcers. Without doing any harm to your body, coconut oil helps treat and prevent ulcers by destroying a wide spectrum of disease-causing organisms. Ulcers can occur anywhere along your digestive tract, and in or near your mouth. Unlike before, it’s now known that a bacterium called Helicobacter pylori causes peptic or

stomach ulcers. Gastric ulcers are brought about by bacteria burrowing into the stomach’s wall. Antibiotics are sometimes prescribed to deal with ulcers. The problem is, antibiotics can lead to other health problems by killing even friendly bacteria.

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CAUSES OF PEPTIC ULCER DISEASE

Helicobacter pylori infection	NSAIDs
Gram-negative, motile spiral rod found in with peptic ulcer disease	5 to 20 percent of patients who use NSAIDs over long periods develop peptic ulcer disease
	NSAID-induced ulcers and complications are More common in older patients, patients with a history of ulcer or gastrointestinal bleeding.

1.H. pylori
Bacteria adhere to the gastric mucosa; the presence of an outer inflammatory protein and a functional cytotoxin-associated gene island in the bacterial chromosome increases virulence and probably ulcerogenic potential. Patients with H. pylori infection have increased resting and meal-stimulated gastrin levels and decreased gastric mucus production and duodenal mucosal bicarbonate secretion, all of which favor ulcer formation.

2.NSAIDs
NSAIDs are the most common cause of peptic ulcer disease in patients without H. pylori infection. Topical

effects of NSAIDs cause submucosal erosions. In addition, by inhibiting cyclooxygenase, NSAIDs inhibit the formation of prostaglandins and their protective cyclooxygenase-2-mediated effects. The annual risk of a life-threatening ulcer-related complication is 1 to 4 percent in patients who use NSAIDs long-term, with older patients at the highest risk. NSAID use is responsible for approximately one half of perforated ulcers, which occur most commonly in older patients who are taking aspirin or other NSAIDs for cardiovascular disease or arthropathy.

3.A bacterium –

An infection of bacteria called helicobacter pylori can cause ulcers to develop. Found in the mucous layer that protects the tissues that line the stomach and small intestine, this bacteria can cause inflammation of the stomach’s inner layer, resulting in an ulcer. Helicobacter pylori has been found to be passed from person to person or through food and water.

4.Regular use of certain pain medications –

Some pain relievers including aspirin and ibuprofen can irritate or inflame the lining of the stomach and small intestine, resulting in an ulcer.

5.signs and Symptoms of Peptic Ulcers

Many people with peptic ulcers upper abdominal pain is the most common symptom.

- Extend from your navel up to your breastbone
- Feel worse when your stomach is empty
- Feel better temporarily when you eat certain foods or take an antacid
- Get worse at night
- Come and go for days or weeks

6.Other symptoms of peptic ulcers may include:

- Nausea
- Vomiting of red or dark blood
- Feeling
- Bloody, black, or tar-like stools
- Unexplained weight loss
- Changes in your appetite

7.Diagnosis

Laboratory tests for H. pylori. Your doctor may recommend tests to determine whether the bacterium H. pylori is present in your

body. He or she may look for H. pylori using a blood, stool or breath test. The breath test is the most accurate.

For the breath test, you drink or eat something that contains radioactive carbon. H. pylori breaks down the substance in your stomach. Later, you blow into a bag, which is then sealed. If you're infected with H. pylori, your breath sample will contain the radioactive carbon in the form of carbon dioxide.

If you are taking an antacid prior to the testing for H. pylori, make sure to let your doctor know. Depending on which test is used, you may need to discontinue the medication for a period of time because antacids can lead to false-negative results.

8.Endoscopy.

Your doctor may use a scope to examine your upper digestive system (endoscopy). During endoscopy, your doctor passes a hollow tube equipped with a lens (endoscope) down your throat and into your esophagus, stomach and small intestine. Using the endoscope, your doctor looks for ulcers.

If your doctor detects an ulcer, a small tissue sample (biopsy) may be removed for examination in a lab. A biopsy can also identify whether H. pylori is in your stomach lining.

Your doctor is more likely to recommend endoscopy if you are older, have signs of bleeding, or have experienced recent weight loss or difficulty eating and swallowing. If the endoscopy shows an ulcer in your stomach, a follow-up endoscopy should be performed after treatment to show that it has healed.

9.Diet

Choose These Foods	Avoid These Foods
• Fruits	• Alcohol
• Vegetables	• Coffee (regular, decaf)
• lean beef	• Caffeinated foods and drinks
• Fish and seafood	• Milk or cream
• Eggs	• Fatty meats
• Fermented dairy foods like kefir or yogurt	• Fried or high-fat foods
• Healthy fats like olive oil, avocados, and nuts	• Heavily spiced foods
• Whole and cracked grains	• Salty foods
• Green tea	• Citrus fruits and juices
	• Tomatoes/tomato products
	• Chocolate

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