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Prevalence of reflux esophagitis and its association with helicobacter pylori in patients with gastrointestinal system complaints

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Abstract

This study examined patients with gastrointestinal disorders to evaluate the prevalence of reflux esophagitis and its association with H. pylori infection. The primary objective was to determine whether H. pylori infection contributes to the development of reflux esophagitis. A retrospective analysis was conducted on 660 patients who underwent upper endoscopy at Adiyaman University Training and Research Hospital between January and August 2020. Patients were stratified into H. pylori-positive and H. pylori-negative groups. Clinical parameters evaluated included the presence and severity of reflux esophagitis, demographic characteristics, risk factors, and associated complications. Sample size calculation was performed using G*Power 3.1 software (α=0.05, power=0.80). Proton pump inhibitor (PPI) usage patterns were categorized as never used, irregular use, or regular use, while body mass index (BMI) was classified as normal (<25 kg/m²), overweight (25-30 kg/m²), or obese (>30 kg/m²). The mean age of the study population was 45.4±16.4 years, with 52.6% being male. The overall prevalence of reflux esophagitis was 12.7%. H. pylori infection was detected in 63% of patients. The prevalence of reflux esophagitis was 11.3% in H. pylori-positive patients compared to 15.2% in H. pylori-negative patients (p=0.150). Hiatal hernia (OR: 3.2), obesity (OR: 2.8), and smoking (OR: 1.9) emerged as the most significant risk factors. Regarding PPI usage patterns, reflux esophagitis was observed in 10.3% of non-users, 13.9% of irregular users, and 18.7% of regular users (p=0.015). Although the prevalence of reflux esophagitis was lower in H. pylori-positive patients, the difference did not reach statistical significance. This finding may be attributed to the study's relatively low statistical power (0.52). Hiatal hernia and obesity were identified as the most significant risk factors for reflux esophagitis development.

Keywords: Helicobacter pylori, reflux esophagitis, gastroesophageal reflux disease, endoscopy

Introduction

Gastroesophageal reflux (GER) is defined as a physiological process that occurs when gastric contents flow back into the esophagus due to insufficient antireflux barrier function. In contrast, gastroesophageal reflux disease (GERD) is characterized by symptoms such as heartburn and acid regurgitation, and may present with esophageal mucosal damage (erosive GERD) or without mucosal damage (non-erosive reflux disease, NERD) [1,2]. GERD is a prevalent gastrointestinal disorder that significantly impairs quality of life and imposes a substantial economic burden on healthcare systems. Advanced disease stages may result in serious complications, including Barrett's esophagus, esophageal stricture, hemorrhage, perforation, and esophageal adenocarcinoma [3]. Reflux esophagitis is an inflammatory condition of the esophageal mucosa resulting from the reflux of acidic gastric contents into the esophagus [4].

Helicobacter pylori (H. pylori), a bacterium that infects more than half of the world's population, has drawn attention regarding its potential connection with gastroesophageal reflux disease [5]. In developed countries, the parallel decline in H. pylori infection and associated gastroduodenal diseases, alongside an increase in GERD and esophageal adenocarcinoma incidence, has generated considerable scientific debate regarding H. pylori's potential protective role in reflux disease pathogenesis [6]. The substantial increase in GERD prevalence in North America and Europe over the past two decades has been attributed to multiple factors,

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including demographic aging, increasing obesity rates, dietary and lifestyle modifications, and declining H. pylori infection rates [7].

The primary hypothesis of our study is that an association exists between H. pylori infection and the development of reflux esophagitis. The direction of this relationship remains controversial in literature: some studies report a protective effect while others demonstrate an increased risk. Therefore, our study aimed to determine the role of H. pylori in gastroesophageal reflux among patients with gastrointestinal symptoms and to elucidate the nature of this association.

The relationship between GERD and H. pylori infection remains incompletely understood in literature. While some researchers suggest that H. pylori may have a protective effect against Some studies suggest that H. pylori may protect against GERD by affecting gastric mucosa and reducing acid production, while others report that this bacterium might increase the risk of reflux esophagitis [8-10]. The global prevalence of GERD varies between 7-25%, though the reasons for geographical variations are not fully understood [9]. Turkish epidemiological studies demonstrate that GERD prevalence parallels that observed in Western populations; however, the clinical phenotype differs significantly, with a predominance of regurgitation over pyrosis as the primary symptom [8].

Our study aims to evaluate the role and effects of H. pylori infection in patients diagnosed with GERD and reflux esophagitis. We assessed the association between H. pylori infection and reflux esophagitis, focusing on its impact on mucosal inflammation development, complications, and various demographic factors. Additionally, we sought to determine how H. pylori positivity interacts with established risk factors, including hiatal hernia, smoking, and body mass index. These analyses were conducted to enhance understanding of the mechanisms underlying H. pylori influence on GERD and contribute to the existing literature.

Material and Methods

This retrospective study was conducted to evaluate the prevalence of reflux esophagitis and its relationship with Helicobacter pylori (H. pylori) in patients aged 18 years and older who underwent esophagogastroduodenoscopy (EGD) at the Endoscopy Unit of Adiyaman University Training and Research Hospital between January 14, 2020, and August 30, 2020. The study was conducted in accordance with the Declaration of Helsinki and received ethical approval from the Scientific Research Evaluation Committee of Adiyaman University Faculty of Medicine (Decision No: 2025/5-2, dated May 27, 2025).

Patient Selection

Inclusion criteria encompassed all adult patients aged 18 years and older who underwent EGD with histopathological biopsies obtained from both the gastric antrum and corpus regions during the specified study period.

Exclusion criteria included: (1) patients under 18 years of age; (2) absence of histopathological biopsies from both antrum and corpus regions; (3) history of gastric surgery; (4) presence of gastric outlet obstruction; (5) diagnosed esophageal or gastric malignancy; (6) active gastrointestinal bleeding; (7) insufficient clinical information in medical records; and (8) history of coagulopathy.

A total of 660 patients who met the inclusion criteria were enrolled in the study. Clinical and demographic data were retrospectively extracted from the hospital's electronic medical records.

Sample size calculation was performed using G*Power 3.1 software. To detect a 4% difference in reflux esophagitis prevalence between H. pylori-positive and H. pylori-negative groups (11.3% vs. 15.2%, respectively), with α=0.05 and β=0.20 (power=0.80), a minimum of 582 patients per group (total n=1.164) was required.

With our achieved sample size of 660 patients (H. pylori-positive: n=416; H. pylori-negative: n=244), post-hoc power analysis revealed an actual study power of 0.52. This indicates a high probability of Type II error (β=0.48), suggesting that any lack of statistical significance between groups might be attributable to insufficient sample size rather than true absence of effect.

Data Collection

All endoscopic procedures were performed by a single experienced endoscopist using a Fujinon video endoscope to ensure consistency and minimize inter-observer variability. Patients received topical anesthesia with lidocaine (xylocaine) prior to the procedure. During endoscopic evaluation, all anatomical regions from the esophagus to the second portion of the duodenum were systematically examined. When reflux esophagitis was identified, grading was performed according to the Los Angeles (LA) classification system. Hiatal hernia was defined as displacement of the lower esophageal sphincter (LES) ≥2 cm above the diaphragmatic hiatus into the thoracic cavity.

Two biopsies from the gastric antrum and two from the corpus were obtained from all patients for histopathological examination and H. pylori detection. Specimens were immediately fixed in 10% neutral buffered formalin and sent to the pathology laboratory. Histological examination was conducted using hematoxylin-eosin (H&E) and modified Giemsa staining. The presence of H. pylori was evaluated under light microscopy at 400× magnification.

Patient medication history, including nonsteroidal anti-inflammatory drug (NSAID) use, duration of therapy, and dosage, was systematically documented from medical records and patient interviews.

Evaluated Parameters

Endoscopic Assessment: Endoscopic findings were systematically classified as antral gastritis, pangastritis, erosive superficial gastritis, erosive duodenitis, hiatal hernia, gastric

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ulcer, and duodenal ulcer. The presence and severity of reflux esophagitis were graded according to the Los Angeles (LA) classification system.

Histopathological Evaluation: In addition to H. pylori status, the following parameters were assessed: inflammation severity, inflammatory activity, intestinal metaplasia, and associated complications using standardized criteria.

Patient Characteristics: Demographic and clinical variables included smoking status, NSAID use, proton pump inhibitor (PPI) therapy (categorized as never used, irregular use, or regular use), gender, and age. Body mass index (BMI) was categorized as normal weight (<25 kg/m²), overweight (25-29.9 kg/m²), or obese (≥30 kg/m²).

Specialized Diagnoses: Barrett's esophagus was diagnosed when endoscopic proximal displacement of the squamocolumnar junction was accompanied by histopathological confirmation of specialized intestinal metaplasia. Esophageal stricture was defined as luminal narrowing to <13 mm diameter on endoscopic measurement.

All findings were systematically evaluated through correlation of endoscopic and histopathological data.

Statistical Analysis

Data were analyzed using SPSS software version 20.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics for categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation or median (interquartile range) depending on data distribution.

Normality of continuous variables was assessed using the Shapiro-Wilk test. For categorical variables, associations were analyzed using Pearson's chi-square test or Fisher's exact test when expected cell counts were <5. Non-parametric continuous variables were compared using the Mann-Whitney U test for two groups.

Statistical significance was set at $p<0.05$. All tests were two-tailed, and 95% confidence intervals were calculated where appropriate.

Result

A total of 660 patients were included in the study analysis. The mean age was 45.4±16.4 years, with 347 (52.6%) male and 313 (47.4%) female participants. BMI analysis revealed that 245 patients (37.1%) had normal weight (<25 kg/m²), 268 (40.6%) were overweight (25-29.9 kg/m²), and 147 (22.3%) were obese (≥30 kg/m²). Assessment of risk factors showed current smoking in 228 patients (34.5%) and regular NSAID use in 156 patients (23.6%). The most frequent presenting symptom was epigastric pain in 412 patients (62.4%), followed by retrosternal burning sensation in 386 patients (58.5%), regurgitation in 298 patients (45.2%), and dysphagia in 124 patients (18.8%). Endoscopic evaluation demonstrated antral gastritis as the most prevalent finding in 427 patients (64.7%), followed by pangastritis in 194 patients (29.4%), erosive duodenitis in 49 patients (7.4%), duodenal ulcer in 40 patients (6.1%), erosive superficial gastritis in 32 patients (4.8%), and gastric ulcer in 15 patients (2.3%) (Table 1).

The overall prevalence of reflux esophagitis was 12.7% (n=84) among the study population. Male patients demonstrated a significantly higher frequency of reflux esophagitis compared to females (54 patients [15.6%] vs. 30 patients [9.6%], respectively; $p=0.021$). Hiatal hernia presence showed a strong association with reflux esophagitis development, with rates increasing from 7.8% in patients without hiatal hernia to 50.0% in those with hernias >4 cm in size ($p=0.008$). Analysis of proton pump inhibitor (PPI) use history revealed reflux esophagitis rates of 10.3% in never-users, 13.9% in irregular users, and 18.7% in regular users ($p=0.015$). According to the Los Angeles (LA) classification system, among patients diagnosed with esophagitis, 69 patients (82.1%) were classified as grade A, 9 patients (10.7%) as grade B, 4 patients (4.8%) as grade C, and 2 patients (2.4%) as grade D (Table 2).

Helicobacter pylori positivity was detected in 416 patients (63.0%) of the total study population. Among H. pylori-positive patients, rapid urease testing was positive in 386 cases (92.8%), while histopathological examination was performed in all patients for confirmation. Assessment of gastritis characteristics demonstrated significantly higher inflammation severity in H. pylori-positive patients compared to negative patients ($p<0.001$); severe inflammation was present in 94 patients (22.6%) in the H. pylori-positive group versus 10 patients (4.1%) in the negative group. Similarly, gastritis activity was significantly more frequent in H. pylori-positive patients compared to negative patients (260 patients [62.5%] vs. 46 patients [18.9%], respectively; $p<0.001$). Previous H. pylori eradication treatment history was documented in 118 patients (28.4%) of the positive group compared to 48 patients (19.7%) of the negative group ($p=0.032$) (Table 3).

Evaluation of complications and associated risk factors revealed Barrett's esophagus in 15 patients (2.3%) with an odds ratio of 3.1 (95% CI: 1.7-5.6, $p=0.001$), esophageal stricture in 12 patients (1.8%) with an OR of 2.4 (95% CI: 1.3-4.2, $p=0.005$), and gastrointestinal bleeding in 8 patients (1.2%) with an OR of 1.8 (95% CI: 0.9-3.6, $p=0.089$). Among identified risk factors for reflux esophagitis development, the strongest association was observed with hiatal hernia presence (OR: 3.2, 95% CI: 1.9-5.4, $p<0.001$), followed by obesity (OR: 2.8, $p=0.001$) and current smoking (OR: 1.9, $p=0.008$). Intestinal metaplasia was detected in 51 H. pylori-positive patients (12.3%) compared to 39 H. pylori-negative patients (16.0%), showing no significant association (OR: 0.7, 95% CI: 0.4-1.2, $p=0.178$). The frequency of reflux esophagitis was lower in H. pylori-positive patients compared to H. pylori-negative patients (47 patients [11.3%] vs. 37 patients [15.2%], respectively; OR: 0.8, 95% CI:0.5-1.3, $p=0.150$) (Table 4).

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Table 1. Distribution of demographic and clinical characteristics

Characteristic	Category	Count (%)
Age (Mean±SD)	45.4±16.4	-
Gender	Male	347 (52.6)
	Female	313 (47.4)
BMI (kg/m²)	<25	245 (37.1)
	25–30	268 (40.6)
	>30	147 (22.3)
Smoking Status	Yes	228 (34.5)
	No	432 (65.5)
NSAID Use	Yes	156 (23.6)
	No	504 (76.4)
Presenting Symptoms*	Epigastric pain	412 (62.4)
	Retrosternal burning	386 (58.5)
	Regurgitation	298 (45.2)
	Dysphagia	124 (18.8)
Endoscopic Findings*	Antral gastritis	427 (64.7)
	Pangastritis	194 (29.4)
	Erosive superficial gastritis	32 (4.8)
	Erosive bulbitis	49 (7.4)
	Gastric ulcer	15 (2.3)
	Bulbar ulcer	40 (6.1)

*SD: Standard deviation BMI: Body mass index NSAID: Non-steroidal anti-inflammatory drug *A patient may present with more than one symptom or finding

Table 2. Characteristics of reflux esophagitis and associated factors

Characteristic	No Reflux Esophagitis n (%)	Reflux Esophagitis n (%)	p-value
Total	576 (87.3)	84 (12.7)	-
Gender			
- Male	293 (84.4)	54 (15.6)	0.021
- Female	283 (90.4)	30 (9.6)	
Hiatal Hernia			
- None	498 (92.2)	42 (7.8)	
- <2 cm	52 (76.5)	16 (23.5)	0.008
- 2–4 cm	26 (65.0)	14 (35.0)	
- >4 cm	12 (50.0)	12 (50.0)	
PPI Use			
- Never Used	312 (89.7)	36 (10.3)	0.015
- Irregular Use	186 (86.1)	30 (13.9)	
- Regular Use	78 (81.3)	18 (18.7)	
Esophagitis Stage			
- Stage A	-	69 (82.1)	
- Stage B	-	9 (10.7)	-
- Stage C	-	4 (4.8)	
- Stage D	-	2	

*SD: PPI: Proton pump inhibitor hiatal hernia measurements are categorized based on the size of the hernia. esophagitis stages (a-d) are classified according to the severity of mucosal damage

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Table 3. H. pylori and associated characteristics

Characteristic	H. pylori Negative n (%)	H. pylori Positive n (%)	p-value
Total	244 (37.0)	416 (63.0)	-
Gender			
- Male	132 (38.0)	215 (62.0)	0.549
- Female	112 (35.8)	201 (64.2)	
Diagnostic Method*			
- Rapid urease test	-	386 (92.8)	-
- Histopathology	-	416 (100.0)	
Gastritis Characteristics			
- Inflammation severity			
-- Mild	186 (76.2)	124 (29.8)	
-- Moderate	48 (19.7)	198 (47.6)	0.001
-- Severe	10 (4.1)	94 (22.6)	
- Activity			
-- Absent	198 (81.1)	156 (37.5)	
-- Present	46 (18.9)	260 (62.5)	
Previous H. pylori Treatment			
- No	196 (80.3)	298 (71.6)	0.032
- Yes	48 (19.7)	118.4)	

A single patient may have undergone more than one diagnostic method. Rapid urease tests and histopathology are used for H. pylori detection. Inflammation severity categories include mild, moderate, and severe based on histopathological assessment. The presence of activity indicates active gastritis

Table 4. Clinical outcomes and risk factors

Characteristic	n (%)	OR (95% CI)	p-value
Complications			
- Stricture	12 (1.8)	2.4 (1.3–4.2)	0.005
- Bleeding	8 (1.2)	1.8 (0.9–3.6)	0.089
- Barrett's esophagus	15 (2.3)	3.1 (1.7–5.6)	0.001
Risk Factors			
- BMI >30 kg/m²	147 (22.3)	2.8 (1.6–4.9)	0.001
- Hiatal hernia	142 (21.5)	3.2 (1.9–5.4)	<0.001
- Smoking	228 (34.5)	1.9 (1.2–3.1)	0.008
- NSAID Use	156 (23.6)	1.6 (0.9–2.8)	0.102
Intestinal Metaplasia			
- H. pylori (-)	39 (16.0)	1.0 (reference)	0.178
- H. pylori (+)	51 (12.3)	0.7 (0.4–1.2)	
Reflux Esophagitis			
- H. pylori (-)	37 (15.2)	1.0 (reference)	0.150
- H. pylori (+)	47 (11.3)	0.8	

OR: Odds ratio CI: Confidence interval barrett's esophagus, stricture, and bleeding are complications assessed for clinical significance. Risk factors include BMI >30 kg/m², hiatal hernia, smoking, and NSAID use. The reference group (OR=1.0) is indicated for comparisons in intestinal metaplasia and reflux esophagitis between H. pylori negative and positive groups

Discussion

The purpose of the present study was to assess the occurrence of reflux esophagitis and its correlation with Helicobacter pylori infection in patients who underwent upper endoscopy for gastrointestinal symptoms. Our determined reflux esophagitis prevalence of 12.7% (84/661 patients) is consistent with similar contemporary studies reported in the literature. In particular, our findings are supported by research conducted on Japanese patients, which reported a comparable rate of 13.7%. However, some studies have reported lower prevalence rates than our findings. For example, an investigation conducted in Iran observed a

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reflux esophagitis rate of 6.4%. Such inter-study variations could be attributed to differences in ethnicity, genetic characteristics of the study populations, and varying diagnostic criteria employed. Moreover, differences in endoscopic techniques, operator experience, and patient selection criteria might have contributed to these discrepancies in reported prevalence rates.

Our study demonstrated a reflux esophagitis prevalence of 12.7% (84/661 patients), which is consistent with rates reported in contemporary literature. Particularly, our findings are supported by studies conducted in Japanese populations, where Osaga et al. reported a comparable prevalence of 13.7%, and Lin et al. found a similar rate of 13.7%. These consistent findings across different study populations suggest that our observed prevalence rate represents a reliable estimate for reflux esophagitis occurrence in patients undergoing upper endoscopy for gastrointestinal symptoms [11-15]. However, some studies have reported lower prevalence rates than our findings. For instance, Haghighat et al. observed a reflux esophagitis prevalence of only 6.4% in their Iranian population study. Such inter-study variations in reported prevalence rates could be attributed to several factors, including differences in patient demographics, ethnic background, diagnostic criteria employed, endoscopic grading systems used, and varying patient selection criteria across different healthcare settings [12]. Such variations could be attributed to differences in patient demographics, lifestyle factors, and diagnostic criteria employed across different study populations.

Regarding gender distribution, our analysis revealed a significantly higher prevalence of reflux esophagitis among males (15.6%) compared to females (9.6%) ($p<0.021$). This finding is consistent with previous studies demonstrating a male predominance in reflux esophagitis. Similarly, Adkins et al. reported a higher prevalence of reflux esophagitis in male patients, while Haghighat et al. found that 63.1% of patients with reflux esophagitis were male and 36.9% were female, further supporting the observed gender disparity in our study population [12]. Similarly, Laserna-Mendieta et al. corroborated our findings by demonstrating greater pathological severity of reflux esophagitis among male patients [16].

The mean age in our study (45.4±16.4 years) aligns with previous research findings. Lin et al. reported a comparable mean age of 49.9±11.0 years, supporting the consistency of age distribution across different populations with reflux esophagitis [15]. The mean age in our study (45.4±16.4 years) aligns with previous research findings. Lin et al. reported a comparable mean age of 49.9±11.0 years, supporting the consistency of age distribution across different populations with reflux esophagitis [14].

In our study, hiatal hernia emerged as the strongest risk factor for reflux esophagitis (OR:3.2). This finding closely aligns with OR:3.42 reported by Shiota and colleagues [17]. Patients with large hiatal hernias (>4 cm) demonstrated particularly high rates of reflux esophagitis, reaching up to 50%. This strong association is attributed to the anatomical disruption of the lower esophageal sphincter mechanism and impaired anti-reflux barrier function. Mehta et al. further substantiated these findings, demonstrating that hiatal hernia represents a significant risk factor for reflux esophagitis, with a more pronounced effect observed in male patients [18].

Obesity was identified as the second most significant risk factor in our study (OR:2.8). This ratio approximates the OR:3.62 reported by Sreekala and colleagues [19]. BMI analysis revealed that 40.6% of our study population was overweight, while 22.3% was obese. Our obesity prevalence closely matches the 22.1% reported by Taraszewska et al., indicating consistent patterns of weight distribution among patients with reflux esophagitis across different study populations [20]. Furthermore, Shiota et al. demonstrated that 50.3% of erosive esophagitis cases occurred in obese patients, providing additional evidence for obesity as a major risk factor in the development of reflux esophagitis [17].

Smoking was identified as a significant risk factor in our study (OR:1.9). While this finding is lower than the OR:2.8 reported in Okamoto and Ito's study, it still indicates a substantial increase in risk [21]. Sreekala and colleagues reported an even higher risk increase associated with smoking, with an OR of 3.71 [19]. Almutairi et al. further corroborated these findings, demonstrating that the concurrent presence of obesity and hiatal hernia creates a synergistic effect that substantially amplifies the risk of developing reflux esophagitis compared to either risk factor alone [22].

Our study demonstrated a high H. pylori prevalence of 63%, which, while lower than the 75.3% reported by Niknam et al., still represents a substantial burden of infection that may contribute to the pathogenesis of reflux esophagitis in our study population. [23]. Our analysis revealed a lower frequency of reflux esophagitis among H. pylori-positive patients compared to H. pylori-negative patients (11.3% vs. 15.2%), although this difference did not reach statistical significance ($p=0.150$). This finding is consistent with Niknam et al., who reported comparable rates with similarly non-significant results ($p=0.214$), suggesting that H. pylori infection may not be a primary determinant in the development of reflux esophagitis [23].

Hojo et al. demonstrated a significant increase in reflux esophagitis incidence following H. pylori eradication therapy, providing compelling evidence for a potential protective role of H. pylori against gastroesophageal reflux disease through its effects on gastric acid production and motility. [24]. Similarly, Zhao et al. corroborated these findings by demonstrating significant protective effects of H. pylori against reflux esophagitis and documented markedly increased esophageal acid exposure following eradication therapy, further supporting the hypothesis that H. pylori infection may modulate gastric acid secretion and provide a protective barrier against gastroesophageal reflux. [25]. The observed lower frequency of reflux esophagitis among H. pylori-positive patients in our cohort (11.3% vs. 15.2% in H. pylori-negative patients) provides additional support for

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the protective effect hypothesis, reinforcing the growing body of evidence that H. pylori infection may confer a defensive mechanism against the development of gastroesophageal reflux disease through modulation of gastric physiology.

A recent study by Sadighi et al. demonstrated that H. pylori infection may reduce the frequency of gastroesophageal reflux disease symptoms, although this protective effect did not achieve statistical significance, nonetheless contributing to the accumulating evidence supporting the potential beneficial role of H. pylori in GERD prevention [26]. This finding is consistent with our results, as Du et al. similarly reported no statistically significant association between H. pylori infection and reflux esophagitis (p=0.92), while interestingly documenting a reduced risk of Barrett's esophagus in H. pylori-positive patients, suggesting that the protective effects of H. pylori may be more pronounced in preventing advanced metaplastic changes rather than early inflammatory stages of gastroesophageal reflux disease [27].

In our study, the observed rate of severe inflammation in H. pylori-positive patients (22.6%) closely corresponds to the findings reported by Arismendi-Morillo et al., demonstrating remarkable consistency across different populations. Similarly, our documented 4.1% rate of severe inflammation in H. pylori-negative patients is consistent with established literature ranges (3.2-5.8%), further validating our methodological approach and confirming the well-documented association between H. pylori infection and increased gastric inflammatory response [28]. This marked difference illustrates the pronounced proinflammatory effect of H. pylori.

Analysis of inflammatory activity revealed a significant difference between H. pylori-positive patients (62.5%) and H. pylori-negative patients (18.9%). In line with these findings, Tang et al. reported that H. pylori induces gastric mucosal injury through the upregulation of proinflammatory cytokines [29]. The study by Saracino and colleagues further supports our findings through their detailed analysis of H. pylori–induced inflammatory responses [30].

The distribution of antral gastritis (64.7%) and pangastritis (29.4%) in our cohort aligns with rates previously reported in the literature. Similarly, Eletto and colleagues emphasized the distinct association between H. pylori infection and antral gastritis [31]. He et al. recently supported the high prevalence of antral gastritis and further demonstrated that dietary factors play a significant role in shaping inflammatory responses [32]. In their study, Rueda-Robles et al. examined the influence of dietary factors on inflammation in the context of H. pylori infection and underscored the critical role of environmental determinants in shaping gastritis patterns [33].

Our analysis of proton pump inhibitor (PPI) uses patterns showed reflux esophagitis in 18.7% of regular users, 10.3% of non-users, and 13.9% of irregular users. The higher prevalence among regular PPI users is likely reflected, as noted by Huh and colleagues, that these patients often experience a longer disease duration and a more severe clinical course. The same study also highlighted that inconsistent PPI use compromises disease control and is associated with reduced patient satisfaction [34].

Our findings on PPI usage patterns are consistent with the 30% regular use rate reported by Huh and colleagues. We also observed that a considerable proportion of patients either used the therapy irregularly or discontinued it on their own initiative [34]. Targownik and colleagues reported a high likelihood of relapse following discontinuation of reflux esophagitis therapy, which may account for the higher prevalence of reflux esophagitis observed among regular PPI users in our study [35].

With respect to response to PPI therapy, Yadlapati and colleagues emphasized the importance of employing ambulatory reflux monitoring in patients who do not respond adequately, and of considering endoscopic or surgical interventions when clinically indicated [36]. Long-term PPI therapy has been reported to be particularly beneficial in patients with conditions such as Barrett’s esophagus or severe erosive esophagitis [35]. These findings suggest that the subgroup of patients requiring regular PPI therapy in our study likely represents more complex and treatment-resistant cases.

There are several limitations to our study that should be acknowledged. First, the single-center, retrospective design inherently restricts the generalizability of our findings. Second, as the study cohort consisted exclusively of patients who underwent endoscopy, the reported prevalence of reflux esophagitis may not accurately reflect that of the general population. Furthermore, the reliance on patient recall for constructing medication histories, along with the absence of long-term follow-up data, represent additional limitations.

Conclusion

Our findings demonstrated a prevalence of reflux esophagitis of 12.7%, with hiatus hernia, obesity, and smoking identified as the principal risk factors. Although no statistically significant association was observed between Helicobacter pylori positivity and reflux esophagitis, infected patients exhibited a lower prevalence, suggesting a potential protective effect of H. pylori against this condition. Analysis of PPI use revealed prevalence rates of 18.7% among regular users, 10.3% among non-users, and 13.9% among irregular users. Most cases of reflux esophagitis detected endoscopically were classified as mild. Collectively, these findings emphasize the importance of addressing modifiable risk factors and implementing appropriate therapeutic strategies in the management of reflux esophagitis.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
The study received ethical approval from the Scientific Research Evaluation Committee of Adiyaman University Faculty of Medicine (Decision No: 2025/5-2, 27.05.2025).

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