A Review On Peptic Ulcer Disease

Wrushabh Dilip Tarare[1], Nutan Khemraj Pustode[2]
Maharashtra Institute of Pharmacy (Dpharm), betala, Bramhapuri, Dr. Babasaheb Ambedkar Technological University, Lonere

Abstract- *Peptic ulcer disease is an important gastrointestinal illness characterized by the development of mucosal breaks into the stomach or duodenum. Its history runs from early clinical observations to major developments in the twentieth century, especially the recognition of Helicobacter pylori as a primary cause. The discovery shifted the focus from acid alone to a more complex interaction between infection, mucosal defense, and environmental triggers. Pathophysiologically, the development of ulcers is linked to an imbalance of aggressive factors such as acid, pepsin, H. pylori, and NSAIDs and protective mechanisms of the mucosal barrier, blood flow, and secretion of bicarbonate. Despite this, it remains a common disease, the prevalence of which varies in different geographic locations due to socioeconomic status, sanitation, medication, and lifestyle.*

Clinically, patients may have burning epigastric pain, early satiety, nausea, vomiting, bloating, or discomfort related to meals. Many patients remain asymptomatic until complications arise. Major risk factors include H. pylori infection, chronic NSAID or aspirin intake, smoking, alcohol use, stress-related mucosal injury, and family history of ulcer disease. If left unattended, peptic ulcers can result in gastrointestinal bleeding, perforation, peritonitis, gastric outlet obstruction, and a risk of recurrent ulceration.

Diagnosis is based on upper gastrointestinal endoscopy, which can visualize the disease directly and provides an opportunity for biopsy to investigate the presence of H. pylori. Non-invasive tests such as urea breath tests, stool antigen assays, and serology assist in diagnosis and follow-up. Prevention includes the rational use of NSAIDs, eradication of H. pylori infection, avoiding tobacco and excessive alcohol intake, and improving diet and stress management. The management focuses on the use of proton pump inhibitors, antibiotics to eradicate H. pylori, cessation of drugs that cause irritation, and supportive lifestyle modifications. The long-term prognosis is good with early detection and appropriate management, and complications may be significantly reduced.

Keywords- Peptic ulcer disease, Helicobacter pylori, gastric ulcer, duodenal ulcer, pathophysiology, epidemiology, NSAIDs, risk factors, diagnosis,prevention, treatment

I. INTRODUCTION

Ulcer

Ulcers are deep wounds that go all the way through the lining and muscle layers of the gastrointestinal tract. Peptic ulcers have been a big health problem in the twentieth century. Studies show that how common and severe this disease is varies a lot from place to place. There are several types of ulcers, but the most common ones are peptic ulcers. These include gastric ulcers, which happen when the stomach lining gets damaged, and duodenal ulcers, which are linked to too much stomach acid. For a long time, doctors argued about what causes peptic ulcers. Now, it's thought that ulcers happen when harmful factors like Helicobacter pylori, NSAIDs, and stomach acid outweigh the body's natural defenses, such as mucin, bicarbonate, and prostaglandins. Many factors can lead to ulcers, such as a sedentary lifestyle, drinking alcohol, eating spicy food, taking certain drugs, and bacterial infections. In animals, several substances are known to cause damage to the gut lining. These include bacteria, drugs, chemicals, stomach acid, lipid byproducts, nerve-related chemicals, inflammation-related substances, and harmful free radicals. Oxidative stress is a major factor in ulcer development because it harms cells and damages their important parts like mitochondria, lysosomes, and the nucleus. Nitric oxide is also important for protecting the stomach lining, and a lack of this substance can lead to ulcers. This study looks at how these substances cause ulcers and helps us better understand the causes of peptic ulcers.

II. TYPES OF ULCER

There are three types of ulcer,as follows:

1.Esophageal Ulcer
2.Aphthous Ulcer
3.Peptic Ulcer

Esophageal Ulcer

Esophageal ulcers are painful sores that form in the lining of the esophagus, usually near its lower end. They often cause a burning discomfort behind the breastbone and can resemble heartburn. These ulcers most often develop due to

acid reflux or GERD, but long-term use of medications like NSAIDs can also contribute. Early recognition and treatment help prevent complications and promote healing.

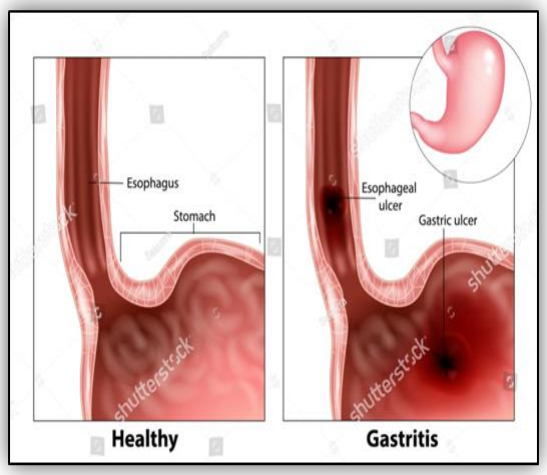

Fig.1: Esophageal Ulcer

1. Apthous Ulcer

Sores that appear on the inside of the mouth are called mouth ulcers. These ulcers are quite common and often happen because of injuries like those from poorly fitting dentures, broken teeth, or fillings. Some other causes include anemia, measles, viral infections, oral candidiasis, long-term infections, throat cancer, mouth cancer, and a lack of vitamin B. One of the most common types of mouth ulcers is called aphthous minor, and it affects about 15 to 20% of people around the world. In some groups, the rate can be as high as 50 to 66%, and it is especially common in North America. Studies have also found that people who smoke are less likely to get aphthous ulcers compared to those who don't smoke.[1]

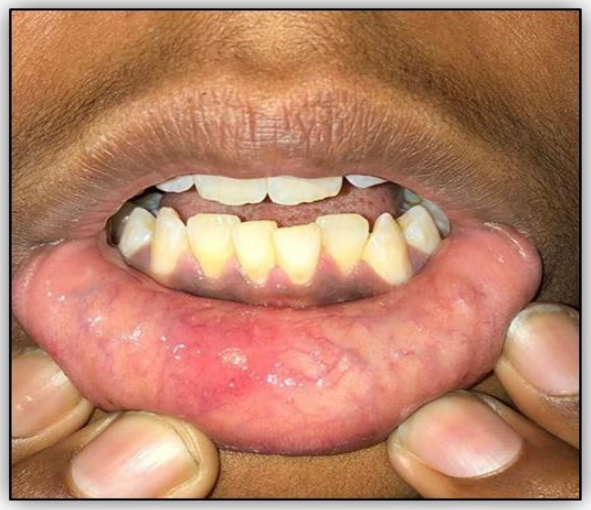

Fig.2: Apthous Ulcer

2. Peptic Ulcer

A peptic ulcer is when there is a deep loss of tissue in the lining of the stomach and the first part of the small intestine, going past the thin layer under the lining and usually reaching into the muscle layer, which is usually caused by the stomach's strong acid.[2]

Peptic ulcer is a general term for sores that form in the stomach or the first part of the small intestine called the duodenum. In the past, people thought stress and spicy food were the main causes of ulcers. But now, doctors know that these things can make ulcers worse, not cause them. The real causes are usually an infection from a bacteria called H. pylori or the use of certain medicines like non-steroidal anti-inflammatory drugs (NSAIDs). Symptoms of peptic ulcers often include stomach pain or discomfort. Other common signs are losing weight, not feeling like eating, feeling bloated, having nausea, and throwing up. Some people might also see blood in their stool or vomit, or have black stools, which can be a sign of bleeding in the digestive system.[3]

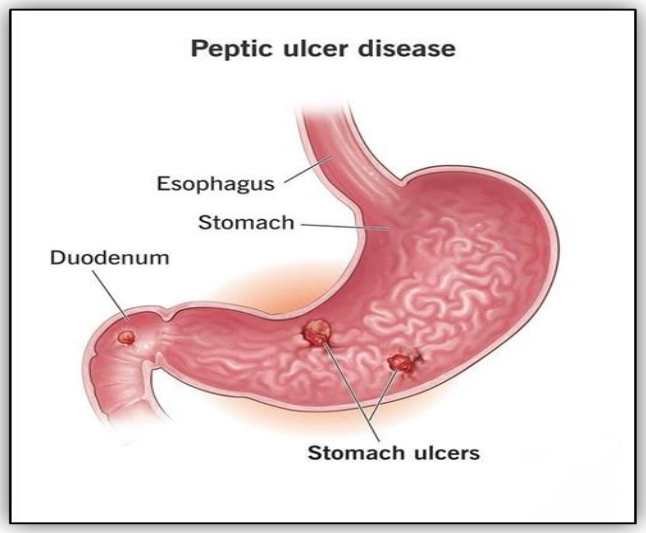

Fig.3: Peptic Ucer Disease

III. TYPES OF PEPTIC ULCER

Stomach and duodenal ulcers, commonly known as peptic ulcers, develop in the digestive tract. Earlier, people thought spicy food and stress were the main causes, but they actually just aggravate the condition. The real triggers are H. pylori infection and long-term use of medicines like NSAIDs. These ulcers may lead to weight loss, nausea, vomiting, a sense of fullness, and dark stools that can signal internal bleeding.

Mouth ulcers are small, painful sores inside the mouth. They often result from irritation caused by rough dentures, broken teeth, or damaged fillings. Other contributors include anemia, viral infections, measles, oral candidiasis, persistent infections, and certain cancers, along with vitamin B deficiency. Amonpg the different types, minor aphthous ulcers are the most common, affecting 15–20 percent of people worldwide. Rates are even higher in North America, where up to two-thirds of the population may experience them. Studies also show that smokers tend to develop aphthous ulcers less frequently than non-smokers.

There are 3 types of peptic ulcer:

1. Gastric ulcer

Gastric ulcers are a significant health concern in the United States and cost the healthcare system millions each year. These ulcers are deeper than 5 mm and extend through the stomach's mucosal layer into the muscle. The good news is that they can be treated and prevented, depending on the underlying cause. The stomach usually protects itself from acid through mucus, prostaglandins, growth factors, and steady blood flow. When this protection breaks down, the lining becomes vulnerable to damage, erosion, and ulcer formation. Factors that weaken this barrier include alcohol use, smoking, excess acid, poor blood flow, NSAID use, low oxygen levels, and infection by H.pylori(Helicobacter pylori).

2. Esofageal Ulcer

An esophageal ulcer is a distinct tear in the mucosal lining of the esophagus. This type of mucosal injury is commonly caused by severe, prolonged esophagitis from other sources or by gastroesophageal reflux disease. It is estimated that 2% to 7% of individuals will develop esophageal ulcers. The system utilizes Amazon Web Services (AWS), including Lambda, S3, IAM, CloudWatch, and Docker, to compress images immediately after they are uploaded. Endoscopic examination often reveals some degree of hiatal hernia in most patients. Normally, the lower esophageal sphincter (LES) prevents the reflux of stomach contents; however, when the LES weakens, this protective mechanism is compromised, exposing the esophageal mucosa to stomach acid and increasing the risk of ulceration. Additionally, as observed in patients with bulimia nervosa, recurrent vomiting exposes the esophageal mucosa to stomach contents, which can either lead to an ulcer or worsen an existing one.

3. Duodenal Ulcer

Peptic ulcer disease includes conditions like duodenal ulcers and involves damage to the protective lining of the stomach or the first part of the small intestine. This damage occurs when the mucosal barrier is disrupted. Dyspepsia is the most common symptom, but patients may also experience bleeding, obstruction, perforation, or fistula formation. Treatment depends on how the patient presents and how advanced the condition is. Anyone with a history of NSAID use or known Helicobacter pylori infection who reports upper abdominal pain or dyspepsia should be evaluated for a gastric or duodenal ulcer. Because H. pylori is a leading cause of peptic ulcer disease, all patients diagnosed with duodenal ulcers should be tested for the infection[4]

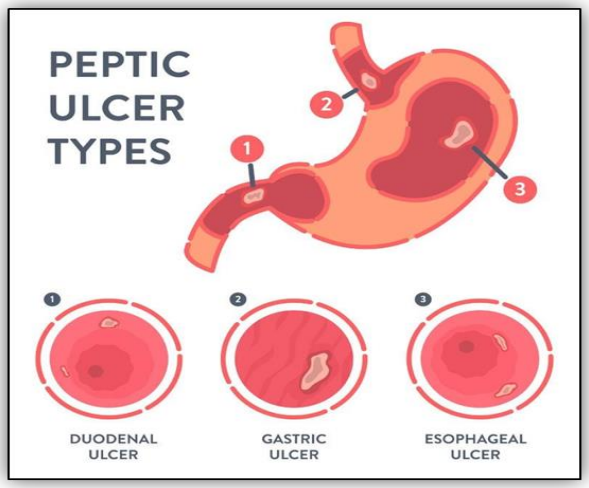

Fig.4: Types of Peptic Ulcer

IV. HISTORY

In the early 1900s, research on peptic ulcer disease (PUD) is often cited as an example of scientific investigation that went in the wrong direction. Scholars like Thagard, Gilbert, Solomon, Zollman, Wray, Miller, Šešelja, Straßer, and O'Connor have written about this case. From the 1800s onward, there were two main theories about what caused PUD. One theory, called the acidity hypothesis, suggested that too much stomach acid was the main cause. The other theory, the bacterial hypothesis, claimed that bacteria were primarily responsible. In the middle of the 20th century, the bacterial theory was mostly ignored, and scientists continued to focus on the acidity theory. This approach involved studying different ways to balance stomach chemicals, such as using antacids or surgery, instead of looking for bacteria or trying to get rid of them. For about thirty years, research on PUD was based on the less accurate of the two theories. It wasn't until the 1980s that Robin Warren and Barry Marshall discovered Helicobacter pylori, a type of bacteria that was actually the main cause of PUD. Their discovery, which earned them a

Nobel Prize in Physiology or Medicine, led to a renewed interest in the bacterial theory of PUD.

According to the received view on the history of this episode (originating primar- ily in Warren and Marshall, 1983; Marshall, 2002),[1] the main reason for the aban- donment of the bacterial hypothesis was a large-scale study by a prominent gastro- enterologist, Palmer (1954). Palmer examined 1,180 subjects, fifth of whom were healthy individuals, while the remainder of the group were patients with gastrointes- tinal complaints. The study showed no presence of bacteria in the gastric mucosa of the subjects. As scientists Fukuda et al. (2002), reflecting on the history of this case, write:

The hypothesis that PUD was caused by bacteria in the mucosa of the human stomach was rejected in 1954 by the major authority in American gastroenterology, Palmer, 1954, despite consistent information in the preceding 50 years about bacteria that adhered to the gastric mucosa. His words ensured that the development of bacteriology in gastroenterology would be closed. The study established the dogma that bacteria could not live in the human stomach, and as a result, investigation of gastric bacteria attracted little attention for the next 20 years.

Today, we understand that Palmer's study was very misleading because it used a method that wasn't right for finding spiral bacteria (see Fukuda et al., 2002). Because of this, this event in history is now one of the main examples of a scientific investigation where everyone followed the rules—each scientist had good reasons to stop believing in the bacterial theory—but the whole scientific community ended up going in the wrong direction for a long time (Zollman, 2010; Kummerfeld and Zollman, 2016; O'Connor, 2020). So, the PUD case shows how individual rational decisions don't always lead to the best outcome for the whole group. It also shows how spreading wrong findings can lead the entire scientific community off track.

However, looking at the events this way still leaves some questions unanswered. For example, if Palmer's study was so influential, why didn't anyone in the scientific community point out possible problems with it? After all, Freedberg and Barron had already warned about the method used in Palmer's study back in 1940, and Palmer even cited them in his own paper. This is especially strange if we believe Šešelja and Straßer (2014), who say that bacterial research had promising directions in the 1950s, even though it was mostly abandoned after Palmer's work. Also, there's no solid historical evidence to back up the claim that Palmer's study had a big impact. Plus, the idea that the bacterial theory was widely popular before Palmer's paper has never been properly supported by evidence

In this paper, we want to add to this discussion by looking closely at the common belief that the bacterial hypothesis was given up mainly because of Palmer's study. If this common story is true, then the reason the bacterial hypothesis was abandoned is because Palmer's work influenced the medical community a lot. As Zollman (2010) says: "It was because many people accepted Palmer's findings that the variety of scientific work that existed a few years before was abandoned too early."

However, claims about Palmer's influence are often made without considering the research situation before 1954. Our goal is to check whether the bacterial theory of peptic ulcer disease had already been mostly forgotten before Palmer published his work. To understand this better, we used digital text analysis on English-language literature about peptic ulcers from the ten years before Palmer's study. This is especially interesting because, if true, it would have major effects on how this episode is discussed in philosophy. First of all, Palmer's work would not be as important as it is often said to be: even if his ideas made some scientists stop looking into the bacterial theory, they would just be the final blow.

Second, trying to explain the PUD episode by saying that Palmer's results spread widely would not work. Because of this, the case doesn't show well how a lot of communication between scientists can lead to inefficient research. This idea comes from Zollman's work on how knowledge spreads through networks, but it's now used a lot in philosophy, not just in studies about formal models of science (for example, Wray, 2010; Douven& Kelp, 2011; Nunn, 2012; Vickers, 2020; Peters, 2020; Killin& Pain, 2021). Since it's hard to find real examples of bad things happening because of too much communication, the PUD case has been especially useful in showing this kind of effect. So, if our idea is proven right, philosophers will need to look for other historical examples to explain this kind of social and knowledge-related process. But as we'll argue in Section 4, this might not be easy.

So, if it turns out that there wasn't much research on the bacterial theory back in the 1940s, then the question of why the bacterial hypothesis was abandoned in the 1950s wouldn't seem strange anymore. Instead, we would have to think about other questions that come from different possible stories about this case. One possibility is that the bacterial hypothesis was popular at the end of the 1800s, but then slowly lost support over time. Another idea is that its decline wasn't slow, but happened suddenly at some point between the late 1800s and the early 1940s. There's also the chance

that the bacterial theory wasn't popular at all during the first half of the 1900s and was always a small, unusual area of study. Until now, discussions about this case have mainly used qualitative analysis, but new quantitative methods, using digital tools, could help find better evidence and figure out which of these stories is most supported. As we will explain, these new possibilities bring up specific philosophical questions that haven't been looked at before.

Here is how we will proceed. In Section 2, we provide a historical overview of this case study, focusing on the question of which factors may have led to the abandonment of the bacterial research program. In Section 3, we introduce the method of digital textual analysis, which we use to look into the historical claim that the bacterial research program was largely abandoned before Palmer's study was published. In Section 4, we discuss the implications of our findings for philosophical discussions of this episode, especially in the area of network epistemology.[5]

V. PATHOPHYSIOLOGY

Peptic ulcers, which were once thought to be purely a physiological problem, are now recognized to have major psychological components. Peptic ulcer formation and exacerbation have been found to be significantly influenced by stress, anxiety and depression . NSAIDs or H. pylori are the main causes of PUD. Stress is also a physiological risk factor for peptic ulcers, according to several studies, such as the Danish study, H. pylori contribute to the development of different disorders in the gastroduodenal mucosa is not fully understood.

H. pylori Infection, which can result in either hyperchlorhydria or hypochlorhydria, can identify the kind of peptic ulcer . Under normal circumstances, the mucus-bicarbonate Barrier, a neutral PH, and ongoing epithelial cell renewal preserve the integrity of the duodenal and gastric mucosa. PGE2 promotes a crucial function in mucosa maintenance by stimulating cell proliferation, mucus, and H3CO3 secretion. A unique and important component at stomach equilibrium, there is adequate blood flow . An imbalance between protective and damaging elements of the stomach mucosa is the mechanism of PUD incidence. Risk factors that increase the possibilities of developing PU.[6][7]

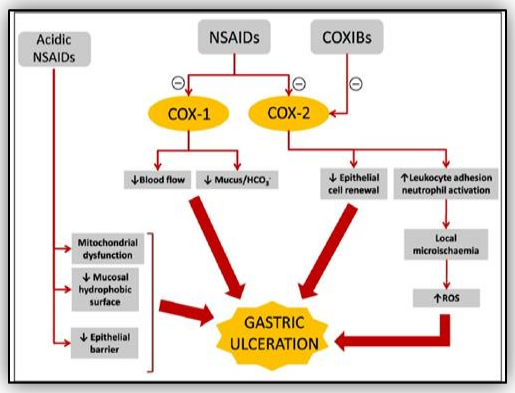

Fig.5: Pathophysiology of Peptic Ulcer

5.1 Role Of H.Pylori Infection

Numerous host and bacterial factors influence the development of ulcers caused by H. pylori. Many ulcers develop at sites of the most severe mucosal inflammation. Reduced acid production can lead to gastric ulcer disease and is typically found in the stomach transitional zone between the corpus and antrum. When acid production is normal to high, the proximal duodenum and distal stomach usually suffer the most severe inflammation. The system uses Amazon Web Services (AWS), including Lambda, S3, IAM, CloudWatch, and Docker, to compress images right after they are uploaded, determining a person's clinical outcome. Duodenal ulceration is predisposed by increased stimulated acid production, while pangastritis or corpus gastritis is predisposed by decreased acid formation. These disorders increase the risk of developing cancer of the abdomen, atrophic gastritis, and ulcers. The distribution of gastritis is thought to be influenced by environmental factors, including host inheritance, bacterial pathogenicity factors, and the age at which the infection begins to manifest.[8]

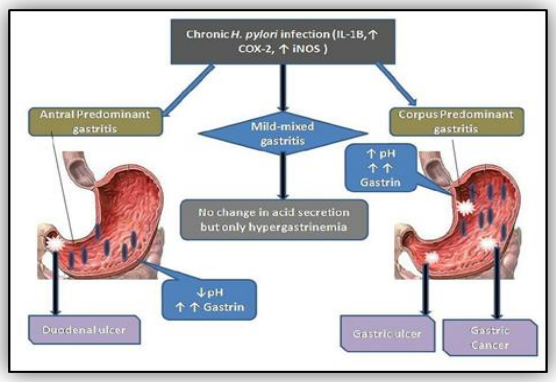

Fig.6: Showing how H. pylori bacteria take over different parts of the stomach and cause various stomach and small intestine diseases.

VI. ETIOLOGY

In this section, we give a historical look at English-language research on PUD from the first half of the twentieth century. We focus on the question of what other factors, apart from Palmer's paper, might have indirectly led to the rejection of the bacterial theory of PUD. To do this, we mainly use secondary sources from the history of medicine and first-hand accounts from gastroenterologists who worked on PUD during that time. These sources include studies from Britain (Christie and Tansey, 2002) and around the world (Warren, 2005). They provide insight into personal reasons that led researchers to move away from the correct theory.

Before discussing the factors that contributed to the decline of bacterial research on PUD, or the 'germ theory of PUD,' we briefly summarize the various theories about the causes of this disease that were studied in the first half of the twentieth century.[9]

6.1 H.Pylori

Peptic ulcers develop when stomach acid injures the lining of the stomach or the first part of the small intestine. This leads to an open sore that can cause discomfort, burning pain, and sometimes bleeding. Under normal conditions, a layer of mucus shields these areas from acid. When acid levels rise or the lining becomes weak, this protection breaks down and an ulcer can form.

Helicobacter pylori is a major contributor to several stomach disorders, including gastritis, stomach ulcers, duodenal ulcers, and even gastric cancer. Treating this infection is essential for proper ulcer healing, and its discovery has greatly changed how doctors approach peptic ulcer disease.

H. pylori is considered the leading cause of stomach ulcers. Once it settles in the stomach lining, it releases harmful substances, weakens the natural protective mechanisms, and exposes the tissue to acid damage. The bacteria hide beneath or within the mucus layer, attach to stomach cells, and produce enzymes that injure the lining. The immune response triggered by the infection adds to the irritation and tissue damage.

These bacteria produce enzymes like urease, catalase, phospholipase, and various proteolytic enzymes that break down the mucus barrier and make the lining more vulnerable. Around half of the global population carries H. pylori, usually in the stomach or upper small intestine, though most infected individuals never develop symptoms. Infection commonly begins in childhood and is more frequent in areas with poor sanitation. In the United States, about five percent of children under ten have this infection. Crowded living conditions and poor hygiene increase the risk, and the bacteria spread through close contact or contaminated food and water.[10]

6.2 NSAIDS

Nonsteroidal anti-inflammatory drugs, or NSAIDs, are the main cause of the first stomach ulcer and can make it bleed or rupture. Using NSAIDs can quickly irritate the stomach lining, which leads to ulcers. A group of chemicals called prostaglandins may help protect the stomach lining. NSAIDs can affect these chemicals. Long-term use of NSAIDs, like diclofenac, naproxen, and ibuprofen, can harm the stomach. These drugs reduce the stomach's protective layer by stopping the production of prostaglandins, making the stomach more likely to get ulcers. While NSAID side effects only affect a small number of people, their widespread use has led to more people having serious stomach problems.

NSAIDs can harm the stomach in several ways, such as irritating the lining, damaging the mucosa, reducing prostaglandin production, lowering blood flow to the stomach lining, and stopping the repair of small damages. Prostaglandins help keep the stomach lining stable. When the activity of cyclooxygenase (COX 1 and COX 2), especially COX 2, goes down, it can cause ulcers. Neutrophils can harm the stomach lining by releasing harmful chemicals and reducing blood flow. The stomach lining is also protected by hydrogen sulphide (H2S) and nitric oxide (NO), which NSAIDs can block. Because of these factors, people who take NSAIDs are at higher risk of ulcer complications. Studies show that people with ulcers have about 12 times more risk of getting another ulcer compared to people who have never had one and haven't used NSAIDs.

Many studies have found that genes can play a role in developing stomach ulcers. Identical twins are more likely to get peptic ulcers than zygotic twins. People who have relatives with ulcers are also more likely to get them. Both duodenal and gastric ulcers have different genetic links: relatives of people with duodenal ulcers but not gastric ones are three times more likely to get duodenal ulcers, while relatives of those with gastric ulcers but not duodenal ones are three times more likely to get gastric ulcers. It is still unknown which specific genes are responsible for this increased risk.

6.3 Life Style Factor

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.

6.4 Stress and diet

Anxiety can contribute to problems triggered by stomach acid, and these issues may appear during periods of intense stress, especially in people dealing with serious medical conditions or critical care treatments. Coffee and caffeine are often blamed for worsening symptoms, but their effects may not be as harmful as commonly assumed. During acute stress, anxiety, heart rate, and blood pressure rise. In people with duodenal ulcers, the main change is a sharp increase in stomach acid production.

There is no specific "ulcer personality." People with ulcers generally share the same psychological traits as the rest of the population, though they may react more strongly to stress. Emotional stress is sometimes believed to aggravate peptic ulcers. Severe burns, major illnesses, nervous system injuries, and surgeries are all stressful events that can increase the risk of ulcer formation. Conditions such as sepsis and low blood pressure also raise the chance of developing ulcers. Many severe injuries, widespread infections, and respiratory problems are linked with high stress levels.

Stress ulcers and bleeding in the upper digestive tract are becoming more common among critically ill patients in intensive care units. This increased risk is related to serious illness and changes in stomach acid levels. Studying how psychological factors influence peptic ulcers is challenging because stress is hard to measure and ulcers have multiple causes. To understand the role of psychological stress, it must be connected to established ulcer pathways, like NSAID use or H. pylori infection. This is why the role of psychological factors in ulcer development is still debated.

Patients in critical care are particularly vulnerable to stress-related ulcers. These ulcers arise from physical stress on the body rather than emotional stress, and they can cause bleeding in the upper digestive tract. They occur when the stomach lining is weakened by shock, infection, major injuries, or long-term illness.

6.5 Zollinger-Ellison syndrome

Zollinger-Ellison syndrome is a uncommon condition that might lead to peptic ulcer disease. This condition makes the cells in the digestive tract that make acid grow into tumors. These tumors can be either cancerous or not. Too much acid from these cells can damage the stomach lining.[11]

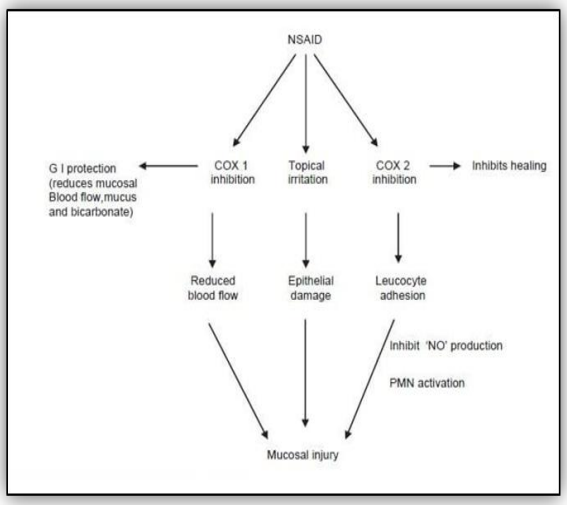

Fig.7: Etiology of Peptic Ulcer

VII. SIGN AND SYPTOMS

Peptic ulcer disease (PUD) symptoms commonly include a gnawing or burning pain in the upper abdomen, along with bloating and nausea. However, many people with peptic ulcers experience no symptoms at all until a complication arises.

The signs and symptoms can also vary depending on whether the ulcer is in the stomach (gastric ulcer) or the upper part of the small intestine (duodenal ulcer).

Common signs and symptoms

- Abdominal pain: A dull or burning pain between the breastbone and the navel is the most frequent symptom. The timing of the pain can offer clues:
  - Gastric ulcer: Pain often gets worse shortly after eating, as the stomach produces more acid.
  - Duodenal ulcer: Pain typically improves with eating but returns a few hours later when the stomach is empty. It may also wake you up at night.
- Bloating and feeling full: Many people experience a sense of fullness or bloating, especially after meals.

- Nausea or vomiting: These are common symptoms that occur because the ulcer can disrupt normal digestion.
- Burping: Frequent burping is also a possible sign.
- Weight changes: Loss of appetite can lead to unexplained weight loss. In contrast, people with duodenal ulcers may experience weight gain as they eat more to relieve the pain.

Serious and emergency symptoms

Untreated peptic ulcers can lead to serious complications, such as bleeding, perforation, or obstruction. You should seek immediate medical attention if you experience:

- Black or tarry stools (melena): A sign of bleeding in the digestive tract.
- Vomiting blood: The vomit may be red or look like "coffee grounds".
- Sudden, sharp abdominal pain: This could indicate a perforation or hole in the stomach or intestine.
- Feeling dizzy or faint: A symptom of significant blood loss.
- Rapid pulse.[12][13]

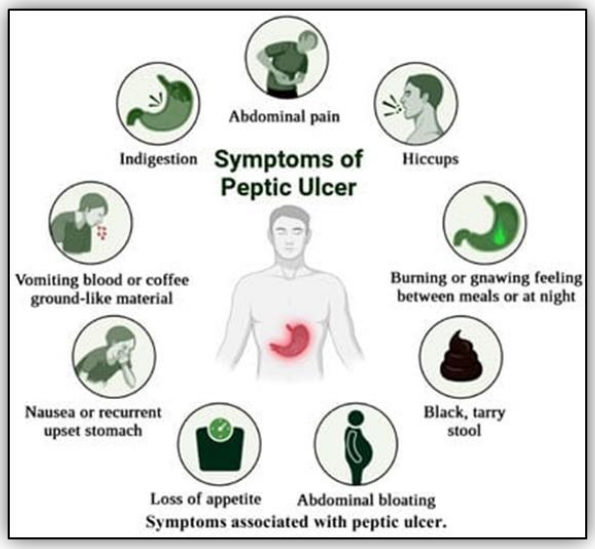

Fig.8: Symptoms of Peptic Ulcer

VIII. RISK FACTORS

I. Helicobacter pylori Infection

Infection with H. pylori is a primary cause of gastric and duodenal ulcers. Research, including studies from India, indicates a strong correlation between this bacterial presence and the onset of ulcers. The magnitude of this danger varies globally; for example, one investigation documented a 20-fold greater chance in the Netherlands, compared to an 8-fold elevation in Japan.

II. Usage of Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

Extended or high-dose consumption of NSAIDs can damage the protective layer of the stomach and significantly heighten the probability of developing an ulcer. This danger is further compounded when multiple NSAIDs are consumed or when they are taken alongside other medications that irritate the stomach. Furthermore, evidence shows that individuals over the age of 55 are more susceptible to experiencing NSAID-related ulcers.

III. Tobacco Use

Smoking not only increases a person's chances of developing a peptic ulcer but also impedes the recovery process for existing ulcers. For those already infected with H. pylori, smoking acts as an additional risk multiplier, making ulcer formation more probable.

IV. Alcohol Consumption

The ingestion of alcohol can aggravate the stomach lining and stimulate greater acid secretion, thus elevating ulcer risk. Large-scale population studies have also established a clear connection between alcohol use and a heightened risk of ulcer-related bleeding.

V. Advanced Age

Older age is a critical predisposing factor, particularly for those using NSAIDs. Individuals over 60 face an increased likelihood of developing these lesions. Furthermore, studies suggest that natural, age-related alterations to the stomach's mucosal barrier may make seniors more susceptible.

VI. Prior Ulcer History

Individuals who have previously suffered from a peptic ulcer have a greater propensity for recurrence. A history of ulcers also increases the vulnerability to serious complications, such as hemorrhage (bleeding).

VII. Heredity and Genetic Predisposition

Having an immediate family member with peptic ulcer disease boosts one's own chances of being affected. Specific genetic characteristics, such as possessing Blood

Type O or having elevated pepsinogen levels, may also contribute to a higher overall risk.

VIII. Societal and Environmental Influences

In certain regions, a lower socioeconomic position is associated with higher rates of H. pylori infection, likely stemming from poor hygiene and crowded living conditions. Environmental factors, such as frequently dining out in unsanitary venues or consuming unfiltered water, have also been correlated with an elevated infection risk.

IX. Severe Physiological Stress

Intense physical stress resulting from serious illnesses, surgical procedures, severe burns, generalized infection (sepsis), or head trauma can lead to the formation of stress ulcers, especially in critically ill patients. These ulcers are frequently observed in individuals requiring care in intensive care units (ICUs).[14][15][16]

IX. DIAGNOSIS

Until the early 1900s, doctors mostly diagnosed peptic ulcers by looking at symptoms and how patients felt. In the 1950s, the use of flexible endoscopies changed how doctors could see ulcers up close. To properly understand a patient's condition, a complete medical history and a thorough physical exam are needed. This helps list all the symptoms and signs related to peptic ulcer disease. It's also important to note the patient's past medical history, including how long they've been drinking alcohol, using NSAIDs, smoking, and any previous episodes of ulcers. When diagnosing peptic ulcer disease, there are two main things to consider. The first is to check if the symptoms are not caused by something else like functional dyspepsia. The second step is to find out exactly where the ulcer is located.

9.1 Radiology

Even though endoscopies have mostly taken over from barium studies in routine diagnosis, barium tests can still be useful for a few patients who don't want an endoscopy or when endoscopy isn't possible because of a narrow esophagus. The skill of the radiologist, the method used, how deep the ulcer is, the size of the lesion (if it's smaller than 0.5 cm, it might be hard to see), and the overall technique all affect how well barium tests can detect and correctly identify issues. Signs that suggest a condition is not cancerous include smooth edges, even folds in the lining, a clear and shiny band or collar, and the opposite wall pulling in around the ulcer. On the other hand, big ulcers, uneven folds, lack of contrast, or irregular filling patterns may point to cancer.

9.2 Blood Test

A blood test can help identify issues related to peptic ulcers, including complications or an H. pylori infection. During the test, a healthcare professional draws a small blood sample and sends it to a laboratory for evaluation.

9.3Urea Breath Test

- The "Meal" with a Tracer: You'll be asked to swallow a small amount of something—like a pill, a liquid, or a tiny bit of pudding—that has a chemical called urea in it. This urea is tagged with a special, safe kind of carbon that helps track it.
- The Bacteria's Job: If there are H. pylori bacteria in your stomach, they act like tiny chemists.They make an enzyme called urease that can break down the urea very quickly.
- The Tell-Tale Gas: When the bacteria break down the urea, one of the things they make is carbon dioxide (CO_2).Because this CO_2 has the special carbon tag, it can be easily found.
- The Exhale: The gas made in your stomach goes into your bloodstream and moves up to your lungs.Then you breathe out into a bag or container to collect the gas.
- The Result: The collected gas is tested.If the special tagged carbon dioxide is found, it means that the H. pylori bacteria are alive and in your stomach.

9.4Stool Test

To check for an H. pylori infection, doctors might use a stool test. Your doctor will give you a container to collect and store the stool sample. You will also get instructions on how to send the kit for testing or where to pick it up.[17][18][19][20]

X. TREATMENT AND MANAGEMENT

Today's treatment for peptic ulcers includes medicines like PPIs and H2 receptor blockers, and surgery for more serious cases. Doctors are also looking into new options such as new drugs, probiotics, and less invasive procedures. When Helicobacter pylori is found, it is treated with antibiotics. Acid-suppressing drugs like PPIs are used to reduce stomach acid, and early endoscopy helps manage any complications. Patients at higher risk may take PPIs to prevent ulcers from coming back. Current treatment plans involve using PPIs and H2 receptor blockers, and surgery when

needed. Research is ongoing into new drugs, probiotics, and minimally invasive techniques to improve care. Modern treatment focuses on reducing stomach acid with H2-receptor antagonists and PPIs, and treating H. pylori with antibiotics, which has helped lower complications and the need for surgery.[21]

10.1 Drugs(Antibiotics,Acid Suppressants)

Peptic ulcer drugs include H2 blockers, anticholinergics, antacids, prostaglandin analogs, ulcer protectants, and PPIs. Recently, potassium-competitive acid blockers (P-CABs) have been found to be better than PPIs for treating these conditions. The main drugs used for peptic ulcer disease are proton pump inhibitors, along with amoxicillin and clarithromycin, which are used together in triple therapy to get rid of H. pylori. In cases where treatment doesn't work, anti-secretory drugs and selective COX-2 inhibitors are also used. Proton pump inhibitors are important for treating peptic ulcers, helping to relieve symptoms and allow healing within 4 to 6 weeks. Also, eradicating H. pylori is usually done using a triple therapy that combines a proton pump inhibitor with two antibiotics for 7 to 14 days.[22]

10.2 Lifestyle Modification

Lifestyle changes that help with peptic ulcers include eating healthy foods, staying active, managing stress well, not smoking, drinking alcohol only in small amounts, getting enough sleep, and using less pain medicine and steroids. These changes are important for helping ulcers heal and stop them from coming back. Along with these lifestyle changes, medicine like proton pump inhibitors and antibiotics are also used to treat and manage peptic ulcers.[23]

10.3 Surgical Management

Surgical treatment for peptic ulcers is important, especially when the ulcer has caused a hole in the stomach or intestine. Even though some doctors prefer to use non-surgical treatments, it's important to understand how getting rid of the bacteria Helicobacter pylori affects the results of surgery. This knowledge helps in giving the best care and lowers the chances of problems and the ulcer coming back. Today, surgery for peptic ulcers is mostly done when there are serious issues like heavy bleeding, a hole in the stomach or intestine, or a blockage in the digestive system. Most of the time, doctors avoid doing surgery unless it's really needed and instead use endoscopy, which is less invasive. There's no clear advantage to using laparoscopic surgery over traditional open surgery for these conditions.[23]

10.4 Endoscopic Therapy

Endoscopic treatment for peptic ulcer disease uses methods like injections, heat, clips, special sprays, and stitching to stop bleeding. This method works best for certain types of ulcers called Forrest 1a, 1b, and 2a. However, ulcers labeled as Forrest 2b are usually treated with proton-pump inhibitors alone. Peptic ulcer disease is still one of the main causes of bleeding in the upper digestive tract, which is a serious emergency that can cause serious health issues. Even though medicines like proton-pump inhibitors and treatments to get rid of the Helicobacter pylori bacteria have made long-term care for peptic ulcers much better, endoscopic techniques are still the main way to stop bleeding from ulcers, especially in patients who show signs of being at high risk, like active bleeding or visible blood vessels.[24][25]

XI. PREVENTION

Some important steps are suggested by experts to lower the chance of getting an ulcer. Some of these steps are meant to stop the disease from happening, like avoiding smoking, not drinking alcohol, and cutting down on aspirin and other NSAIDs. In many cases, taking NSAIDs can lead to an ulcer without any warning. This risk is especially high for older people and those who have had peptic ulcer disease before. Although peptic ulcers are uncommon in children and young adults, especially in developed countries, they can still be at risk due to NSAIDs or ulcers caused by Helicobacter pylori. Some cases have been reported even in medical papers from developed countries. In fact, serious stomach problems in infants and children are not very common. Therefore, diagnosing the possible causes needs careful attention from specialists and various tests. However, there is not much information in the literature about peptic ulcers in children, especially in western countries, and such reports are rare and go back to many years ago. Changing diet and avoiding bad eating habits, like fast food and salty or fried snacks, are best ways to reduce the risk of peptic ulcers in modern societies. Considering the fast-paced world and busy lifestyles, the risk of peptic ulcers can be greatly reduced by lowering oxidative stress, especially in older people. A more relaxed lifestyle and using natural antioxidants can help prevent ulcers. For example, it has been found that oral use of ethanol extracts from the bark of Combretumleprosum can have a protective effect on the stomach and help prevent ulcers by reducing acid production and increasing the body's defenses. Turmeric, which is widely used in Asian foods, contains curcumin, a substance that strongly inhibits the growth of H. pylori. This effect has been tested both in the lab and in living organisms. Because of this, using turmeric in cooked food can help prevent H. pylori-related peptic ulcers.[26]

To reduce your risk of developing a peptic ulcer:

- Avoid tobacco products
- Avoid alcohol
- Use caution with aspirin and/or NSAIDs
- Don't ignore your ulcer symptoms
- Protect yourself from infections by washing hands regularly and consuming foods that have been cooked thoroughly.[27]

XII. PROGNOSIS AND COMPLICATION

Prognosis

Most peptic ulcers heal within a few weeks. Most people will only need medication for about two months. Medications are very effective in treating peptic ulcers. People with chronic conditions, like Zollinger-Ellison syndrome, may need to take them for life.Rarely, some people have persistent stomach ulcers that don't respond to treatment or that keep coming back after treatment. They can cause chronic pain, excessive scarring and other complications. These cases might require surgery to:

- Remove the scar tissue or open up the outlet (Pyloroplasty)
- Seve the nerve that triggers the stomach acid (vagotomy)

Unfortunately, recurrence is common with rates exceeding 60% in most series. NSAID-induced gastric perforation occurs at a rate of 0.3% per patient per year. However, unlike in the past, mortality rates for peptic ulcer disease have decreased significantly.

Complications

PUD can cause bleeding, perforation, penetration, obstruction of the stomach outlet, persistent symptoms, and gastric cancer (adenocarcinoma and MALT lymphoma). The most frequent consequence, bleeding, affects 15-20% of individuals. A significant amount (about 40–60%) of acute upper GI bleeding is caused by PUD. Prolonged ulcerative inflammation and scarring can restrict the duodenum, potentially obstructing the gastric exit.Moreover, it triggers an overactive immunological response and chronic inflammation, both of which lead to carcinogenesis. Less than 2% of people worldwide ever have stomach cancer, despite the fact that 50% of people have H. pylori infection.[28]

XIII. CONCLUSION

Peptic ulcer disease represents a condition that has been recognized for centuries, and its understanding has steadily improved through advances in science and clinical practice. Early historical descriptions were based mainly on symptoms, but modern research has clarified how ulcers form when the natural balance between gastric acid and the protective mucosal barrier breaks down. This imbalance is usually driven by infection with H. pylori or prolonged use of NSAIDs, though lifestyle habits such as smoking, alcohol use and stress can add to the risk. These factors damage the lining of the stomach or duodenum, which leads to inflammation and the development of open sores.The disease usually presents with burning or gnawing abdominal pain, discomfort related to meals, nausea, bloating and, in more severe cases, bleeding or weight loss. These signs and symptoms highlight the importance of timely diagnosis. Endoscopy remains the most reliable method to visualize the ulcer directly, while laboratory tests help identify H. pylori and assess complications.

Treatment has evolved to become highly effective. Acid-reducing medicines, antibiotics to eradicate H. pylori and protective agents that support healing form the core of therapy. When these are used correctly, the majority of ulcers heal well and recurrence becomes less common. Prevention also plays a major role. Avoiding unnecessary NSAID use, quitting smoking, limiting alcohol and managing stress help protect the gastric mucosa. Identifying at-risk individuals and treating H. pylori early can further reduce disease burden. Overall, the prognosis for peptic ulcer disease is positive when it is diagnosed early and treated properly. Most people recover fully, and complications such as bleeding, perforation or obstruction are far less frequent than in the past. Continued attention to lifestyle habits, appropriate medication use and regular follow-up help maintain long-term health and prevent relapse.

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