Bangladesh Med J. 2025 Sept; 54(3)
DOI: https://doi.org/10.3329/bmj.v54i3.90549

Original Article

Clinico-endoscopic Patterns of Dyspepsia in *Helicobacter pylori* Positive Adults

Mahfuz SM[1], Islam MS[2], *Khokan MAI[3], Newaz AAS[4], Chowdhury MFK[5], Islam S[6]

Abstract

Dyspepsia is a common upper gastrointestinal symptom complex with overlapping functional and organic etiologies. Among Helicobacter pylori (H. pylori)-positive patients, symptom patterns frequently demonstrate poor correlation with endoscopic findings. This study evaluated the clinicoendoscopic distribution of dyspepsia subtypes and their associated predictors among H. pylori-positive adults without alarm features. This cross-sectional study analyzed baseline data from a randomized controlled trial conducted among treatment-native adults aged ≥18 years with H. pylori-positive dyspepsia. Dyspepsia was categorized according to Rome IV criteria into Epigastric Pain Syndrome (EPS), Postprandial Distress Syndrome (PDS), and mixed subtype. Upper gastrointestinal endoscopy classified patients as functional dyspepsia (FD) or organic dyspepsia (OD). Descriptive statistics, chi-square test, Student's t-test, ANOVA, and multinomial logistic regression were performed. Odds ratios (OR) with 95% confidence intervals (CI) were reported, and p<0.05 was considered statistically significant. A total of 80 participants were included, with a mean age of 35.8±11.4 years; 53.7% were female, and 52.5% resided in rural areas. Mixed dyspepsia was the most frequent subtype (55.0%), followed by EPS (28.8%) and PDS (16.3%). Functional dyspepsia accounted for 51.2% of cases, whereas organic dyspepsia was observed in 48.8%. Abnormal endoscopic findings were significantly more frequent among EPS and PDS subtypes (p<0.001). Erosive gastritis was the most common abnormality (23.8%), followed by nonerosive gastritis (15.0%). Epigastric pain, epigastric burning, postprandial fullness, and early satiety were significantly more common in FD than OD (all p<0.01). Organic dyspepsia was significantly associated with age ≥34 years (OR 1.90; p=0.008) and smoking history (OR 1.76; p=0.019). Multinomial logistic regression demonstrated that symptom duration ≥12 months (OR 9.04; p=0.005) and normal endoscopy/FD (OR 17.00; p<0.001) independently predicted mixed dyspepsia. Mixed dyspepsia was the predominant symptom phenotype among H. pylori-positive adults. Functional dyspepsia was strongly associated with overlapping symptom patterns, whereas EPS and PDS were more frequently associated with organic abnormalities. Symptom duration and endoscopic status were stronger predictors of dyspepsia subtype than demographic variables, supporting an integrated clinicoendoscopic approach for evaluation and stratification.

Keywords: *Functional dyspepsia; organic dyspepsia; epigastric pain syndrome; postprandial distress syndrome; helicobacter pylori*

1. Dr. Syeda Mubashsharah Mahfuz, OSD, Directorate General of Health Services, Dhaka, Bangladesh
2. Dr. Md. Saiful Islam, Resident Physician, National Gastroliver Institute and Hospital, Dhaka
3. *Dr. Md. Ahid Iqbal Khokan, Medical Officer, UHC, Tongibari, Munshiganj, Bangladesh, Email: ahidiqbaldmc65@gmail.com
4. Dr. Abdullah Al Shah Newaz, Assistant Professor, Department of Gastroenterology, Bangladesh Medical University (BMU), Dhaka.
5. Dr. Md. Fazlul Karim Chowdhury, Assistant Professor, Department of Gastroenterology, BMU, Dhaka.
6. Dr. Susmita Islam, Assistant Professor, Department of Gastroenterology, BMU, Dhaka.

* For Correspondence

INTRODUCTION

Dyspepsia is characterized by chronic or recurrent upper abdominal symptoms, including epigastric pain, epigastric burning, early satiety, postprandial fullness, bloating, nausea, and upper abdominal discomfort.[1] Dyspepsia significantly impairs quality of life, increases healthcare utilization, and imposes a considerable socioeconomic burden, particularly in low and middle-income countries.[2]

Globally, the prevalence of dyspepsia varies widely, ranging from 10% to 40% depending on the population studied and diagnostic criteria applied.[3] In South Asian countries, the burden of dyspepsia remains high because of dense population, dietary variation, environmental factors, smoking, widespread *H. pylori* infection, and limited access to early gastrointestinal evaluation.[4] Functional gastrointestinal disorders are increasingly recognized as major contributors to dyspepsia; however, clinically significant organic lesions continue to be common in developing countries.[5]

The Rome IV classification divides functional dyspepsia into two principal symptom-based subtypes: Epigastric Pain Syndrome (EPS) and Postprandial Distress Syndrome

(PDS).[6] EPS is characterized predominantly by epigastric pain or burning, whereas PDS is associated with meal-related symptoms such as postprandial fullness and early satiety. Nevertheless, many patients demonstrate overlapping manifestations of both syndromes, often referred to as mixed dyspepsia.[7]

Upper gastrointestinal endoscopy remains the standard investigation for identifying structural or mucosal abnormalities among dyspeptic patients. Endoscopic evaluation differentiates functional dyspepsia (FD), in which no structural explanation is found, from organic dyspepsia (OD), where identifiable lesions such as gastritis, erosions, peptic ulcer disease, or malignancy are detected.[8] Despite its diagnostic importance, previous studies have demonstrated inconsistent relationships between dyspeptic symptom patterns and endoscopic findings.[9]

Among the recognized etiological factors of dyspepsia, *H. pylori* occupies a particularly important role. *H. pylori* infection affects nearly half of the global population and remains highly prevalent in South Asia and Bangladesh.[10] The organism contributes to chronic gastritis, peptic ulcer disease, mucosal inflammation, altered gastric motility, visceral hypersensitivity, and gastric carcinogenesis.[11] Several studies have demonstrated that *H. pylori* infection may influence both symptom severity and dyspepsia phenotype, although findings remain inconsistent across populations.[12]

In Bangladesh, data regarding symptom subtype distribution and its association with endoscopic findings remain limited. A recent Bangladeshi study reported that mixed EPS-PDS overlap represented the most common symptom phenotype among functional dyspepsia patients.[13] Another community-based endoscopy study identified substantial coexistence of functional dyspepsia, peptic ulcer disease, and *H. pylori* infection among South Asian adults.[14]

Previous international studies have reported variable endoscopic yields among dyspeptic populations. A Cambodian study found that erosive gastritis and peptic ulcer disease were common endoscopic findings among chronic dyspepsia patients.[15] Similarly, contemporary literature suggests that functional dyspepsia is strongly influenced by altered gut-brain interaction, gastric hypersensitivity, motility disturbances, and chronic low-grade inflammation.[16] Chronic gastritis related to *H. pylori* may further contribute to symptom generation through mucosal injury and inflammatory pathways involving the gut-brain axis.[17,18] These mechanisms may explain why symptom overlap is frequently observed and why symptom severity often does not accurately predict endoscopic pathology.

A thorough understanding of the relationship between dyspeptic symptoms and endoscopic abnormalities in H. pylori–positive patients is crucial from a clinical perspective. This knowledge may enhance risk stratification and guide the effective use of endoscopy in settings where resources are limited. Identifying symptom patterns that are associated with organic pathology can help clinicians prioritize their patients effectively. Moreover, characterizing overlapping symptom patterns may lead to a better understanding of the pathophysiology of dyspepsia and support the development of individualized management strategies. The purpose of this study was to evaluate the clinico-endoscopic patterns of dyspepsia in adults who are *H. pylori*–positive and do not present alarm features.

MATERIALS AND METHODS

This study was designed as a cross-sectional analytical examination conducted at the tertiary care hospital of Bangladesh Medical University, Bangladesh. It used baseline (pre-treatment) data from a randomized controlled trial (RCT) focused on *H. pylori* eradication, conducted from January 2024 to June 2025. All eligible participants from the baseline cohort prior to randomization were recruited into this analysis. The sample size was determined by the parent RCT.

A total of 80 adult patients (aged ≥18 years) presenting with dyspeptic symptoms without alarm features were enrolled consecutively. Dyspepsia was defined according to the Rome IV criteria. This study included dyspeptic individuals confirmed to have *H. pylori* infection via both the rapid urease test and stool antigen test, who were treatment-naïve, participated in symptom assessment, and underwent upper gastrointestinal endoscopy before being randomized in the RCT. Patients with gastrointestinal bleeding, persistent vomiting, significant weight loss, progressive dysphagia, malignancy, prior gastric surgery, severe systemic illness, pregnancy, recent proton pump inhibitor or antibiotic use, or previous *H. pylori* eradication therapy were excluded.

A structured, interviewer-administered questionnaire was used to collect clinical and demographic data. All eligible patients underwent detailed clinical evaluation, including demographic characteristics (age, sex, residence), smoking history, body mass index (BMI), symptom profile, and

symptom duration. Dyspeptic symptoms were categorized into Epigastric Pain Syndrome (EPS), Postprandial Distress Syndrome (PDS), and mixed subtype based on Rome IV criteria.

Upper gastrointestinal endoscopy (UGIE) was performed in all participants before the RCT. Patients with normal endoscopy were classified as functional dyspepsia (FD), while those with identifiable mucosal or structural abnormalities were categorized as organic dyspepsia (OD). Endoscopic findings, including erosive gastritis, non-erosive gastritis, gastric ulcer, and duodenal ulcer, were documented.

Only baseline data were analyzed to ensure a uniform group of untreated individuals. The primary outcome was the distribution of dyspepsia subtypes (EPS, PDS, mixed). Secondary outcomes included functional versus organic dyspepsia, the association between symptoms and endoscopic findings, and predictors of dyspepsia phenotype.

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 26. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequency and percentage. Pie charts were used to illustrate the distribution of dyspepsia subtypes and the FD-OD group, and bar diagrams were used to show the distribution of individual dyspeptic symptoms across subtypes. Student's t-test and ANOVA were used for comparison of normally distributed continuous variables, whereas the chi-square test was applied for categorical variables. Multinomial logistic regression analysis was performed using EPS as the reference category to identify predictors of dyspepsia subtypes. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. A p-value <0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board of Bangladesh Medical University. Written informed consent was obtained from all participants before enrolment.

RESULTS

A total of 80 *H. pylori*–positive adults with dyspepsia were included in the study. The mean age of the participants was 35.8±11.4 years. Females constituted 53.7% of the study population, while 52.5% of participants resided in rural areas. The mean BMI was 24.8±3.0 kg/m², and 35.0% had a history of smoking.

Figure 1 displays the distribution of dyspepsia subtypes. Among the patients with dyspeptic symptoms, 23 (28.75%) patients were categorized as having epigastric pain syndrome (EPS), 13 (16.25%) as postprandial distress syndrome (PDS), and 44 (55.0%) as mixed dyspepsia.

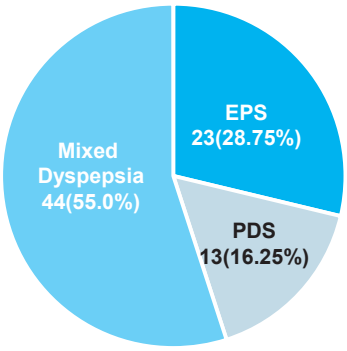

Figure 1. Distribution of dyspepsia subtypes (n=80)

Figure 2 shows a pie chart illustrating the proportions of patients diagnosed with Functional Dyspepsia (FD) and Organic Dyspepsia (OD) based on endoscopic findings. FD was found in 51.3% of study participants, and OD in 48.7%.

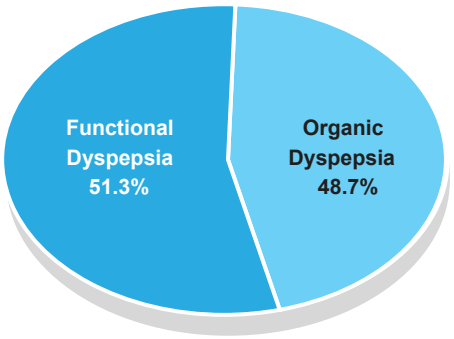

Figure 2: Functional Dyspepsia and Organic Dyspepsia based on endoscopic findings (n=80).

Table I presents the baseline demographics, lifestyle, and clinical features of study participants. The mean age of the study population was 35.8 ± 11.4 years. There were no statistically significant differences across dyspepsia subtypes in terms of age (p = 0.995), sex distribution (p = 0.290), body mass index (BMI) (p = 0.751), residence (p = 0.994), smoking status (p = 0.635), or symptom duration (p = 0.447).

Endoscopic findings normal (FD) versus abnormal (OD) differed significantly among subtypes, normal (FD) versus abnormal (OD) (p < 0.001). Normal endoscopy was more frequent in the mixed subtype (72.7%) compared to EPS (21.7%) and PDS (30.8%). Conversely, abnormal endoscopic findings were more common in EPS (78.3%) and PDS (69.2%) than in mixed dyspepsia (27.3%).

Among specific findings, non-erosive gastritis showed a significant association with dyspepsia subtype (p < 0.001), being more frequent in PDS (38.5%) and EPS (26.1%) compared to the mixed group (2.3%). No statistically significant differences were observed for erosive gastritis (p = 0.105), gastric ulcer (p = 0.179), or duodenal ulcer (p = 0.329).

Table I: Baseline Demographics, Lifestyle, and Clinical features study participants (n=80)

Variable	Total (n=80)	EPS (n=23)	PDS (n=13)	Mixed (n=44)	p-value
Age (years)	35.8 ± 11.4	35.9 ± 9.5	35.5 ± 6.4	35.9 ± 13.4	0.995
Sex					0.290
Male	37 (46.3%)	8 (34.8%)	8 (61.5%)	21 (47.7%)	
Female	43 (53.7%)	15 (65.2%)	5 (38.5%)	23 (52.3%)	
BMI (kg/m²)	24.8 ± 3.0	24.9 ± 3.0	24.2 ± 2.7	24.8 ± 3.2	0.751
Residence					0.994
Urban	38 (47.5%)	11 (47.8%)	6 (46.2%)	21 (47.7%)	
Rural	42 (52.5%)	12 (52.2%)	7 (53.8%)	23 (52.3%)	
Smoking status					0.635
Ever smoker	28 (35.0%)	8 (34.8%)	6 (46.2%)	14 (31.8%)	
Never smoker	52 (65.0%)	15 (65.2%)	7 (53.8%)	30 (68.2%)	
Symptom duration (months)	21.0 ± 12.1	18.3 ± 9.2	22.2 ± 9.7	22.0 ± 13.9	0.447
Endoscopic findings of Dyspepsia					<0.001
Normal/ FD	41 (51.2%)	5 (21.7%)	4 (30.8%)	32 (72.7%)	
Abnormal/ OD	39 (48.8%)	18 (78.3%)	9 (69.2%)	12 (27.3%)	
Abnormal Endoscopic Findings					
Erosive gastritis	19 (23.8%)	9 (39.1%)	3 (23.1%)	7 (15.9%)	0.105
Non-erosive gastritis	12 (15.0%)	6 (26.1%)	5 (38.5%)	1 (2.3%)	<0.001
Gastric ulcer	4 (5.0%)	0 (0.0%)	0 (0.0%)	4 (9.1%)	0.179
Duodenal ulcer	7 (8.8%)	3 (13.0%)	2 (15.4%)	2 (4.5%)	0.329

Note: Values are expressed as mean ± SD or frequency (%). BMI = Body Mass Index; EPS = Epigastric Pain Syndrome; PDS = Postprandial Distress Syndrome; FD = Functional Dyspepsia; OD = Organic Dyspepsia. Statistical tests: ANOVA for continuous variables; Chi-square test for categorical variables.

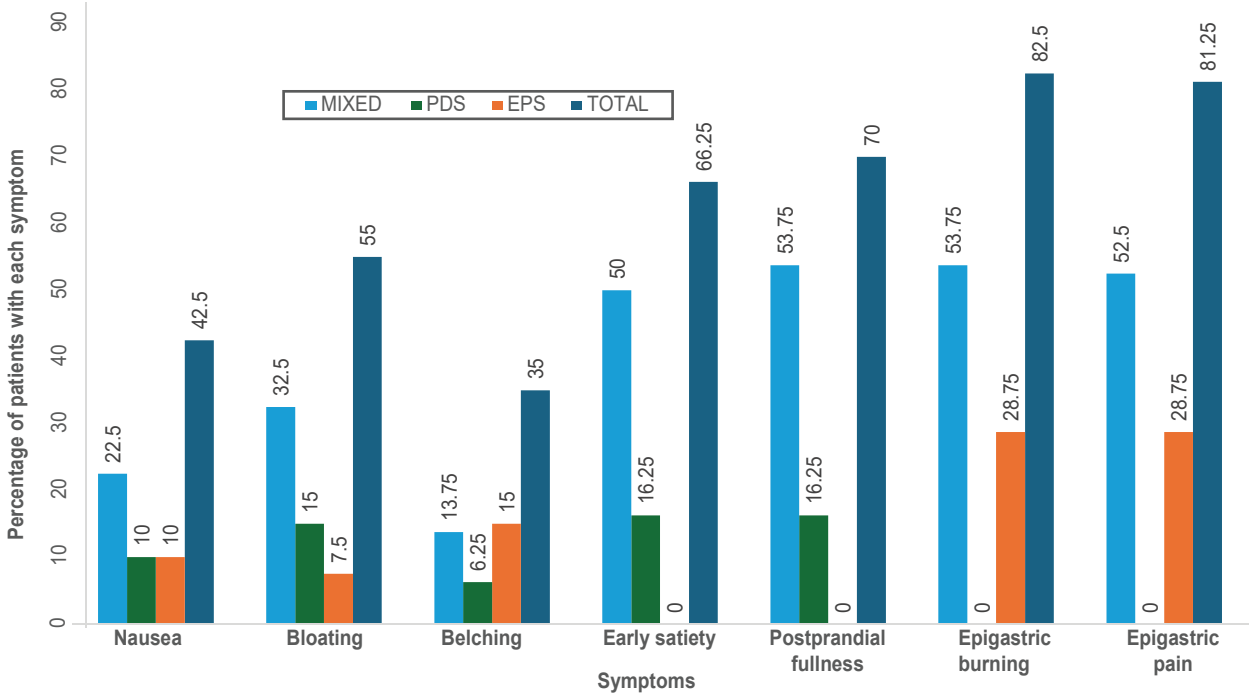

Figure 3: Distribution of dyspeptic symptoms in different subtypes of dyspepsia (n = 80)

Figure 3 illustrates the distribution of dyspeptic symptoms in different subtypes of dyspepsia. Among *H pylori* positive adults, in the EPS group, epigastric pain and epigastric burning were each reported by 28.75 % of patients, while postprandial fullness and early satiety were absent (0.0 %). Belching occurred in 15.0 %, bloating in 7.5 %, and nausea in 10.0 %. In the PDS group, postprandial fullness and early satiety were each present in 16.25 %, belching in 6.25 %, bloating in 15.0 %, and nausea in 10.0 %, whereas epigastric pain and burning were not reported (0.0 %). In the Mixed subtype, epigastric pain was observed in 52.5 %, epigastric burning in 53.75 %, postprandial fullness in 53.75 %, early satiety in 50.0 %, belching in 13.75 %, bloating in 32.5 %, and nausea in 22.5 %.

Table II presents the comparison of dyspeptic symptoms between functional and organic dyspepsia. Among the 80 participants, functional dyspepsia (FD) was present in 41 patients (51.2%), while organic dyspepsia (OD) was identified in 39 patients (48.8%).

Both epigastric pain (p = 0.006) and epigastric burning (p = 0.004) were significantly more frequent in FD (each was present in 90.2%) compared to OD, where 71.8% in epigastric pain and 74.4% in epigastric burning. Postprandial fullness and early satiety were significantly more common in FD, observed in 87.8% vs 51.3% (p = 0.001) and 80.5% vs 51.3% (p = 0.003), respectively. There were no statistically significant differences between the two groups in terms of bloating (p = 0.742), belching (p = 0.631), or nausea (p = 0.518).

Table II. Comparison of dyspeptic symptoms between functional and organic dyspepsia

Symptom	FD (n=41)	OD (n=39)	p value*
Epigastric pain	37 (90.2%)	28 (71.8%)	**0.006**
Epigastric burning	37 (90.2%)	29 (74.4%)	**0.004**
Postprandial fullness	36 (87.8%)	20 (51.3%)	**0.001**
Early satiety	33 (80.5%)	20 (51.3%)	**0.003**
Bloating	22 (53.7%)	22 (56.4%)	0.742
Belching	12 (29.3%)	16 (41.0%)	0.631
Nausea	20 (48.8%)	14 (35.9%)	0.518

FD = Functional Dyspepsia; OD = Organic Dyspepsia.
*Chi-Square test

Table III shows the comparison of associated factors between functional and organic dyspepsia. Age ≥34 years was significantly associated with organic dyspepsia (OR 1.90; p = 0.008). Smoking was also significantly associated with organic dyspepsia (OR 1.76; p = 0.019). No statistically significant associations were observed for sex (p = 1.000), residence (p = 0.512), obesity (p = 0.509), or symptom duration (p = 0.260). Dyspepsia subtype distribution differed significantly between functional and organic groups (p < 0.001). Functional dyspepsia was more frequent in the mixed subtype (78.0%), whereas organic dyspepsia was more frequent in EPS (46.2%).

Table III: Comparison of associated factors between functional and organic dyspepsia (n=80)

Variable		Functional (n=41)	Organic (n=39)	OR for Organic (95% CI)	P value*
Age	≥34 years	15 (36.6%)	26 (66.7%)	**1.90 (1.15–3.14)**	**0.008**
	<34 years	26 (63.4%)	13 (33.3%)		
Sex	Male	19 (46.3%)	18 (46.2%)	1.00 (0.64–1.56)	1.000
	Female	22 (53.7%)	21 (53.8%)		
Residence	Urban	21 (51.2%)	17 (43.6%)	0.85 (0.54–1.35)	0.512
	Rural	20 (48.8%)	22 (56.4%)		
Obesity	Obese	20 (48.8%)	16 (41.0%)	0.85 (0.54–1.35)	0.509
	Non-obese	21 (51.2%)	23 (59.0%)		
Smoking history	Smoker ever	9 (22.0%)	19 (48.7%)	**1.76 (1.15–2.71)**	**0.019**
	Smoker never	32 (78.0%)	20 (51.3%)		
Duration	≥12 months	15 (36.6%)	20 (51.3%)	1.35 (0.87–2.12)	0.260
	<12 months	26 (63.4%)	19 (48.7%)		
Subtype distribution	EPS	5 (12.2%)	18 (46.2%)		**<0.001**
	PDS	4 (9.8%)	9 (23.1%)		
	Mixed	32 (78.0%)	12 (30.8%)		

CI = Confidence Interval, EPS=Epigastric Pain Syndrome, OR = Odds ratio, PDS=Postprandial Distress Syndrome. *Chi-Square test

Table IV displays the multinomial logistic regression analysis, which was performed using EPS as the reference category. There were no statistically significant associations between age, sex, smoking status, or BMI and either PDS or mixed dyspepsia (all $p > 0.05$). Symptom duration ≥12 months was significantly associated with mixed dyspepsia (OR 9.04; $p = 0.005$), but not with PDS ($p = 0.170$). Normal endoscopy was also significantly associated with mixed dyspepsia (OR 17.00; $p < 0.001$), while no significant association was observed for PDS ($p = 0.414$).

Table IV. Multinomial logistic regression analysis of predictors of dyspepsia subtypes(n=80)

Variable	PDS vs EPS OR (95% CI)	p-value	Mixed vs EPS OR (95% CI)	p-value
Age ≥34 years vs <34 years	1.07 (0.20–5.64)	0.941	0.45 (0.10–1.96)	0.289
Male sex vs female	0.33 (0.05–2.05)	0.234	0.73 (0.15–3.53)	0.694
Smoking (yes vs no)	0.87 (0.13–5.79)	0.882	1.58 (0.29–8.68)	0.598
BMI ≥25 kg/m² vs <25 kg/m²	0.71 (0.14–3.74)	0.690	1.10 (0.28–4.43)	0.889
Duration ≥12 months vs <12 months	3.18 (0.61–16.61)	0.170	9.04 (1.93–42.32)	0.005*
Normal endoscopy vs abnormal findings	2.12 (0.35–12.97)	0.414	17.00 (3.59–80.54)	<0.001*

*Reference category: Epigastric Pain Syndrome (EPS), *statistical significance (p < 0.05).*
BMI= Body Mass Index, CI= Confidence Interval, EPS=Epigastric Pain Syndrome, OR=Odd ratio, PDS=Postprandial Distress Syndrome

DISCUSSION

This study evaluated dyspepsia in *H. pylori*–positive, non-alarm patients by combining symptom patterns with endoscopic findings in a single, well-defined cohort. The mean age of the participants was 35.8 years, and females slightly predominated (53.7%). These demographic findings are consistent with regional dyspepsia studies reporting higher healthcare-seeking behavior among younger and middle-aged adults, particularly women.[13] Although baseline characteristics such as age, sex, BMI, residence, smoking status, and symptom duration did not significantly differ among dyspepsia subtypes, the findings suggest that symptom pattern is influenced more strongly by pathophysiological mechanisms than by demographic variables alone.

This study appraised the clinicoendoscopic patterns of dyspepsia among *H. pylori*–positive adults without alarm features and demonstrated that mixed dyspepsia was the predominant symptom pattern, accounting for 55.0% of cases. EPS and PDS constituted 28.8% and 16.3% respectively. These findings are comparable with previous Bangladeshi observations, where overlapping EPS-PDS symptoms were also the most frequently reported dyspepsia pattern.[13] Similar findings from community-based South Asian studies further suggest that symptom overlap is common among dyspeptic patients, particularly in populations with high *H. pylori* prevalence.[14]

The predominance of mixed dyspepsia in the study may reflect the multifactorial pathophysiology of dyspepsia, involving gastric hypersensitivity, impaired accommodation, delayed gastric emptying, mucosal inflammation, and altered gutbrain interaction.[17,18] The coexistence of pain-related and meal-related symptoms indicates that rigid symptom-based classification may not adequately represent the complexity of dyspeptic disorders.

An important finding of the present study was the nearly equal distribution between functional dyspepsia (51.2%) and organic dyspepsia (48.8%). Previous studies have similarly demonstrated that organic lesions remain common among dyspeptic patients in developing countries because of persistent *H. pylori* infection and chronic gastritis.[19]

Normal endoscopy was significantly more frequent among patients with mixed dyspepsia, whereas abnormal endoscopic findings were predominantly observed in EPS and PDS subtypes. This finding suggests that overlapping symptom patterns may be more closely related to functional gastrointestinal disturbances than to discrete structural lesions.

Among abnormal endoscopic findings, erosive gastritis was the most common lesion (23.8%), followed by nonerosive gastritis (15.0%). Duodenal ulcer and gastric ulcer were

identified in smaller proportions. Similar endoscopic profiles have been reported in studies from Cambodia and other Asian populations, where gastritis and peptic ulcer disease represented the most frequent abnormalities among chronic dyspepsia patients.[15] Chronic *H. pylori* infection induces mucosal inflammation and epithelial injury, which may contribute to these findings.[11,17]

The present study also demonstrated significant differences in symptom distribution between functional and organic dyspepsia. Epigastric pain, epigastric burning, postprandial fullness, and early satiety were all significantly more frequent in FD compared with OD. These findings indicate that symptom severity and overlap may be more pronounced in functional disorders, potentially reflecting heightened visceral sensitivity and altered central pain processing.[18,20] In contrast, bloating, belching, and nausea did not differ significantly between FD and OD, suggesting lower discriminatory value for these symptoms.

Another important observation was the significant association between age ≥34 years and organic dyspepsia. Older age has consistently been associated with increased prevalence of structural gastrointestinal pathology because of cumulative mucosal injury, prolonged *H. pylori* exposure, and higher likelihood of chronic gastritis or ulcer disease. Smoking history was also significantly associated with organic dyspepsia. Smoking may impair gastric mucosal defense and potentiate inflammation, thereby increasing susceptibility to peptic ulceration.[21]

Multinomial logistic regression analysis demonstrated that symptom duration ≥12 months independently predicted mixed dyspepsia. Chronicity of symptoms may reflect persistent functional disturbances, prolonged inflammatory exposure, or recurrent symptom overlap. In addition, normal endoscopy/functional dyspepsia was strongly associated with mixed dyspepsia (OR 17.00; $p<0.001$), supporting the hypothesis that overlapping symptom phenotypes are predominantly functional in origin.

The findings of this study have important clinical implications. In resource-limited settings such as Bangladesh, symptom-based assessment alone may not reliably predict endoscopic pathology among *H. pylori*–positive patients. Although mixed dyspepsia was commonly associated with functional disease, EPS and PDS subtypes demonstrated higher frequencies of organic lesions. Therefore, integration of symptom profile with endoscopic evaluation may improve patient stratification and optimize use of diagnostic resources.

LIMITATION OF THE STUDY

The study was conducted in a single tertiary care center with a relatively small sample size, which may limit generalizability. Histopathological correlation and longterm followup were not performed.

CONCLUSION

Mixed dyspepsia was the predominant symptom phenotype among *H. pylori*–positive adults without alarm features. Functional dyspepsia was slightly more common than organic dyspepsia and was strongly associated with overlapping symptom patterns and normal endoscopy. In contrast, EPS and PDS demonstrated a greater association with abnormal endoscopic findings. Age and smoking were significantly associated with organic dyspepsia, while prolonged symptom duration and normal endoscopy independently predicted mixed dyspepsia. These findings support the importance of an integrated clinicoendoscopic approach for evaluation and stratification of dyspeptic patients in clinical practice.

ACKNOWLEDGMENT

The authors thank all patients who participated in this study. We also acknowledge the support of the Department of Gastroenterology, Bangladesh Medical University.

Conflict of Interest: The authors declare no conflict of interest.

Funding: No external funding was received.

REFERENCES

1. Harer KN, Hasler WL. Functional dyspepsia: a review of the symptoms, evaluation, and treatment options. Gastroenterol Hepatol (N Y). 2020;16(2):66–74.
2. Chuah KH, Cheong SY, Lim SZ, Mahadeva S. Functional dyspepsia leads to more healthcare utilization in secondary care compared with other functional gastrointestinal disorders. J Dig Dis. 2022; 23(2):111-117.
3. Francis P, Zavala SR. Functional dyspepsia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. [Updated 2024 Jun 8]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554563/
4. Miwa H, Ghoshal UC, Gonlachanvit S, Gwee KA, Ang TL, Chang FY, et al. Asian consensus report on functional dyspepsia. J Neurogastroenterol Motil. 2012;18(2):150-168

5. Lee K, Kwon CI, Yeniova AÖ, Koyanagi A, Jacob L, Smith L, et al. Global prevalence of functional dyspepsia according to Rome criteria, 1990–2020: a systematic review and meta-analysis. Sci Rep. 2024;14(1):4172.
6. Drossman DA, Hasler WL. Rome IV—functional GI disorders: disorders of gut-brain interaction. Gastroenterology. 2016;150(6):1257–1261.
7. Shin CM. Overlap between postprandial distress and epigastric pain syndromes in functional dyspepsia: its implications for research and clinical practice. J Neurogastroenterol Motil. 2013;19(3):409–411.
8. Mao LQ, Wang SS, Zhou YL, Chen L, Yu LM, Li M, et al. Clinically significant endoscopic findings in patients of dyspepsia with no warning symptoms: a cross-sectional study. World J Clin Cases. 2021;9(15):3597–3606.
9. Jung HK, Kim SE, Shim KN, Jung SA. The role of Rome III criteria in the diagnosis of functional dyspepsia. Korean J Gastroenterol. 2012;59(4): 275–281.
10. Kharel S, Bist A, Shrestha S, Homagain S. Helicobacter pylori in healthy South Asians. JGH Open. 2020;4(6):1037–46.
11. Malfertheiner P, Camargo MC, El-Omar E, Liou JM, Peek R, Schulz C, et al. Helicobacter pylori infection. Nat Rev Dis Primers. 2023;9(1):19.
12. Talley NJ, Hunt RH. What role does Helicobacter pylori play in dyspepsia and nonulcer dyspepsia? Arguments for and against H. Pylori being associated with dyspeptic symptoms. Gastroenterology. 1997;113(6 Suppl):S67–77.
13. Sarkar MAM, Arefin MS, Khan MMR, Saha M, Alam MR, Ghosh CK, et al. Demographic and clinical characteristics of functional dyspepsia and its subtypes in adult patients: an experience from a tertiary care centre in Bangladesh. Mymensingh Med J. 2024; 33(2):426–32.
14. Rahman MM, Ghoshal UC, Kibria MG, Sultana N, Yusuf MA, Nahar S, et al. Functional dyspepsia, peptic ulcer, and Helicobacter pylori infection in a rural community of South Asia: an endoscopy-assisted household survey. Clin Transl Gastroenterol. 2021;12(4):e00334.
15. Oung B, Chea K, Oung C, Saurin JC, Ko CW. Endoscopic yield of chronic dyspepsia in outpatients: a single-center experience in Cambodia. JGH Open. 2019;4(1):61–8.
16. Wang L, Shi Y, Li Y, Wang X, Xu L, Zhang Y. Pathophysiological mechanisms of functional dyspepsia: a narrative review. Front Med (Lausanne). 2025;12:1624242.
17. Kim SH. Gastritis: pathophysiology, diagnosis, and clinical implications. Korean J Helicobacter Up Gastrointest Res. 2026;26(1):8–14.
18. Sgambato D, Capuano A, Sullo MG, Miranda A, Federico A, Romano M. Gut-brain axis in gastric mucosal damage and protection. Curr Neuropharmacol. 2016;14(8):959–66.
19. Mohammed M. Correlation of endoscopic findings with various Helicobacter pylori tests among dyspeptic patients. Int J Clin Med. 2014;5: 1180-1188.
20. Corsetti M, Fox M. The management of functional dyspepsia in clinical practice: what lessons can be learnt from recent literature? F1000Res. 2017;6:1778.
21. Maity P, Biswas K, Roy S, Banerjee RK, Bandyopadhyay U. Smoking and the pathogenesis of gastroduodenal ulcer—recent mechanistic update. Mol Cell Biochem. 2003;253(1–2):329–338.