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REVIEW ON MANAGEMENT IN PEPTIC ULCER DISEASE

[1]Sahil Siraj Makhani , [2] Prof. AkshayKumar Meshram , [3]Prof. Shrikant Mahajan

B.Pharmacy

Maharashtra Institute Of Pharmacy, (Betala) Bramhapuri , Maharashtra.

ABSTRACT

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.

INTRODUCTION

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 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 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.[1]

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.

Approximately 10% of Americans develop chronic PUD during their lifetime. The incidence Varies with ulcer type, age, gender, and geographic location. Race, occupation, genetic Predisposition, and societal factors may play a minor role in ulcer pathogenesis but are Attenuated by the importance of HP infection and NSAID use. The prevalence of PUD in the United States has shifted from predominance in men to nearly comparable prevalence

in men And women.Recent trends suggest a declining rate for younger men and an increasing rate for Older women. Factors that have influenced these trends include the declining smoking rates in Younger men and the increased use of nsaids in older adults.

Since 1960, ulcer-related Physician visits, hospitalizations, operations, and deaths have declined in the United States by More than 50%, primarily because of decreased rates of PUD among men. The decline in Hospitalizations has resulted from a reduction in hospital admissions for uncomplicated Duodenal ulcer. However, hospitalizations of older adult for ulcer-related complications (bleeding and perforation) have increased.[1] [3]

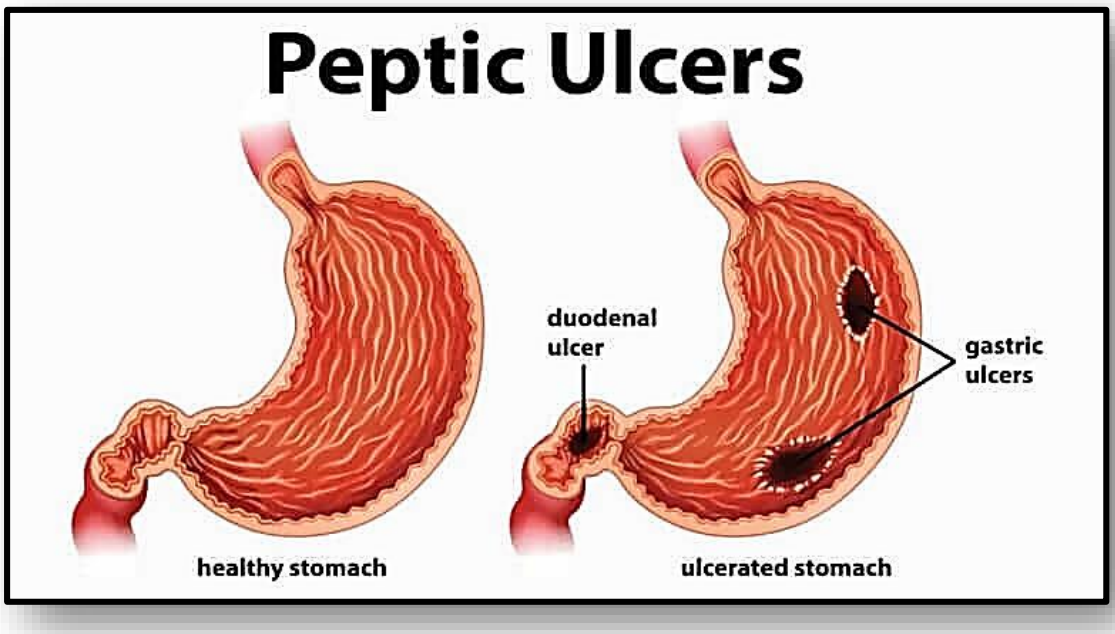

Fig. No. :- 01:- Healthy Stomach Vs Ulcerated Stomach

ETIOLOGY

Most peptic ulcers occur in the presence of acid and pepsin when HP (*Helicobacter pylori),* Nsaids, or other factors disrupt normal mucosal defense and healing mechanisms. Hypersecretion of acid is the primary pathogenic mechanism in hypersecretory states such as ZES. Ulcer location is related to a number of etiologic factors. Benign gastric ulcers can Occur anywhere in the stomach, although most are located on the lesser curvature, just

distal To the junction of the antral and acid-secreting mucosa. Most duodenal ulcers occur in the First part of the duodenum (duodenal bulb).[4]

Although H pylori is the major cause for peptic ulcers, other factors which cause peptic ulcer include:

Table No : 01 :- Common And Uncommon Causes (Peptic Ulcer).

Common Causes :-
Helicobacter pylori infection
Nonsteroidal anti-inflammatory drugs
Critical illness (stress-related mucosal damage)
Uncommon Causes :-
Hypersecretion of gastric acid (e.g., Zollinger-Ellison syndrome)
Viral infections (e.g., cytomegalovirus)
Vascular insufficiency (crack cocaine–associated)
Radiation
Chemotherapy (e.g., hepatic artery infusions)
Rare genetic subtypes

ZOLLINGER -ELLISON SYNDROME (GASTREINOMA)

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. Zollinger-Ellison syndrome (ZES) is a group of symptoms comprised of severe peptic ulcer disease, gastroesophageal reflux disease (GERD), and chronic diarrhea caused by a gastrin-secreting tumor of the duodenum or pancreas (gastrinoma triangle) that results in increased stimulation of acid-secreting cells of the stomach. Gastrinoma is a functional neuroendocrine tumor that secretes gastric acid which causes ZES.

The earlier misconception was that the location of gastrinoma is in the pancreas. However, gastrinomas occur in the duodenum more than the pancreas by three times especially in the first portion of the duodenum. There are other non-neuroendocrine tumors secreting gastrin, but not adequate amounts to cause significant symptoms. Gastrinoma causing ZES occurs sporadically in about 80% of cases and is reported to be 20% to 25% as multiple endocrine neoplasia type 1 (MEN1) from multiple reports in the literature. Approximately 50% of patients with MEN1 have ZES; therefore, MEN1 must be included in a workup if ZES is highly suspicious. It takes an average of 8 years from the start of symptoms to diagnosis due to the widespread use of proton pump inhibitors (ppis). One study of two referral centers in Italy and the United States showed a 62% decrease in referrals and diagnosis of ZES compared to a time when ppis were used less frequently. In 1955, ZES made first its appearance in Annals of Surgery as a case series compiled by two surgeons from Ohio State University, Dr. Robert M. Zollinger and Edwin H. Ellison. Both cases had ulcers at the upper jejunum with excess gastric acid production refractory to medical therapy and surgical therapy including gastrectomy. The ultimate finding that led to ZES with hypersecretion, hyperacidity, and recurrent peptic ulceration was a non-beta cell islet tumor of the pancreas. The pioneer case series induced enough interest to lead to thepublication of multiple studies that recognize the connections between gastrinoma and ZES.[2][5]

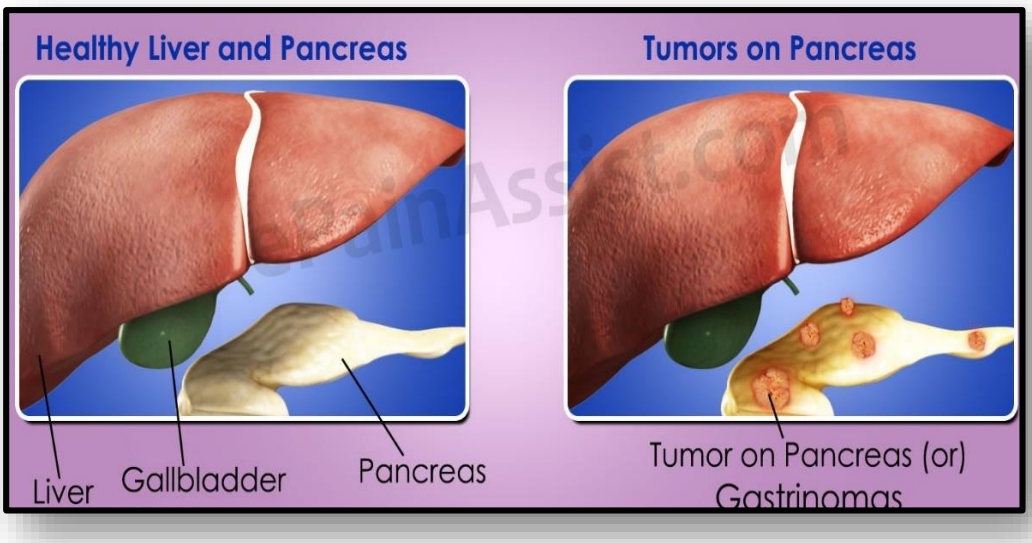

Fig . No :- 02:- Healthy Liver and Pancreas Vs Tumors On pancreas.

SIGNS AND SYMPTOMS ASSOCIATED WITH PEPTIC ULCER

1. Burning pain mainly abdominal pain. Pain may get better or worse after eating a meal.
2. Nausea, vomiting
3. Weight loss
4. Fatigue
5. Belching
6. Chest pain
7. Anorexia
8. Vomiting blood

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. [2]

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. [2]

The role of pepsin in the pathogenesis of peptic ulcer has been the subject of intense study and debate for many years. Two difficulties inherent in distinguishing between the role of acid alone vs acid and pepsin are that a) acid-containing gastric juice always contains pepsin, and, b) that hydrogen ion concentration (ph) is a major determinant of the activity of pepsin. However, studies in animal models of peptic ulcer indicate clearly that pepsin, in combination with acid, produces much more severe and more extensive mucosal damage than acid alone. Recent interest in pepsin and its precursor, pepsinogen, has stemmed from the finding that each is remarkably heterogeneous, and that the heterogeneity has a genetic basis. Results of studies using radioimmunoassays specific for the 2 major forms of pepsinogen, pepsinogen I and pepsinogen II, have shown that serum levels of pepsinogen I and pepsinogen II, and the ratio of pepsinogen I to pepsinogen II, can be used as noninvasive probes of gastric mucosal structure and function, indicators of the genetics.[2

HELICOBACTER PYLORI

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 produces acid inhibiting proteins which allow it to adapt to the low Ph environment of the stomach. H pylori attaches itself to gastric type epithelium by adherence pedestals, which prevent the organism from being shed during cell turn over and mucus secretion. [2]

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. H pylori infection alter the inflammatory response and damages epithelial cells directly by cell mediated immune mechanisms or indirectly by activated neutrophils or macrophages attempting to phagocytose bacteria or bacterial products. H pylori infection may increase gastric acid secretion in patients with duodenal ulcer, or diminish acid output in patients with gastric cancer infection of gastric antrum leads to post prandial hypergastrinemia and hyper secretion of gastric acid.[2]

Responsible mechanisms includes :-

- Cytokines, such as tumor necrosis factor (TNF) a released in H pylori gastritis.
- Products of H pylori, such as ammonia; and diminished expression of somatostatin.

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 inhibition 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. [2]

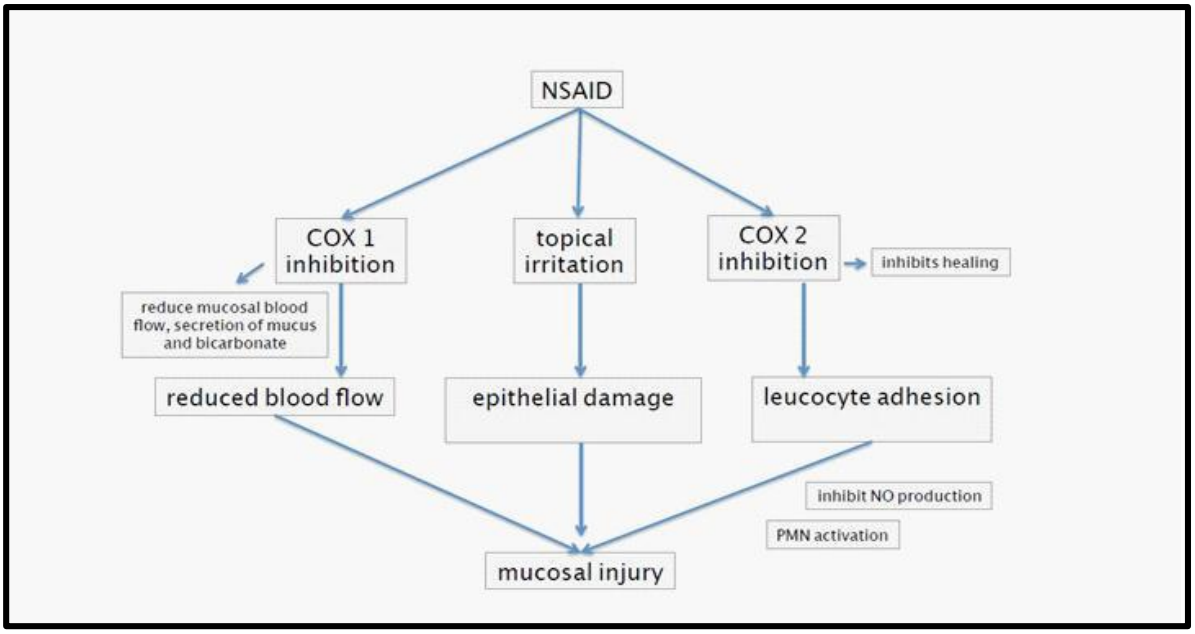

Fig.No - 3: Nonsteroidal antiinflammatory drugs induced mucosal injury.

DIAGNOSIS AND 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. [3]

Various tests for detection of H pylori is given in table below.

Table No :- 02 :- Test For Detetction Of H.Pylori.

Tests	Description	Advantages	Disadvantages
Antibody Detection (Laboratory Based)	Detects Antibodies To H. Pylori In Serum	Endoscopy Not Required Inexpensive Most Accurate	Low Specificity
Antibody Detection	Qualitative Detection Of (Igg) Antibody To H. Pylori In Whole Blood	Quick Inexpensive	May Yield Invalid Results

Urea Breath Test	H Pylori Urease Breaks Down Ingested Labeled(Urea,Patients Exhales Labeled CO_2)	Endoscopy Not Required Less Expensive	Results Are Not Immediate
Histology	Microbial Examination	High Specificity And Sensitivity	Require Endoscopy Expensive
Culture	Culture Of Biopsy	Used To Test For Antibiotic Resistance High Specificity	Require Endoscopy 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 (Non radio active isotope) and carbon 14 (Radio active 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 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.[20]

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, [20]

- H_2 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.
- 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.

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. [20]

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 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. [20]

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 H_2 receptor antagonist or a pump inhibitor, which has proved to provide a faster and more reliable healing than antacids. Either an H_2 receptor blocker or a proton pump inhibitor can be used as a first line agent. In refractory cases, sucralfate also may be indicated.

The treatment of chronic PUD varies depending on the etiology of the ulcer (HP or NSAID), Whether the ulcer is initial or recurrent, and whether complications have occurred. Overall treatments aimed at relieving ulcer pain, healing the ulcer, preventing ulcer recurrence, and reducing ulcer-related complications.

The goal of therapy in HP-positive patients with an active ulcer, a previously documented ulcer, or a history of an ulcer-related complication, is to eradicate HP, heal the ulcer, and cure the disease. Successful eradication heals ulcers and reduces the risk of recurrence to less than 10% at 1 year. The goal of therapy in a patient with a NSAID-induced ulcer is to heal the ulcer as rapidly as possible. Patients at high risk of developing NSAID ulcers should be switched to a COX-2 inhibitor or receive prophylactic drug co-therapy to reduce ulcer risk and ulcer-related complications. When possible, the most Cost-effective drug regimen should be utilized.[3]

CLASSIFICATION OF DRUGS FOR PEPTIC ULCER

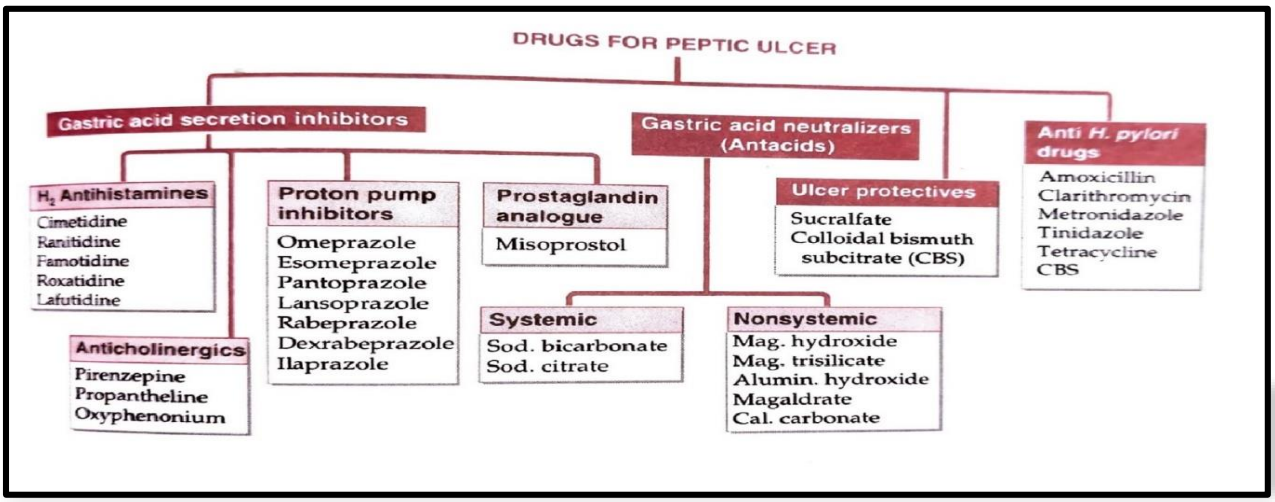

Fig . No. :- 03 :- Classification Of Drugs For Peptic Ulcer. [4]

DRUGS FOR PEPTIC ULCER DISEASE

Table No:- 03.:- Drugs For Peptic Ulcer Disease.

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
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.
Contrindications	Documented hypersensitivity.
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
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
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
Adult Dose	30 mg qid for 4-8 week
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
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
	PROTON PUMP INHIBITERS
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
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. [4]
Adult Dose	20 mg tab PO qid 4-8 week
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
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
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
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 [4]

AYURVEDIC MEDICATION

ALOE M.P PLUS

Aloe m.p plus is a powerful natural, 100% organic aloe mucilaginous polysaccharides (amp) based non toxic 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 aliments.[20]

MANUKA HONEY

- Manuka honey has been found to inhibit the growth of the bacteria, helicobacter pylori.
- The procedure to take summer glow umf 16 + manuka honey for digestive health is given.
- Take a teaspoon to a table spoon of summerglow umf 16 + manuka honey three-four times a day. Ideally one hour before meals and again at bed time.
- 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.
- Many people have experienced good results if they have the honey straight from the teaspoon.

- Summerglow 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. 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). [20]

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[20].

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.
- 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.
- Slippery elm (ulmus fulva): although there has been little scientific research on slippery elm, it has along history of use based on clinical experience. [20][1]
- Angelica (angelica archangelica).
- German chamomile (matricaria recutita).
- Lemon balm (melissa officinalis).
- Licorice.
- Milk thistle (silybum marianum).
- Peppermint (mentha piperita). [20][1]

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 H_2 antagonist. Three regimens are mentioned here.[4]

- 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 H_2 antagonist or proton pump inhibitor, but the benefit of this have not been evaluated. The H_2 antagonist or proton pump inhibitor is stopped once healing has occurred. [4]

- 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. [4]

- 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. [4]

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.[1]

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.[2]

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. [1][20]

OMEGA-3-FATTY ACIDS :-

Treatment with Omega-3-fatty acids reduced the risk of ulcers caused by NSAIDS.[1][20]

PROBIOTICS :-

Probiotics inhabit the lining of the intenstine and protect from the entry of bad infections that can cause disease. Lactobacillus acidophilus is the most commonly used probiotics. [1]

VITAMIN C :-

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

HEALING OPTIONS :-

Tabe No:-04 :- Healing Options.

Herbal home remedy • Banana • Chandan • Lime • Vegetable juice • Almond milk
Ayurvedic supplements • Avucid • Avipattikar Churna • Mahashankha Bati
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

CONCLUSION

Peptic ulcer can be cured. The incidence can be reduced by proper diet control. 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. Peptic Ulcer a disease which can be avoidable. Although continuous exposure of different Etiological factor which try to disrupt the gastric mucosa, but gastric mucosa likely to Maintain its structural integrity and functioning. However, mucosal defence were loss to Different etiological factors leads to gastric mucosal injuries. The action of etiological factors On gastric mucosa were lead to the development of potential therapies to treat PUD. *H. Pylori* And nsaids are the leading cause for the gastric mucosal damage which is eventually Countered by magical drugs for PUD are PPI's and H2RA's. Both the class of drugs play a Mainstay in treatment of PUD. Even the refractory PUD can be effectively treated with the Well developed treatment regimen. Not only the drug regimen but also some home remedies Have prove to be control PUD.

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