JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

A REVIEW ARTICLE: RECENT TRENDS IN THE TREATMENT AND MEDICATION FOR PEPTIC ULCER

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ABSTRACT:

Peptic ulcer is a chronic disease affecting upto 10% of the world's population. Peptic ulcers are open sores that develop on the inside lining of esophagus, stomach and Upper portion of small intestine. The two most common types of peptic ulcers are called Gastric ulcers or duodenal ulcers. The most common symptom of a peptic ulcer is abdominal pain. Approximately 500,000 new cases are reported each year, with 5 million people affected in the United States alone ulcer disease has become a disease predominantly affecting the older population, with the peak incidence occurring between 55 and 65 years of age. Interestingly, those at the highest risk of contracting peptic ulcer disease generations born around the middle of the 20th century. In men duodenal ulcers were common than gastric ulcers in women, The converse was to be found to be true. 35% of patients diagnosed with gastric ulcers will suffer serious complications. *H. pylori* infection is associated with about 60% of duodenal ulcers and 40% of gastric ulcers. overuse of NSAIDS.H. pylori infection and the use of non-steroidal anti-inflammatory drugs (NSAIDS) are the predominant causes of peptic ulcer disease. which major gastric cancer and minor factors are stress, smoking, spicy food and nutritional deficiencies .The idea behind treating ulcers is to lower the amount of acid that your stomach makes to neutralize the acid that is made and to protect the injured area so it can have time to heal.

Key words: peptic ulcer, Helicobacter pylori infection, pathogenesis and mediators

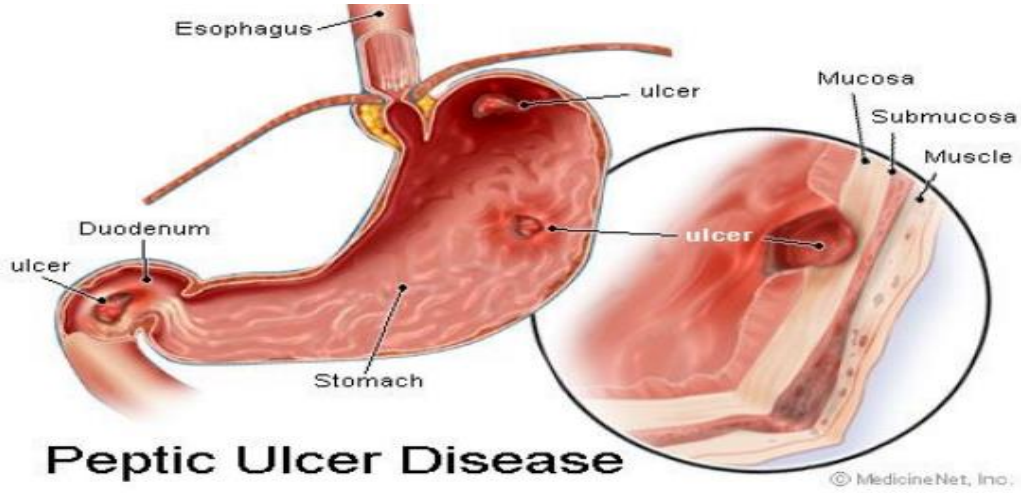
INTRODUCTION:

Peptic ulcer is a condition in which a person experience severe abdominal pain, especially around the inner part of the stomach. In this disease, mucus membrane around the digestive organs gets weakens. peptic ulcer is a chronic disease that results from imbalance between endogenous protective factors of gastric mucosa (mucosa and bicarbonate secretion, adequate blood flow,prostaglandinE2,nitric oxide ,and antioxidants enzymes and others) and aggressive factors (acid and pepsin secretions).behavioral and environmental factors such as smoking, poor diet ,alcohol and non- steroidal inflammatory drugs ingestion ,and helicobacter pylori infections ,among others have been

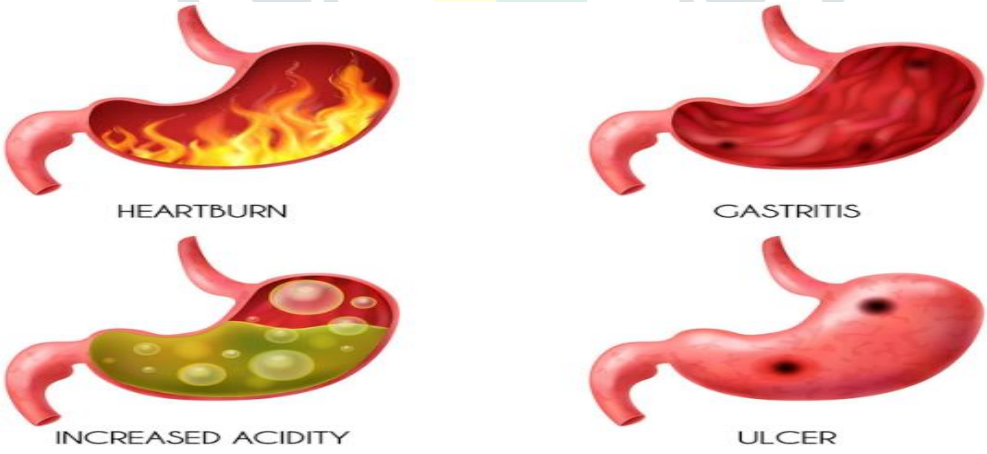
implicated in the etiology of gastric ulcer[1]. Peptic ulcer is often break greater than 3-5 mm in the stomach or duodenum with a visible depth. Peptic ulcer is one of the world major gastrointestinal disorders and affecting the 10 % world population. about 19 out of 20 peptic ulcers are duodenal. An estimated 15000 deaths occur each year as a consequence of peptic ulcer annual incidence estimates of peptic ulcer hemorrhage and perforation was 19.4 -15.7 and 3.8- 14 per 100,000 incidence estimates of peptic ulcer hemorrhoge and perforation were respectively[2]

ANATOMY OF PEPTIC ULCER:

The stomach is located in the upper part of the abdomen just beneath the diaphragm.



FORMATION OF ULCER:

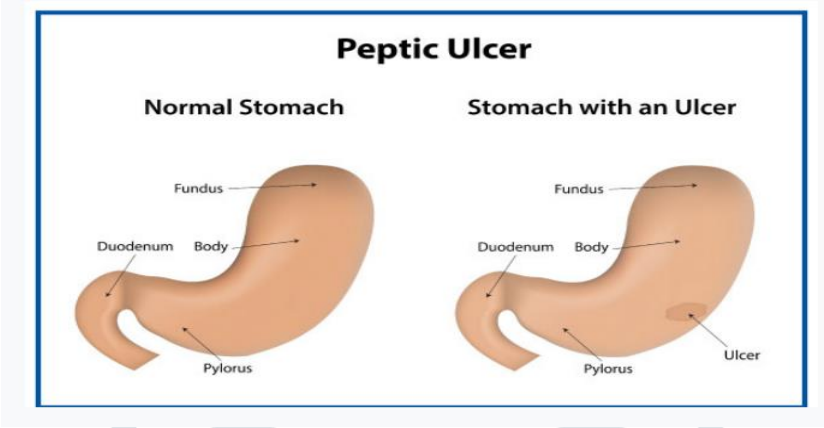


TYPES OF PEPTIC ULCER:

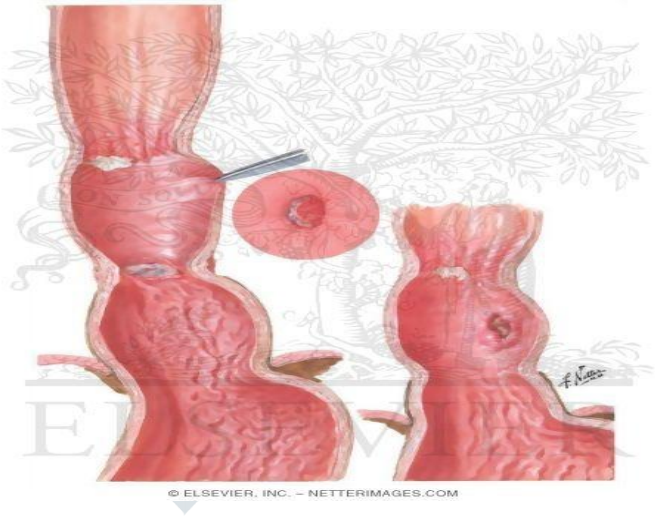
1. Peptic ulcer:

Peptic ulcer is a broad term which includes ulcers of digestive tract in the stomach or the Peptic ulcer is an acid-induced lesion of the digestive tract that is usually located in the stomach or duodenum. Earlier it was believed that one developed this type of ulcers due to stress and spicy food However, recent research has shown that these are just aggravating factors. The causative agent is infection caused by the bacteria H. pylori or reaction to

certain medicines like non- steroidal anti- inflammatory drugs [NSAIDS][3]. Symptoms of peptic ulcers include abdominal discomfort and pain other symptoms are include weight loss, appetite, bloating nausea and vomiting. Some may experience blood in stool and vomit, [4]

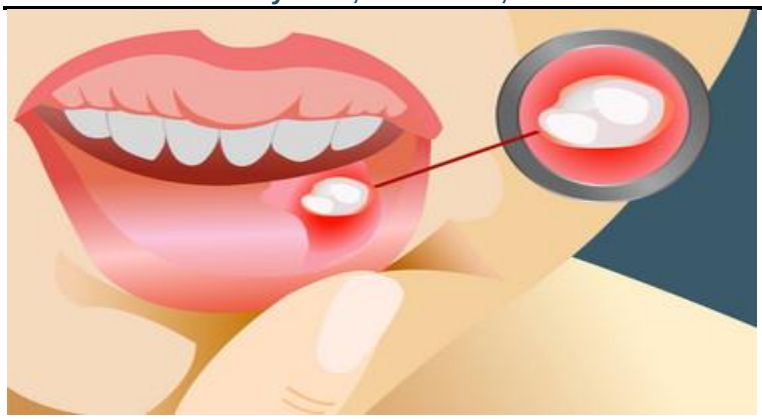


Esophageal ulcer: Esophageal ulcers are lesions that occur in the esophagus these are most commonly formed at the end of the food pipe and can be felt as a pain right below the breastbone, in the same area where symptoms of heartburn are felt, Esophageal ulcers are associated with acid reflux, associated with acid reflux, or GERD, prolonged NSAIDS] [4]



APTHOUS ULCER:

Sores that develop in the inner lining of the mouth are referred to as mouth ulcers. mouth ulcers are common and are usually due to trauma such as from ill-fitting dentures, fractured teeth, or fillings, anemia, measles, viral infection, chronic infections, throat cancer, mouth cancer, vitamin B deficiency are some of common causes of ulcers sore s in mouth. The incidence of aphthous ulcers has been found to be lower in smokers than in nonsmokers **[6].**



EPIDEMIOLOGY:

Peptic ulcer disease (PUD) is a global problem with a lifetime risk of development ranging from 5% - 10% Overall, there is a decrease in the incidence of PUD worldwide due to improved hygienic and sanitary conditions combined with effective treatment and judicious use of NSAIDs. Duodenal ulcers are four times more common than gastric ulcers. Also, duodenal ulcers are more common in men than in the women[7]. It is fairly heterogeneous disease with worldwide distribution, but the disease effects people from all countries and different races. its average prevalence is between 5-10% of the general population over a life time [8-9].This represents approximately 10-20% of people infected with helicobacter pylori globally, with wide variations between different races and countries of the world have been confirmed. The average incidence of peptic ulcer among the persons infected by HP, is approximately 1% per year.[10]

PATHOGENESIS OF PEPTIC ULCER:

The pathology can be divided in 3 broad categories:

1.*H. pylori* positive

2.*H. pylori* negative and non- NSAIDS associated

3. NSAID associated

NSAIDS plays an important role in pathogenesis of peptic ulcer[11].

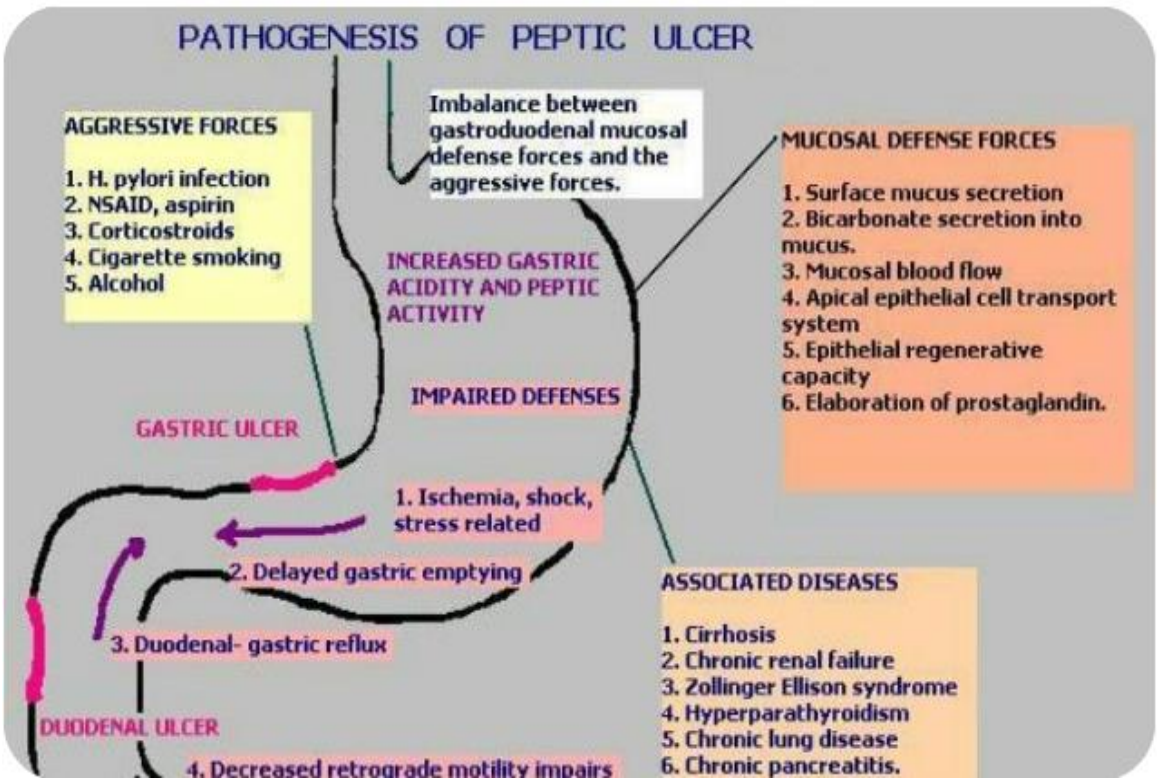
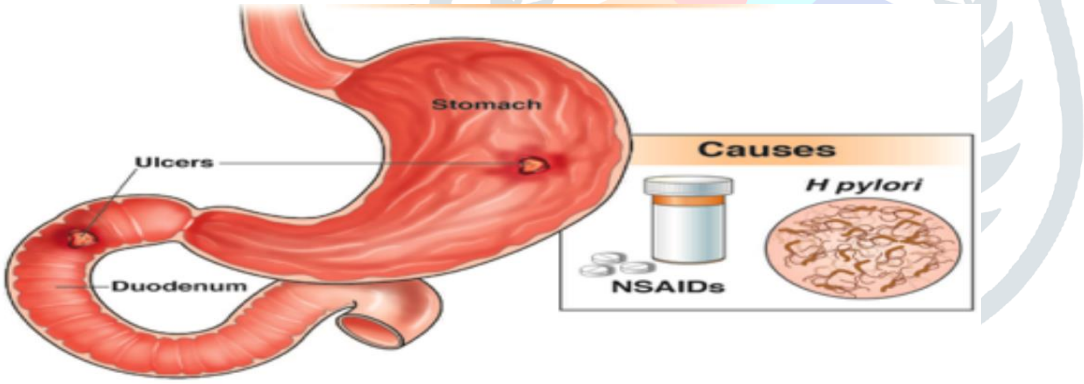


FIG: pathogenesis of peptic ulcer

ETIOLOGY / CAUSES:



Till the last decade it has been estimated that 95% of duodenal ulcer and 70% of gastric ulcer is attributed due to H.pylori. about 14%- 25% of gastric duodenal ulcers are found to be associated with NSAIDS[12]. Peptic ulcer disease [PUD]has various however Helicobacter pylori – associated PUD and NSAID – associated PUD account for the majority of the disease etiology [13]

COMMON CAUSES	RARE CAUSES
	1.Gastinoma [Zollinger – Ellison syndrome] 2. Hyperplasia / Hyper function of antral G cells

1.Helicobacter pylori infection 2.NSAIDS 3. Medications	3. Systemic Masto cytosis 4. Myeloproliferative Syndrome with Basophilia 5. Viral infections [Herpes simplex virus Tipo1 and Cytomegalovirus] 6. Vascular insufficiency [Cocaine] 7. Ischemia caused by Stenosis of Celiac artery 8. Radiation 9. Chemoembolization [Via hepatic artery] 10. Chron's disease 11. Type 2 amyloidosis 12. Neuhauser Syndrome [tremor- nystagmus-ulcer] 13. porphyria cutanea tarda 14. Other drugs [potassium chloride, bisphosphonates, mycophenolate] 15. Idiopathic	**1.**

HELICOBACTER-PYLORI-ASSOCIATED PUD

H pylorus is a gram-negative bacillus that is found within the gastric epithelial cells. This bacterium is responsible for 90% of duodenal ulcers and 70% to 90% of gastric ulcers. H. pylori infection is more prevalent among those with a lower socioeconomic status and is commonly acquired during childhood. The organism has wide spectrum of virulence factors allowing it to adhere to and inflame the gastric mucosa. This results in hypochlorhydria or achlorhydria leading to gastric ulceration

Virulence Factors of Helicobacter pylori

1. Urease Secretion of urease breaks down urea into ammonia and protects the organism by neutralizing the acidic gastric environment.

2. Toxins: CAGA/ VACA are associated with stomach mucosal inflammation and host tissue damage

3. Flagella: Provides motility and allows movement toward the gastric epithelium

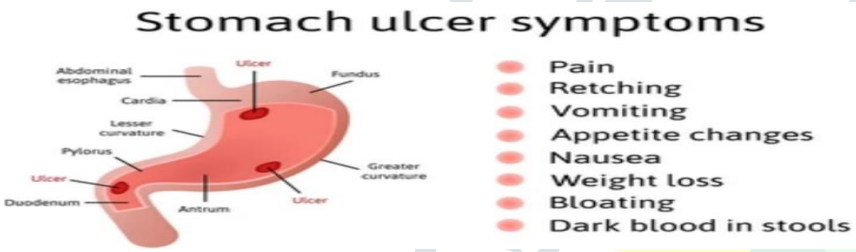
2.NSAIDS- ASSOCIATED PUD

Nonsteroidal anti-inflammatory drugs and aspirin use is the second most common cause of PUD after H. pylori infection [14-15]. Thegastric mucosa is normally protected by secretion of prostaglandin. NSAIDs block prostaglandin synthesis by inhibiting COX-1 enzyme resulting in a decrease in gastric mucus and bicarbonate production and a decrease in mucosal blood flow

3.MEDICATIONS:

Apart from NSAIDs, bisphosphonates, potassium chloride, steroids, and fluorouracil have been implicated in etiology of PUD.

SYMPTOMS OF PEPTIC ULCER:



1. fullness, bloating or bleching
2. Intolerance to fatty foods
3. Heart burn
4. Nausea
5.. Burning stomach pain

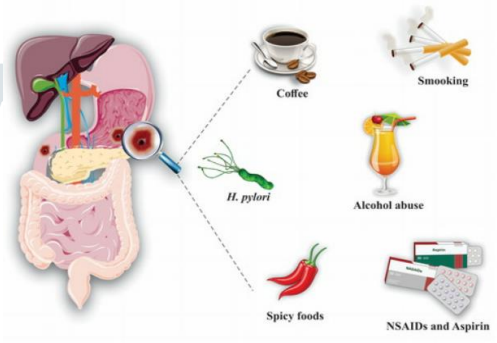
The most typical sign of a peptic ulcer is burning stomach discomfort. stomach acid, as well as having an empty stomach. aggravates the pain Eat some meals that buffer stomach acid or take an acid – reducing medication to alleviate the pain, but it may return. its possible that the discomfort will be worse between meals and at night

Many persons with peptic ulcers have no symptoms at all ulcers. Ulcers can also generate severe indications and symptoms, such as:

1. Vomiting or vomiting blood [which can be red or black in color]

2. Feces with dark blood, or stools that are black or tarry
3. Breathing problems
4. Feeling dizzy
5. Vomiting or nausea
6. Weight loss that isn't explained
7. Appetite shifts

TREATMENT OF PEPTIC ULCER [16-17]



Exploiting drug delivery systems for oral route in the peptic ulcer disease treatment

1. Initial treatment of peptic ulcer disease depends on its cause.

A]. H. pylori infection. Treatment to eliminate Helicobacter pylori (H. pylori) bacteria usually involves combining two antibiotics with an acid reducer such as a proton pump inhibitor or sometimes a bismuth compound. Curing the infection speeds the healing of an ulcer and makes the ulcer less likely to recur. It is important to take all the medicine to prescribes so that the bacteria are killed . If the bacteria are not eliminated by the antibiotics, they may become even more difficult to kill later (resistant).

B]. NSAIDs. If at all possible, taking nonsteroidal anti-inflammatory drugs (NSAIDs). If must continue taking an NSAID, other medicines may be used to protect the stomach.

C]. Hypersecretory condition. Acid reducers are most often used to treat an ulcer caused by a hypersecretory condition (a condition in which your stomach produces excessive acid). In addition, To conduct other tests to determine whether there is another cause for the ulcer.

D]. Unknown cause. If no cause can be found (idiopathic ulcer), The ulcer will usually be treated with an acid reducer. Long-term treatment depends on the severity of the ulcer and other factors, such as the size of the ulcer, whether you have had complications, and what other treatments have been used.

E]. Ongoing treatment: Use of NSAIDs can slow the healing of an ulcer or prevent it from healing altogether. If must continue to use NSAIDs, may recommend that to take a prostaglandin analog, such as misoprostol, or a proton pump inhibitor. Misoprostol does not help ulcers heal, but it can help prevent ulcers from coming back.

2. Eradication of H. pylori infection:

Over the past 20 years. H. pylori eradication therapies have mainly consisted of antimicrobial agents combined with anti -secretory drugs. there is now a worldwide consensus that the first line treatment should be triple therapy with a PPI twice daily plus clarithromycin 500mg twice daily and either amoxicillin 1g twice daily [PPI-CA] or metronidazole 500mg twice daily [PPI-CM] for 7-14days [18]. Treatment with PPIs is superior to treatment once daily [19]] Successful eradication with first line treatments varies from 70%-95%and 10-day and 14-day treatments are generally 7-9% more effective than the most commonly used 7-day regimens [20]

DO"S AND DON"TS :

This necessitates following a set of do's and don'ts to manage it and related symptoms. Life style changes, dietary modifications and healthy discipline is needed to counter this condition.

DO"S	DON"TS
1.Eat light foods	Eat half – cooked food, meat etc.
2.wash your hands very frequently	Take over- the- counter medications
3.limit certain medicines that may aggravate symptoms 4. Take medicines after food	Do smoking Eat foods that irritate the stomach
5.Limit soft drink beverages or alcoholic beverages	Take an overdose on iron supplement

Following the healthy guidelines and following crucial to cure ulcers. Regular monitoring of this condition is also required to check for the effectiveness of the treatment. Remember, if left untreated, the consequences can be serious, as the ulcer will spread causing a lot of discomfort.

RISK FACTORS:

Genetic factors may predispose you to developing an ulcer,

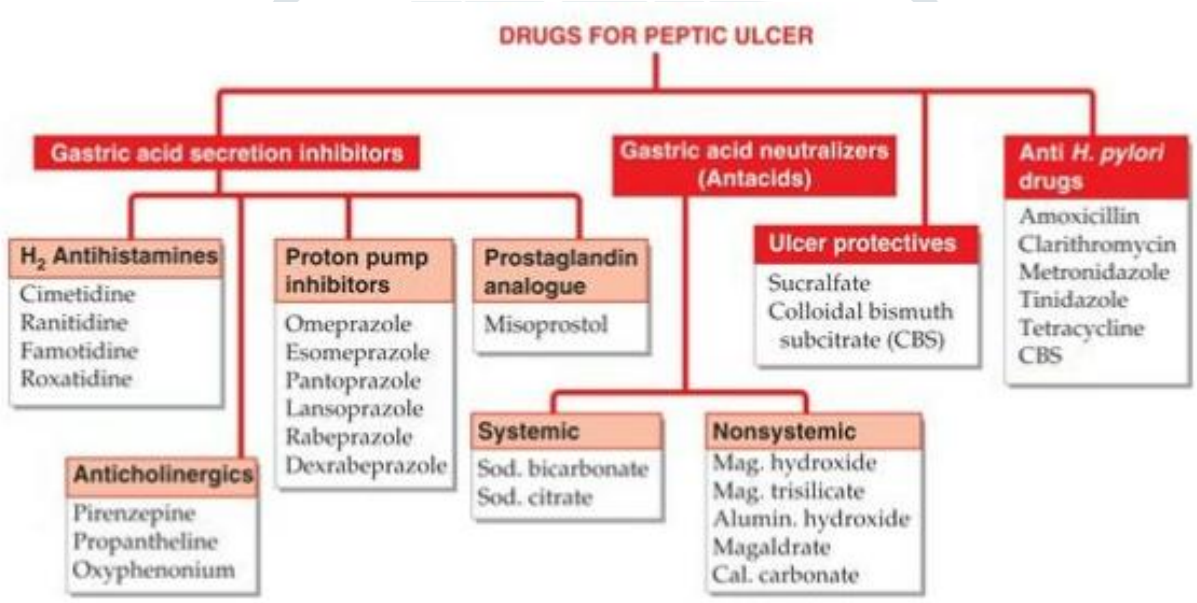
1. Increasing age
2. Alcohol abuse
3. Diabetes may increase your risk of having H. pylori.

4. Lifestyles factors, including chronic stress, coffee drinking [decaf] and smoking, may make more susceptible to damage from NSAIDS or H. pylori if you are a carrier of this organism. Again however, these factors do not cause an ulcer on their own.

LIFE STYLE RISK FACTORS:

That cigarette smoking can increase chance of getting an ulcer, especially if infected with *H. pylori* [21]. While a link has not been found between alcohol consumption and peptic ulcers, ulcers are more common in people who have cirrhosis of the liver, a disease often linked to heavy alcohol consumption. Drinking alcohol can increase discomfort to have an ulcer [22]

CLASSIFICATION OF DRUGS:



1. GASTRIC ACID SECRETORY AGENT

1. H_2- Antihistamines:

The H2 antihistamines are a class of drugs used to block the action of histamine on parietal cells in the stomach., decreasingly the production of acid by these cells. these are used in the treatment of dyspepsia and are highly selective because they do not affect H1 or H3 receptors.

Mechanism of action:

The H2 receptor are competitive antihistamines at the parietal H2 receptor they suppress the normal secretion of acid by parietal cells and the meal stimulated secretion of acid. they accomplish by two mechanisms:**.** Histamine released by ECL cells in the stomach is al cells blocked from binding on parietal cell h2 receptors, which stimulate acid secretion.

2]. In the presence of H2 blockers direct stimulation of parietal cells by gastrin or acetyl choline results in reduced acid secretion.

Therapeutic uses:

- peptic ulcer disease [PUD]
- Dyspepsia
- Zollinger- Ellison syndrome
- prevention of stress ulcer [a specific indication of ranitidine]

Adverse Effects:

Adverse effects occur in less than 5% patients. these includes:

- Headache
- Dizziness
- Diarrhea
- Rashes
- Confusion

EXAMPLES : Cimetidine, Ranitidine, Famotidine, Roxatidine.

2.Proton pump inhibitors:

Proton pump inhibitors are the class of drugs that are irreversible inhibitors of the gastric parietal cell proton pump. This enzyme promotes the exchange of h+ or k+ ions required for mediating HCL secretion. Omeprazole is a prodrug and is absorbed from small intestine, and its hepatic metabolite is responsible for acid production

Mechanism of drugs:

PPIS are inactive prodrugs. After reaching into acidified compartment is undergoes a molecular conversion to active thiophilic sulphonamide cation that reacts with H+ /K+ ATPase covalently and inactivates the enzymes. They reduce the production of acid by blocking the enzyme in the wall of the stomach that produces acid, reduction of acid prevents ulcers and allows any ulcers that exist in the oesophagus, stomach, and duodenum heal.

Therapeutic uses:

it is used to prevent and treatment of acid – related conditions are:

- Peptic ulcers
- stress ulcers
- Zollinger- Ellison syndrome
- In combination with antibiotics. it is used for eradicating Helicobacter pylori
- GERD

Adverse effects:

- Headache
- Abdominal pain
- Diarrhoea
- Constipation

EXAMPLES: omeprazole, esomeprazole, pantoprazole, Lansoprazole, rabeprazole, dexarabeprazole.

3.Anticholinergics :

Anticholinergics agents block the neurotransmitter acetylcholine in the central peripheral nervous system, antimuscarinic agent is a type of anti- cholinergic which is found on the muscarinic acetylcholine receptors. Most of them synthetic but scopolamine and atropine are naturally extracted alkaloids.

Mechanism of action :

Probanthine , oxyphenonium, etc, are older non selective muscarinic agents that block M1and M2 receptor sub types. While the recently discovered drugs pirenzepine and telenzepine act selectively on muscarinic M1 receptors, and retain acid secretion inhibiting effect with minimal effects on heart, intestine, and urinary bladder [M2 receptor].

Individual drug – pirenzepine:

Pirenzepine reduces gastric acid secretion, therefore is used in peptic ulcers, it also reduces muscle spasm.

Mechanism of action:

Pirenzepine selectively blocks M1 muscarinic receptors and inhibits gastric secretion without producing typical atropine side effects.

Therapeutic uses:

Pirenzepine effectively relieves the pain of peptic ulcer and promotes its healing.

4. Prostaglandins analogues:

Prostaglandins are important in the pathophysiology of peptic ulcer disease and possibility in its prevention and treatment as well. Prostaglandins have been shown to inhibit gastric secretion stimulate bicarbonate secretion. And increases gastric blood volume.

Examples for prostaglandins: Misoprostol

2.Gastric acid neutralizers

Antacids:

Chemically antacids are basic substances that neutralize the gastric acid and increase the pH of gastric contents. At pH above 5, peptic activity is indirectly reduced as pepsin is secreted as a complex with an inhibitory terminal moiety that dissociates if pH is more than 5: at pH between 2to 4 optimum peptic activity occurs.

Examples of antacids: antacids can be following 2 type are

1. Non systemic Antacids Ex: Mag.hydroxide, Mag. Trisilicate, Aluminium Hydroxide, Magaldrate, cal. Carbonate

2. System antacids: sodium bicarbonate and citrate

3.Ulcer protectives:

Ulcer protective drugs can increase mucosal resistance

Examples: Sucralfate, colloidal bismuth sub citrate [CBS]

4.Anti H- pylori drugs:

Helicon bacter pylori are gram – negative bacillus bacteria, which live in the GIT. if they remain present for many years, they cause ulcers in the lining of stomach or in the upper part of small intestine. Which eliminate the bacteria. these drugs having urease activity attach to the surface epithelium beneath the mucus and produce ammonia, the

ammonia maintains a neutral micro environment for the bacteria and promotes back diffusion of H+ ions. some of the drugs used for inhibiting the growth of Helicobacter pylori.

Examples: Amoxicillin, Clarithromycin, CBS, Tinidazole, Tetracycline [23]

DIFFERENT CLASSES OF DRUGS AND THEIR SIDE EFFECTS:

Different pharmacological classes of drugs which are used for the treatment of ulcer [24] Table 1:

CLASS	EXAMPLE	SIDE EFFECTS
1.antacids	Sodium bicarbonate Calcium carbonate Aluminum hydroxide Magnesium hydroxide	Rebound acid secretion
2.H2 receptor antagonist	Cimetidine, Ranitidine, Famotidine, Nizatidine, Roxatidine, Lafutidine	Diarrhea, Headache, Drowsiness, Fatigue, Muscle pain.
3.Anticholinergics	Pirenzepine, Terenzepine	Dry mouth, blurred vision, tachycardia, bladder dysfunction
4.Acid pump [H+/K+ ATPase] inhibitor.	Omeprazole, lansoprazole, pantoprazole, Rabeprazole	Nausea, Abdominal pain, constipation, Diarrhoea.
5.Cholecystokinin-2-receptor antagonist	Proglumide, L365260 YM022,	Under clinical trail
6.Mucosal defensive agents	Prostaglandins analogue [misoprostal] sucralftate	Diarrhoea, contraindicated in pregnancy, constipation

CLASSIFICATION OF DRUGS AND EXAMPLES WITH THEIR DOSE:[25]

CLASS	EXAMPLE	DOSE
Proton pump inhibitors	Omeprazole, Esomeprazole,	20mg Twice Daily

	Lansoprazole, Rabeprazole, pantoprazole.	40mg once or 20mg twice daily 30 mg once or twice daily 20 mg once or twice daily 40mg once or twice daily
2.Bismuth compounds	Bismuth subcitrate[BSC] Bismuth subsalicylates [BSS] Ranitidine bismuth citrate [RBC]	120mg four times daily 524mg four times daily 400 mg twice daily

PATIENT EDUCATION:

Patients with peptic ulcer disease should be counseled about potentially injuries agents like non -steroidal, anti -inflammatory drugs NSAIDS, aspirin ,alcohol, tobacco and caffeine. If it is necessary to use NSAIDS use the lowest possible dose and also consider prophylaxis for patients who use NSAIDS, obesity has a strong association with peptic ulcer disease, and patients should be asked to lose weight. Stress reduction counselling can be helpful in some cases.

PREVENTION OF H. PYLORI PEPTIC ULCER DISEASE:

Prevention of h. pylori associated disease benefits from predictors of who will become clinically ill. accordingly current treatment guidelines advise prophylactic h. pylori eradication for some individuals at higher risk for disease.[26].Antibiotic treatment has been reported to increase regression of cancer precursor lesions [27]

In case of gastric ulcer disease we should uses of NSAIDS we select altered treatments we select alternative approaches to relieve pain. in over daily life some factors responsible for decreasing the possibilities of peptic ulcers[28].We use mainly healthy oils. During the disease we should reduce the consumption of coffee, alcoholic and soda pop[29]In over daily life we should avoid refined foods like pasta, sugar, and white bread.

LEAVING SMOKING:

It is also a vital approach for the reason that cigarettes ingredients block with the defensive lining of stomach. Cigarette smoking, it also escalations acid production in stomach [30]

PHYSIOLOGICAL MANAGEMENT;

Anxiety and stress both are play a vital role in ulcer prevention. It also symptoms in gastric ulcer [31]

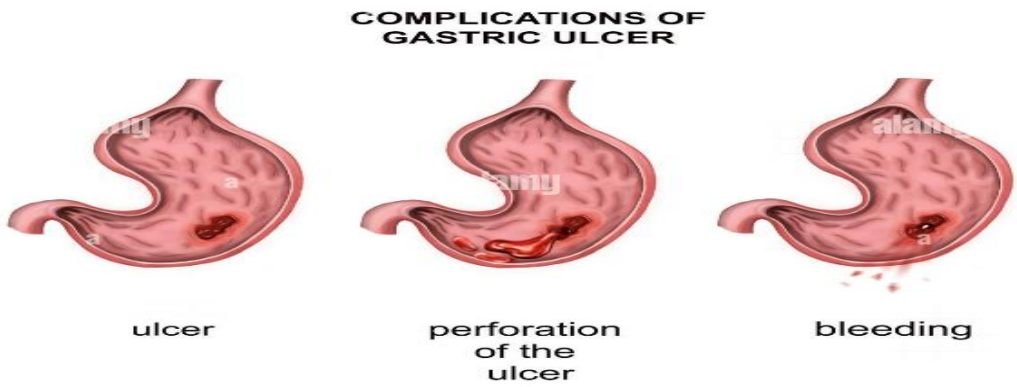
PROBIOTIC COMPLEMENTS:

Lactobacillus acidophilus, this probiotics food supplements ,it may be protecting peptic ulcer, and how ever it may help to maintain a balance between digestive systems . These type of supplements, it may help suppress HP infection and reduce side effects [32]

VITAMIN C;

Vitamin c supplements used with the dose of 500 to 1000mg and twice in a day, vit. c help to detect the proliferation of HP and another work is treating the bleeding in gastric ulcer, which is caused by aspirin [33]

COMPLICATIONS:



The complications occurs in 3 types:

1.bleeding

2.perforation

3.penetration

4.obstruction

1.Bleeding:

The acute clinical complications of ulcers is bleeding and approximately occurs in 15% -20% of ulcers. Bleeding is also the major cause of mortality in patients older than 65years [34].Ulcers related with NSAIDS are more likely to bleed than those causes only by H. pylori chronic infections. Populations at greatest risk are the elderly and those with other serious conditions, such as respiratory, cardiac, cerebrovascular renal problems [35].A total of 80 – 90% of upper gastro intestinal hemorrhage are not from variceal bleeding origin , and around 40 – 50% of these are caused by peptic ulcer disease. The mean associated mortality is around 5.5% [36] It can take various clinical manifestations

:15% have melenas, 30% hematemesis, 50% have both and about 5% have hematochezia caused by severe bleeding [37]

2.Perforation:

It occurs in up to 5% of patients with peptic ulcer, usually correspond to 60% of duodenal ulcers cases, most of them located at the anterior wall of the duodenal bulb and 40% of gastric ulcers, often affecting lesser curvature, free perforation of a duodenal or gastric ulcer into cavity may endanger the patients life.

3.Penetration:

This complication occurs when an ulcer across the wall of stomach or duodenum, but instead of drilled, freely into the peritoneal cavity, the clinical presentation may be similar to that of uncomplicated ulcer but the pain is usually more severe and persistent [38]rarely penetrating peptic ulcers may from fistulas between the duodenum and bile duct [choledoco – duodenal fistula] or between the stomach and colon .

4.Obstruction:

It is a un common complication which represents approximately 5% of ulcer-related complications. until about 1970, peptic ulcers represented the most common cause of obstruction to gastric emptying [39].In the last years, however, has decreased the frequency of obstructions. secondary to peptic ulcer and currently gastric malignancies are the leading cause of gastric outlet obstruction[40]

DIAGNOSIS AND EVALUATION:

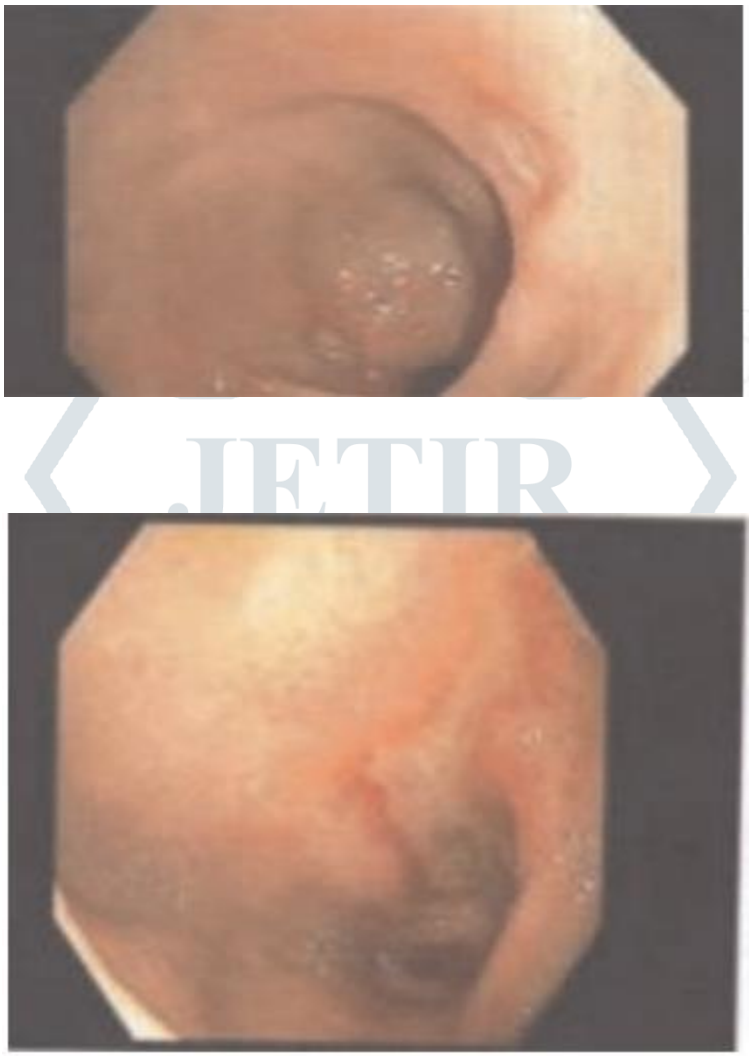
A through history and physical examination is necessary to evaluate the patient. In each case, we should look for alarm features which include [41]

1. Evidence of overt or occult gastrointestinal bleeding: hematemesis, melena, anemia, heme-positive stool.
2. Iron deficiency
3. Dysphagia
4. Left supraclavicular lymphadenopathy
5. Palpable abdominal mass
6. Symptom of impending perforation: severe persistent epigastric pain.
7. Symptom of obstruction: persistent vomiting
8. Malignancy: anorexia: unintended weight loss
9. Age :> 55 years.

Diagnosis tests:

should include complete blood count esophagogastroduodenoscopy or upper gastrointestinal [UGI] series and tests for detection H. pylori infection. EGD is preferred over UGI series as it has much higher diagnostic yield and mucosal

biopsy can be taken. Endoscopic views of clean-based duodenal ulcer and gastric ulcer are shown in Figures 1 and 2. During endoscopy, the location, size, depth, and any sign or stigmata of bleeding can be evaluated, and gastric biopsy from antrum, body, and incisura can be taken to detect H. pylori infection [42]. Although endoscopic evaluation is the gold standard of diagnosis of PUD, it is not cost-effective to perform EGD in all suspected cases of PUD.



DEUODENAL ULCERGASTRIC ULCER

Alternative non-endoscopic strategies can be considered in the absence of alarm features: **H. pylori test and treat**: In a population where the prevalence of H. pylori infection exceeds 20%, patients should get tested for H. pylori infection and, if positive, should be treated by anti-H. pylori therapy [43]. If H. pylori test is negative or patients still remain symptomatic after anti-H. pylori therapy, they should be given a 4–6 week course of proton pump inhibitor (PPI) therapy. If PPI therapy fails, patients should be reassured, diagnosis should be reassessed, and EGD should be considered. If patients respond to anti-H. pylori treatment or PPI therapy, they can be managed without further investigation [44]

Stool for H. pylori antigen and urea breath tests are most accurate not only for identification of active H. pylori infection but also for confirmation of eradication of infection. Serology for H. pylori antibody is less reliable and cannot be used for confirmation of cure.

Empiric acid suppression therapy: In a population where the prevalence of H. pylori infection is 10% or less, empiric PPI therapy is most cost-effective. In case of PPI failure, test-and-treat strategy should be applied as[45]

Physicians should make decision between test and treat strategy and empiric therapy for 4-6 weeks in the absence of alarm features. EGD should be considered in the presence of alarm features.

CONCLUSION:

Peptic ulcer disease is a common clinical problem in our environment predominately affecting all age of people. Peptic ulcer disease is predicted to continue to have a large global influence on health care delivery health economics, and patient quality of life as the prevalence of illness rises with age.The two common risk factors are H. pylori infection and NSAIDS. The H. pylori and NSAIDS possess the highest risk of ulcer development, however, the changing trends show increase in risk due to augmented use of NSAIDS. peptic ulcer illness continues to be a problem in medical visits. The majority of people present with dyspepsia should be examined for peptic ulcer disease.

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