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Review Article

Helicobacter pylori Eradication Therapies: Current Strategies and Future Perspectives in Peptic Ulcer Disease

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A B S T R A C T

Helicobacter pylori (H. pylori) infection is a significant global health concern, linked to peptic ulcers, gastric adenocarcinoma, and mucosa-associated lymphoid tissue lymphoma. Standard triple therapy with proton pump inhibitors (PPIs) and antibiotics, while effective in many cases, faces rising failure rates due to antibiotic resistance, patient non-compliance, and adverse side effects. Quadruple and sequential therapies have been introduced, utilising additional agents like bismuth and adjusting antibiotic administration. However, these regimens still confront challenges, including drug resistance and high recurrence rates. Probiotics, particularly *Lactobacillus* strains, have emerged as potential adjuncts to standard therapy, and they have been shown to enhance eradication rates and reduce side effects. Alternative therapies like vonoprazan, a potassium-competitive acid blocker, and personalised dosing based on body size and genetic factors, particularly CYP2C19 variations, are under investigation to optimise efficacy. Innovative strategies, such as antibiotic stewardship guided by susceptibility testing, have also been proposed to enhance treatment outcomes while mitigating resistance. Patient education on adherence and proper medication usage has been shown to significantly impact eradication success rates. Additionally, broad eradication programmes aimed at high-risk populations have demonstrated substantial reductions in gastric cancer incidence and mortality, emphasising lifestyle adjustments and early detection. As research advances, future therapies may include antivirulence agents targeting *H. pylori's* virulence factors and new mucolytic drugs, promising better outcomes while preserving gut microbiota. Overall, a multifaceted approach incorporating personalised medicine, probiotic adjuncts, and patient-focused strategies appears essential for effectively managing *H. pylori* infection and minimising associated health risks.

Keywords: *Helicobacter pylori*, Peptic Ulcer, Personalised Medicine, Pharmacotherapy, Microbiota

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Introduction

Helicobacter pylori (H. pylori) is the bacteria that majorly causes diseases such as peptic ulcer, gastric adenocarcinoma, chronic gastritis, and mucosa-associated lymphoid tissue lymphoma, which is a gastric malignancy.[1] It is a major pathogen that has a significant effect on human mortality and morbidity. It is a helical s-shaped gram-negative short spiral bacterium with a width of 0.5–1 µm and a length of 2–4 µm.[2] The infection can be symptomatic as well as asymptomatic, and progress to gastric cancer which was initially identified in 1991. It is also connected to idiopathic thrombocytopenic purpura.[3] Signs of peptic ulcer caused by *H. pylori* can include nausea, bloating, burning, or empty discomfort in the stomach, along with black, tarry stools or blood in the vomit. It is typically spread by contaminated food, water, or saliva. Both gastric and duodenal peptic ulcers can be caused by a number of things, such as an H. pylori infection, chronic NSAID use, excessive alcohol consumption, smoking, or stress.

The diagnosis involves invasive (rapid urease test, biopsy, culture) or non-invasive (breath tests, stool studies, serology) methods. Treating *H. pylori* is vital not just for alleviating symptoms, but also for minimising the risk of stomach cancer. The traditional approach is a combination of antibiotics and PPIs , although rates of efficacy are decreasing due to drug resistance.[4] Concerns regarding the impact of prescribed antibiotics on Gastro intestinal (GI) health resulted in an investigation for natural remedies. Probiotics, which refers to helpful bacteria, have emerged to be potential therapy options for *H. pylori.*

Treatment, tailored to resistance rates, typically involves multiple antibiotics and acid suppression. Eradicating *H. pylori* is crucial to reduce complications and prevent cancer progression, with post-treatment monitoring equally vital. Thus, early detection, timely treatment, and vigilant follow-up are essential in managing *H. pylori* infection and mitigating associated risks, especially gastric cancer. [5]

Current Treatment Strategies

The most commonly used treatment plan for the eradication of *H. pylori* uses a standard three-drug regimen which is a combination of PPIs and antibiotics. The commonly used one is amoxicillin with metronidazole or clarithromycin have been considered as the first drug of choice for *H. pylori* treatment. The ineffectiveness of the therapy is seen due to the resistance to key antibiotics like metronidazole, clarithromycin, and levofloxacin.[6] Triple therapy centred around PPIs and antibiotics is said to be decreasing in effectiveness for treating *H. pylori,* mainly due to the heightened antibiotic resistance, the prevalence of antibiotic-related side effects, faster metabolism of PPI and diminished patient compliance. Antibiotics that are acid-fast and bactericidal like metronidazole, clarithromycin, and amoxicillin are the commonly used drugs for the treatment of *H. pylori.* In addition to that use of a single drug fails to eradicate the bacteria well, so we recommend using a multi-drug regimen.[7]

Due to the increased failure of triple therapy quadruple therapy containing bismuth is opted, for 10–14 days, based on how rapidly the disease advances, patients ought to consume a PPI of standard dose which is given twice a day, then a dose of 500 mg of tetracycline thrice a day, followed by 500 mg of metronidazole thrice a day, and bismuth thrice a day.[8]

Sequential treatment can also be used where the above-mentioned same antibiotics are administered sequentially. It is a five-day dual therapy that consists of the usual dose of PPI administered twice daily alongside 1 gram of amoxicillin twice a day. It is followed by a five-day triple therapy which comprises 500 mg of clarithromycin twice a day, followed by 500 mg of metronidazole twice a day, and a usual dose of PPI administered twice a day. Concomitant therapy also known as the non-bismuth tetrad therapy is the first choice for treatment in case of clarithromycin resistance. [9]

Treatment Challenges

The major treatment challenges faced in the case of *H. pylori* were increased resistance to antibiotics used for the treatment, patient-related factors like complexity of the disease, increased cost of medications, polypharmacy, tolerability, ineffective eradication of *H. pylori,* and unavailability of drugs in some geographical areas leading to treat ment with available drugs. [10]

Future Perspectives

In the coming years, the development of *Helicobacter pylori (H. pylori)* eradication medicines will be focused on overcoming the challenges posed by H. pylori infection, including its associations with chronic inflammation peptic ulcer disease, and gastric cancer. While the current standard treatments include acid-suppressing medications and antibiotics, the decreasing success rates and unintended consequences of prolonged broad-spectrum antibiotic use have emphasised the necessity for alternative options.

The traditional method of treating *H. pylori* through trial and error has given way to a new approach that focuses on antimicrobial resistance. To deal with the increasing incidence of antibiotic resistance, this strategy focuses on maximising the use of antibiotics. The initial action is to stop using antibiotics in instances where susceptibility testing results reveal resistance in previous years.[11] Also being tested as adjunct therapies that reduce side effects and increase eradication rates are probiotics, including *Lactobacillus* strains. It eliminates *H. pylori* and reduces side effects like diarrhoea and stomach discomfort. Promising

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modifications to *H. pylori* treatment regimens, likely probiotic combinations have been shown in meta-analyses to significantly raise eradication rates and reduce side effects.[12] In most studies, *Limosilactobacillus (L. reuteri)* combined with standard therapy to reduce the side effects and improve recurrence rates. [13]

H. pylori eradication therapy causes some modifications in gut microbiota and changes in potential functioning genes, as well as taxonomic changes and declines in the alpha diversity index. The safety of probiotics is still under study so some other alternatives were needed to improve the gut microbiota. In this case, treatment is done by adding butyric acid and inulin, which may help preserve the gut microbiota in its initial state and promote recovery along with bacterial eradication. [14]

The most effective regimen can be hard for gastroenterologists to figure out since it depends heavily on a number of variables that have been thoroughly examined in randomised clinical studies in the past. Vonoprazan, a new potassium-competitive acid blocker, could potentially improve the effectiveness of standard therapy. However, further research is needed to develop more effective drugs.[15]

A seven-day combination therapy that involved vonoprazan and amoxicillin resulted in a 93% *H. pylori* eradication rate, according to a study. Based on different studies, individuals who had smaller sizes had greater recovery rates with this therapy. Modifying the amoxicillin dose corresponding to the patient's body size could enhance the effectiveness of treatment, reduce disturbance of the gut microbiota, and lower antibiotic resistance. Before employing this regimen as the primary therapy for H. pylori eradication, additional studies from a variety of fields are necessary.[16]

Other therapeutic options for *H. pylori* eradication studies include nonantibiotic-like mucolytic agents and antivirulence therapy. However, erdosteine and NAC, both mucolytic medications which showed success in studies in humans, are costly and require large quantities, which inhibits their widespread adoption. Antivirulence therapeutics focus on *H. pylori's* virulence factors to enhance the specificity and efficacy of eradication therapy to reduce antibiotic resistance and limitations in current treatments for *H. pylori* infections.[13]

However, there were no studies to conclude about the prolonged negative effects of *H. pylori* eradication. In the beginning, it was considered that treating atrophic gastritis with medication could worsen acid reflux into the oesophagus. Animals that have been subjected to antibiotics have experienced changes in their gut microbiota, which may affect immune system function, metabolic rate, weight gain, and bone growth. There were also fears that massive elimination programmes could make bacteria resistant to antibiotics. In contrast, further studies proved no increased likelihood of colorectal or oesophageal cancer after the organism's removal. The latest research discovered that even though the infection's treatment raises the diversity of the gut microbiota, gut dysbiosis persists after eradication therapy. *H. pylori* eradication therapy is still an essential part of care despite these factors.[17]

The latest suggestions which have been included in the Maastricht 2-2000 Consensus Report, refer to giving PPIs twice daily in addition to metronidazole or clarithromycin, and to continue this dosage plan for at least seven days. Considering possible incentives, it is advised to give amoxicillin and clarithromycin at the same time as second-line therapy. As part of second-line therapy, a proton pump inhibitor (twice daily), bismuth salt (four times daily), metronidazole (three times daily), and tetracycline (four times daily) are usually given for not less than seven days. Additional proton pump inhibitor-based triple therapy might be an acceptable choice if bismuth is not available. Treatment failures must be treated by professionals since they frequently arise from inadequate compliance.[18]

The objective of the wide widespread screening and *H. pylori* eradication programme is to dramatically lower the incidence of cancer of the gastric tract. Policymakers, researchers, and healthcare providers collaborate on establishing a programme which targets people at high risk employing an array of screening methods, followed by elimination therapies and lifestyle changes that are specifically tailored to the individual. Microbial incidence and gastrointestinal cancer collision have been demonstrated to drastically decrease as a result of proper implementation in areas such as the Matsu Islands. The programme aims to further minimise related mortality by 39% and gastric cancer rates by 68% by 2025. [19] It emphasises significant value on integration into current healthcare systems, acceptability, and feasibility while illustrating affordability and population-wide impact. Efficacy is supported by evidence, particularly in zones where the presence of *H. pylori* is excessive. This evidence-based system actually suggests the importance of changes in lifestyle, eradication, and specific detection in the struggle toward cancer of the gastric tract.

The Japanese Society for the Research and Study of the bacteria Helicobacter recently revised its criteria, which now include evidence of the bacterial eradication of diseases apart from ulcers. The serum pepsinogen levels, endoscopic, and X-ray examination are now used as diagnostic techniques. It is more successful to use a one-week triple therapy consisting of amoxicillin, clarithromycin, and a potassium-competitive acid blocker (P-CAB). PPIs or P-CAB when combined with amoxicillin and metronidazole should be considered if screening for susceptibility is not

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acceptable. For this reason, and to provide appropriate strategies for infection elimination and subsequent cancer prevention, there is an intense emphasis on avoiding gastric cancer through the eradication of *H. pylori* bacteria in all age groups.

When primary and secondary therapy fails, patients seek advice from a physician for further treatment. While various treatment approaches often fail continuously because of the increase in infection rates and change in eradication indication. It is very important to identify the problems in the current treatment by considering factors like the stability of drugs, intestinal pH, antimicrobial properties, and genetic changes in CYP2C19. In some studies, they prove that PPIs can alter the metabolism by producing genetic variations in CYP2C19 in some patients, so here dose adjustment is better. The physician can add some tailoring methods to improve the outcomes in complex cases.

Novel approaches to eliminating bacteria comprise fourfold combination regimens, a 14-day high-dose regimen containing amoxicillin and esomeprazole, and a four-drug combination comprising famotidine, furazolidone, tripotassium dicitrato bismuthate, and josamycin. These treatments include fluoroquinolone sequential therapy, 10-day bismuth fourfold, 14-day bismuth quadruple anti-*H. pylori* therapy, and first-line bismuth-containing quadruple therapies. Furthermore, there are recovery medications such as triple therapy, including levofloxacin, concomitant therapy, a 14-day rifabutin triple salvage regimen, and high-dose double therapy. [20] Supplemental therapies like probiotics and statins are also being studied, but more convincing data is required to validate their significance in *H. pylori* eradication.

In the case of both adult and geriatric populations, the treatment provided is the same but repetitive prescription orders, quadruple therapy, high-dose PPIs, and longer duration of prescriptions lead to a decrease in the efficacy of therapy.

Patient education has a significant impact on improving the success rate of *H. pylori* eradication and poor patient adherence. If patients are provided adequate knowledge about the disease and proper patient counselling regarding the usage of drugs, to a greater extent we can improve resistance to antibiotics. Patient counselling is inexpensive, safe, and convenient and has a beneficial impact on patient adherence and the eradication rate of *H. pylori.*

Table 1.Key Strategies and Assessments in the Eradication and Treatment Optimisation of *H. pylori*

New Strategies	References	Key Findings
Antibiotic stewardship based on susceptibility testing	Cardos et al., 2021	Focusing on the fight against antimicrobial resistance and reducing the use of antibiotics when prior susceptibility testing has shown resistance.
Probiotic adjunct therapy (e.g., *Lactobacillus* strains)	Mestre et al., n.d.	Probiotics, especially strains of *Lactobacillus,* enhance eradication rates, alleviate side effects like diarrhoea and stomach pain, and support the eradication of *H. pylori.*
Limosilactobacillus (L. reuteri) as part of standard therapy	Liang et al., 2022	*L. reuteri,* when combined with standard *H. pylori* therapy, reduces side effects and recurrence rates, leading to improved outcomes
Alternative adjuncts (butyric acid and inulin)	Abdulkhakov et al., 2024	After eradication therapy, butyric acid and inulin help to maintain gut microbiota and promote recovery, possibly neutralising variations in gut microbiota and gene activity.
Potassium-competitive acid blocker (vonoprazan) in standard therapy	Matsumoto et al., 2019	*H. pylori* was 93% eradicated when vonoprazan and amoxicillin were taken together for seven days. Results could be improved by adjusting the amoxicillin dosage according to the patient's body size.
Dose modification of amoxicillin	Matsumoto et al., 2019	Adapting the dosage of amoxicillin to the patient's size may improve efficacy, lessen disruption of the gut microbiota, and decrease antibiotic resistance.
Nonantibiotic therapies (e.g., erdosteine, NAC, antivirulence Agents)	Liang et al., 2022	Antivirulence medicines target *H. pylori* virulence factors to increase specificity and decrease resistance; mucolytics, such as erdosteine and NAC, have demonstrated efficacy but are expensive.

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Assessment of long-term and microbiota effects of eradication therapy	Chiang et al., 2022	Early concerns about the adverse consequences of eradication therapy, like immunological disturbance and acid reflux, have mainly been eliminated, and there is no evidence of a higher risk of cancer. Despite being essential, eradication therapy may result in intestinal dysbiosis; if microbiota diversity increases but dysbiosis persists, this suggests that microbiota-supportive medicines may be helpful.
First- and second-line therapy options	Japanese Society, 2023	P-CAB combined with amoxicillin and clarithromycin, or with amoxicillin and metronidazole, is effective for *H. pylori* eradication when susceptibility screening is not possible.
Advanced combination regimens and supplemental therapies	Aumpan et al., 2023	Future potential new high-dose regimens include quadruple therapies (e.g., famotidine, furazolidone, bismuth, and josamycin) and a 14-day combination of amoxicillin and esomeprazole. The 14-day rifabutin triple therapy and levofloxacin-based triple therapy are viable salvage options for cases that are resistant. More research is required to confirm the benefits of supplemental treatments like probiotics and statins, but they are being investigated.

Conclusion

H. pylori eradication therapies including triple therapy have been a standard treatment option for *H. pylori* infections for over a decade. However, as resistance to marcolodes is a major hurdle leading to their effectiveness dropping, poor patient adherence and failure of treatment regimens demanding an alternate course of treatment. In an effort to tackle this, newly developed regimens that exhibit greater effectiveness against strains resistant to antibiotics have been proposed as the initial therapies. At present, the most prevalent treatments are non-bismuth quadruple therapy and vonoprazan-based triple therapy. These approaches have 90% eradication rates, even in regions with a substantial number of drug-resistant strains. Yet these regimens have limitations, notably, there is a chance of high antibiotic resistance and causing dysbiosis of the gut microbiota. An easy, affordable, and well-tolerated treatment which has a minimal impact on gut microbiota and future development of antibiotic resistance would be ideal. Nowadays, probiotics are added with quadruple therapies to *H. pylori* eradication. We believe it is vital to redefine the optimal therapeutic notion to include fewer adverse effects on gut microbiota balance and the potential growth of antibiotic resistance. Using amoxicillin alongside vonoprazan as a dual therapy is an appealing alternative that meets these criteria. This straightforward regimen offers a high degree of eradication, greater security, and minimal disruption to the flora of the gut, which could be beneficial in avoiding future resistant strains of antibiotics. We expect that this dual therapy can be extensively utilised across the world and that substantial data concerning its efficacy will ultimately be acquired. Further studies and research are essential to develop a novel approach to *H. pylori* eradication.

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