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INDICATIONS FOR THE TREATMENT OF GASTROINTESTINAL ULCERS WITH ANTIBACTERIAL DRUGS

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Abstract: This article analyzes the clinical indications for the use of antibacterial therapy in the management of gastrointestinal (GI) ulcers. Particular emphasis is placed on Helicobacter pylori (H. pylori)-associated peptic ulcers, non-H. pylori-related infections, and cases involving secondary bacterial complications. The paper outlines diagnostic criteria, pharmacological strategies, and current international guidelines for implementing antibacterial treatment. The importance of antibiotic stewardship and resistance prevention is also discussed.

Keywords: Gastrointestinal ulcers, peptic ulcer, Helicobacter pylori, antibacterial therapy, triple therapy, antimicrobial resistance, gastroenterology, ulcer complications.

INTRODUCTION

Gastrointestinal ulcers remain a prevalent and clinically significant pathology worldwide, with substantial implications for patient quality of life and healthcare systems. Among the various etiological factors, infection with *Helicobacter pylori* has been established as a leading cause of both gastric and duodenal ulcers. While acid suppression remains central to symptom control and mucosal healing, it is the eradication of infectious agents that provides definitive treatment in selected cases. Antibacterial therapy, therefore, plays a pivotal role in the modern management of gastrointestinal ulcers — but only in

specific, evidence-based scenarios. This article explores the precise indications for antibiotic use in ulcer treatment, the rationale behind various therapeutic regimens, and the challenges posed by emerging antimicrobial resistance.

MATERIALS AND METHODS

The principal indication for the use of antibacterial drugs in gastrointestinal ulcer therapy is the presence of *Helicobacter pylori* infection. This Gram-negative, spiral-shaped bacterium colonizes the gastric mucosa and disrupts mucosal defenses through urease production, cytotoxin release, and induction of local inflammation. A causal relationship between *H. pylori* infection and ulcer formation has been firmly established by epidemiological, clinical, and interventional studies. Consequently, all patients diagnosed with active peptic ulcers (either gastric or duodenal) and confirmed *H. pylori* infection are recommended to receive eradication therapy, regardless of symptom severity or ulcer size.

Diagnosis of *H. pylori* may be made through non-invasive methods such as urea breath testing, stool antigen testing, or serological assays, as well as invasive procedures like gastric mucosal biopsy during endoscopy with histology, culture, or rapid urease testing. Confirmation of infection is essential before initiating antibacterial treatment to avoid unnecessary antibiotic exposure and to ensure appropriate targeting of therapy.

RESULTS AND DISCUSSION

The most commonly employed antibacterial regimen is the so-called triple therapy, which typically consists of a proton pump inhibitor (PPI) such as omeprazole or esomeprazole, combined with clarithromycin and either amoxicillin or metronidazole for 10–14 days. In regions with high clarithromycin resistance, quadruple therapy that includes bismuth compounds and tetracycline is recommended. Alternative regimens — such as sequential therapy or hybrid therapy — may be employed based on local resistance patterns and treatment response history. The goal is complete eradication of the pathogen to reduce ulcer recurrence and prevent complications such as bleeding or perforation [1].

Beyond *H. pylori*, there are limited but important scenarios in which antibacterial therapy may be warranted in gastrointestinal ulcer management. These include ulcers complicated by secondary bacterial infection, such as infected ulcer craters or phlegmonous gastritis. In such cases, broad-spectrum antibiotics targeting likely pathogens (e.g., *Staphylococcus aureus*, *Streptococcus spp.*, or anaerobes) may be administered alongside supportive care and acid suppression.

Another context includes the prevention of postoperative infectious complications following ulcer-related surgical interventions. While not a primary treatment for the ulcer itself, prophylactic antibiotics may be indicated perioperatively in patients undergoing gastrectomy or duodenal repair, especially in cases involving perforation or peritonitis.

Conversely, in non-*H. pylori*, non-NSAID-related ulcers — often referred to as idiopathic peptic ulcers — antibacterial therapy holds no proven benefit and should not be routinely used. Likewise, empirical use of antibiotics in uncomplicated ulcers without microbiological confirmation is discouraged due to the growing global threat of antimicrobial resistance.

Indeed, resistance to clarithromycin, metronidazole, and even amoxicillin has been increasingly reported, particularly in parts of Asia, Europe, and South America. This underscores the importance of region-specific guidelines and periodic surveillance of local resistance profiles. Molecular testing for resistance genes may eventually become a standard tool for guiding antibiotic selection, though such approaches remain limited in availability.

Clinicians must also consider patient adherence, potential drug interactions, and side effects when prescribing eradication regimens. Common adverse reactions include gastrointestinal upset, altered taste, diarrhea, and, in some cases, antibiotic-associated colitis. These factors further support the principle that antibacterial therapy should be reserved for clearly defined indications supported by diagnostic evidence [2].

In addition to established indications, the decision to initiate antibacterial therapy for gastrointestinal ulcers must take into account various special patient populations that present unique diagnostic and therapeutic challenges. Immunocompromised individuals, including those undergoing chemotherapy, solid organ transplant recipients, and patients with HIV/AIDS, may exhibit atypical presentations of peptic ulcer disease and altered immune responses to *H. pylori* infection. In such cases, early and aggressive eradication therapy may be warranted even in the absence of classic ulcer symptoms, as delayed treatment may contribute to mucosal complications, impaired healing, or secondary opportunistic infections.

Pediatric and geriatric populations also require tailored approaches. In children, although *H. pylori* infection is common, its correlation with symptomatic ulcer disease is less straightforward, and indiscriminate use of antibiotics is discouraged. In contrast, elderly patients often present with atypical symptoms, and coexisting conditions (e.g., cardiovascular disease, chronic kidney disease) may complicate the pharmacological profile and increase the risk of adverse drug reactions. For these reasons, antibacterial therapy should only be initiated after careful benefit-risk assessment and laboratory confirmation of infection.

An increasingly critical issue is the growing resistance of *H. pylori* strains to commonly used antibiotics. The primary mechanisms of resistance include point mutations in bacterial genes encoding ribosomal proteins and enzymes such as 23S rRNA (conferring clarithromycin resistance) or nitroreductases (affecting metronidazole activity). These mutations lead to reduced antibiotic binding or impaired drug activation, rendering standard regimens ineffective. The alarming global rise in dual or triple-resistant *H. pylori* strains has prompted the development of susceptibility-guided therapy, in which tailored antibiotic combinations are prescribed based on culture or molecular diagnostics. While this approach

shows improved eradication rates, it is often limited by cost, access, and laboratory infrastructure, particularly in low-resource settings [3].

To mitigate resistance-related treatment failures, adjunctive strategies have been explored. The use of probiotics — live microorganisms that confer health benefits — is one such approach. Several randomized controlled trials suggest that supplementation with strains like *Lactobacillus rhamnosus*, *Bifidobacterium bifidum*, and *Saccharomyces boulardii* during eradication therapy can improve patient compliance, reduce gastrointestinal side effects (such as diarrhea and bloating), and possibly enhance eradication rates. The proposed mechanisms include suppression of *H. pylori* colonization through competitive inhibition, immunomodulatory effects, and maintenance of mucosal integrity. Although probiotics are not substitutes for antibiotics, they represent a promising adjunct in comprehensive treatment protocols.

Once antibacterial therapy has been administered, post-treatment testing is essential to confirm eradication, particularly in patients with complicated ulcers, prior treatment failures, or high-risk features (e.g., gastric MALT lymphoma, early gastric cancer, or a family history of gastric malignancy). Non-invasive urea breath tests and stool antigen tests are preferred due to their high sensitivity and specificity, but should only be performed at least four weeks after completing therapy to avoid false negatives from residual antibiotic or proton pump inhibitor effects. Endoscopic confirmation with histology or culture may be required in refractory cases [4].

On a broader scale, the strategic use of antibacterial therapy in gastrointestinal ulcers also carries important public health implications. Mass screening and treatment initiatives for *H. pylori* have been proposed in regions with high gastric cancer incidence, such as East Asia, with the aim of reducing long-term disease burden. However, such programs must be carefully balanced against the risks of widespread antibiotic use, including disruption of gut microbiota, allergic reactions, and propagation of resistant bacterial species. This

necessitates the implementation of robust surveillance systems, public education campaigns, and adherence to international guidelines to ensure that antibiotic use remains judicious and evidence-based.

In summary, while the core indication for antibacterial treatment in gastrointestinal ulcers remains the eradication of *H. pylori*, clinical decision-making must incorporate patient-specific factors, emerging resistance trends, post-treatment follow-up strategies, and public health considerations. Future advancements in molecular diagnostics, pharmacogenomics, and personalized medicine are expected to further refine the appropriate use of antibiotics in this complex and evolving field [5].

CONCLUSION

Antibacterial therapy represents a cornerstone in the treatment of gastrointestinal ulcers caused by *Helicobacter pylori*, offering curative potential and reducing the risk of recurrence and serious complications. However, its use must be based on confirmed microbiological evidence and aligned with local resistance patterns to maintain therapeutic efficacy and minimize the risk of resistance development. Antibiotics have no role in idiopathic or NSAID-induced ulcers unless secondary infection is suspected. Adhering to evidence-based indications, implementing diagnostic testing protocols, and practicing antibiotic stewardship are essential for the responsible and effective management of gastrointestinal ulcers in the modern clinical landscape.

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