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AN OVERVIEW ON PEPTIC ULCERS & ITS MANAGEMENT

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ABSTRACT

A ulcer happens while the coating of these organs is consumed by the acudic stomach related juices which are discharged by the stomach cells.1Peptic ulcer illness is common,affecting a great many americansyearly.The clinical expense of treating peptic ulcer and it's difficulties runs in the billions of dollars yearly . Ulcers can foster in the throat, stomach or duodenum, at the edge of a gastroenterostomy, in the jejunum, in Zollinger-Ellison disorder, and in relationship with a Meckel's diverticulum containing ectopicgastricmucosa [5].

A peptic ulcer, also known as PUD or peptic ulcer disease, is the most common ulcer of an area of the gastritestinal tract that is usually acidic and thus extremely painful. It is defined as mucosal erosions equal to or greater than 0.5 cm. As many as 70–90% of such ulcers are associated with Helicobacter pylori, a spiral-shaped bacterium that lives in the acidic environment of the stomach; however, only 40% of those cases go to a doctor. Patients who respond to optimal therapy for peptic ulcer disease do not require specialized testing.

In any case, those with recalcitrant (not recuperated following two months of treatment) or repetitive illness ought to have serumgastrin and serum calcium estimated to evaluate for gastrinoma and numerous endocrineneoplasia (MEN). These patients ought to likewise go through gastric corrosive examination to decide if the ulcer is brought about by gastric corrosive hypersecretion (basal corrosive result surpassing 10 mEq/hr) or diminished mucosal assurance.

Keywords*: Peptic Ulcer, endoscope,*

1. INTRODUCTION

Peptic ulcer illness addresses a serious clinical issue. Around 500,000 new cases are accounted for every year, with 5 million individuals impacted in the US alone. Strangely, those at the most noteworthy gamble of contracting peptic ulcer sickness are those ages brought into the world around the center of the twentieth 100 years. Ulcer illness has turned into a sickness transcendently influencing the more established populace, with the pinnacle Peptic ulcer infection addresses a serious clinical issue. Around 500,000 new cases are accounted for every year, with 5 million individuals impacted in the US alone. Strangely, those at the most elevated chance of contracting peptic ulcer illness are those ages brought into the world around the center of the twentieth 100 years. Ulcer illness has turned into an infection dominatingly influencing the more seasoned populace, with the pinnacle rate happening somewhere in the range of 55 and 65 years old. In men, duodenal ulcers were more normal than gastric ulcers; in ladies, the opposite was viewed as obvious. 35% of patients determined to have gastric ulcers will experience serious complications[4]. Despite the fact that death rates from peptic ulcer illness are low, the high predominance and the subsequent aggravation, enduring, and cost are expensive. Ulcers can develop in the esophagus, stomach or duodenum, at the margin of a gastroenterostomy, in the jejunum, in Zollinger-Ellison syndrome, and in association with a Meckel's diverticulum containing ectopicgastricmucosa[5]. Peptic ulcer disease is one of several disorders of the upper gastrointestinal tract that is caused, at least partially, by gastric acid. Patients with peptic ulcer disease may present with a range of symptoms, from mild abdominal discomfort to catastrophic perforation and bleeding[5].

History: John Lykoudis, an overall specialist in Greece, treated patients for peptic ulcer sickness with anti-infection agents, starting in 1958, well before it was usually perceived that microbes were a predominant reason for the disease.[2]Helicobacter pylori was rediscovered in 1982 by two Australian researchers, Robin Warren and Barry J. Marshall as a causative element for ulcers.[2] In their unique paper, Warren and Marshall fought that most stomach ulcers and gastritis were brought about by colonization with this bacterium, not by stress or hot food as had been expected before.[3]

The H. pylori theory was ineffectively received,3 so in a demonstration of self-trial and error Marshall drank a Petri dish containing a culture of creatures removed from a patient and after five days created gastritis. His side effects vanished following fourteen days, yet he took anti-toxins to dispense with the excess microbes at the encouraging of his significant other, since halitosis is one of the side effects of infection.[3]

AIM: To Study Peptic Ulcer and It's Management.

OBJECTIVE:

- A peptic ulcer is a hole in the gut lining of the stomach, duodenum, or esophagus.
- A peptic ulcer of the stomach is called a gastric ulcer; of the duodenum, a duodenal ulcer, and of the esophagus, an esophagus ulcer .[1]
- Peptic ulcer illness is normal, influencing a huge number of Americans yearly. The clinical expense of treating peptic ulcer and it's difficulties runs in the billions of dollars yearly.
- Ongoing clinical advances have expanded how we might interpret ulcer arrangement. Improved and extended treatment choice are presently available[1]

2. 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. [5] Peptic ulcers are generally caused by acid resistant bacteria 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.

3. GENETIC FACTOR

Severe Stress [8] (e.g.: Trauma, Burns):

Numerous studies have revealed conflicting conclusions regarding the role of psychological factors in the pathogenesis and natural history of peptic ulcer disease. The role of psychological factors is far from established. Acute stress results in increases in pulse rate, blood pressure and anxiety, but only in those patients with duodenal ulcers did acute stress actually result in significant increases in basal acid secretion[8][9]. There is no clearly established “ulcer-type” personality. Ulcer patients typically exhibit the same psychological makeup as the general population, but they appear to perceive greater degrees of stress. In addition, there is no evidence that distinct occupational factors influence the incidence of ulcer disease.

Alcohol, Diet:

Although alcohol has been shown to induce damage to the gastricmucosa in animals, it seems to be related to the absolute ethanol administered (200 proof). Pure ethanol is lipid soluble and results in frank, acute mucosal damage[9]. Because most humans do not drink absolute ethanol, it is unlikely there is mucosal injury at ethanol concentrations of less than 10% (20 proof). Ethanol at low concentrations (5%) may modestly stimulate gastric acid secretions; higher concentrations diminish acid secretion. Though physiologically interesting, this has no direct link to ulcerogenesis or therapy[9]

Smoking [10]:

The writing uncovers areas of strength for a connection between's cigarette smoking and the occurrence of ulcer sickness, mortality, complexities, repeats and defer in mending rates. Smokers are twice bound to foster ulcer infection than nonsmokers. Cigarette smoking and H. pylori are co-factors for the arrangement of peptic ulcer illness. There is areas of strength for a between H. pylori disease and cigarette smoking in patients with and without peptic ulcers[7]. Cigarette smoking might increment defenselessness, decrease the gastric mucosal protective factors, or may give a better milieu to H. [7][8][9]

4. SIGNS AND SYMPTOMS ASSOCIATED WITH PEPTIC ULCER

Symptoms of a peptic ulcer can be:

- Stomach torment, traditionally epigastric with seriousness connecting with eating times, after close to three hours of taking a feast swelling and stomach fullness[11];
- water reckless (surge of spit after an episode of disgorging to weaken the corrosive in throat - albeit this is more connected with gastroesophageal reflux illness); sickness, and overflowing spewing; loss of hunger and weight loss[11]
- Hematemesis (retching of blood); this can happen because of draining straightforwardly from a gastric ulcer, or from harm to the throat from serious/keeping on regurgitating [11]. melena (delay, putrid excrement because of oxidized iron from hemoglobin);
- Once in a blue moon, a ulcer can prompt a gastric or duodenal hole, which prompts intense peritonitis. This is very difficult and requires prompt surgery[11][12].

Epidemiology:

A minority of cases of *H. pylori* infection will eventually lead to an ulcer and a larger proportion of people will get non-specific discomfort, abdominal pain or gastritis.

Peptic ulcer disease had a tremendous effect on morbidity and mortality until the last decades of the 20th century, when epidemiological trends started to point to an impressive fall in its incidence.[16] The reason that the rates of peptic ulcer disease decreased is thought to be the development of new effective medication and acid suppressants and the discovery of the cause of the condition, *H. pylori*.

In the United States about 4 million people have active peptic ulcers and about 350,000 new cases are diagnosed each year. Four times as many duodenal ulcers as gastric ulcers are diagnosed.

5. DIAGNOSIS

Radiological Diagnosis:

Though less invasive than endoscopy, the barium x-ray is limited by being less sensitive and accurate at defining mucosal disease, or distinguishing benign from malignant ulcer disease (Figure no-2). In patients who have anatomic deformities from previous gastric surgery or scarring from chronic inflammation, barium x-rays may be difficult to interpret medication [19] . Generally, these x-rays have up to a 30% false negative and a 10% false positive rate. Until 1970, peptic ulcers were diagnosed almost exclusively by radiological methods [20]

The most common inaccuracies of radiological diagnosis include the failure to recognize true ulcers, or the misdiagnosis of a scar or a deformed duodenal bulb as a true ulcer. Since the 1970s, increasing numbers of peptic ulcers are diagnosed by endoscopy [24].

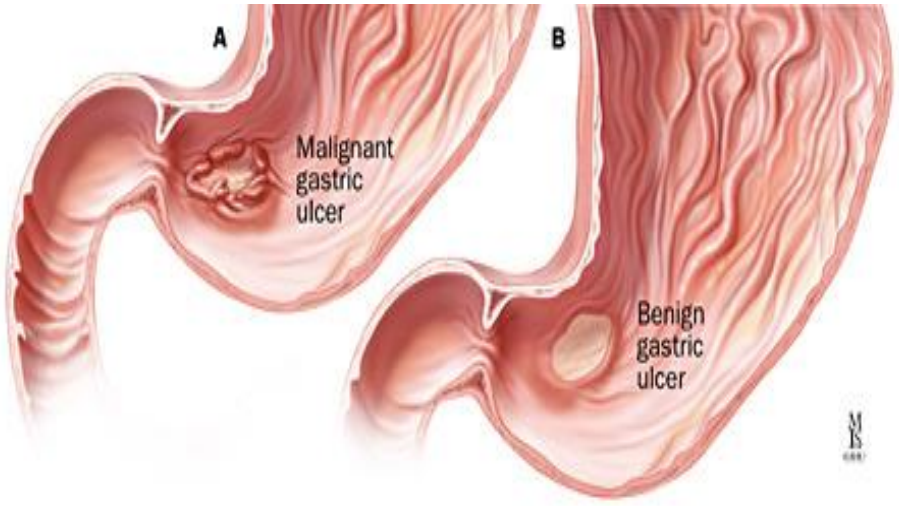


Figure no-2. Peptic ulcers; A. malignant; B. benign.

Laboratory Testing:

Patients who respond to optimal therapy for peptic ulcer disease do not require specialized testing. However, those with refractory (not healed after 8 weeks of therapy) or recurrent disease should have serumgastrin and serum calcium measured to screen for gastrinoma and multiple endocrineneoplasia (MEN). These patients should also undergo gastric acid analysis to determine whether the ulcer is caused by gastric acid hypersecretion (basal acid output exceeding 10 mEq/hr) or decreased mucosal protection[20][21].Urea breath tests are simple and noninvasive, and have been used to diagnose H. pylori infection. Because H. pylori produce large quantities of the enzyme urease, these breath tests have the potential to be quite useful. 13C- and 14C-urea breath tests offer excellent diagnostic yield. Patients ingest a solution containing 13C- or 14C-labeled urea and an exhaled breath is sampled for isotope-labeled CO2 released by intragastric H. pylori urease activity. The test can be completed within 20 minutes and is highly sensitive and specific (Figure no-3).

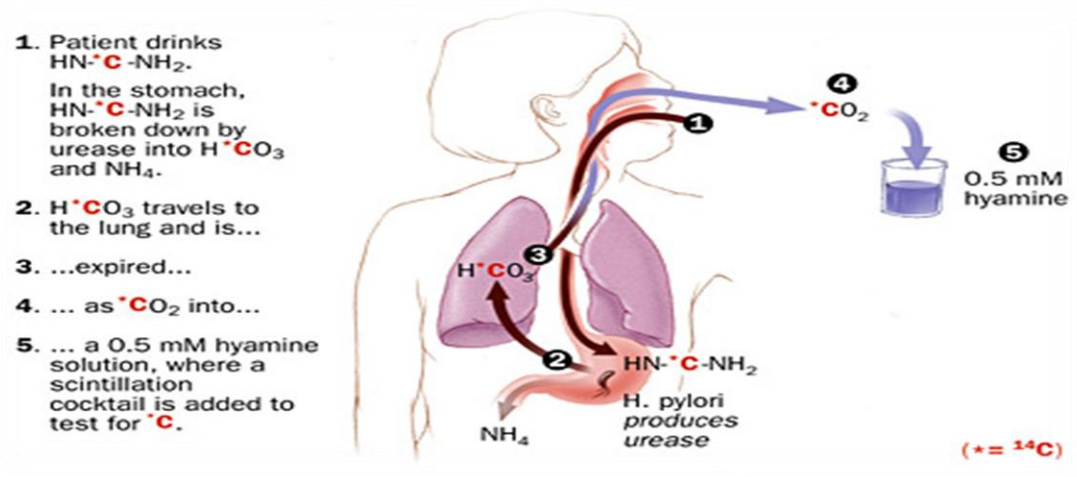


Fig. No. 3 urea breath test determines the presence of H. pylori

Blood Tests:

Blood tests, for example, the protein connected immunosorbent examine (ELISA) and fast office-based tests recognize and gauge H. pylori antibodies. The body produces antibodies against H. pylori trying to battle the bacteria19. Blood tests are modest and simple to use for specialists. Nonetheless, the disservice is that there is an expanded gamble of getting a bogus positive test, particularly in individuals who have had a H. pylori contamination before.

Breathe Tests:

Breath tests measure the amount of carbon dioxide in exhaled breath. Patients are given a substance called urea with carbon to drink. Bacteria break down this urea and the carbon is absorbed into the blood stream and lungs, and exhaled in the breath. By collecting the breath, doctors can measure this carbon and determine whether H. pylori are present or absent. Urea breath tests are at least 90 percent accurate for diagnosing the bacteria and are particularly suitable to follow-up treatment to see if bacteria have been eradicated[20][21]].

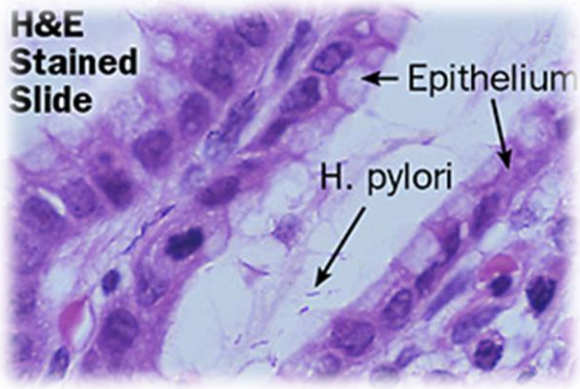
Endoscopic Diagnosis:

Gastrointestinal endoscopy allows the physician to visualize and biopsy the upper gastrointestinal tract including the esophagus, stomach and duodenum [22].The enteroscope (a longer endoscope) allows visualization of at least 50% of the small intestine, including most of the jejunum and different degrees of the ileum. During these procedures, the patient is given a numbing agent to help prevent gagging. Pain medication and a sedative may be administered prior to the procedure. The patient is placed in the left lateral position.

An endoscope (a thin, flexible, lighted tube) is passed through the mouth and pharynx and into the esophagus. The forward-viewing scope transmits an image of the esophagus, stomach and duodenum to a monitor visible to the physician (Figure no-4). Air may be introduced into the stomach, expanding the folds of tissue, and enhancing examination of the stomach .[22]

Fig no. 4-Endoscope

Esophagogastroduodenoscopy (EGD) is the most direct and most accurate method of establishing the diagnosis of peptic ulcer disease. In addition to identifying the ulcer, its location and size, EGD also provides an opportunity to detect subtle mucosal lesions and to biopsy lesions to establish histopathological basis. Endoscopic biopsies are indicated for all gastric ulcers at the time of diagnosis, whereas duodenal ulcers are almost always benign, not requiring biopsy in usual circumstances. [23]Endoscopic biopsy also appears the best and most accurate diagnostic method for H. pylori. Histological examination with standard hematoxylin and eosin staining provides an excellent means of diagnosis (Figure no-5).



In an effort to speed up the diagnosis of H. pylori following a biopsy of the gastricmucosa, urease activity has been used. Biopsy specimens are placed in a urea and phenol red solution or gel. If urease from H. pylori is present in the specimen, urea is hydrolyzed to release ammonia, increasing pH in the solution and giving a pink color to the gel or solution. At 3 hours, this test has a sensitivity of 90%. Using this technique, the diagnosis can be made sooner than standard histopathological examination. [23]

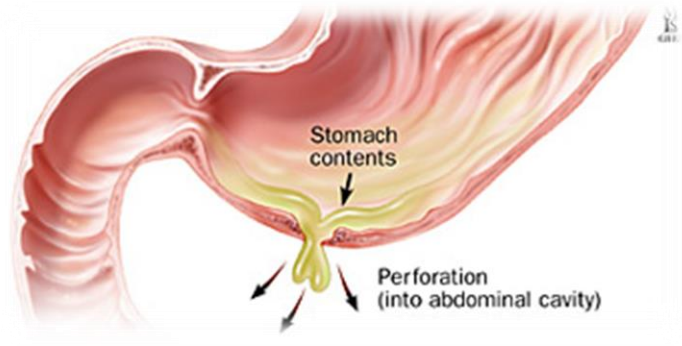
6. COMPLICATIONS

Hemorrhage:

Discharge, hole/entrance, and gastric outlet hindrance keep on being the significant complexities related with peptic ulcer infection, regardless of the accessibility of powerful ulcer medications14. In the US, the yearly confusion rate ranges between 2-5%. Gastrointestinal hemorrhage affects 5–20% of patients (more often those with duodenal ulcers) and is the most common complication of peptic ulcer disease. Bleeding occurs more often in men than in women. Hemorrhage from ulcers stops spontaneously in approximately 75–80% of cases.[26]

Perforation:

Roughly 5-10% of patients with peptic ulcers experience a hole into the stomach pit (Figure no-6). This rate is higher in men than in ladies. Roughly 15% of patients bite the dust from ulcer perforation.[27]



Two sorts of hole of the stomach and duodenum have been noticed. Free hole happens when duodenal or gastric items spill into the stomach cavity with peritoneal tainting by gastric, pancreatic and biliary juices[24]. Clinically this creates an acuteabdomen, which is effortlessly analyzed. Contained hole happens when the ulcer creates a full-thickness opening in the duodenum or stomach, yet the omentum or other neighboring organs forestall peritoneal defilement.

Penetration:

The acute onset of associated complications, such as pancreatitis, cholangitis, or diarrhea of undigested food, may diagnose penetration.[26] The diagnosis of penetration is more difficult than perforation, and is based on a combination of severe ulcer symptoms, atypical pain distribution, and diminished response to the usual therapy. Surgery is usually not recommended in the management of penetration unless biliary complications are present or the underlying peptic disease is severe.

Gastric Outlet Obstruction:

Fewer than 5% of patients develop gastric outlet obstruction from pyloric stenosis. Duodenal ulcers give rise to pyloric stenosis more often than gastric ulcers. Peptic ulcer disease may be accompanied by varying degrees of obstruction caused by inflammatory swelling of the pyloric channel or chronic scarring associated with fibrosis.

Patients with gastric outlet obstruction usually have a history of nausea, vomiting, and epigastric pain or fullness. Laboratory findings may show anemia, low serumalbumin, and hyperkalemicalkalosis. Radiological exam is usually diagnostic, showing a large gastric shadow with an air/fluid level (Figure no-7). An upper GI series yields valuable information by showing marked delay in gastric emptying and a large atonic stomach. Endoscopy is the best test for evaluating gastric outlet obstruction after decompression of the stomach for 12–24 hours. [26][27]

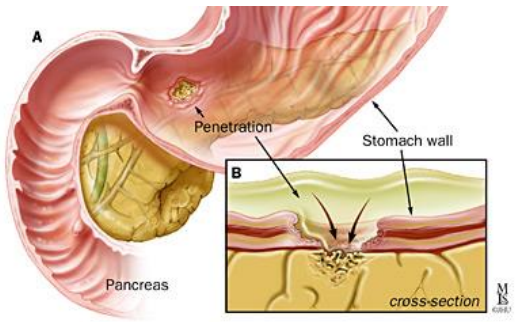


Fig no-7. Penetration and Gastric outlet obstruction

7. CLASSIFICATION OF DRUGS USED IN TREATMENT

A) Reduction of gastric acid secretion

- **a) H2 antihistamines :** Cimetidine, Famotidine, Nizatidine, Rantidine
- **b) Proton pump inhibitors :** Lansoprazole, Omeprazole, Esomeprazole, rabeprazole, Pantoprazole.
- **c) Anticholinergics :** Pirenzepine, Propantheline, Oxyphenonium.
- **d) Prostaglandin analogues:** Misoprostol, Enprostil, Rioprostil.

B) Neutralization of gastric acid (Antacids)

a) Systemic antacid : Sodium bicarbonate, Sodium citrate

b) Non systemic antacids : Magnesium hydroxide, Magnesium trisilicate, Magaldrate, Aluminium hydroxide gel,

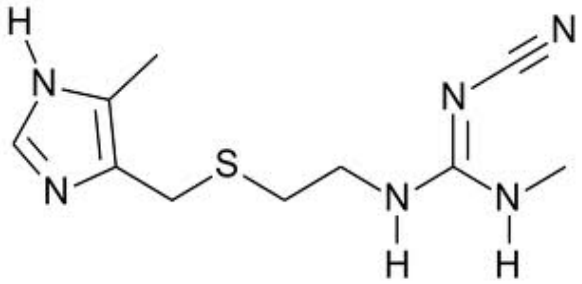
c) Anti H pylori drugs : Amoxicillin, Clarithromycin, Metronidazole, Tinidazole, Tetracyclin.

8. DRUGS USED IN PEPTIC ULCER

A. H2-RECEPTOR ANTAGONISTS:

Hinder the activity of receptor on the parietal cell, which represses corrosive discharge. The four medications in this class are similarly successful and are available without a prescription in half original potency for acid reflux treatment. Albeit the IV organization of H2 blockers might be utilized to treat intense inconveniences, the advantages are yet to be demonstrated

1. Cimetidine [30]:



Cimetidine is generally utilized in the treatment of acid reflux and peptic ulcers. It has been advertised by GlaxoSmithKline (which is offering the brand to Esteem Brands) under the exchange nameTagamet(sometimes Tagamet HB or Tagamet HB200). Cimetidine was supported in the UK in 1976 and was endorsed in the US by the Food and Medication Organization for remedies beginning January 1,1979.

Drug Class And Mechanism:

Cimetidine belongs to a class of medications called histamine H2-antagonists. Histamine is a natural chemical that stimulates stomach cells to produce acid. Histamine H2-antagonists inhibit the action of histamine on the acid-producing cells of the stomach and reduce stomach acid.

Adverse Effects:

- Galactorrhea
- Gynecomastia
- Antimalarial medication hydroxychloroquine
- Enhancing Estrogen Activity[30]

Interactions:

The development of longer-acting H_2-receptor antagonists with reduced adverse effects, such as ranitidine, proved to be the downfall of cimetidine and, though it is still used, it is no longer among the more widely used H_2-receptor antagonists. Side effects can include dizziness, and more rarely, headache.

It is likewise critical to perceive that cimetidine can cooperate with various different psychoactive prescriptions, remembering those for the classes of Tricyclic Antidepressants and Particular Serotonin Reuptake Inhibitors, causing expanded blood levels of these medications and ensuing harmfulness.

Dosing:

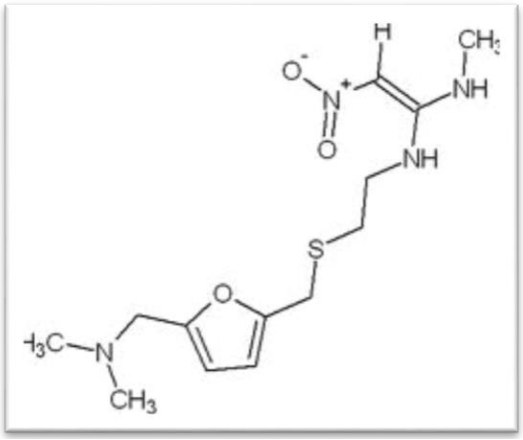
- Duodenal ulcers are treated with 800 mg at bedtime, 300 mg 4 times a day at meal times and bedtime, or 400 mg twice a day for 4-6 weeks. Maintenance therapy is 400 mg at bedtime.
- Active gastric ulcers are treated with 800 mg at bedtime or 300 mg 4 times a day at meal times and bedtime for up to 8 weeks.

- The regimen for GERD is 800 mg twice a day or 400 mg 4 times a day for 12 weeks.
- Pathological hypersecretory conditions are treated with 300 mg 4 times daily up to 2400 mg daily.

Uses:

1. Cimetidine is utilized to treat ulcers of the stomach and digestive organs and keep them from returning after they have recuperated.
2. This prescription is likewise used to treat specific stomach and throat (throat) issues brought about by an excessive amount of stomach corrosive.
3. Diminishing additional stomach corrosive can assist with alleviating side effects, for example, stomach torment, indigestion, trouble gulping, tireless hack, and inconvenience resting.
4. It can likewise forestall serious corrosive harm to your stomach related framework (e.g., ulcers, malignant growth of the throat).
5. Cimetidine has a place with a class of medications usually called H2 blockers.

i. Ranitidine[32][l]:



Drug Class And Mechanism:

Ranitidine is an oral drug that blocks the production of acid by acid-producing cells in the stomach. It belongs to a class of drugs called H2 (histamine-2) blockers that also includes cimetidine (Tagamet), nizatidine (Axid), and famotidine (Pepcid)[32][33]. Histamine is a naturally-occurring chemical that stimulates cells in the stomach (parietal cells) to produce acid. H2-blockers inhibit the action of histamine on the cells, thus reducing the production of acid by the stomach. Since excessive stomach acid can damage the esophagus, stomach, and duodenum and lead to inflammation and ulceration, reducing stomach acid prevents and heals acid-induced inflammation and ulcers [32][33].

Dosing:

- Ranitidine may be taken with or without food.
- Usual oral doses for treating ulcers and GERD are 150 mg twice daily or 300 mg at bedtime. The maintenance dose is 150 mg daily.
- Erosive esophagitis is treated with 150 mg 4 times daily.
- Zollinger Ellison syndrome may be treated with as much as 6 g daily.
- Heartburn is treated with 75 mg or 150 mg once or twice daily 30-60 minutes before consuming meals or beverages that cause heartburn. Self-medication should not last longer than 2 weeks unless advised by a physician.

Drug Interactions:

Ranitidine, like other drugs that reduce stomach acid, may interfere with the absorption of drugs that require acid for adequate absorption. Examples include iron salts (for example iron sulphate), itraconazole (Sporanox), and ketoconazole (Nizoral, Extina, Xolegel, Kuric).

- **Pregnancy:** There are no adequate studies of ranitidine in pregnant women. Available evidence suggests that there is little risk when used during pregnancy.
- **Nursing Mothers:** Ranitidine is secreted into human breast milk and may pose a potential risk to the infant.

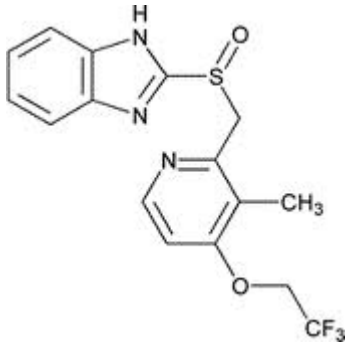
Side Effects:

Minor side effects include constipation, diarrhea, fatigue, headache, insomnia, muscle pain, nausea, and vomiting. Major side effects are rare; they include: agitation, anemia, confusion, depression, easy bruising or bleeding, hallucinations, hair loss, irregular heartbeat, rash, visual changes, and yellowing of the skin or eyes.

B. 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

i. **Lansaprazole [33]:**



Drug Class and Mechanism:

Lansoprazole is in a class of medications called proton siphon inhibitors (PPI) which block the creation of corrosive by the stomach. Different medications in a similar class incorporate rabeprazole(Aciphex), omeprazole (Prilosec), pantoprazole (Protonix), and esomepra zole (Nexium)[33]. Proton siphon inhibitors are utilized for the treatment of conditions like ulcers, gastroesophageal reflux sickness (GERD) and Zollinger-Ellison disorder that are brought about by stomach corrosive. Lansoprazole, as other proton-siphon inhibitors, impedes the compound in the mass of the stomach that produces corrosive. By hindering the protein, the creation of corrosive is diminished, and this permits the stomach and throat to heal.[33]

Dosing:

For initial treatment of duodenal ulcers the recommended dose for adults is 15 mg daily for 4 weeks. For the treatment of GERD, the recommended initial treatment is 15 mg for up to 8 weeks. For maintaining healing (long-term) in duodenal ulcer and GERD the recommended treatment is 15 mg daily. For initial treatment of severe (erosive) esophagitis and gastric ulcer, the recommended dose for adults is 30 mg daily for 4-8 weeks.

Drug Interactions:

Lansoprazole is less likely than omeprazole (Prilosec, Zegerid) to interact with other drugs.The absorption of certain drugs may be affected by stomach acidity, and, as a result, lansoprazole and other PPIs that reduce stomach acid also reduce the absorption and concentration in blood of ketoconazole (Nizoral) and increase the absorption and concentration in blood of digoxin (Lanoxin). This may lead to reduced effectiveness of ketoconazole or increased digoxin toxicity, respectively.

Uses:

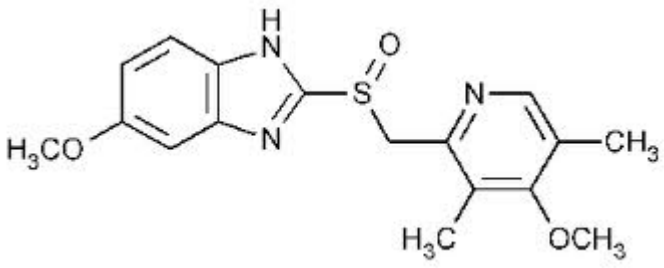
Lansoprazole works by blocking acid production in the stomach. This medication is known as a proton pump inhibitor (PPI). It is used to treat acid-related stomach and throat (esophagus) problems (e.g., acid reflux or GERD, ulcers, erosive esophagitis, Zollinger-Ellison syndrome). Decreasing excess stomach acid can help relieve symptoms such as heartburn, difficulty swallowing, persistent cough, and trouble sleeping. It can also prevent serious acid damage to your digestive system (e.g., ulcers, cancer of the esophagus).Lansoprazole may also be used to treat ulcers due to the long-term

use of certain drugs (nonsteroidal anti-inflammatory drugs or NSAIDs) for pain or swelling. In addition, this medication may be used in combination with antibiotics to treat certain types of ulcers caused by bacterial infection.

Side Effects:

Constipation or diarrhea may occur. If any of these effects persist or worsen, notify your doctor or pharmacist promptly.Remember that your doctor has prescribed this medication because he or she has judged that the benefit to you is greater than the risk of side effects. Many people using this medication do not have serious side effects.Tell your doctor immediately if this unlikely but serious side effect occurs: stomach pain.Tell your doctor immediately if any of these highly unlikely but very serious side effects occur: signs of vitamin B-12 deficiency with long-term (over 3 years) treatment[35] (e.g., unusual weakness, sore tongue, numbness or tingling of the hands/feet).A serious allergic reaction to this drug is unlikely, but seek immediate medical attention if it occurs. Symptoms of a serious allergic reaction include: rash, itching/swelling (especially of the face/tongue/throat), dizziness, trouble breathing. This is not a complete list of possible side effects [33][34]

i. **Omeprazole[35]:**



Drug Class and Mechanism:

Omeprazole is in a class of drugs called proton pump inhibitors (PPI) that block the production of acid by the stomach. Other drugs in the class include lansoprazole (Prevacid), rabeprazole (Aciphex), pantoprazole (Protonix), and esomeprazole (Nexium). Proton pump inhibitors are used for the treatment of conditions such as ulcers, gastroesophageal reflux disease (GERD) and the Zollinger-Ellison syndrome, which are all caused by stomach acid.[34][35] Omeprazole, like other proton-pump inhibitors, blocks the enzyme in the wall of the stomach that produces acid. By blocking the enzyme, the production of acid is decreased, and this allows the stomach and esophagus to heal. Zegerid contains omeprazole and an antacid (sodium bicarbonate).

Dosing:

For ulcers, GERD, erosive esophagitis and eradication of H. pylori the recommended dose for adults is 20-40 mg daily. Ulcer healing usually occurs within 4-8 weeks. H. pylori infections are treated for 10-28 days. The usual dose for prevention of upper gastrointestinal bleeding in critically ill patients is 40 mg daily for 14 days. [33][34][35]

Drug Interactions:

Omeprazole potentially can increase the concentrations in blood of diazepam (Valium), warfarin (Coumadin), and phenytoin (Dilantin) by decreasing the elimination of these drugs by the liver.

Uses:

Omeprazole is combined with the antibiotics clarithromycin and amoxicillin (or metronidazole in penicillin-hypersensitive patients) in the 7–14 day eradication triple therapy for Helicobacter pylori. Infection by H. pylori is the causative factor in the majority of peptic and duodenal ulcers.

Adverse effects:

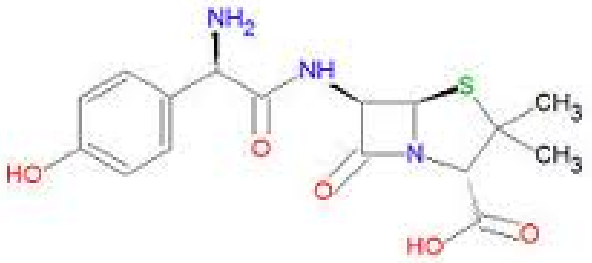
Some of the most frequent side effects of omeprazole (experienced by over 1% of those taking the drug) are headache, diarrhea, abdominal pain, nausea, dizziness, trouble awakening and sleep deprivation, although in clinical trials the incidence of these effects with omeprazole was mostly comparable to that found with placebo.[33]Other side effects may include iron and vitamin B12 deficiency, although there is very little evidence to support this.[34]

Interaction:-

Omeprazole is a cutthroat inhibitor of the compounds CYP2C19 and CYP2C9, and may in this way communicate with drugs that rely upon them for digestion, like diazepam, escitalopram, and warfarin; the groupings of these medications might increment on the off chance that they are utilized

correspondingly with omeprazole.[35]Clopidogrel (Plavix) is a dormant prodrug that to some extent relies upon CYP2C19 for change to its dynamic structure; restraint of CYP2C19 blocks the enactment of clopidogrel, hence decreasing its belongings and possibly expanding the gamble of stroke or cardiovascular failure in individuals taking clopidogrel to forestall these events.[33][34] Omeprazole is additionally a serious inhibitor of p-glycoprotein, as are other PPIs.[35]

ii. Amoxicillin[39]:



Amoxicillin (INN), formerly amoxycillin (BAN), and abbreviated amox, is a moderate-spectrum, bacteriolytic, β-lactamantibiotic used to treat bacterialinfections caused by susceptible microorganisms. It is usually the drug of choice within the class because it is better absorbed, following oral administration, than other β-lactam antibiotics. Amoxicillin is one of the most common antibiotics prescribed for children.is[36]Amoxicillin is susceptible to degradation by β-lactamase-producing bacteria, which are resistant to a broad spectrum of β-lactam antibiotics, such as penicillin.[37][39] For this reason, it is often combined with clavulanic acid, a β-lactamase inhibitor, and marketed under one name. This increases effectiveness by reducing its susceptibility to β-lactamase resistance

Interaction:

Amoxicillin may interact with the following groups of drugs:

- Anticoagulants (e.g. warfarin, pradaxa)[39]
- Allopurinol (gout treatment)
- Birth control pills[*citation needed*]
- Certain antibiotics
- Cancer treatment (methotrexate)
- Uricosuric drugs
- Typhoid vaccine

9. MECHANISM OF ACTION

Beta-lactam antibiotic:

This drug acts by inhibiting the synthesis of bacterial cell walls. It inhibits cross-linkage between the linear peptidoglycan polymer chains that make up a major component of the cell walls of both Gram-positive and Gram-negative bacteria.[36]It has two ionizable groups in the physiological range (the amino group in alpha-position to the amide carbonyl group and the carboxyl group).[37][39]

Dosing:

For most infections in adults the dosing regimens for amoxicillin are 250 mg every 8 hours, 500 mg every 8 hours, 500 mg every 12 hours or 875 mg every 12 hours, depending on the type and severity of infection.

For the treatment of adults with gonorrhea, the dose is 3 g given as one dose.

For most infections, children older than 3 months but less than 40 kg are treated with 25 mg/kg/day in divided doses every 12 hours, 20 mg/kg/day in divided doses every 8 hours, 40 mg/kg/day in divided doses every 8 hours or 45 mg/kg/day in divided doses every 12 hours depending on type and severity of the infection.

Side Effects:

Side effects due to amoxicillin include diarrhea, dizziness, heartburn, insomnia, nausea, itching,vomiting, confusion,abdominal pain, easy bruising, bleeding, rash, and allergic reactions. Individuals who are allergic to antibiotics in the class of cephalosporins may also be sensitive to amoxicillin.

Medical uses:

Amoxicillin is used in the treatment of a number of infections including: acute otitis media, streptococcal pharyngitis, pneumonia, skin infections, urinary tract infections, salmonella, lyme disease, and chlamydia infections.[36] It is used to prevent bacterial endocarditis in high risk people who are having dental work done, to prevent strep pneumococus infections in those without a spleen, and for both the prevention and the treatment of anthrax.[37] It is also a treatment for cystic acne.[39] The UK however does not recommend its use for infectious endocarditis prophylaxis.[39] These recommendations have not appeared to have changed the rates of infection.[39]

10. CONCLUSION

Peptic ulcer can be cured. The incidence can be reduced by proper diet control. Research is 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.

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