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REVIEW OF PEPTIC ULCER DISEASE AND ITS TREATMENT OPTIONS

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ABSTRACT

Peptic ulcer is a chronic disease that affects up to 10% of the world's population. Composition Peptic ulcers depend on the presence of gastric juice pH and a decrease in mucosal immunity. Non-steroidal anti-inflammatory drugs (NSAIDs) and Helicobacter pylori (H. pylori) are two major factors that interfere with mucosal resistance to damage. Common treatment for peptic ulcers, such as proton pump inhibitors (PPIs) and histamine-2 (H2) receptor antagonists, have been shown side effects, relapses, and various drug interactions. On the other hand, medicinal plants as well their chemicals are useful in preventing and treating many diseases. So, this review identifies common medicinal plants that may be usedfor healing or prevention peptic ulcers.

KEYWORDS: NSAIDs, PPI, H. Pylori.

INTRODUCTION

Peptic ulcer is a painful ulcer or feature that focuses on the formation of the lining of the oesophagus, stomach, or duodenum, from the mucosa membrane to the submucosa and deep into the muscle layer. The moderate spread of peptic ulcer disease in general the population is 5-10 %, but recent epidemiological studies have shown a decrease in incidence, hospital admission rates, and death associated with peptic ulcer. This is very likely secondly in the introduction of new therapies and improved hygiene, which has led to a decline in prices Helicobacter pylori (H. pylori) infections.

Traditionally, mucosal disorders in patients with acid peptic acid are considered a natural effect with hypersecretory acid and dietary factors or stress. Risk factors for development of peptic ulcer including H.pylori infection, alcohol and tobacco use, non-steroidal use of anti- inflammatory drugs (NSAIDs), as well as Zollinger-Ellison syndrome. The main harmful substances of both gastric ulcers and duodenal H pylori infection and NSAID use. However, only a small one the number of people affected by H. pylori or NSAID users develop peptic ulcer disease, i.e., individual involvement is important at the onset of mucosal damage.

H. pylori-negative, NSAID-negative, and aspirin-negative peptic ulcer disease, separated as an idiopathic ulcer, it can be found in about one in five cases. It is caused by inequality among the factors that cause mucosal integrity and aggressive, but pathogenic insults Methods for the development of idiopathic peptic ulcer are unknown. Danish course showed that stress can increase the incidence of peptic ulcer. Other etiologist includes ischemia, drugs, and radiotherapy, antibiotics, histamine, eosinophilic infiltration, gastric bypass surgery, and metabolic disorders. Active polymorphisms of various cytokines are associated with peptic ulcers. For example, polymorphisms of interleukin 1 beta (IL1B) affect the production of mucosal interleukin 1β, which causes H infections. pylori-associated gastroduodenal.

On the other hand, the risk of complications of peptic ulcer increases fourfold for NSAID users, and twice as many aspirin users. Concomitant use of NSAIDs or aspirin with anticoagulants, corticosteroids, as well as selective serotonin reuptake inhibitors increase the risk of high gastrointestinal inflammation. bleeding. Although most people who use NSAIDs or aspirin have an H-infection. pylori simultaneously, their association in the pathogenesis of peptic ulcer disease remains controversial. Meta-analysis of observational studies has concluded that NSAIDs, aspirin use, and H infection. pylori to grow risk of peptic ulcer diseaseindependently.[1]

Causes of peptic ulcer

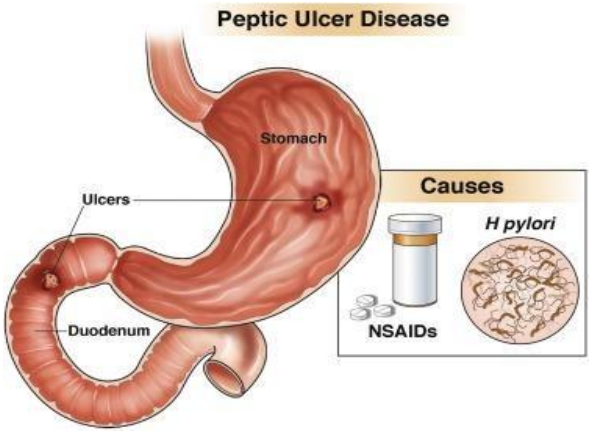
Peptic ulcers occur when acid in the digestive tract eats away at the inner surface of the stomachor small intestine. The acid can create a painful open sore that may bleed.[2]

H. pylori

Type of bacteria that cause a stomach infection and inflammation. Helicobacter pylori is one of the major causative factors of peptic ulcer disease. It secretes urease to create an alkaline environment, which is suitable for its survival. It expresses blood group antigen adhesin and outer inflammatory protein adhesin, which

enables it to attach to the gastric epithelium. The bacterium also expresses virulence factors such as CagA and PicB, which cause stomach mucosal inflammation. The VacA gene encodes for vacuolating cytotoxin, but its mechanism of causing peptic ulcers is unclear. Such stomach mucosal inflammation can be associated with hyperchlorhydria (increased stomach acid secretion)or hypochlorhydria (reduced stomach acid secretion). Inflammatory cytokines inhibit the parietal cell acid secretion. H. pylori also secretes certain products that inhibit hydrogen potassium ATPase; activate calcitonin gene-related peptide sensory neurons, which increases somatostatin secretion to inhibit acid production by parietal cells; and inhibit gastrin secretion. This reduction in acid production causes gastric ulcers. On the other hand, increased acid production at the pyloric antrum is associated with duodenal ulcers in 10% to 15% of H. pylori infection cases. In this case, somatostatin production is reduced andgastrin production is increased, leading to increased histamine secretion from the enterochromaffin cells, thus increasing acid production. An acidic environment at theantrum causes metaplasia of the duodenal cells, causing duodenal ulcers.

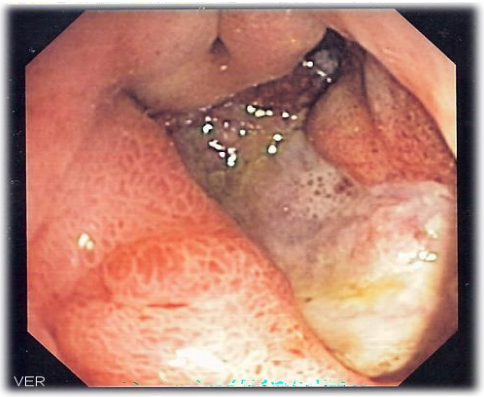
Human immune response toward the bacteria also determines the emergence of peptic ulcer disease. The human IL1B gene encodes for Interleukin 1 beta, and other genes that encode for tumour necrosis factor and Lymphotoxin alpha also play a role in gastric inflammation.[3]



NSAIDs

Taking nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin can increase the risk of peptic ulcer disease by four times compared to non-users. The risk of getting a peptic ulceris two times for aspirin users. Risk of bleeding increases if NSAIDs are combined with selective serotonin reuptake inhibitor (SSRI), corticosteroids, antimineralocorticoids, and anticoagulants. The gastric mucosa protects itself from gastric acid with a layer of mucus,the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (*COX-1*), which is essential for the production of these prostaglandins. Besides this, NSAIDs also inhibit stomach mucosa cells proliferation and mucosal blood flow, reducing bicarbonate and mucus secretion, which reduces the integrity of the mucosa.

Another type of NSAIDs, called COX-2 selective anti-inflammatory drugs (such as celecoxib), preferentially inhibit *COX-2*, which is less essential in the gastric mucosa. This reduces the probability of getting peptic ulcers; however, it can still delay ulcer healing for those who already have a peptic ulcer. Peptic ulcers caused by NSAIDs differ from those caused by *H. pylori* as the latter's appear as a consequence of inflammation of the mucosa (presence of neutrophil and submucosal edema), the former instead as a consequence of a direct damage of the NSAID molecule against COX enzymes, altering the hydrophobic state of the mucus, the permeability of the lining epithelium and mitochondrial machinery of the cell itself. In this way NSAID's ulcers tend to complicate faster and dig deeper in the tissue causing more complications, often asymptomatically till a great portion of the tissue is involved.[3]



Stress

Stress due to serious health problems, such as those requiring treatment in an intensive care unit, is well described as a cause of peptic ulcers, which are also known as stress ulcers.

While chronic life stress was once believed to be the main cause of ulcers, this is no longer the case. It is, however, still occasionally believed to play a role. This may be due to the well-documented effects of stress on gastric physiology, increasing the risk in those with other causes, such as H. pylori or NSAID use.[3]

Diet

Dietary factors, such as spice consumption, were hypothesized to cause ulcers until the late 20th century, but have been shown to be of relatively minor importance. Caffeine and coffee,also commonly thought to cause or exacerbate ulcers, appear to have little effect. Similarly, while studies have found that alcohol consumption increases risk when associated with H. pylori infection, it does not seem to independently increase risk. Even when coupled with H. pylori infection, the increase is modest in comparison to the

primary risk factor.[3]

Other

Other causes of peptic ulcer disease include gastric ischaemia, drugs, metabolic disturbances, cytomegalovirus (CMV), upper abdominal radiotherapy, Crohn's disease, and vasculitis. Gastrinomas (Zollinger–Ellison syndrome), or rare gastrin-secreting tumors, also cause multiple and difficult-to-heal ulcers.

It is still unclear if smoking increases the risk of getting peptic ulcers.[3]

There are three types of peptic ulcers

- Gastric ulcers: ulcers that develop inside the stomach.
- Oesophageal ulcers: ulcers that develop inside the oesophagus.
- Duodenal ulcers: ulcers that develop in the upper section of the small intestines, called theduodenum.

Pathogenesis of Peptic Ulcer

About half the world's population is stimulated by H. pylori, which is already one of many common causes of peptic ulcer. Frequency of H. pylori is high in development countries, especially in Africa, Central America, Central Asia, and Eastern Europe. The body is often found in childhood in an unhygienic, congested environment, especially in low-income countries. H. pylori causes epithelial cell depletion as well injury.

H. pylori is an etiologic factor in many patients with peptic ulcer disease and may put people at the forefront of developing gastric carcinoma. H. pylori colon in the human stomach. How to transfer H. pylori is vague, but appears to be transmitted to the individual through the fecal-oral route. The spread of H. pylori in adults appears to be related to the opposite of socio-economic status. It is also thought that water is the H-transfer dam. pylori.

Method H. pylori induces the development of various types of ulcers in gastroduodenal mucosa is not fully defined. H. pylori infection can lead to hypochlorhydria or hyperchlorhydria, thus determining the type of peptic ulcer. Major mediators of H infection. Pylori cytokines inhibit the production of parietal cells, but H. pylori can directly affect H + K + ATPase α-subunit, activates genetically related peptide calcitonin (CGRP) for somatostatin-related neurons, or inhibit gastrin production. Although the formation of gastric ulcers is associated with hyposecretion, 10-15% of patients with H infection. pylori have increased the number of abortions caused hypergastrinemia and decreased antral somatostatin content. This leads to an increase in histamine production, and later an increase in the production of acid or pepsin from the parietal cells and stomach. In addition, the completion of H. pylori leads to a decrease in gastrin mRNA expression and increased expression ofsomatostatin mRNA. In most cases the rest of the patients, stomach ulcers are associated with hypochlorhydria and mucosal atrophy.[1]

Important features of mucous membranes, bicarbonate, mucosal blood flow, prostaglandin, and epithelial regeneration.

The main cause of NSAID-related damage to the gastroduodenal mucosa is formal inhibition of cyclooxygenase-1 (COX-1), responsible of prostaglandin synthesis, and is associated with a decrease in mucosal blood flow, lower mucus and bicarbonate production, as well as inhibiting cell proliferation. NSAIDs block the enzyme by releasing in a focused manner. Co-administration of external prostaglandins as well Consumption of cyclooxygenase- 2 (COX-2) -selected NSAIDs reduces mucosal damage and the risk of ulcers.[1]

Complications of a peptic ulcer

Untreated ulcers can become worse over time. They can lead to other more serious healthcomplications such as

- **Perforation:** A hole develops in the lining of the stomach or small intestine and causes an infection. A sign of a perforated ulcer is sudden, severe abdominal pain.
- **Internal bleeding:** Bleeding ulcers can result in significant blood loss and thus require hospitalization. Signs of a bleeding ulcer include lightheadedness, dizziness,and black stools.
- **Scar tissue:** This is thick tissue that develops after an injury. This tissue makes it difficult for food to pass through your digestive tract. Signs of scar tissue includevomiting and weight loss.[4]

All three complications are serious and may require surgery. Seek urgent medical attention if you experience the following symptoms

- Sudden, sharp abdominal pain
- Fainting, excessive sweating, or confusion, as these may be signs of shock
- Blood in vomit or stool
- Abdomen that’s hard to the touch
- Abdominal pain that worsens with movement but improves with lying completely still

Outlook for peptic ulcers

With proper treatment, most peptic ulcers heal. However, you may not heal if you stop takingyour medication early or continue to use tobacco, alcohol, and nonsteroidal pain relievers during treatment. Your doctor will schedule a follow-up appointment after your initial treatment to evaluate your recovery.

Some ulcers, called refractory ulcers, don’t heal with treatment. If your ulcer doesn’t heal with the initial treatment, this can indicate:

- An excessive production of stomach acid
- Presence of bacteria other than *H. pylori* in the stomach
- Another disease, such as stomach cancer or Crohn’s

disease.

Your doctor may offer a different method of treatment or run additional tests to rule out stomach cancer and other gastrointestinal diseases.[4]

How to prevent peptic ulcers

Certain lifestyle choices and habits can reduce your risk of developing peptic ulcers. Theseinclude:

- Not drinking more than two alcoholic beverages a day
- Not mixing alcohol with medication
- Washing your hands frequently to avoid infections
- Limiting your use of ibuprofen, aspirin, and naproxen (Aleve)

Maintaining a healthy lifestyle by quitting smoking cigarettes and other tobacco use and eatinga balanced diet rich in fruits, vegetables, and whole grains will help you prevent developing apeptic ulcer.[4]

Peptic Ulcer- Signs and Symptoms

1. Heartburn and bloating.
2. Epigastric pain and discomfort. Pain worsens after meals in gastric ulcer but in duodenal ulcers it is relieved by meals.
3. Nausea and vomiting.
4. Gastrointestinal tract bleeding (hematemesis and/or melena).5- loss of appetite and weight loss
5. 6- Tarry, black, or bloody stools.[5]

Treatment

H. pylori-caused ulcers are treated with a combination of antibiotics and an acid-reducingproton pump inhibitor.

Antibiotics: Usually two antibiotics are prescribed. Among the common choices are amoxicillin, clarithromycin, metronidazole and tetracycline.

Bismuth subsalicylate: Sometimes this drug (eg, Pepto-Bismol) is added to the antibiotics plus proton pump inhibitor combinations mentioned above. This drug protects the stomach lining.

Combination treatment is usually taken for 14 days.

One newer medication, Talicia, combines two antibiotics (rifabutin and amoxicillin) with a proton pump inhibitor (omeprazole) into a single capsule.[6]

H2 receptor antagonists

They are reversible competitive antagonists for H2 receptors thus abolishing HCl secretion mediated by histamine, while that Ach and gastrin mediation is slightly inhibited.

Used for 4-6 weeks + eradication of H.pylori

An evening dose is preferred

Cimetidine: (800mg bed time or 400mg twice daily) - it is the least potent drug of H2bloockers

Ranitidine: (300mg bedtime or 150mg twice daily). Does not bind to the androgen receptors, no enzyme inhibition.

Famotidine: (40mg bedtime or 20mg twice daily). Twice as potent as ranitidine, has longer duration of action. No enzyme inhibiting or antiandrogenic effects.

Nizatidine (300mg bedtime or 150mg twice daily). Potent as Ranitidine, No enzyme inhibitng or antiandrogenic effects.[7]

Adverse effects

1. Inhibitor of cytochrome p450, increase levels of warfarin, theophylline and phenytoin.
2. Headache and confusion in the elderly.
3. Act as androgen receptor antagonist causing reversible gynecomastia, sexual dysfunction in males.[7]

Anticholinergic Drugs

Selective M1 receptor antagonists: (M1 receptors in autonomic ganglia) Pirenzepine,Telenzepine

Inhibit gastric acid secretion with minimal unwanted effects of cholinergic blockade

Less effective than other antisecretory drugs.[7]

Proton Pump Inhibitors: PPIs

- Examples Omeprazole, lansoprazole, Pantoprazole
- Mechanism of Action
- Causes irreversible inhibition of the H^+/ K^+ ATPase, blocking the transport of hydrogeninto the lumen.
- Reduces both basal and stimulated acid secretion
- PPIs are very effective acid suppressors as they do in the final stage of HCl release.
- They are destroyed by gastric acidity.
- They have an enteric coating, where they are easily absorbed by the alkaline mediumof the intestine as they are a weak base.
- They are prodrugs which means that after being absorbed in the intestines, they reach the gastric parietal cells in circulation.

Adverse effects

Diarrhea, colic, headache and diziness.

Inhibit metabolism of warfarin, phenytoin.

Prolonged inhibition of acid secretion risk of gastric neoplasia due to prolonged inhibition of acid secretion.[7]

Antacids

Weak bases, neutralize gastric acid and reduce pepsin activity

- Hydroxide is the most common base but trislicate, carbonate are also used
- Reduce pain and may promote healing
- Tab act more slowly than liquid antacid unless sucked or chewed
- They are weak bases that reduce gastric acidity by neutralizing HCl.

- They should not be used with H2 blockers or PPIs because they only function in acidicmedium

Mechanism of action
- Neutralize gastric acid.
- Decrease peptic activity (pepsin is inactive at PH 4).
- Reduce H.pylori colonization.
- Increase PG synthesis.

Types of Antacids
Sodium bicarbonate
- Absorbed systematically and should not be used for long-term treatment (alkalosis).
- Release Carbon dioxide causing eructation.
- Contraindicated in hypertension due to its high sodium content.[7]

Calcium carbonate
- Partially absorbed, has some systemic effect.
- Can cause hypercalcemia.
- Should not be used for long-term treatment.
- May stimulate gastrin release causing rebound acid production.
- Contraindicated in renal disease.

Magnesium hydroxide
- Not absorbed, no systemic effects.
- Causes diarrhea.

Aluminum hydroxide
- Not absorbed, no systemic effects.
- Causes constipation.
- Combination between aluminium and magnesium salts is logical to produce a balance in adverse effects on the bowel

Adverse Effects of Antacids
- Bind drugs: tetracycline, digoxin and prevent their absorption.
- Change in bowel habits.
- Calcium-based antacids cause rebound acid secretion.
- Milk-alkali syndrome, rare: Excessive doses of either sodium bicarbonate or calcium carbonate with calcium-containing dairy products can lead to hypercalcemia, renal insufficiency, and metabolic alkalosis (milk-alkali syndrome).
- They should be used cautiously in elderly and patients with renal impairment.
- Magnesium containing antacids cause CNS depression.
- Aluminum containing antacids can cause hypophosphatemia.

Cytoprotective Agents Sucralfate
It is sucrose sulphate + aluminium hydroxide gel.
Mechanism of action
1. In acidic medium the negatively charged sulphate groups bind to the positively charged proteins in the ulcer base forming a protective barrier against acid, bile and pepsin.
2. Increases mucous and bicarbonate secretion.
3. Increases PG secretion.
4. Inactivates bile acids and pepsin.

Adverse effects of sucralfate
- Nausea, vomiting, constipation, flatulance and dry mouth.
- Interfere with absorption of some drugs.

Misoprostol
This is a synthetic prostaglandin.

Mechanism of action
Inhibit acid secretion
Stimulate bicarbonate secretion
Stimulate mucus production.
Increase blood flow
Used in patients taking NSAIDs who are at risk for gastric ulcers (cytoprotective effect)

Adverse effects
May cause diarrhea and stimulation of uterine contraction.

Colloidal bismuth
- Combine with proteins in the ulcer base.
- Stimulate mucus production.
- Eradicate H.pylori.
- Dark discoloration of teeth and tongue.
- Promotes healing of both duodenal and gastric ulcers.
- Herbal Antiulcer Treatment

Allium sativum
Allium sativum of the Liliaceae family is commonly known as the “vellapundu” locally and locally called “vellapundu.” It is grown all over India. The chemical ingredients in this plant area contain aromatic acrid oil which is the active ingredient, starch, cereal, albumen, and sugar. Seeds produce a fragrant oil. Juice, especially its oil components, is rich in compounds of sulfur, iodine, and salicylic acid, in addition to essential nutrients and complementary vitamins.[8]

By Ayurvedic. Mustard or coconut oil in which the garlic is fried is not used very well for ulcers, ulcers and wounds. Garlic juice mixed with 3 or 4 parts of ordinary or distilled water has been used as an ingredient in cleansing wounds and ulcers.

In Recent Studies. Extract of A bulb juice A. sativum was given in doses of 250 and 500 mg /kg orally to mice, against cystamine-induced gastric ulcer. Exfoliation greatly enhances the healing of stomach ulcers and prevents the growth of gastric and duodenal ulcers in mice.

Throughout history, the health benefits of garlic have

been well documented, as well as its main uses of Allium sativum was due to its therapeutic properties. Organosulfur components of Allium sativum, which includes S-allyl-L-cysteine (SAC) sulfoxides and δ-glutamyl S-allyl- L-cysteine, is known to be primary. a combination of its bioactivity. Raw Allium sativum is easy to convert into a bioinactive form. Likewise, many types of its releases with different compounds of bioactive compounds have been were developed, and their effectiveness has been recognized and evaluated in many studies. Great role Allium sativum release has been shown to have an antioxidant effect by releasing active oxygen species (ROS), inhibits lipoprotein oxidation and reduces glucose uptake into the antioxidant serum. enzymes. Also, it showed a depressive effect of H. pylori-induced gastric inflammation in vivo, and anti- tumorigenic effect by promoting apoptosis and induction of cell cycle binding. Allicin and allyl-methyl plus methyl-allyl thiosulfinate from acetonic Allium sativum extracts limit the growth of H. pylori in in vitro research.[1]

Galega purpurea

Galega purpurea (papilionaceae) is more commonly known as "purple tephrosia". It is calleda place "Kolluk - kay - welai". Available throughout India, especially in the south India. It grows on rocky ground it is very difficult to concentrate. Chemicals constituents are yield gum, a fresh of albums and coloring matter, resin, chlorophyll, glucoside rutin.

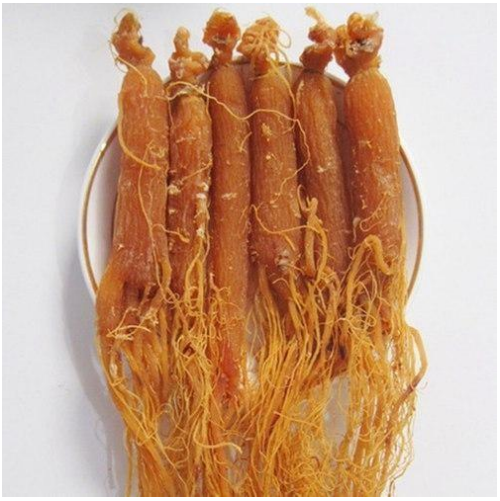
Antiulcer function

The root is powdered and mixed with honey is applied to wounds.[8]

Korean red ginseng

The extraction of Korean red ginseng plays an important role in inhibiting H 5-LOX H activity. pylori-induced, such as inactive c-jun, suppresses NF-κB-DNA binding, inhibits H. pylori-induced 5 (S) -hydroxyeicosatetraenoic acid biosynthesis, and inhibition of pro inflammatory interleukin (IL) -8 or 5-LOX mRNA. Therefore, these mechanisms reduce gastric carcinogenesis.

In addition, Korean red ginseng has been shown to be beneficial in suppressing 5 lipoxygenase (5-LOX) mRNA and enzyme activities, and consequently reducing the concentration of 5-hydroxy-eicosatetraenoic acid. Similarly, the extraction of green tea may prevent the activation of many transcription factors and targeted genes, including COX-2 and inducible nitric oxide synthase (NOS) activation of mitogen-activated protein kinase, as well as H-lipopolysaccharide. pylori-activated TLR- 4. As a result, these inhibitors increase the inflammatory factors that cause gastric mucosal ulcers. Kim et al. reported for the protective effect of Korean red ginseng against H. pylori-induced cytotoxicity in vitro. Meanwhile, in a previous clinical study, additional administration of Korean red ginseng increased H-levels. pylori, reduced gastrointestinal inflammation, as well as reduced DNA oxidative damage andapoptosis.[1]



Camellia Sinensis (Green Tea Polyphenols)

Today, Camellia sinensis is one of the most widely used beverages.

Chemopreventive The effects of Camellia sinensis depend on its function as an antioxidant, but also on its cellular regulation activities in cell growth, development, and apoptosis; and selected career development of bacterial flora in the intestine. Among the many ingredients of green tea, polyphenols and epigallocatechin gallate (EGCG) suppresses genetic expression of tumor necrosis factor-alpha (TNF-α). On the other hand, urease H. pylori is important in colonization, and research is focused. in the release of Camellia sinensis showed inhibitory activity of this enzyme. That causes that to prevent bacterial colonization. Many similar studies have shown the effect of prevention release of Camellia sinensis by increasing vacuolation of cells by normally cytotoxin A (vacA) and urea conduction in H. pylori infection. As a result, it can pursue anti-H. pylori function in vivo.[1]

In 2008, Rao et al. reported gastroprotective activity of 50% ethanolic extract extracted from Ficus fruit glomerata (FGE) in models of gastric ulcers in mice. FGE was applied per mouth (50, 100 and a body weight of 200 mg / kg), twice a day for five days to prevent ethanol (EtOH), pylorus ligation (PL), and cold restraint

stress (CRS), which causes the formation of sores. It showed a volume-dependent wound compression, and played a major role in preventing oxidative damage of the gastric mucosa by inhibiting lipid peroxidation and significantly reducing H + / K + -ATPase again superoxide dismutase. Their results showed that F. glomerate has an important gastroprotective effect that may be the result of contraceptives.

Curcuma Longa and Artemisia Asiatica

Medicinal plants with antioxidant and anti-inflammatory activity have been shown effect on gastroesophageal reflux (GERD). Medicinal plants and herbal preparations with antioxidant and anti-inflammatory systems includes Curcuma longa, Panax quinquefolium, Artemisia asiatica, and Lonicera japonica. In addition, other options include: regulation of regulation genetic proteins that play a key role in major inflammation, including 1 intercellular adhesion molecule-1 (ICAM-1) and cytokine-induced neutrophil chemoattractant-2-beta (CINC-2-2 beta) (Panax quinquefolium); revitalizing function and gastric mucus (Morus alba, Curcuma longa); to reduce stomach acid, such as Curcuma longa, Morus alba, and acididinol syrup, tonic contractions of low esophageal sphincter (LES) (Salvia miltiorrhiza, STW 5), and prevention cytokines containing pro-inflammatory IL-1 b and TNF-a (STW 5). It is important to mention the research on mice when pre-treatment with Artemisia compounds asiatica (DA-9601) reduced total congestion of the esophagus wall and volume of ulcers group ranitidine.

Mahattanadul has shown in his study of rats that the rhizome of Curcuma longa playsa protective role. Role in the formation of acute acid reflux esophagitis (RE), but it has not been effective in preventing chronic acid RE. However, its combination with dimethyl sulfoxide as an antioxidant compound reduced the intensity of the esophagitis wound index to lansoprazole. In contrast, lansoprazole tends to increase the severity of all histopathological changes in addition to control and groups treated with curcumin. Therefore, it appeared to have antioxidant and anti-inflammatory activity

Curcumin plays a major role in its beneficial effects on GERD.

An herbal medicine can be a powerful weapon to suppress or reverse a disease-related condition stages of H infection. pylori and dissolution. Finally, those plant products have proven to be powerful who can be medical personnel in the prevention of gastrointestinal disorders.[1]

Curcuma Longa

Herb-Drug Interactions

With the increasing use of herbal supplements worldwide, the number of adverse events drug interactions are increasing. The interaction between the herbal supplement and the drug can be observed such as pharmacokinetic or pharmacodynamic interactions. Pharmacokinetic interactions are the result of using the same method of absorption, distribution, metabolism, or extraction a supplement and a drug used in combination, which leads to a change in the concentration of the drug in the blood and pharmacologic action. Pharmacodynamic interactions involve a direct effect on the machine the action of a given drug in combination without changing the concentration of the drug, only in opposition or to increase the clinical effects of the drug. Allium sativum release reduces the concentration of P-gp-transporting drugs, such as digoxin, Doxorubicin, rosuvastatin, and verapamil. The most widely studied allium sativum link is one containing warfarin, although this has not yet been confirmed in controlled clinical trials. And, it prevents platelet aggregation, so it should be used with caution in patients with platelet aggregation or those with anticoagulant therapy. Zingiber officinalis increases bleeding time by blocking thromboxane synthetase, but this has not been confirmed in clinical trials. Ginkgo biloba knew increasing the risk of bleeding, especially in combination with anticoagulant drugs, due to prevention platelet aggregation. Flavonoid in Ginkgo biloba has antiplatelet activity, but does not

affect blood coagulation or platelet function in humans. When combined with NSAIDs, it can be very harmful bleeding .

Panax ginseng implements cytochrome P450 3A4 (CYP3A4), which reduces efficiency. of calcium channel blockers, certain antihypertensive drugs and statin, and others antidepressants. Panax ginseng has hypoglycemic activity in patients with diabetes, and it ispossible causes headaches, tremors, and dementia in patients treated with phenelzine. The extract of green tea has been shown to increase simvastatin concentration, or prevent drug parasites organic anion transporting protein 1a1 (OATP1A1) and anion transporting protein 1a12 (OATP1A2), responsible for the transport of fluoroquinolones, beta blockers, and imatinib.

In conventional antiulcer treatment, it is important to emphasize multiple drug interactions cimetidine drugs. Studies have reported significant clinical interactions with warfarin, phenytoin, diazepam, chlormethiazole, propranolol, lidocaine, and a number ofother drugs. Also, cimetidine may increase the quality or effect of green tea as a result of CYP1A2 inhibition, which in turn hepatic oxidative metabolism of caffeine.[1]

Abbreviation

H. pylori - Helicobacter pylori
NSAIDs - Non-steroidal anti-inflammatory drugs PPIs - proton pump inhibitors
SSRI - selective serotonin reuptake inhibitor CGRP - Calcitonin gene-related peptide SAC - S-allyl-L-cysteine
ROS - reactive oxygen species NOS - nitric oxide synthase EGCG - epigallocatechin gallate
TNF-α- tumor necrosis factor-alpha FGE - Ficus fruit glomerata
EtOH - ethanol
PL- pylorus ligation
CRS - cold restraint stress GERD -gastroesophageal reflux LES - low esophageal sphincter RE -reflux esophagitis.

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Finally, I want to thank my parents for supporting me through thick and thin and always giving me the best of everything that I would need and want.

CONCLUSIONS

Now a days peptic ulcer is very common in rural area, because of their lifestyle and unhygienic condition. Moreover 5-10% people are suffering in peptic ulcer. Most of the cases are not curable but still it can be remedial by treating some antibiotics. After knowing all the details about peptic ulcer we can overcome the problem.

The combination of herbal products and common anti-ulcer drugs may introduce a synergistic effect against H. pylori and gastric ulcer disease and improve the outcome in four patients stomach ulcer. With only a few human studies, it is suggested that additional clinical studies be conducted larger samples on the efficacy and safety of medicinal plants with anti-ulcer function. And, it would be it has been useful to design lessons to investigate and clarify the methods medicinal plants used to treat or prevent peptic ulcer.

Finally, herbal products used for therapeutic purposes require a license to be effective their safety and quality, and ensure that a randomized controlled trial confirms its requirements possible efficiency. With growing reports of drug interactions, there is still a shortage research in this field, apart from the steps taken to address this problem. So, pharmacists and doctors should be especially aware of the risks associated with the use of herbal preparations, even if they are open their own or combined with other herbal remedies or conventional therapies.

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