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Peptic Ulcers: Understanding Etiology, Pathophysiology, Diagnosis, Treatment of Peptic Ulcers

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ABSTRACT: Peptic ulcer disease (PUD) is a common gastrointestinal disorder characterized by acid-induced lesions in the digestive tract, predominantly affecting the stomach and the first segment of the duodenum. It results from extensive erosion of the mucosal lining, which can progress to the muscular layer due to the corrosive effects of gastric acid. PUD is classified into two main types: gastric ulcers, which occur in the stomach, and duodenal ulcers, which develop in the duodenum. The primary causes of PUD are infection with Helicobacter pylori and the use of non-steroidal anti-inflammatory drugs (NSAIDs), with additional contributing factors including a sedentary lifestyle, alcohol consumption, spicy foods, and other bacterial infections. Clinical manifestations include abdominal pain, nausea, vomiting, bloating, reduced appetite, weight loss, and, in severe cases, gastrointestinal bleeding evident through blood in vomit or dark stools. Diagnosis involves endoscopy, stool antigen tests, urea breath tests, and blood tests. Treatment strategies focus on eradicating H. pylori, reducing gastric acid production with proton pump inhibitors (PPIs) and H2 receptor antagonists, and using mucosal protective agents. Effective management requires a comprehensive approach addressing both the underlying causes and symptoms to reduce the global burden of morbidity and mortality associated with PUD.

Key Word: Peptic ulcer disease (PUD), Endoscopy, Malignant, Hypochlorhydria, Ulceration, NSAIDS.

I. INTRODUCTION

A peptic ulcer is an acid-induced lesion in the digestive tract, usually in the stomach or the first segment of the duodenum.The occurrence of extensive damage to the stomach lining, mucosa, and/or duodenum that extends over the mucous membrane is known as a peptic ulcer especially to the muscular layer because of the production of gastric acid in the surroundings .The erosion of the stomach or duodenal lining, which is made up of cells that secrete gastric acid and mucosa that shields stomach cells from gastric secretions, is known as Peptic Ulcer Disease (PUD). This erosion continues through the mucosa muscularis .PUD mostly comes in two varieties: gastric ulcer and duodenal ulcer .It is known as a gastric ulcer when it develops in the stomach. This kind of ulcer is caused by the bacteria H. pylori.A duodenal ulcer occurs when a gastric ulcer spreads to the duodenum. This kind of gastric ulcer develops in the first segment of the small intestine . These days, one of the most common chronic gastrointestinal disorders is peptic ulcers. It has now spread throughout the world and is a major cause of morbidity and mortality, impacting a vast number of people . Many factors, including a sedentary lifestyle, alcohol use, spicy food, medicines, and different bacterial infections, were identified that they are crucial in the etiology of ulcerations .But new research has revealed that these are only aggravating factors; the actual cause is an infection brought on by the H. pylori bacteria and a reaction to certain medications, such as non-steroidal anti-inflammatory drugs (NSAIDs).Pain and discomfort in the abdomen are signs of peptic ulcers. Additional symptoms include nausea, vomiting, bloating, reduced appetite, and weight loss. Additionally, some people may have blood in their vomit and faeces, and dark stools, which are a sign of bleeding in the stomach . Proton pump inhibitors (PPIs), H2 receptor antagonists, antacids, antibiotics, and mucosal protective agents are among the several medication regimens that are available for the treatment of peptic ulcers. Endoscopy, stool antigen testing, urea breadth testing, and blood tests are among the diagnostic procedures .

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II. ETIOLOGY:

2.1H. PYLORI

Peptic ulcers develop when the inner surface of the stomach or small intestine gets damaged by acid in the digestive tract, which is the organ through which food passes. An uncomfortable, sometimes bleeding open sore may result from the acid. The mucous layer that covers your digestive tract usually acts as a barrier against acid. However, you may get an ulcer if the amount of acid rises .Numerous gastrointestinal conditions, including gastritis, duodenal and stomach ulcers, gastric cancer, and lymphoproliferative disorders, have been linked to Helicobacter pylori infections . It has been discovered that eliminating this bacterium is of really important to reduce peptic ulcer problems. Thus, it is unavoidable that Helicobacter pylori infection is one of the primary etiologic factors for gastric ulcers, and its identification altered the way peptic ulcer disease patients are treated .Stomach ulcers are primarily caused by H. Pylori infections. Once this bacterium will penetrate the mucous layer of the gastric mucosa in the stomach and release toxins that harm the mucosa, stop the stomach lining's protective components from being produced, and cause scars ulcer . Helicobacter pylori only colonize gastric type epithelium, where it resides inside or beneath the mucus layer. By rupturing the mucus layer, attaching itself to the stomach epithelium, and releasing its contents, it increases the permeability of the underlying mucosa to acid reflux disease. Lastly, tissue damage is further exacerbated by the host immune system's inflammatory reaction to Helicobacter pylori . The synthesis of many enzymes, including phospholipase, catalase, and urease, can harm tissue either directly or indirectly. Proteolytic enzyme activity also breaks down mucus and increases tissue vulnerability to harm.Production of different enzymes such as urease, catalase and phospholipase can directly or indirectly damage tissue. In addition, proteolytic enzyme activity degrades mucus and makes tissue more susceptible to damage.About half of the world's population suffers from this common bacterial disease, which is found in the duodenum andstomach. For most people, it does not appear to cause any issues. The H. pylori infection primarily affects children. It is more common in developing countries. Approximately 5% of kids H. pylori bacteria are present in Americans under the age of ten. Children are more exposed to infection if they reside in crowded areas with unhygienic circumstances. It can be transferred from one person to another through close physical contact. Eating and drinking can also spread H. pylori.

2.2NSAIDS

Nonsteroidal anti-inflammatory drugs, or NSAIDs, are the root cause of the first stomach ulcer and promote its bleeding and rupture. NSAID usage can cause rapid irritation of the stomach lining, which promotes the development of ulcers. One family of chemicals called prostaglandins may be able to control the protective lining of the stomach. These substances may be affected by NSAIDs.

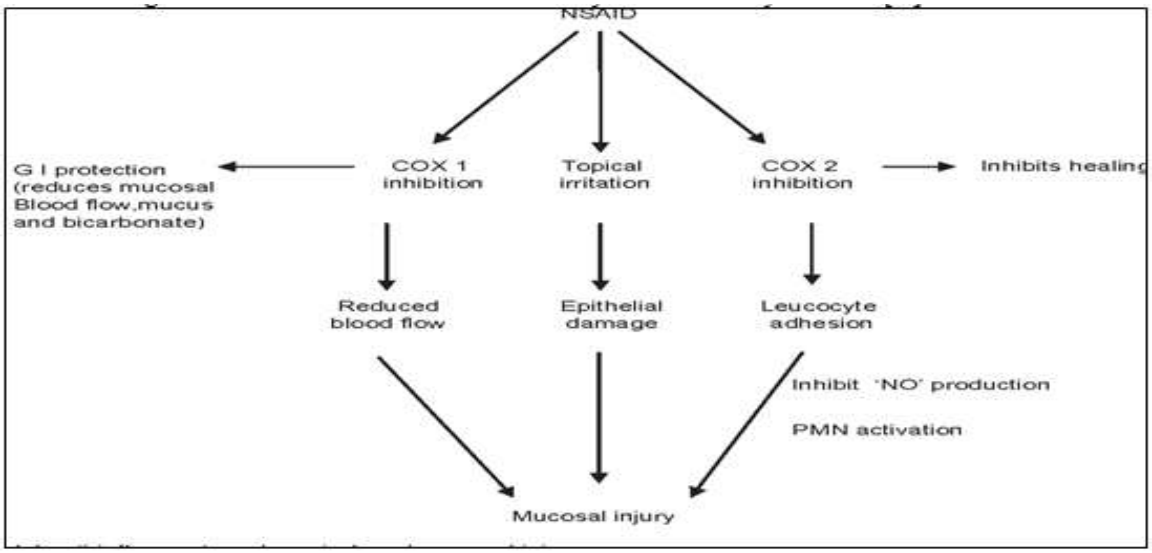

FIG: ETIOLOGY OF PEPTIC ULCERS BY NSAIDS

International Journal of Pharmaceutical Research and Applications
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Long-term usage of NSAIDs, including diclofenac, naproxen, and ibuprofen, can harm the stomach. These medications reduce the protective function of the gastric mucosa by inhibiting prostaglandin production, which leaves the stomach at greater risk of ulcers.Although NSAID side effects only affect a tiny percentage of users, the extensive use of these medications has contributed to an increasing percentage of people suffering from severe gastrointestinal issues. Through a variety of methods, such as the topical irritating action on the epithelium, mucosal impairment, inhibition of gastric prostaglandin production, decrease in stomach mucosal blood flow,andinterferencewiththe repair of minor damage. Mucosal stability depends on prostaglandins. The decreased activity of cyclo-oxygenase (COX 1 and COX 2), particularly COX 2, is thought to be the cause of stomach ulcers. Neutrophils harm the stomach mucosa by releasing oxygen free radicals, proteases, and decreasing capillary blood flow. The stability of the stomach mucosa is maintained by hydrogen sulphide (H2 S) and nitric oxide (NO), which are inhibited by NSAIDs. Because of these factors, NSAIDs remain at a greater risk of ulcer complications. To verify this claim, the level of the risk for ulcer complications in individuals with ulcers When compared to people who have never had an ulcer and have not been exposed to NSAIDs, their risk for ulceration increases by around 12 times..Numerous findings have indicated that there are genetic factors that contribute to the development of gastric ulcer disease. It has been shown that identical twins had a greater incidence for peptic ulcers than zygotic twins, and close relatives of ulcer patients have been shown to have a greater risk of peptic ulcer development. Both duodenal and gastric ulcers seem to have different biological accumulation: close relatives of patients with duodenal ulcers but not gastric ulcers have a threefold higher prevalence of duodenal ulcers, while relatives of patients with gastric ulcers have a threefold higher prevalence of gastric ulcers but not duodenal ulcers. It is unknown which genes are in charge of this ulcer susceptibility.

2.3LIFSTYLE FACTORS

Aspects of Lifestyle The following lifestyle decisions can raise the risk of ulcers, albeit they are not the primary factors:

- Smoking: decreases stomach ph. development, healing, and the mucosa's circulation system.
- Thespirit: Abuse of beverages may damage the stomach lining and increase acid production,bothof which can lead to ulcers.
- DietaryElements: Previously believed to be the primary causes of ulcers, caffeine and spicy foods are now shown to be aggravating factors.

2.4STRESS AND DIET

Anxiety triggered infections, which are induced by stomach acid, are known to be caused by stress gave by serious health problems, such as the need for treatment within a critical illness device . Coffee and caffeine have both frequently been thought to cause pain, but they seem to have less adverse effects as well.Anxiety, heart rate, and blood pressure

all rise in response to acute stress, but only basal acid secretion rise extremely in duodenal ulcer patients. There are no definite "ulcer-type" personalities. Although ulcer patients' psychological composition is essentially the same as that of the general population, they seem to be more susceptible to stress.According to some accounts, peptic ulcers may be made worse by emotional stress. Burns, severe illnesses, CNS damage, and surgery are stressful situations that might result in PUD. Sepsis, hypotension, and other conditions enhance the probability of after ulceration. many traumatic injuries, severe systemic disease, and respiratory failure. Complications such as stress ulceration and upper gastrointestinal bleeding are becoming more common in critically sick patients receiving intensive care. An elevated risk of gastric ulcers and bleeding is associated with severe sickness and a lower pH in the stomach. However, there are many disadvantages to research on the relationship between psychological factors and peptic ulcer illness, including the fact that psychological stress is hard to quantify and that ulcer disease etiology is complex. Psychological factors must be linked to clearly known pathways in the pathophysiology of peptic ulcer disease, such as the use of NSAIDs and Helicobacter pylori infections. Therefore, there is ongoing debate on the significance of psychodynamic variables in the development of peptic ulcers.Patients in critical and intensive care settings are particularly concerned about stress ulcers, which are caused by physiological stress not psychological stress and can result in upper gastrointestinal bleeding. They can be caused by one or more mucosal defects and are frequently linked to shock, sepsis, trauma, or chronic illnesses.

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2.5ZOLLINGER-ELLISON SYNDROME

Zollinger-Ellison syndrome is a relatively rare disorder that can potentially cause peptic ulcer disease. This disorder causes the digestive tract's acid-producing cells to grow into a tumour. These tumours may or may not be malignant. Excessive acid production by the cells harms stomach tissue.

III. PATHOGENESIS OF PEPTIC ULCERS

3.1HELICOBACTOR PYLORI

The duodenal side of the stomach is more informed about how the HP enhances the development of PU.H. pylori testing should be done on all individuals who acquired peptic ulcers. Chronic inflammation associated with an H. pylori infection can cause hypochlorhydria or hyperchlorhydria. which can help determine the kind of stomach ulcer that formed .One of the most usual causes of peptic ulcer illness. H. pylori, colonizes about half of the world's population. In underdeveloped countries, particularly in Africa, Central America, Central Asia, and Eastern Europe, H. pylori are more common.The bacteria usually grow up during childhood in crowded, unclean environments, primarily in lower socioeconomic nations. The inflammatory response including neutrophils, lymphocytes, plasma cells, and macrophages caused by H. pylori results in epithelial cell damage and degeneration, which is often more severe in the antrum. The It is not entirely clear how H. pylori cause the development of various diseases in the gastroduodenal mucosa.Depending on the kind of peptic ulcer, an H. pylori infection can cause either hypochlorhydria or hyperchlorhydria. Cytokines that suppress parietal cell secretion are the primary mediators of H. pylori infection; however, H. pylori can also directly impact the H+/K+ ATPase α-subunit, activate calcitonin gene-related peptide (CGRP) sensory neurons associated with somatostatin, or prevent the synthesis of gastrin.

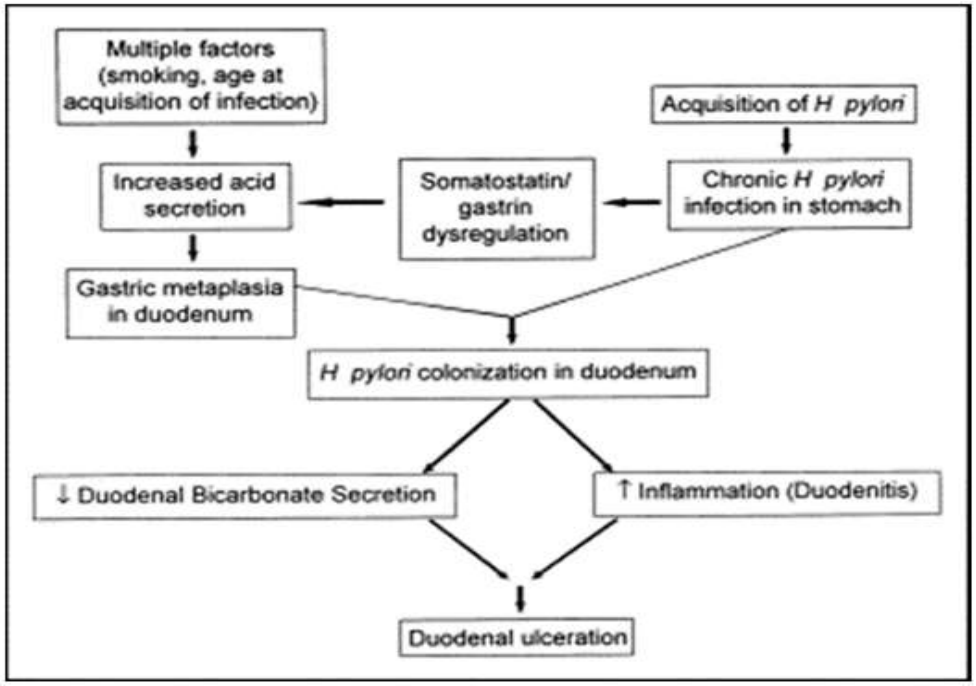

FIG: PATHOGENESIS OF ULCERS BY H. PYLORIBACTERIA

Although hyposecretion is linked to the development of gastric ulcers, 10–15% of individuals infected with H. pylori had lower antral somatostatinlevels andenhanced gastric secretion due to hypergastrinemia.This causes the parietal and gastric cells to secrete more histamine, which in turn causes them to secrete more acid or pepsin. Additionally, gastrin mRNA expression decreases and somatostatin mRNA expression increases when H. pylori is removed. For the vast majority of patients who stay, stomach ulcers are linked to mucosal degeneration and hypochlorhydria.

3.2NSAIDS

The systemic inhibition of constitutively expressed cyclooxygenase-1 (COX-1), which is in

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charge of prostaglandin synthesis, is the primary mechanism of NSAID-associated damage to the gastroduodenal mucosa. This mechanism is linked to reduced mucosal blood flow, low secretion of mucus and bicarbonate, and inhibition of cell division. NSAIDs reversibly and concentration-dependently inhibit the enzyme. Mucosal damage and ulcer risk are decreased when cyclooxygenase-2 (COX-2)-selective NSAIDs and exogenous prostaglandins are administered together. However, the toxicity of NSAIDs varies due to their distinct physicochemical characteristics.

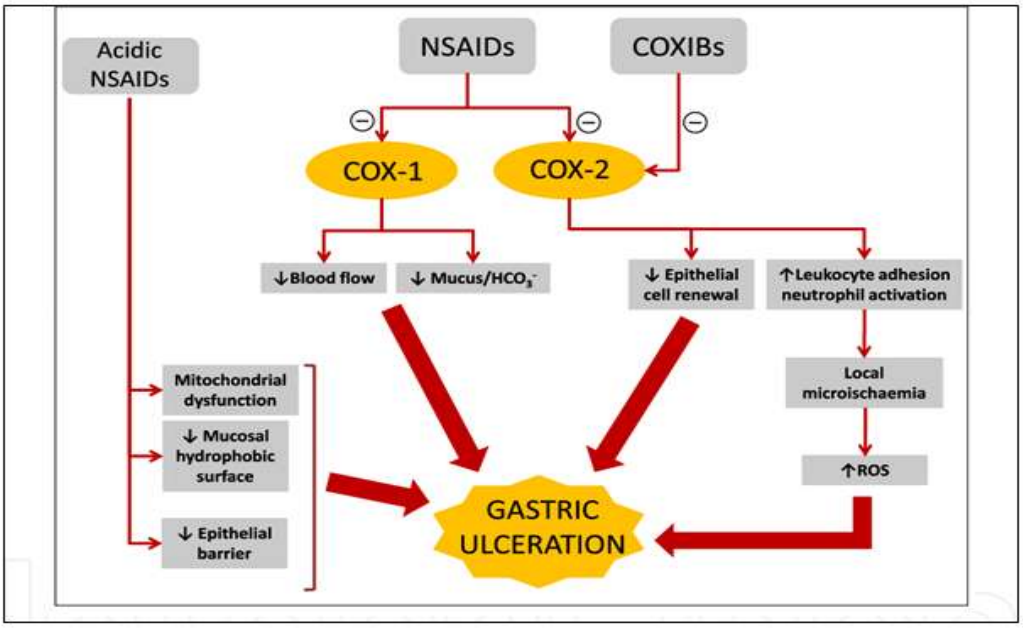

FIG:PATHOGENESIS OF ULCERS BY NSAIDS

NSAIDs interfere with mucus phospholipids and cause mitochondrial oxidative phosphorylation to become separate, which starts mucosal damage. When NSAIDs are exposed to stomach acid (pH 2), they get protonated, pass through lipid membranes, and enter epithelial cells (pH 7.4), where they ionize and release H+. When NSAIDs are stuck in epithelial cells due to their inability to pass the lipid membrane, oxidative phosphorylation becomes uncoupled, mitochondrial energy generation is reduced, decreased cell strength and increased cellular permeability. The patients most at risk for developing NSAID-induced ulcers include those who are over 65, have a history of peptic ulcers or haemorrhage, use steroids or anticoagulants, and take large dosages or combinations of NSAIDs.

IV. STAGES OF PEPTIC ULCERS

4.1. Acute Stage-Acute peptic ulcer disease symptoms are characterized by their fast formation, visible manifestation, and rapid progression. time. The condition can be totally cured at this point if it is identified and treated appropriately. But the majority of patients usually ignore the symptoms and, for whatever reason, do not visit the doctor, which increases the illness.

4.2. Chronic Stage-If acute peptic ulcer disease is not treated; it will cause swelling and inflammation for a long time and can ultimately turn into a chronic condition. The condition becomes more challenging to treat during the chronic stage, when the lesions spread and may even result in serious complications such as intestinal metaplasia, pyloric stenosis, bleeding, perforation, atrophic inflammation, and gastric cancer.

V. SIGN AND SYMPTOMS

Pain in the stomach that burns is the most typical sign of a peptic ulcer. Both stomach acid and an empty stomach increase the pain. Eating specific foods that buffer stomach acid or using an acid-reducing medicine can frequently ease the discomfort, but it may return. The discomfort could be more intense at night and in between meals. Many peptic ulcer sufferers don't even have symptoms . The duodenum, the first segment of the small intestine, is home to the duodenal ulcer. severe chest or lower abdominal discomfort along

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with an upper abdominal burning feeling. Patients with DU typically wake up from sleep in agony. Pain normally starts two hours after the stomach is empty. after eating or at night and feel relieved. Another name for DU is kissing ulcers. Painful gastric ulcers are found higher in the belly. In contrast to DU, eating in GU may make pain worse rather than better. GU symptoms include weight loss, nausea, and vomiting. Although GU patients often produce less acid, ulcers can nevertheless develop even when there is no acid present. Other symptoms might include nausea, vomiting, and weight loss. A blockage of the stomach's outflow, either whole or partial, might be the reason for the vomiting. In addition to being roused from sleep by the discomfort of duodenal ulcers, patients may also feel scorching or gnawing sensations in their upper belly. Sometimes, during the night or two hours after a meal, when the stomach is empty, discomfort may arise in the lower abdomen, back, or chest region. Relief is frequently felt after eating. Epigastric pain, melena from acute or subacute gastrointestinal bleeding, and complete obstruction of the gastric outlet are further symptoms of ulcer disease.

1.Stomachache that burns
2.An intolerance to fatty meals, as well as feelings of fullness,
3.bloating
4.Heartburn
5. Feeling nauseous
6.Many peptic ulcer sufferers are asymptomatic.
7.Less frequently, ulcers might result in serious symptoms like: Vomiting or throwing up blood,
8.which might be dark or red Tarry or black stools or dark blood in the stool Breathing difficulties Feeling lightheaded
9.Vomiting
10.Unexpected loss of weight
11.Changes in appetite.

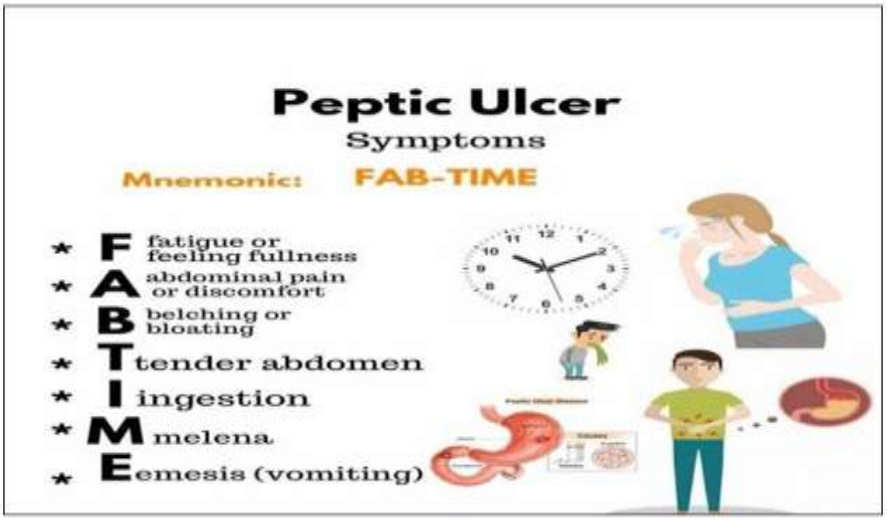

FIG: SYMPTOMS OF PEPTIC ULCER

VI. DIAGNOSIS

Up until the early 1900s, the majority of peptic ulcer diagnoses were made based on clinical indications and symptoms. Observing ulcer disease up close was transformed by a number of flexible endoscopies in the 1950s. A full clinical history and physical examination are necessary to create a complete list of all PUD symptoms and signs. All previous medical history must also be documented, including the duration of alcohol use, NSAID and smoking histories, and any possible peptic ulcer episodes. There are two primary considerations for diagnosing peptic ulcer disease. The first step is to determine whether the symptoms being described have nothing to do with functional dyspepsia, andfinding the exact source of the ulcer is the second step.

6.1. RADIOLOGY

Although endoscopic investigations have mostly replaced barium gastroduodenal studies in regular diagnostic procedures, they can still be helpful in a small number of patients who refuse to have them done or in situations where endoscopy is unavailable due to oesophageal constriction. The radiologist's expertise, the method employed, the ulcer depth, the size of the lesion (if it is less than

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0.5 cm in diameter, it may be hard to detect), and the methodology all affect the sensitivity and specificity of barium radiography investigations. Regular edges, symmetrical mucosal folds, a smooth, transparent band or collar, and indentation of the opposing wall around the ulcer crater are radiologic indicators that point to a benign condition. On the other hand, large ulcers, uneven mucosal folds, lack of contrast, or irregular filling are indicators of malignancy .

6.2BLOOD TEST

A blood test Blood tests can be used by doctors to look for symptoms of peptic ulcer complications or an H. pylori infection. A medical practitioner will draw blood from you for an NIH blood test, and the sample will be sent to a laboratory.

6.3UREA BREATH TEST

To screen for an H. pylori infection, doctors may do a urea breath test. You will take a pill, drink, or pudding containing urea that has been "labelled" with a unique carbon atom for thetest. The bacteria will turn the urea into carbon if H. pylori are present. Carbon dioxide. You will exhale carbon dioxide into a container after a few minutes.

6.4STOOL TEST

To screen for an H. pylori infection, doctors may use stool testing. A stool sample collection and keeping container will be provided by your physician. Instructions on where to mail or pick up the kit for testing will be sent to you.

VII. TREATMENT

An imbalance between protective mechanisms (such mucus and bicarbonate) and aggressive forces (like gastric acid) can lead to peptic ulcers, which are mucosal lesions in the stomach, duodenum, or oesophagus. The usage of nonsteroidal anti-inflammatory medicines (NSAIDs) and Helicobacter pylori infection are the main contributing causes. Recurrence prevention, healing promotion, and symptom relief are the main goals of effective management.

7.1ANTACIDS

Although antacids have mostly been replaced by histamine 2 antagonists and PPIs as the main therapy for the majority of acid-peptic illnesses, their usage is still justified since they are safe, affordable, and widely accessible. Antacids are useful for quickly relieving moderate or intermittent symptoms since they start working almost instantly. Antacids primarily affect the stomach by inhibiting the proteolytic enzyme pepsin and partially neutralizing gastric hydrochloric acid. Acid neutralization in the stomach lumen avoids the requirement for the medication to be absorbed systemically. All of them are taken orally, and the quantity of acid that an antacid dosage neutralizes is often used to gauge how effective they are. Antacids work to lower stomach acidity for a comparatively short period of time when taken empty, but this can be extended to one to three hours when taken with meals.Sodium bicarbonate, calcium carbonate, magnesium hydroxide, and aluminium hydroxide are among the commonly used antacids; the most common formulations are liquid suspensions or solid tablets. Sodium bicarbonate was first used as an antacid and is still used occasionally as a self-prescribed regimen of "baking soda" mixed in water or in combination products containing aspirin, but its soluble nature has made it less desirable because larger doses can cause systemic alkalosis and a high sodium load, which can be problematic in patients with systolic cardiac dysfunction or renal insufficiency.The small intestine's alkaline environment transforms the soluble calcium chloride produced by the reaction with HCl back into calcium carbonate. This reduces absorption by precipitating out into the stool. The insoluble antacids magnesium hydroxide and aluminium hydroxide are other frequently used agents. Constipation may result from items that include calcium and aluminium. In order to combat this, these substances are frequently mixed with magnesium hydroxide, which by itself can result in loose stools and diarrhoea.Compliance has been another factor affecting efficacy of antacids and it appears to be limited by the need for frequent dosing and the poor correlation between symptomatic relief and ulcer healing.

7.2H2-RECEPTOR ANTAGONIST

The basal rate of acid release during nonfeeding times is mostly mediated by histamine. This is especially crucial during the nighttime fasting periods, which is why H2RA dosage is used before bedtime. With a pH of over 3 for duodenal ulcers and a pH of over 4 for GERD, models based on data from clinical trials and 24-hour pH monitoring have shown that ulcer healing is dependent on the degree of acid suppression and the length of the 24-hour cycle with decreased acidity. With their heterocyclic rings, cimetidine, ranitidine, famotidine, and nizatidine often share

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structural and pharmacokinetic traits. Following oral dosage, H2RAs are efficiently absorbed in the small intestine and reach peak concentrations in 1-3 hours. Food consumption rarely affects this rate, however concurrent antacid treatment usage may. All drugs have linear pharmacokinetics and are mostly removed by the kidneys; between 30 and 60 percent of the medication is eliminated unaltered in the urine. Patients with renal impairment require dose modifications, whereas those with liver illness do not.In nonnuclear dyspepsia, histamine receptor antagonists are somewhat helpful, although they fall short of PPIs in terms of effectiveness. When it comes to treating oesophageal reflux disease, H2RAs are better than a placebo but worse than PPIs. In a similar vein, PPIs outperform H2RAs in preventing acute peptic ulcer rebleeding , as well as in reducing ulcer symptoms and promoting ulcer healing. While double-dose H2RAs were beneficial in lowering the risk of endoscopically visualized duodenal and gastric ulcers, regular dosages of H2RAs are effective in lowering the risk of duodenal but not gastric ulcers in the prevention of NSAID-induced damage.

7.3PROTON PUMP INHIBITOR

Inhibiting this stage in gastric acid generation has transformed the therapy of GI tract disorders since the gastric H+K+-ATPase was found to be the common mechanism for acid formation. The strongest inhibitors of stomach acid secretion are PPIs. Omeprazole, esomeprazole, lansoprazole, pantoprazole, and rabeprazole are the five commonly used PPIs.While intravenous omeprazole is used in other countries, intravenous formulations of pantoprazole, lansoprazole, and esomeprazole are available in the United States. In order to inhibit the H+K+-ATPase, PPIs, which are weak bases that function as prodrugs, require an acidic environment. All these compounds share a common structure consisting of substituted pyridylmethyl sulfinyl benzimidazoles that varies in terms of the substitutes on either the pyridine or the benzimidazole rings. As a result of their acid dissociation constant (pKa) levels, they accumulate in the secretion canaliculus of the parietal cell, achieving higher concentrations here when compared with plasma.After being protonated, the PPI transforms into the active sulfenamide species, which combines with cysteine residues in the H+K+-ATPase's α-subunit to generate disulfide bonds. This leads to an inhibitory mechanism that is not dependent on gastrin, cholinergic, or histamine stimulation for acid production, and a duration of action that surpasses plasma half-life. In contrast to H2RAs, PPIs also inhibit the release of pepsin, which helps to lessen injury to the mucosa.Furthermore, PPI dosing in the morning is linked to noticeably better acid suppression than H2RAs, whose ideal dosage occurs at night. PPIs should be taken before breakfast since eating will attract H+K+-ATPase to become active and susceptible to medication action, and the quantity of H+K+-ATPase in the parietal cells is at its highest following a protracted fast. With repeated administration, the effects of the PPIs intensify, and by the third day, a steady state is usually reached, with around 70% of the pumps remaining blocked for two hours. Furthermore, when the enzyme's recruitment rises, acid suppression gradually gets better. consequently, the periodic use of a PPI taken on a ‘as needed’ basis does not reliably provide adequate acid inhibition and does not produce a consistent or satisfactory clinical response. Proton-pump inhibitors undergo metabolism via hepatic CYP2C19. Of the PPIs, rabeprazole is particular as only 15–20% of its metabolism involves the CYP system. There is differential metabolism between individuals due to pharmacogenetic variation. Poor metabolizers constitute nearly 2–6% of Caucasian and 15–20% of Asian populations and they tend to have higher plasma drug levels, more profound acid inhibition and higher healing rates in PPI-containing H. pylori regimens. However, pharmacogenetic testing of patients has not been displayed to routinely improve outcomes and is not worked.

7.4MUCOSAL PROTECTIVE AGENTS

- **SUCRALFATE**

An aluminium salt of sulphated sucrose and aluminium hydroxide are the basic compounds that form sucralfate. It is a nonabsorbable medication that binds to gastric mucosa and an ulcer tissue. These properties support healing and provide cytoprotective effects. When exposed to gastric acid the sulphate ions bind to proteins in the damaged the gastric tissue of ulcer holes and stimulate vascular development, delivery of growth factors and formation of granulation tissue. This binding is supported by a low pH and is the reasons for use 30–60 min before meals. The drug is excreted in stool and only a minor increase on serum and urinary aluminium has been reported with its use, because of this issue that sucralfate is best avoided in patients with kidney failure.When compared to H2RAs, sucralfate is equally effective in repairing gastric and duodenal ulcers. The main use is to prevent stress ulcers in individuals who

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are in severe condition. Although there is a lack of clinical trial evidence, sucralfate seems to be just as effective as H2RAs at preventing haemorrhage and stress ulcers in critical care. By keeping the intragastric pH lower and preserving the sterilizing effects of an acidic stomach, sucralfate may be superior to H2RAs and maybe PPIs in the prevention of stress-related mucosal damage. As a result, compared to antacids and H2RAs, it has been shown to have a decreased incidence of nosocomial pneumonia.

- **BISMUTH**

Bismuth salicylate, a frequently used salt of salicylic acid, has antacid qualities. The US FDA has authorized the use of bismuth in conjunction with other treatments for eliminating H. pylori because it inhibits the bacteria. Inhibition of pepsin activity, an increase in mucosal prostaglandin synthesis, and the release of mucus and bicarbonate are other mechanisms that may aid in ulcer healing. It is eliminated in faces and is mostly unabsorbed. The faces turn black as a result of its reaction with hydrogen sulphide in the colon to produce bismuth sulphide. It is now used in H. pylori regimens and has moderate effectiveness in treating nonnucleardyspepsia.

7.5PROSTAGLANDINS ANALOGS

The theoretical basis for prostaglandin therapy is to fight the systemic effects of NSAIDs and improve epithelial cell growth and repair. Early work resulted to the development of misoprostol, arbaprostil, enprostil and rioprostil. Of these, analogue approved by misoprostol is the only prostaglandin E2 the FDA for the prevention of NSAID-related ulcers and is designed to help change the NSAID-induced deficiency of prostaglandins in the gastric mucosa It is frequently administered by mouth with a good absorption reaching a peak plasma concentration in 30 min and a half-life of 1.5 h. The drug has no effect in the enzyme CYP system and its metabolites are removed through urine.The only prostaglandin analogue that has been shown to lessen severe gastroenterological side effects from NSAID treatment is misoprostol, which has also been shown to be more effective than H2RAs at preventing stomach ulcers but not duodenal ones.

VIII. CONCLUSION:

In conclusion, peptic ulcer disease (PUD) remains a prevalent and significant global health concern, characterized by acid-induced erosions in the stomach or duodenum. The primary etiological factors include Helicobacter pylori infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs), with additional contributors such as lifestyle choices, alcohol consumption, and dietary habits acting as aggravating factors. The clinical presentation often involves abdominal pain, discomfort, and associated symptoms like nausea, bloating, and signs of gastrointestinal bleeding in severe cases. Effective management of PUD relies on a combination of pharmacological treatments, including proton pump inhibitors (PPIs), H2 receptor antagonists, antibiotics for H. pylori eradication, and mucosal protective agents. Accurate diagnosis through endoscopy, stool antigen tests, urea breath tests, and blood tests is critical for targeted therapy. Addressing both the underlying causes and symptoms is essential to reduce the morbidity and mortality associated with PUD, emphasizing the importance of early detection, appropriate treatment, and lifestyle modifications in the prevention and management of this common gastrointestinal disorder.

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