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# ORIGINAL ARTICLE

# Association between Helicobacter Pylori Infection and Platelet Indices among the pediatric population in Aswan

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## ABSTRACT

**Keyword**: H. Pylori, platelets, children

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**Background:** Clinical studies on adults in developed countries have linked Helicobacter pylori infection with thrombocytopenia. However, there is extremely limited study in children, particularly in developing countries.**Objectives**: To investigate the effect of H. pylori infection on platelet indices in children. **Methods:** This observational cross-sectional study included 195 children with dyspepsia recruited from the gastroenterology pediatric department of Aswan University Hospital. **Results:** Children with *H. pylori* infection had significantly lower hemoglobin levels (11.015 ± 1.109 g/dL vs. 11.553 ± 1.00 g/dL, p=0.015), reduced platelet counts (p=0.004), and higher prevalence of low MCV (64.6% vs. 25.5%, p<0.001), low MCH (20.8% vs. 2.13%, p=0.004), leucopenia (27.1% vs. 8.5%, p=0.018), and low MPV (22.9% vs. 4.3%, p=0.014) compared to *H. pylori*-negative children. **Conclusion:** *H. pylori* infection is associated with hematological alterations, including lower hemoglobin levels, reduced platelet indices, and altered red cell indices, suggesting its potential role in anemia and nutritional deficiencies.

## INTRODUCTION

Helicobacter pylori is a widespread chronic infection globally. Its prevalence varies by geographic region, often persisting due to reinfection and inadequate eradication efforts. Socioeconomic factors significantly contribute to its transmission [(1)]. Beyond its well-established role in gastrointestinal disorders, *H. pylori* has been linked to conditions such as inflammatory bowel disease, gastroesophageal reflux disease, non-alcoholic fatty liver disease, and even hematological and metabolic disorders [(2)]. Infected children exhibit higher rates of anemia and stunted growth than their uninfected counterparts [(3)].

Evidence suggests a relationship between *H. pylori* and idiopathic thrombocytopenia (ITP). Although the exact mechanism remains unclear, one hypothesis involves the cytotoxin-associated gene A (CagA), which may trigger the production of cross-reactive antibodies that affect platelet function [(5)]. While studies in adults from high-income countries suggest a link, there is limited data on pediatric populations in low-income regions where *H. pylori* prevalence is high [(6)].

This study evaluates the effect of *H. pylori* infection on platelets in children presenting to Aswan University Hospital.

## PATIENTS AND METHODS

This observational cross-sectional study included children aged 6-15 who were presented with dyspepsia at the gastroenterology outpatient clinic of Aswan University Hospital. A stool antigen test confirmed an H. pylori infection. Children with chronic liver disease, obesity, metabolic syndrome, or diabetes mellitus were excluded.

Comprehensive data collection included medical history, demographic details, anthropometric measurements, and systemic examination. Laboratory investigations comprised of complete blood count (CBC), fasting plasma glucose, glycosylated hemoglobin (HbA1c), and *H. pylori* stool antigen testing.

To evaluate the H. pylori antigen, approximately 1 g of stool from each child was collected in a 5 ml sample diluent using an Enzyme Immunoassay Test (ELISA) technique kit (Eagle Biosciences, Inc., Amherst, NH, USA) [(7)].

**Statistical Analysis:**

We used IBM SPSS version 27 to analyze the data. The Shapiro-Wilk and Kolmogorov-Smirnov tests were used to determine normalcy. Numerical variables were presented as means and standard deviations, whereas categorical variables were displayed as frequencies and percentages. Depending on the situation, chi-square, Fisher's exact, independent t-tests, or Mann-Whitney tests were used to compare the groups. Statistical significance was defined as a p-value <0.05.

**Ethical Approval:** The Institutional Ethics and Research Review Board of Aswan University approved the study. Parents gave written informed consent before enrollment, adhering to the Declaration of Helsinki guidelines.

## RESULTS

This study included 195 children with a mean age of 10.5 ± 2.57 years; 43.6% were male, and 50.3% resided in urban areas (table 1).

**Table (1): Patients' demographic characteristics (n=195)**

| Variables | | Frequency (Percentage %) | |
|---|---|---|---|
| **Gender** | Male | 85 (43.6%) | |
| | Female | 110 (56.4%) | |
| **Age (years)** | 6 - 9.50 | 105 (53.8%) | |
| | 10-15 | 90 (46.2%) | |
| | **Mean ± SD** | 10.5 ± 2.5697 | |
| **Residence** | Urban | 98 (50.3%) | |
| | Rural | 97 (49.8%) | |
| | | **Mean ± SD** | **Median (range)** |
| **Weight (kg)** | | 24 ± 7.8898 | 23 (14.50-54) |
| **Height (cm)** | | 119.1 ± 15.328 | 119 (92-160) |

| | | |
|---|---|---|
| **Body mass index (Kg/m$^2$)** | 16.35 ± 1.8 | 16.1 (12.30-21.50) |
| **H. Pylori + ve cases** | 102 (52.3%) | |

Table 2 shows children with *H. pylori* infection had significantly lower median Height-for-Age Z-scores (p<0.001) and higher rates of stunting (25.4% vs. 6.5%, p=0.013). Additionally, *H. pylori*-positive children had lower BMI-for-Age Z-scores, with a higher prevalence of wasting (27.45% vs. 4.3%, p<0.001). No significant difference in Weight-for-Age Z-scores was observed (p>0.05).

**Table (2): Impact of H. pylori infection on nutritional status**

| **Parameters** | | **H. Pylori status** | | **P value** |
|---|---|---|---|---|
| | | Positive (n=102) | Negative (n=93) | |
| | | N (%) | N (%) | |
| **Height-for-age Z-score (HAZ)** | Normal | 76 (74.6%) | 87 (93.5%) | **0.013*** |
| | Stunted | 26 (25.4%) | 6 (6.5%) | |
| | **Median (IQR)** | -1.445 (-1.975-0.8775) | -2.23 (-2.69-(-1.78)) | **<0.001**** |
| **Weight-for-age Z-score (WAZ)** | Normal | 53 (52%) | 41 (44.1 %) | 0.470 |
| | Underweight | 49 (48%) | 52 (55.9 %) | |
| | **Median (IQR)** | -0.945 (-1.84- (-0.3875)) | -1.030 (-1.410- (-0.68) | 0.967 |
| **BMI for age Z-score** | Normal | 72 (70.59 %) | 68 (73.1 %) | **<0.001*** |
| | Wasted | 28 (27.45%) | 4 (4.3 %) | |
| | Overweight risk | 2 (1.96 %) | 21 (22.6 %) | |
| | **Median (IQR)** | 0.285 (-0.965-0.46) | 0.60 (0.16-1.06) | **<0.001**** |
| **Weight for height Z-score (WHZ)** | **Median (IQR)** | 0.325 (-0.7775-0.7125) | 0.750 (0.32-1.30) | **0.005**** |

Table 3 shows laboratory findings indicated significant differences between *H. pylori*-positive and negative children in terms of hemoglobin levels (p=0.015), MCV (p<0.001), MCH (p=0.004), leucopenia (p=0.018), and platelet counts (p=0.004). Mean platelet volume (MPV) was also significantly lower in *H. pylori*-positive children (p=0.014).

**Table (3): Laboratory findings of study children (n=195)**

| **Parameters** | **Mean ± SD** | **Median (range)** |
|---|---|---|
| **HBA1c (%)** | 5.001 ± 0.2946 | 5 (4.50-5.70) |
| **Complete blood count (CBC)** | | |
| **WBCs (×10$^3$/ µL)** | 6.938 ± 2.2231 | 7 (2-12.7) |
| **Hemoglobin (g/ dL)** | 11.281 ± 1.0853 | 11.50 (9-13.50) |
| **MCV (FL)** | 75.624 ± 9.8873 | 77 (8.2-90) |
| **MCH (pg.)** | 27.814 ± 2.7736 | 28 (20.1-34) |
| **Platelets (×10$^3$/ µL)** | 209.04 ± 52.849 | 199 (88-420) |
| **MPV (FL)** | 8.141 ± 0.7017 | 8 (7-10.6) |

Table 4 reveals a statistically significant correlation of H. pylori infection with MCV and MCH (p = 0.004), WBC (p = 0.004). Leucopenia was found in 27.1% of H. pylori-positive patients and 8.6% of H. pylori-negative patients. Besides, platelet count was highly reduced in H. pylori-positive patients compared to H. pylori-negative patients. Finally, a significant correlation was determined for H. pylori infection and mean platelet volume (MPV), being low in 20.59% of the H. pylori infections versus 2.15% of the H. pylori negativities (p = 0.014).

**Table (4): Association between H. Pylori infection and hematological parameters**

| Parameters | | H. Pylori status | | P value |
|---|---|---|---|---|
| | | Positive (n=102) | Negative (n=93) | |
| | | N (%) | N (%) | |
| **Hemoglobin (gm/dL)** | Anemia (<11.5) | 59 (57.8%) | 35 (37.6%) | 0.051 |
| | Normal (≥11.5) | 43 (42.2%) | 58 (62.4%) | |
| | **Mean ± SD** | 11.015 ± 1.109 | 11.553 ± 1.00 | **0.015**** |
| **MCV (FL)** | Low (≤75 FL) | 64 (62.7%) | 24 (25.8%) | **<0.001*** |
| | Normal (> 75 FL) | 38 (37.2%) | 69 (74.2%) | |
| | **Mean ± SD** | 73.44 ± 6.9896 | 77.855 ± 11.823 | **0.029**** |
| **MCH (Pg.)** | Low (<25) | 21 (20.6 %) | 2 (2.15%) | **0.004*** |
| | Normal (>25) | 81 (79.4%) | 91 (97.85 %) | |
| | **Mean ± SD** | 27.423 ± 3.302 | 28.213 ± 2.0635 | 0.165 |
| **WBCs (×$10^3$/µL)** | Leucopenia | 13 (27.1%) | 8 (8.6%) | **0.018*** |
| | Normal | 35 (72.9%) | 85 (91.4%) | |
| | **Mean ± SD** | 6.571 ± 2.4003 | 7.313 ± 1.9822 | 0.104 |
| **Platelets (×$10^3$/µL)** | Thrombocytopenia | 13 (12.7 %) | 2 (2.15 %) | 0.053 |
| | Normal count | 89 (87.3 %) | 91 (97.85 %) | |
| | **Mean ± SD** | 193.77 ± 42.22 | 224.64 ± 58.258 | **0.004**** |
| **MPV (FL)** | Low (<7.5) | 21 (20.59 %) | 2 (2.15 %) | **0.014*** |
| | Normal (7.5-10) | 79 (77.45%) | 89 (95.7 %) | |
| | High (>10) | 2 (1.96 %) | 2 (2.15%) | |
| | **Mean ± SD** | 7.84 ± 0.6153 | 8.449 ± 0.654 | **<0.001**** |

## DISCUSSION

Platelets play a crucial role in hemostasis and immune regulation. Previous studies indicate that *H. pylori* affect platelet count and function, potentially contributing to hematological abnormalities. In chronic ITP patients, platelet counts often improve following *H. pylori* eradication [(8)].

In this study, *H. pylori*-infected children exhibited significantly lower hemoglobin levels, platelet counts, and MPV. These findings align with prior research indicating an association between *H. pylori* and anemia, due to chronic gastritis-related blood loss and impaired iron absorption. The bacterium reduces gastric acid secretion, which may promote enteropathogen colonization, diarrhea, malabsorption, and subsequent iron deficiency anemia [(9)].

Several studies support the relationship between *H. pylori* and hematological alterations. **Bille et al.** [(10)] reported a high prevalence of microcytic hypochromic anemia in infected children. Similarly, studies from Ethiopia, Sudan [(11)], and Pakistan [(12)] found lower platelet counts among *H. pylori*-infected

individuals. However, research from the Netherlands [13] did not identify significant differences, due to variations in study populations and methodologies.

Elevated MPV levels in some *H. pylori*-infected individuals may indicate a compensatory response to platelet destruction. Increased MPV reflects young platelet production, a common response to systemic inflammation. This variability underscores the complexity of *H. pylori*-related hematological effects and the need for further investigation [14].

In the current research, H. pylori-positive cases also presented significantly lower median Height-for-Age Z-scores, BMI-for-age Z-score, and a higher prevalence of wasting ($p < 0.001$). The median Weight-for-Height Z-score was significantly lower among H. pylori-positive cases ($p = 0.005$). No significant difference between the groups was noted for Weight-for-Age Z-scores ($p > 0.05$). Early childhood growth measures have been correlated with H. pylori. There is a great heterogeneity in the findings of research looking at the impact of H. pylori infection on a child's growth. H. pylori infection has been correlated in numerous studies with a decrease in the growth of children [15, 16, 17]. Cross-sectional studies from several countries, however, show no correlation [18].

This may be explained by the fact that H. pylori decrease the production of gastric acid, which can result in an enteropathogenic infection that can cause diarrhea, nutrient malabsorption, decreased food intake because of dyspepsia, and iron-deficiency anemia [19]. However, with so many confounding variables, such as diet and socioeconomic status, it is difficult to prove that H. pylori alone cause growth impairment in children.

## CONCLUSION

This study highlights a significant association between *H. pylori* infection and reduced platelet indices in children, supporting a potential role in anemia and altered hematological profiles. Further longitudinal research is warranted to establish causality and explore the underlying mechanisms.

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