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Review Article

A REVIEW ON PEPTIC ULCER, ITS CAUSES AND TREATMENT

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The most common causes of peptic ulcer disease (PUD) are Helicobacter pylori infection and use of nonsteroidal anti-inflammatory drugs (NSAIDs). The test-and-treat strategy for detecting H. pylori is appropriate in situations where the risk of gastric cancer is low based on age younger than 55 years and the absence of alarm symptoms. Most other patients should undergo upper endoscopy to rule out malignancy and other serious causes of dyspepsia. Urea breath tests and stool antigen tests are most accurate for identifying H. pylori infection and can be used to confirm cure; serologic tests are a convenient but less accurate alternative and cannot be used to confirm cure. Treatment choices include standard triple therapy, sequential therapy, quadruple therapy, and levofloxacin-based triple therapy. Standard triple therapy is only recommended when resistance to clarithromycin is low. Chronic use of NSAIDs in patients with H. pylori infection increases the risk of PUD. Recommended therapies for preventing PUD in these patients include misoprostol and proton pump inhibitors. Complications of PUD include bleeding, perforation, gastric outlet obstruction, and gastric cancer. Older persons are at higher risk of PUD because of high-risk medication use,including antiplatelet drugs,warfarin, selective serotonin reuptake inhibitors, and bisphosphonates.

Key words: peptic ulcer, H. pylori, duodenal ulcer, gastric ulcer

INTRODUCTION

Peptic ulcer disease (PUD) is a break in the lining of the stomach, first part of the small intestine or occasionally the lower esophagus. [1,2] An ulcer in the stomach is known as a gastric ulcer while that in the first part of the intestines is known as a duodenal ulcer.[1] The small intestine comprises the duodenum, ileum, and jejunum. Duodenal ulcers usually occur in younger people.

Causes of peptic ulcer:

•H. pylori bacteria (Helicobacter pylori). Most ulcers are caused by an infection from a bacteria or germ called H. pylori. This bacteria hurts the mucus that protects the lining of your stomach and the first part of your small intestine (the duodenum). Stomach acid then gets through to the lining.

•NSAIDs (nonsteroidal anti-inflammatory drugs). These are over-the-counter pain and fever medicines such as aspirin, ibuprofen, and naproxen. Over time they can damage the mucus that protects the lining of your stomach.

•Anti-bleeding drugs can also cause ulceration. Smoking and obesity also cause ulceration.

•Zollinger-Ellison Syndrome is a condition that leads to excess acid production. This can overwhelm the protective layer in the stomach and intestines and lead to ulcers.

•In a few cases, the ulcer is actually due to a cancer in the stomach or intestine.

Symptoms:

•Almost all people will have pain in the upper, central part of the abdomen (just under the breastbone). The pain can be a dull ache,

throbbing, sharp, burning, "gas-like," cramping, etc.[3]

- Nausea and vomiting
- Feeling full after eating a small amount of food
- Burping
- Not feeling hungry
- Losing weight without trying
- Bloody or black stool
- Vomiting blood

Diagnosis techniques:

1.Upper GI (gastrointestinal) series or barium swallow

In this procedure, doctor inserts a long tube with a camera down in throat and into the stomach and small intestine to examine the area for ulcers.This instrument also allows doctor to remove tissue samples for examination.

2.Upper endoscopy or EGD (esophagogas troduodenoscopy).

3.Blood tests. These check for infection-fighting cells (antibodies) that mean you have H. pylori.

4.Stool culture. A small sample of your stool is collected and sent to a lab. In 2 or 3 days, the test will show if you have H. pylori.

5.Urea breath test. This checks to see how much carbon dioxide is in your breath when you exhale.

6.In cases where Zollinger-Ellison Syndrome is suspected, a Gastrin level can be measured. In people with this disease, the Gastrin level will be elevated.

Risk factors

1.Use of NSAIDs. The higher the dose, the higher the risk of developing ulcers. Also, some NSAIDs have a lower risk of ulcer formation.

2.Infection with H. pylori.

3.Diseases such as Zollinger-Ellison Syndrome that lead to increased production of acid.

4.Increased stress may be a risk factor.

5.Smoking increases the risk of ulcers and slows ulcer healing.

6.In countries other than the U.S., lower socioeconomic status is a risk factor because those people have a higher rate of infection with H. pylori.

Pathophysiology of H. pylori

H. pylori, a gram-negative, helical, rod-shaped bacterium, colonizes the gastric mucosa of approximately one-half of the world population[4] and an estimated 30% to 40% of the U.S. population.[5] H. pylori is present in 95% of patients with duodenal ulcers and in 70% of those with gastric ulcers.[6] It is typically transmitted via the fecal-oral route during early childhood and persists for decades. The bacterium is a known cause of gastric and duodenal ulcers[7] and is a risk factor for mucosa-associated lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma.[11][12]

Treatment:

Treatment Eradication of H.pylori is recommended in all patients with PUD.[10]First-line therapy should have an eradication rate of more than 80%.[6] Because pretreatment susceptibility is rarely known to the primary care physician, therapy must be chosen empirically based on regional bacterial resistance patterns, local recommendations, and drug availability. standard triple therapy is a reasonable initial therapy where clarithromycin

resistance is low.[4][6][11][12]

Eradication heals most duodenal ulcers and greatly diminishes the risk of recurrent bleeding.[13] A systematic review found that treatment of H. pylori infection is more effective than antisecretory noneradicating therapy (with or without long-term maintenance antisecretory therapy) in preventing recurrent bleeding from peptic ulcer.[14]Current data suggest that increasing the duration of therapy to 14 days significantly increases the eradication rate.

Test of cure:

Test of cure for all patients after therapy is neither cost-effective nor practical. Indications for eradication testing with the urea breath test or stool antigen test include H. pylori–associated ulcer,continueddyspeptic symptoms H. pylori, pylori–associated MALT lymphoma, and resection for gastric cancer.[4] When indicated, eradication testing should be performed at least four weeks after completion of therapy.[4]

Standard Triple Therapy: A seven to 10-day triple drug regimen consisting of a PPI, amoxicillin 1 g, and clarithromycin 500 mg (Biaxin) twice daily has long been the first-line therapy to eradicate H. pylori. However, increasing resistance to clarithromycin is associated with declining eradication ratesnow well below 80%[15] Therefore, thisregimen is not recommended where the prevalence of clarithromycinresistant strains of H. pylori exceeds 15% to 20%.[12] An alternative triple drug regimen substitutes metronidazole 500 mg twice daily for amoxicillin. Adding probiotics to triple therapy, specifically Saccharomyces boulardii and Lactobacillus, has been shown to increase eradication rates (absolute increase of 9% and 5%, respectively) and decrease adverse effects of treatment, particularly diarrhea (absolute decrease of 14% and 7%, respectively).

Sequential Therapy: Sequential therapy consists of a five-day course of a PPI and amoxicillin 1 g taken twice daily, followed by a five-day course of a PPI, clarithromycin 500 mg, and metronidazole 500 mg (Flagyl) or tinidazole 500 mg (Tindamax) taken twice daily. The overall eradication rate is 84%, with an eradication rate of 73% for clarithromycin-resistant strains. A recent meta-analysis of available global data revealed that sequential therapy is superior to seven-day triple therapy, but it is not superior to 14-day triple therapy, bismuth-based quadruple therapy, or non–bismuth-based quadruple therapy.[11]

Compliance and tolerance rates of sequential therapy are similar to those of triple therapy but cost is lower, especially when the cost of failure of first-line therapy is considered. However, most studies were performed in Italy, and the ACG guideline states that sequential therapy requires validation in the United States.[5]

Non–Bismuth-Based Quadruple Therapy (Concomitant Therapy):

This approach involves the addition of metronidazole 500 mg or tinidazole 500 mg twice daily to the standard triple regimen. It is less complex than sequential therapy with similar eradication rates.[16,17] Additionally, non– bismuth-based quadruple therapy may be more effective than sequential therapy in patients with dual

antibiotic resistance to clarithromycin and metronidazole. [18] It has the highest eradication rate, about 90%, even in areas with high clarithromycin and metronidazole resistance, but would presumably cost more than sequential therapy because clarithromycin is taken for 10 days.

Bismuth-Based Quadruple Therapy: This is the traditional quadruple regimen and includes a bismuth salt (subsalicylate 525 mg or subcitrate potassium 420 mg), metronidazole 250 mg, and tetracycline 375 to 500 mg, all taken four times daily, in addition to a PPI taken twice per day.[4] Bismuth-based quadruple therapy is often employed as salvage therapy if first-line treatment fails, but it may be used as first-line therapy in areas of high resistance or when cost is an important consideration. A three-in-one combination capsule containing bismuth subcitrate potassium, metronidazole, and tetracycline has been developed to help reduce the pill burden, but patients still have to take three capsules four times per day in addition to a PPI. The regimen is usually given for 10 to 14 days.

Levofloxacin-Based Triple Therapy: This is a 10-day regimen of a PPI and amoxicillin 1 g twice daily, and levofloxacin 500 mg (Levaquin) once daily. The ACG states that this regimen requires validation in the United States. [5] It should be reserved for second-line therapy and is better tolerated than bismuthbased quadruple therapy.[12]

•Treatment will depend on the type of ulcer you have. Your healthcare provider will create a care plan for you based on what is causing your ulcer.

•Treatment can include making lifestyle changes, taking medicines, or in some cases having surgery. Lifestyle changes may include:[19]

•Not eating certain foods

•Quitting smoking.

•Limiting alcohol and caffein

•Not using NSAIDs (non-steroidal anti-inflammatory medicines.

Medicines to treat ulcers may include:

• **Antibiotics.** These bacteria-fighting medicines are used to kill the H. pylori bacteria. Often a mix of antibiotics and other medicines is used to cure the ulcer and get rid of the infection.

• **H2-blockers (histamine receptor blockers).** These reduce the amount of acid your stomach makes by blocking the hormone histamine. Histamine helps to make acid.

• **Proton pump inhibitors or PPIs**. These lower stomach acid levels and protect the lining of your stomach and duodenum.

• **Mucosal protective agents**. These medicines protect the stomach's mucus lining from acid damage so that it can heal.

• **Antacids.** These quickly weaken or neutralize stomach acid to ease your symptoms.

• **Ulcer healing agent**.

In most cases, medicines can heal ulcers quickly. Once the H. pylori bacteria is removed, most ulcers do not come back.

Conclusion:

Duodenal ulcer perforation is the second most common abdominal emergency in our study. After

invention of the H2 blockers and proton pump inhibitors the role of elective surgery for duodenal ulcer has been drastically decreasing, but the incidence of perforation is not much changing.

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