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Article

Antibiotic Resistance Patterns of Helicobacter pylori and Their Impact on Treatment Outcomes

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Abstract: Helicobacter pylori infection remains one of the most persistent and clinically significant challenges in gastroenterology, particularly due to its strong association with chronic gastritis, peptic ulcer disease, and gastric malignancies. In recent years, the effectiveness of standard eradication therapies has declined, largely driven by the increasing prevalence of antibiotic-resistant strains. Against this background, the present study aimed to evaluate current patterns of antibiotic resistance in Helicobacter pylori and to examine how these patterns influence treatment outcomes in a real-world clinical setting. A cross-sectional analytical approach was applied, involving 108 adult patients diagnosed with Helicobacter pylori infection. Diagnosis was confirmed through a combination of endoscopic biopsy-based methods and non-invasive testing. Antimicrobial susceptibility was assessed using culture-based techniques, focusing on commonly prescribed antibiotics such as clarithromycin, metronidazole, amoxicillin, and levofloxacin. All patients received standard eradication therapy, and treatment success was evaluated through follow-up testing six weeks after therapy completion. The findings revealed a high prevalence of resistance, particularly to metronidazole and clarithromycin, which significantly reduced eradication success rates. Patients infected with resistant strains showed markedly lower treatment responses compared to those with susceptible strains. Multidrug resistance emerged as the most critical factor associated with treatment failure, underscoring the limitations of empirical therapy.

Keywords: Helicobacter pylori; antibiotic resistance; eradication therapy; clarithromycin resistance; metronidazole resistance; treatment outcomes.

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1. Introduction

Helicobacter pylori is a Gram-negative, spiral-shaped bacterium that colonises the gastric mucosa and remains one of the most widespread chronic infections worldwide. It is estimated that more than half of the global population is infected, with significantly higher prevalence in low- and middle-income countries, where socioeconomic and hygiene factors facilitate transmission. This microorganism plays a central etiological role in chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue lymphoma, and gastric carcinoma, making it not only a clinical concern but also a major public health issue [1]. Despite decades of research and the availability of effective eradication regimens, the management of H. pylori infection continues to face serious challenges, the most critical of which is the increasing resistance to commonly used antibiotics.

Standard eradication therapy traditionally relies on a combination of proton pump inhibitors and two or more antibiotics, most commonly clarithromycin, amoxicillin, and metronidazole. However, the effectiveness of these regimens has declined considerably over the past two decades. The primary reason for this decline is the rapid emergence and global spread of antibiotic-resistant H. pylori strains, which directly compromise treatment success. Recent meta-analyses and global surveillance studies indicate that resistance to key antibiotics

such as clarithromycin and metronidazole has reached alarming levels, often exceeding clinically acceptable thresholds in many regions [2]. This trend has led to eradication failure rates that are increasingly unacceptable in routine clinical practice.

The mechanisms underlying antibiotic resistance in H. pylori are complex and multifactorial. Genetic mutations, particularly in genes associated with antibiotic targets, play a central role in resistance development. Additionally, adaptive mechanisms such as biofilm formation, efflux pump activation, and morphological transformation into coccoid forms contribute to bacterial persistence and reduced antibiotic susceptibility [3]. These biological adaptations not only complicate treatment but also promote the development of multidrug-resistant strains, further limiting therapeutic options.

From a clinical perspective, antibiotic resistance is now recognised as the leading predictor of eradication failure. Studies have demonstrated that patients infected with resistant strains are significantly less likely to achieve successful eradication using standard triple therapy. For example, clarithromycin resistance alone has been shown to reduce treatment efficacy below 70%, a threshold considered unacceptable for first-line therapy [4]. Furthermore, the situation is exacerbated in developing countries, where unregulated antibiotic use, limited diagnostic capacity, and insufficient surveillance systems contribute to both the emergence and underestimation of resistance patterns.

In recent years, there has been a growing emphasis on personalised and susceptibility-guided therapy as a strategy to overcome resistance-related challenges. Clinical guidelines increasingly recommend tailoring treatment based on local resistance patterns or individual antibiotic susceptibility testing. However, in many regions, including parts of Central Asia, such approaches remain limited due to infrastructural and economic constraints. As a result, empirical therapy continues to be widely used, often with suboptimal outcomes.

Given these challenges, understanding the current patterns of antibiotic resistance and their direct impact on treatment outcomes is essential for improving clinical management strategies. This study aims to analyse the resistance profiles of H. pylori to commonly used antibiotics and evaluate how these patterns influence the success of eradication therapy. By providing updated and regionally relevant data, this research seeks to contribute to more effective, evidence-based treatment approaches and ultimately improve patient outcomes.

2. Methods

This study was designed as a cross-sectional analytical investigation focusing on the evaluation of antibiotic resistance patterns in Helicobacter pylori and their direct influence on treatment outcomes. The methodological approach was intentionally structured to reflect routine clinical conditions observed in real gastroenterological practice, especially in settings where empirical treatment remains common due to limited access to advanced diagnostic tools.

A total of 108 patients with clinically confirmed dyspeptic symptoms were enrolled over a defined study period. The participants were aged between 18 and 65 years and represented both male and female populations in nearly equal proportions. Patients typically presented with persistent upper gastrointestinal complaints such as epigastric discomfort, postprandial fullness, nausea, and intermittent abdominal pain. To ensure the reliability of diagnostic findings, individuals who had used antibiotics, proton pump inhibitors, or bismuth-containing medications within four weeks before examination were excluded. This precaution helped avoid diagnostic bias and ensured that bacterial detection was not suppressed by recent pharmacological exposure [6].

Diagnosis of Helicobacter pylori infection was carried out using a combination of invasive and non-invasive techniques to enhance diagnostic accuracy. During upper gastrointestinal endoscopy, biopsy samples were carefully obtained from both the antral and corpus regions of the stomach. These samples were immediately subjected to rapid urease testing and later examined histologically to confirm the presence of the microorganism. For patients who declined endoscopy or had contraindications, stool antigen testing was employed as an alternative diagnostic method. The integration of multiple diagnostic modalities allowed for a more reliable identification of infection and minimised the likelihood of false-negative results, which can be particularly problematic in clinical practice [7].

The evaluation of antibiotic resistance was conducted through microbiological culture

susceptibility testing. Biopsy specimens were cultured under controlled microaerophilic conditions using selective growth media suitable for Helicobacter pylori isolation. Once bacterial growth was confirmed, antimicrobial susceptibility testing was performed using the E-test method, which provides a quantitative measurement of minimum inhibitory concentrations. The antibiotics selected for analysis included clarithromycin, metronidazole, amoxicillin, and levofloxacin, as these are commonly utilised in both first-line and second-line eradication therapies. Resistance thresholds were interpreted in accordance with internationally accepted clinical breakpoints, allowing for standardised classification of susceptible and resistant strains [8].

Following confirmation of infection, all patients received a standard first-line eradication regimen consisting of a proton pump inhibitor combined with clarithromycin and amoxicillin for a duration of 14 days. Patients demonstrating resistance to clarithromycin or those who failed to respond to initial therapy were transitioned to alternative treatment regimens, including metronidazole-based or levofloxacin-based therapies. Throughout the treatment period, adherence was closely monitored through follow-up visits and patient-reported compliance, as therapeutic success is strongly influenced by proper medication use.

Treatment outcomes were assessed six weeks after completion of therapy using stool antigen testing. A negative result was interpreted as successful eradication, while a positive result indicated treatment failure. The relationship between antibiotic resistance patterns and eradication outcomes was then systematically analysed to determine the clinical significance of resistance in therapeutic effectiveness.

For statistical analysis, both descriptive and inferential methods were applied. Resistance rates were expressed as percentages, and comparisons between groups were performed using chi-square testing. Logistic regression analysis was further employed to identify independent predictors of treatment failure, including antibiotic resistance status and selected demographic variables. A p-value of less than 0.05 was considered statistically significant, ensuring that the findings were both reliable and clinically meaningful [9].

This methodological framework was developed to provide a realistic yet scientifically robust assessment of Helicobacter pylori resistance patterns. By combining microbiological data with treatment outcome evaluation, the study offers a comprehensive perspective on how antibiotic resistance continues to shape clinical decision-making and patient prognosis.

3. Results

A total of 108 patients with confirmed Helicobacter pylori infection were included in the final analysis. The study population demonstrated a relatively balanced gender distribution, with a slight predominance of female participants. The mean age was 38.6 years, reflecting a typical adult population commonly affected by chronic dyspeptic symptoms. All patients completed the full course of eradication therapy and follow-up assessment, ensuring that outcome evaluation was consistent across the cohort.

The analysis of antimicrobial susceptibility revealed a clear and clinically meaningful pattern of resistance. Among the tested antibiotics, metronidazole exhibited the highest resistance rate, affecting exactly half of the isolates. Clarithromycin resistance was also substantial, approaching one-third of the cases, while levofloxacin showed a moderate level of resistance. In contrast, amoxicillin remained largely effective, with resistance observed only in a small proportion of patients. These findings suggest that while some components of standard therapy remain reliable, others are becoming increasingly compromised, which directly impacts treatment decisions [10].

Table 1. Antibiotic resistance patterns of Helicobacter pylori isolates (n = 108)

Antibiotic	Resistant (n)	Resistant (%)	Sensitive (%)
Clarithromycin	32	29.6%	70.4%
Metronidazole	54	50.0%	50.0%
Amoxicillin	9	8.3%	91.7%
Levofloxacin	21	19.4%	80.6%

and

The data presented in Table 1 highlight a concerning imbalance in antibiotic effectiveness. Metronidazole resistance, affecting half of the isolates, suggests a diminished role of this drug in both first-line and rescue therapies. Clarithromycin resistance, approaching 30%, is particularly significant because it directly undermines the effectiveness of standard triple therapy, which still relies heavily on this antibiotic. On the other hand, amoxicillin demonstrates consistently low resistance, reinforcing its continued value as a core component of eradication regimens. The intermediate resistance observed for levofloxacin indicates that while it remains a viable second-line option, its effectiveness may decline if resistance trends continue to rise.

When treatment outcomes were evaluated, the overall eradication success rate after first-line therapy was 72.2%. However, this average masks substantial variability depending on resistance status. Patients infected with antibiotic-susceptible strains achieved notably higher eradication rates, whereas those with resistant strains experienced significantly lower success. The most pronounced decline in treatment effectiveness was observed in cases involving clarithromycin resistance and multidrug resistance.

Table 2. Treatment outcomes based on antibiotic resistance status

Resistance Status	Patients (n)	Eradication Success (%)	Failure (%)
No resistance	41	90.2%	9.8%
Clarithromycin resistance only	23	65.2%	34.8%
Metronidazole resistance only	28	71.4%	28.6%
Multi-drug resistance	16	50.0%	50.0%

Table 2 clearly demonstrates the clinical consequences of antibiotic resistance. Patients without detectable resistance achieved a high eradication rate exceeding 90%, confirming the effectiveness of standard therapy in favourable conditions. In contrast, the presence of clarithromycin resistance resulted in a marked reduction in treatment success, with nearly one-third of patients failing therapy. Metronidazole resistance also contributed to lower success rates, although its impact was less pronounced when present alone. The most critical finding is the dramatic decline in eradication success among patients with multidrug-resistant strains, where treatment failure reached 50%. This highlights the urgent need for tailored therapeutic strategies in such cases.

Further statistical analysis confirmed that antibiotic resistance was a strong and independent predictor of treatment failure. The association between clarithromycin resistance and unsuccessful eradication was particularly significant, with resistant cases showing more than a threefold increase in failure risk. Multidrug resistance amplified this risk even further, indicating a cumulative negative effect when multiple antibiotics lose their efficacy simultaneously. Overall, the results provide compelling evidence that antibiotic resistance is not merely a laboratory finding but a decisive factor that shapes real clinical outcomes. The observed patterns underscore the limitations of empirical therapy and emphasise the importance of adapting treatment strategies based on resistance profiles. These findings are consistent with recent global reports, which also highlight the growing burden of resistance and its direct impact on eradication success rates [11].

5. Discussion

The findings of this study provide a clear and clinically meaningful picture of how antibiotic resistance in Helicobacter pylori continues to reshape treatment outcomes. The observed resistance rates, particularly for clarithromycin and metronidazole, are not isolated phenomena but reflect a broader global trend that has been steadily intensifying over the past decade. What becomes evident from these results is that the traditional reliance on empirical therapy is increasingly difficult to justify in settings where resistance prevalence exceeds clinically acceptable thresholds.

One of the most striking observations in this study is the strong association between clarithromycin resistance and treatment failure. Patients infected with resistant strains demonstrated a substantial reduction in eradication success, which aligns closely with findings from recent international studies. Clarithromycin resistance is primarily driven by

point mutations in the 23S rRNA gene, which interfere with antibiotic binding and render standard therapy ineffective. As resistance levels approach or exceed 15–20% in many regions, clinical guidelines now recommend avoiding clarithromycin-based regimens unless susceptibility is confirmed [14]. The results presented here reinforce that recommendation and highlight the risks of continuing outdated empirical approaches.

Metronidazole resistance, although more prevalent, showed a comparatively moderate impact on treatment outcomes when present alone. This observation can be explained by the partial reversibility of metronidazole resistance under certain physiological conditions, as well as the possibility of overcoming resistance through higher doses or prolonged therapy. Nevertheless, its high prevalence remains clinically relevant, particularly when combined with resistance to other antibiotics. In such cases, the cumulative effect significantly compromises eradication success, as demonstrated by the markedly lower outcomes in patients with multidrug-resistant strains [15].

Another important aspect of this study is the relatively low resistance observed for amoxicillin. This finding is consistent with global data suggesting that resistance to amoxicillin remains rare due to the essential role of its target sites in bacterial cell wall synthesis. As a result, amoxicillin continues to serve as a reliable backbone in most eradication regimens. However, its effectiveness is ultimately dependent on the susceptibility of companion antibiotics, which underscores the importance of combination therapy strategies [16].

From a broader perspective, the results highlight the growing gap between recommended treatment strategies and real-world clinical practice. In many developing regions, including Central Asia, routine susceptibility testing is not widely available, leading clinicians to rely heavily on empirical therapy. This approach, while practical, often fails to account for local resistance patterns and may contribute to repeated treatment failures. The findings of this study suggest that even a moderate level of resistance can significantly alter treatment outcomes, emphasising the need for region-specific data to guide therapeutic decisions. The clinical implications are substantial. First, there is a clear need to move toward more individualised treatment approaches, where therapy is tailored based on known resistance patterns whenever possible. Second, the increasing prevalence of multidrug resistance calls for the reconsideration of current first-line regimens, potentially incorporating alternative combinations or novel therapeutic strategies. Finally, the importance of antimicrobial stewardship cannot be overstated. Reducing unnecessary antibiotic use and improving prescribing practices are essential steps in slowing the progression of resistance.

Despite its strengths, this study has certain limitations that should be acknowledged. The sample size, although sufficient to demonstrate significant associations, may not fully capture the variability of resistance patterns across different populations. Additionally, the lack of molecular analysis limits the ability to identify specific genetic mechanisms underlying resistance. Future research incorporating genomic techniques and larger, multicenter cohorts would provide a more comprehensive understanding of this issue. In conclusion, the discussion of these findings underscores a critical reality: antibiotic resistance in Helicobacter pylori is no longer a secondary concern but a central factor determining treatment success. Addressing this challenge requires a shift in both clinical practice and research priorities, with a stronger emphasis on evidence-based, locally adapted strategies.

6. Conclusion

The results of this study clearly demonstrate that antibiotic resistance in Helicobacter pylori has become a decisive factor influencing the success of eradication therapy. The declining effectiveness of commonly used antibiotics, particularly clarithromycin and metronidazole, reflects a shift in the clinical landscape where traditional treatment approaches are no longer universally reliable. What was once considered a predictable and manageable infection now requires more careful evaluation and strategic decision-making.

One of the key insights from this analysis is that treatment success is no longer determined solely by adherence to standard protocols but is heavily dependent on the infecting strain's resistance profile. Patients with susceptible strains continue to respond well to conventional therapy, while those with resistant or multidrug-resistant infections face significantly higher risks of treatment failure. This contrast highlights the limitations of

empirical therapy in environments where resistance rates are steadily increasing. Moving forward, a more individualised approach to treatment is essential. Whenever possible, therapeutic decisions should be guided by local resistance patterns or, ideally, by direct susceptibility testing. At the same time, efforts to improve antibiotic stewardship must be strengthened to slow the progression of resistance and preserve the effectiveness of existing.

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