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Helicobacter Pylori Infection among Children with Chronic Liver Diseases

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Abstract

Background: *Helicobacter pylori* (*H.pylori*) infection is a gastrointestinal pathogen with emerging evidence linking it to chronic liver diseases (CLD). **Aim:** To investigate the prevalence of *H. pylori* infection among children with CLD, its endoscopic and histopathological associations, and its effect on disease severity and severity of bleeding. **Methods:** This cross-sectional study included 63 children diagnosed with CLD at the National Liver Institute, Menoufia University, Egypt. Patients were categorized into *H. pylori*-positive and *H. pylori*-negative groups. All patients underwent gastrointestinal endoscopy, with

histopathological examination and *H. pylori* stool antigen in stools. Demographic, clinical, laboratory, endoscopic, and histopathological findings were analyzed. **Results:** *H. pylori*-positive patients showed higher prevalence of abdominal pain, dyspepsia, anemia, melena, and hematemesis ($P < 0.05$). *H. pylori* infection was significantly associated with lower hemoglobin and elevated liver enzymes: ALT (89.3% vs. 62.9%, $P = 0.000$), AST (75% vs. 37.14%, $P = 0.003$), and total serum bilirubin (92.86% vs. 51.43%, $P < 0.001$). Endoscopic findings showed a significant association with gastritis ($P = 0.007$) and gastric nodularity ($P = 0.006$) in *H. pylori*-positive patient. A significantly higher infiltration of inflammatory cells and lymphoid follicle formation in both duodenal and gastric biopsies of *H. pylori*-positive patients. The need for intervention (sclerotherapy/banding) was significantly higher in *H. pylori*-positive patients. GBS was not statistically different between the two groups. **Conclusion:** *H. pylori* infection in children with CLD was associated with a higher prevalence of gastritis, gastric nodularity, and increased gastrointestinal bleeding symptoms with increased rates of sclerotherapy/banding in patient with CLD.

1- Introduction:

Helicobacter Pylori (*H. pylori*) is a microaerophilic, gram-negative bacillus which can penetrate the gastric mucosal layer. *H. pylori*-induced inflammation can lead to chronic gastritis, peptic ulcer, gastric adenocarcinoma, gastric mucosa-associated lymphoid tissue lymphoma, gastroesophageal reflux disease and functional dyspepsia. In the developed world, the frequency of *H. pylori* infection is quickly decreasing, whereas it stays largely steady in poor nations. In children, the overall prevalence of *H. pylori* infection was 32.3%. [1]

Children with *H. pylori* infection may exhibit various gastrointestinal symptoms, although these can vary in severity and may not always be specific to *H. pylori*. Recurrent or persistent abdominal pain, often described as a dull or burning sensation, is one of the hallmark symptoms of *H. pylori* infection in children. Some children may experience

intermittent nausea or episodic vomiting. These symptoms may be more pronounced in the morning or when the stomach is empty. Children with *H. pylori* infection may also complain of a bloated feeling or early satiety, even after consuming small amounts of food and decreased appetite in some children, resulting in weight loss or poor weight gain. [2]

In pediatric practice, endoscopy is warranted in the case of familial history of stomach cancer, or in children with iron deficiency anemia that fails to respond to standard medical management. In such cases biopsy and endoscopy facilitates detection of *H. pylori* and treatment can be directed towards the identified pathogen. Endoscopy is performed to show inflammation, mucosal atrophy, erythema, nodularity, intestinal metaplasia, ulcerations and bleeding. Histopathological examination for gastric and duodenal tissues, In the gastric samples the analysis focused on the presence of *H. pylori* (detected using Giemsa stain), lymphoid follicles, inflammatory cell infiltration, neutrophil activity, and any evidence of dysplasia or malignancy. For the duodenal tissue, the assessment included villous atrophy, crypt hypertrophy, the villous-to-crypt ratio, presence of inflammatory cells, features of duodenitis, lymphocyte infiltration, lymphoid follicle formation or aggregation, and signs of dysplasia or malignancy. [3]

However, increasing research data open possibilities of strong associations between *H. pylori* infection with chronic liver diseases that include cirrhosis, Non-alcoholic fatty liver disease (NAFLD), and hepatocellular carcinoma. In children with cirrhosis, the presence of *H. pylori* is particularly challenging due to the compromised immune system resulting from chronic liver disease. A meta-analysis investigation identified the increased proportion of *H. pylori*-positive individuals in patients with cirrhosis and chronic hepatitis. [4] In a case-control study involving children under 18 years, the prevalence of *H. pylori* infection was found to be significantly higher among those with liver cirrhosis compared to healthy children. [5]

The treatment of *H. pylori* in pediatric CLD requires a careful balance between eradication efficacy and minimizing drug-related hepatotoxicity. Current guidelines from ESPGHAN and NASPGHAN recommend a standard triple therapy consisting of a proton pump inhibitor (PPI) combined with two antibiotics, typically amoxicillin and clarithromycin or metronidazole, for 10–14 days. In

children with CLD, drug metabolism may be altered necessitating dose adjustment and careful monitoring for adverse effects. Liver function should be assessed before initiating therapy especially in cases of advanced fibrosis or cirrhosis. [6]

The aim of the study was to investigate the prevalence of *H. pylori* infection in children with CLD and to report the interrelationship of *H. pylori* infection and CLD in children.

2- Material and methods:

Study design and population:

This cross-sectional study was carried out to investigate *H. pylori* infection in children with chronic liver diseases. The study included all pediatric patients presenting to endoscopic unit in the National Liver institute Menoufia university between August 2023 until August 2024. The patients included in the study were children up to 18 years old with chronic liver disease and were all tested for *H. Pylori* positivity using endoscopy and histopathological biopsies. Patient with risk of GIT bleeding due to tissue biopsy during endoscopy session with INR>1.9, and those with thrombocytopenia (platelet count less than 100 x 103 platelet/µl). Patients who did not wish to participate in the study and did not sign the informed consent for tissue biopsy were also excluded.

Sampling method and grouping:

This study in children included 63 children. Demographic data and detailed medical history of the patient including the family history, weight, height, Body Mass Index (BMI), *H. pylori* stool antigen linking Immune Sorbent test, liver function tests and complete blood count were collected. Patients were divided into two groups according to result of histopathology confirming the presence or absence of *H. pylori* infection; Group 1: *H. pylori*- positive, and Group 2: *H. pylori*-negative.

Stool Antigen Testing by ELISA

The Enzyme-Linked Immunosorbent Assay (ELISA) was used to test for *H. pylori* antigens in stool samples and was performed using Perkin Elmer, inc, USA (Cat. No 10224). [7]

Endoscopy and histopathology:

Esophagogastroduodenoscopy (EGD) examination was performed in the endoscopy unit at the National Liver institute, Menoufia University, Egypt, using an Olympus (GIF-H170) device with light source CLV-U40 and processor C.V240 and Pentax (EPK-I5000) endoscopes with OPTIVISTA EPK- i7010 HD Video Processor (Tokyo, Japan). All children underwent flexible EGD by an experienced endoscopist. All patients were kept nothing per oral for 4-6

hours prior to the procedure and assessed by anesthesiologist for general conditions and all vital signs. Endoscopy examination was applied to assess varices (esophageal and gastric) and its grades, the findings of *H. pylori* gastritis, erythema, erosions, ulcers, nodularity and signs of portal hypertensive gastropathy. Esophageal, gastric, and duodenal biopsies were taken for histopathological examination.

Histopathological analysis:

Biopsies were taken from the stomach lesser and greater curvatures of the antrum, lesser and greater curvature of the corpus, incisura angularis for optimal gastric biopsy and a biopsy from the duodenum according to Lee and Kim et al., [8] a total 6 biopsies were collected and sent for histopathological examination. Biopsy specimens were fixed in 10% formalin and embedded into paraffin wax before receiving staining with hematoxylin and eosin (H&E) (Loba Chemie Pvt, Ltd, India). Giemsa stain (Loba Chemie Pvt, Ltd, India) was used to detect *H. pylori*. [9] Histopathological features of the gastric tissue were examined, including gastric lymphoid follicles, infiltration of inflammatory cells, neutrophil activity, dysplasia or malignancies, and the presence of *H. pylori* detected by Giemsa stain. For the duodenum, histopathological features of the duodenum included inflammatory cell presence, villous atrophy, crypt hypertrophy, villous-to-crypt ratio, duodenitis, lymphocyte infiltration, lymphocyte aggregation or follicle formation, and malignancy or dysplasia. [3]

Scoring systems:

The pediatric end-stage liver disease (PELD) and Model for end-stage liver disease (MELD) scores calculated for each child, as PELD was used for children aged under 12 years and MELD was used for children aged 12 years or more, according to McDiamrid et al. and Kamath et al.. [10-12]

Modified glasgow blatchford score:

GBS was designed to predict the need for clinical intervention such as endoscope, surgical or blood transfusion. The modified version created by Abo Hussein et al. [13] to adapt pediatric patients was applied in this study.

Ethical considerations:

This research was conducted according to the guidelines laid down in the Declaration of Helsinki and was recommended and approved by the institutional revie board of the National Liver Institute, Menoufia University (approval No. 00672/2024). Ethical considerations included Patients' consent compliance, the researchers sought the parents' consent for the children, but the children assured their

cooperation during the study. To protect the privacy of the participants the data collected was disguised, and the storage of data was done under the recognized regulations such as the American Academy of Pediatrics. [14]

Statistical analysis:

In this study, Chi-square test was useful in determining the relationship between the categorical variables used. The categorical variables were *H. pylori* positivity/negativity, gender, the socio-economic level of the participants and clinical results concerning C. L. D. To compare the differences of the steady variables like age, BMI, level of liver enzymes or virtually any other biochemical parameter, the t-test was applied. Logistic regression was used to test the variables capable of influencing the occurrence of a specific type of liver disease in relation to variables.

3- Results:

A total of 63 children with chronic liver disease were included in this study. Among which, there was 28 males accounting for 44.4% of the whole sample, and 35 females representing 55.6% of the included 63 children. The mean age was of 10.16 ± 4.16 years, with the youngest patient 2 years old and the oldest 17 years old. Around half of the patients (50.8%) were living in rural areas and the remainder were living in urban areas, with 53 (84.13%) living in overcrowding conditions, while 10 (15.87%) were living in non-crowded homes. Most patients (68.3%) had uneducated mothers while 47.6% had uneducated fathers. As for nutritional habits, 61.9% of patients had history of eating from street venders as shown in **Table 1**.

The etiology of the cases among a total of 63 pediatric patients is shown in **Figure 1**. The most prevalent condition was Autoimmune Hepatitis (AIH), identified in 33 patients (52.38%), reflecting the dominant presence of autoimmune pathology in the cohort. Biliary Atresia Post-Kasai was the second most common diagnosis, found in 17 patients (26.98%), underscoring the significant burden of biliary disorders. Other diagnoses included Progressive Familial Intrahepatic Cholestasis (PFIC) in 5 patients (7.93%), and Wilson Disease and Undiagnosed Chronic Liver Disease, each in 2 patients (3.17%). Rare etiologies such as Ductal Hypoplasia, Biliary Hamartoma, Caroli Syndrome, and Congenital Hepatic Fibrosis were each reported in 1 patient (1.59%).

The analysis of factors associated with *H. pylori* positivity among the studied population showed gender related association (P=0.02), as males being more

likely to test positive for *H. pylori* compared to females. There was a significant relationship between *H. pylori* positivity and the region of residence, with patients living in rural areas showing a markedly higher prevalence (89.29%) of *H. pylori* positivity compared to urban residents (10.71%). Additionally, a higher number of patients who are *H. pylori* negative (80%) were living in urban cities. Similarly, crowding index was significantly associated with *H. pylori* positivity, as patients from crowded households demonstrated a higher prevalence (96.43%) compared to non-crowded households (3.7%). Nutritional habits indicated that patients consuming food from street vendors were having a higher prevalence of *H. pylori* positivity (82.1%) than those who did not (17.9%). Regarding anthropometric measurements, there was no significant changes in weight and BMI between both groups. However, in length/height differences were marginally significant, showing that *H. pylori* positive patients had lower average (normal) length/height (57.14%) compared to *H. pylori* negative patients (80%). Length/height below average was found in 42.86% in *H. pylori* positive patients, while in *H. pylori* negative patients only 20% were shorter than average, as shown in **Table 2**.

Patients with positive *H. pylori* showed more frequent occurrence of anemia, dyspepsia, abdominal pain, followed by hematemesis, abdominal enlargement, melena, and a history of encephalopathy, compared to those with negative *H. pylori*. Although requirement for blood transfusion was more common in *H. pylori*-positive patients (35.71% vs. 17.14%), this difference did not reach statistical significance (P = 0.092, OR: 2.69, 95% CI: 0.83–8.66). Other symptoms, such as fatigue, jaundice, ascites, and lower limb oedema, showed no significant association with *H. pylori* positivity. **Table 3**

H. pylori-positive patients had significantly lower hemoglobin levels (85.71% vs. 45.71%, P ≤ 0.001), indicating a strong association with anemia. Elevated ALT (89.3% vs. 62.9%, P ≤ 0.001), AST (75% vs. 37.14%, P = 0.003, OR: 5.08), and total serum bilirubin (92.86% vs. 51.43%, P < 0.001) were also more frequent in the *H. pylori*-positive group. Other hematologic and biochemical markers showed no significant differences. Ultrasonographically, *H. pylori* infection was significantly associated with portal hypertension (P < 0.001) and cirrhosis (39.29% vs. 17.14%, P = 0.049). Splenomegaly was more common in *H.*

pylori-positive patients but was not statistically significant (42.86% vs. 22.86%, P = 0.090). Hepatomegaly showed no significant association. Stool antigen test showed 85.71% sensitivity with a P value of 0.0001. **Table 4**

H. pylori-positive patients showed a higher prevalence of esophageal varices (71.43%) compared to *H. pylori*-negative patients (60%), this difference was not statistically significant. A trend toward more severe varices (Grade 2) in the *H. pylori*-positive group was noted, though also not statistically significant. Gastritis and gastric nodularity were significantly more frequent in *H. pylori*-positive patients (P = 0.007 and P = 0.006, respectively), with *H. pylori* infection increasing the odds of gastritis more than fourfold. The need for endoscopic intervention (sclerotherapy) was significantly associated with *H. pylori* positivity (P = 0.001). **Table 5**

The need for intervention due to GIT bleeding was evaluated using the Modified Glasgow-Blatchford Score (GBS) with cut-off point at 8.5 which was calculated for all studied patients according to Abo Hussein et al., [13]. However, scores were slightly higher in *H. pylori positive patients*, differences were not statistically significant. Score distribution is shown in **Figure 2**.

Histopathological analysis showed significantly more inflammatory cell infiltration in *H. pylori*-positive duodenal biopsies (100% vs. 85.71%, P = 0.037), with higher lymphocyte infiltration (75% vs. 42.86%, P = 0.010) and increased lymphoid follicle formation (53.57% vs. 11.43%, P < 0.001). No significant differences were found in villous atrophy, crypt hypertrophy, villous-to-crypt ratio, or duodenitis, and no malignancy or dysplasia was detected. In gastric biopsies, lymphoid follicles were more frequent in *H. pylori*-positive patients (57.14% vs. 28.57%, P = 0.022), and neutrophil activity was significantly elevated (P < 0.001), while inflammatory cell infiltration showed no significant variation. **Table 6**

The analysis compared MELD and PELD scores in relation to *H. pylori* positivity among pediatric patients, stratified by age group, as shown in (Figure 3.A). Overall, no statistically significant association was found between *H. pylori* infection status and liver disease severity based on MELD or PELD scores. **Figure 3.B and C**

4- Discussion:

The infection of *H. pylori* in pediatric patients is a major global health concern, especially in poorer nations where the prevalence is higher due to inadequate sanitation and overcrowding. [1]

Regarding the relationship between *H. pylori* and CLD in children, only few articles in the literature investigated similar relationships. Lupu et al. [15] found a significant relationship between *H. pylori* infection and liver cytolysis syndrome in children, suggesting an increased risk of liver cytolysis in infected children. Barakat et al. [16] found that *H. pylori* infection was more prevalent in children with NAFLD (64%) than in controls (25%), with Cag A-positive strains linked to more severe NAFLD. An earlier study in 2010 showed that *H. pylori* has been detected in 65.6% of liver tissue samples from children with chronic liver diseases. Prevalence varies by geography and ethnicity, suggesting environmental influences. [17]

This is a cross-sectional study included 63 children diagnosed with CLD, 44.5% were males while 55.5% were females. Zou et al. and Kim and Lim, [18, 19] agreed with the findings of this study regarding the gender distribution of children with chronic liver disease**.** However, Xiao et al. [20] opposed these findings, suggesting no gender differences**.** The difference may be due to the nature of the study population as the study focused on metabolic liver diseases rather than chronic pediatric liver conditions.

In the present study, patients were categorized into *H. pylori*-positive, and *H. pylori*-negative groups using endoscopy confirmed with histopathology. *H. pylori*-positive group represents 28 (44.4%) of the pediatric patients of CLD.

In similarity with our results, Ibrahim et al. [21] show in their meta-analysis that the *H. pylori* infection was more frequent in males than in females**.** But in contrast, Lupu et al. [22] reported that the female sex was affected in 68.9% of cases and 32.8% had the bacteria present**.** Although further research is needed to understand the mechanisms by which sex may influence the acquisition and/or persistence of infection.

In the present study, there was a statistical significant difference according to region (P=<0.001) as pediatrics inhabit rural areas being more likely to be positive for *H. pylori* compared to urban residents. Balas et al. [23] confirmed our results, suggesting that limited access to clean water and sanitation in rural areas plays a crucial role in the transmission of *H. pylori*.

As per our findings, there was a statistical significant difference according to crowding index (P=0.017) as patients from crowded households demonstrated a higher prevalence *H. pylori* infection. Similarly, Pornsiripratharn et al. [24] found

that individuals living in crowded conditions were more susceptible due to close contact and increased exposure to infected individuals.

In our results, there was a statistical significant difference according to nutritional habits (P=0.011) as patients consuming food from street vendors were having a higher prevalence of *H. pylori* positivity. Also, Dănilă et al. [25] demonstrated that individuals consuming food from unregulated sources had a higher prevalence of gastric pathology linked to *H. pylori* infection. In contrast, Barakat et al. [26] argued that dietary habits alone do not significantly contribute to *H. pylori* infection. Their study suggested that genetic factors and immune responses play a more dominant role in determining infection risk.

In the present study, educational levels of both mothers and fathers were not significantly associated with *H. pylori* positivity (P = 0.628 and P = 0.397, respectively). Awuku et al. [27] found no significant association between the educational level of either parents and the prevalence of *H. pylori* infection. Darko et al. and [28]. Malaty et al., [29] however, found that children whose mothers did not complete high school had higher rates of *H. pylori* infection**.** This can be because that better-educated parents are more likely to adopt improved hygiene practices.

In the present study, according to anthropometric measures (height, weight, BMI), there was a statistical significant difference according to Length / height when *H. pylori* positive group (P=0.049). Jishnu et al. [30] reported a noticeable link between *H. pylori* infection and reduced height in children**.** Lupu *et al.*, [15] however, did not establish a clear relationship between *H. pylori* infection and anthropometric parameters.

As per our results, regarding clinical symptoms, we found that patients with *H. pylori* positivity were significantly present more frequently with anemia, dyspepsia, abdominal pain, hematemesis, abdominal enlargement, melena, and a history of encephalopathy compared to those without infection (P<0.05 for each). In addition, Attallah *et al.* identified a higher frequency of hematemesis among *H. pylori*-positive patients with chronic hepatitis C and emphasized the role of *H. pylori* infection in exacerbating bleeding tendencies. [31] Zhang et al. [32] found no significant relationship between *H. pylori* infection and gastrointestinal bleeding in their study of asymptomatic school-age children. Their findings suggested that other factors, such as genetic predisposition and environmental

influences, may play a more dominant role in the development of upper gastrointestinal bleeding, offering a different perspective from the current results.

In the present study, according to laboratory and radiological results, there was a statistically significant low Hb in *H. pylori* positive group (P=0.001) indicating a strong association with anemia. *H. pylori* positivity was significantly associated with certain ultrasonographic findings including portal hypertension (P < 0.001) and cirrhosis (P = 0.049) correlated with *H. pylori* positivity. For the stool antigen test, it showed high sensitivity (85.71%) and 100% specificity, with a *P* value of 0.0001, used as a non-invasive *H. pylori* marker. Kim and Lim, [18] reported a notable association between *H. pylori* infection and anemia in pediatric patients. Their research demonstrated that children with iron deficiency anemia frequently exhibited gastrointestinal lesions, which were often linked to *H. pylori.* On the other hand, Haghi-Ashtiani et al. [33] did not find any associations with iron deficiency anemia in children. Lupu et al. and Bolia & Srivastava, [15, 34] highlighted how chronic *H. pylori* infection can exacerbate hepatic vascular complications, including portal hypertension and cirrhosis**.** *H. pylori* stool antigen test was found to be quick, non-invasive, and reliable for the detection of *H. pylori* in children with 91.3% sensitivity [35]. However, the one step polyclonal stool antigen test was occasionally reported to be unreliable [36].

In the present study, according to esophageal and gastric varices, we found that esophageal varices were more frequently observed in *H. pylori*-positive patients, However, this difference was not statistically significant, suggesting that while *H. pylori* infection might be associated with varices, it does not strongly influence their occurrence. According to varices grades, while not statistically different, grade 2 varices were about three times more common in *H. pylori*-positive patients, suggesting a potential trend toward more advanced CLD in this group. According to our endoscopic findings related to *H. pylori* infection we found a strong and significant association with gastritis and nodularity (P=0.007 & P=0.006, respectively). While no significant differences were observed in other endoscopic findings. In similarity, Jishnu et al. [30] found a similar pattern of varices grades distribution**.** Zou et al. [19] did not find a significant association between *H. pylori* infection and esophageal varices.

Similarly, Miao et al. [37] aligned with the observation that ulcers were absent in the infected group. They hypothesized that other factors, such as NSAID use or acid reflux, might be more influential in ulcer development than *H. pylori* itself. In contrary, Hashim et al. [38] found that some pediatric patients with chronic *H. pylori* infection developed esophageal ulcers, possibly due to prolonged inflammation and acid imbalance. They proposed that while *H. pylori* may not be the primary cause, it could exacerbate underlying esophageal conditions, leading to a higher risk of ulceration in some cases**.** Similarly, Miao et al. [37] reported the finding that *H. pylori* infection showed a strong and significant association with gastritis, whereas Van Veen et al. [39] revealed that gastric nodularity was more prevalent in *H. pylori*-positive patients.

In the present study, on investigating the relationship between the need for interventions (banding or sclerotherapy) and *H. pylori* positivity, it was evident that sclerotherapy was significantly higher (P=0.001) in *H. pylori* positive patients. Attallah et al., [31] observed the same finding while Zhang et al. [32] did not find a statistically significant relationship between *H. pylori* infection and the necessity for endoscopic interventions in asymptomatic school-age children.

In this study, according to the modified Glasgow Blatchford score, the requirement for hospital-based intervention for GIT bleeding, such as endoscopic, medical, and blood transfusion, was comparable in *H. pylori*-positive and negative patients. This could be explained by the cross-sectional nature of our study, in which the included patients were recruited during endoscopic intervention, either diagnostic or treatment, and were more or less hemodynamically stable.

In agreement with our findings, Moreno Trigos et al. [40] suggested that *H. pylori* infection alone does not significantly increase the need for endoscopic intervention. They argued that concurrent gastrointestinal disorders and patient comorbidities, play a more substantial role in determining the severity of bleeding and the necessity for intervention. However, Van Veen et al. [39] highlighted that *H. pylori*-positive patients often present with more severe gastrointestinal symptoms, increasing the likelihood of requiring endoscopic intervention. Their study emphasized that chronic inflammation and mucosal damage caused by *H. pylori* infection contribute to a higher risk of gastrointestinal bleeding, necessitating more frequent endoscopic procedures.

According to our histopathological findings, lymphocyte infiltration was significantly more common in *H. pylori*-positive patients (P = 0.010). Similarly, lymphocyte aggregation or the presence of lymphoid follicles was significantly higher in the *H. pylori*-positive group (P < 0.001). We also found significantly more inflammatory cell infiltration in *H. pylori*-positive duodenal biopsies. No cases of malignancy or dysplasia were detected in either group. These findings highlight a significant association between *H. pylori* infection and increased inflammatory activity, particularly lymphocyte infiltration and aggregation, emphasizing the potential role of *H. pylori* in triggering immune responses.

While Moreno Trigos et al. [40] suggested that the presence of lymphoid follicles may not be exclusively linked to *H. pylori* but could also be influenced by other co-existing gastric pathogens. They argued that while *H. pylori* plays a role, other microbiota alterations might contribute to histopathological changes, explaining variability across different studies. Attallah et al. [31] reported significant immune activation, particularly through lymphocyte infiltration and aggregation, aligning with the observed histopathological changes in *H. pylori*-positive patients. This reinforces the role of *H. pylori* in promoting duodenal inflammation, despite the lack of statistical significance in some cases**.** In addition, Van Veen et al. [39] suggested that not all cases of *H. pylori* infection led to significant neutrophilic infiltration. They proposed that host genetic factors and bacterial strain variability influence the severity of the inflammatory response, meaning that some infected individuals may exhibit minimal neutrophilic activity despite active infection**.** Also, Zhang et al. [32] presented a differing perspective and suggested that duodenal inflammation might not be exclusively attributed to *H. pylori*. They proposed that other factors, such as dietary habits, gut microbiota composition, and genetic predisposition, could contribute to duodenal inflammatory changes.

In the present study, concerning the MELD and PELD scores among patients with and without *H. pylori* infection, the median PELD score remained comparable between both groups, indicating no significant difference in disease severity based on this parameter. Xiao et al. [20] also aligned with the results regarding survival rates and disease prognosis. Their research demonstrated that *H. pylori* infection was not a determining factor in liver disease severity or survival predictions, further supporting the observation that other

underlying conditions may play a more significant role in patient outcomes.

In contrast, Kim and Lim [18] suggested that *H. pylori* infection could contribute to worsening gastrointestinal lesions and chronic inflammation, which may influence overall disease progression, particularly in pediatric patients. They argued that chronic *H. pylori* infection might aggravate pre-existing conditions, potentially affecting long-term survival rates even if short-term outcomes appear stable.

We also point out the need for further analyses evaluating the efficacy of *H. pylori* eradication, the long-term clinical effects of such a potential relationship and the evaluation of multiple *H. pylori* strains in CLD among the pediatric population.

5- Conclusion:

H. pylori infection in children with CLD was associated with a higher prevalence of gastritis, gastric nodularity, and increased gastrointestinal bleeding symptoms in the form of hematemesis, melena and anemia with increased rates of sclerotherapy/banding in patient with CLD. *H. pylori* infection may be accused in significant increase in GIT symptoms and more deterioration in their liver condition. Further studies are needed on large number of patients to clarify the impact of *H. pylori* treatment on liver disease status and liver disease progression.

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Table 1: Demographic data of the studied patients

Demographic data		Mean ± SD	Min - Max
Age (years)		10.16 ± 4.16	2 - 17
Gender	**Male**	28 (44.4%)	
	Female	35 (55.6%)	
Socioeconomic factors		N (%)	
Region	**Rural**	32 (50.8%)	
	Urban	31 (49.2%)	
Crowding index*	**Crowded**	53 (84.13%)	
	Non crowded	10 (15.87%)	
Mother's education	**Educated**	20 (31.7%)	
	Uneducated	43 (68.3%)	
Father's education	**Educated**	33 (52.4%)	
	Uneducated	30 (47.6%)	
Nutritional habits	**Eat from street venders**	39 (61.9%)	
	Don't eat from street venders	24 (38.1%)	

Data are presented as mean ± SD or frequency (%).* Crowding Index = Total number of residents/Total number of rooms used for sleeping. An index greater than or equal to 2.0 indicated overcrowding [41].

Table 2: Correlation between *H. pylori* positivity and patients' characteristics in studied patients

Demographics						
Parameters		**Positivity for *H. pylori***		***P*-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Age group (years)	**<3**	0 (0%)	3 (8.57%)	0.274		
	3 – 6	7 (25%)	4 (11.43%)			
	>6 – 10	7 (25%)	11 (31.43%)			
	>10	14 (50%)	17 (48.57%)			
Gender	**Male**	17 (60.71%)	11 (31.43%)	**0.02**	3.37	(1.19, 9.55)
	Female	11 (39.29%)	24 (68.57%)			
Socioeconomic factors						
Parameters		**Positivity for *H. pylori***		***P*-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Region	**Rural**	25 (89.29%)	7 (20%)	**<0.001**	33.33	(7.77, 142.97)
	Urban	3 (10.71%)	28 (80%)			
Crowding index	**Crowded**	27 (96.43%)	26 (74.29%)	**0.017**	9.35	(1.11, 79.04)
	Non crowded	1 (3.7%)	9 (25.71%)			
Mother education	**Educated**	8 (28.57%)	12 (34.29%)	0.628	1.3	(0.44, 3.83)
	Uneducated	20 (71.43%)	23 (65.71%)			
Father education	**Educated**	13 (46.43%)	20 (57.14%)	0.397	1.54	(0.57. 4.18)
	Uneducated	15 (53.57%)	15 (42.86%)			
Nutritional habits	**Eat from street vendors**	23 (82.1%)	18 (51.43%)	0.011	4.34	(1.35, 14.03)
	Don't eat from street vendors	5 (17.9%)	17 (48.57%)			
Anthropometric measurements						
Parameters		**Positivity for *H. pylori***		***P*-value**	**OR**	**95%CI**
		Yes (n=28)	**Yes (n=28)**			
Length / height	**Above average**	0	0	**0.049**	0.33	(0.1,1.02)
	Average	16 (57.14%)	28 (80%)			
	Below average	12 (42.86%)	7 (20%)			
Weight	**Overweight**	1 (3.57%)	2 (5.71%)	0.776	0.8	

	Average	21 (75%)	29 (82.86%)			(0.17, 3.71)
	Underweight	6 (21.43%)	4 (11.43%)			
BMI index	**Underweight**	2 (7.14%)	6 (17.14%)	0.144	2.98	(0.38, 25.43)
	Healthy weight	20 (71.43%)	17 (48.57%)			
	Risk of overweight	3 (10.71%)	10 (28.57%)			
	Overweight	3 (10.71%)	2 (5.71%)			

Data are presented as frequency (%).

Table 3: Correlation between *H. pylori* positivity and symptoms in studied patients

Variable	Positivity for *H. pylori*		P-value	OR	95%CI
	Yes (n=28)	No (n=35)			
Anemia	24 (85.71%)	16 (45.71%)	**0.001**	7.13	(2.04, 24.87)
Dyspepsia	21 (75%)	13 (37.14%)	**0.003**	5.08	(1.7, 15.2)
Abdominal pain	22 (78.57%)	11 (31.43%)	**<0.001**	8	(2.53, 25.28)
Fatigue	11 (39.29%)	13 (37.14%)	0.862	1.1	(0.39, 3.04)
Hematemesis	16 (57.14%)	7 (20%)	**0.002**	5.33	(1.75, 16.29)
Abdominal enlargement	11 (39.29%)	5 (14.29%)	**0.023**	3.88	(1.15, 13.06)
Blood transfusion need	10 (35.71%)	6 (17.14%)	0.092	2.69	(0.83, 8.66)
Jaundice	7 (25%)	8 (22.86%)	0.843	1.13	(0.35, 3.6)
Melena	9 (32.14%)	1 (2.86%)	**0.002**	16.11	(1.89, 137.01)
Bleeding per rectum	4 (14.29%)	2 (5.72%)	0.249	1.38	(0.47, 16.26)
Encephalopathy history	7 (25%)	2 (5.71%)	**0.030**	5.5	(1.04, 29.04)
Ascites	3 (10.71%)	3 (8.57%)	>0.999	1.28	(0.24, 6.89)
Lower limb oedema	1 (3.57%)	0 (0%)	0.444	--	--
Clubbing	0 (0%)	1 (2.9%)	>0.999	--	--

Data are presented as frequency (%).

Table 4: Correlation between *H. pylori* positivity and laboratory and radiological data in studied patients

Results of laboratory analysis						
Variables		**Positivity for *H. pylori***		P-value	OR	95%CI
		Yes (n=28)	**No (n=35)**			
WBC	**Abnormal (Leukopenia / Leucocytosis)**	11 (39.29%)	12 (34.29%)	0.682	1.24	(0.44, 3.48)
	Normal	17 (60.71%)	23 (65.71%)			
Hb	**Low**	24 (85.71%)	16 (45.71%)	**0.001**	7.13	(2.04, 24.87)
	Normal	4 (14.29%)	19 (54.29%)			
HCT	**Low**	16 (57.14%)	20 (57.14%)	>0.999	1	(0.37, 2.73)
	Normal	12 (42.86%)	15 (42.86%)			
MCV	**Low**	12 (42.86%)	13 (37.14%)	0.645	1.27	(0.46, 3.5)
	Normal	16 (57.14%)	22 (62.86%)			
PLT	**Low**	14 (50%)	14 (40%)	0.427	1.5	(0.55, 4.09)
	Normal	14 (50%)	21 (60%)			
ALT	**High**	25 (89.3%)	22 (62.9%)	**≤0.001**	4.92	(1.24,19.27)
	Normal	3 (10.7%)	13 (37.1%)			
AST	**High**	21 (75%)	13 (37.14%)	**0.003**	5.08	(1.7, 15.2)
	Normal	7 (25%)	22 (62.86%)			
ALK	**High**	18 (64.29%)	18 (51.43%)	0.306	1.7	(0.61, 4.71)
	Normal	10 (35.71%)	17 (48.57%)			
GGT	**High**	20 (71.43%)	19 (54.29%)	0.164	2.1	(0.73, 6.05)
	Normal	8 (28.57%)	16 (45.71%)			
Albumin	**Low**	12 (42.86%)	11 (31.43%)	0.349	1.64	(0.58, 4.6)
	Normal	16 (57.14%)	24 (68.57%)			
TSB	**High**	26 (92.86%)	18 (51.43%)	**<0.001**	12.28	(2.52, 59.83)
	Normal	2 (7.14%)	17 (48.57%)			
DSB	**High**	15 (53.57%)	19 (54.29%)	0.955	0.97	(0.36, 2.63)
	Normal	13 (46.43%)	16 (45.71%)			
Creatinine	**High**	10 (35.71%)	12 (34.29%)	0.906	1.06	(0.38, 3.02)
	Normal	18 (64.29%)	23 (65.71%)			
Urea	**High**	10 (35.71%)	11 (31.43%)	0.720	1.21	(0.42, 3.47)
	Normal	18 (64.29%)	24 (68.57%)			
Ultrasonography findings						
Variable		**Positivity for *H. pylori***		**P-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Portal hypertension		18 (64.29%)	6 (17.14%)	**<0.001**	8.7	(2.7, 28.05)
Splenomegaly		12 (42.86%)	8 (22.86%)	0.090	2.53	(0.85, 7.51)

Cirrhosis	11 (39.29%)	6 (17.14%)	**0.049**	3.13	(0.98, 9.99)
Hepatomegaly	5 (17.86%)	3 (8.57%)	0.449	2.32	(0.5, 10.69)
Stool antigen findings					
	Positivity for *H. pylori*		**P-value**	**OR**	**95%CI**
	Yes (n=28)	**No (n=35)**			
Positive	**24 (85.71%)**	**0 (0%)**	**<0.001**	210	(17, 2600)
Negative	**4 (14.29%)**	**35 (100%)**			

Data are presented as frequency (%). WBCs: White blood cells, Hb: Hemoglobin, HCT: Hematocrit, MCV: Mean Corpuscular Volume, PLT: Platelets, ALT: Alanine transaminase, AST: Aspartate aminotransferase, ALK: Alkaline phosphatase, GGT: Gamma-glutamyl transpeptidase, TSB: Total bilirubin, DSB: Direct bilirubin, PTT: Partial Thromboplastin Time, PT: Prothrombin time, INR: International Normalized Ratio. Numerical data are presented as mean ± SD or median (IQR) as appropriate and categorical data are presented as frequency (%), OR: Odds ratio, CI: Confidence interval, Statistical significance at P-value<0.05.

Table 5: Correlation between *H. pylori* positivity and endoscopic findings in studied patients

Esophageal Endoscopy						
Variable		**Positivity for *H. pylori***		**P-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Varices		20 (71.43%)	21 (60%)	0.344	1.67	(0.58, 4.82)
Varices grade	**No varices**	8 (28.57%)	14 (40%)			
	Grade 1	13 (46.43%)	18 (51.14%)	0.96	8 (28.57)	12 (34.29)
	Grade 2	6 (21.43%)	2 (5.71%)	0.152	13 (46.43)	19 (54.29)
	Grade 3-4	1 (3.57%)	1 (2.86%)	0.78	5 (17.86)	2 (5.71)
Nodularity		2 (7.14%)	1 (2.86%)	0.470	2.62	(0.22, 30.44)
Ulcer		0 (0%)	2 (5.71%)	0.498		
Erosion		2 (7.14%)	1 (2.86%)	0.580	2.62	(0.22, 30.44)
Mucosal erythema		2 (7.14%)	2 (5.71%)	>0.999	1.27	(0.17, 9.63)
Gastric endoscopy						
Variable		**Positivity for *H. pylori***		**P-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Total Varices		3 (10.71%)	3 (8.57%)	>0.999	1.28	(0.24, 6.89)
Varices grade	**No varices**	25 (89.29%)	32 (91.43%)	>0.999		
	Grade 1	2 (7.14%)	2 (5.71%)		1.28	(0.17, 9.73)
	Grade 2	1 (3.57%)	1 (2.86%)		1.28	(0.08, 21.49)
	Grade 3-4	0 (0%)	0 (0%)			
Total PHG		21 (75%)	30 (85.71%)	0.282	0.5	(0.14, 1.79)
PHG grade	**No**	7 (25%)	5 (14.29%)			
	Mild	13 (46.43%)	23 (65.71%)		0.4	(0.11, 1.53)
	Moderate	4 (14.29%)	5 (14.29%)		0.57	(0.1, 3.27)
	Severe	4 (14.29%)	2 (5.71%)		1.43	(0.18, 11.09)
Gastritis		20 (71.43%)	13 (37.14%)	**0.007**	4.23	(1.45, 12.32)
Nodularity		9 (32.14)	2 (5.71)	**0.006**	2	(0.56, 7.16)

Ulcers		0 (0%)	1 (2.86%)	>0.99		
Mucosal erythema		1 (3.57%)	1 (2.86%)	>0.99	1.26	(0.08, 21.07)
Duodenal Endoscopy						
Variable		**Positivity for *H. pylori***		**P-value**	**OR**	**95%CI**
		Yes (n=28)	**No (n=35)**			
Duedenopathy		23 (82.14%)	33 (94.29%)	0.226	0.28	(0.05, 1.56)
Nodularity		2 (7.14%)	1 (2.86%)	0.427	2.62	(0.22, 30.44)
Ulcers		0 (0%)	2 (5.71%)	0.498		
Erosions		0 (0%)	1 (2.86%)	>0.99		
Mucosal erythema		1 (3.57%)	2 (5.71%)	>0.99	0.61	(0.05, 7.11)

Data are presented as frequency (%).

Table 6: Correlation between *H. pylori* positivity and histopathological findings in studied patients

Duodenal Histopathology					
Variables	**Positivity for *H. pylori***		**p-value**	**OR**	**C.I**
	Yes (n=28)	**No (n=35)**			
Inflammatory cells	28 (100%)	30 (85.71%)	**0.037**		
Villous atrophy	3 (10.71%)	5 (14.29%)	0.672	0.72	(0.16,3.31)
Crypt hypertrophy	1 (3.57%)	0 (0%)	0.260		
Decreased villous to crypt ratio	8 (29.63%)	9 (25.71%)	0.732	0.82	(0.27,2.52)
Duodenitis	24 (88.9%)	26 (76.47%)	0.210	2.46	(0.58,10.37)
Lymphocyte infiltration	21 (75%)	15 (42.86%)	**0.010**	4	(1.35,11.85)
Lymphocyte aggregation or follicles	15 (53.57%)	4 (11.43%)	**0.000**	8.94	(2.49,32.13)
Malignancy or dysplasia	0 (0%)	0 (0%)	0.000	-	---
Gastric Histopathology					
Gastric lymphoid follicles	16 (57.14%)	10 (28.57%)	**0.022**	3.33	(1.17, 9.51)
Infiltration of inflammatory cells	27 (96.43%)	29 (82.86%)	0.120	5.59	(0.63, 49.47)
Neutrophil activity	20 (71.43%)	3 (8.57%)	**<0.001**	26.67	(6.32, 112.52)
Dysplasia or malignancies	0 (0%)	0 (0%)			
Detection of *H. pylori* organism by Giemsa stain	28 (100%)	0 (0%)			

Data are presented as frequency (%).

Figure legends

Figure 1: The etiology of the cases.

Figure 2: Shows the distribution of GBS scores among patients with negative or positive *H. pylori* scores. Negative *H. pylori* patients are represented by purple circles (n=35) while positive *H. pylori* patients are represented by brown triangles (n=28). Cut-off value was set at 8.5 and denoted by the dotted blue line.

Figure 3: (A) the distribution of PELD / MELD scores among patients with negative or positive *H. pylori* scores. Negative *H. pylori* patients are represented by purple circles (n=35) while positive *H. pylori* patients are represented by brown triangles (n=28). (B) scatterplot of PELD scores with a cut-off value set at 6 indicating minimum score and denoted by the dotted blue line. Moderate liver disease indicating required increased monitoring are set between 7-14. Scores above 15 (black dotted line) indicate severe liver disease and higher priority for transplantation. For MELD score (C), Values at 10 or lower are indicative of Mild liver disease; low mortality, 10-14 indicate moderate disease, increased risk of complications and to consider specialist referral. Scores of 15 or higher indicate severe liver disease, survival benefit and prioritize for transplantation.