FREQUENCY OF *HELICOBACTER PYLORI* INFECTION AND ASSOCIATED FACTORS AMONG PATIENTS WITH PEPTIC ULCER DISEASE AT HOSPITAL 199, DA NANG, VIET NAM

TỶ LỆ NHIỄM *HELICOBACTER PYLORI* VÀ CÁC YẾU TỐ LIÊN QUAN CỦA NGƯỜI BỆNH VIÊM LOÉT DẠ DÀY, TÁ TRÀNG TẠI BỆNH VIỆN 199, ĐÀ NẴNG, VIỆT NAM

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Abstract - A total of 285 patients presenting to the Gastroenterology Department at Hospital 199, Da Nang, were enrolled in this study. The frequency of *Helicobacter pylori* infection was 35.4%, by the rapid urease test (CLO test). Infection rates did not differ significantly by sex, age, education, or occupation ($p > 0.05$). Recent use of other gastrointestinal medications (excluding prohibited antibiotics and PPIs) within four weeks before endoscopy was associated with reduced *H. pylori* detection (OR = 0.53; $p = 0.023$). A significant difference in infection rate was observed between treatment-naïve patients and those with prior treatment ($p = 0.011$). *H. pylori* infection was significantly associated with gastritis ($p = 0.041$). Although ulcer differences were not statistically significant ($p > 0.05$), odds ratios suggested a clinically relevant trend, with *H. Pylori*-positive individuals having approximately 2.11- and 2.78-fold higher odds of gastric and duodenal ulcers, respectively, compared with non-infected patients.

Key words - *Helicobacter pylori*; Gastroenterology; Gastroduodenal; Hospital 199, Danang, Vietnam

Tóm tắt - Nghiên cứu phân tích trên 285 người bệnh đến khám tại khoa Nội tiêu hóa Bệnh viện 199 Đà Nẵng. Tỷ lệ nhiễm *Helicobacter pylori* là 35,4% bằng phương pháp test nhanh urease (CLO test). Tỷ lệ nhiễm *Helicobacter pylori* và các yếu tố nhân khẩu học như giới tính, độ tuổi, học vấn và nghề nghiệp không tạo ra sự khác biệt có ý nghĩa thống kê ($p > 0,05$). Sử dụng các thuốc tiêu hóa khác (không tính kháng sinh và PPI đã bị loại trừ trong tiêu chuẩn nghiên cứu) trong vòng 4 tuần trước khi nội soi làm giảm tỉ lệ phát hiện *H. pylori* (OR = 0,53; $p = 0,023$). Tỷ lệ nhiễm *H. pylori* của người bệnh đã từng điều trị trước đó và không điều trị là khác biệt có ý nhĩa thống kê ($p = 0.011$). Sự khác biệt có ý nghĩa thống kê giữa *H. pylori* và bệnh lý viêm dạ dày ($p = 0,041$). Với bệnh lý loét, dù chưa đạt ý nghĩa thống kê ($p > 0,05$), OR gợi ý xu hướng tăng nguy cơ: loét dạ dày (OR = 2,11) và loét tá tràng (OR = 2,78).

Từ khóa - Vi khuẩn *Helicobacter pylori*; Nội tiêu hóa; Dạ dày-tá tràng; Bệnh viện 199 Đà Nẵng, Việt Nam

1. Introduction

Helicobacter pylori (*H. pylori*) is a motile, Gram-negative spiral bacterium that primarily colonizes the gastric mucosal layer in humans. It is recognized as the most prevalent agent of chronic infection globally, affecting approximately 50% of the world's population [1]. *H. pylori* plays a critical role in gastrointestinal pathology, serving as the primary etiological factor for chronic gastritis and peptic ulcer disease. Furthermore, it is a major risk factor for gastric adenocarcinoma and Mucosa-Associated Lymphoid Tissue (MALT) lymphoma [2]. Recent evidence suggests that the eradication of *H. pylori* not only facilitates the healing of inflammatory lesions but also constitutes the most vital primary prevention strategy for reducing gastric cancer mortality within the community [3]. The bacterium thrives in the hostile acidic environment of the stomach through the localized neutralization of pH, a process mediated by the urease enzyme. The pathogenesis of *H. pylori* is highly complex, involving a multi-faceted interaction between bacterial virulence factors, host immune responses, and environmental variables. Key virulence factors, such as cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA), are decisive in inducing epithelial cell damage and stimulating systemic inflammatory responses via the secretion of pro-inflammatory cytokines, including IL-1β, IL-6, and IL-8 [4]. Numerous domestic and international studies have elucidated the correlation between *H. pylori* prevalence and various factors, such as clinical symptomatology, lifestyle, residential location, educational attainment, and medical history. In Vietnam, research indicates an increasing trend in infection rates among the working-age population, peaking within the 40-55 age demographic. According to Huong et al., this surge is associated with social behavioral patterns and the frequency of communal dining among middle-aged individuals. Conversely, while the prevalence in children remains lower, it exhibits an upward trajectory due to intra-familial transmission [5]. Furthermore, a 2024 study by Tuan et al. revealed significant disparities based on residence and living conditions. Inhabitants of suburban areas or provinces characterized by high population density and limited water and sanitation facilities exhibit positive rates 15-20% higher than those in major

metropolitan centers like Hanoi or Ho Chi Minh City. This disparity is closely linked to occupational factors; manual laborers and industrial workers show a disproportionately higher prevalence compared to office-based professionals [6]. Research by Bich et al. further identified low educational levels and unstable employment as independent risk factors, with the Odds Ratio (OR) for infection among unskilled laborers being 1.85 times higher than that of individuals with tertiary education or higher [7]. Regarding gender and marital status, Vietnamese data from the 2023-2024 period indicate a higher prevalence in males, largely attributed to high-risk behaviors such as tobacco use and alcohol consumption at informal outdoor eateries [8]. Notably, marital status plays a pivotal role in maintaining the reservoir of infection. The cultural habit of sharing utensils and dipping sauces within Vietnamese households results in a high cross-infection rate between spouses (approximately 62.5%), establishing the family as the most critical epidemiological unit for bacterial control [9]. The correlation between demographic characteristics and current *H. pylori* prevalence clearly reflects the impact of socio-economic conditions on public health status. Regarding age, cross-sectional studies conducted between 2022 and 2024 confirm a "cumulative temporal effect," where infection rates progressively increase with age, peaking in the 40-60 age group. In conclusion, *H. pylori* is not only a causative agent of chronic gastroduodenal diseases but also a primary risk for gastric malignancy, representing a substantial disease burden. Although medical advancements have clarified its pathogenesis and virulence mechanisms, community infection rates remain high due to barriers related to lifestyle habits, sanitary conditions, and intra-familial transmission. Specifically, variations in demographic characteristics-such as age, occupation, and education-among patients admitted to hospitals present unique challenges for preventive efforts. Consequently, this study was conducted to determine the current status of infection and analyze specific risk factors. The findings aim to provide a scientific foundation for optimizing screening strategies, prevention, eradication therapy, and health education tailored to patient populations in Da Nang and across Vietnam.

2. Subjects and methods

2.1. Subjects

The study population comprised patients presenting with symptomatic gastroduodenal inflammation or peptic ulcer disease who were indicated for gastrointestinal endoscopy. The recruitment was conducted at the Department of Gastroenterology, Hospital 199, Da Nang City, Vietnam, from August 2025 to February 2026.

2.2. Methods

2.2.1. Study design

This study used a cross-sectional design and a convenience sample of 285 patients. Data were collected using structured interview questionnaires based on pre-specified variables. The sample size was determined using the following formula, and based on a previously reported prevalence of 24.6% [10], a sample size of 285 cases was calculated. Where: $Z_{1-\alpha/2}$=1.96 (corresponding to a 95% confidence level). p = 0.246 (reported prevalence). d = 0.05 (margin of error).

Inclusion criteria: Male and female patients who provided informed consent and presented with one or more clinical symptoms-including epigastric pain, eructation, pyrosis, or abdominal distension-admitted to 199 Hospital and indicated for endoscopy.

Exclusion criteria: Patients with active gastrointestinal hemorrhage or gastric malignancy, and individuals who had received antibiotics (e.g., amoxicillin, tinidazole, bismuth) or proton pump inhibitors (PPIs) within the four weeks preceding enrollment.

2.2.2. Gastroscopy

Upper gastrointestinal endoscopy was performed using a flexible video endoscope. The procedure involved a systematic examination of the esophageal, gastric, and duodenal mucosa to identify macro-pathological lesions. Gastritis was diagnosed based on endoscopic features such as erythema, edema, and mucosal atrophy according to the Updated Sydney System. Peptic ulcers were defined as a mucosal defect of at least 5 mm in diameter with perceptible depth.

2.2.3. Detection of H. pylori

The prevalence of *H. pylori* was determined using the rapid urease test (CLO test). This indirect diagnostic assay detects urease activity on the gastric mucosa. Urease secreted by *H. pylori* hydrolyzes urea into ammonia (NH_3) and carbon dioxide (CO_2), leading to alkalinization of the medium. The test uses phenol red as a pH indicator, which changes from yellow to pink/red as pH increases. For optimal accuracy, biopsy specimens were obtained from both the gastric antrum and the gastric body. Diagnostic thresholds were defined as follows: Urease concentration 100 UI/mL: positive within 10-20 seconds; Urease concentration 20 UI/mL: positive within 1-2 minutes and Urease concentration 4 UI/mL: positive within 10-15 minutes.

2.2.4. Data analysis

Data were analyzed using standard medical statistical methods with SPSS (Statistical Package for the Social Sciences) version 20.0. Categorical variables are presented as numbers and percentages. Continuous variables are reported as mean ± standard deviation (SD). Comparisons between two proportions were performed using the chi-square test or Fisher's exact test as appropriate. A two-tailed p-value < 0.05 was considered statistically significant.

2.3. Limitations

This study was conducted on a symptomatic patient population at a single hospital; therefore, the observed *H. pylori* infection rate may be higher than that of the general population and may not be representative of the entire region. Future multicenter or community-based studies are essential to provide a more comprehensive overview.

2.4. Ethical considerations

The study was conducted in accordance with ethical principles for research involving human subjects, ensuring confidentiality and informed consent. All patient data were anonymized and reported only in aggregate form to protect individual privacy. The study protocol received formal approval from the Biomedical Ethics Committee of the University of Da Nang - School of Medicine and Pharmacy (Approval No. 70/TYD-HĐYS).

3. Results and Discussion

3.1. Study population characteristics

A total of 285 patients were included in the analysis. As shown in Table 3.1, the prevalence of *H. pylori* infection was 35.4% (101/285). This rate is lower than figures previously reported by Tung et al. in Central Vietnam (60-70%) [11] and the global pooled prevalence of 44.3% reported by Li et al. [12]. The discrepancy may be partly explained by the demographic profile of our cohort: most participants resided in urban areas (82.1%), and more than half had tertiary education or higher (54.7%). These subgroups generally have better access to healthcare and greater awareness of food safety and hygiene, which may reduce environmental transmission risk. Nonetheless, the observed prevalence indicates a substantial community burden of *H. pylori*, consistent with Huy, who described the complex epidemiology of gastroduodenal diseases in Vietnam during the current decade [13]. Regarding associated factors, neither sex (p = 0.911) nor mean age (43.15 ± 16.5 years) was significantly associated with *H. pylori* infection. These findings are in agreement with Huong et al. in Northern Vietnam [5], suggesting relatively uniform exposure across genders and age groups within the working-age population, which is characterized by high levels of social interaction and communal dining. Although some studies have highlighted the contribution of poor rural sanitation to higher infection rates [6], our data (p = 0.321) indicate that transmission via oral-oral or fecal-oral routes remains relevant in high-density urban settings. Marital status was also not associated with infection (p = 0.177), despite household environments being recognized as potential reservoirs for transmission [9]. Educational level (p = 0.499) and occupational category (p = 0.106) likewise showed no significant differences in *H. pylori* positivity. This may reflect homogenized exposure risks in contemporary urban and workplace environments in Southeast Asia, where collective settings and crowded public spaces facilitate transmission across professional and educational strata [14]. Overall, these results suggest that *H. pylori* infection in our study population is relatively widespread and largely independent of the individual demographic variables examined. The absence of significant associations may be influenced by the sample size and the single-center design of the study. Nevertheless, the findings support the need for broad-based preventive measures that target the general population rather than focusing exclusively on specific demographic subgroups.

Table 1. *General characteristics of the study population and their association with H. pylori infection (n = 285)*

Characteristics		Total n (%)	*H. pylori* (+) (%)	p-value
Age (Mean ± SD): 43.15 ± 16.5				
Gender	Male	137 (48.1)	49 (48.5)	0.911
	Female	148 (51.9)	52 (51.5)	
Residence	Rural/Mountainous	51 (17.9)	15 (14.9)	0.321
	Urban	234 (82.1)	86 (85.1)	
Marital status	Single	74 (26.0)	31 (30.7)	0.177
	Married	211 (74.0)	70 (69.3)	
Education Level	Primary/Lower Secondary	62 (21.8)	20 (19.8)	0.499
	Upper Secondary	67 (23.5)	21 (20.8)	
	Tertiary (College/University)	156 (54.7)	60 (59.4)	
Occupation	Civil servants/ Office workers	47 (16.5)	13 (12.9)	0.106
	Military/Police personnel	39 (13.7)	19 (18.8)	
	Pupils/Students	20 (7.0)	11 (10.9)	
	Workers	58 (20.4)	19 (18.8)	
	Business/Self-employed	55 (19.3)	20 (19.8)	
	Unemployed/Homemakers	66 (20.2)	19 (18.8)	

3.2. Association between H. pylori infection and lifestyle characteristics and medical history

The results presented in Table 2 indicate significant variations in infection rates based on medication history and prior gastric pathologies. In this study, lifestyle factors such as smoking (OR = 0.78; p = 0.429) and alcohol consumption (OR = 1.09; p = 0.712) were not significantly associated with *H. pylori* infection. This finding is consistent with Hanh et al. in Vietnam [15], suggesting that, when considered independently, these behaviors are not direct risk factors in an endoscopic study population. By contrast, a 2025 multicenter study in Southeast Asia [17] reported that alcohol consumption increases the risk of mucosal damage and thus facilitates bacterial colonization (OR = 2.15). This discrepancy may be due to our study sample, which consisted of patients already presenting with gastrointestinal symptoms. Regarding family history, cohabitation with *H. pylori*-infected individuals was not associated with a significantly increased risk (p = 0.429), which contradicts the commonly held view of intra-familial transmission. This finding may reflect improved hygienic eating practices (e.g., using separate chopsticks and bowls) in recent years, as noted by Lam et al. [16]. Medical and treatment history emerged as the most statistically significant group of variables in this study. Notably, the use of other gastrointestinal medications (excluding the prohibited antibiotics and PPIs) prior to the 4-week enrollment window significantly influenced the *H. pylori* detection rate (OR = 0.53; p = 0.023). This suggests that various pharmacological interventions, even those not strictly listed in standard exclusion criteria, may still affect the gastric microenvironment and the sensitivity of the rapid urease test. This outcome aligns perfectly with the latest 2025 international clinical guidelines [18], which emphasize that the administration of acid suppressants or antibiotics

before testing can lead to false-negative results due to the temporary inhibition of bacterial growth. Notably, the group of patients with a history of peptic ulcer disease had a lower infection rate compared to those without such a history (41.6% vs. 59.8%, p = 0.003). This may be explained by the "treatment effect": patients with a medical history often have undergone previous eradication therapies or used supportive medications, which reduce bacterial density. This finding is consistent with observational studies at major hospitals in Vietnam, where reinfection rates are gradually declining due to effective eradication protocols [15, 16]. The study results indicate a statistically significant association between a history of prior *H. pylori* treatment and current infection status (p = 0.011). An odds ratio (OR) of 0.52 (95% CI: 0.32-0.86) suggests that previous treatment history acts as a protective factor, correlating with an approximately 48% reduction in the risk of testing positive for the bacterium compared to the patients group with no prior treatment history.

Table 2. *Association between H. pylori infection status and lifestyle characteristics and medical history*

Characteristics	Category	*H. pylori* (+) N (%)	*H. pylori* (-) N (%)	OR (95% CI)	p – value
Lifestyle Habits					
Smoking	No	82 (81.2%)	142 (77.2%)	0.78 (0.42-1.43)	0.429
	Yes	19 (18.8%)	42 (22.8%)		
Alcohol consumption	No	46 (45.5%)	88 (47.8%)	1.09 (0.67-1.78)	0.712
	Yes	55 (54.5%)	96 (52.2%)		
Family History					
Living with an *H. pylori*–infected person.	No	70 (69.3%)	119 (64.7%)	0.81 0.482-1.36	0.429
	Yes	31 (30.7%)	65 (35.3%)		
Medical and Treatment History					
Medication use (4 weeks prior to endoscopy)	No	76 (75.2%)	114 (62.0%)	0.53 (0.31-0.92)	**0.023**
	Yes	25 (24.8%)	70 (38.0%)		
History of Peptic Ulcer Disease (PUD)	No	59 (58.4%)	74 (40.2%)	0.47 (0.29-0.78)	**0.003**
	Yes	42 (41.6%)	110 (59.8%)		
Previously treated for *H. pylori*	No	84 (45.7%)	62 (61.4%)	0.52 (0.32-0.86)	**0.011**
	Yes	100 (54.3%)	39 (38.6%)		

3.3. Association between H. pylori infection and gastroduodenal diseases

The analytical results shown in Table 3 reveal a complex relationship between *Helicobacter pylori* infection and gastroduodenal lesions. In the gastritis subgroup, the frequency of inflammation was higher among *H. pylori*-negative patients (85.9%) compared to those who were *H. pylori*-positive (76.2%; p = 0.041; OR = 0.52). While *H. pylori* is globally recognized as a primary etiological factor for gastritis, this apparent inverse association in our cohort likely reflects a selection bias or the influence of unmeasured confounders. Many patients in our setting frequently self-medicate with over-the-counter acid suppressants or NSAIDs prior to clinical evaluation. These medications can lead to false-negative CLO test results or cause non-infectious gastric mucosal injury. Furthermore, factors such as alcohol consumption and psychological stress may predominate as causes of gastritis in the *H. pylori*-negative group, thereby skewing the observed distribution of inflammation [2, 8, 10, 13, 21]. It is consistent with the report by Phuong et al. in Vietnam [19] and with research on gastric microbiota alterations and the role of chemical stimuli in maintaining chronic inflammation [10, 25]. Regarding organic ulcerative lesions, the odds ratios for gastric and duodenal ulcers in the *H. pylori*-infected group were 2.11 and 2.78, respectively. However, these associations did not reach statistical significance (p > 0.05) and were characterized by wide confidence intervals. These findings suggest a potential association that should be interpreted with caution due to the limited sample size in these subgroups. This trend aligns with biological evidence on the direct role of cagA-related virulence factors and proteolytic enzymes secreted by *H. pylori* in disrupting the mucosal barrier and promoting ulcer formation, as well as with the Southeast Asian meta-analysis by Arisaka et al. [17] and the Maastricht VI/Florence consensus [1]. The lack of statistical significance may reflect limited sample size within the ulcer subgroups rather than evidence refuting the etiologic role of *H. pylori*; this interpretation is also consistent with national guidelines and consensus statements from 2024-2025 [18, 20].

Furthermore, the similar prevalence of combined inflammatory–ulcerative conditions between the two groups (11.9% vs. 10.3%; p = 0.687) may indicate a rising proportion of non-*H. pylori* (Non-Hp) cases associated with modern lifestyle factors, a point emphasized by the Vietnam Association of Gastroenterology and by recent authors such as Jn et al. [20, 21]. In summary, while gastritis is often iniitiated or sustained by non-infectious factors, *H. pylori* remains a central etiological agent in the development of gastroduodenal ulcers. Therefore, management should combine targeted bacterial eradication with interventions addressing modifiable non-infectious risk factors, in line with national and international recommendations [1, 18, 20].

Table 3. *Association between H. pylori infection and gastroduodenal diseases*

Characteristics	Category	*H. pylori* (+) N (%)	*H. pylori* (-) N (%)	OR (95% CI)	p – value
Gastritis	No	24 (23.8%)	26 (14.1%)	0.52 (0.28-0.98)	**0.041**
	Yes	77 (76.2%)	158 (85.9%)		
Gastric Ulcer	No	88 (87.1%)	172 (93.5%)	2.11 (0.92-4.83)	0.070
	Yes	13 (12.9%)	12 (6.5%)		
Duodenal Ulcer	No	98 (97.0%)	182 (98.9%)	2.78 (0.45-16.95)	0.247
	Yes	3 (3.0%)	2 (1.1%)		
Combined Gastroduodenal Ulceration	No	89 (88.1%)	165 (89.7%)	1.17 (0.54-2.52)	0.687
	Yes	12 (11.9%)	19 (10.3%)		

4. Conclusion

Based on the results of this study, the following conclusions are drawn:

The mean age of the study population was 43.15 ± 16.5 years. The prevalence of *Helicobacter pylori* infection, determined by the rapid urease test, was 35.4%. No statistically significant differences in infection prevalence were observed across examined demographic variables

(sex, age group, area of residence, educational level, and occupation) ($p > 0.05$), suggesting that bacterial exposure risk is broadly distributed within the patient population of the study area.

Recent use of other gastrointestinal medications (excluding the prohibited antibiotics and PPIs) prior to the 4-week enrollment window significantly influenced the *H. pylori* detection rate (OR = 0.53; $p = 0.023$).

Patients with a history of peptic ulcer disease (PUD) had a lower infection rate than those without such a history (41.6% vs. 59.8%; $p = 0.003$). In contrast, lifestyle factors, including smoking and alcohol consumption, were not clearly associated with *H. pylori* infection status in this cohort.

A statistically significant association was identified between prior *H. pylori* treatment and current infection status ($p = 0.011$). The odds ratio of 0.52 (95% CI: 0.32–0.86) indicates that prior treatment reduced the odds of a positive test by approximately 48% compared with treatment-naïve patients. Nevertheless, clinical data also showed that 54.3% of currently infected patients had a history of prior treatment, a proportion higher than that of previously treated patients who were currently negative (38.6%), suggesting incomplete eradication or reinfection in a substantial subset.

H. pylori infection was significantly associated with gastritis ($p = 0.041$) and was linked to increased odds of ulcer disease, with ORs of approximately 2.11 for gastric ulcer and 2.78 for duodenal ulcer.

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