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A REVIEW ON ULCER WITH A BASIC DETAILS AND TREATMENT

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ABSTRACT

Ulcer disease is a common gastrointestinal disorder characterized by the development of erosions in the lining of the stomach, duodenum, or esophagus. This review provides a concise overview of ulcers, encompassing their basic definition, etiology, common symptoms, diagnostic approaches, and current treatment strategies. The primary causes, including *Helicobacter pylori* infection and nonsteroidal anti-inflammatory drug (NSAID) use, are discussed. Typical clinical manifestations such as epigastric pain are highlighted. The review outlines the importance of diagnostic methods like endoscopy and *H. pylori* testing. Finally, it summarizes the current treatment paradigm, which focuses on acid suppression, eradication of *H. pylori* when present, and lifestyle modifications to promote ulcer healing and prevent recurrence. Pathogenesis Briefly explaining the mechanisms involved in the formation of the ulcer, such as the imbalance between aggressive and protective factors in the gastrointestinal lining. Symptoms Outlining the common signs and symptoms associated with ulcers, such as abdominal pain (often epigastric), bloating, nausea, vomiting, and potentially more severe symptoms like bleeding. Complications Briefly noting potential complications of untreated ulcers, such as bleeding, perforation, obstruction, and in rare cases, an increased risk of gastric cancer.

KEYWORDS: *Helicobacter pylori*, Non-steroidal anti-inflammatory drug, Stomach, ulcer disease.

INTRODUCTION

An ulcer is an open sore that develops on the lining of the stomach or the upper part of the small intestine, known as the duodenum. Peptic ulcers, the most common type, are caused by damage to the stomach's protective lining, often due to acid. Excessive Stomach Acid

Production. An overproduction of stomach acid, known as hyperacidity, can also contribute to the development of ulcers. Conditions such as Zollinger-Ellison syndrome, which causes the stomach to produce excessive acid, can increase the risk of ulcers. It's worth noting that while excessive stomach acid can be a contributing factor, many ulcers occur even in the presence of normal or reduced acid production. The most common symptom is stomach pain, which may be burning or gnawing.[1]

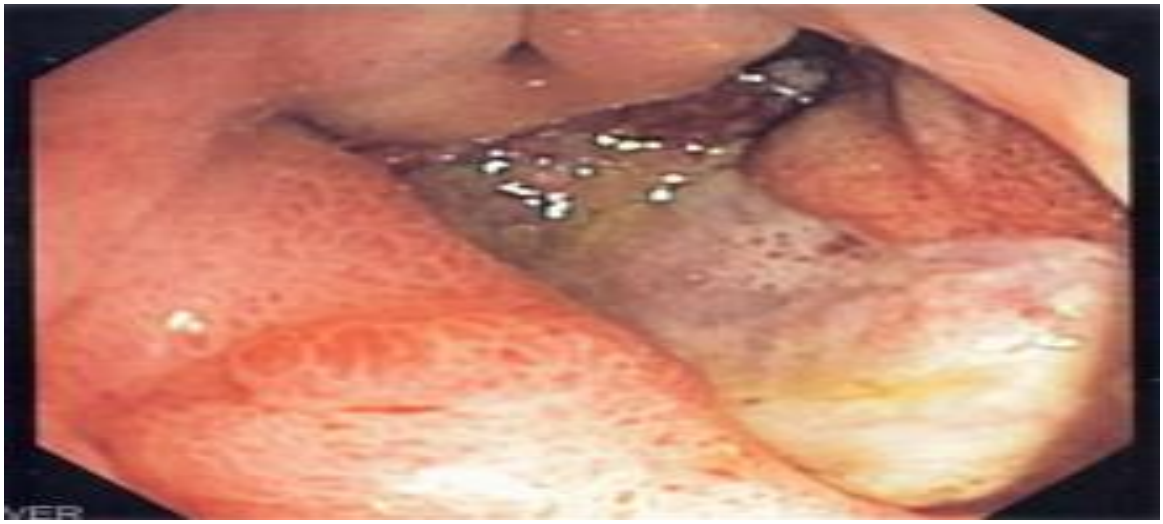

Figure 1: Peptic ulcer.

Due to life style factor arise and causes ulcer

1: Stress: Although stress doesn't directly cause ulcers, it can worsen symptoms and delay healing. Managing stress through relaxation techniques and lifestyle modifications can be beneficial.

2: Spicy Foods and Caffeine: Contrary to popular belief, spicy foods and caffeine don't directly cause ulcers. However, they can exacerbate symptoms and increase discomfort in individuals already affected by ulcers.

3: Smoking and Alcohol Consumption: Smoking and excessive alcohol consumption can impair the healing process and increase the risk of complications in individuals with ulcers. Quitting smoking and moderating alcohol intake can significantly improve outcomes.[2]

In ulcer show some Symptoms Stomach pain Often described as burning or gnawing, and may be worse on an empty stomach or at night. Nausea and vomiting. Loss of appetite or weight loss. Bloating or burping. There some Types of Ulcers here Gastric Ulcer An ulcer located in the stomach and Duodenal Ulcer An ulcer located in the duodenum (the first part

of the small intestine). Helicobacter pylori (H. pylori) infection A bacteria that can cause ulcers. Long-term use of NSAIDs Medications like aspirin and ibuprofen can irritate the stomach lining. Treatment Medications: Antibiotics to treat H. pylori infection, proton pump inhibitors (PPIs) to reduce acid production, and antacids Lifestyle changes: Avoiding smoking, alcohol, and foods that trigger symptoms. Surgery: In rare cases, surgery may be needed to remove the ulcer or a portion of the stomach. Complications Bleeding: An ulcer can bleed, causing vomiting blood or black, tarry stools. Perforation: A hole can develop in the stomach or duodenum, leading to a life-threatening infection. Gastric obstruction: The ulcer can block the flow of food through the digestive system.[3]

TYPE OF ULCER

These are sores in the lining of the stomach or duodenum (the first part of the small intestine).

Gastric ulcers: Occur in the stomach.

Duodenal ulcers: Occur in the duodenum.

Venous Ulcers: These are open sores that develop on the lower legs due to poor blood flow in the veins, often caused by varicose veins.[4]

Diabetic Foot Ulcers: These ulcers are a common complication of diabetes, often occurring on the feet and toes due to nerve damage and poor circulation.

Mouth Ulcers (Canker Sores): These are painful sores that can develop inside the mouth, often appearing as small, white or yellow sores with a red border.

Genital Ulcers: These are sores that can develop on the genitals, often caused by sexually transmitted infections like herpes.

Pressure Ulcers (Bedsores): These sores occur on the skin due to prolonged pressure, often on bony areas like the heels or hips.[5]

Corneal Ulcers: Sores on the cornea (the clear front part of the eye).

Esophageal Ulcers: Sores in the lining of the esophagus.[6]

PATHOPHYSIOLOGY: The peptic ulcer disease (PUD) mechanism results from an imbalance between gastric mucosal protective and destructive factors. Risk factors predisposing to the development of PUD:

- *H. pyl*ori infection
- NSAID use
- First-degree relative with PUD

- Emigrant from a developed nation
- African American/Hispanic ethnicity.

With peptic ulcers, there is usually a defect in the mucosa that extends to the muscularis mucosa. Once the protective superficial mucosal layer is damaged, the inner layers are susceptible to acidity. Further, the ability of the mucosal cells to secrete bicarbonate is compromised.[7]

H. pylori is known to colonize the gastric mucosa and cause inflammation. *H. pylori* also impair the secretion of bicarbonate, promoting the development of acidity and gastric metaplasia.[8]

MEDICAL TESTS FOR ULCER: Physical examination, and invasive/non-invasive medical tests. A careful history should be obtained and noted for the presence of any complications. Patient reporting of epigastric abdominal pain, early satiety, and fullness following a meal raise suspicion of PUD. The pain of gastric ulcers increases 15 to 30 minutes after a meal and may result in weight loss, whereas the pain of duodenal ulcers decreases with a meal, which can result in weight gain. Any patient presenting with anemia, melena, hematemesis, or weight loss should be further investigated for complications of PUD, predominantly bleeding, perforation, or cancer. A physical exam may reveal epigastric abdominal tenderness and signs of anemia.[9]

Investigations

1. *Esophagogastroduodenoscopy (EGD):* Gold standard and most accurate diagnostic test with sensitivity and specificity up to 90% in diagnosing gastric and duodenal ulcers. The American Society of Gastrointestinal Endoscopy has published guidelines on the role of endoscopy in patients presenting with upper abdominal pain or dyspeptic symptoms suggestive of PUD.[10] Patients over 50 years of age and new onset dyspeptic symptoms should be evaluated by an EGD. Anyone with the presence of alarm symptoms should undergo EGD, irrespective of age.
2. *Barium swallow:* It is indicated when EGD is contraindicated.
3. Complete blood work, liver function, and levels of amylase and lipase.
4. Serum gastric is ordered if Zollinger-Ellison syndrome is suspected.
5. *Helicobacter pylori* testing:

- *Serologic testing*

- *Urea breath test:* High sensitivity and specificity. It may be used to confirm eradication after 4 to 6 weeks of stopping treatment. In the presence of urease, an enzyme produced by H.pylori, the radiolabeled carbon dioxide produced by the stomach is exhaled by the lungs.[11]
- Antibodies to H.pylori can also be measured.
- *Stool antigen test*
- *Urine-based ELISA and rapid urine test*
- *Endoscopic biopsy:* Culture is not generally recommended as it is expensive, time-consuming, and invasive. It is indicated if eradication treatment fails or there is suspicion about antibiotic resistance. Biopsies from at least 4-6 sites are necessary to increase sensitivity. Gastric ulcers are commonly located on the lesser curvature between the antrum and fundus. The majority of duodenal ulcers are located in the first part of the duodenum.[12]

6. Computerized tomography of the abdomen with contrast is of limited value in the diagnosis of PUD itself but is helpful in the diagnosis of its complications like perforation and gastric outlet obstruction.[13]

Differential Diagnosis: The following conditions can present with symptoms similar to peptic ulcer disease and it is important to be familiar with their clinical presentation in order to make the correct diagnosis.

- Gastritis - an inflammatory process of the gastric mucosa from immune-mediated or infectious etiology presenting with upper abdominal pain and nausea. Clinical presentation is very similar to that of peptic ulcer disease.
- Gastroesophageal reflux disease (GERD) - patients usually describe a burning sensation in the epigastrium and lower retrosternal area, excessive salivation, or intermittent regurgitation of food material.
- Gastric cancer - apart from abdominal pain, patients usually describe alarm symptoms like weight loss, melena, recurrent vomiting, or evidence of malignancy elsewhere in case of metastasis.
- Pancreatitis - epigastric or right upper quadrant pain that is more persistent and severe, worse in the supine position, and patients usually have a history of alcoholism or gallstones. Elevated serum amylase and lipase are useful in the diagnosis.

- Biliary colic - intermittent, severe deep pain in the right upper quadrant or epigastrium precipitated by fatty meals.
- Cholecystitis - right upper quadrant or epigastric pain that usually lasts for hours, is exacerbated by fatty meals, and is associated with nausea and vomiting. Fever, tachycardia, positive Murphy sign, leukocytosis, and abnormal liver functions help further distinguish this from biliary colic.[14,15]

TREATMENT / MANAGEMENT

Medical Treatment

Antisecretory drugs used for peptic ulcer disease (PUD) include H2-receptor antagonists and proton pump inhibitors (PPIs). PPIs have largely replaced H2 receptor blockers due to their superior healing and efficacy. PPIs block acid production in the stomach, providing relief of symptoms and promoting healing. Treatment may be incorporated with calcium supplements as long-term use of the PPIs can increase the risk of bone fractures. NSAIDs induced PUD can be treated by stopping the use of NSAIDs or switching to a lower dose. Corticosteroids, bisphosphonates, and anticoagulants should also be discontinued if possible. Prostaglandin analogs (misoprostol) are sometimes used as prophylaxis for NSAID-induced peptic ulcers. First-line treatment for H. pylori-induced PUD is a triple regimen comprising two antibiotics and a proton pump inhibitor. Pantoprazole, clarithromycin, and metronidazole, or amoxicillin are used for 7 to 14 days. Antibiotics and PPIs work synergistically to eradicate *H. pylori*. The antibiotic selected should take into consideration the presence of antibiotic resistance in the environment. If first-line therapy fails, quadruple therapy with bismuth and different antibiotics is used.[16]

Medications that may be used include

- **Proton pump inhibitors (PPIs):** These drugs reduce stomach acid production and are commonly prescribed to treat ulcers regardless of the cause. Examples include omeprazole, lansoprazole, and pantoprazole.
- **H2 blockers (histamine H2-receptor antagonists):** These also reduce stomach acid production, although they are generally less potent than PPIs. Examples include famotidine and ranitidine.[17]
- **Antacids:** These medications neutralize stomach acid and can provide quick pain relief, but they don't heal ulcers.[18]

- **Cytoprotective agents:** These medications, such as sucralfate and misoprostol, help protect the stomach lining.[19]

Refractory Disease and Surgical Treatment

Surgical treatment is indicated if the patient is unresponsive to medical treatment, noncompliant, or at high risk of complications. A refractory peptic ulcer is one over 5 mm in diameter that does not heal despite 8-12 weeks of PPI therapy.[20] The common causes are persistent H. pylori infection, continued use of NSAIDs, or significant comorbidities that impair ulcer healing or other conditions like gastrinoma or gastric cancer. If the ulcer persists despite addressing the above risk factors, patients can be candidates for surgical treatment. Surgical options include vagotomy or partial gastrectomy.[21]

CONFLICT OF INTEREST: No conflict of interest.

CONCLUSION

In conclusion, ulcers represent a significant health concern characterized by disruptions in the protective lining of various bodily organs, most notably the gastrointestinal tract. The understanding of ulcer pathogenesis has evolved considerably, with *Helicobacter pylori* infection and the widespread use of NSAIDs identified as primary etiological factors for peptic ulcers. While the characteristic symptom is often abdominal pain, the clinical presentation can vary, and diagnosis relies on endoscopic evaluation and testing for *H. pylori*. Fortunately, significant advancements in medical treatment have dramatically improved the prognosis for individuals with ulcers. The advent of acid-suppressing medications and effective antibiotic regimens for *H. pylori* eradication have made ulcer healing and recurrence prevention highly achievable in most cases. However, it remains crucial to recognize and manage risk factors, such as NSAID use and lifestyle choices, to minimize the incidence and complications of ulcers.

REFERENCE

1. Dağ MS, Öztürk ZA, Akın I, Tutar E, Çıkman Ö, Gülşen MT. Drug-induced esophageal ulcers: case series and the review of the literature. Turk J Gastroenterol., 2014; 25(2): 180-4. doi: 10.5152/tjg.2014.5415. PMID: 25003679.
2. Carlborg B, Densert O, Lindqvist C. Tetracycline induced esophageal ulcers. a clinical and experimental study. Laryngoscope, 1983; 93(2): 184-7. doi: 10.1288/00005537-198302000-00011. PMID: 6823189.

3. Akbulut S, Caliskan AR, Saritas H, Demyati K, Bilgic Y, Unsal S, Koc C, Yilmaz S. Analysis of risk factors affecting the development of peptic ulcer perforation: case-control study. Prz Gastroenterol., 2021; 16(1): 23-28. doi: 10.5114/pg.2020.94744. Epub 2020 Apr 25. PMID: 33986884; PMCID: PMC8112271.
4. Yegen BC. Lifestyle and Peptic Ulcer Disease. Curr Pharm Des., 2018; 24(18): 2034-2040. doi: 10.2174/1381612824666180510092303. PMID: 29745325.
5. Tominaga K, Tsuchiya A, Sato H, Mizusawa T, Morita S, Ishii Y, Takeda N, Natsui K, Kawata Y, Kimura N, Arao Y, Takahashi K, Hayashi K, Yokoyama J, Terai S. Esophageal Ulcers Associated with Ulcerative Colitis: A Case Series and Literature Review. Intern Med., 2020 Aug 15; 59(16): 1983-1989. doi: 10.2169/internalmedicine.4437-20. Epub 2020 May 23. PMID: 32448835; PMCID: PMC7492127.
6. Akkus G, Sert M. Diabetic foot ulcers: A devastating complication of diabetes mellitus continues non-stop in spite of new medical treatment modalities. World J Diabetes, 2022; 13(12): 1106-1121. doi: 10.4239/wjd.v13.i12.1106. PMID: 36578865; PMCID: PMC9791571.
7. Hu SW, Chen AC, Wu SF. Drug-Induced Esophageal Ulcer in Adolescent Population: Experience at a Single Medical Center in Central Taiwan. Medicina (Kaunas), 2021; 57(12): 1286. doi: 10.3390/medicina57121286. PMID: 34946231; PMCID: PMC8708022.
8. Narayanan M, Reddy KM, Marsicano E. Peptic Ulcer Disease and *Helicobacter pylori* infection. Mo Med., 2018; 115(3): 219-224. PMID: 30228726; PMCID: PMC6140150.
9. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clin Microbiol Rev., 2006; 19(3): 449-90. doi: 10.1128/CMR.00054-05. PMID: 16847081; PMCID: PMC1539101.
10. armon RC, Peura DA. Evaluation and management of dyspepsia. Therap Adv Gastroenterol., 2010; 3(2): 87-98. doi: 10.1177/1756283X09356590. PMID: 21180593; PMCID: PMC3002574.
11. Kim SY, Park JM. Quality indicators in esophagogastroduodenoscopy. Clin Endosc., 2022; 55(3): 319-331. doi: 10.5946/ce.2022.094
12. Kuo FC, Wang SW, Wu IC, Yu FJ, Yang YC, Wu JY, Wang WM, Wu DC. Evaluation of urine ELISA test for detecting Helicobacter pylori infection in Taiwan: a prospective

study. World J Gastroenterol., 2005; 11(35): 5545-8. doi: 10.3748/wjg.v11.i35.5545. PMID: 16222752; PMCID: PMC4320369.

13. Di Saverio S, Bassi M, Smerieri N, Masetti M, Ferrara F, Fabbri C, Ansaloni L, Ghersi S, Serenari M, Coccolini F, Naidoo N, Sartelli M, Tugnoli G, Catena F, Cennamo V, Jovine E. Diagnosis and treatment of perforated or bleeding peptic ulcers: 2013 WSES position paper. World J Emerg Surg., 2014; 9: 45. doi: 10.1186/1749-7922-9-45. PMID: 25114715; PMCID: PMC4127969.
14. Femia G, French JK, Juergens C, Leung D, Lo S. Right ventricular myocardial infarction: pathophysiology, clinical implications and management. Rev Cardiovasc Med., 2021; 22(4): 1229-1240. doi: 10.31083/j.rcm2204131. PMID: 34957766.
15. Chhabra P, Ingole N. Gastroesophageal Reflux Disease (GERD): Highlighting Diagnosis, Treatment, and Lifestyle Changes. Cureus, 2022; 14(8): 28563. doi: 10.7759/cureus.28563. PMID: 36185857; PMCID: PMC9517688.
16. Begg M, Tarhuni M, N Fotso M, Gonzalez NA, Sanivarapu RR, Osman U, Latha Kumar A, Sadagopan A, Mahmoud A, Khan S. Comparing the Safety and Efficacy of Proton Pump Inhibitors and Histamine-2 Receptor Antagonists in the Management of Patients With Peptic Ulcer Disease: A Systematic Review. Cureus, 2023; 15(8): 44341. doi: 10.7759/cureus.44341. PMID: 37779765; PMCID: PMC10538946.
17. Shin JM, Sachs G. Pharmacology of proton pump inhibitors. Curr Gastroenterol Rep., 2008; 10(6): 528-34. doi: 10.1007/s11894-008-0098-4. PMID: 19006606; PMCID: PMC2855237.
18. Garg V, Narang P, Taneja R. Antacids revisited: review on contemporary facts and relevance for self-management. J Int Med Res., 2022 Mar; 50(3): 3000605221086457. doi: 10.1177/03000605221086457. PMID: 35343261; PMCID: PMC8966100.
19. Ballinger A. Cytoprotection with misoprostol: use in the treatment and prevention of ulcers. Dig Dis., 1994; 12(1): 37-45. doi: 10.1159/000171435. PMID: 8200123.
20. Gurusamy KS, Pallari E. Medical versus surgical treatment for refractory or recurrent peptic ulcer. Cochrane Database Syst Rev., 2016; 3(3): CD011523. doi: 10.1002/14651858.CD011523.pub2. PMID: 27025289; PMCID: PMC7078917.
21. Bazzoli F, De Luca L, Graham DY. Helicobacter pylori infection and the use of NSAIDs. Best Pract Res Clin Gastroenterol., 2000; 15(5): 775-85. doi: 10.1053/bega.2001.0234. PMID: 11566040.