



A Brief Review Of Conventional Therapy & Herbal Remedies of Peptic Ulcer

¹Naziya Pathan , ²Mayuri Shrikhande , ³Varsha Avhad, ⁴Sumit Devkar, ⁵Aman Shaikh ,

¹Student , ^{2,3}Assistant Professor , ^{4,5}Student

¹Department of Pharmacognocny

¹Arihant College Of Pharmacy , Kedgaon , Ahmednagar(M.S.),India, 414005

Abstract :

The presence of gastric juice pH and the lowering of mucosal defenses are prerequisites for the development of peptic ulcers. Helicobacter pylori (H. pylori) infection and non-steroidal anti-inflammatory drugs (NSAIDs) are the two main elements that comprise the mucosal defense against damage. traditional methods of treating peptic ulcers, such as histamine-2 (H2) receptor antagonists and proton pump inhibitors (PPIs) have shown side effects, drug interactions, and relapses. Conversely, however, therapeutic plants and Many diseases can be prevented and treated with their chemical compounds. Therefore, The common medicinal plants included in this review can be used to treat or prevent stomach ulcers. Attempts have been made in this review to learn about some medicinal plants that have potential applications in both modern science and Ayurvedic medicine for the treatment or prevention of peptic ulcers.

Keywords : *Peptic Ulcer, Conventional drugs, Ayurvedic treatment, Polyherbal formulation.*

I. INTRODUCTION :

Peptic ulcer is a prevalent ailment that affects the submucosal layer of the stomach and duodenum. It is mostly linked to the overuse of non-steroidal anti-inflammatory medicines (NSAIDs) and H. pylori infection [1].

In the previous 20 to 30 years, there has been a notable decline in the disease's occurrence, and this development has been linked to the development of pharmaceutical therapy and the expansion of healthcare services [2].

Besides the risk factors and epidemiology, talk about the pathophysiology of peptic ulcers [3-5]. We will talk about the working closely with the peptic ulcer treatment strategy and any associated complications to diagnose the condition

Patients with acid peptic disease are thought to experience mucosal disruption as a result of an acidic environment that is hypersecretory, in addition to dietary variables or stress. Alcohol and tobacco use, non-steroidal antidepressants, H. pylori infection, and other risk factors

Use of nonsteroidal anti-inflammatory medications (NSAIDs) with Zollinger-Ellison syndrome.

There are several synthetic medications available to treat ulcers. But when compared to herbal remedies, these medications are more costly and probably have more adverse effects. The literature showed that numerous ayurvedic physicians and conventional medical practitioners treat ulcers with a variety of medicinal plants and polyherbal formulations.



Figure 1: Stomach Ulcer

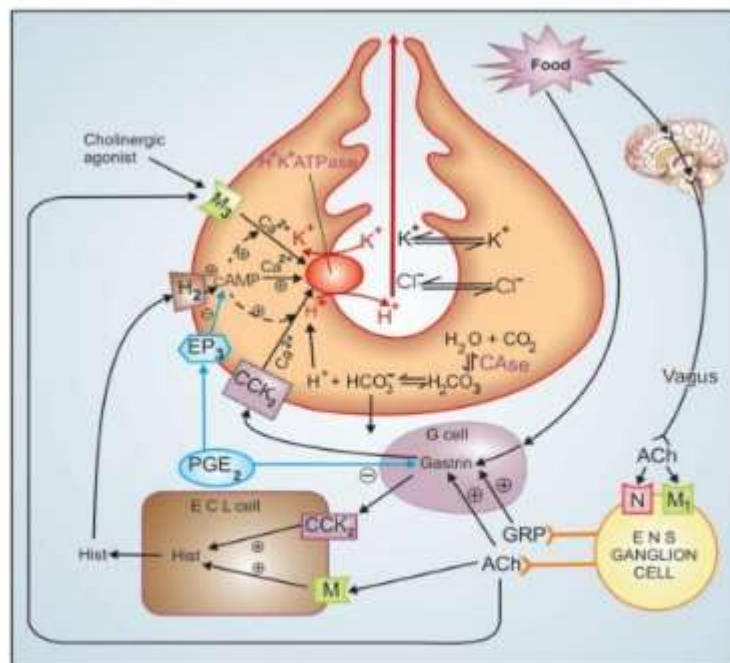
II. PATHOPHYSIOLOGY :

Peptic ulcer can be identified by the presence or absence of hypo- or hyperchlorhydria, which is caused by an H. pylori infection. Although cytokines that block parietal cell secretion are the primary mediators of H. pylori infection, H. pylori can also directly impact the H+/K+ ATPase.

α -subunit, which stimulates sensory neurons connected to somatostatin through calcitonin gene-related peptide (CGRP), could prevent the gastrin from being produced [16]. Despite the fact that stomach ulcer development is linked to hyposecretion; in 10-15% of cases, H. pylori infection is associated with increased stomach secretion as a result of decreased antral somatostatin concentration and hypergastrinemia [17]. This causes histamine levels to rise, secretion, followed by a rise in the gastric and parietal cells' release of acid or pepsin.

Fig. summarizes the mechanisms at work at the gastric parietal cells. In the apical canaliculi, the terminal enzyme H+K+ATPase (proton pump) secretes H+ ions that are capable of stimulating parietal cells through histamine, ACh, and gastrin operating through their own basolateral receptors in these cells' membranes. Among the three physiological Histamine and other secretagogues function by binding to H2 receptors. the primary function because the other two, ACh and gastrin, act more indirectly and partially directly by releasing Paracrine enterochromaffin-like (ECL) cells' histamine found in the oxyntic glands and referred to as "histaminocytes." As though H2 receptors produce cAMP, which in turn activates H+K+ATPase.

Gastrin/cholecystokinin (CCK2) and muscarinic receptors seem to operate via the phospholipase C → IP3–DAG pathway, mechanism that releases intracellular calcium ions.



Secretion of HCl by gastric parietal cell and its regulation
 C.Ase.—Carbonic anhydrase; Hist.—Histamine; ACh.—Acetylcholine; CCK₂—Gastrin cholecystokinin receptor; M.—Muscarinic receptor; N—Nicotinic receptor; H₂—Histamine H₂ receptor; EP₃—Prostaglandin receptor; ENS—Enteric nervous system; ECL cell—Enterochromaffin-like cell; GRP—Gastrin releasing peptide; + Stimulation; – Inhibition.

Figure 2: Pathophysiology of Peptic Ulcer

Ca²⁺ is also involved in the cAMP-mediated proton pump activation. When exposed to cholinergic agonists and gastrin, the secretomotor response is fully expressed. only when cAMP produced by H₂ activation is present.

Histamine thus takes part in the acid response to gastrin and ACh on multiple levels, while H₂ antagonists inhibit in addition to histamine, pentagastrin, ACh, and actually any stimulus that secretes gastric acid.

The antrum releases glutathione in reaction to an increase in food ingredients, vagally mediated reflexes, and antral pH involving the enteric nervous system's ganglion cells (ENS). Gastrin release is induced by the postganglionic ENS neurons from gastrin-secreting "G" cells by developing both gastrin and ACh peptide release (GRP).

The M₁ subtype of muscarinic receptors is the predominant receptor mediating vagal responses. Its location on the intramural plexuses' ganglion cells has been verified. The muscarinic receptor subtype of parietal cells is M₃, whereas the subtype of muscarinic receptor on ECL cells is unknown. Vagus releases acetylcholine (ACh) in close proximity to G and ECL cells, but seemingly apart from the cells of the parietal region. Consequently, vagal effects are mostly indirectly by way of gastrin and histamine.

One description of prostaglandins is "cytoprotective."

function in the mucosa of the stomach by increasing mucus and secretion of bicarbonate. from cells of the stomach mucosal epithelium, in addition to additional acts.

TREATMENT :

1. Allopathic Aspects :

1. Reduction of gastric acid secretion

(a) H₂ antihistamines: Cimetidine, Ranitidine, Famotidine, Roxatidine

(b) Proton pump inhibitors: Omeprazole, Esomeprazole, Lansoprazole, Pantoprazole, Rabeprazole,

(c) Anticholinergic drugs: Pirenzepine, Propantheline,

(d) Prostaglandin analogue: Misoprostol

2. Neutralization of gastric acid (Antacids)

(a) Systemic: Sodium bicarbonate, Sod. citrate

(b) Nonsystemic: Magnesium hydroxide, Mag. trisilicate, Aluminium hydroxide gel, Magaldrate, Calcium carbonate

3. Ulcer protectives: Sucralfate, Colloidal bismuth subcitrate (CBS)

4. Anti-H. pylori drugs: Amoxicillin, Clarithromycin, Metronidazole, Tinidazole.

Mechanisms of action and adverse effects of the most commonly used antiulcer treatment options

Medicine	Mechanism Of Action	Adverse Effect
1. Proton Pump Inhibitors (PPIs) : Omeprazole Lansoprazole Rabeprazole Esomeprazole Pantoprazole	Inhibition of the gastric H ⁺ /K ⁺ -ATPase (proton pump) enzyme system	Headache Abdominal pain Diarrhea Nausea Vomiting Constipation Flatulence Vitamin B12 deficiency Osteoporosis
2. H ₂ Receptor Blockers : Cimetidine Famotidine Nizatidine Ranitidine	Blocking the action of histamine at the histamine H ₂ receptors of parietal cells	Headache Anxiety Depression Dizziness Cardiovascular events Thrombocytopenia
3. Antacids : Aluminum hydroxide		Hypophosphatemia Chalky taste Constipation

Magnesium hydroxide	Increases gastric pH to greater than four, and inhibits the proteolytic activity of pepsin Causes osmotic retention of fluid	Abdominal cramping Diarrhea Electrolyte imbalance
4. Cytoprotective Agents : Sucralfate	Stimulate mucus production and enhance blood flow throughout the lining of the gastrointestinal tract	Diarrhea Abdominal pain Headache Constipation Backpain

CLASSIFICATION OF DRUGS :-

CLASS	SUBCLASS	DRUGS
Gastric acid secretion inhibitors	Proton Pump Inhibitors	Omeprazole, Lansoprazole, Pantoprazole, Rabeprazole,
	H ₂ Receptor Antagonist	Cimetidine, Ranitidine, Famotidine, Loxatidine
	Anticholinergic	Pirenzepine, Telenzepine, Propantheline.
	Prostaglandin analogue	Misoprostol, Enprostil
Gastric acid neutralizers (Antacid)	Systemic	Sodium bicarbonate, sodium citrate
	Non - systemic	Magnesium hydroxide, Aluminum hydroxide
Ulcer protective	Sucralfate, Colloidal Bismuth Subcitrate (CBS)	

I] Gastric Acid Secretion Inhibitors :**1. Proton Pump Inhibitor :**

MOA - These drugs inhibit H⁺K⁺ATPase in parietal cells of the stomach

PPIs are prodrugs, given orally in the early morning in an empty stomach

These drugs have a short half-life (1.5 hours) but can inhibit acid secretion for more than 24 hours

E. g - Omeprazole, Pantoprazole, Rabeprazole

Lansoprazole is more potent than omeprazole and the safest PPI in pregnancy and has higher oral bioavailability.

Pharmacokinetics -

These drugs are weak bases and can be destroyed by gastric acid, so they are given in enteric-coated forms.

Bioavailability of all PPIs is reduced by food.

- Lansoprazole is the safest PPI in pregnancy.
- Esomeprazole, lansoprazole, and pantoprazole can be given by i.v. route.
- Omeprazole and esomeprazole are microsomal enzyme inhibitors. These may decrease the metabolism of diazepam.
- Lansoprazole enhances the metabolism of theophylline

The side effects of the PPIs, such as a headache, diarrhea, constipation, and abdominal discomfort, are minor and easily managed

2. H₂ Receptor Antagonist :

MOA - These drugs competitively inhibit H₂ receptors in the parietal cell, thus inhibiting acid secretion. ACh and gastrin act partly by causing the release of histamine, therefore the acid-secreting capacity of these agents is also decreased by H₂ blockers.

These drugs are less potent than PPIs

E. g. - Cimetidine, Ranitidine, Famotidine

3. Anticholinergic :-

These drug shows anticholinergic activity

E.g - Pirenzepine, Telenzepine, Propantheline

4. Prostaglandin Analogue

PGE1, PGE2 and PGI2 are produced in gastric mucosa and appear to serve a protective role by

Promoting mucus and H₂CO₃ secretion

Inhibiting acid secretion

E. g.- Misoprostol (PGE1 Analogue) , Enprostil and Rioprostil

III] Antacids (Gastric Acid Neutralizers)

These drugs do not decrease acid production but neutralize gastric acid.

There are two types of Antacids

1.Systemic 2.Nonsystemic

1.Systemic Antacids -

These drugs are absorbed systemically in systemic circulation, which disturb acid base balance it further induces alkalosis on large dose

E. g - Sodium bicarbonate, Sodium citrate.

2.Non Systemic Antacids -

These drugs are poorly absorbed from GIT , not absorbing systematically

These are basic compounds reacts with gastric HCl and forms salt

It has no effect on the acid-base balance.

E. g - Magnesium hydroxide, Aluminum hydroxide

III] Ulcer protective :-

In the stomach's acidic environment, it dissociates into its anionic form, which attaches itself to the base of the ulcer. This stops gastric acid from diffusing and forms a barrier that is resistant to bile and pepsin

E. g - Sucralfate, Colloidal Bismuth Subcitrate

• Sucralfate: It is aluminum salt of sulfated sucrose. At pH below 4, its molecules polymerize to form a sticky layer that covers the ulcer base and acts as a physical

barrier to prevent acid exposure. It can bind phosphates also and can result in hypophosphatemia. It should not be given with antacids because it acts only in acidic medium (antacids raise the pH by neutralizing the gastric acid). Most common side effect of sucralfate is constipation.

• Colloidal bismuth subcitrate: It also forms an acid resistant coating over the ulcer. It also dislodges H. pylori from the surface of gastric mucosa and kills it. Adverse effects include blackening of tongue and bismuth toxicity

Alternative Therapy :-

Phytotherapy, or the use of medicinal plants to treat a variety of illnesses, is as old as humanity. Additionally, there has been an increase in interest in complementary therapies and the use of herbal products in recent years, particularly those made from herbal remedies. Additionally, because of the emergence of different side effects from using conventional medicines for a variety of illnesses, medicinal plants are thought to be the primary source of potentially novel medications. Crude plant extracts are the primary source of novel pharmaceuticals, and have demonstrated encouraging outcomes when used to treat stomach ulcers.

Many pharmaceuticals, including sucralfate, bismuth, anticholinergics, antacids, antimicrobials, and proton pump inhibitors, are known to be ineffective in certain situations and have a host of negative consequences, including hematopoietic changes, arrhythmia, impotence, gynecomastia, hypersensitivity. Consequently, studies of the novel pharmacologically active agents by evaluating various plant extracts resulted in the identification of efficient and safe medications that have a gastroprotective effect.

The ability of medicinal plants to generate diverse and renewable secondary metabolites, also referred to as phytochemical constituents, is what gives them their medicinal qualities. As a result, many plants have employed these phytochemicals as a defense mechanism against infections

HERBS AND AYURVEDIC MEDICINES FOR ULCER

Using certain herbs and ayurvedic medications is very helpful for ulcers.

Shatavari balances the levels of stomach acid by increasing mucous secretion, regulating hormonal rage, and reducing acid production.

Indravaruni's antioxidant qualities repair the intestines' damaged linings and lower free radical levels. It also has purgative qualities that calm the pitta.

Amalaki is an effective acidity regulator.

Kushmanda: strengthens digestion and regulates stomach secretion

Along with these, there are more medicinal plant that shows anti ulcer effects such as :-

Medicinal Plant	Mechanism of Action	Effect
1. Glycyrrhiza glabra L	Anti ulcerogenic and anti inflammatory	Anti-inflammatory, anti tussive, hepatoprotective
2. Carica papaya L	Treatment with aqueous and methanolic extract causes decrease in gastric acidity and increase mucus production	Antioxidant, Antiulcer activity
3. Korean red ginseng	Inhibition of H. pylori-induced 5-lipoxygenase (5-LOX) activity; preventing pro-inflammatory interleukin (IL)-8 or 5-LOX mRNA	Anti-inflammatory effect; increase eradication rates of H. pylori; reduction of gastric inflammation and oxidative DNA damage
4. Allium sativum	Inhibition of lipoprotein oxidation and lower serum glucose induction of antioxidant enzymes; mechanisms need to be more investigated	Antioxidant; suppressive effect of H.pylori-induced gastric inflammation in vivo and in vitro
5. Curcuma longa	Inhibition of H. pylori-induced 5-LOX activity	Anti-inflammatory; antioxidant
6. Zingiber officinalis	Inhibition of PGE2 and parietal cell H+K+-ATPase	Anti-inflammatory effect; antioxidant
7. Camellia sinensis (Green tea polyphenols)	Suppression of tumor necrosis factor-alpha (TNF- α) gene expression; inhibition of urease	Antioxidant; improvement in the function of intestinal bacterial flora
8. Aloe vera	Through reducing leukocyte adhesion, increasing IL-10 levels, decreasing TNF alpha levels, and inhibiting IL-6 and IL-8, aloe vera effectively reduces inflammatory responses	Antioxidant, anti-inflammatory, mucus secreting,

1. Glycyrrhiza glabra L

It is commonly known as Liquorice belonging to the family Fabaceae.

The glycyrrhetic acid of Liquorice showed potent in vitro activity against H. pylori indicating its antiulcer effect on peptic ulcers



Figure 3: Glycyrrhiza Glabra

2. Carica papaya L

Carica papaya (Caricaceae) is commonly known as “papaya.” It is locally called “pappali-pazham.”

Chemical constituents in this plant are Papain, chymopapain, pectin, carposide, carpaine, carotenoids, and antheraxanthin

Active Constituents. Chymopapain and papain are widely known as being useful for digestive disorders and disturbances of the gastrointestinal tract .



Fig. 3: Reported biological activities of different parts of *Carica papaya* L.

Carica papaya L

3.Korean red ginseng

Korean ginseng (*Panax ginseng* Meyer, Araliaceae) is traditionally used as an important herbal medicine in Far East Asia. These include ginseng saponins, ginseng oils and phytosterol, carbohydrates and sugars, organic acids, nitrogenous substances, amino acids and peptides, vitamins and minerals, and certain enzymes that have been isolated and characterized. Korean red ginseng has been shown to be beneficial in suppressing 5-lipoxygenase (5-LOX) mRNA and enzyme activities, and consequently the decreased synthesis of 5-hydroxy-eicosatetraenoic acid



Korean red ginseng

4.Allium sativum

Allium sativum belonging to the family Liliaceae is commonly known as “garlic” and locally called as “vellapundu.” Active Constituents. Volatile oil, alliin, and allicin are considered.



Allium sativum

Antiulcer activity : - Mustard or coconut oil in which garlic has been fried is an excellent application for maggots infesting ulcers, ulcerated surfaces, and wounds. Garlic juice mixed with 3 or 4 parts of ordinary or distilled water has been used as a lotion for washing wounds and foul ulcers

5.Curcuma longa

Curcuma longa L. is commonly known as Turmeric, which belongs to the family Zingiberaceae.

Antiulcer activity :- Its combination with the antioxidant dimethyl sulfoxide decreased the esophagitis ulcer index severity to a level comparable to lansoprazole. However, lansoprazole tended to increase all histopathological changes' severity above the control and groups given curcumin. For this reason, it appears that the anti-inflammatory and antioxidant effects of One important factor in curcumin's positive effects on GERD is its presence [101]. Herbal therapy can be a potent tool for reducing or controlling the disease-related signs and symptoms of *H. pylori* infection and removal. In the end, those plant-based products have demonstrated strong prospective use as drug candidates in the prevention of stomach disorders.



Curcuma longa

6. Zingiber officinalis

Synonyms : Rhizoma zingiberis, Zingibere

Biological Source : Ginger consists of the dried rhizomes of the Zingiber officinale Roscoe, belonging to family Zingiberaceae.

Anti ulcer activity : By stopping the growth of the colon cancer cells, increasing DNA synthesis, and inducing apoptosis, the plant extract showed antitumor effects on the cells [92]. Additionally, the primary strong phenolic component found in Zingiber officinalis is 6-gingerol, a substance with a wide range of pharmacological effects. Extracts from Zingiber officinalis that contain Gingerols are essential for inhibiting prostaglandin E2 (PGE2) [73]. Conversely, however, the active Phenolic substances like zingerone and gingerol are important in preventing parietal cell H⁺

In K⁺-ATPase. Because of this, the actions of zingerone and gingerol are crucial in inhibition of the proton pump and a decrease in stomach acid production. It also exhibits a shielding effect against ulcers caused by H. pylori .



Zingiber officinalis

7. Camellia sinensis (Green tea polyphenols)

The botanical name of tea is Camellia sinensis. The biological source of tea is prepared leaves and leaf buds. It belongs to the theaceae family. Commonly it is known as tea plant or tea shrub

Anti ulcer activity - In addition to its antioxidant activity, Camellia sinensis's molecular regulatory actions on cellular growth, development, and apoptosis, as well as a specific enhancement in the function of these mechanisms, all contribute to its chemopreventive effects. of the bacterial flora in the intestines. Among green tea's many components are polyphenols and The expression of the tumor necrosis factor-alpha (TNF- α) gene is suppressed by epigallocatechin gallate (EGCG).



Camellia sinensis

However, since H. pylori's urease is essential to its colonization, research focused on Camellia sinensis extract revealed this enzyme's inhibitory activity. This leads to the inhibition of the colonization of bacteria. Several related studies showed the inhibitory effect of

Camellia sinensis extract by vacuolating urea and cytotoxin A (vacA), which increases cell vacuolation conduction in an infection with *H. pylori*

8. Aloe Vera

Aloevera belonging to the family Liliaceae is commonly known as "aloe gel."

Chemical constituents in this plant are aloin, isobarbaloin, and emodin



Aloevera

Antiulcer Activity In Ayurvedic : Leaves are being used successfully in America in the local treatment of chronic ulcers. First the pain diminishes and after a few weeks the ulcers heal.

In Recent Studies. Aloe vera powder was mixed with gum acacia; the solution was administered orally in rats at dose of 200 mg/kg against indomethacin induced gastric ulcer. The extract showed significant antiulcer activity comparable to control.

Active Constituents : Barbalin, isobarbolin, and saponins are considered.

AYURVEDIC MEDICATED FORMULATION :-

1. Nature Vedic Jsgut -
2. Zandu Pancharishta -
3. Medinutrica - Pachan fatafat Capsule
4. Alsarex Tablet :
5. Yashtimadhu Capsule

1. Jsgut - A. V. Syrup

Composition :- Each 10 ml syrup contains

Herbs	Active Constituents	Quantity
Curcumin	NLT95% Curcumoids	500mg
Mulethi	Glycyrrhiza Glabra	300mg
Ashwagandha	Withania Somnifera	150mg
Guduchi	Tinospora Cordifolia	300mg
Saunf	Foeniculum Vulgarae	150 mg
Arand	Ricinus Communis	150mg
Garlic	Allium Sativum	150mg
Chamomile	Matricaria Chamomilla	150mg
Aloevera	Aloe Barbadensis	150mg
Honey		0.5ml
Sugar		q. s



Jasgut -A. V. Syrup

2 Zandu Pancharishta:-

Ingredients:

- Draksha (Dried Grapes) - *Vitis vinifera*
- Kumari or Aloe vera juice – *Aloe barbadensis*
- Dashamoola (Combination of ten roots)
- Ashwagandha - *Withania somnifera*
- Shatavari - *Asparagus racemosus*
- Triphala (Combination of three herbs, i.e. Amalaki, Bahera, and Haritaki)
- Guduchi - *Tinospora cordifolia*
- Bala - *Sida cordifolia*
- Yashtimadhu or Licorice - *Glycyrrhiza glabra*
- Trikatu (Combination of three herbs, i.e. Kali Mirch, Pippali and Sunthi)
- Trijat (Combination of three herbs, i.e. Dalchini, Elaichi, and Tejpatra)
- Arjuna - *Terminalia arjuna*
- Manjistha - *Rubia Cordifolia*
- Ajamoda - *Apium Graveolens Linn*
- Dhanyaka or dhanian powder - *Coriandrum sativum*
- Haridra or Turmeric - *Curcuma Longa*
- Shati - *Hedychium spicatum*
- Sveta Jiraka - *Carum bulbocastanum*
- Lavanga or Clove - *Syzygium aromaticum*

The dosage of Pancharishta is 6 teaspoons or 2 tablespoons (i.e. 30 ml) for adults. It should be infused in an equal quantity of water (i.e. 30 ml) and should be taken immediately after a meal.



Zandu Pancharishta:

3. MEDINUTRICA Pachan Fatafat Capsule :-

It is 100% Pure Herbal Extract Veg Ayurvedic Medicine for Constipation, Acidity, Gas, Indigestion, IBS & Gastric Ulcer - 30 Veg Capsules



Pachan Fatafat Capsule

4. Alsarex Tablet :-

Alsarex Tablet is a proprietary Ayurvedic medicine manufactured by Charak Pharma

It acts as an anti-ulcerant and natural antacid.

Ingredients of Alsarex Tablet:

Amalaki [Embelica officinalis] – 50 mg

Yastimadhu [Glycyrrhiza glabra] – 50 mg

Usheera [Vetiveria zizanioides] –50 mg

Udumbara [Ficus racemosa] – 50 mg

Shatavari [Asperagus racemosus] –25 mg

Suthashekhara rasa – 50 mg – Sutshekhhar Ras is an Ayurvedic medicine in tablet or powder form. It is used in the treatment of dyspepsia, gastritis, vomiting, abdominal pain etc.

Pravala pisti – 50 mg

Apamarga kshara – 20 mg – Apamarga Kshara is an alkaline Ayurvedic medicine, in powder form. It is prepared from an herb called Apamarga – Prickly Chaff-flower – Achyranthes aspera. The medicine has been in use since the time of Sushruta, that is for thousands of years.

Jaharmohra pisti – 10 mg

Jatamansi [Nardostachys jatamansi] – 10 mg – Also called Spikenard, atamansi is a famous Ayurvedic herb used in neuro-psychiatric diseases and skin diseases.

Jeeraka [Cuminum cyminum] – 10 mg

Kapardika bhasma – 10 mg

Mukta shukti bhasma – 10 mg

Shankha bhasma – 10 mg

Yashada bhasma – 10 mg

Chandana [Santalum album] – 25 mg – Sandalwood is a coolant, very useful in gastritis, burning sensation in hands and feet. It is also a good ingredient to include in your juice mixes, in small amounts, of course.

Lodhra [Symplocos racemosa] – 25 mg

Triphala [Embelica officinalis, Terminalia chebula, Terminalia bellerica] – 25 mg



Alsarex Tablet

Yashtimadhu

Himalaya Yashtimadhu Tablet is an ayurvedic medicine that is primarily used for the treatment of GERD, Digestive Disorders. Secondary and off-label uses of Himalaya Yashtimadhu Tablet have also been mentioned below. The key ingredients of Himalaya Yashtimadhu Tablet are Mulethi (Yashtimadhu)



Yashtimadhu Tablet

Balances excessive gastric secretion, acts as a demulcent and produces thick, protective mucus for the stomach lining, accelerating the healing of gastric and duodenal ulce

REFERENCE :-

- 1.Narayanan, M.; Reddy, K.M.; Marsicano, E. Peptic ulcer disease and Helicobacter pylori infection. *Mo. Med.*2018, 115, 219–224. [PubMed]
- 2.Lanas, A.; Chan, F.K.L. Peptic ulcer disease. *Lancet* 2017, 390, 613–624. [CrossRef]
- 3.Lanas, A.; García-Rodríguez, L.A.; Polo-Tomás, M.; Ponce, M.; Quintero, E.; Perez-Aisa, M.A.; Gisbert, J.P.;Bujanda, L.; Castro, M.; Muñoz, M.; et al. The changing face of hospitalization due to gastrointestinal bleeding and perforation. *Aliment. Pharmacol. Ther.* 2011, 33, 585–591. [CrossRef] [PubMed]
- 4.Sonnenberg, A. Review article: Historic changes of helicobacter pylori-associated diseases.*Aliment. Pharmacol. Ther.* 2013, 38, 329–342. [CrossRef] [PubMed]
- 5.Søreide, K.; Thorsen, K.; Harrison, E.M.; Bingener, J.; Møller, M.H.; Ohene-Yeboah, M.; Søreide, J.A.Perforated peptic ulcer. *Lancet* 2015, 386, 1288–1298. [CrossRef]
- 6.Zhang, B.B.; Li, Y.; Liu, X.Q.; Wang, P.J.; Yang, B.; Bian, D.L. Association between vacA genotypes and the risk of duodenal ulcer: A meta-analysis. *Mol. Biol. Rep.* 2014, 41, 7241–7254. [CrossRef] [PubMed]
- 7.Datta De, D.; Roychoudhury, S. To be or not to be: The host genetic factor and beyond in Helicobacter pylori mediated gastro-duodenal diseases. *World J. Gastroenterol.* 2015, 21, 2883–2895. [CrossRef]
- 8.Lanas, Á.; Carrera-Lasfuentes, P.; Arguedas, Y.; García, S.; Bujanda, L.; Calvet, X.; Ponce, J.; Perez-Aísa, Á.;Castro, M.; Muñoz, M.; et al. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. *Clin. Gastroenterol. Hepatol.* 2015, 13,906–912.e2. [CrossRef]
- 9.Masclée, G.M.; Valkhoff, V.E.; Coloma, P.M.; de Ridder, M.; Romio, S.; Schuemie, M.J.; Herings, R.; Gini, R.;Mazzaglia, G.; Picelli, G.; et al. Risk of upper gastrointestinal bleeding from different drug combinations.*Gastroenterology* 2014, 147, 784–792. [CrossRef]
- 10.Huang, J.Q.; Sridhar, S.; Hunt, R.H. Role of helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: A meta-analysis. *Lancet* 2002, 359, 14–22. [CrossRef]
- 11.Charpignon, C.; Lesgourgues, B.; Pariente, A.; Nahon, S.; Pelaquier, A.; Gatineau-Sailliant, G.;Roucaïrol, A.M.; Courillon-Mallet, A.; Group de l'Observatoire National des Ulcères de l'Association Nationale des Hépatogastroentérologues des Hôpitaux Généraux (ANGH). Peptic ulcer disease: One in five is related to neither Helicobacter pylori nor aspirin/NSAID intake. *Aliment. Pharmacol. Ther.* 2013, 38946–954. [CrossRef] [PubMed]
- 12.Levenstein, S.; Rosenstock, S.; Jacobsen, R.K.; Jorgensen, T. Psychological stress increases risk for peptic ulcer,regardless of Helicobacter pylori infection or use of nonsteroidal anti-inflammatory drugs. *Clin. Gastroenterol.Hepatol.* 2015, 13, 498–506.e1. [CrossRef] [PubMed]
- 13.McColl, K.E. Helicobacter pylori-negative nonsteroidal anti-inflammatory drug-negative ulcer.*Gastroenterol. Clin. N. Am.* 2009, 38, 353–361. [CrossRef] [PubMed]
- 14.Siddique, O.; Ovalle, A.; Siddique, A.S.; Moss, S.F. Helicobacter pylori infection: An update for the internist in the age of increasing global antibiotic resistance. *Am. J. Med.* 2018, 131, 473–479. [CrossRef] [PubMed]
- 15.Hooi, J.K.Y.; Lai, W.Y.; Ng, W.K.; Suen, M.M.Y.; Underwood, F.E.; Tanyingoh, D.; Malfertheiner, P.;Graham, D.Y.; Wong, V.W.S.; Wu, J.C.Y.; et al. Global prevalence of Helicobacter pylori infection: Systematic review and meta-analysis. *Gastroenterology* 2017, 153, 420–429. [CrossRef] [PubMed]
- 16.Zaki, M.; Coudron, P.E.; McCuen, R.W.; Harrington, L.; Chu, S.; Schubert, M.L. H. Pylori acutely inhibits gastric secretion by activating CGRP sensory neurons coupled to stimulation of somatostatin and inhibition of histamine secretion. *Am. J. Physiol. Gastrointest. Liver Physiol.* 2013, 304, G715–G722. [CrossRef] [PubMed]
- 17.El-Omar, E.M.; Oien, K.; El-Nujumi, A.; Gillen, D.; Wirz, A.; Dahill, S.; Williams, C.; Ardill, J.E.; McColl, K.E.Helicobacter pylori infection and chronic gastric acid hyposecretion. *Gastroenterology* 1997, 113, 15–24. [CrossRef]
- 18.Moss, S.F.; Legon, S.; Bishop, A.E.; Polak, J.M.; Calam, J. Effect of helicobacter pylori on gastric somatostatin in duodenal ulcer disease. *Lancet* 1992, 340, 930–932. [CrossRef]
- 19.Bhala, N.; Emberson, J.; Merhi, A.; Abramson, S.; Arber, N.; Baron, J.A.; Bombardier, C.; Cannon, C.; Farkouh, M.E.; FitzGerald, G.A.; et al. Vascular and upper gastrointestinal effects of non-steroidal anti-inflammatory drugs: Meta-analyses of individual participant data from randomized trials. *Lancet* 2013,382, 769–779.
- 20.Bjarnason, I.; Scarpignato, C.; Takeuchi, K.; Rainsford, K.D. Determinants of the short-term gastric damage caused by NSAIDs in man. *Aliment. Pharmacol. Ther.* 2007, 26, 95–106. [CrossRef]
- 21.Mössner, J. The indications, applications, and risks of proton pump inhibitors. *Dtsch. Arztebl. Int.* 2016, 113,477–483. [CrossRef] [PubMed]
- 22.Maes, M.L.; Fixen, D.R.; Linnebur, S.A. Adverse effects of proton-pump inhibitor use in older adults: A review of the evidence. *Ther. Adv. Drug Saf.* 2017, 8, 273–297. [CrossRef] [PubMed]
- 23.Pension, J.; Wormsley, K.G. Adverse reactions and interactions with H2-receptor antagonists. *Med. Toxicol.*1986, 1, 192–216. [CrossRef]
- 24.Maton, P.N.; Burton, M.E. Antacids revisited: A review of their clinical pharmacology and recommended therapeutic use. *Drugs* 1999, 57, 855–870. [CrossRef] [PubMed]

25. Mizokami, Y.; Oda, K.; Funao, N.; Nishimura, A.; Soen, S.; Kawai, T.; Ashida, K.; Sugano, K. Vonoprazan prevents ulcer recurrence during long-term NSAID therapy: Randomised, lansoprazole-controlled non-inferiority and single-blind extension study. *Gut* 2018, 67, 1042–1051. [CrossRef] [PubMed]
26. Laine, L.; Ahnen, D.; McClain, C.; Solcia, E.; Walsh, J.H. Review article: Potential gastrointestinal effects of long-term acid suppression with proton pump inhibitors. *Aliment. Pharmacol. Ther.* 2000, 14, 651–668. [CrossRef]
27. Lam, J.R.; Schneider, J.L.; Zhao, W.; Corley, D.A. Proton pump inhibitor and histamine 2 receptor antagonist use and vitamin B12 deficiency. *JAMA* 2013, 310, 2435–2442. [CrossRef] [PubMed]
28. Koivisto, T.T.; Rautelin, H.I.; Voutilainen, M.E.; Heikkinen, M.T.; Koskenpato, J.P.; Färkkilä, M.A. First-line eradication therapy for *Helicobacter pylori* in primary health care based on antibiotic resistance: Results of three eradication regimens. *Aliment. Pharmacol. Ther.* 2005, 21, 773–782. [CrossRef] [PubMed]
29. Lew, E.A. Review article: Pharmacokinetic concerns in the selection of anti-ulcer therapy. *Aliment. Pharmacol. Ther.* 1999, 13 (Suppl. S5), 11–16. [CrossRef] [PubMed]
30. Gilard, M.; Arnaud, B.; Le Gal, G.; Abgrall, J.F.; Boschhat, J. Influence of omeprazol on the antiplatelet action of clopidogrel associated with aspirin. *J. Thromb. Haemost.* 2006, 4, 2508–2509. [CrossRef] [PubMed]
31. Ghebremariam, Y.T.; Lee, J.C.; LePendou, P.; Erlanson, D.A.; Slaviero, A.; Shah, N.H.; Leiper, J.M.; Cooke, J.P. Response to letters regarding article, “unexpected effect of proton pump inhibitors.

