

PEPTIC ULCER AND ITS MANAGEMENT

Kaur Diljot

Seth GL Bihani SD College of Technical Education, Sri Ganganagar, Rajasthan, India.

Email: dsidhu789@gmail.com

(Received on Date:

Date of Acceptance:)

ABSTRACT

Ulcers are the open sore in the skin or mucous membrane. Usually ulcers are seen in duodenum, which is the first part of intestine, in stomach referred as gastric ulcer and esophagus called esophageal ulcers. Peptic ulcers are generally caused by an acid resistant bacteria called *Helicobacter pylori* (*H pylori*) which infect the stomach. *H pylori* is gram negative spiral shaped bacteria. In human it colonizes in stomach and the likelihood of infections increases with age. Peptic ulcer describes a condition in which there is a discontinuity in the entire thickness of the gastric mucosa that persists as a result of acid and pepsin in the gastric juice. The word peptic refers to the pepsin i.e., stomach enzyme, which helps in breakdown of proteins.

No. of Tables: 3

No. of References:

EPIDEMIOLOGY

Prevalence of *H pylori* infection correlates with socio-economic status rather than race with a prevalence of 80% in developing countries compared to prevalence of 20-90% in developed countries. Peptic ulcer is common among older age individual and females. *H pylori* infection is commonly seen in adult population. *H pylori* infections occur in 10% of children annually between the ages of 2 and 8 years. It is clear from the surveys conducted that the majority of person in the world are infected with *H pylori*. *H pylori* infection was diagnosed in 82% of all peptic ulcer patients and also seen in 75% of Non steroidal anti-inflammatory drugs (NSAID) users. 5-10% of the adult population have peptic ulcer in life time.

ETIOLOGY

Although *H pylori* is the major cause for peptic ulcers, other factors which cause peptic ulcer include: Non steroidal anti-inflammatory drugs, Aspirin; Zollinger Ellison Syndrome (Gastrinoma); Severe stress (e.g.: Trauma, Burns); Alcohol, smoking; Bile reflux; Pancreatic enzyme reflux; Radiation; Staphylococcus aureus exotoxin; Bacterial or viral infection

The most important one is the *H pylori* infection, non steroidal anti-inflammatory drugs, smoking and alcohol consumption.

ZOLLINGER -ELLISON SYNDROME SIGNS AND SYMPTOMS ASSOCIATED WITH PEPTIC ULCER

Burning pain mainly abdominal pain. Pain may get better or worse after eating a meal; Nausea, vomiting; Weight loss; Fatigue;

Belching; Chest pain; Anorexia & Vomiting blood

PATHOPHYSIOLOGY

Gastric acid secretion

Gastric acid secretion is a complex, continuous process in which multiple central and peripheral factors contribute to a common endpoint: the secretion of H^+ by parietal cells. Neuronal (acetylcholine, ACh), paracrine (histamine), and endocrine (gastrin) factors all regulate acid secretion (Figure 36-1). Their specific receptors (M_3 , H_2 , and CCK_2 receptors, respectively) are on the basolateral membrane of parietal cells in the body and fundus of the stomach. The H_2 receptor is a GPCR that activates the G_s -adenylyl cyclase-cyclic AMP-PKA pathway. ACh and gastrin signal through GPCRs that couple to the G_q -PLC- IP_3 - Ca^{2+} pathway in parietal cells. In parietal cells, the cyclic AMP and the Ca^{2+} -dependent pathways activate H^+, K^+ -ATPase (the proton pump), which exchanges hydrogen and potassium ions across the parietal cell membrane. This pump generates the largest known ion gradient in vertebrates, with an intracellular pH of about 7.3 and an intracanalicular pH of about 0.8.

The most important structures for CNS stimulation of gastric acid secretion are the dorsal motor nucleus of the vagal nerve, the hypothalamus, and the solitary tract nucleus. Efferent fibers originating in the dorsal motor nuclei descend to the stomach *via* the vagus nerve and synapse with ganglion cells of the enteric nervous system. ACh release from postganglionic vagal fibers directly stimulates gastric acid secretion through

muscarinic M₃ receptors on the basolateral membrane of parietal cells. The CNS predominantly modulates the activity of the enteric nervous system *via* ACh, stimulating gastric acid secretion in response to the sight, smell, taste, or anticipation of food (the "cephalic" phase of acid secretion). ACh also indirectly affects parietal cells by increasing the release of histamine from the enterochromaffin-like (ECL) cells in the fundus of the stomach and of gastrin from G cells in the gastric antrum.

ECL cells, the source of gastric histamine secretion, usually are in close proximity to parietal cells. Histamine acts as a paracrine mediator, diffusing from its site of release to nearby parietal cells, where it activates H₂ receptors. The critical role of histamine in gastric acid secretion is dramatically demonstrated by the efficacy of H₂ receptor antagonists in decreasing gastric acid secretion (*see* below).

Gastrin, which is produced by antral G cells, is the most potent inducer of acid secretion. Multiple pathways stimulate gastrin release, including CNS activation, local distention, and chemical components of the gastric contents. Gastrin stimulates acid secretion

indirectly by inducing the release of histamine by ECL cells; a direct effect on parietal cells also plays a lesser role.

Somatostatin (SST), which is produced by antral D cells, inhibits gastric acid secretion. Acidification of the gastric luminal pH to <3 stimulates SST release, which in turn suppresses gastrin release in a negative feedback loop. SST-producing cells are decreased in patients with *H. pylori* infection, and the consequent reduction of SST's inhibitory effect may contribute to excess gastrin production.

DIAGNOSIS

Test for *H Pylori*

The diagnosis of *H pylori* can be made using invasive or non invasive tests. The invasive method requires upper GI endoscopy with a mucosal biopsy taken for histology, culture or detection of urease activity.

The non invasive tests for *H pylori* do not require endoscopy or a mucosal biopsy and include the urea breath test and antibody detection test. These are less expensive than endoscopic tests. Various tests for detection of *H pylori* is given in table below.

Table 1:

Tests	Description	Advantages	Disadvantages
Antibody detection (Laboratory based)	Detects antibodies to <i>H pylori</i> in serum	Endoscopy not required	Low specificity
		Inexpensive	
		Most accurate	
Antibody detection	Qualitative detection of IgG antibody to <i>H pylori</i> in whole blood	Quick	May yield invalid results
		Inexpensive	
Urea breath test	<i>H pylori</i> urease breaks down ingested labeled	Endoscopy not required	Results are not immediate

	(urea, patients exhales labeled CO ₂)	Less expensive		
Histology	Microbial examination	High specificity and sensitivity	Require endoscopy	
			Expensive	
Culture	Culture of biopsy	Used to test for antibiotic resistance	Require endoscopy	
			High specificity	Patient discomfort
				Expensive
Biopsy urease	Urease of H pylori generates ammonia which causes a colour change	High specificity and sensitivity	Require endoscopy	
				expensive

In case of invasive test histological identification has a sensitivity and specificity greater than 95% and allows classification of gastritis that may be present. Culture has a specificity of 100% and enables susceptibility testing of anti-microbial agents to detect resistance and permit appropriate treatments.

The sensitivity and specificity of Biopsy urease test which detect H pylori urease enzyme activity are above 90%. Urea breath test is based on urease production by H pylori. The carbon 13 (Nonradioactive isotope) and carbon 14 (Radioactive isotope) tests require that the patient ingest labeled urea, which is broken down in the stomach to ammonia and labeled bicarbonate. The labeled bicarbonate is absorbed in the blood and excreted in breath.

Antibody detection test are used to detect circulating immuno globulin IgG directed against *H pylori*. ELISA (Enzyme Linked Immuno Sorbent Assay) have been approved for use and have a sensitivity and specificity of 90%. The method use an enzyme to detect the binding of Ag & Ab. This enzyme convert's colorless substrate to colored

product indicate the presence of Ag-Ab binding. (Ag-Antigen & Ab-Antibody).

Tests for H pylori (with the exception of antibody detection) may produce false negative results. If antibiotics or bismuth are taken within the previous four weeks, or if a proton pump inhibitor is taken within the previous two weeks. These agents temporarily suppresses H pylori and cause false negative results.

The selection of a specific H pylori test depends on patient specific factor and the clinical situation. Antibody (Ab) detection tests are the initial screening test of choice because they are quick, inexpensive and less invasive than endoscopic biopsy tests.

TREATMENT

Recommended treatment may include:

Life Style Changes

Doctors used to recommend eating bland foods with milk and only small amount of food with each meal. People who find that certain foods cause irritation should discuss the problem with their physicians. Smoking has been shown to delay ulcer healing and has been linked to ulcer recurrence. Therefore people with ulcer should not smoke.

Medications

Several types of medication are given for the treatment of stomach and duodenal ulcer.

Which include,

H₂ blockers to reduce the amount of acid in the stomach by blocking histamine, which is a powerful stimulant of acid secretion; Acid pump inhibitors which completely block stomach acid production by stopping the stomach's acid pump the final step of acid secretion; Mucosal protective agents to shield the stomach's mucous lining from the damage of acid and they do not inhibit the release of acid. When treating *H. pylori*, these medications are often used in combination with antibiotics.

Surgery

In most cases, antiulcer medicines heal ulcers quickly and effectively, and eradication of *Helicobacter pylori* prevents most ulcers from recurring. However, people who do not respond to medication or who develop complications may require surgery.

At present, laparoscopic surgery is performed to treat ulcers and include,

Vagotomy: A procedure that involves cutting parts of the vagus nerve (a nerve that transmits message from the brain to the stomach) to interrupt messages sent through it and thereby, reducing the acid

secretion. ;Antrectomy: An operation to remove the lower part of the stomach (antrum), which produces a hormone that stimulates the stomach to secrete digestive juices. Sometimes a surgeon may also remove an adjacent part of the stomach that secretes pepsin and acid. The vagotomy is usually done in conjunction with an antrectomy; Pyloroplasty: A surgical procedure that may be performed along with a vagotomy, in which the opening in to the duodenum and small intestine (pylorus) are enlarged, enabling contents to pass more freely from the stomach. In the future laproscopic methods may become the standard surgical treatment.

Goals of treatment

Treatment goals are the relief of discomfort and protection of the gastric mucosal barrier to promote healing. Cessation of the causative agent and antacids may be sufficient for treating mild cases. Most patients require an H₂ receptor antagonist or a pump inhibitor, which has proved to provide a faster and more reliable healing than antacids. Either an H₂ receptor blocker or a proton pump inhibitor can be used as a first line agent. In refractory cases, sucralfate also may be indicated.

ANTI MICROBIAL AGENTS	H2 RECEPTOR BLOCKERS	ANTACIDS
Amoxicycline	Cimitidine	Sodium bicarbonate
Bismuth compounds	Rantidine	Aluminium hydroxide
Clarithromycin	Famotidine	Magnesiumhydroxide
Tetracycline	Nizatidine	Calcium carbonate
Metronidazole		

PROTON PUMP INHIBITORS	MUCOSAL PROTECTIVE AGENTS	ANTIMUSCRANIC AGENTS
Esomperazole	Sucrafalate	Dicyclomine
Omeprazole	Bismuth subsalicylate	PROSTAGLANDINS
Lansoprazole		Misoprostol

CLASSIFICATION OF DRUGS USED IN TREATMENT:

Table 2: Drug Category

Antacids	Aluminium containing and magnesium containing antacids can be helpful in relieving symptoms of gastritis by neutralizing gastric acids. These agents are inexpensive and safe
Drug Name	Aluminium and magnesium hydroxide (Maalox, Mylanta). It neutralizes gastric acidity, resulting in increase in stomach and duodenal bulb pH. Aluminium ions inhibit smooth muscle contraction, thus inhibiting gastric emptying. Magnesium and aluminum antacid mixtures are used to avoid bowel function changes.
Adult dose	2 – 4 tsp PO qid prn.
Pediatric Dose	0.5 ml/kg PO qid prn
Contraindications	Documented hypersensitivity
Interactions	Both drugs reduce efficacy of fluoroquinolones, corticosteroids, benzodiazepines, and phenothiazines; aluminum and magnesium potentiate effects of valproic acid, sulfonylurea, quinidine, and levodopa
Pregnancy	C-Safety for use during pregnancy has not been established
Precautions	Use aluminum containing antacids with caution in patients who have recently suffered a massive upper GI hemorrhage
Drug Category	H ₂ -receptor antagonists – inhibit the action of histamine on the parietal cell, which inhibits acid secretion. The four drugs in this class are all equally effective and are available over the counter in half prescription strength for heartburn treatment. Although the IV administration of H ₂ blockers may be used to treat acute complications, the benefits are yet to be proven.
Drug Name	Cimetidine (Tagamet) – inhibits histamine at H ₂ receptors of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen ion concentrations.
Adult Dose	150 mg PO qid; not exceed 600 mg/d 50 mg/dose IV/IM q6-8h; not to exceed 400 mg/d.

Pediatric Dose	Not established. Suggested Dose: 20-40mg/kg/d PO/IV/IM divided q4h.
Contraindications	Documented hypersensitivity.
Interactions	Can increase blood levels of theophylline, warfarin, tricyclic antidepressants, triamterene, phenytoin, quinidine, propranolol, metronidazole, procainamide, and lidocaine.
Pregnancy	B-Usually safe but benefits must outweigh the risks.
Precautions	Elderly patients may experience confusional states; may cause impotence and gynecomastia in young males; may increase levels of many drugs; adjust dose or discontinue treatment if changes in renal function occur.
Drug Name	Famotidine (Pepcid) – competitively inhibits histamine at the H ₂ receptor of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations.
Adult Dose	40mg PO qhs 20mg/dose IV qhs; not to exceed 40 mg/d
Pediatric dose	Not established Suggested dose: 1-2 mg/kg/d PO/IV divided qhs; not to exceed 40 mg/dose
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of ketoconazole and itraconazole
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	If changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment
Drug Name	Nizatidine (AxiD) - Competitively inhibits histamine at H ₂ receptors of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations.
Adult Dose	300 mg PO hs or 150 mg PO bid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	None reported
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment
Drug Name	Ranitidine (Zantac) - Competitively inhibits histamine at the H ₂ receptors of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations.
Adult Dose	150 mg PO bid or 300 mg PO qhs; not to exceed 300 mg/d 50 mg/dose IM/IV qhs
Pediatric Dose	<12 years: Not established

	>12 years: 1.25-2.5 mg/kg/dose PO qhs; not to exceed 300 mg/d 0.75-1.5 mg/kg/dose IV/IM qhs; not to exceed 400 mg/d
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of ketoconazole and itraconazole; may alter serum levels of ferrous sulfate, diazepam, nondepolarizing muscle relaxants, and oxaprozin
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment
Drug Category: <i>Proton pump inhibitors</i> - Bind to the proton pump of parietal cell, inhibiting secretion of hydrogen ions into gastric lumen. Proton pump inhibitors relieve pain and heal peptic ulcers more rapidly than H ₂ antagonists do. Drugs in this class are equally effective. They all decrease serum concentrations of drugs that require gastric acidity for absorption, such as ketoconazole or itraconazole. Five drugs are now FDA approved in this category. Omeprazole will soon go off patent and be available as a generic. Side effect is achlorhydria	
Drug Name	Lansoprazole (Prevacid) - Decreases gastric acid secretion by inhibiting the parietal cell H ⁺ /K ⁺ ATP pump.
	Used for up to 4 week to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 wk to treat all grades of erosive esophagitis.
Adult Dose	30 mg qid for 4-8 week
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of ketoconazole and itraconazole; may increase theophylline clearance
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Adjust dose in liver impairment
Drug Name	Omeprazole (Prilosec) -- Decreases gastric acid secretion by inhibiting the parietal cell H ⁺ /K ⁺ ATP pump.
	Used for up to 4 week to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 wk to treat all grades of erosive esophagitis.
Adult Dose	20 mg PO qid for 4-8 week
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of itraconazole and ketoconazole; may increase toxicity of warfarin, digoxin, and phenytoin
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Bioavailability may be increased in elderly patients

Drug Name	Esomeprazole (Nexium) - S-isomer of omeprazole. Decreases gastric acid secretion by inhibiting the parietal cell H ⁺ /K ⁺ ATP pump.
	Used for up to 4 week to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 week to treat all grades of erosive esophagitis.
Adult Dose	20-40 mg PO qid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	Amoxicillin or clarithromycin may increase plasma levels of esomeprazole when used concurrently; may reduce absorption of dapsone; may increase levels of diazepam and GI absorption of digoxin; may decrease absorption of iron, ketoconazole and itraconazole
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Symptomatic relief with proton pump inhibitors may mask symptoms of gastric malignancy
Drug Name	Rabeprazole (Aciphex) -Decreases gastric acid secretion by inhibiting the parietal cell H ⁺ /K ⁺ ATP pump. For short-term (4-8 week) treatment and symptomatic relief of gastritis.
	Used for up to 4 week to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 wk to treat all grades of erosive esophagitis.
Adult Dose	20 mg tab PO qid 4-8 week
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of itraconazole and ketoconazole; may increase toxicity of warfarin, digoxin, and phenytoin
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Symptomatic relief with proton pump inhibitors may mask symptoms of gastric malignancy
Drug Name	Pantoprazole (Protonix) -Decreases gastric acid secretion by inhibiting the parietal cell H ⁺ /K ⁺ ATP pump. For short-term (4-8 week) treatment and symptomatic relief of gastritis.
	Used for up to 4 week to treat and relieve symptoms of active duodenal ulcers. Physicians may prescribe for up to 8 week to treat all grades of erosive esophagitis.
Adult Dose	40 mg PO qid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity

Interactions	May decrease effects of itraconazole and ketoconazole; may increase toxicity of warfarin, digoxin, and phenytoin
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Symptomatic relief with proton pump inhibitors may mask symptoms of gastric malignancy
Drug Category: <i>Gastrointestinal agents</i> - Are effective in the treatment of peptic ulcers and in preventing relapse. Their mechanism of action is not clear. Multiple doses are required, and they are not as effective as the other options. Side effects are constipation, dry mouth and nausea.	
Drug Name	Sucralfate (Carafate) - Binds with positively charged proteins in exudates and forms a viscous adhesive substance that protects the GI lining against pepsin, peptic acid, and bile salts. Used for short-term management of ulcers.
Adult Dose	1 g PO qid
Pediatric Dose	Not established Suggested dose: 40-80 mg/kg/ divided qhs
Contraindications	Documented hypersensitivity
Interactions	May decrease effects of ketoconazole, ciprofloxacin, tetracycline, phenytoin, warfarin, quinidine, theophylline, and norfloxacin
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in renal failure and impaired excretion of absorbed aluminum
Drug Name	Misoprostol (Cytotec) - A prostaglandin analog that protects the lining of the GI tract by replacing depleted prostaglandin E1 in prostaglandin inhibiting therapies.
Adult Dose	200 mcg PO qid with food; if not tolerated, decrease to 100 mcg qid or 200 mcg bid with food
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	None reported
Pregnancy	X - Contraindicated in pregnancy
Precautions	Caution with elderly patients and in renal impairment

AYURVEDIC MEDICATION

Aloe M.P Plus

Aloe M.P Plus is a powerful natural, 100% organic aloe mucilaginous polysaccharides (AMP) based non toxic supplement. Aloe vera polysaccharides are believed to neutralize harmful enzymes and proteins and work together with your body to reverse

stomach disorders, immune disease and many other common ailments.

Manuka honey

Manuka honey has been found to inhibit the growth of the bacteria, *Helicobacter pylori*. The procedure to take summe rglow

UMF 16 + Manuka honey for digestive health is given as

i. Take a teaspoon to a table spoon of summergeglow UMF 16 + Manuka honey three-four times a day. Ideally one hour before meals and again at bed time.

ii. Should not drink water immediately after having the honey so as not to dilute the honey.

iii. Take honey with bread, toast or cracker biscuit holds the honey in the stomach for as long as possible.

iv. Many people have experienced good results if they have the honey straight from the teaspoon.

v. Summergeglow UMF 16 + Manuka honey is pure honey. So it does not interfere with regular medications.

vi. Adjust the amount and frequency to suit your own needs. Most people have a generous amount of the honey initially they reduce it as they feel warranted.

vii. A little discomfort was experienced by a few for a very short period.

Summer glow UMF 16 + Manuka honey is 100% pure honey so it is safe to have as much as desired and as often as wished. (excepts for diabetics)

- **Extra virgin Siberian pine nut oil**

Extra virgin Siberian pine nut oil stops ulcer related stomach and duodenal ulcer quickly. It has got natural antioxidant property. Clinical studies proves that peptic ulcer are also caused by free radicals. So this oil acts as an effective healing agent for such type of ulcers.

HERBS

Certain herbs are recommended by herbal specialist for peptic ulcers. They are

Astragalus (*Astragalus membranaceus*): Used traditionally to treat stomach ulcers.

Barberry (*Berberis vulgaris*): This herbs contains active substances called berberine alkaloids. These substances have been shown to combat infection and bacteria. For this reason barberry is used to ease inflammation and infection of the gastro intestinal tract.

Barberry has also been used traditionally to improve appetite. Bilberry (*Vaccinium myrtillus*): Bilberry fruits helps to prevent stomach ulcer related to a variety of factors including stress, medications and alcohol.

Cat's claw (*Uncaria tomentosa*): The bark and root of this herb have been used among indigenous people of the rainforest for centuries to treat a variety of health problems including ulcers and other gastro intestinal disorders. The benefits of this herb may be due to its ability to reduce inflammation.

Cranberry (*Vaccinium spp*): May have properties that help to prevent

H pylori infection. Dong Quai (*Angelica sinensis*): Animal studies with dong quai, soothe ulcers, but studies in needed before a definitive conclusion can be drawn.

Garlic (*Allium sativum*): Some studies suggest that high amounts of garlic may protect against stomach cancer, which is a potential complication of H pylori peptic ulcers. This is controversial, however and high amounts of garlic may infact cause gastro intestinal distress.

Licorice (*glycyrrhiza glabra*): This herb is a demulcent (soothing, coating agent) that has long been valued for its use in food and medicinal remedies, including treatment of ulcers. Some licorice root extracts, known as deglycyrrhizenated licorice (DGL), still have the healing properties of licorice without the harmful effects. DGL may be

better for stomach or duodenal ulcers than Glycyrrhiza glabra and may even prove as effective as some prescription drugs for stomach ulcers. Slippery elm (Ulmus fulva): Although there has been little scientific research on slippery elm, it has a long history of use based on clinical experience. Gastritis (stomach inflammation) and peptic ulcer are among the conditions that seem to respond well to slippery elm. Turmeric (Curcuma longa): Turmeric has long been used in both Ayurvedic and Chinese medicine to treat digestive disorders. In an animal study, for example, extracts of turmeric root reduced the release of acid from the stomach and protect intestinal walls and ulcers from injuries such as gastritis or inflammation. Further studies are needed to know to what extent these protective effects apply to human volunteers as well (Note: at very high doses, turmeric may induce ulcers). It is very important to stick with the dose recommended by an herbal specialist. Angelica (Angelica archangelica) German chamomile (Matricaria recutita) Lemon balm (Melissa officinalis) Licorice Milk thistle (Silybum marianum) Peppermint (Mentha piperita) Additional herbs that have been used clinically by herbal specialists to treat peptic ulcers include; Calendula (Calendula officinalis): Used in the United States during the 19th century to treat stomach ulcers. Capsaicin: The active ingredient in cayenne (Capsicum frutescens/Capsicum Spp) Marshmallow (Althea officinalis)

HELICOBACTER PYLORI

ERADICATION

Effective treatment

Treatment regimens which have been shown in randomized controlled trials (RCTs) to be most effective consist of two antibiotics combined with either bismuth or a proton pump inhibitor or H₂ antagonist. Three regimens are mentioned here.

Standard triple therapy

Consist of a two week course of bismuth subcitrate (4 □ 120mg daily) with metronidazole (3 □ 400 mg daily) and tetracycline (4 □ 500mg daily). It is cheap and has been shown to eradicate H pylori in almost 95% of patients. This standard triple therapy have given along with H₂ antagonist or proton pump inhibitor, but the benefit of this have not been evaluated. The H₂ antagonist or proton pump inhibitor is stopped once healing has occurred.

Other triple therapies

Five new regimen which has found to achieve eradication rates of 95% or over have been evaluated. One week of omeprazole (2 □ 20mg daily), Amoxicillin (2 □ 1000mg daily), clarithromycin (2 □ 500mg daily).

Alternatively one week of : Omeprazole (2 □ 20 mg daily), metronidazole (2 □ 400 mg daily), clarithromycin (2 □ 250mg daily). These treatment have not been compared directly to the standard triple therapy. Several other alternative regimens have been proposed.

Success of eradication therapy

Success depends on:

Patient compliance: Patient should be counseled concerning the importance of completing the course of therapy and warn against the possible side effects they may

experience. Metronidazole and Tinidazole medications are not effective in populations with resistance to H pylori organisms. The

standard triple therapy has proven to be very effective and its low cost make it a choice of treatment.

HEALING OPTIONS

Table 3

Herbal Home Remedy	Banana
	Chandan
	Lime
	Vegetable juice
	Almond milk
Ayurvedic supplements	Avucid
	Avipattikar churna
	Mahashankha Bati
	Prawal Bhasma
Diet	The diet of the patient suffering from a peptic ulcer should be planned so as to provide adequate nutrition, while affording rest to the disturbed organs, maintaining the neutralization of excess gastric acid and there by inhibiting the production of acid and reducing mechanical and chemical irritation. Milk, cream, butter, fruits, fresh raw and boiled vegetables, natural foods and natural vitamin supplements constitute the best diet.
Yoga	Yoga will also help in the healing of peptic ulcer disease.
	Vajrasana
	Padmasana
	Pawanmuktasan
	Bhujangasana
	Paschl mothanasana

LIFE STYLE:

Doctors generally recommend eating bland foods with mild and only small amounts of food with each meal. Dietary and other life style measures that should help include;

Eat a diet rich in fiber, especially from fruits and vegetables; this may reduce the risk of developing an ulcer in the first place and may speed up the recovery. The vitamin A may be an added benefit from these foods.

Foods containing flavanoids, like apples, celery, cranberries (including cranberry

juice), onions, and tea may inhibit the growth of *Helicobacter pylori*.

Quit smoking

Receive treatment for alcohol abuse

Avoid coffee, including decaffeinated coffee, as well as carbonated beverages all of which can increase the production of stomach acid

Reduce stress with regular use of relaxation techniques such as yoga or medication. These practices may help to lessen pain and reduce

the need for non-steroidal anti-inflammatory agent.

Nutrition and dietary supplements

Dietary measures that should help are, Eat a diet rich in fiber, especially from fruits (including fruit juices) and vegetables. It may reduce the risk of developing ulcer. The vitamin A may be an added benefit from these foods.

Foods containing flavanoids, like apples celery, cranberries onions and tea may inhibit the growth of H pylori.

Avoid beverages that should irritate the lining of the stomach or increase acid production including coffee, alcohol and carbonated beverages.

Gamma-linolenic acid (GLA)

Gamma linolenic acid (GLA) from evening primrose oil (EPO) may have anti ulcer properties. Gamma linolenic acid is an essential fatty acid (EFA) in the Omega 6 family that is found primarily in plant based oils, including EPO and borage seed oil.

Omega-3-fatty acids

Treatment with Omega-3-fatty acids reduced the risk of ulcers caused by NSAIDS.

Probiotics

Probiotics inhabit the lining of the intestine and protect from the entry of bad infections that can cause disease. Lactobacillus acidophilus is the most commonly used probiotics.

Vitamin C

High dose of vitamin C treatment for four weeks are effectively in the treatment of H pylori infection in some people.

CONCLUSION

Peptic ulcer can be easliy cured by a proper diet control and well lifestyle. Research are carried out for the development of optimal and most cost effective drug regimen. Future research will surely provide us with safer drug with minimal side effect regimen and perhaps a vaccine. Newer drug that spare the GI tract and decrease NSAID related morbidity and mortality will be soon available in market.

REFERENCES

Anderson J, Gona Zales. J.H. Pylori infection Review of the guidelines for diagnosis and treatment. 2000, 55; 44-49.

Barr.M.Buckley M,O. Moracin Non Steroidal anti inflammatory drugs and H pylori, Aliment pharmacol

Blaser M.J Parsonnet J: The bacteria behind ulcers I. AM 274, 104, 1996.

Danesh J. H pylori infection and gastric cancer systematic review of the epidemiological studies. Aliment pharmacol ther 1999; 13; 851-856.

Del.Valle J. Cohen H, Laine L et.al. Acid peptic disorders In Yamada.T, Aplers DH, Laine L et.al, eds. Textbook of Gastro enterology 3rd edition Philadelphia, Lippincott Williams and Wilkins 1999; 1370-1444.

Delvalle J, Scherman J.M. Zollenger – Ellison syndrome 1999, 1445-1462.Fifth edition, Pharmacotherapy, A pathophysiologic Approach.,

Joseph .T. Dipiro, Robert L. Talbert, Gary C. Yee, Gary R. Matzke, Barbara G. Wells, L. Mechael Posey. Graham Dy. Therapy of H pylori; Current status and issues. Gastro enterology 2000; 118 (suppl) S2-S8.

Huang J2, Hunt RH H-pylori and gastric cancer – Alimetn pharmacol ther 2000 (suppl 3); 48-54; Review.

Issel bacher K.J, Podolsky D.K: Approach to the patient with GI disease in Wilson JD, Braunwald E. et al, Harrison's principles of internal medicine, New York, MC. GrawHill, 1991: 1213-1216.

James.M. Crawford M.D, PhD, Associate professor of pathology, Director – Programme in gastro intestinal pathology.

Yale University School of Medicine, New Haven.

Janowitz HD: Approach to the patient with GI symptoms in Sachar DB, Waye J.D et.al eds. Pocket guide to Gastro enterology Baltimore, Williams and Wilkins 1989: 1-7.

Kuipers E.J: Exploring the link between H pylori and gastric cancers. Aliment pharmacol ther 1993; 13 (suppl); 3-11.

MacGowan et al: Helicobacter Pylori and gastric acid, biological and therapeutic implication, Gastro enecology 110; 926; 1996.

Megrand. F: How should H pylori infection be diagnosed? Gastro entrology 1997, 113 (suppl); S93-S98.

Morson DC et al: Gastro intestinal Pathology 3rd edition, Oxford Black well Scientific Publications 1992.

Ng TM, Fock K.M, Khor J.L, et. al NSAIDS, H pylori and bleeding Gastric ulcer. Aliment Pharmacol Ther 2000; 14; 203-209.

Robbins and Cotran pathological basis of disease, 7th edition. Kumar Abbas Fausto, Page No: 816-820.

Robbins pathological basis of disease, Cotran, Kumar, Collins Sixth edition Page No: 793-796.Text book of pathology, Harsh Mohan, Page No: 556-568.

Sachs G. Shin M, Munson K et.al. The control of gastric acid and H pylori eradication. Aliment pharmacol ther 2000; 14; 1384-401 Review.

Talley N.J, Holtmann G. Approach to the patient with dyspepsia and related functional GI complaints in Yamada T, Aplers DH, Laine L et. al, eds. Text book of Gastroenterology, 3rd edition, Philadelphia,

Lippincott Williams and Wilkins 1999; 660-693.

Vaira D, Holton J, Menegatti M. et. al. Invasive and non invasive test for H pylori infection Aliment pharmacol ther 2000, (suppl), 13-22 Review.

Vaira. D, Menegatti M, Miglioli M. Gastro enterology 1997, 113 (suppl); S78-S84.

Walsh JH, Peterson WL. The treatment of Helicobacter Pylori infection in the management of ulcer disease 1995, 333, 984-991.

White head R: Gastro intestinal and Oesophageal Pathology; 2nd edition London, Churchill Livingstone, 1995.

Williams M.P, Pounder RE H.Pylori, from the benign to the malignant Am J. Gastro enterol 1999, 94 (suppl); S11-S16.

Yardely JH, Hendrix T.R Gastritis, duodenitis and associated ulcerative lesions. In Yamada T, Aplers DH, Laine L et.al, eds. Text book of gastro enterology, 3rd ed. Philadelphia, Lippincott Williams and Wilkins; 1999; 1463-1499.

