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Duodenogastric reflux does not influence *Helicobacter pylori* colonization in children

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ABSTRACT

Aims: Duodenogastric reflux (DGR) and *Helicobacter pylori (H. pylori)* infection are two common etiologies implicated in gastric mucosal injury during childhood. However, their interplay remains poorly understood. This study aimed to investigate the relationship between DGR and *H.pylori* colonization through comprehensive endoscopic and histopathological evaluation in a pediatric cohort.

Methods: In this retrospective study, the medical records of 698 children who underwent esophagogastroduodenoscopy between January 2024 and March 2025 were reviewed. Patients were classified into DGR and non-DGR groups, based on the presence or absence of bile residues in the stomach during endoscopy. Histopathological assessments were conducted using the modified Sydney classification to evaluate *H.pylori* presence and density. Demographic, endoscopic, and histological parameters were compared across groups.

Results: DGR was identified in 21.6% of patients and was significantly associated with older age and female sex ($p<0.001$ and $p=0.017$, respectively). However, no significant differences were observed between the DGR and non-DGR groups regarding the frequency or density of *H.pylori* colonization ($p=0.647$ and $p=0.731$). Both DGR and *H.pylori* positivity were independently associated with increased endoscopic abnormalities ($p<0.001$ for both) and gastric mucosal inflammation ($p=0.047$, and $p<0.001$ respectively).

Conclusions: DGR and *H.pylori* infection independently contribute to gastric mucosal pathology in children. However, DGR does not appear to influence *H.pylori* colonization significantly. These findings underscore the need for further prospective and molecular studies to elucidate the mechanistic interactions between bile reflux and *H.pylori* in pediatric populations.

Introduction

Duodenogastric reflux (DGR) is characterized by the backward flow of duodenal contents into the gastric lumen, typically resulting from pyloric sphincter dysfunction. Although the exact cause of DGR remains unclear, factors such as gastroduodenal dysmotility, hormonal disturbances, dietary habits, and *Helicobacter pylori (H. pylori)* infection have been implicated. Gastrointestinal hormones, including gastrin, cholecystokinin, and secretin, may increase the risk of DGR by influencing gastric acid secretion and regulating gastric motility. In addition, *H. pylori* infection may further impair motility and contribute to the development of DGR. Pyloric dysfunction, however, is considered the primary underlying cause. It can occur primarily or develop secondarily to upper gastrointestinal surgeries such as cholecystectomy or gastrectomy.

DGR can cause irritation, inflammation, and various histopathological changes in the gastric mucosa in both adults and children. Recent pediatric esophagogastroduodenoscopy (EGD) studies have reported an increasing frequency of DGR, likely due to changes in dietary patterns, the more frequent use of EGD in children, and a higher prevalence of gastric surgery (1-5).

H. pylori is capable of establishing long-term colonization within the gastric mucosa, which may result in chronic gastritis, peptic ulceration, and, in progressive cases, the development of gastric adenocarcinoma. The development of *H. pylori* infection is influenced by host-related factors, environmental conditions, and bacterial virulence mechanisms (1,6-8).

The relationship between DGR and *H. pylori* remains uncertain. Only a limited number of studies have investigated this association in the pediatric population, and it is still debated whether the chemical gastritis environment caused by bile acids in DGR affects *H. pylori* colonization. Some studies suggest that DGR may reduce *H. pylori* colonization, while others report no significant effect (1,4,9-11).

This study investigates the association between DGR and *H. pylori* in children by combining endoscopic findings with histopathological analyses. The results are expected to clarify whether DGR has a reducing effect or no effect on *H. pylori*, thereby informing diagnostic and therapeutic strategies in pediatric patients.

Methods

Research design and setting

This retrospective analysis was conducted in the Pediatric Gastroenterology Department of a university-based tertiary care hospital and included the clinical records of 1,168 pediatric patients who underwent EGD between January 1, 2024, and March 31, 2025.

Children aged 1-18 years who underwent EGD during the study period for symptoms such as nausea, weight loss, dyspepsia, vomiting, chronic diarrhea, abdominal pain, malnutrition, or gastroesophageal reflux, and who had gastric biopsies obtained, were included. Written informed consent was obtained from their legal guardians. Patients were excluded if they had Crohn's disease, celiac disease, ulcerative colitis, eosinophilic esophagitis, immunodeficiency, a history of gastrointestinal surgery, chronic liver disease, a gastrostomy or nasogastric tube, or required EGD for foreign body retrieval. Patients with a history of antibiotic use targeting *H. pylori* before EGD were also excluded. After applying these criteria, a total of 698 pediatric cases were included in the final analysis.

Patients in whom bile residues were observed upon initial entry into the stomach during EGD and who also had evidence of gastritis, either endoscopically or histopathologically, were classified into the DGR group. Patients without bile residue at initial entry, as well as those in whom bile was observed later during the procedure, were assigned to the non-DGR group. (1,4,10,11). In addition, the characteristics of patients with confirmed *H. pylori* colonization (*H. pylori*-positive) and those without (*H. pylori*-negative) were analyzed. The same inclusion and exclusion criteria were applied across all groups. A power calculation was performed to determine the minimum number of participants required to achieve sufficient statistical power for the study's main objective.

Data collection and EGD

Patient data, including age, sex, presenting symptoms, EGD indications, endoscopic findings, and histopathological results, were retrospectively collected from the hospital information management system. EGD procedures were performed by an experienced pediatric gastroenterologist using the FUJINON ELUXEO VP-7000 endoscopy system, under deep sedation administered by an anesthesiologist. During the procedure, findings such as hyperemia, edema, nodularity, friability, bleeding, erosion, ulceration, and the presence of bile in the stomach were recorded for the esophageal, gastric, and duodenal mucosa. In accordance with European Society for Paediatric Gastroenterology, Hepatology and Nutrition guidelines, at least two biopsy samples were obtained from the corpus, antrum, and duodenum of each patient during EGD, regardless of whether any visible lesions were present (12).

Histopathological evaluation

The detection and colonization density of *H. pylori* were assessed using the criteria outlined in the modified Sydney system. In this context, the degree of inflammation was determined based on the infiltration of mononuclear cells in the lamina propria, while the level of activity was assessed by the extent of neutrophil infiltration. The severity of inflammation and *H. pylori* colonization density were graded as "none", "mild", "moderate", and "severe" (13).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was assessed with histograms, quantile-quantile plots, and the Kolmogorov-Smirnov test. Group comparisons were conducted using the independent samples t-test for normally distributed variables and the Pearson chi-square test for categorical data. All tests were two-sided, with $p<0.05$ considered significant. Sample size, determined by prior power analysis, was sufficient for statistical validity.

A binary logistic regression was performed to evaluate the effects of DGR, age, and gender on *H. pylori* infection, with *H. pylori* positivity as the dependent variable. Model fit was assessed using Nagelkerke R^2 and the Hosmer-Lemeshow test, and results included β coefficients, odds ratios [Exp(β)], Wald test, and p-values, with significance set at $p<0.05$.

An ordinal logistic regression was conducted to identify factors associated with gastric inflammation severity, using *H. pylori* positivity, age, DGR, and gender as independent variables. Results included β coefficients and odds ratios Exp(β) for interpreting the effects of each variable.

Ethical approval

The study received ethical approval from the Scientific Research Ethics Committee of the University of Health Sciences Türkiye, Gülhane (decision no: 2025-320, date: 03.06.2025). All procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Results

This study included 698 pediatric patients, and DGR was identified in 151 of them (21.6%). The mean age of patients in the DGR group (13.88±2.81 years) was significantly higher than that of the non-DGR group (11.79±4.27 years) (p<0.001). Female patients were more frequently represented in the DGR group (p=0.017). There was no statistically significant variation in *H. pylori* detection or colonization intensity between the DGR and non-DGR groups (p>0.05) (Table 1).

The frequency of pathological findings on gastric endoscopy was 97.4% in the DGR group and 84.3% in the non-DGR group (p<0.001). In histopathological examination of the stomach, the severity of inflammation was significantly greater in the DGR group (p=0.047). This difference corresponded to a Cramér's V of 0.106, indicating a weak effect size. Neutrophil activity was similar between the two groups (p>0.05) (Table 2).

When evaluated in terms of *H. pylori* colonization, 184 patients (26.4%) constituted the *H. pylori*-positive group. This group had a higher mean age, while the gender distribution was similar to that of the *H. pylori*-negative group (p=0.03 and p>0.05, respectively). Abnormal findings on gastric endoscopy were more frequent in the *H. pylori*-positive group (95.1%) compared to the *H. pylori*-negative group (84.4%) (p<0.001). Histopathological examination of the stomach revealed significantly higher degrees of inflammation and neutrophil activity in the *H. pylori*-positive group (p<0.001) (Table 3).

According to the results of the binary logistic regression analysis, the β value for age was calculated as 0.069, and Exp(β)=1.072. This means that each increase in age is associated with a 7.2% increase in the likelihood of *H. pylori* positivity. The p-value for the significance test was found to be 0.03.

The gender variable was found to have no significant effect on *H. pylori* positivity. The β value for gender was 0.068, Exp(β)=1.071, and the p-value was 0.705. This indicates that the effect of gender on *H. pylori* positivity is minimal and not statistically significant. This result suggests that gender is not a determining factor in the development of *H. pylori* infection. Similarly, the relationship between DGR and *H. pylori* positivity was not statistically significant. The β value for DGR was 0.029, Exp(β)=1.029, and the p-value was 0.892. This result indicates that DGR does not have a significant effect on *H. pylori* positivity.

The overall model fit was calculated as Nagelkerke R^2=0.20. Additionally, the Hosmer-Lemeshow test used to assess the model's fit yielded a p-value of 0.076, indicating that the model fits the data well but does not provide statistically significant alignment.

The ordinal logistic regression analysis demonstrated that *H. pylori* positivity had the strongest impact on gastric inflammation (β=1.64). Additionally, age was identified as an important factor in increasing inflammation (β=0.35). DGR contributed very little to gastric inflammation (β=0.09). Gender did not have a significant effect on inflammation.

When patients with both DGR and *H. pylori* were compared to those with either DGR or *H. pylori* alone, a significant difference was observed between the groups in terms of histopathological inflammation (p=0.023) and activity (p=0.001). The DGR and *H. pylori* group had markedly higher rates of moderate-to-severe inflammation (73.8%) and moderate-to-severe activity (61.9%). No statistically significant difference was found in endoscopic gastric findings (p=0.062); however, the DGR and *H. pylori* group showed a higher tendency for erosion (14.3%) and ulcer (11.9%) (Table 4).

Table 1. Distribution of age, gender, and *H. pylori* colonization in the DGR and non-DGR groups

Variable	Category	Non-DGR group (n=547)	DGR group (n=151)	p-value
Age (mean ± SD)	Years	11.79±4.27	13.88±2.81	**<0.001***
		n (%)	**n (%)**	
Gender	Male	218 (39.8)	44 (29.1)	**0.017****
	Female	329 (60.2)	107 (70.9)	
***H. pylori* colonization**	Absent	405 (74.0)	109 (72.2)	0.647**
	Present	142 (25.9)	42 (27.8)	
***H. pylori* colonization density**	Mild	41 (7.5)	15 (9.9)	0.731**
	Moderate	66 (12.1)	16 (10.6)	
	Severe	35 (6.4)	11 (7.3)	

*Independent samples t-test, **Pearson chi-square test
H. pylori: Helicobacter pylori, DGR: Duodenogastric reflux, SD: Standard deviation

Table 2. **Endoscopic and histopathological gastric evaluation in non-DGR and DGR groups**

Variable	Category	Non-DGR group (n=547) n (%)	DGR group (n=151) n (%)	p-value*
Gastric endoscopic evaluation	Normal	86 (15.7)	4 (2.6)	**0.001**
	Hyperemia/edema	315 (57.5)	109 (72.2)	
	Nodularity	74 (13.5)	18 (11.9)	
	Erosion	44 (8.0)	9 (6.0)	
	Ulcer	28 (5.1)	11 (7.3)	
Gastric inflammation severity	Normal	59 (10.7)	6 (4.0)	**0.047**
	Mild	362 (66.1)	105 (69.5)	
	Moderate	83 (15.1)	30 (19.9)	
	Severe	43 (7.8)	10 (6.6)	
Gastric neutrophil activity	Normal	396 (72.3)	108 (71.5)	0.971
	Mild	51 (9.3)	15 (9.9)	
	Moderate	71 (12.9)	21 (13.9)	
	Severe	29 (5.3)	7 (4.6)	

*Pearson chi-square test
DGR: Duodenogastric reflux

Table 3. **Age, gender, gastric endoscopic and histopathological evaluation of *H. pylori*-positive and *H. pylori*-negative groups**

Variable	Category	*H. pylori*-negative (n=514)	*H. pylori*-positive (n=184)	p-value
Age (mean ± SD)	Years	11.98±4.29	13.02±3.35	**0.030***
		n (%)	**n (%)**	
Gender	Male	193 (37.5)	69 (37.5)	0.091**
	Female	321 (62.5)	115 (62.5)	
Gastric endoscopic evaluation	Normal	80 (15.6)	9 (4.9)	**<0.001****
	Hyperemia/edema	352 (68.5)	73 (39.7)	
	Nodularity	21 (4.1)	71 (38.6)	
	Erosion	33 (6.4)	20 (10.9)	
	Ulcer	28 (5.4)	11 (6.0)	
Gastric inflammation severity	Normal	65 (12.6)	0 (0.0)	**<0.001****
	Mild	427 (83.1)	40 (21.7)	
	Moderate	18 (3.5)	95 (51.6)	
	Severe	4 (0.8)	49 (26.6)	
Gastric neutrophil activity	Normal	491 (95.5)	13 (7.1)	**<0.001****
	Mild	15 (2.9)	51 (27.7)	
	Moderate	6 (1.2)	86 (46.7)	
	Severe	2 (0.4)	34 (18.5)	

*Independent samples t-test, **Pearson chi-square test
H. pylori: Helicobacter pylori, SD: Standard deviation

Table 4. Endoscopic and histopathological gastric evaluation in patients with both DGR and *H. pylori* vs. those with either DGR or *H. pylori* alone

Variable	Category	DGR and *H. pylori* group (n=42) n (%)	DGR or *H. pylori* alone group (n=251) n (%)	p-value*
Gastric endoscopic evaluation	Normal	0 (0.0)	13 (5.2)	0.062
	Hyperemia/edema	19 (45.2)	144 (57.4)	
	Nodularity	12 (28.6)	65 (25.9)	
	Erosion	6 (14.3)	17 (6.8)	
	Ulcer	5 (11.9)	12 (4.8)	
Gastric inflammation severity	Normal	0 (0.0)	6 (2.4)	**0.023**
	Mild	11 (26.2)	123 (49.0)	
	Moderate	21 (50.0)	83 (33.1)	
	Severe	10 (23.8)	39 (15.5)	
Gastric neutrophil activity	Normal	5 (11.9)	111 (44.2)	**0.001**
	Mild	11 (26.2)	44 (17.5)	
	Moderate	19 (45.2)	69 (27.5)	
	Severe	7 (16.7)	27 (10.8)	

*Pearson chi-square test
H. pylori: Helicobacter pylori, DGR: Duodenogastric reflux

Discussion

This study investigated the association between DGR and *H. pylori* in children by integrating both endoscopic and histopathological assessments. Our findings indicate that the presence of DGR was associated with older age and female sex, yet it had no significant impact on the frequency or density of *H. pylori* colonization. Importantly, both conditions were independently linked to abnormal endoscopic findings and increased gastric mucosal inflammation. These results suggest that each contributes to the pathogenesis of gastric disorders in the pediatric population.

In our study, no significant difference was detected in *H. pylori* positivity between children with and without DGR (27.8% vs. 25.9%). Colonization density was also comparable across groups. These findings suggest that DGR does not exert a marked suppressive effect on *H. pylori* in children. However, any potential influence might be offset by the bacterium's adaptive mechanisms. The high environmental adaptability of *H. pylori* and its ecological advantage within the gastric microbiota may allow it to maintain colonization despite bile exposure.

Previous studies have examined the relationship between these two conditions, but results remain inconsistent. Some pediatric studies have reported lower rates of *H. pylori* gastritis in children with DGR, attributing this to the possible bactericidal effects of bile acids (10,11). In contrast, some studies suggested that DGR did not influence *H. pylori* colonization rates in children (1,4).

A recent study demonstrated that the presence of DGR is associated with distinct alterations in the gastric mucosal microbiota, marked by an increase in non-*H. pylori* species (14). Supporting this, two prospective investigations showed a substantial reduction in the relative abundance of commensal taxa in both the duodenal bulb and gastric mucosa of *H. pylori*-infected children, alongside a predominance of the *Helicobacter* genus (15,16). These observations suggest that *H. pylori* may activate adaptive responses to maintain a competitive edge within a dynamically shifting microbial environment. Our findings appear to support this hypothesis, suggesting that despite exposure to hostile conditions such as bile acids, *H. pylori* might influence competing microbiota and contribute to the establishment of a more favorable ecological niche for its persistence.

The interaction between bile acids and *H. pylori* is influenced by factors such as the type, concentration, and duration of exposure. Conjugated bile acids may damage bacterial membranes and inhibit adaptive mechanisms, while bile-induced epithelial injury may reduce bacterial adherence. Certain bile acids have been reported to suppress metabolism and virulence gene expression, potentially limiting colonization. Despite these effects, *H. pylori* has genetic adaptations that may provide resistance to prolonged bile exposure (14,17). This multifaceted interplay may help explain the inconsistencies observed across studies examining the bile and *H. pylori* interaction, including our study.

In our cohort, gastric mucosal lesions such as hyperemia, edema, and ulceration were significantly more common among children with DGR compared to the non-DGR group, supporting the notion of direct mucosal injury by bile acids, as previously

reported in the literature (2,18,19). Histopathologically, inflammation severity was also higher in the DGR group, suggesting an elevated risk of chemically induced gastritis. Notably, no significant difference was observed in neutrophil activity, potentially indicating a stronger association of DGR with chronic rather than acute inflammation.

Meanwhile, in children with *H. pylori* infection, gastric endoscopy revealed more extensive pathology, and histological analysis showed significantly elevated inflammation and neutrophil activity scores. These findings reaffirm *H. pylori* as a principal driver of gastric inflammation and highlight its critical role in the extent of mucosal damage.

Regression analyses indicated that both DGR and *H. pylori* independently contribute to gastric mucosal inflammation (β=1.64 and β=0.35, respectively). Each condition was associated with higher rates of hyperemia, edema, and ulceration. The coexistence of conditions may further increase the risk of gastritis. Clinically, this highlights the importance of considering both entities in therapeutic strategies. However, the combination of DGR and *H. pylori* may negatively impact treatment outcomes, potentially necessitating longer durations of acid-suppressive therapy in these patients. In addition, dietary modifications to minimize mucosal irritation remain important in affected children. Comprehensive education and structured follow-up plans should also be provided to families to support treatment adherence.

One of the notable strengths of our study lies in the inclusion of a large, systematically evaluated pediatric cohort, in which the relationship between DGR and *H. pylori* infection was assessed using both endoscopic and histopathological criteria. While few pediatric studies have addressed this interaction, our findings provide valuable insights into this underexplored area. The detailed evaluation of multi-site biopsy specimens based on the modified Sydney classification further enhances the reliability of our results.

Nonetheless, the retrospective nature of our study posed certain limitations in accessing complete clinical data. As in previous studies, the diagnosis of DGR was based on the visual detection of bile residues in the stomach and was supported by endoscopic or histopathological evidence of gastritis (1,4,10,11). Although DGR was assessed upon initial entry into the stomach to minimize the effects of sedation and the endoscopic procedure, only patients with bile residues were classified as having chronic DGR, and this approach may still have influenced the accuracy of diagnosis. In addition, as with any visually based assessment, the diagnosis of DGR is subject to observer variability, which represents another limitation of our study.

In future research, prospective studies using more objective methods such as intragastric bile acid quantification or pH monitoring could help improve diagnostic precision. Likewise, directly measuring bile acid concentrations could provide a more detailed understanding of the effect of DGR on *H. pylori* colonization.

Conclusion

In conclusion, our findings suggest that, contrary to common assumptions, DGR may not significantly influence the frequency or intensity of *H. pylori* colonization in children, indicating that the presumed antibacterial effects of bile may be counteracted by the bacterium's strong adaptive mechanisms. Importantly, we demonstrated that DGR and *H. pylori* each independently contribute to mucosal inflammation, and that their coexistence may amplify bile-induced chemical gastritis. This study is among the first to systematically evaluate the independent and combined effects of DGR and *H. pylori* in a large pediatric cohort. These results advance current understanding in pediatric gastroenterology by emphasizing the need for an integrated diagnostic and therapeutic approach in children presenting with gastrointestinal symptoms, and may guide future strategies aimed at improving management and outcomes.

Ethics

Ethics Committee Approval: The study received ethical approval from the Scientific Research Ethics Committee of the University of Health Sciences Türkiye, Gülhane (decision no: 2025-320, date: 03.06.2025).

Informed Consent: Written informed consent was obtained from their legal guardians.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.T., Y.M.E., Concept: S.T., Design: S.T., Data Collection or Processing: S.T., Y.M.E., Analysis or Interpretation: S.T., Y.M.E., Literature Search: S.T., Y.M.E., Writing: S.T.

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

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