



Peptic Ulcer Disease and Its Management

*Keerti Phalswal, Ruchika Singh and Parul Saini

Dept. of Medical Surgical Nursing, Faculty of Nursing, SGT University, Gurugram, Haryana, India

ABSTRACT

Globally, peptic ulcer disease seems as a major cause of morbidity and mortality. According to WHO it has been estimated that up to 10% of people worldwide are affected by the chronic condition known as peptic ulcer. There are about two thirds of individuals who are suffering from peptic ulcer disease. Peptic ulcer disease shows various symptoms which include epigastric discomfort, which can be accompanied by dyspepsia, bloating, abdominal fullness, nausea, or early satiety. The development of disease is influenced by the pH of gastric juice and a decline in mucosal defenses. *Helicobacter pylori* (*H. pylori*) infection and non-steroidal anti-inflammatory drugs (NSAIDs) are the main factors which damage the mucosal resistance. Proton pump inhibitors (PPIs) and histamine-2 (H₂) receptor antagonists, two common therapies for peptic ulcers, have been shown to cause side effects, relapses, and a variety of pharmacological interactions.

KEY WORDS: PUD- Peptic ulcer disease, *H. pylori*- *Helicobacter pylori* infection, PPI- Proton pump inhibitor, EGD- Esophagogastroduodenoscopy, COX-1- cyclooxygenase 1.

Received 12.10.2022

Revised 23.10.2022

Accepted 21.11.2022

INTRODUCTION

Peptic ulcer disease is characterized by a breakdown of the inner lining of the Gastro-intestinal tract caused either by gastric acid secretion or the production of pepsin in excess. It penetrates the muscularis propria layer of stomach. Stomach and proximal duodenum are the most common areas where PUD is present. It can also affect the lower part of esophagus, the distal duodenum, or the jejunum. In gastric ulcer, epigastric pain usually occurs within 15-30 minutes of eating; however, pain in duodenal ulcer usually occurs 2-3 hours after eating. It was once widely believed that stress can also lead to peptic ulcer disease as it increases the production of gastric acid. *H. pylori* testing is done in most of the patients with PUD. Endoscopy is also recommended for the confirmation of diagnosis, mostly for those patients who are having serious symptoms. Patients are usually managed using triple-drug therapy based on a proton pump inhibitor (PPI). PUD includes:

- Gastric ulcers that occur anywhere inside the stomach
- Duodenal ulcers are the ulcers which occur inside the upper portion of small intestine (duodenum)

Until late 1970s, Antacids and anti-cholinergic medications were used to treat PUD in the United States, and surgery was commonly required to manage ulcer disease. Histamine-2 receptor antagonists, were first made available in 1976. *Helicobacter pylori*, formerly known as *Campylobacter pyloridis*, had been discovered in gastric biopsies by Dr. J. Robin Warren, and Dr. Barry Marshall had cultivated *H. pylori* from patients with ulcers and gastritis in 1982.¹ The prevalence of peptic ulcer disease had decreased by the 20th century's end. It is believed that improving hygiene standards was the reason for the decline in *H. pylori* infection rates in the general population [1].

RISK FACTORS

Smoking- Smoking can cause PUD in individuals with *H. pylori* infection [2].

Consumption of Alcohol-Alcohol consumption is harmful and it irritate and erodes the inner lining of stomach and also increases the acid production.

Stress- It is considered as one of the factors that cause PUD.

Spicy Foods- Consumption of spicy food on regular basis

Epidemiology and Etiological Factor

Globally, PUD is major causes that cause illness and death. Consequences can include everything from gastrointestinal bleeding and abdominal pain to perforation and occlusion of the gastric outlet.

There are 8.4% more Americans who have peptic ulcer disease than there were in 2000.

Male sex, smoking, and chronic medical disorders have all been proven to be connected with a higher risk of PUD. Additionally, it was discovered that peptic ulcer disease increases with age [3]. The rare causes of PUD are Zollinger-Ellison syndrome, malignancy, stress, viral infection, vascular insufficiency, radiation therapy, Crohn disease and chemotherapy [4].

H. pylori Associated PUD

It is the first most common cause of PUD. This bacterium causes 90 percent of duodenal ulcers and 70- 90 percent of gastric ulcers. It is common in people with low socioeconomic status and is mostly acquired as a child. The organism allows it to stick and causes inflammation of the inner lining of stomach. This leads to gastric ulceration [5].

NSAIDs Associated PUD

The second most common cause of PUD is the use of NSAIDs. Prostaglandin secretion normally protects the gastric mucosa. By inhibiting the COX-1 enzyme, NSAIDs inhibit prostaglandin synthesis, resulting in decreased gastric mucus and bicarbonate production and decreased the blood flow of mucosa.

Clinical Manifestations

The most common presenting symptom in symptomatic patients is pain in epigastric region, dyspepsia and bloating, abdominal fullness, nausea and heart burn. Symptoms may be intermittent in many patients.

DIAGNOSIS

It can be diagnosed easily by the presence of symptoms. If ulcers develop without taking NSAIDs, it is most likely caused by an H. pylori infection. One of these tests will be required to confirm the diagnosis.

Endoscopy-If severe symptoms are present then upper endoscopy is required to see if an ulcer is present or not. An endoscope is used to look for abnormalities in the throat and stomach during this procedure.⁶

Esophago Gastroduod Enoscopy (EGD) -EGD is the most accurate and gold standard test used for the diagnosis of gastric and duodenal ulcers, with sensitivity and specificity up to 90%. Patients over the age of 50 who have recently developed symptoms of dyspepsia should undergo an EGD. Anyone, regardless of age, who has alarm symptoms should have an EGD [6].

Barium Swallow- If EGD is contraindicated only then barium swallow is indicated.

Urea Breath Test-This test is highly sensitive and specific. Four to six weeks after discontinuing the treatment it can be used to confirm the diagnosis.

H. Pylori Antibody Blood Test-If there was infection in past then these antibodies will also be present and can be detected 12-18 months after the treatment [7].

Imaging Test-CT scans and X-Rays detect ulcers less frequently. Ulcers are visible to imaging machines when a liquid is consumed which will coat the digestive tract.

MANAGEMENT

Non-pharmacological Management

- Reduce stress
- Cessation of cigarette smoking
- Stop the use of NSAIDs
- Avoid the consumption of spicy foods, caffeine, alcohol
- Drink plenty of water on daily basis
- Avoid fasting and maintain optimum gap between meals

Pharmacological Management

Proton Pump Inhibitors (PPI) -It reduce the secretion of gastric acid, which heals the ulcers. PPIs include pantoprazole, omeprazole etc [8].

Histamine Receptor Blockers (H₂ blockers) -These drugs are used to reduce the production of acid and it includes ranitidine, cimetidine etc.

Antibiotics- It kills the growth of bacteria.

Protective Medications- These medications prevent further damage of mucosal lining by covering the ulcers with a protective layer [9].

Management of H. Pylori Positive PUD

Triple therapy	PPI Amoxicillin Clarithromycin	Double dose*/12h 1gm/12h 500mg/12h	14 days
Triple Therapy (Penicillin Allergy)	PPI Metronidazole Clarithromycin	Double dose*/12h 500mg/12h 500mg/12h	10-14 days
Quadruple Therapy (Concomitant Therapy)	PPI Amoxicillin Clarithromycin Metronidazole	Standard dose/12h 1gm/12h 500mg/12h 500mg/12h	10-14 days
Rescue Treatment (After failure of Triple or Concomitant Therapies)	PPI Amoxicillin Clarithromycin Levofloxacin	Standard dose/12h 1gm/12h 500mg/12-24h	14 days
Classic Quadruple therapy	PPI Bismuth Tetracycline Metronidazole	Standard dose/12h 120mg/6h 500mg/6h 500mg/8h	10-14 days

Refractory Disease and Surgical Treatment

If an individual is not responding to medical treatment, not taking the medication regularly, or have high risk of complications, surgical treatment is indicated. An ulcer that is larger than 5mm and does not heal even after 12 weeks of PPI therapy is known as refractory peptic ulcer [9]. The commonest causes are chronic H.pylori infection, significant comorbidities continuous use of NSAIDs that interfere with healing of ulcers, such as gastrinoma [9]. If the ulcer are still present, the surgery is indicated. Partial gastrectomy or Vagotomy are surgical options [10].

COMPLICATION

Internal Bleeding- Bleeding can be slow and cause anemia, or it can be severe and either blood transfusion or hospitalization is required. Massive loss of blood can result in black or bloody stools or vomit [11].

Perforation- Serious infection of abdomen (peritonitis) can occur as peptic ulcers erodes through the mucosal lining of stomach or small intestine [12].

Obstruction- Peptic ulcers can cause the obstruction of the digestive tract, which causes a feeling of fullness quickly, weight loss and vomiting as a result of swelling from inflammation or scarring.

Cancer- Patients are at high risk for gastric cancer due to H. pylori infection [13].

CONCLUSION

Peptic ulcer disease is not a common disorder of childhood. PUD is a common clinical problem. The two most common associated risk factors are NSAIDs and H. pylori infection, its clinical features are nonspecific. Prompt treatment and eradication of H-pylori bacteria results in improvement of symptoms and cure along with complete healing for long term.

REFERENCES

1. Ando T, Tsuzuki T, Mizuno T, Minami M, Ina K, Kusugami K et al. Characteristics of Helicobacter pylori-Induced Gastritis and the Effect of H. Pylori Eradication in Patients With Chronic Idiopathic Thrombocytopenic Purpura. *Helicobacter*. 2004; 9(5):443-452.
2. Zhang B, Li Y, Liu X, Wang P, Yang B, Bian D. Association between vacA genotypes and the risk of duodenal ulcer: a meta-analysis. *Molecular Biology Reports*. 2014; 41(11):7241-7254.
3. Fletcher P, Craig D. The role and treatment of Helicobacter pylori infection in peptic ulcer disease: a review of the relationship between Helicobacter pylori infection and peptic ulcer disease. *Journal of Clinical Pharmacy and Therapeutics*. 1993; 18(5):311-316.
4. Lanás, A., Garcia-Rodriguez, L., Polo-Tomas, M., Ponce, M., Quintero, E., Perez-Aisa, M., Gilbert, J., Bujanda, L., Castro, M., Muñoz, M., Del-Pino, M., Garcia, S. and Calvet, X., 2011. The changing face of hospitalization due to gastrointestinal bleeding and Perforation. *Alimentary Pharmacology & Therapeutics*, 33(5), pp.585-591.

5. Sidahmed H, Hashim N, Abdulla M, Ali H, Mohan S, Abdelwahab S et al. Antisecretory, Gastroprotective, Antioxidant and Anti-Helicobacter Pylori Activity of Zerumbone from Zingiber Zerumbet (L.) Smith. PLOS ONE. 2015; 10(3):e0121060.
6. Naika N, Zasshi G., Peptic ulcer. Peptic ulcer: Advance on diagnosis and treatment. 2. Disease state. 3. Infantile peptic ulcer. 1995; 84(6):885-889.
7. Fallone C, Chiba N, van Zanten S, Fischbach L, Gisbert J, Hunt R et al. The Toronto Consensus for the Treatment of Helicobacter pylori Infection in Adults. Gastroenterology. 2016; 151(1):51-69.e14.
8. Zheng Q, Chen W, Lu H, Sun Q, Xiao S. Comparison of the efficacy of triple versus Quadruple therapy on the eradication of Helicobacter pylori and antibiotic resistance. Journal of Digestive Diseases. 2010; 11(5):313-318.
9. Kuna L, Jakab J, Smolic R, Raguz-Lucic N, Vcev A, Smolic M. Peptic Ulcer Disease: A Brief Review of Conventional Therapy and Herbal Treatment Options. Journal of Clinical Medicine. 2019; 8(2):179.
10. Orel R, Turk H. Re: Might the Use of Acid-Suppressive Medications Predispose to the Development of Eosinophilic Esophagitis. American Journal of Gastroenterology. 2010; 105(2):468.
11. Sun Q, Liang X, Zheng Q, Liu W, Xiao S, Gu W et al. High Efficacy of 14-Day Triple Therapy-Based, Bismuth-Containing Quadruple Therapy for Initial Helicobacter pylori Eradication. Helicobacter. 2010; 15(3):233-238.
12. Ang T, Fock K, Ng T, Teo E, Chua T, Tan Y. Efficacy of quadruple therapy for Helicobacter pylori eradication after failure of proton pump inhibitor-based triple therapy in Singapore. Chinese Journal of Digestive Diseases. 2003; 4(1):35-39.
13. Sonnenberg A. Review article: historic changes of Helicobacter pylori-associated diseases. Alimentary Pharmacology & Therapeutics. 2013; 38(4):329-342.

CITATION OF THIS ARTICLE

Keerti Phalswal, Ruchika Singh and Parul Saini. Peptic Ulcer Disease and Its Management. Bull. Env. Pharmacol. Life Sci., Spl Issue [4]: 2022: 610-613