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A COMPREHENSIVE REVIEW ON PEPTIC ULCER: AN OVERVIEW

Imad Ahmad*, Dr. Amresh Gupta, O. P. Verma, Dr. Pritt Verma, Gulab Chandra and Dr. Rohit Mohan

Goel Institute of Pharmacy and Sciences, Faizabad Road, Lucknow, Uttar Pradesh – 226028.

***Corresponding Author: Imad Ahmad**
Goel Institute of Pharmacy and Sciences, Faizabad Road, Lucknow, Uttar Pradesh - 226028.

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ABSTRACT
Background: The prevalence of peptic ulcer Disease has decreased as did its elective surgery; however its complications still occur. Gastric outlet obstruction is that the least frequent complication of peptic ulceration disease. **Case Summary:** During this report, we present a case of massive gastric dilation thanks to gastric outlet obstruction that needed emergency surgery thanks to perforation. **Discussion:** Non-operative management is usually tried first, with medical therapy and endoscopic dilation. Emergent surgery is never needed, but during this patient, despite trying to optimize his condition first, the ulcer perforation precipitated surgical management. **Conclusion:** Although Gastric Outlet Obstruction is that the least frequent complication of peptic ulceration Disease and typically non-operative treatment is tried first, surgery could also be necessary.

INTRODUCTION

Peptic ulcer disease (PUD) may be a break within the inner lining of the stomach, the primary a part of the tiny intestine, or sometimes the lower esophagus. An ulcer within the stomach is called a gastric ulcer, while one in the first part of the intestines is a duodenal ulcer. With a gastric ulcer, the pain may worsen with eating. The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. A few third of older people haven't any symptoms. Complications may include bleeding, perforation, and blockage of the stomach. Bleeding occurs in as many as 15% of cases. Common causes include the bacteria Helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAIDs). Other, less common causes include tobacco smoking, stress thanks to serious illness, Behcet disease, Zollinger-Ellison syndrome, Cohn disease, and liver cirrhosis. Older people are more sensitive to the ulcer-causing effects of NSAIDs. The diagnosis is usually suspected due to the presenting symptoms with confirmation by either endoscopy or barium swallows. H. pylori are often diagnosed by testing the blood for antibodies, a urea breath test, testing the stool for signs of the bacteria, or a biopsy of the stomach. Other conditions that produce similar symptoms include stomach cancer, coronary heart condition, and inflammation of the stomach lining or gallbladder inflammation.[1-8]

SINGS AND SYMPTOMS

- Abdominal pain, epigastria, strongly correlated with mealtimes. In case of duodenal ulcers, the pain appears.
- Bloating and abdominal fullness.
- A rush of saliva after an episode of regurgitation to dilute the acid in esophagus, although this is more associated with gastro-esophageal reflux disease.
- nausea and copious vomiting
- Appetite loss and weight loss, in gastric ulcer.
- Duodenal ulcer as the pain is relieved by eating, weight gain
- Hematemesis (vomiting of blood), this can occur due to bleeding directly from a gastric ulcer or from damage to the esophagus from severe/continuing vomiting.
- Melina (tarry, foul-smelling feces due to presence of oxidized iron from hemoglobin)
- Rarely, an ulcer can lead to a gastric or duodenal perforation, which leads to acute peritonitis and extreme, stabbing pain,[9] and requires immediate surgery.

A history of heartburn or gastro-esophageal reflux disease (GERD) and use of certain medications can raise the suspicion for peptic ulcer. In people over the age of 45 with more than two weeks of the above symptoms, the odds for peptic ulceration are high enough to warrant rapid investigation by esophagogastro-duodenoscopy. The timing of symptoms in reference to the meal may differentiate between gastric and duodenal ulcers. A peptic ulcer would give epigastria pain during the meal, related to nausea and vomiting, as gastric acid production is increased as food enters the stomach. Pain in duodenal ulcers would be aggravated by hunger and relieved by a meal and is associated with night pain.[9-10] Furthermore, typical ulcers tend to heal and recur, and as a result the pain may occur for few days and weeks and then wane or

disappear.[11] Usually, children and elderly not develop any symptoms unless complications have arisen. Pain is typically caused by the ulcer, but it's going to be aggravated by the stomach acid when it comes into contact with the ulcerated area. The pain caused by peptic ulcers are often felt anywhere from the navel up to the sternum, it's going to last from jiffy to many hours, and it's going to be worse when the stomach is empty. Also, sometimes the pain may flare at night, and it can commonly be temporarily relieved by eating foods that buffer stomach acid or by taking anti-acid medication.[12] However, peptic ulcer disease symptoms could also be different for each sufferer.[13]

TYPE OF PEPTIC ULCER[14-17]

Peptic ulcers are a form of acid–peptic disorder. Peptic ulcers classified according to their location and other factors.

By location

- Duodenum (called duodenal ulcer)
- Esophagus (called esophageal ulcer)
- Stomach (called gastric ulcer)
- Meckler's diverticulum

Modified Johnson

- **Type I** - In stomach, ulcer along the body. Not associated with acid hyper-secretion.
- **Type II** - In body ulcer combined with duodenal ulcers. Associated with acid over secretion.
- **Type III** - In pyloric channel within 3 cm of pylorus. Associated with acid over secretion.
- **Type IV** - Proximal gastro-esophageal ulcer.
- **Type V** - Can occur throughout the stomach.

Macroscopic appearance

Gastric ulcers are most frequently localized on the lesser curvature of the stomach. The ulcer may be round to oval parietal defect ("hole"), 2–4 cm diameter, with a smooth base and perpendicular borders. These borders aren't elevated or irregular within the acute sort of peptic ulceration, and regular but with elevated borders and inflammatory surrounding within the chronic form. In the ulcerative sort of gastric cancer, the borders are irregular. A gastric peptic ulceration may be a mucosal perforation that penetrates the muscular is mucosa and lamina propria, usually produced by acid-pepsin aggression. Ulcer margins are perpendicular and present chronic gastritis. During the active phase, the bottom of the ulcer shows 4 zones: fibrinoid necrosis, inflammatory exudates, granulation and animal tissue. The fibrous base of the ulcer may contain vessels with thickened wall or with thrombosis.[18-19]

Proton pump inhibitors - It also called **PPIs**, it reduce stomach acid by blocking the action of the parts of cells that produce acid. These drugs include the prescription and over-the-counter medications, omeprazole, lansoprazole, **rabeprazole** (**Aciphex**), **esomeprazole** (**N exium**) and pantoprazole (Protonix).

Esomeprazole

It is the S-enantiomer of omeprazole; claimed to have higher oral bioavailability and to produce better control of intragastric pH than omeprazole in GERD patients because of slower elimination and longer t½. Higher healing rates of erosive esophagitis and better GERD symptom relief are reported in comparative trials with omeprazole. Side effect and drug interaction profile is similar to the racemic drug.

Rabeprazole

This newer PPI is claimed to cause fastest acid suppression. Due to higher pKa, it is more rapidly converted to the active pieces. However, potency and efficacy are similar to omeprazole.

USES

The H2 blockers are used in conditions in which it is profitable to suppress gastric acid secretion. Appropriate doses used, all available agents have similar efficacy. However, PPIs, because of higher efficacy and equally good tolerability, have outstripped H2 blockers.

1. Peptic ulcer - Omeprazole 20 mg OD is equally or more effective than H2 blockers. Relief of pain is rapid and excellent. Faster healing has been demonstrated with 40 mg/day: some duodenal ulcers heal even at 2 weeks and the remaining (over 90%) at 4 weeks. Gastric ulcer generally requires 4–8 weeks. Peptic ulcer caused healing ulcers in patients not responding to H2 blockers. PPIs are an integral component of anti-H. pylori therapy. Drugs choice for PPIs Like, NSAID induced gastric/duodenal ulcers. However, higher doses given for longer periods are generally required. When the NSAID cannot be stopped, it is advisable to switch over to a COX-2 selective NSAID. PPIs Maintenance treatment reduces recurrence of NSAID associated ulcer.[20-21]

Adverse effects

- Nausea
- loose stools
- Headache
- Abdominal pain
- Muscle and joint pain
- Dizziness are complained by 3–5%

EPIDEMIOLOGY AND ETIOLOGIC FACTORS

Peptic ulcer disease may be a source of serious morbidity and mortality worldwide. The prevalence of peptic ulcer disease in the United States is estimated to be 8.4%.6 Higher peptic ulcer disease incidence has been found to be associated with male sex, smoking, and chronic medical conditions. Peptic ulcer disease has also been found to be associated with increasing age.[15,16] Over time, a significant decrease in peptic ulcer disease diagnoses, as well as its associated complications, has been observed in both the United States and elsewhere in the world. The majority of peptic ulceration disease cases

are now known to be related to H. pylori infection or the use of non steroidal anti inflammatory drugs (NSAIDs), or both.[17] H. pylori is a Gram-negative bacterium that colonizes the gastric mucosa, progressing to gastritis and potentially peptic ulcer disease and gastric cancer.[18,19] H. pylori affects an outsized segment of the population; however, only a little subset will develop clinical disease. NSAID use, including aspirin, is common and results in an increased risk of gastrointestinal adverse events, including peptic ulceration disease. The relative risk of developing a symptomatic ulcer is 4.0 for non aspirin NSAID users and 2.9 for patients taking aspirin.[20] While H. pylori and NSAID use are the cause of the vast majority of peptic ulcers, other less common causes have been identified, including gastrinoma (Zollinger-Ellison syndrome), Diagnostic testing for H. pylori infection includes urea breath testing, stool antigen testing, rapid urea's testing or histology of gastric biopsies taken at the time of upper endoscopy, and serologic testing. In most circumstances, tests for active infection (urea breath testing, stool antigen testing, rapid urea's testing, or histology) are preferable compared with serologic antibody testing thanks to low pretest probability of infection. In those with documented peptic ulceration disease, serologic testing for H. pylori immunoglobulin G antibody is acceptable thanks to a better pretest probability. Those with a peptic ulceration disease history who are treated for H. pylori in the past are advised to undergo testing for eradication with either stool antigen testing or urea breath testing 21. Due to the possibility of false negative testing, testing to confirm eradication of H. pylori infection should be performed no sooner than 1 month after completing antibiotic treatment.[22-23]

CONCLUSION

Timely diagnosis and management of peptic ulcer disease and its sequel are crucial, as is prevention of peptic ulcer disease among patients at high risk. Prompt diagnosis of H. pylori and appropriate therapy is important, as is cautious use of NSAIDs. Our study strongly suggests that empirical H. pylori eradication, strategy associated with PPI prophylaxis in patients remaining on NSAID is the dominant strategy for preventing Re-current peptic ulcer bleeding. Pharma-coeconomic model stress the need to confirm the effectiveness of the empirical eradication strategy by clinical studies in patients with bleeding ulcer.

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