International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Upper Gastrointestinal Endoscopic Findings in Dyspeptic Patients with and without Helicobacter pylori Infection: A Comparative Study in Western Uttar Pradesh

Dr. Shreekar B. Savaliya[1], Dr. Vipul Sangal[2], Dr. Izhar Qadir[3], Dr. Akhil kr. Gupta[4], Dr. Foram Hirapara[5]

[1]Junior resident, Department Of Surgery, Muzaffarnagar medical college and hospital, Muzaffarnagar, Uttar Pradesh
[2]Associate Professor, Department Of Surgery, Muzaffarnagar medical college and hospital, Muzaffarnagar, Uttar Pradesh
[3]Assistant Professor, Department Of Surgery, Muzaffarnagar medical college and hospital, Muzaffarnagar, Uttar Pradesh
[4]Professor and Head, Department Of Surgery, Muzaffarnagar medical college and hospital, Muzaffarnagar, Uttar Pradesh
[5]Junior Resident, department of medicine, SMIMER medical college, Surat, Gujrat

OPEN ACCESS

Corresponding Author:

Dr. Shreekar B. Savaliya
Junior resident, Department of Surgery, Muzaffarnagar medical college and hospital, Muzaffarnagar, Uttar Pradesh

Received: 21-06-2026
Accepted: 06-07-2026
Available online: 15-07-2026

ABSTRACT

Background: Dyspepsia is one of the most common gastrointestinal complaints encountered in clinical practice, presenting with symptoms such as epigastric pain, postprandial fullness, early satiety, and nausea. Although many patients have functional dyspepsia, a significant number have underlying organic lesions. Helicobacter pylori infection plays an important role in the development of chronic gastritis and peptic ulcer disease. Upper gastrointestinal (UGI) endoscopy, along with *H. pylori* testing, is widely used to identify these abnormalities. The present study was undertaken to compare endoscopic findings in dyspeptic patients with and without *H. pylori* infection. ***Aim:*** To compare upper gastrointestinal endoscopic findings in patients presenting with dyspepsia with and without Helicobacter pylori infection. ***Materials and Methods:*** This prospective, hospital-based observational study was conducted in the Department of General Surgery, Muzaffarnagar Medical College, Uttar Pradesh, over a period of 18 months. A total of 100 adult patients with dyspeptic symptoms were enrolled. All patients underwent upper gastrointestinal endoscopy, and gastric antral biopsy specimens were obtained for the detection of *H. pylori* using the rapid urease test. Based on the test results, patients were divided into *H. pylori*-positive (n = 50) and *H. pylori*-negative (n = 50) groups. Clinical profile, endoscopic findings, and histopathological features were compared using appropriate statistical methods. ***Results:*** Of the 100 patients studied, abnormal endoscopic findings were seen more frequently in those with *H. pylori* infection. Gastritis was the most common finding, followed by duodenal and gastric ulcers. In contrast, normal endoscopic findings were more often observed in patients without *H. pylori* infection. Histopathological examination also showed more frequent chronic inflammatory changes in the *H. pylori*-positive group, suggesting a clear association between the infection and gastroduodenal mucosal disease. ***Conclusion:*** Helicobacter pylori infection was associated with a higher frequency of abnormal upper gastrointestinal endoscopic findings in patients with dyspepsia. As clinical symptoms alone cannot reliably identify infected patients, combining endoscopy with *H. pylori* testing provides a more accurate diagnosis and helps guide appropriate treatment and management.

Keywords*: Dyspepsia, Helicobacter pylori, Upper gastrointestinal endoscopy, Gastritis, Peptic ulcer disease, Rapid urease test.*

INTRODUCTION

Dyspepsia is a common gastrointestinal disorder characterized by pain or discomfort in the upper abdomen [1]. It may be acute, chronic, or recurrent and is often associated with symptoms such as epigastric pain or burning, postprandial fullness, early satiety, nausea, bloating, belching, and vomiting [2]. According to the Rome III criteria, dyspepsia is defined as upper abdominal pain or discomfort associated with one or more of these symptoms in the absence of an identifiable structural or metabolic disease, with symptom onset at least six months before diagnosis [3].

Dyspepsia is a major health concern worldwide and has a considerable impact on patients' quality of life and healthcare resources. Its prevalence ranges from 25–50% in Western countries and 8–30% in Asian populations [4-6]. Clinically, dyspepsia is classified into structural (organic) and functional types. Structural dyspepsia is caused by conditions such as peptic ulcer disease, gastritis, gastroesophageal reflux disease, and upper gastrointestinal malignancy, whereas functional dyspepsia accounts for nearly 50–90% of cases and is diagnosed when no structural cause is identified [7]. Functional dyspepsia is further categorized into Epigastric Pain Syndrome (EPS) and Postprandial Distress Syndrome (PDS), or a combination of both [8-9].

Among the various causes of dyspepsia, *Helicobacter pylori* infection plays a central role. It is a spiral-shaped, Gram-negative, urease-producing bacterium that colonizes the gastric mucosa and infects nearly half of the world's population, with a higher prevalence in developing countries [10-13]. In India, the prevalence increases with age, and about one-third of adults presenting with dyspepsia are infected with *H. pylori*. Although many infected individuals remain asymptomatic, persistent infection can lead to chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric adenocarcinoma [14-18]. Because of its strong association with gastric cancer, the World Health Organization (WHO) has classified *H. pylori* as a Group I carcinogen [19-22].

Several methods are available for diagnosing *H. pylori* infection, including both invasive and non-invasive tests. Non-invasive tests such as the urea breath test, stool antigen test, and serology are commonly used for initial evaluation because they are simple and cost-effective [23-24]. However, upper gastrointestinal (UGI) endoscopy with gastric biopsy remains the gold standard, as it allows direct visualization of the mucosa, identification of structural lesions, and histopathological confirmation of *H. pylori* infection. Common endoscopic findings in dyspeptic patients include gastritis, erosive oesophagitis, duodenitis, and peptic ulcer disease [25-28].

Current guidelines recommend empirical proton pump inhibitor therapy and non-invasive *H. pylori* testing for patients without alarm features, while UGI endoscopy is reserved for those with persistent symptoms or warning signs such as gastrointestinal bleeding, unexplained weight loss, progressive dysphagia, persistent vomiting, anaemia, or suspected malignancy [29-30].

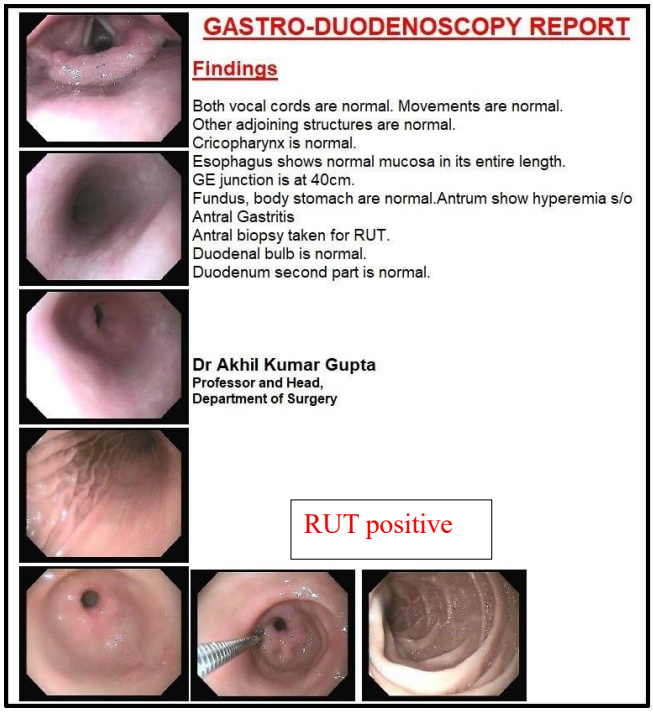

Figure 1: Gastro-duodenoscopy report showing antral hyperemia suggestive of antral gastritis with biopsy taken for RUT.

AIM AND OBJECTIVES

Aim: To compare the upper gastrointestinal endoscopic findings in patients presenting with dyspepsia according to their *Helicobacter pylori* infection status.

Objectives

- To determine the prevalence of *Helicobacter pylori* infection among patients presenting with dyspepsia.
- To assess the pattern and spectrum of upper gastrointestinal endoscopic findings in patients with dyspepsia.
- To compare the upper gastrointestinal endoscopic findings between *Helicobacter pylori*-positive and *Helicobacter pylori*-negative patients.
- To evaluate the association between *Helicobacter pylori* infection and specific upper gastrointestinal endoscopic abnormalities in dyspeptic patients.

MATERIALS AND METHODS

Study Design and Setting

This prospective, observational, hospital-based cross-sectional study was conducted in the Department of General Surgery, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh. The study was initiated after obtaining approval from the Institutional Ethics Committee, and all procedures were carried out in accordance with ethical principles for research involving human participants.

Study Duration

The study was conducted over a period of 18 months, comprising 12 months of patient recruitment and data collection, followed by 6 months dedicated to data compilation, statistical analysis, and interpretation of the findings.

Study Population

The study included adult patients presenting with symptoms of dyspepsia who attended the outpatient and inpatient services of the Department of General Surgery during the study period. Patients who fulfilled the eligibility criteria and were willing to participate were enrolled after providing written informed consent.

Sample Size

A total of 100 patients with dyspepsia were included in the study. Based on the results of *Helicobacter pylori* testing, patients were categorized into two equal groups: 50 *Helicobacter pylori*-positive patients and 50 *Helicobacter pylori*-negative patients. The upper gastrointestinal endoscopic findings were then compared between the two groups.

Sampling Method

Patients were recruited using a purposive consecutive sampling technique. Consecutive eligible patients presenting with dyspeptic symptoms during the study period were enrolled until the required sample size of 100 participants was achieved.

Inclusion Criteria

Patients fulfilling the following criteria were included in the study:

- Adults aged **18 years or older** presenting with symptoms of dyspepsia.
- Patients of either sex.
- Patients who provided written informed consent for participation in the study and for undergoing upper gastrointestinal endoscopy.

Exclusion Criteria

Patients were excluded if they met any of the following criteria:

- Inability to undergo or tolerate upper gastrointestinal endoscopy.
- Previously diagnosed active peptic ulcer disease or gastric malignancy.
- History of *Helicobacter pylori* eradication therapy within the preceding three months.
- History of partial or total gastrectomy.
- Presence of active upper gastrointestinal bleeding.
- Refusal or inability to provide written informed consent.

RESULTS

Comparison of age groups by *Helicobacter pylori* infection status

The age distribution of participants was comparable between the *Helicobacter pylori*-positive and *Helicobacter pylori*-negative groups. Among *H. pylori*-negative patients, the highest proportion (26.0%) was in the 36–45 years age group, while among *H. pylori*-positive patients, the 26–35 years age group constituted the largest proportion (24.0%) (figure 2). However, the differences in age distribution between the two groups were not statistically significant ($\mathbf{p} > 0.05$), suggesting that *H. pylori* infection was not associated with age in the present study.

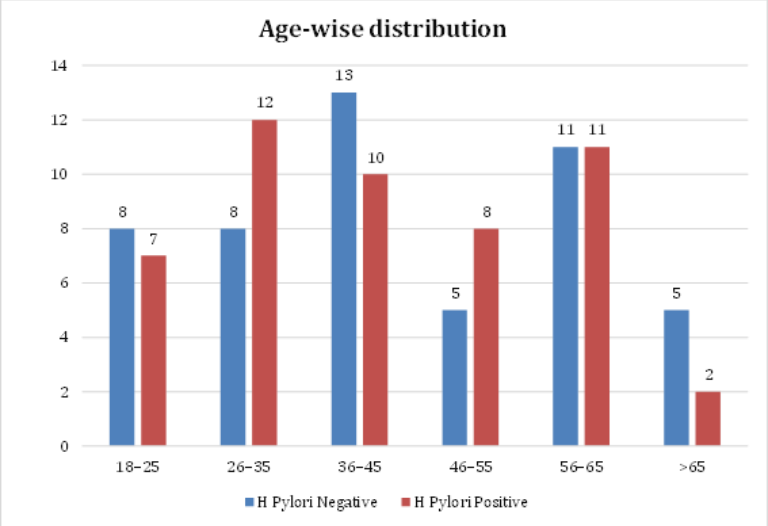

Figure 2: Comparison of age distribution between Helicobacter pylori-positive and Helicobacter pylori-negative participants

Distribution of study participants by sex and *Helicobacter pylori* infection status

Male participants outnumbered females in both the *Helicobacter pylori*-positive and *Helicobacter pylori*-negative groups. In the *H. pylori*-negative group, 62.0% were males and 38.0% were females, while in the *H. pylori*-positive group, males and females constituted 60.0% and 40.0% of participants, respectively are presented as figure 3. However, the difference in sex distribution between the two groups was not statistically significant (**p** > 0.05).

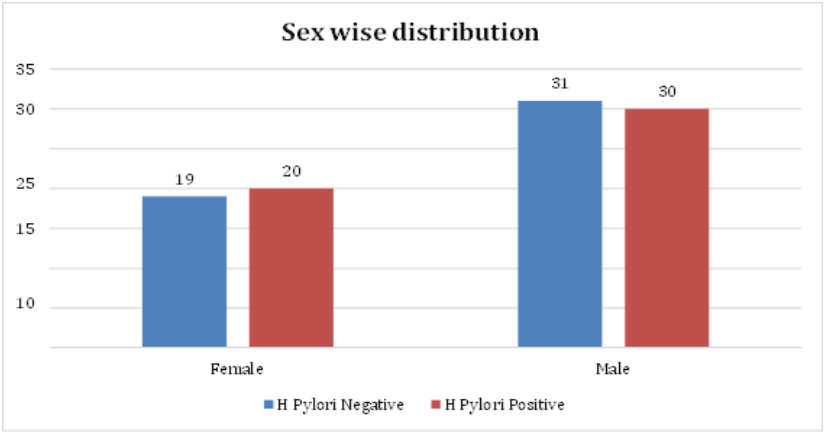

Figure 3: Sex distribution of study participants according to *Helicobacter pylori* status

Table-1: Distribution of BMI categories among study participants based on *Helicobacter pylori* infection status

BMI Category		H Pylori		P Value
		Negative	Positive	1.00
Underweight	<18.5	3(6%)	2(4%)	
Normal	18.5-24.9	27(54%)	25(50%)	
Overweight	25-29.9	18(36%)	20(40%)	
Obese Type I	30-40	2(4%)	3(6%)	
Total		50 (100%)	50 (100%)	

Normal BMI was the most common category in both the *Helicobacter pylori*-negative (54.0%) and *Helicobacter pylori*-positive (50.0%) groups, followed by overweight are presented in table 1. Only a small proportion of participants were underweight or obese. The distribution of BMI categories was similar between the two groups, with no statistically significant difference (**p** > 0.05).

Table 2: Prevalence of epigastric pain by *Helicobacter pylori* infection status

	H Pylori		p Value
Epigastric Pain	Negative	Positive	0.841

No	27 (54.0%)	26 (52.0%)	
Yes	23 (46.0%)	24 (48.0%)	
Total	50 (100%)	50 (100%)	

Epigastric pain was a common symptom among the study participants and was reported with similar frequency in both groups. It was present in 46.0% of *Helicobacter pylori*-negative patients and 48.0% of *Helicobacter pylori*-positive patients, with no statistically significant difference between the groups (**p** > 0.05) (table 2).

Table-3: Prevalence of early satiety by *Helicobacter pylori* infection status

	H Pylori		**p Value**
Early Satiety	**Negative**	**Positive**	**0.401**
No	41 (82.0%)	44 (88.0%)	
Yes	9 (18.0%)	6 (12.0%)	
Total	50 (100%)	50 (100%)	

Early satiety was observed in a small proportion of participants in both study groups. It was reported by 18.0% of *Helicobacter pylori*-negative patients and 12.0% of *Helicobacter pylori*-positive patients. However, this difference was not statistically significant (**p** > 0.05), (table 3) indicating no meaningful association between early satiety and *H. pylori* infection

Table 4: Rome IV dyspepsia subtypes according to *Helicobacter pylori* infection status

	H Pylori		**p Value**
Rome IV Subtype	**Negative**	**Positive**	**0.730**
EPS	16 (32.0%)	19 (38.0%)	
Overlap	7 (14.0%)	5 (10.0%)	
PDS	9 (18.0%)	6 (12.0%)	
Unclassified	18 (36.0%)	20 (40.0%)	
Total	50 (100%)	50 (100%)	

The distribution of Rome IV dyspepsia subtypes was broadly similar in both study groups. Epigastric Pain Syndrome (EPS) was the predominant subtype, affecting 32.0% of *Helicobacter pylori*-negative and 38.0% of *Helicobacter pylori*-positive patients, while unclassified dyspepsia was the next most frequent subtype (table 4). No statistically significant difference was observed between the groups (**p** > 0.05).

Table 5: Prevalence of NSAID use according to *Helicobacter pylori* infection status

	H Pylori		**P Value**
NSAID use	**Negative**	**Positive**	**0.362**
No	35 (70.0%)	39 (78.0%)	
Yes	15 (30.0%)	11 (22.0%)	
Total	50 (100%)	50 (100%)	

NSAID use was observed in both the *Helicobacter pylori*-positive and *Helicobacter pylori*-negative groups. It was reported by 22.0% of *H. pylori*-positive patients and 30.0% of *H. pylori*-negative patients. However, the difference in NSAID use between the two groups was not statistically significant (**p** > 0.05) (table 5).

Table 6: Comparison of upper gastrointestinal endoscopic diagnoses between *Helicobacter pylori*-positive and *Helicobacter pylori*-negative participants

Final Endoscopic Diagnosis	**H Pylori**		**P Value**
	Negative	**Positive**	**0.001**
Duodenal ulcer	2 (4.0%)	11 (22.0%)	
Erosions	5 (10.0%)	4 (8.0%)	
Esophagitis	6 (12.0%)	2 (4.0%)	
Gastric ulcer	0 (0.0%)	4 (8.0%)	
Gastritis	16 (32.0%)	22 (44.0%)	
Normal	21 (42.0%)	7 (14.0%)	
Total	50 (100%)	50 (100%)	

A significant difference in upper gastrointestinal endoscopic findings was observed between the two groups (**p** = 0.001). Normal endoscopic findings were the most frequent observation among *Helicobacter pylori*-negative patients (42.0%),

whereas gastritis was the predominant finding in *H. pylori*-positive patients (44.0%). Peptic ulcer disease, particularly duodenal ulcer, was more common in the *H. pylori*-positive group, while normal endoscopy was observed more frequently in patients without *H. pylori* infection (table 6).

Table 7: Relationship between *Helicobacter pylori* infection and clinically significant upper gastrointestinal endoscopic lesions

Clinically significant lesion	H Pylori		P Value
	Negative	Positive	0.001
No	48 (96.0%)	35 (70.0%)	
Yes	2 (4.0%)	15 (30.0%)	
Total	50 (100%)	50 (100%)	

The presence of clinically significant upper gastrointestinal endoscopic lesions was substantially higher among *Helicobacter pylori*-positive patients than among *H. pylori*-negative patients (30.0% vs. 4.0%). The association was statistically significant ($\mathbf{p} < 0.001$), indicating that *H. pylori* infection was associated with an increased likelihood of significant endoscopic lesions (Table 7).

Table 8: Histopathological findings in biopsied patients based on *Helicobacter pylori* infection status

Histopathology Done	H Pylori	
	Negative (n=22)	Positive (n=29)
Active chronic gastritis	5 (22.7%)	5 (17.2%)
Chronic duodenitis	2 (9.1%)	6 (20.7%)
Chronic gastritis	4 (18.2%)	8 (27.6%)
Chronic gastritis with ulcer	0 (0.0%)	2 (6.9%)
No significant pathology	8 (36.4%)	4 (13.8%)
Reactive gastropathy/erosive changes	2 (9.1%)	2 (6.9%)
Reflux esophagitis	1 (4.5%)	2 (6.9%)
Total	22 (100%)	29 (100%)

Among the H. pylori–negative patients (n = 22), the most common histopathological finding was no significant pathology, observed in 36.4% of cases. This was followed by active chronic gastritis (22.7%) and chronic gastritis (18.2%). Chronic duodenitis and reactive gastropathy/erosive changes were each present in 9.1% of patients, while reflux esophagitis was identified in 4.5%. Notably, no cases of chronic gastritis with ulcer were detected in the H. pylori–negative group. In contrast, among H. pylori–positive patients (n = 29), chronic gastritis was the predominant histopathological finding, accounting for 27.6% of cases, followed by chronic duodenitis (20.7%) and active chronic gastritis (17.2%). Chronic gastritis with ulcer, reactive gastropathy/erosive changes, and reflux esophagitis were each observed in 6.9% of patients. The proportion of patients with no significant pathology was considerably lower in the H. pylori–positive group (13.8%) than in the H. pylori–negative group (36.4%), suggesting that H. pylori infection was associated with a higher frequency of histopathological abnormalities (table 8).

DISCUSSION

The present study found no significant association between *Helicobacter pylori* infection and demographic factors such as age, sex, or body mass index (BMI). These findings suggest that demographic characteristics alone are not reliable predictors of *H. pylori* infection in patients with dyspepsia, consistent with previous reports by Agarwal et al. and Mwangi et al. [31,32]. Although increased BMI has been linked to clinically significant endoscopic lesions, it does not appear to independently predict *H. pylori* infection [33].

Similarly, common dyspeptic symptoms, including epigastric pain, postprandial fullness, and early satiety, were comparable between *H. pylori*–positive and –negative patients. This supports earlier studies showing that symptom patterns alone cannot reliably distinguish *H. pylori* infection, as dyspeptic symptoms are influenced by multiple functional and organic mechanisms [34,35]. Likewise, the comparable distribution of Rome IV dyspepsia subtypes between the two groups further indicates that symptom-based classification has limited value in predicting infection [36].

No significant association was observed between *H. pylori* infection and lifestyle factors such as alcohol consumption, tobacco use, or NSAID exposure. Although these factors contribute to upper gastrointestinal symptoms, their effects appear to be independent of *H. pylori* infection. The difference from the findings of Subedi et al. may reflect variations in study population and regional risk factors [37].

In contrast, *H. pylori* infection was strongly associated with abnormal endoscopic findings. Gastritis, duodenal ulcer, and gastric ulcer were significantly more common among infected patients, whereas normal endoscopic findings predominated

in those without infection. These results are consistent with the established role of *H. pylori* in chronic gastritis and peptic ulcer disease and are in agreement with previous studies [32,33]. Furthermore, clinically significant endoscopic lesions were significantly more frequent in *H. pylori*–positive patients, reinforcing the importance of endoscopic evaluation and *H. pylori* testing in dyspeptic individuals with suspected organic disease [32,34].

Histopathological findings further supported these observations. Chronic gastritis and chronic duodenitis were more common among *H. pylori*–positive patients, whereas normal histology was more frequent in *H. pylori*–negative patients. These findings are consistent with the chronic inflammatory changes induced by *H. pylori* [32,37].

Overall, the study demonstrates that demographic characteristics, symptom profile, and lifestyle factors are poor predictors of *H. pylori* infection. In contrast, endoscopic and histopathological findings show a strong association with infection, highlighting the importance of upper gastrointestinal endoscopy with biopsy for accurate diagnosis and appropriate management of dyspeptic patients.

CONCLUSION

This study showed that *Helicobacter pylori* infection is associated with a higher occurrence of abnormal upper gastrointestinal endoscopic findings in patients with dyspepsia, particularly inflammatory and ulcerative lesions. Clinical symptoms alone were not sufficient to differentiate infected and non-infected patients, emphasizing the importance of diagnostic evaluation. The findings support the role of *H. pylori* in gastric mucosal changes and highlight the need for its assessment in dyspeptic patients undergoing endoscopy for better diagnosis and management.

REFERENCES

1. Bektas, M., Soykan, I., Altan, M., Alkan, M., & Özden, A. (2009). The effect of Helicobacter pylori eradication on dyspeptic symptoms, acid reflux and quality of life in patients with functional dyspepsia. *European journal of internal medicine*, *20*(4), 419-423.
2. Stanghellini, V., Chan, F. K., Hasler, W. L., Malagelada, J. R., Suzuki, H., Tack, J., & Talley, N. J. (2016). Gastroduodenal disorders. *Gastroenterology*, *150*(6), 1380-1392.
3. Conroy, R. T., & Siddiqi, B. (2007). Dyspepsia. *Primary Care: Clinics in Office Practice*, *34*(1), 99-108.
4. Tytgat, G. N. J. (2002). Role of endoscopy and biopsy in the work up of dyspepsia. *Gut*, *50*(suppl 4), iv13-iv16.
5. Niknam, R., Seddigh, M., Fattahi, M. R., Dehghanian, A., & Mahmoudi, L. (2014). Prevalence of Helicobacter pylori in patients with dyspepsia. *Jundishapur journal of microbiology*, *7*(10), e12676.
6. Ghoshal, U. C., Singh, R., Chang, F. Y., Hou, X., Wong, B. C. Y., & Kachintorn, U. (2011). Epidemiology of uninvestigated and functional dyspepsia in Asia: facts and fiction. *Journal of neurogastroenterology and motility*, *17*(3), 235.
7. Lu, C. L., Lang, H. C., Chang, F. Y., Chen, C. Y., Luo, J. C., Wang, S. S., & Lee, S. D. (2005). Prevalence and health/social impacts of functional dyspepsia in Taiwan: a study based on the Rome criteria questionnaire survey assisted by endoscopic exclusion among a physical check-up population. *Scandinavian journal of gastroenterology*, *40*(4), 402-411.
8. Talley, N. J., Silverstein, M. D., Agreus, L., Nyren, O., Sonnenberg, A., & Holtmann, G. (1998). AGA technical review: evaluation of dyspepsia. *Gastroenterology*, *114*(3), 582-595.
9. Talley, N. J., Weaver, A. L., & Zinsmeister, A. R. (1995). Impact of functional dyspepsia on quality of life. *Digestive diseases and sciences*, *40*(3), 584-589.
10. Farooqi, J. I., & Farooqi, R. J. (2011). Non-steroidal-anti-inflammatory-drugs induced gastrotoxicity. *JOURNAL-COLLEGE OF PHYSICIANS AND SURGEONS OF PAKISTAN*, *11*, 650-655.
11. Aminde JA, Dedino GA, Ngwasiri CA, Ombaku KS, Makon CA, Aminde LN. (2019). Helicobacter pylori infection among patients presenting with dyspepsia at a primary care setting in Cameroon: seroprevalence, five-year trend and predictors. BMC infectious diseases,19(1):1–9.
12. Kusters, J. G., Van Vliet, A. H., & Kuipers, E. J. (2006). Pathogenesis of Helicobacter pylori infection. *Clinical microbiology reviews*, *19*(3), 449-490.
13. Brown, L. M. (2000). Helicobacter pylori: epidemiology and routes of transmission. *Epidemiologic reviews*, *22*(2), 283-297.
14. Ieni, A., Barresi, V., Rigoli, L., Fedele, F., Tuccari, G., & Caruso, R. A. (2016). Morphological and cellular features of innate immune reaction in Helicobacter pylori gastritis: a brief review. *International Journal of Molecular Sciences*, *17*(1), 109.
15. Mandeville, K. L., Krabshuis, J., Ladep, N. G., Mulder, C. J., Quigley, E. M., & Khan, S. A. (2009). Gastroenterology in developing countries: issues and advances. *World journal of gastroenterology: WJG*, *15*(23), 2839–54.
16. Das, J. C., & Paul, N. (2007). Epidemiology and pathophysiology of Helicobacter pylori infection in children. *The Indian Journal of Pediatrics*, *74*(3), 287-290.

17. Srinivas, Y., Prasad, P. K., & Sai, N. D. (2016). Prevalence and impact of Helicobacter pylori in dyspepsia. *International Surgery Journal*, *3*(1), 305-309.
18. Wai, C. T., Yeoh, K. G., Ho, K. Y., Kang, J. Y., & Lim, S. G. (2002). Diagnostic yield of upper endoscopy in Asian patients presenting with dyspepsia. *Gastrointestinal endoscopy*, *56*(4), 548-551.
19. Kate, V., Ananthakrishnan, N., Badrinath, S., & Ratnakar, C. (1998). Prevalence of Helicobacter pylori infection in disorders of the upper gastrointestinal tract in south India. *National Medical Journal of India*, *11*, 5-8.
20. Mapel, D., Roberts, M., Overhiser, A., & Mason, A. (2013). The Epidemiology, Diagnosis, and Cost of Dyspepsia and H elicobater pylori Gastritis: A Case–Control Analysis in the S outhwestern U nited S tates. *Helicobacter*, *18*(1), 54-65.
21. Özbek, A., Özbek, E., Dursun, H., Kalkan, Y., & Demirci, T. (2010). Can Helicobacter pylori invade human gastric mucosa?: an in vivo study using electron microscopy, immunohistochemical methods, and real-time polymerase chain reaction. *Journal of clinical gastroenterology*, *44*(6), 416-422.
22. Mishra, S., Singh, V., Rao, G., Jain, A. K., Dixit, V. K., Gulati, A. K., & Nath, G. (2008). Detection of Helicobacter pylori in stool specimens: comparative evaluation of nested PCR and antigen detection. *The Journal of Infection in Developing Countries*, *2*(03), 206-210.
23. Potamitis, G. S., & Axon, A. T. (2015). Helicobacter pylori and nonmalignant diseases. *Helicobacter*, *20*, 26-29.
24. Smith, S. M., O'Morain, C., & McNamara, D. (2014). Antimicrobial susceptibility testing for Helicobacter pylori in times of increasing antibiotic resistance. *World journal of gastroenterology: WJG*, *20*(29), 9912.
25. Krogfelt, K. A., Lehours, P., & Mégraud, F. (2005). Diagnosis of Helicobacter pylori infection. *Helicobacter*, *10*, 5-13.
26. Iwańczak, F., Pytrus, T., Iwańczak, B., Gościniak, G., Witkowska-Vogtt, E., Dzierzanowska, D., & Laszewicz, W. (2005). Assessment of selected tests in diagnostics of Helicobacter pylori infections. *Polski Merkuriusz Lekarski: Organ Polskiego Towarzystwa Lekarskiego*, *19*(109), 52-56.
27. Mégraud, F., & Lehours, P. (2007). Helicobacter pylori detection and antimicrobial susceptibility testing. *Clinical microbiology reviews*, *20*(2), 280-322.
28. Faintuch, J. J., Silva, F. M., Navarro-Rodriguez, T., Barbuti, R. C., Hashimoto, C. L., Rossini, A. R. A. L., ... & Eisig, J. N. (2014). Endoscopic findings in uninvestigated dyspepsia. *BMC gastroenterology*, *14*(1), 19.
29. Malfertheiner, P., Megraud, F., O'Morain, C. A., Atherton, J., Axon, A. T., Bazzoli, F., ... & European Helicobacter Study Group (EHSG). (2012). Management of Helicobacter pylori infection—the Maastricht IV/Florence consensus report. *Gut*, *61*(5), 646-664.
30. Moayyedi, P. M., Lacy, B. E., Andrews, C. N., Enns, R. A., Howden, C. W., & Vakil, N. (2017). ACG and CAG clinical guideline: management of dyspepsia. *Official journal of the American College of Gastroenterology| ACG*, *112*(7), 988-1013.
31. Agarwal, P. K., Badkur, M., Agarwal, R., & Patel, S. (2018). Prevalence of Helicobacter pylori infection in upper gastrointestinal tract disorders (dyspepsia) patients visiting outpatient department of a hospital of North India. *Journal of family medicine and primary care*, *7*(3), 577-580.
32. Marzio, L., Cappello, G., & Ballone, E. (2003). Evaluation of dyspeptic symptoms in patients with and without Helicobacter pylori infection and normal upper gastrointestinal endoscopy. *Digestive and liver disease*, *35*(3), 138-142.
33. Ayana, S. M., Swai, B., Maro, V. P., & Kibiki, G. S. (2014). Upper gastrointestinal endoscopic findings and prevalence of Helicobacter pylori infection among adult patients with dyspepsia in northern Tanzania.18;16(1).
34. Mwangi, C. N., Njoroge, S., Rajula, A., Laving, A., Kamenwa, R., Devani, S., ... & Revathi, G. (2020). Prevalence and endoscopic findings of Helicobacter pylori infection among dyspeptic patients in Kenya. *Open J Med Microbiol*, *10*(04), 233.
35. Mao, L. Q., Wang, S. S., Zhou, Y. L., Chen, L., Yu, L. M., Li, M., & Lv, B. (2021). Clinically significant endoscopic findings in patients of dyspepsia with no warning symptoms: A cross-sectional study. *World Journal of Clinical Cases*, *9*(15), 3597.
36. Subedi, R. C., Regmi, B. U., Pathak, B. D., Dhakal, B., Sitaula, D., Paudel, U., ... & Roka, K. (2023). Prevalence of Helicobacter pylori infection in dyspeptic patients presenting to a tertiary care center of a developing country: a cross-sectional study. *F1000Research*, *12*, 1195.
37. Mabeku, L. B. K., Nguefak, L. D. T., Nintewoue, G. F. F., Ngemeshe, S. N., Mengang, J. M. N. N., Ngatcha, G., ... & Talla, P. (2025). Conventional Upper-Intestinal Endoscopy Value in Predicting Histopathological Features Related to Helicobacter pylori Infection: A Comparative Study Between Endoscopy and Histology Findings Among Dyspeptic Outpatients in Cameroon. *JGH Open*, *9*(8), e70222.