Vol.4, No.7, Page 2390- 2398, 2026 https://doi.org/10.59628/jast.v4i7.2963 ISSN 2958-9568 JAST

SUN PUBLISHER

مجلة جامعة صنعاء للعلوم التطبيقية والتكنولوجيا

Sana'a University Journal of Applied Sciences and Technology

https://journals.su.edu.ye/index.php/jast/

Determinants and Clinical Manifestations of Helicobacter pylori Infection among Gastrointestinal Patients in Dhamar, Yemen: A Hospital-Based Cross-Sectional Study

Sara Mohammed Mayas[1,2*], Salwa H. Alkhyat[1], Maged Ahmed Al-Garadi[3] and Aref Noman[4]

[1]**Department of Biological Sciences, Faculty of Sciences, Sana'a University, Sana'a, Yemen,**
[2]**Department of Microbiology Section, Biology, Faculty of Sciences, Dhamar University, Dhamar, Yemen,**
[3]**Department of Microbiology, Faculty of Veterinary Medicine, Dhamar University, Dhamar, Yemen,**
[4]**Department of Microbiology and Immunology, Faculty of Medicine and Health Sciences, Sana'a University, Sana'a, Yemen**

*Corresponding author: s.mayas@su.edu.ye

Abstract

Background: *Helicobacter pylori* infection remains a major global public health challenge, particularly in low-resource settings where disease burden remains high. Despite its clinical importance, data regarding associated risk factors and symptom patterns remain limited in several populations, including Yemen.

Objective: This study aimed to investigate the prevalence of *H. pylori* infection and evaluate associated factors and clinical symptoms among gastrointestinal patients attending a tertiary healthcare center in Dhamar.

Methods: A hospital-based cross-sectional study was conducted using a quota-based sampling approach. *H. pylori* infection was diagnosed using a stool antigen test. Data were analyzed using univariate statistical analyses followed by multivariable binary logistic regression to identify independent predictors while controlling for potential confounding variables..

Results: The study demonstrated a high prevalence of *H. pylori* infection among the investigated population. Although univariate analysis initially identified several significant sociodemographic associations, multivariable logistic regression revealed that age >30 years was the only statistically significant independent predictor associated with infection (AOR = 0.086; 95% CI: 0.017–0.437; P = 0.003). Clinical manifestations, including upper abdominal pain (98.0%), nausea (97.6%), and vomiting (96.9%), showed significant associations with *H. pylori* positivity (P < 0.001). In contrast, lifestyle and dietary variables, including hot pepper consumption, did not retain significance following multivariable adjustment

Conclusions: *H. pylori* infection was highly prevalent and independently associated with older age and gastrointestinal symptoms. These findings provide preliminary epidemiological evidence and support the need for future longitudinal studies using representative sampling and confirmatory diagnostic methods.

Article Info

Keywords:
Helicobacter pylori, prevalence, associated factors, gastrointestinal symptoms, epidemiology, stool antigen test, Yemen, cross-sectional study.

Article History:
Received: 31-March-2026,
Revised: 05-May-2026,
Accepted: 14-June-2026,
Published: 28 July 2026.

1. INTRODUCTION

Helicobacter pylori colonizes the gastric mucosal lining and is one of the most widespread bacterial infections globally, affecting more than half of the world's population. It is strongly implicated in a spectrum of gastrointestinal diseases, most notably chronic gastritis, and may progress to severe complications, such as peptic ulcer disease. *H. pylori* is recognized as the most significant established risk factor for gastric cancer and mucosa-associated lymphoid tissue (MALT) lymphoma, conditions that collectively contribute substantially to global cancer-related mortality. In this context, the International

Agency for Research on Cancer (IARC) has classified *H. pylori* as a Group I carcinogen. Although eradication therapy has been associated with a reduced risk of gastric cancer in clinical settings, definitive population-based evidence remains limited [1, 2]

A large proportion of infected individuals remain asymptomatic; however, others develop a wide range of gastrointestinal manifestations, including persistent epigastric burning pain, dyspepsia, bloating, flatulence, nausea, vomiting, and anorexia. A smaller but clinically significant proportion of infected individuals may ultimately develop gastric malignancies

[3, 4].

The prevalence of *H. pylori* infection varies widely across populations and is strongly influenced by a complex interaction between socioeconomic, environmental, and host-related factors [5]. Infection is typically acquired during childhood and may persist for a lifetime. Higher prevalence rates are consistently observed among children living in poverty, overcrowded households, and regions with poor sanitation and limited access to clean water [6]. In many developing countries, prevalence rates may reach 80–90%, reflecting the combined effects of low socioeconomic status, inadequate hygiene, overcrowding, and consumption of contaminated or unboiled water, particularly in rural areas. In contrast, industrialized countries have experienced a marked decline in prevalence, largely attributed to improved sanitation, hygiene practices, and living standards [7].

Transmission is believed to occur primarily via the fecal–oral, oral–oral, and gastro–oral routes [8]. The frequent clustering of infections within households supports the possibility of intrafamilial transmission or shared exposure to common environmental sources of contamination [9].

An accurate diagnosis of *H. pylori* infection is essential for the effective management of gastroduodenal diseases. Multiple diagnostic modalities have been developed, each with distinct advantages and limitations. These methods are broadly categorized into invasive and non-invasive methods. Invasive techniques, including endoscopic biopsy, histopathology, rapid urease testing, culture, and polymerase chain reaction (PCR), provide a definitive diagnosis but require specialized infrastructure and resources [10]. Noninvasive methods include the urea breath test (UBT), serological assays for *H. pylori* IgG antibodies, and stool antigen testing (SAT). Although non-invasive tests generally demonstrate high sensitivity and specificity, each has limitations: UBT accuracy may be reduced in patients receiving proton pump inhibitors or those with altered gastric anatomy, while serological tests cannot distinguish between active and past infections [11].

Despite the recognized global burden of *H. pylori*, data on its associated factors and clinical manifestations in Yemen, particularly in Dhamar City, remain limited. Existing local studies have predominantly focused on descriptive prevalence estimates, with limited analytical assessments of risk factors and symptom associations. Therefore, this study aimed to investigate the factors associated with *H. pylori* infection and its clinical manifestations using stool antigen testing among patients with gastrointestinal disorders attending a tertiary care center in Dhamar. The findings are expected to provide preliminary evidence to guide future large-scale population-based investigations.

2. MATERIALS AND METHODS

2.1. Study setting

Owing to the exploratory nature of the study and resource constraints in the local setting, a convenience sample of 96 patients was recruited. **Inclusion criteria:** adult gastrointestinal patients (≤ 17 years). **Exclusion criteria:** Patients who had used antibiotics or proton pump inhibitors (PPIs) within the four weeks prior to stool collection were excluded to prevent false-negative results.

2.2. Specimen collection & storage

Fresh fecal samples were collected in stool sample collection containers. A minimum of 1–2 mL of liquid stool or 1–2 g of solid stool was collected. All stool samples were tested using the SD BIOLINE *H. pylori* Antigen Rapid Test kit (STANDARD DIAGNOSTIC, INC., Korea). The test results were obtained within 10–15 minutes, with a performance sensitivity of 98.4% and specificity of 100%. **(STANDARD DIAGNOSTIC, INC**. **Korea**).

2.3. Questionnaire

Information was collected using a structured questionnaire. The collected data encompassed the patient's and family's demographics (age, gender, Place of Residence**)**, socioeconomic characteristics (marital status, education level, household population, family income, employment status), personal habits, household hygiene practices, and assessment of clinical symptoms associated with *H. pylori* infection, including upper abdominal discomfort such as epigastric pain, nausea, vomiting, loss of appetite, and headache.

2.4. Statistical analysis

Statistical analyses were conducted using SPSS software (version 21). Data from the questionnaire were analyzed using appropriate statistical methods; specifically, the chi-square test was applied to examine the association between *H. pylori* infection and selected associated factors. Statistical significance was defined as a P-value < 0.05, and odds ratios (ORs) were calculated along with their corresponding 95% confidence intervals

(CIs).

2.5. Ethical considerations

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Thamar University (validation number: TUMEC-21022).

3. RESULTS

3.1. Sociodemographic and Behavioral Factors Associated with *H. pylori* Infection

A total of 96 participants were recruited for the study, with a mean age of 34.25 ± 14.82 years. As shown in Table 1, several factors were significantly associated with *H. pylori* infection status. Regarding sociodemographic characteristics, age was associated with infections. Univariate analysis indicated that younger patients aged ≤ 30 years had significantly lower odds of *H. pylori* infection than older adults aged >30 years (OR = 0.09, 95% CI: 0.03–0.40, P = 0.001).

Social status also played a significant role (P = 0.02), with married individuals having higher odds of infection (82.5%) than single participants (60.6%), with an OR of 3.1. Furthermore, education level was significantly associated with infection (P = 0.04); specifically, university-educated participants had lower odds of being infected (OR = 0.1) than illiterate individuals. In terms of behavioral and dietary habits, the intake of hot pepper was negatively associated with *H. pylori* infection (P = 0.012). Further studies are required to confirm this finding.

However, no statistically significant associations (P > 0.05) were observed between *H. pylori* infection and other studied variables, including sex, household population, place of residence, family income, water type, smoking, khat chewing, dental caries, intake of non-steroid medicines(NSAID), spices, vegetables, fatty foods, and tea/coffee drinking.

3.2. Clinical Symptoms According to *H. pylori* Infection Status

The distribution of various clinical symptoms among the study participants in relation to their *H. pylori* infection status is shown in Table 2.

The analysis focused on the prevalence differences between the infected and non-infected groups. The results revealed that most of the assessed clinical signs were significantly more prevalent in patients who tested positive for *H. pylori*. Specifically, the vast majority of patients presenting with upper stomach pain (98.0%), nausea (97.6%), vomiting (96.9%), loss of appetite (93.9%), and headache (94.1%) were infected with *H. pylori*, showing highly significant statistical differences (P = 0.001 for all) compared to non-infected individuals. General stomach pain also differed significantly (P = 0.004). In contrast, the presence of heartburn did not show a statistically significant difference between *H. pylori-infected* and non-infected (P = 0.2).

3.3. Multivariable Logistic Regression Analysis of Associated Factors

A multivariable binary logistic regression analysis was performed, including variables that showed significance in the univariate analysis (age, social status, education level, and hot pepper intake). After adjusting for confounders, only age remained a statistically significant independent factor associated with *H. pylori* infection (AOR = 0.086; 95% CI: 0.017–0.437, p = 0.003). Participants in the younger age group (17-30 years) were significantly less likely to be infected than those older than 30 years. The other variables (social status, education, and hot pepper) lost their statistical significance, indicating that they were likely confounding factors in the univariate analysis. This suggests that their initial association was likely due to confounding by age or other sociodemographic factors (Table 3).

4. DISCUSSION

Helicobacter pylori infection remains one of the most prevalent chronic bacterial infections worldwide and continues to represent a substantial public health challenge, particularly in developing countries, where socioeconomic and environmental factors facilitate transmission. Understanding the epidemiological determinants of *H. pylori* infection is essential for identifying high-risk populations and developing targeted preventive strategies aimed at reducing the disease burden and improving healthcare outcomes [12]. In the present study, age > 30 years emerged as an independent predictor significantly associated with *H. pylori* infection (AOR = 0.086, *P* = 0.003), suggesting that cumulative lifetime exposure may contribute substantially to the observed infection pattern.

This observation is consistent with previous reports demonstrating a progressive increase in *H. pylori* prevalence with age [13, 14].The age-related increase in infection rates may reflect prolonged exposure to environmental risk factors and increased opportunities for bacterial acquisition over time. However, this finding contrasts with other studies that reported lower infection frequencies among older populations [15]. Such discrepancies may be attributed to differences in population characteristics, geographical settings, diagnostic methodologies, healthcare access, and variations in bacterial eradication practices. Furthermore, our findings differ from those of previous studies that have reported significant associations between infection prevalence and dietary practices, particularly among pediatric populations [16].

Table 1. Sociodemographic and Behavioral Factors Associated with *H. pylori* Infection

Associated Factors	Total (N=96)	*H. pylori* (+) (N = 72) n (%)	*H. pylori* (-) (N = 24) n (%)	OR (95% CI)	P-value
Age group					
(17-30) Young adult	50	29 (58.0%)	21 (42.0%)	0.09 (0.03-0.4)	**0.001***
>30 Old-aged adults	46	43 (93.5%)	3 (6.5%)	Ref.	
Gender					
Male	48	36 (75.0%)	12 (25.0%)	1.00(0.39-2.51)	1.0
Female	48	36 (75.0%)	12 (25.0%)	Ref.	
Social status					
Married	63	52 (82.5%)	11 (17.5%)	3.1 (1.2-7.9)	**0.02***
Single	33	20 (60.6%)	13 (39.4%)	Ref.	
Household population					
≤ 4	7	6 (85.7%)	1 (14.3%)	2.1 (0.2-18.3)	0.8
> 4	89	66 (74.2%)	23 (25.8%)	Ref.	
Room in house					
≤ 3	43	34 (79.1%)	9 (20.9%)	1.5 (0.6-3.8)	0.4
> 3	53	38 (71.7%)	15 (28.3%)	Ref.	
Place of Residence					
Urban	82	59 (72.0%)	23 (28.0%)	0.2 (0.02-1.6)	0.1
Rural	14	13 (92.9%)	1 (7.1%)	Ref.	
Education level					
Illiterate	15	14 (93.3%)	1 (6.7%)	Ref.	
Basic education	26	21 (80.8%)	5 (19.2%)	0.3 (0.03-2.8)	0.5
Secondary	17	14 (82.4%)	3 (17.6%)	0.3 (0.03-3.6)	0.7
University	38	23 (60.5%)	15 (39.5%)	0.1 (0.03-0.9)	**0.04***
Family income					
High	56	40 (71.4%)	16 (28.6%)	0.6 (0.2-1.6)	0.3
Low	40	32 (80.0%)	8 (20.0%)	Ref.	
Water type					
Tap water	48	35 (72.9%)	13 (27.1%)	0.8 (0.3-2.0)	0.6
Non-tap water	48	37 (77.1%)	11 (22.9%)	Ref.	
Smoking					
Yes	21	17 (81.0%)	4 (19.0%)	1.5 (0.5-5.1)	0.5
No	75	55 (73.3%)	20 (26.7%)	Ref.	
Khat chewing					
Yes	60	42 (70.0%)	18 (30.0%)	0.5 (0.2-1.3)	0.1
No	36	30 (83.3%)	6 (16.7%)	Ref.	
Dental caries					
Yes	39	28 (71.8%)	11 (28.2%)	0.7 (0.3-1.9)	0.5
No	57	44 (77.2%)	13 (22.8%)	Ref.	
NSAID					
Yes	68	53 (77.9%)	15 (22.1%)	1.7 (0.6-4.5)	0.3
No	28	19 (67.9%)	9 (32.1%)	Ref.	
Intake of spices					

Yes	86	63 (73.3%)	23 (26.7%)	0.3 (0.04-2.5)	0.4
No	10	9 (90.0%)	1 (10.0%)	Ref.	
Intake of hot pepper					
Yes	64	43 (67.0%)	21 (33.0%)	0.2 (0.06-0.8)	**0.012***
No	32	29 (90.6%)	3 (9.4%)	Ref.	
Intake of vegetables					
Yes	92	69 (75.0%)	23 (25.0%)	1.0 (0.09-10.1)	1.0
No	4	3 (75.0%)	1 (25.0%)	Ref.	
Intake of fat food					
Yes	82	60 (73.2%)	22 (26.8%)	0.5 (0.09-2.2)	0.3
No	14	12 (85.7%)	2 (14.3%)	Ref.	
Tea drinking					
Yes	90	68 (75.6%)	22 (24.4%)	1.5 (0.3-9.0)	0.6
No	6	4 (66.7%)	2 (33.3%)	Ref.	
Coffee drinking					
Yes	66	50 (75.8%)	16 (24.2%)	1.3 (0.4-3.0)	0.8
No	30	22 (73.3%)	8 (26.7%)	Ref.	

Symptomatic (N=24), asymptomatic (N=24), Gastric Cancer (N=24), and healthy (N=24) groups; *CI: Confidence Interval

These inconsistencies suggest that the influence of dietary behaviors on *H. pylori* acquisition may vary according to age group and population-specific environmental context.

The present study also demonstrated no significant association between *H. pylori* infection and demographic or lifestyle variables, including sex, smoking status, khat chewing, and source of drinking water. Similar observations have been reported in previous regional and international studies that found no meaningful relationship between infection status and behavioral or socioeconomic variables [15, 17–20]. The absence of such associations in our cohort may reflect the relatively homogeneous behavioral characteristics of the participants recruited from a hospital-based setting, thereby limiting the variability between groups. Additionally, the strong influence of age as an independent predictor may have overshadowed the contribution of secondary lifestyle factors.

However, these findings are not universally consistent with the literature. Other studies have identified significant relationships between *H. pylori* infection and variables such as sex, smoking habits, khat consumption, and tobacco use [21, 22]. Variations across studies may arise from differences in cultural practices, regional behavioral patterns, sample composition, and study design, highlighting the multifactorial nature of *H. pylori* transmission and the potential interactions between host, environmental, and lifestyle determinants.

Regarding dietary factors, univariate analysis demonstrated an inverse association between *H. pylori* positivity and hot pepper consumption; however, this relationship lost statistical significance after adjustment for potential confounding variables. This finding suggests that the apparent protective effect may not be an independent determinant of infection risk. Previous investigations have proposed that certain dietary components, including capsaicin-containing foods and high-sugar dietary patterns, may influence bacterial growth or gastric microenvironment characteristics that affect colonization dynamics [23]. However, the current study did not identify significant associations between *H. pylori* infection and the consumption of spices, vegetables, coffee, tea, or fatty food. Similar findings have been documented elsewhere, indicating that individual dietary habits alone may exert a limited influence on infection prevalence compared with broader environmental and host-related factors [24].

A notable finding of this study was the strong association between *H. pylori* infection and gastrointestinal symptoms, including upper abdominal pain, nausea, and vomiting ($P = 0.001$). These observations are in agreement with previous reports that identified significant relationships between *H. pylori* positivity and gastrointestinal manifestations in symptomatic patient populations [24, 25]. This association is biologically plausible because *H. pylori* colonization induces chronic gastric mucosal inflammation, which contributes to gastric dysfunction and symptom development [26]. However, the interpretation of these findings should consider the characteristics of the study population. The remarkably high prevalence of gastrointestinal symptoms among participants reflects the symptomatic nature of this hospital-based cohort rather than the epidemiological profile of *H. pylori* infection in the general population, where colonization frequently remains asymptomatic. Therefore, these clinical manifestations should be considered indications for clinical investigation rather than definitive diagnostic markers or direct causal consequences of infection.

Limitations

Several limitations should be considered when interpreting the findings of this study. First, the relatively small sample size ($N = 96$) and recruitment from a single tertiary healthcare center may have reduced the statistical power and limited the external validity of the results. Therefore, caution should be exercised when extrapolating these findings to broader populations. Second, the use of a convenience/quota-based sampling strategy introduced the possibility of selection bias. This approach was primarily adopted because of the limited availability of patients with gastric cancer and concomitant *H. pylori* infection; however, such a recruitment strategy restricts the representativeness of the study population and limits the applicability of the observed prevalence estimates to the wider population of Yemen. Third, the cross-sectional nature of the study permits the identification of associations but does not allow the establishment of temporal or causal relationships between potential risk factors and the development of infection or clinical manifestation. Finally, the diagnosis of *H. pylori* infection was based solely on stool antigen tests. Although this method demonstrates acceptable sensitivity and specificity and is practical for resource-constrained settings, the absence of confirmation using gold-standard diagnostic approaches, such as endoscopic assessment with histopathological or molecular evaluation, may have introduced a degree of diagnostic misclassification in our study.

5. CONCLUSION

The present hospital-based investigation demonstrated a substantial burden of *H. pylori* infection among patients presenting with gastrointestinal disorders and identified age > 30 years as the major independent predictor associated with infection status. While several sociodemographic and behavioral variables initially appeared to be associated with infection, these relationships were not maintained following multivariable adjustment, suggesting that age may exert a more prominent influence within this population. Furthermore, the significant association between *H. pylori* positivity and upper abdominal pain, nausea, and vomiting underscores the potential clinical

Table 2. Prevalence of Clinical Symptoms According to *H. pylori* Infection Status

Clinical signs	Total (N = 96)	*H. pylori* (+) n (%)	*H. pylori* (-) n (%)	P-value
Stomach pain				**0.004***
Yes	70	58 (82.9%)	12 (17.1%)	
No	26	14 (53.8%)	12 (46.2%)	
Upper stomach pain				**0.001***
Yes	50	49 (98.0%)	1 (2.0%)	
No	46	23 (50.0%)	23 (50.0%)	
Heartburn				0.2
Yes	48	41 (85.4%)	7 (14.6%)	
No	48	31 (64.6%)	17 (35.4%)	
Nausea				**0.001***
Yes	42	41 (97.6%)	1 (2.4%)	
No	54	31 (57.4%)	23 (42.6%)	
Vomiting				**0.001***
Yes	32	31 (96.9%)	1 (3.1%)	
No	64	41 (64.1%)	23 (35.9%)	
Loss of appetite				**0.001***
Yes	49	46 (93.9%)	3 (6.1%)	
No	47	26 (55.3%)	21 (44.7%)	
Headache				**0.001***
Yes	51	48 (94.1%)	3 (5.9%)	
No	45	24 (53.3%)	21 (46.7%)	

value of these manifestations as indicators for further diagnostic evaluation. Although the cross-sectional design limits causal inference, these findings provide important epidemiological insights and establish a foundation for future multicenter longitudinal studies employing larger sample sizes and gold-standard diagnostic methods to further elucidate the determinants and clinical outcomes of *H. pylori* infection.

Declarations

Ethical Approval

This article does not contain any studies involving animals or human participants performed by any of the authors. We declare that this manuscript is original and is not currently under consideration for publication elsewhere. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

Conflict of interest

The authors declare no competing interests.

Competing interests

The authors have no financial or proprietary interest in any material discussed in this article.

Authors' contributions

All authors contributed to the study's conception and design. All authors performed the simulations, data collection, and analysis, and commented on the present version of the manuscript. All authors have read and approved the final manuscript.

Funding: No funding was received from any organization for this study.

Availability of data and materials

No datasets are used in the present study.

Consent for publication

The authors confirm that informed consent was obtained for the publication of the data contained in this **article.**

Consent to participate

Informed consent was obtained from all authors.

Declaration of AI-Assisted Technologies

"The authors declare that artificial intelligence (AI) tools were used only for limited purposes, such as language editing, grammar improvement, and formatting assistance. All scientific content, data analysis, interpretations, conclusions, and final manuscript revisions were reviewed and verified by the authors, who took full responsibility for the integrity and originality of the work."

Table 3. Multivariable Logistic Regression Analysis for Factors Associated with *H. pylori* Infection

	Adjusted Odds Ratio (AOR)	95% C.I. for AOR	P-value
Age			
(17–30) Young adult vs. >30 Old-aged	0.086	0.017–0.437	**0.003***
Social status			
Single vs. Married	0.474	0.123–1.823	0.277
Intake of hot pepper			
Yes vs. No	0.267	0.056–1.285	0.100
Education level			
Overall	–	–	0.292

*Statistically significant at $P < 0.05$.

Note: The education variable subcategories were included in the multivariable model but did not show statistical significance as independent factors.

REFERENCES

[1] A. Ali and K. I. AlHussaini, "Helicobacter pylori: A contemporary perspective on pathogenesis, diagnosis and treatment strategies," *Microorganisms*, vol. 12, no. 1, p. 222, 2024. DOI: 10.3390/microorganisms12010222. [Online]. Available: https://doi.org/10.3390/microorganisms12010222.

[2] P. Malfertheiner et al., "Management of helicobacter pylori infection: The maastricht vi/florence consensus report," *Gut*, vol. 71, no. 9, pp. 1724–1762, 2022. DOI: 10.1136/gutjnl-2022-327745. [Online]. Available: https://doi.org/10.1136/gutjnl-2022-327745.

[3] S. Venero-Fernandez et al., "Prevalence of and factors associated with helicobacter pylori infection in preschoolers in havana, cuba: A population-based study," *Revista de Gastroenterol. de Mexico (English Ed.*, vol. 85, no. 2, pp. 151–159, 2020. DOI: 10.1016/j.rgmxen.2019.03.010.

[4] H. Ghazanfar, N. Javed, R. Reina, O. Thartori, A. Ghazanfar, and H. Patel, "Advances in diagnostic modalities for helicobacter pylori infection," *Life*, vol. 14, no. 9, p. 1170, 2024. DOI: 10.3390/life14091170. [Online]. Available: https://doi.org/10.3390/life14091170.

[5] Y. Li, H. Choi, K. Leung, F. Jiang, D. Y. Graham, and W. K. Leung, "Global prevalence of helicobacter pylori infection between 1980 and 2022: A systematic review and meta-analysis," *The Lancet Gastroenterol. & Hepatol.*, vol. 8, no. 6, pp. 553–564, 2023. DOI: 10.1016/S2468-1253(23)00070-5. [Online]. Available: https://doi.org/10.1016/S2468-1253(23)00070-5.

[6] J. Nguyen, K. Kotilea, P. Bontems, and V. Y. Miendje Deyi, "Helicobacter pylori infections in children," *Antibiotics*, vol. 12, no. 9, p. 1440, 2023. DOI: 10.3390/antibiotics12091440. [Online]. Available: https://doi.org/10.3390/antibiotics12091440.

[7] R. Borka Balas, L. E. Meliț, and C. O. Mărginean, "Worldwide prevalence and risk factors of helicobacter pylori infection in children," *Children*, vol. 9, no. 9, p. 1359, 2022. DOI: 10.3390/children9091359. [Online]. Available: https://doi.org/10.3390/children9091359.

[8] M. Duan et al., "Transmission routes and patterns of helicobacter pylori," *Helicobacter*, vol. 28, no. 1, e12945, 2023. DOI: 10.1111/hel.12945. [Online]. Available: https://doi.org/10.1111/hel.12945.

[9] S. Kayali et al., "Helicobacter pylori transmission routes and recurrence of infection: State of the art," *Acta Bio Medica: Atenei Parmensis*, vol. 89, no. 8-S, pp. 72–76, 2018. DOI: 10.23750/abm.v89i8-S.7947. [Online]. Available: https://doi.org/10.23750/abm.v89i8-S.7947.

[10] R. Spagnuolo, G. G. M. Scarlata, M. R. Paravati, L. Abenavoli, and F. Luzza, "Change in diagnosis of helicobacter pylori infection in the treatment-failure era," *Antibiotics*, vol. 13, no. 4, p. 357, 2024. DOI: 10.3390/antibiotics13040357. [Online]. Available: https://doi.org/10.3390/antibiotics13040357.

[11] Y. Wen et al., "Artificial intelligence for the diagnosis of helicobacter pylori infection in endoscopic and pathological tissue images: A systematic review and meta-analysis," *Intell. Med.*, vol. 10, p. 100 244, 2025. DOI: 10.1016/j.ibmed.2025.100244. [Online]. Available: https://doi.org/10.1016/j.ibmed.2025.100244.

[12] A. Elbehiry, E. Marzouk, M. Aldubaib, and A. Abalkhail, "Helicobacter pylori infection: Current status and future prospects on diagnostic, therapeutic and control challenges," *Antibiotics*, vol. 12, no. 2, p. 191, 2023. DOI: 10.3390/antibiotics12020191. [Online]. Available: https://doi.org/10.3390/antibiotics12020191.

[13] R. X. Chen et al., "A survey on helicobacter pylori infection rate in hainan province and analysis of related risk factors," *BMC Gastroenterol.*, vol. 23, no. 1, p. 338, 2023. DOI: 10.1186/s12876-023-02973-3. [Online]. Available: https://doi.org/10.1186/s12876-023-02973-3.

[14] X. Z. Zhou et al., "Large-scale, national, family-based epidemiological study on helicobacter pylori infection in china: The time to change practice for related disease prevention," *Gut*, vol. 72, no. 5, pp. 855–869, 2023. DOI: 10.1136/gutjnl-2022-328965. [Online]. Available: https://doi.org/10.1136/gutjnl-2022-328965.

[15] F. Zhang et al., "Prevalence and associated risk factors of helicobacter pylori infection in the wuwei cohort of northwestern china," *Trop. Med. & Int. Health*, vol. 26, no. 3, pp. 290–300, 2021. DOI: 10.1111/tmi.13517. [Online]. Available: https://doi.org/10.1111/tmi.13517.

[16] H. Ye et al., "Prevalence and risk factors of helicobacter pylori infection in urban children aged 6–13 years in fuzhou, china: A cross-sectional study in a high gastric cancer incidence region," *Front. Pediatr.*, vol. 13, p. 1 457 195, 2025. DOI: 10.3389/fped.2025.1457195. [Online]. Available: https://doi.org/10.3389/fped.2025.1457195.

[17] C. R. G. Mikhail, A. Abd El Maksoud Mohamed, O. G. Shaker, E. El Desouky, and R. H. Shalaby, "Frequency and risk factors of helicobacter pylori infection among dental students: An observational cross-sectional study," *Sci. Reports*, vol. 13, p. 14 264, 2023. DOI: 10.1038/s41598-023-41445-6. [Online]. Available: https://doi.org/10.1038/s41598-023-41445-6.

[18] B. Al-absi, N. Al-Henhen, M. A. W. Almorish, and A. M. Othman, "The relationship between helicobacter pylori infection and iron deficiency anemia in yemeni adults," *Sana'a Univ. J. Appl. Sci. Technol.*, vol. 19, no. 1, pp. 164–169, 2025. DOI: 10.59628/jchm.v19i1.1435. [Online]. Available: https://doi.org/10.59628/jchm.v19i1.1435.

[19] D. Pete, N. R. Salama, J. W. Lampe, M. C. Wu, and A. I. Phipps, "The prevalence and risk factors of helicobacter pylori infection and caga virulence gene carriage in adults in the navajo nation," *Microbiota Health Dis.*, vol. 6, e1007, 2024. DOI: 10.26355/mhd_20247_1007. [Online]. Available: https://doi.org/10.26355/mhd_20247_1007.

[20] L. Mezmale et al., "Prevalence and potential risk factors of helicobacter pylori infection among asymptomatic individuals in kazakhstan," *Asian Pac. J. Cancer Prev.*, vol. 22, no. 2, pp. 597–603, 2021. DOI: 10.31557/APJCP.2021.22.2.597. [Online]. Available: https://doi.org/10.31557/APJCP.2021.22.2.597.

[21] S. H. Alkhyat, M. A. AL-Garadi, S. M. A. Mayas, M. A. AL Zubairy, A. A. M. AL-Harethi, and S. S. S. Baraba'a, "Prevalence and risk factors for helicobacter pylori infection among gastrointestinal patients in dhamar city, yemen," *Sana'a Univ. J. Appl. Sci. Technol.*, vol. 3, no. 5, pp. 1155–1163, 2025. DOI: 10.59628/jast.v3i5.1643. [Online]. Available: https://doi.org/10.59628/jast.v3i5.1643.

[22] A. M. Awad et al., "Prevalence of helicobacter pylori and associated risk factors in yemeni patients with gastritis in the city of aden: A cross-sectional study," *The J. Med. Res.*, vol. 10, no. 4, pp. 106–111, 2024. DOI: 10.31254/jmr.2024.10404. [Online]. Available: https://doi.org/10.31254/jmr.2024.10404.

[23] D. Xie, W. Xu, Z. Zhang, F. Huang, and X. Dai, "Epidemiological surveys, antibiotic resistance, and related risk factors of helicobacter pylori in quanzhou, china: A cross-sectional study," *Sci. Reports*, vol. 15, no. 1, p. 4410, 2025. DOI: 10.1038/s41598-025-89073-2. [Online]. Available: https://doi.org/10.1038/s41598-025-89073-2.

[24] N. K. A. A. Naser, M. B. M. Bashir, and A. S. M. A. Ali, "Prevalence and associated risk factors of helicobacter pylori infection among medical students at shendi university, sudan," *BMC Gastroenterol.*, vol. 25, no. 1, pp. 1–7, 2025. DOI: 10.1186/s12876-025-04074-9. [Online]. Available: https://doi.org/10.1186/s12876-025-04074-9.

[25] L. B. Kouitcheu Mabeku, M. L. Noundjeu Ngamga, and H. Leundji, "Potential risk factors and prevalence of helicobacter pylori infection among adult patients with dyspepsia symptoms in cameroon," *BMC Infect. Dis.*, vol. 18, no. 1, p. 278, 2018. DOI: 10.1186/s12879-018-3146-1. [Online]. Available: https://doi.org/10.1186/s12879-018-3146-1.

[26] N. Kim, "Symptoms of acute and chronic h. pylori infection," in *Helicobacter pylori*, 2nd ed., Springer, 2024, pp. 205–213. DOI: 10.1007/978-981-97-0013-4_16. [Online]. Available: https://doi.org/10.1007/978-981-97-0013-4_16.