REVIEW

HERBS USED TO TREAT PEPTIC ULCER: A SYSTEMIC REVIEW

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ABSTRACT:

Any open wound is known as Ulcer. Peptic ulcer, which is also known as PUD or peptic ulcer disease, is an ulcer (mucosal erosions equal to or greater than 0.5 cm) of an area of the gastrointestinal tract that is usually acidic and results extreme pain. Symptoms include abdominal pain with severity relating to mealtimes, after around 3 hours of taking a meal; starvation; oily and spicy food intake; bloating and abdominal fullness; nausea, and copious vomiting; loss of appetite and weight loss etc. There are many herbs, nutrients, and plant products that have been found to play a role in protecting or helping to heal stomach and peptic ulcers. Few human trials are available, but many have shown good potential in animal or in vitro studies. And the present study was aimed to collect information on various herbs which are used in treating Peptic Ulcer in various parts of the world, depending upon the data's provided by various researchers.

KEY WORDS: Peptic Ulcer, Helicobacter pylori, Gastrin, PPI, H_2 blocker, PG Analogue, Herbs.

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INTRODUCTION:

Any internal open wound is called Ulcer. Peptic ulcer, also known as PUD or peptic ulcer disease, is an ulcer (defined as mucosal erosions equal to or greater than 0.5 cm) of an area of the gastrointestinal tract that is usually acidic and thus extremely painful. Symptoms includes abdominal pain, classically epigastric with severity relating to mealtimes, after around 3 hours of taking a meal (duodenal ulcers are classically relieved by food, while gastric ulcers are exacerbated by it); bloating and abdominal fullness, nausea, and copious vomiting; loss of appetite and weight loss; hematemesis (vomiting of blood); this can occur due to bleeding directly from a gastric ulcer, or from damage to the oesophagus from severe/continuing vomiting; melena (tarry, foul-smelling faeces due to oxidized iron from haemoglobin). Rarely, an ulcer can lead to a gastric or duodenal perforation, which leads to acute peritonitis. This is extremely painful and requires immediate surgery. A history of heartburn, gastroesophageal reflux disease (GERD) and use of certain forms of medication can raise the suspicion for peptic ulcer. Medicines associated with peptic ulcer include NSAID (non-steroid anti-inflammatory drugs) that inhibit cyclooxygenase, and most glucocorticoids (e.g. dexamethasone and prednisolone).

In patients over age of 45-50 with more than two weeks of the above symptoms, the odds for peptic ulceration are high enough to warrant rapid investigation by EGD 2,3The timing of the symptoms in relation to the meal may differentiate between gastric and duodenal ulcers: A gastric ulcer would give epigastric pain during the meal, as gastric acid production is increased when food enters the stomach. Symptoms of duodenal ulcers would initially be relieved by a meal, as the pyloric sphincter closes to concentrate the stomach contents, therefore acid is not reaching the duodenum. Duodenal ulcer pain would occurs mostly 2-4 hours after the meal intake, when the stomach begins to release digested food and acid to digest into the duodenum. Also, the symptoms of peptic ulcers may vary with the location of the ulcer and the patient's age. Furthermore, typical ulcers tend to heal and cure and as a result the pain may occur for few days and weeks and then disappear. Usually, children and the elderly do not develop any symptoms unless complications have arisen.[1]

ETHIOLOGY[2]:

Helicobacter pylori: A major causative factor (60% of gastric and up to 90% of duodenal ulcers) is chronic inflammation due to *Helicobacter pylori* that colonizes the antral mucosa. The immune system is unable to clear the infection, despite the appearanceof antibodies. Thus, the bacterium can cause achronic active gastritis (type B gastritis), resulting ina defect in the regulation of gastrin production by thatpart of the stomach, and gastrin secretion can either be decreased (most cases) resulting in hypo- or achlorhydria or increased. Gastrin stimulates the production of gastric acid by parietal cells and, in *H.pylori* colonization responses that increase gastrin, the increase in acid can contribute to the erosion ofthe mucosa and therefore

NSAIDs: Another major cause is the use of NSAIDs. The gastric mucosa protects itself from gastric acid with a layer of mucous, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (COX-1), which is essential for the biosynthesis of these prostaglandins. COX-2 selective anti-inflammatories (such as celecoxib or the since withdrawn rofecoxib) preferentially inhibit cox-2, which is less essential in the gastric mucosa, and roughly halve the risk of NSAID-related gastric ulceration.

Stress: Researchers also continue to look at stress as a possible cause, or at least complication, in thedevelopment of ulcers. There is debate as to whether psychological stress can influence the developmentof peptic ulcers.

Gastrinomas (Zollinger Ellison syndrome): it is a rare gastrin-secreting tumours, also cause multiple and difficult to heal ulcers.

Smoking: Studies show that cigarette smoking can increase a person's chance of getting an ulcer. Smoking also slows the healing of existing ulcers and contributes to ulcer recurrence.

Caffeine: Many beverages and foods that contain caffeine, which can stimulate acid secretion in the stomach. This canaggravate an existing ulcer, but

the stimulation of stomach acid can't be attributed solely to caffeine.

Alcohol: While a link hasn't been found between alcohol consumption and peptic ulcers, ulcers are more common in people who have cirrhosis of the liver, a disease often linked to heavy alcohol consumption daily.

Genetic factor: People with blood group O appear to be more prone to develop peptic ulcer than thosewith other blood groups. Genetic influences appear to have greater role in duodenal ulcers as evidence bytheir occurrence in family's monozygotic twins and association with HLB-B5 antigen.

Spicy & Alkaline food: Food enriched with spices liberates more gastric juice to digest. This over acid secretion damages mucosal cell wall and causes peptic ulcer. More alkaline food intake occurs also peptic ulcer because of more bicarbonate ions.

Regulation of Acid secretion:

The regulation of acid secretion is important in the pathogenesis of peptic ulcer, and constitutes a particular target for drug action. The secretion of the parietal cells is an isotonic solution of HCl (150 mmol/l) with a pH less than 1, the concentration of hydrogen ions being more than a million times higher than that of the plasma. The Cl− isactively transported into canaliculi in the cells that communicate with the lumen of the gastric glands and thus with the stomach itself. This Cl_2secretion is accompanied by K+, which is then exchanged for H+ from within the cell by a K+-H+-ATPase (the 'proton pump', Fig. 29.1). Carbonic anhydrase catalyses the combination of carbon dioxide and water to give carbonic acid, which dissociates into H+ and bicarbonate ions. The latter exchanges across the basal membrane of the parietal cell for Cl–.[3]

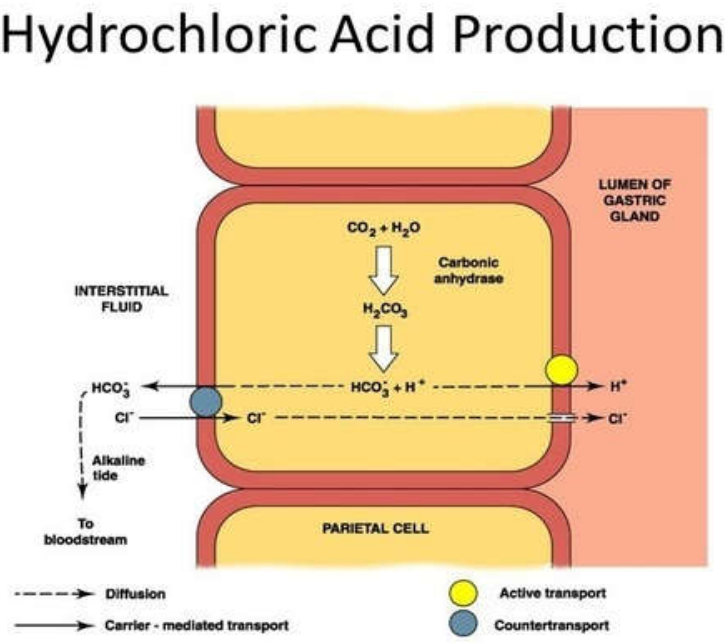

Fig.1: Production of Hcl

SYMPTOMS:

Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be lifethreatening. It occurs when the ulcer ruptures one of the blood vessels, such as the gastroduodenal artery. Perforation (a hole in the wall) often leads to catastrophic consequences. Erosion of the gastrointestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain. Posterior wall perforation leads to pancreatitis; pain in this situation often radiates to the back. Penetration is when the ulcer continues

into adjacent organs such as the liver and pancreas. Scarring and swelling due to ulcers causes narrowing in the duodenum and gastric outlet obstruction. Patient often presents with severe vomiting. Cancer is included in the differential diagnosis (elucidated by biopsy), Helicobacter pylori as the etiological factor making it 3 to 6 times more likely to develop stomach cancer from the ulcer.[4]

PLANT USED FOR PEPTIC ULCER:

There are many herbs, nutrients, and plant products that have been found to play a role in protecting or helping to heal stomach and peptic ulcers. Few human trials are available, but many have shown good potential in animal or in vitro studies. A variety of botanical products have been reported to possess antiulcer activity but the documented literature has centred primarily on pharmacological action. The plants are –

SHATAVARI:

Biological source: The plant contains dried root & leaves of *Asparagus racemosus*belonging to the family *Asparagaceae*.

Parts used: Dried root& leaves

Chemical constituent:

Shatavari roots contain 4 steroid saponin, shatavarin I-IV (0.2%). Shatavarin I is the major glycoside with3 glucose and rhamnose moieties attached to sarapogenin,whereas
in shatavarin IV 2 glucose and 1-rhamnose moieties are attached. Flowers and fruits contain quercetin, rutin and hyperoside, while leaves contains diosgenin and hyperoside, while leaves contain diosgenin and quercetin.[5]

Use in Peptic Ulcer:

Shatavari act on the ulcer bed by covering the area and also it decreases the activity of proton pump by binding with receptors and it is having acid neutralizing property. Root is boiled in milk and the milk is administered to relieve bilious dyspepsia and Gastric Ulcer.[6]

Fig.2: Shatavari

Fig.3: Aloe

ALOE VERA:

Biological source: Aloe contains fleshy leaf of *Aloe barbadensis* is belonging to the family *Liliaceae.*

Parts used **:** Fleshy leaf

Chemical Constituents:

Aloe contain anthraquinone derivatives (10% to 40%) like aloin, mucilage (30%), resinous substances (16% to 63%) like aloesin and aloesone, sugars (about 25%), polysaccharides like acemannan and betamannan, fatty acids and cholesterol, campe-sterol, P-sistosterol, glycoproteins (aloctins A and B), lectins, a gibberellin-like substance, enzymes such as cyclo-oxygenase and bradykininase, together with other compounds such as lupeol, salicylic acid, urea, cinnamic acid, phenol, sulphur, magnesium lactate, salicylates, and amino acids. Aloin (barbaloin) is an impure mixture of barbaloin A and barbaloin B, which inter-convert through the anthranol form.[7]

Use in Peptic Ulcer:
Aloe vera juice is used for Ulcer healing agent because of Aloin binds with receptor of paraietal cell and create inhibitory effect of gastric juice secretion. Beside this aloe having healing effect to the ulcer bed and having a latex type form, it create a protective bed on ulcer. [8]

AMLA:

Biological source: Amla contains dried ripe fruit of *Embelica officinalis* belonging to family *Euphorbiaceae.*
Parts used : Dried ripe fruit.

Chemical Constituent:
The fruit is a very rich source of vitamin C. It was proposed that superior effect of the mistaken "vitamin C" component is actually the more stable and potent anti-oxidant effect of the tannins that appeared to be the vitamin. A repeated laboratory test showed that every 100g of fresh fruit provides 470 - 680mg of vitamin C. The vitamin value of Amla increased further when the juice was extracted from the fruit. The dehydrated berry provided 2428 - 3470mg of vitamin C per 100g. Its mineral and vitamin contents include calcium, phosphorous, iron, carotene, Thiamine, riboflavin, and niacin. The seeds of the Indian gooseberry contains a fixed oil, phosphatides, and an essential oil. The fruits, bark, and the leaves of this tree are rich in tannin.[9]

Use in Peptic Ulcer:
Amla is having very good anti-inflammatory activity on ulcer and it also having healing property to the damaged wall. It is natural antibacterial agent and it regulated the hyperacidity.[10]

Fig.4: Amla

Fig.5: Ashwagandha

ASHWAGANDHA:

Biological source: Ashwagandha contains dried root and stem bases of *Withania somnifera* belonging to the family *Solanaceae.*
Parts used: Dried root and stem base.

Chemical constituents:
The main constituents of Ashwagandha are alkaloids and steroidal lactones. Among the various alkaloids, withanine is the main constituent. The other alkaloids are somniferine, somnine, somniferinine, withananine, pseudowithanine, tropine, pseudo-tropine, cuscohygrine, anferine and anhydrine. Two acyl steryl glucoside viz. sitoindoside VII and sitoindoside VIII have been isolated from root. The leaves contain steroidal lactones, which are commonly called withanolides. The withanolides have C28 steroidal nucleus with C9 side chain, having six membered lactone ring.[11]

Use in Peptic Ulcer:
Ashwagandha treats on the gastric ulcer three mechanism-

- Reducing the amount of acid secreted in the stomach.
- Increasing secretions that counteract the acid levels (and balance the pH).
- Act as physical protective barriers of the lining.[12]

PAPAYA:

Biological source: Papaya contains unripe fruit of *Carica papaya* belonging to the family *Caricaceae*.

Parts used: Unripe fruit

Chemical constituents:

Papaya is rich inenzyme called papain. It also contains Carotenoidsnamely β –carotene, kryptoxanthin and zeaxa in thefruits, alkaloids from the leaves arecarprine,carpinine. Monoterpenoids includes 4-terpiniol, linalool and linalool oxides. WhereasFlavonoids marks their presence viaquercetin, myrecetin and kaemferol in the shoot. Thefruits also contain potassium, calcium, magnesium, copper, zinc, iron.[13]

Use in Peptic Ulcer:

Papaya has been used for digestion problems. This product should not be used for intestinal parasite infections because it may be ineffective. The effects of Carica papaya Linn on exogenous ulcer and histamine-induced acid secretion were studied in rats. The latex of the unripen fruit of C. papaya was effective in protecting the exogenous ulcer. It significantly lessened the acid secretion induced by intravenous infusion of histamine in chronic gastric fistulae rats. Crystalline papain was also effective in protecting the exogenous ulcer and in decreasing the histamine-induced acid secretion in rats. The conclusion is that papain is the active principle in C. Papaya that exerts the ulcer-protective effect.[14]

Fig.6: Papaya

Fig.7: Banana

BANANA:

Biological source : Banana contains fruit and whole plant of *Musa paradisca* belonging to the family *Musaceae.*

Parts used : Fruit and Whole plant

Chemical constituents:

Plant contains glycoside sitoindoside IV, 14-methyl-9Beta, and 19-cyclo-5-ergost- 24 (28)-en-3Beta-ol (I). Flowers contain diglycosides of delphinidine and leucocyanidin. Unripe fruit contains starch. Fruit contains sugars, vitamin B6 & C, Folate, Fibre, proteins albumin and globulin. Flesh of mature fruit contains tannin, also contain serotonin and norepinephrine in addition to dopamine and an unidentified catecholamine. Peel contains tannins.[15]

Role in peptic ulcer:

Banana is a natural antacid and astringent, which protects against ulcer development. Leucocyanidin which is a component of banana, also increase the thickness of the mucous membrane of stomach. It protects against potential damage of stomach wall for acids.[16]

Fig.8: Ginseng

GINSENG:

Biological source: Ginseng contains dried root of *Panax ginseng* belonging to the family *Araliaceae.*

Parts used : Dried root

Chemical constituents:

The main active ingredient in the Panax species are a group of dammarrane-type triterpenoid glycosides. They are referred to as saponins and termed as ginsenosides.In Russia they termed as panaxosidess.These are in the ginseng root. There are more than 30 ginsenoside.One of them is an oleanolic acid derivative. It is the type and composition of the Ginsennosides which give their different qualities. There are 8 main ginsenosides and the composition in American and Asian is quite different. There are many more ginsenosides in American ginseng than there are in Asian ginseng. The most abundant ginsenoside in both species is ginsenosides Rb1.This ginsenosides is reported to have sedative effect. Ginsenoside Rg1 is
Said to have a stimulant effect. The levels of Rg1 in Asian ginseng are much higher than in American Ginseng.Asian ginseng also contains ginsenosides RF and Rg2,whereas American ginseng is virtually devoid of these ginsenosides.Pseudoginsenosides F11 is noted in American ginseng,but it is almost absent from Asian ginseng. The root of ginseng contain resin,sugar,starch,mucilage, a saponin, a volatile oil and several steroids compound.It contain strong antioxidant and an another compound germanium.

Role in peptic ulcer:

Ginseng is containing Ginsenoside Rb1 which is an ulcer protective agent because it increases the mucous secretion on the stomach wall.[17]

CONCLUSION: The present study was aimed at the medicinal plants for the treatment of Gastric Ulcer. Some of them arealready reported as anti-ulcer drug, but for some still no work has been done and these are only used traditionally. It is high time efforts should be made to use the vast ethno-pharmacological knowledge ourtraditional practitioners have to develop safer herbal preparations, for the people which will be less toxic and cheaper than the Modern day Medicaments. In view of increasing popularity of alternative system of medicine, it is necessary to conduct research to support the therapeutic claim and also to ensure that the plants are given importance according to their therapeutic value, in modern herbal medicines. Safety is not a matter of concern for these plants as it has been proved over the years by their traditional use. The point where more study is needed is to develop Standard Procedures for Standardization of Herbals.[18]

DISCUSSION:

Nowadays the synthetic drugs are used for the treatment of peptic ulcer, which is having more side effects. But the herbs are also effectively act on peptic ulcer to cure up with less side effects. So, it is better to isolate the Active phytoconstituent for the peptic ulcer treatment as an herbal medicine that giving more potent action on peptic ulcer with decreased side effects.

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CONFLICT OF INTEREST REPORTED: NIL ; SOURCE OF FUNDING: NIL