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Evaluation of *helicobacter pylori* prevalence in patients with bile reflux using antral and corpus biopsies

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ABSTRACT

Introduction: Alkaline reflux gastritis (ARG) is a chronic inflammatory condition caused by exposure of gastric mucosa to bile and duodenal contents. The relationship between bile reflux and *Helicobacter pylori* infection remains controversial. This study aimed to evaluate *H. pylori* prevalence and histopathological findings in patients with endoscopically detected bile reflux.

Materials and Methods: This retrospective observational study included 136 patients with bile reflux who underwent simultaneous antrum and corpus biopsies between January 2022 and January 2024. Histopathological examinations were performed using hematoxylin-eosin and modified Giemsa stains. *H. pylori* status, gastritis type, inflammation severity, activity, atrophic changes, intestinal metaplasia, and lymphoid aggregates were evaluated according to Sydney classification.

Results: *H. pylori* was positive in 76 patients (55.9%) overall, with higher prevalence in antrum (51.5%) compared to corpus (43.4%). Chronic active gastritis was significantly more common in *H. pylori* positive patients in both antrum (74.3% vs 18.2%, p<0.001) and corpus (78.0% vs 14.3%, p<0.001). Inflammation severity was significantly higher in *H. pylori* positive patients in both locations (p<0.001). Intestinal metaplasia was three times more frequent in antrum than corpus (14.7% vs 4.5%). Lymphoid aggregates were significantly more common in *H. pylori* positive patients in antrum (48.6% vs 21.2%, p<0.001).

Conclusion: Despite bile reflux presence, *H. pylori* prevalence remains high (55.9%), suggesting that endoscopically observed bile may reflect transient reflux rather than chronic alkaline reflux gastritis. The synergistic effect of *H. pylori* and bile reflux leads to more severe inflammatory changes. Histopathological confirmation is essential for alkaline reflux gastritis diagnosis, as endoscopic bile presence alone is insufficient.

Keywords: Alkaline reflux gastritis, bile reflux, chronic gastritis, duodenogastric reflux, *helicobacter pylori*, intestinal metaplasia

Introduction

Alkaline reflux gastritis (ARG) is a chronic inflammatory condition that occurs as a result of exposure of the gastric mucosa to bile and other duodenal contents.[1] This condition, which manifests with symptoms such as epigastric pain, nausea, and vomiting, is diagnosed through endoscopic and histopathological examinations.[2] However, whether the presence of bile on endoscopy alone is sufficient for the diagnosis of ARG remains a controversial topic in the literature.

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The presence of bile in the gastric lumen may not always result in mucosal damage and inflammation. In some patients, despite evident bile reflux on endoscopy, histopathological examination may reveal minimal inflammation or normal mucosal findings. This suggests that the effect of bile on the gastric mucosa may be related to individual factors, duration of exposure, and other accompanying factors.[2,3] The effect of *H. pylori* infection on ARG is not clear in the literature. Different findings have been reported regarding the effect of bile reflux on *H. pylori* infection and gastric inflammation.[4-6]

The aim of this retrospective study is to evaluate the frequency of histopathological gastritis findings and *H. pylori* prevalence in patients with endoscopically detected bile reflux. Our study investigates whether bile reflux observed on endoscopy actually leads to alkaline reflux gastritis and aims to examine the relationship between bile presence and histopathological changes.

Materials and Methods

Study Design and Patient Population

This study was designed as a retrospective observational study. Patients who underwent upper gastrointestinal endoscopy and were found to have bile reflux in the gastric lumen at the Ministry of Health Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital between January 2022 and January 2024 were included in the study.

Inclusion Criteria

- Age 18 years and above
- Presence of bile in the gastric lumen during endoscopy
- Biopsies obtained from both antrum and corpus
- Available histopathological examination results
- Complete clinical and pathological data

Exclusion Criteria

- Incomplete clinical or pathological data
- Previous gastric surgery (gastrectomy, antrectomy, gastroenterostomy, sleeve gastrectomy, etc.)
- Diagnosis of malignancy
- Patients under 18 years of age

Patient Selection and Data Collection

Patient data were collected retrospectively from the hospital information management system. A total of 311 patients with bile reflux were identified during the study period. After applying exclusion criteria, 45 patients were excluded from the study. The distribution of excluded patients was as follows:

- Patients with malignancy diagnosis (n=20): Gastric cancer (n=13), esophageal cancer (n=2), both malignancy and surgical history (n=5)
- Patients with previous gastric surgery (n=20): Total/subtotal gastrectomy (n=8), gastroenterostomy (n=8), antrectomy (n=3), sleeve gastrectomy (n=1)
- Incomplete clinical/pathological data (n=5)

After applying exclusion criteria, 266 patients with bile in the gastric lumen on endoscopy and complete histopathological evaluation results were included in the study. Of these patients, simultaneous biopsies were obtained from both corpus and antrum in 136 (51.1%).

Endoscopic Evaluation

All endoscopic procedures were performed by experienced gastroenterologists. The diagnosis of bile reflux was made based on the presence of bile in the gastric lumen during endoscopy.

Histopathological Evaluation

In 136 patients with simultaneous corpus and antrum biopsies, at least 2 biopsies were taken from each region. Biopsy specimens were fixed in 10% formalin solution and processed through routine histopathological procedures. Sections were stained with hematoxylin and eosin (H&E) and modified Giemsa stains.

Statistical Analysis

Data were analyzed using R statistical software (version 4.3.0, R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics were presented as number and percentage for categorical variables, and as mean ± standard deviation or median (interquartile range) for continuous variables. Chi-square test or Fisher's exact test was used for comparison of categorical variables, and Wilcoxon rank-sum test was used for comparison of continuous variables. P values <0.05 were considered statistically significant.

Ethical Approval

This study was approved by the Ethics Committee of Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital (Date: 12/03/2025, No: 2024/58) and conducted in accordance with the principles of the Declaration of Helsinki.

Results

Demographic and Clinical Characteristics

A total of 136 patients with bile reflux who underwent simultaneous antrum and corpus biopsies were included in the study. The median age was 49.5 (IQR: 38.0-63.0) years, with 60 (44.1%) males and 76 (55.9%) females. No significant differences were observed between *H. pylori* positive and negative groups regarding age (p=0.806) or sex distribution (p=0.120).

Smoking was present in 59 patients (43.4%), with a higher prevalence in the *H. pylori* positive group (50.0%) compared to the negative group (35.0%), though this difference was not statistically significant (p=0.080). The most common comorbidities were hypertension (42.6%), dyslipidemia (26.5%), and gastroesophageal reflux disease (22.1%). No significant differences in comorbidity distribution were found between *H. pylori* positive and negative groups (Table 1).

H. pylori Prevalence

Overall, *H. pylori* was positive in 76 patients (55.9%) and negative in 60 patients (44.1%). When analyzed by location, *H. pylori* was detected in 70 patients (51.5%) in antrum biopsies and 59 patients (43.4%) in corpus biopsies, indicating a higher colonization tendency in the antrum despite the presence of bile reflux.

Histopathological Findings in Antrum

In antrum biopsies, chronic inactive gastritis (50.0%) and chronic active gastritis (47.1%) were the most common findings. The prevalence of chronic active gastritis was significantly higher in *H. pylori* positive patients (74.3%) compared to negative patients (18.2%, p<0.001).

Inflammation was present in 94.1% of patients, with a higher rate in *H. pylori* positive (98.6%) versus negative groups (89.4%, p=0.030). According to the Sydney classification, inflammation severity showed significant differences between groups (p<0.001): Mild inflammation was more common in *H. pylori* negative patients (86.4% vs 44.9%), while moderate (39.1% vs 11.9%) and severe inflammation (15.9% vs 1.7%) were more prevalent in *H. pylori* positive patients.

Table 1. Demographic characteristics and comorbidities of patients with bile reflux

Characteristic	Overall N=136[1]	H.pylori Positive N=76[1]	H.pylori Negative N=60[1]	p[2]
Age	49.5 (38.0, 63.0)	50.0 (38.0, 63.0)	48.0 (39.0, 63.5)	0.806
Sex, n (%)				0.120
Male	60 (44.1)	38 (50.0)	22 (36.7)	
Female	76 (55.9)	38 (50.0)	38 (63.3)	
Smoking, n (%)	59 (43.4)	38 (50.0)	21 (35.0)	0.080
Diabetes mellitus, n (%)	21 (15.4)	12 (15.8)	9 (15.0)	0.899
Hypertension, n (%)	58 (42.6)	31 (40.8)	27 (45.0)	0.622
Chronic arterial disease, n (%)	9 (6.6)	5 (6.6)	4 (6.7)	>0.999
Chronic pulmonary disease, n (%)	4 (2.9)	3 (3.9)	1 (1.7)	0.630
Dyslipidemia, n (%)	36 (26.5)	17 (22.4)	19 (31.7)	0.222
GERD, n (%)	30 (22.1)	16 (21.1)	14 (23.3)	0.750
Cholelithiasis, n (%)	11 (8.1)	7 (9.2)	4 (6.7)	0.755
CKD, n (%)	14 (10.3)	7 (9.2)	7 (11.7)	0.640
Other comorbidities, n (%)	58 (42.6)	31 (40.8)	27 (45.0)	0.622

[1]Median (IQR); n (%) [2]Wilcoxon rank sum test; Pearson's chi-square test; Fisher's exact test GERD, Gastroesophageal reflux disease; CKD, Chronic kidney disease; H. pylori, Helicobacter pylori.

Activity (neutrophil infiltration) was present in 74.3% of *H. pylori* positive patients compared to 18.2% of negative patients (p<0.001). Intestinal metaplasia was observed in 14.7% of patients with no significant difference between groups (p=0.531). Atrophic changes were found in 6.6% of patients. Lymphoid aggregates/follicles were significantly more common in *H. pylori* positive patients (48.6% vs 21.2%, p<0.001) (Table 2).

Histopathological Findings in Corpus

In corpus biopsies, chronic inactive gastritis (48.5%) and chronic active gastritis (41.9%) were the predominant findings. Chronic active gastritis was significantly more frequent in *H. pylori* positive patients (78.0% vs 14.3%, p<0.001).

Inflammation was present in 90.4% of patients, with higher rates in *H. pylori* positive (98.3%) compared to negative groups (84.4%, p=0.006). Inflammation severity according to Sydney classification showed significant differences (p<0.001): Mild inflammation was predominant in *H. pylori* negative patients (87.7% vs 55.2%), while moderate (31.0% vs 9.2%) and severe inflammation (13.8% vs 3.1%) were more common in *H. pylori* positive patients.

Activity was present in 81.4% of *H. pylori* positive patients versus 13.0% of negative patients (p<0.001). Intestinal metaplasia was markedly less frequent in corpus compared to antrum (4.5% vs 14.7%) with no significant difference between *H. pylori* groups (p=0.694). Atrophic

Table 2. Histopathological findings in antrum biopsies according to h. pylori status

Characteristic	Overall N=136[1]	H.pylori Positive N=70[1]	H.pylori Negative N=66[1]	p[2]
Type of gastritis, n (%)				<0.001
Chronic active gastritis	64 (47.1)	52 (74.3)	12 (18.2)	
Chronic atrophic gastritis	0 (0.0)	0 (0.0)	0 (0.0)	
Chronic inactive gastritis	68 (50.0)	18 (25.7)	50 (75.8)	
Normal mucosa	4 (2.9)	0 (0.0)	4 (6.1)	
Activity, n (%)				<0.001
0	72 (52.9)	18 (25.7)	54 (81.8)	
1	29 (21.3)	21 (30.0)	8 (12.1)	
2	29 (21.3)	25 (35.7)	4 (6.1)	
3	6 (4.5)	6 (8.6)	0 (0.0)	
Inflammation, n (%)	128 (94.1)	69 (98.6)	59 (89.4)	0.030
Inflammation severity, n (%)				<0.001
Mild	82 (64.1)	31 (44.9)	51 (86.4)	
Moderate	34 (26.5)	27 (39.1)	7 (11.9)	
Severe	12 (9.4)	11 (15.9)	1 (1.7)	
Atrophic changes, n (%)	9 (6.6)	7 (10.0)	2 (3.0)	0.167
Atrophic changes severity, n (%)				0.250
Mild	6 (66.7)	5 (71.4)	1 (50.0)	
Moderate	2 (22.2)	2 (28.6)	0 (0.0)	
Severe	1 (11.1)	0 (0.0)	1 (50.0)	
Intestinal metaplasia, n (%)	20 (14.7)	9 (12.9)	11 (16.7)	0.531
Intestinal metaplasia severity, n (%)				0.160
Mild	12 (60.0)	4 (44.4)	8 (72.7)	
Moderate	7 (35.0)	5 (55.6)	2 (18.2)	
Severe	1 (5.0)	0 (0.0)	1 (9.1)	
Lymphoid Aggregates/Follicles, n (%)	48 (35.3)	34 (48.6)	14 (21.2)	<0.001

[1]n (%); [2]Fisher's exact test; Pearson's Chi-squared test.

changes were rare (2.9%) and found only in *H. pylori* negative patients. Lymphoid aggregates/follicles were present in 27.9% of patients with no significant difference between groups (p=0.843) (Table 3).

Comparison of Antrum and Corpus Findings

When comparing antrum and corpus findings, intestinal metaplasia was three times more frequent in antrum than corpus (14.7% vs 4.5%). Similarly, lymphoid aggregates/follicles were more common in antrum (35.3% vs 27.9%). Both locations showed strong associations between *H. pylori* positivity and chronic active gastritis, inflammation presence and severity.

Endoscopic Findings

Endoscopic findings were relatively uncommon: Antral polyps in 1.5%, corpus polyps in 1.5%, cardia/fundus polyps in 2.2%, and submucosal lesions in 2.9% of patients. No significant differences were observed between *H. pylori* positive and negative groups for any endoscopic findings (Table 4).

Discussion

Helicobacter pylori is a global health problem, affecting a significant portion of the world's population and playing a central role in the pathogenesis of gastroduodenal dis-

Table 3. Histopathological findings in corpus biopsies according to h. pylori status

Characteristic	Overall N=136[1]	H.pylori positive N=59[1]	H.pylori negative N=77[1]	p[2]
Type of gastritis, n (%)				<0.001
Chronic active gastritis	57 (41.9)	46 (78.0)	11 (14.3)	
Chronic inactive gastritis	66 (48.5)	13 (22.0)	53 (68.8)	
Normal mucosa	12 (8.8)	0 (0.0)	12 (15.6)	
Chronic atrophic gastritis	1 (0.7)	0 (0.0)	1 (1.3)	
Activity, n (%)				<0.001
0	78 (57.4)	13 (22.0)	65 (84.4)	
1	38 (27.9)	29 (49.2)	9 (11.7)	
2	15 (11.0)	13 (22.0)	2 (2.6)	
3	5 (3.7)	4 (6.8)	1 (1.3)	
Inflammation, n (%)	123 (90.4)	58 (98.3)	65 (84.4)	0.006
Inflammation severity, n (%)				<0.001
Mild	89 (72.4)	32 (55.2)	57 (87.7)	
Moderate	24 (19.5)	18 (31.0)	6 (9.2)	
Severe	10 (8.1)	8 (13.8)	2 (3.1)	
Atrophic changes, n (%)	4 (2.9)	0 (0.0)	4 (5.2)	0.133
Atrophic changes severity, n (%)				>0.999
Mild	2 (50.0)	0 (NA)	2 (50.0)	
Moderate	1 (25)	0 (NA)	1 (25.0)	
Severe	1 (25)	0 (NA)	1 (25.0)	
Intestinal metaplasia, n (%)	6 (4.5)	2 (3.4)	4 (5.3)	0.694
Intestinal metaplasia severity, n (%)				0.333
Mild	5 (83.3)	1 (50.0)	4 (100.0)	
Moderate	1 (16.7)	1 (50.0)	0 (0.0)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Lymphoid Aggregates/Follicles, n (%)	38 (27.9)	17 (28.8)	21 (27.3)	0.843

[1]n (%); [2]Fisher's exact test; Pearson's Chi-squared test.

Table 4. Endoscopic findings according to h. pylori status

Characteristic	Overall N=136[1]	H.pylori positive N=70[1]	H.pylori negative N=66[1]	p[2]
Antral polyp, n (%)	2 (1.5)	1 (1.3)	1 (1.7)	>0.999
Corpus polyp, n (%)	2 (1.5)	2 (2.6)	0 (0.0)	0.503
Cardia or Fundus polyp, n (%)	3 (2.2)	1 (1.3)	2 (3.3)	0.583
Submucosal Lesion, n (%)	4 (2.9)	4 (5.3)	0 (0.0)	0.130

eases.[7] *H. pylori* eradication is critical in the treatment of peptic ulcer disease and in reducing the risk of developing gastric cancer and MALT lymphoma.[8] Therefore, understanding the prevalence and colonization patterns of *H. pylori* in different clinical conditions is important.

In our study, *H. pylori* was positive in 76 (55.9%) of 136 patients with bile reflux. *H. pylori* positivity was detected in 70 patients (51.5%) in antrum biopsies and 59 patients (43.4%) in corpus biopsies. Notably, 6 patients were *H. pylori* positive in the corpus while negative in the antrum. This finding confirms that the combined biopsy protocol increases diagnostic success.[9]

One of the most important findings of our study is that the presence of bile in the gastric lumen on endoscopy is not equivalent to alkaline reflux gastritis. The detection of histopathologically normal mucosa in some patients with endoscopic bile reflux suggests that transient bile presence may not be sufficient to cause mucosal damage. This indicates that chronic exposure to bile, duration of direct mucosal contact, or differences in bile composition may be determinants in the development of mucosal damage.

The significantly high rate of chronic active gastritis in *H. pylori* positive patients in both the antrum (74.3%) and corpus (78.0%) demonstrates that *H. pylori* remains the main determinant of gastric inflammation even in the presence of bile. *H. pylori* and duodenogastric reflux cause synergistic damage to the gastric mucosa.[10] The significantly higher severe inflammation in both the antrum and corpus in *H. pylori* positive patients confirms this synergistic effect. These findings suggest that inflammatory processes in gastrointestinal diseases require more detailed investigation.[11]

Conflicting results have been reported in the literature regarding bile reflux inhibiting *H. pylori* colonization.[12,13] Our prevalence of 55.9% suggests that endoscopically observed bile may reflect transient reflux, which may be different from chronic alkaline reflux gastritis. Gastric pH changes may also affect *H. pylori* colonization.[14]

The three-fold higher prevalence of intestinal metaplasia in the antrum (14.7%) compared to the corpus (4.5%) is an important finding. Literature reports that primary bile reflux gastritis has different characteristics from *H. pylori* gastritis, but their coexistence leads to more severe premalignant changes.[15] The association of bile reflux with gastric cancer and precancerous lesions has been demonstrated, and the risk of intestinal metaplasia has been found to increase in alkaline reflux developing after cholecystectomy.[16,17] In light of these findings, our higher intestinal metaplasia rate in the antrum suggests that this region is more sensitive to both *H. pylori* and bile exposure. Risk factors and prediction models developed for endoscopic bile reflux may help determine which patients require closer follow-up.[18]

The complex effects of bile reflux on gastric microbiota are important. Hua et al.[19] showed that *H. pylori* infection reduces gastric microbial diversity and that close relationships develop between Helicobacter and non-Helicobacter bacteria, especially in patients with chronic atrophic gastritis. Additionally, bile reflux has been reported to increase gastric colonization of oral bacteria (Neisseria, Staphylococcus). These complex microbiological interactions may explain the high *H. pylori* prevalence in our study.

This study has several limitations. Due to its retrospective design, the degree, duration, and symptom severity of bile reflux could not be evaluated. The inability to perform quantitative measurement of bile reflux and the subjective nature of endoscopic evaluation are other limitations. Including only patients with simultaneous antrum and corpus biopsies limited our sample size but ensured reliable comparison. Only histopathological methods were used for *H. pylori* diagnosis; additional methods such as PCR or urea breath test were not applied.

Conclusion

In conclusion, *H. pylori* prevalence is high in patients with bile reflux. The presence of bile in the gastric lumen on endoscopy alone is not sufficient for the diagnosis of alkaline reflux gastritis; histopathological confirmation is required. Considering the synergistic effect of *H. pylori* and bile reflux, biopsies should be obtained from both antrum and corpus, *H. pylori* eradication should be performed when positive, and close surveillance should be implemented in patients with intestinal metaplasia.

Disclosures

Ethics Committee Approval: This study was approved by the Ethics Committee of Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital (Date: 12/03/2025, No: 2024/58).

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