Turkish Journal of Pediatric Disease

Original Article

From stomach to liver: The impact of *Helicobacter pylori* gastritis on pediatric hepatic steatosis

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Received : 31.10.2025, Accepted : 13.04.2026 DOI: 10.12956/TJPD.2025.1263

ABSTRACT

Objective: The purpose of our study was to examine the association between endoscopically diagnosed *Helicobacter pylori* (HP) gastritis and hepatosteatosis, and to investigate the correlation between the presence of HP and biochemical and anthropometric measurements in children.

Materials and Methods: Patients who were followed up in the Pediatric Gastroenterology outpatient clinic of Kayseri City Training and Research Hospital and underwent esophagogastroduodenoscopy performed by the attending gastroenterologist were evaluated. Patients aged between 2 and 18 years, with a histopathological diagnosis of gastritis, both HP positive and negative, were enrolled in the study.

Results: In patients with HP-positive gastritis, the incidence of hepatosteatosis was found to be statistically significantly higher compared to those with HP-negative gastritis ($\chi^2 = 22.704$; $p < 0.001$). A statistically significant weak positive correlation was found between the density of HP and the grade of hepatosteatosis (rho = 0.344; $p < 0.001$).

Conclusion: The HP-positive gastritis in children is associated with the development of hepatosteatosis, and the higher grade of hepatosteatosis in patients with HP-positive gastritis suggests that HP gastritis may impact the development of fatty liver.

Keywords: Children, gastritis, *Helicobacter pylori*, hepatosteatosis

Introduction

Helicobacter pylori (HP) is a gram-negative bacillus that infects more than half of the world's population. It plays a role in the development of important gastroduodenal diseases such as peptic ulcer, gastric mucosa-associated lymphoid tissue lymphoma, and gastric adenocarcinoma (1). Children are most commonly infected with HP during preschool (2). A systematic meta-analysis examining the global prevalence of HP infection in children and associated risk factors reported that HP infection is present in 32.3% of children worldwide, reflecting differences according to the diagnostic methodologies used (3). Moreover, recent evidence suggests that the effects of HP are not limited to the gastrointestinal tract but may also modulate both the development and clinical course of several extragastric diseases (4). Particularly in gastrointestinal diseases, significant associations have been reported between HP positivity and conditions such as gastroesophageal reflux disease, inflammatory bowel disease, non-alcoholic fatty liver disease, and cholelithiasis in adulthood (5). On the contrary, very few studies have examined this relationship in the childhood age group. It has been reported that HP infection should be considered in the investigation of the causes of chronic immune thrombocytopenic purpura in childhood (6). A recent meta-analysis reported that HP infection in children is associated with growth retardation and may be specifically linked to height-for-age scores (7).

Childhood fatty liver disease is increasingly prevalent worldwide. Fatty liver disease that begins at an early age can progress to steatohepatitis, fibrosis, cirrhosis, and even hepatocellular carcinoma in later years. Therefore, early detection of hepatosteatosis and identification of associated risk factors are crucial for preventing long-term metabolic complications and progressive liver damage (8).

Various mechanisms have been proposed in the literature to suggest that HP may be implicated in the development of hepatosteatosis. These mechanisms suggest that HP may reach the liver via the bile and trigger a local inflammatory response (9). Infection-induced proinflammatory cytokines such as C-reactive protein (CRP) and tumor necrosis factor-alpha (TNF-α) may contribute to the development of insulin resistance (10). Furthermore, HP infection may facilitate hepatic steatosis by decreasing adiponectin levels, which is known to inhibit fatty acid accumulation in the liver (11). Indeed, a meta-analysis of 21 studies found a significant and positive association between HP infection and hepatosteatosis. However, none of the studies included in this meta-analysis evaluated the pediatric age group (12).

The limited data available to evaluate the relationship between HP infection and hepatosteatosis in children necessitated the conduct of this study. The primary aim was to compare the presence and degree of hepatosteatosis in children with HP-positive and HP-negative gastritis and to evaluate a possible relationship. Secondly, this research was conducted to examine the relationships between HP colonization density and the degree of hepatosteatosis and selected clinical, laboratory, and anthropometric variables. This approach aimed to contribute to a better understanding of the relationship between HP infection and hepatosteatosis in childhood.

Materials and Methods

This retrospective observational study included 272 patients who underwent esophagogastroduodenoscopy between April 2024 and May 2025 and were diagnosed with gastritis based on histopathological findings. Endoscopic procedures were performed by the responsible investigator, and ultrasonographic examinations were performed by radiologists; this information was obtained from medical records.

The presence of HP gastritis was determined by reviewing the patients' endoscopy and histopathology reports. The presence of HP in the antral biopsy samples was evaluated based on Giemsa staining and scoring. In determining the severity of HP-associated gastric inflammation, bacterial density was assessed semi-quantitatively according to the amount of microorganisms observed on the mucosal surface and was graded from mild to severe as +1, +2, and +3, which were expressed as a score reflecting HP colonization density. Patients with gastritis without HP constituted the HP-negative gastritis group.

In patients with hepatosteatosis, other secondary causes of hepatic steatosis, including endocrine disorders, metabolic abnormalities, viral hepatitis, drug-induced liver damage, and genetic/metabolic liver diseases were evaluated and excluded. The grade of hepatosteatosis was assessed using liver ultrasonography findings from the same patients. Patients were evaluated as having Grade 1 (mild), Grade 2 (moderate) and Grade 3 (severe) hepatosteatosis based on the increase of liver parenchymal echogenicity (13). Biochemical parameters and anthropometric measurements were also obtained from patient records.

Statistical analysis

Data were evaluated using the IBM Statistical Package for the Social Sciences, version 22.0 (SPSS Inc., Armonk, NY, IBM Corp., USA). Descriptive statistics are presented as number (n) and percentage (%) for categorical variables, and as mean and standard deviation and median and minimum–maximum values for continuous variables. The normality of continuous variables was evaluated by examining the skewness and kurtosis coefficients; values within the range of ±1.5 were considered as an indicator of approximately normal distribution. The chi-square test was used to compare categorical variables including the presence of HP-positive gastritis according to the presence of hepatosteatosis. Associations between the grade of hepatosteatosis and continuous variables were evaluated using Spearman correlation coefficient. Binary logistic regression analysis was applied to examine the variables associated with the presence of hepatosteatosis; the results were reported with odds ratio (ORs) and 95% confidence interval. Variables included in the logistic regression models were selected based on clinical relevance and prior evidence from the literature. In addition, variables showing an association with hepatosteatosis in univariate analyses were considered for inclusion in the multivariate models. Model fit was evaluated using Cox and Snell and Nagelkerke R^2 values. The statistical significance level was accepted as $p < 0.050$.

Results

The study included 272 patients. Of the patients, 173 were female (63.6%). The mean age of study group was 13±3.9 years. HP-positive gastritis were statistically higher in females than in males. Histopathological examinations showed the HP-positive gastritis in 47.5% of all patients (n= 29). Of these, 57 showed mild, 31 had moderate, and 41 had severe HP colonization density. Hepatosteatosis was detected in 13.6% of all patients (n=37); of these cases, hepatic steatosis was graded as mild in 19 patients, moderate in 14 patients, and severe in 4 patients. Among the patients with HP-positive gastritis, 31 patients had hepatic steatosis, corresponding to 24% of this group. Descriptive characteristics of the cohort are displayed in Table I.

When the relationships between the degree ofx hepatosteatosis and independent variables were examined, a moderately positive and statistically significant correlation was found between the degree of HP colonization density and the grade of hepatosteatosis (rho =0.344; p<0.001). A weak but statistically significant positive correlation was found between AST and hepatosteatosis grade (rho =0.205; p=0.001), whereas no significant correlation was observed between ALT levels and hepatosteatosis grade (rho= 0.119; p=0.077). A weak but statistically significant positive correlation was also found between the ESR and the grade of hepatosteatosis (rho=0.205; p<0.001). The relationship between BMI-SD and the grade of hepatosteatosis was found to be moderately positive (r =0.334; p<0.001). These findings suggest that the grade of hepatosteatosis varies with BMI-SD and HP colonization density (Table II). There was a weakly negative correlation between the HP-positive gastritis and CRP

Table I: Demographic and clinical characteristics of the study population

Variables	Descriptive Statistics
Gender*	
Female	173 (63.6)
Male	99 (36.4)
Age (years)†	13.0±3.9
Hemoglobin (g/dl)†	13.61±1.71
Albumin (g/dl)†	4.71±0.27
Platelet (×10³/μL)‡	315 (158–646)
White blood cell (×10³/μL)‡	7.18 (4.14–22.31)
AST (U/L)‡	18 (9–61)
ALT (U/L)‡	13(5–108)
CRP (mg/dl)‡	4.7 (3.9–5.6)
ESR (mm/h)‡	4 (1–42)
BMI (SD)‡	–0.31 (–3.54–3.23)
Endoscopic Findings of Esophagus*	
Normal	201 (73.9)
Hyperemia	71 (26.1)
Endoscopic Findings of Antrum*	
Normal	115 (42.3)
Hyperemia	135 (49.6)
Ulcer	13 (4.8)
Nodularity	9 (3.3)
Endoscopic Findings of Duodenum*	
Normal	220 (80.9)
Hyperemia	46 (16.9)
Ulcer	5 (1.8)
Cobblestone appearance	1 (0.4)
Histopathologic Findings of Esophagus*	
Normal	150 (55.1)
Esophagitis	118 (43.4)
Eosinophilic esophagitis	4 (1.5)
Histopathologic Findings of Antrum*	
Lymphoplasmacytic infiltration	119 (43.8)
Chronic inflammation	58 (21.3)
Active inflammation and epithelial erosion	50 (18.4)
Active inflammation	45 (16.5)
Histopathologic Findings of Duodenum*	
Normal	247 (90.8)
Duodenitis	23 (8.5)
Villous atrophy	2 (0.7)

: n(%), †: mean±SD, ‡: median (min–max), **ALT: Alanine aminotransferase, **AST:** Aspartate aminotransferase, **BMI:** Body mass index, **CRP:** C-reactive protein, **ESR:** Erythrocyte Sedimentation Rate*

(rho=–0.180 p=0.006), and ESR (rho= –0.138 p=0.045). There was no correlation between the presence of HP and BMI-SD.

On the other hand, the hepatosteatosis was statistically higher in patients with HP-positive gastritis compared to patients with HP-negative gastritis (Table III).

In the logistic regression analysis conducted to evaluate factors associated with the presence of hepatosteatosis, metabolic and laboratory variables were included in the first model. In this model, only BMI SDS was found to be statistically significantly associated with the presence of hepatosteatosis, with a one-unit increase in BMI-SD approximately doubling the probability of hepatosteatosis (OR=2.13; 95% CI: 1.48–3.07; p<0.001). No statistically significant relationship was found between AST, ALT, and

Table II: Correlations between hepatosteatosis grade and independent variables

Variables	rho	p*
Hp colonization density (score)	0.344	0.001
Age	0.000	0.994
Hemoglobin	0.060	0.323
Platelet	–0.005	0.940
White blood cell	0.087	0.154
AST	0.205	0.001
ALT	0.119	0.077
Albumin	–0.019	0.756
CRP	0.074	0.266
ESR	0.205	0.001
BMI-SD	0.334	0.001

: Spearman rank correlation analysis. **ALT: Alanine aminotransferase, **AST:** Aspartate aminotransferase, **BMI:** Body mass index, **CRP:** C-reactive protein, **ESR:** Erythrocyte Sedimentation Rate*

Table III: Comparison of groups based on the presence of hepatosteatosis

	Hepatosteatosis*		χ^2	p‡
	Absent	Present		
Gastritis				
HP-Negative	137 (58.3)	6 (16.2)	22.704	<0.001
HP-Positive	98 (41.7)	31 (83.8)		

**: n(%), ‡: Chi-square test*

ESR levels and the presence of hepatosteatosis. In the second model, when HP-positive gastritis was added, the association between BMI-SD and hepatosteatosis was maintained, while HP-positive gastritis was also found to be significantly associated with the presence of hepatosteatosis (OR = 9.81; 95% CI: 3.32–28.99; p < 0.001). It was observed that adding the HP variable to the model improved model fit and increased the Nagelkerke R^2 value (Table IV).

Discussion

This study indicates that hepatosteatosis may accompany HP-positive gastritis in childhood and emphasizes that HP may contribute to the pathogenesis of fatty liver. The objective of this research was to contribute to the literature by demonstrating the relationship between HP and HS in children.

HP is generally asymptomatic in childhood. Current pediatric data and meta-analysis results report that HP gastritis occurs at similar rates in girls and boys (3). In contrast, the rate of HP gastritis was higher in girls at 63.6%. The risk of developing gastric or duodenal ulcers due to HP reported in the pediatric data ranges from 0.4% to 12% (14). In our study, ulcers were observed in less than 5% of patients. The ESPGHAN guidelines for HP screening recommend testing only in children with suspected gastric or duodenal ulcers presenting with complaints such as epigastric pain, weight loss, and loss of appetite (15).

While the long-term development of ulcers or gastric cancer depends on the extent of the persistence of the infection, the risk of malignancy associated HP in childhood is considered to be quite low (16,17). HP initiates primary gastric adenocarcinoma by direct contact with the gastric epithelium

Table IV: Factors influencing the presence of hepatosteatosis

	B	S.E.	Sig.	Exp(B)	95% C.I.for EXP(B)	
					Lower	Upper
Model 1						
AST	0.030	0.032	0.350	1.031	0.967	1.098
ALT	–0.009	0.027	0.742	0.991	0.939	1.046
ESR	–0.012	0.031	0.710	0.988	0.930	1.051
BMI-SD	0.757	0.186	0.001	2.132	1.479	3.073
Constant	–2.490	0.606	0.001	0.083	-	-
Model 2						
AST	0.046	0.035	0.193	1.047	0.977	1.122
ALT	–0.019	0.029	0.496	0.981	0.927	1.037
ESR	–0.003	0.039	0.944	0.997	0.925	1.076
BMI-SD	0.778	0.194	0.001	2.177	1.487	3.186
HP-positive gastritis	2.283	0.553	0.001	9.810	3.320	28.987
Constant	–3.034	0.726	0.001	0.048	-	-

ALT: *Alanine aminotransferase,* ***AST:*** *Aspartate aminotransferase,* ***BMI-SD:*** *Body mass index standard deviation score,* ***ESR:*** *Erythrocyte sedimentation rate,* ***HP:*** *Helicobacter pylori,* ***B:*** *Regression coefficient,* ***S.E:*** *Standard error,* ***Exp(B):*** *Odds ratio,* ***CI:*** *Confidence interval. Model fit was evaluated using Cox and Snell and Nagelkerke R^2 values. Model 1: Cox ve Snell R Square = 0.116; Nagelkerke R Square = 0.205, Model 2: Cox ve Snell R Square = 0.208; Nagelkerke R Square = 0.366*

and transmembrane translocation. Immune cell infiltration triggered by the microorganism induces proinflammatory cytokines, and chronic inflammation leads to the production of reactive oxygen species, cellular damage, and ultimately, carcinogenesis (18). Beyond well-known gastric effects of HP, the association between HP and various systemic diseases is increasingly being reported (19). Among all these, iron deficiency anemia is the most widely recognized; its prevalence occurs at a high rate in adolescents with HP-positive gastritis compared to adolescents with HP-negative gastritis (20). Furthermore, Type 1 Diabetes Mellitus, atherosclerosis, and pernicious anemia have also been found to be associated with HP (21,22). Studies based on pathogenetic mechanisms have reported that HP induces chronic systemic inflammation by stimulating T and B cells, macrophages, and other inflammatory mediators (23). Furthermore, HP treatment has been shown to reduce IgE, IL-4, and IL-13 levels and to increase IFN-γ and IL-10 levels, producing a potential anti-inflammatory effect (24).

Literature generally reports an increased correlation between HP infection and inflammatory parameters such as ESR and CRP (25,26). However, in our study, a weak and negative correlation was found between HP gastritis and CRP and ESR. This difference is thought to be related to the fact that HP infection in childhood mostly presents with a local and low-grade inflammatory response, and non-significant systemic acute phase response.

A weak symptom-disease severity correlation has been emphasized in the literature, HP colonization density and inflammation severity are considered to indicate the clinical presentation (16,27). Due to an insufficient number of patients regarding HP density, patients could not be evaluated according to the presence and severity of HP symptoms. In addition to the gastointestinal outcomes, HP may also be linked with under-recognized systemic implications, including hepatic inflammation, especially in pediatric patients where information is scarce. Fatty liver disease is a type of liver damage caused by metabolic stress. While numerous research from Asia have proposed that HP could be an individual contributor for fatty liver, others have not supported this relationship (28,29).

Previous studies have shown that the relationship between HP infection and hepatosteatosis may be associated with obesity and metabolic dysfunction (30). However, studies in the pediatric population report that HP infection may be associated with fatty liver disease independently of obesity and may be considered a potential risk factor (31). In our study, HP presence was related to hepatosteatosis, and increased HP positivity associated with a greater hepatosteatosis severity. Although an association was observed, its clinical implications appear modest and should be interpreted cautiously. The persistence of the association observed between HP-positive gastritis and hepatosteatosis after adjusting for BMI-SD in our study suggests that HP infection may contribute to hepatic steatosis through mechanisms beyond just fat accumulation, possibly via chronic low-grade inflammation and metabolic dysregulation, as previously suggested in pediatric and adult studies (9-11,19,28). This result suggests that HP may have a negative impact on liver health. However, the variable virulence of HP strains as well as host genetic predisposition also influence clinical outcomes (15). The virulence factors of HP strains and metabolic markers associated with hepatosteatosis were not evaluated. More comprehensive studies may reveal a link between hepatosteatosis and strain and host characteristics.

In the population we studied, no association was observed between BMI-SD and HP positivity. This supports the notion that hepatosteatosis was not largely mediated by obesity, indicating the plausibility of an HP-related link. Consistent with this, earlier research has also showed no association between overweight status or obesity and HP infection (32). Our study showed that BMI-SD was one of the strongest factors associated with the hepatosteatosis. However, it is noteworthy that HP-positive gastritis also occurs together with hepatosteatosis, and this relationship cannot be explained solely by BMI. The inclusion of HP positivity in the evaluation allowed for a more comprehensive consideration of the possible determinants of hepatosteatosis and suggested that HP infection may have an independent contribution to the hepatosteatosis process. This indicates

that HP gastritis may play a role in the development of hepatosteatosis through non-obesity mechanisms.

Emerging evidence suggests that HP infection may be associated with a growing number of extragastric pathologies. However, these pathologies do not typically manifest in childhood and may be influenced by various factors, such as age, gender, race, and geographic location. Therefore, large-scale, multicenter research is warranted to elucidate the role of extragastric manifestations, such as liver disease, in the pathogenesis of these conditions.

Strengths of this study include its focus on a childhood patient group, the grading of hepatosteatosis, the largely elimination of the influence of obesity, and the use of multifaceted clinical and histopathological data. Furthermore, demonstrating a relationship between HP colonization and the severity of hepatosteatosis increases the biological consistency supporting the possible metabolic effects of bacterial infection. The single-center, retrospective design of our research limited comprehensive evaluation of clinical symptoms and the identification of a causal association.

Limitations

The main limitations of this study include its retrospective design and the fact that hepatosteatosis was evaluated solely by ultrasonography. Due to the retrospective nature of the study, the inability to ensure imaging standardization, inter-observer agreement, and blinding conditions may have limited the reliability of hepatosteatosis grading.

Furthermore, this study does not allow for determining whether HP gastritis occurs before or after the development of hepatosteatosis. Accordingly, the findings only show a correlation between HP gastritis and hepatosteatosis, not a causal or temporal relationship.

Conclusion

This study highlights the relationship between HP gastritis and hepatosteatosis in childhood, demonstrating that the potential extragastric effects of HP infection should not be overlooked. The findings suggest that HP-positive gastritis may be associated with hepatosteatosis, even in children without metabolic risk factors. Therefore, hepatic health should also be monitored in patients with HP-positive gastritis who have gastric complaints. However, as the findings reflect an association rather than a causal relationship, prospective and longitudinal studies are needed to elucidate the potential effects of HP eradication on hepatosteatosis and to clarify the temporal aspects of this relationship.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Kayseri City Training and Research Hospital (15.04.2025, reference number: 407).

Contribution of the authors

Study conception and design: BDS, EZE; data collection: BDS, EZE; analysis and interpretation of results: BDS; draft manuscript preparation: BDS, EZE. All authors reviewed the results and approved the final version of the article.

Source of funding

The authors declare the study received no funding.

Conflict of interest

The authors declare that there is no conflict of interest.

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