WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Peptic Ulcer and Its Treatment

Mr. Gulzar Ahmad Najar[1], Ms Tanya Sharma[2]

Student at Mewar University[1]
Assistant Professor at Mewar Universty[2]

ABSTRACT:

The development of open sores on the stomach lining or the proximal portion of the small intestine is a common gastrointestinal ailment known as peptic ulcer disease (PUD). The genesis, pathophysiology, clinical symptoms, diagnostic techniques, and current treatment modalities of peptic ulcers are all thoroughly examined in this review. Recent developments in pharmaceutical treatment are highlighted, as is the significance of lifestyle changes in the management of this illness.

Introduction

Peptic ulcer disease is a major global health concern that can occasionally result in death. Helicobacter pylori infection and nonsteroidal anti-inflammatory drug (NSAID) use are the main reasons. PUD can have a wide range of symptoms, from mild cases to serious side effects like bleeding or perforation. For efficient management, it is essential to comprehend the mechanisms causing ulcer development and the therapeutic approaches that are accessible.

Pathophysiology and Etiology

Infection with Helicobacter pylori:

- By generating urease, the bacterium breaks down the mucosal barrier, raising ammonia levels that counteract stomach acid and harming epithelial cells.
- It sets off an inflammatory reaction that damages and ulcerates the mucosa.

NSAID

NSAIDs decrease the synthesis of prostaglandins, which shield the stomach mucosa, by inhibiting the cyclooxygenase enzymes COX-1 and COX-2. Because of the lower prostaglandin levels, there is less blood flow to the mucosa, less synthesis of mucus and bicarbonate, and more discharge of acid.

Factors:

Ulcer formation may be influenced by genetic susceptibility, smoking, excessive alcohol use, and psychological stress.
Clinical Signs and Indications
As the defining symptom, epigastric pain is frequently characterized as burning or gnawing.

Use of NSAIDs:

By blocking the cyclooxygenase enzymes COX-1 and COX-2, NSAIDs lower the synthesis of prostaglandins, which shield the stomach mucosa. Lower prostaglandin levels cause greater acid output, decreased mucus and bicarbonate synthesis, and decreased mucosal blood flow.

1. **Methods of Diagnosis**

Non-invasive Examinations: H. pylori serology, stool antigen testing, and urea breath testing.

2. **Invasion of Tests:**

biopsies combined with endoscopy for histological analysis and urease tests. Additionally, endoscopy is useful for determining the location, size, and consequences of ulcers.

3. **Imaging Research:**

When endoscopy is not an option, barium meal studies may be utilized.

Methods of Treatment

Pharmacological Treatment: a. H. pylori Elimination: Triple therapy: amoxicillin/metronidazole + clarithromycin + proton pump inhibitor (PPI). PPI + bismuth subsalicylate + tetracycline + metronidazole is known as quadruple treatment. b. Acid Reduction: PPIs are first-line medications, such as omeprazole and lansoprazole. Alternatives include H2-receptor antagonists, such as famotidine and ranitidine. c. Agents that are cytoprotective: Over the ulcer, sucralfate creates a barrier of defense. NSAID-induced ulcers can be effectively treated with misoprostol, however its use is constrained by adverse effects.

Modifications to Lifestyle:

Both drinking and smoking should be stopped. NSAID avoidance or COX-2 selective inhibitor use, if feasible. management of stress and dietary changes to stay away from irritants. Surgery: Only used in cases of refractory ulcers, blockage, or perforation. These procedures include pyloroplasty, antrectomy, and vagotomy.

Conclusion

There are recognized methods for diagnosing and treating peptic ulcer disease, which is still a common ailment. The elimination of H. pylori and the prudent application of acid-suppressive medication have greatly enhanced results. Future studies on the gut microbiota, resistance patterns, and non-invasive diagnostic techniques could improve PUD treatment.

References

1. "Helicobacter pylori infection and gastric disease." Gastroenterology 2017; 152(5): 1215-1230; Malfertheiner, P., et al.
2. F. L. Lanza and colleagues published "Guidelines for prevention of NSAID-related ulcer complications." 2009; 104(3): 728-738; American Journal of Gastroenterology.
3. N. Vakil et al. "Management of Helicobacter pylori infection: the Maastricht V/Florence consensus report." Gut 66(1): 6-30, 2016.
4. W. D. Chey and B. C. Wong, "The management of Helicobacter pylori infection." 2007; 102(8): 1808-1825; American Journal of Gastroenterology.
5. Laine, L., et al. "An approach to adult upper gastrointestinal bleeding." 2012; 366(19): 1791-1801; New England Journal of Medicine.
6. J. Sung et al., "Systematic review: the global incidence and prevalence of peptic ulcer disease." 2009; 29(9): 938-946; Alimentary Pharmacology & Therapeutics.
7. A. C. Ford et al. "Impact of NSAIDs on the gastrointestinal tract: mechanisms and clinical relevance." 2014; 11(6): 347-357; Nature Reviews Gastroenterology & Hepatology.
8. "H. pylori and the risk of gastric cancer." Alimentary Pharmacology & Therapeutics 1999; 13(Suppl 1): 13-18 and Kuipers, E. J.
9. D. Y. Graham and colleagues, "New treatments and novel therapeutic strategies for H. pylori infections." North American Gastroenterology Clinics 2010; 39(4): 803-820.
10. M. Robinson and colleagues, "The role of proton pump inhibitors in the management of PUD." Drugs, 60(Suppl 1), 2000: 1–23.