Management of Peptic Ulcer-A Review

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Abstract- Peptic ulcer disease (PUD) is a break in the inner lining of the stomach, the first part of the small intestine, or sometimes the lower esophagus. An ulcer in the stomach is called a gastric ulcer, while one in the first part of the intestines is a duodenal ulcer. The most common symptoms of a duodenal ulcer are waking at night with upper abdominal pain and upper abdominal pain that improves with eating. With a gastric ulcer, the pain may worsen with eating. The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. About a third of older people have no symptoms. Complications may include bleeding, perforation, and blockage of the stomach. Bleeding occurs in as many as 15% of cases. Common causes include the bacteria Helicobacter pylori and nonsteroidal anti-inflammatory drugs (NSAIDs). Other, less common causes include tobacco smoking, stress due to serious illness, Behcet disease, Zollinger-Ellison syndrome, Crohn disease, and liver cirrhosis. Older people are more sensitive to the ulcer-causing effects of NSAIDs. The diagnosis is typically suspected due to the presenting symptoms with confirmation by either endoscopy or barium swallow. H. pylori can be diagnosed by testing the blood for antibodies, a urea breath test, testing the stool for signs of the bacteria, or a biopsy of the stomach. Other conditions that produce similar symptoms include stomach cancer, coronary heart disease, and inflammation of the stomach lining or gallbladder inflammation. Diet does not play an important role in either causing or preventing ulcers.

Index terms- epidemiology, etiology, pathophysiology, diagnosis, treatment

DEFINITION

Ulcers are the open sore in the skin or mucous membrane. Usually ulcers are seen in duodenum, which is the first pact of intestine, in stomach refused as gastric ulcer and esophagus called esophageal ulcers. Peptic ulcers are generally caused by an acid resistant bacterium called Helicobacter pylori (H pylori) which infect the stomach. H pylori are 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 persist 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.

EPIDEMOLOGY

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 (Gastreinoma)
- 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 antiinflammatory drugs, smoking and alcohol consumption.

ZOLLINGER -ELLISON SYNDROME

Zollinger Ellison syndrome occurs from a gastrienoma, a tumor in the pancreas. Zollinger Ellison syndrome is characterized by gastric acid hyper secretion and also by the recurrent peptic ulceration that results from a gastrin producing tumor (gastrinoma). This disease can be distinguished from peptic ulcer by the demonstration of fasting hyper gastrinoma. This tumor may be localized or diffuse tumor. The presence of hyper gastrinoma leads to hyper secretion. The treatment is based on the presence or absence of peptic ulcers, esophagitis, diarrhoea, and gastrinoma, which may be malignant. The major drug of choice is the proton pump inhibitors for managing hyper secretion of gastric acid. Treatment should be instituted with omeprazole 60 mg/d.

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 lose
- Fatigue
- Belching
- Chest pain
- Anorexia
- Vomiting blood

• In children certain medical condition can contribute to the development of ulcer.

PATHOPHYSIOLOGY

Gastric acid secretion

A minimal level of gastric acid secretors is necessary for the formation of peptic ulcers. Thus gastric acid serves as a cofactor with H pylori infection or use of non-steroidal anti-inflammatory drugs. Basal or nocturnal acid secretion is generally increased in a patient with duodenal ulcer. Factor responsible for acid hyper secretion include increased parietal cell mass, increased basal secretory drive and increased post-prandial secretory drive. Acid hyper secretions may also be a consequence of H pylori infection

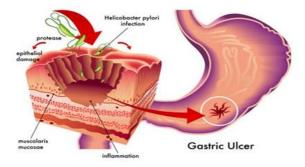


Figure 3: Peptic ulcer diseas

Pepsin

It appears to play a critical role in the proteolytic activity involved in ulcer formation. Gastric mucosal cell secrete two types of proteolytic proenzymes. Pepsinogen is produced only in the chief and mucous neck cells of the acid secreting mucosa, where as pepsinogen II is found in antral mucosa. Pepsin is activated by acidic pH, inactivated reversibly at pH 4 and irreversibly destroyed at pH 7. Pepsinogen I secretion is directly proportional to the rate of acid secretion.

Helicobacter pylori-



Figure 3: Peptic ulcer disease

It is a spiral shaped pH sensitive, gram negative micro aerophilic bacterium that resides between the mucous layer and surface epithelial cells in the stomach or any location where gastric type epithelium is found.

The combination of its spiral shape and flagella permits the H pylori to move from the lumen of the stomach, where the pH is low to the mucous layer where the local pH is neutral. The acute infection is accompanied by transient hypochlorhydria, which permits the organism to survive in the acidic gastric juice. The exact method by which H pylori initially induces hypochlorhydria is unclear. One theory is that H pylori produces large amount of urease, which hydrolyse urea in the gastric juice and converts it to ammonia and carbon dioxide. The local buffering effect of ammonia protects the organism from the lethal effect of acid. H pylori also produce acid inhibiting proteins which allow it to adapt to the low pH environment of the stomach. H pylori attach itself to gastric type epithelium by adherence pedestals, which prevent the organism from being shed during cell turn over and mucus secretion.

- H pylori contributes to gastric mucosal injury by
- Direct mechanisms
- Alteration in the immune / inflammatory response
- Hyper gastrinemia leading to increased acid secretion.,

Direct mucosal damage is produced by elaborating bacterial enzymes (lipases, proteases and urease), virulence factors like (vacuolating cytotoxin, cytotoxin associated gene proteins and growth inhibiting factor) and adherence. Lipases and protease degrade gastric mucus, ammonia produced by urease may be toxic to gastric epithelial cells, and bacterial adherence enhances the uptake of toxins into gastric epithelial cells.

Non-Steroidal Anti Inflammatory Drugs

Non-steroidal anti-inflammatory drugs cause gastric mucosal damage by two important mechanisms (a) direct or topical irritation of the gastric epithelium and (b) Systemic inhibition of endogenous GI mucosal prostaglandin synthesis

Systemic inhibitions of prostaglandin play the predominant role in the development of gastric ulcer. Cycloxygenase (COX) is the rate limiting enzyme in

the conversion of arachidonic acid to PGs and is inhibited by Non-steroidal anti-inflammatory drugs.

DIAGNOSIS

Test for H Pylori

- The diagnosis of H pylori can be made using invasive or noninvasive tests. The invasive method requires upper GI endoscopy with a mucosal biopsy taken for histology, culture or detection of urease activity.
- The noninvasive 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.
- 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 (Non radio active 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) has been approved for use and has a sensitivity and specificity of 90%. The methods use an enzyme to detect the binding of Ag & Ab. This enzyme converts 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 with in the previous four weeks, or if a proton pump inhibitor is taken with in 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 stomachs acid pump the final step of acid secretion.

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- c. Anticholenergics Pirenzepine, Propantheline, Oxyphenonium.
- d. Prostaglandin analogues: Misoprostol, Enprostil, Rioprostil.
- 2. Neutralization of gastric acid (Antacids)
- a. Systemic antacid: Sodium bicarbonate, Sodium citrate
- b. Non systemic antacids: Magnesium hydroxide, Magnesi trisilicate, Magaldrate, Aluminiu hydroxide gel, Calcium carbonate.
- 3. Ulcer protective: Sucralfate, Colloidal bismuth sub citrate CBS).
- 4. Ulcer healing drugs: Carbenxolone sodium.
- 5. Anti H pylori drugs : Amoxicillin, Clarithromy Metronidazole, Tinidazole, Tetracyclin

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
- Contraindictions

- 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

- H2-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 H2 blockers may be used to tret acute complications, the benefits are yet to be proven.
- Drug Name
 - Cimetidine (Tagamet) inhibits histamine at H2 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, warfrain, 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 makes; 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 H2 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 H2 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 H2 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:

Proton pump inhibitors - 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 H2 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: Gastrointestinal agents - 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.t

AYURVEDIC MEDICATION

Aloe M.P Plus

Aloe M.P Plus is a powerful natural, 100% organic aloe mucilaginous polysaccharides (AMP) based nontoxic supplement. Aloevera polysaccharides are believed to neutrilise 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 summerglow UMF 16 + Manuka honey for digestive health is given as

- 1. Take a teaspoon to a table spoon of summerglow UMF 16 + Manuka honey three-four times a day.
- 2. Ideally one hour before meals and again at bed time.
- 3. Should not drink water immediately after having the honey so as not to dilute the honey.
- Take honey with bread, toast or cracker biscuit holds the honey in the stomach for as long as possible.
- 5. Many people have experienced good results if they have the honey straight from the teaspoon.
- 6. Summer glow UMF 16 + Manuka honey is pure honey. So it does not interfere with regular medications.
- 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.
- 8. 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 apetite.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 back 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.

- 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 specialist 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 cryenne (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 makes it a choice of treatment.

HEALING OPTIONS

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.

PREVENTIONOF PEPTIC ULCER

1 Avoidance of drug-induced gastric ulcers

Patients receiving medications as non-steroidal anti-inflammatory drugs, the anticoagulant drug warfarin, corticosteroids or the anti-osteoporotic drug alendronate may be more prone to gastric ulcer. Non-steroidal anti-inflammatory drugs are valuable therapeutics that act not only as anti-inflammatory, but also as analgesics and antipyretics. They are used in a wide variety of clinical scenarios, including arthritis and other musculoskeletal disorders. Unfortunately, their use has been limited by their gastric ulcer-inducing effects. Nearly 25 % of chronic users of these drugs develop gastric ulcer disease

2 Life style risk factors

Several. studies implied that modulating life style factors as dietary factors, controlling stress, reducing smoking and alcohol intake may directly prevent the initiation of gastric ulcers, especially in predisposed people. Some even suggested certain physical exercises to reduce the risk of ulcer formation or recurrence. Such exercises were seen to directly improve psychological and cardiovascular conditions and thus may be indirectly related to decreasing gastric ulcer development.

3 Diet

Diet rich in fibers may decrease the risk of developing gastric ulcers by about 50%. Fiber found in fruits and vegetables is particularly protective, as vitamin A contained in many of these foods may increase the benefit. Milk, previously thought to aid in decreasing ulcer symptoms, actually encourages the production of acid in the stomach, although moderate amounts (2-3 cups/day) appear to do no harm. However, yogurt may protect against gastric ulcer, as it contains probiotics. Coffee (caffeinated and decaffeinated), soft drinks and fruit juices with citric acid increase stomach acid production. Although no studies have proven that any of these drinks contribute to ulcers, consuming more than 3 cups of coffee per day may increase susceptibility to Helicobacter pylori infection

As a body response to stress, many diseases may develop. There is debate as to whether psychological stress can influence the development of gastric ulcers. Some studies still suggest that stress may predispose a person to ulcers or prevent existing ulcers from healing. Some even believe that the relationship between stress and ulcers is so strong that people with ulcers should be treated for psychological conditions. Stress causes the digestive tract to slow down and more gastric acid is allowed to accumulate in the stomach. Increased stomach acidity may predispose to or aggravate an already present ulcer.

5 Smoking

Cigarette smoking appears to be a risk factor for the development and recurrence of gastric ulcer. The incidence of gastric ulcer is higher among smokers than non-smokers. Compared with non-smokers, people who smoke cigarettes are twice as likely to develop gastric ulcer. Smoking may lead to initiation of ulceration, slow ulcer healing and an increased risk of gastric ulcer recurrence. Smoking may have an inconsistent effect on gastric acid secretion.

CONCLUSION

Gastric ulcer is a multi-factorial disease that has become a real socio-economic burden and opposes a great challenge in its treatment. Prevention is better than cure, as they say. Usage of medications designed for treatment of gastric ulcer as a means for its prevention is faced by several drawbacks; as limited effectiveness of these drugs in ulcer prevention, numerous side effects of available anti-ulcer drugs and the cost of gastric ulcer medications. Consequently, separate line of research has been devoted to investigate preventive measures of gastric ulcer. Despite of the size of investigations done on this subject, prevention of gastric ulcer is still a challenge especially in predisposed groups. Herbal compounds can provide an alternative preventive means for gastric ulcer as they are safer, cheaper and usually having limited, if any, side effects. For reaching the optimal remedy that can prevent gastric ulcer formation, more investigations are definitely still needed.

REFERENCES

4 Psychological factors: stress

- [1] Abdiev, S.; Ahn, K.S.; Khadjibaev, A.; Malikov, Y.; Bahramov, S.; Rakhimov, B.; Sakamoto, J.; Kodera, Y.; Nakao, A. & Hamajima, N. (2010) Helicobacter pylori infection and cytokine gene polymorphisms in Uzbeks. Nagoya J. Med. Sci., vol. 72, p.p. 167-172.
- [2] Aburaya, M.; Tanaka, K.; Hoshino, T.; Tsutsumi, S.; Suzuki, K.; Makise, M.; Akagi, R. & Mizushima, T. (2006). Heme oxygenase-1 protects gastric mucosal cells against nonsteroidal anti-inflammatory drugs. J. Biol. Chem., vol. 281, p.p. 33422-33432.
- [3] Adachi, M.; Horiuchi, G.; Ikematsu, N.; Tanaka, T.; Terao, J.; Satouchi, K. & Tokumura, A. (2011) Intragastrically Administered Lysophosphatidic Acids Protect Against Gastric Ulcer in Rats Under Water-Immersion Restraint Stress. Dig. Dis. Sci. [Epub ahead of print-PMID: 21298479]
- [4] Al-Howiriny, T.; Alsheikh, A.; Alqasoumi, S.; Al-Yahya, M.; ElTahir, K. & Rafatullah, S. (2010). Gastric antiulcer, antisecretory and cytoprotective properties of celery (Apium graveolens) in rats. Pharm. Biol., vol. 48, p.p. 786-793.
- [5] Alam, M.S.; Alam, M.A.; Ahmad, S.; Najmi, A.K.; Asif, M. & Jahangir, T. (2010) Protective effects of Punica granatum in experimentallyinduced gastric ulcers. Toxicol. Mech. Methods, vol. 20, p.p. 572-578
- [6] .Allen, A. & Flemstrom, G. (2005) Gastroduodenal mucus bicarbonate barrier: protection against acid and pepsin. Am. J. Physiol Cell Physiol, vol. 288, p.p. C1-19. Alsarra, I.A.; Ahmed, M.O.; Alanazi, F.K.; Eltahir, K.E.; Alsheikh, A.M. & Neau, S.H. (2010). Influence of cyclodextrin complexation with NSAIDs on NSAID/cold stressinduced gastric ulceration in rats. Int. J. Med. Sci., vol. 7, p.p. 232-239.
- [7] Graham, D.Y.; Javed, S.U.; Keihanian, S.; Abudayyeh, S. & Opekun, A.R. (2010). Dual proton pump inhibitor plus amoxicillin as an empiric anti-H. pylori therapy: studies from the United States. J. Gastroenterol., vol. 45, p.p. 816-820.
- [8] Joseph, J.M.; Sowndhararajan, K. & Manian, S. (2010), Protective effects of methanolic extract of Hedyotis puberula (G. Don) R. Br. ex Arn.

- against experimentally induced gastric ulcers in rat. J. Ethnopharmacol., vol. 131, p.p. 216-219.
- [9] Konturek, P.C.; Brzozowski, T.; Burnat, G.; Szlachcic, A.; Koziel, J.; Kwiecien, S.; Konturek, S.J. & Harsch, I.A. (2010). Gastric ulcer healing and stress-lesion preventive properties of pioglitazone are attenuated in diabetic rats. J. Physiol Pharmacol., vol. 61, p.p. 429-436
- [10]. Lakshmi, V.; Singh, N.; Shrivastva, S.; Mishra, S.K.; Dharmani, P.; Mishra, V. & Palit, G. (2010). Gedunin and photogedunin of Xylocarpus granatum show significant antisecretory effects and protect the gastric mucosa of peptic ulcer in rats. Phytomedicine, vol. 17, p.p. 569-574.
- [11] Lanas, A. (2008). Role of nitric oxide in the gastrointestinal tract. Arthritis Res. Ther., vol. 10, Suppl. 2, p.p. S4. Lanza, F.L.; Chan, F.K. & Quigley, E.M. (2009). Guidelines for prevention of NSAID-related ulcer complications. Am. J. Gastroenterol., vol. 104, p.p. 728-738.