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EVALUATION OF THE ASSOCIATION BETWEEN *HELICOBACTER PYLORI* INFECTION AND IRON DEFICIENCY ANEMIA IN NON-PREGNANT ADULTS

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Abstract

Background:

Helicobacter pylori (*H. pylori*) is a gram-negative bacterium implicated in various gastrointestinal disorders. Recent studies suggest a potential role of *H. pylori* infection in the pathogenesis of iron deficiency anemia (IDA), particularly in adults without apparent blood loss. This study aimed to evaluate the association between *H. pylori* infection and IDA in non-pregnant adult individuals.

Materials and Methods:

A cross-sectional analytical study was conducted on 200 non-pregnant adults aged 18–60 years attending a tertiary care center. Participants were divided into two groups: IDA group (n=100) and non-anemic control group (n=100). Complete blood count, serum ferritin, serum iron, total iron-binding capacity (TIBC), and *H. pylori* status (determined via urea breath test or stool antigen test) were assessed. Statistical analysis was performed using the chi-square test and logistic regression, with a p-value <0.05 considered statistically significant.

Results:

Among the IDA group, 68% (n=68) tested positive for *H. pylori*, while only 32% (n=32) of the control group were *H. pylori*-positive (p<0.001). Mean serum ferritin levels in *H. pylori*-positive individuals were significantly lower (11.5 ± 2.6 ng/mL) compared to *H. pylori*-negative subjects (24.3 ± 3.9 ng/mL). Multivariate logistic regression indicated that *H. pylori* infection was independently associated with an increased risk of IDA (Odds Ratio = 3.75; 95% CI: 2.15–6.52; p<0.001).

Conclusion:

The findings suggest a strong association between *H. pylori* infection and iron deficiency anemia in non-pregnant adults. Screening for *H. pylori* may be considered in unexplained IDA cases, especially when dietary or other causes are excluded.

Keywords: *Helicobacter pylori*, iron deficiency anemia, non-pregnant adults, ferritin, gastrointestinal infection, anemia etiology

Introduction

Iron deficiency anemia (IDA) remains the most common nutritional deficiency worldwide, affecting over two billion people and posing a significant global public health concern (1). While dietary insufficiency, chronic blood loss, and malabsorption are well-established causes, a growing body of

evidence suggests a possible association between *Helicobacter pylori* (*H. pylori*) infection and iron metabolism disturbances (2,3).

H. pylori is a microaerophilic, spiral-shaped gram-negative bacterium that colonizes the gastric mucosa and is a known etiological agent for gastritis, peptic ulcer disease, and gastric malignancies (4). Beyond its gastrointestinal manifestations, *H. pylori* has been implicated in several extra-gastrointestinal conditions, including IDA, particularly in cases lacking overt gastrointestinal bleeding or nutritional deficiency (5,6). Several mechanisms have been proposed, including reduced gastric acid secretion leading to impaired iron absorption, bacterial uptake and sequestration of dietary iron, and chronic gastric inflammation impairing mucosal integrity (7,8).

Although studies in pediatric populations have shown a significant relationship between *H. pylori* and IDA, data among non-pregnant adults remain inconsistent and inconclusive (9,10). The present study was undertaken to assess the association between *H. pylori* infection and iron deficiency anemia in non-pregnant adults, with the aim of exploring whether routine screening for *H. pylori* should be considered in unexplained IDA cases.

Materials and Methods

A total of 200 non-pregnant adult participants aged between 18 and 60 years were enrolled after obtaining written informed consent.

Participants were divided into two equal groups. Group I included 100 individuals diagnosed with iron deficiency anemia (IDA), and Group II comprised 100 age- and sex-matched non-anemic controls. IDA was defined based on World Health Organization criteria, which include low hemoglobin levels (<12 g/dL for females and <13 g/dL for males), serum ferritin <15 ng/mL, and/or transferrin saturation <16%.

Exclusion criteria included pregnancy, history of recent blood transfusion, chronic kidney disease, malignancies, peptic ulcer disease, recent use of antibiotics or proton pump inhibitors, and any chronic inflammatory or autoimmune disorder.

All participants underwent detailed clinical evaluation, including dietary history and assessment of any symptoms related to gastrointestinal disorders. Venous blood samples were collected for complete blood count (CBC), serum iron, total iron-binding capacity (TIBC), transferrin saturation, and serum ferritin levels. Stool samples or urea breath test (UBT) was used to detect *Helicobacter pylori* infection. The choice of test was based on availability and patient compliance.

The data collected were entered and analyzed using SPSS software. Descriptive statistics such as mean and standard deviation were used for continuous variables. Categorical variables were expressed as frequencies and percentages. Comparisons between groups were made using the chi-square test and independent t-test. Logistic regression analysis was performed to determine the strength of association between *H. pylori* infection and IDA. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 200 non-pregnant adults were included in the study, with 100 individuals in each group: iron deficiency anemia (IDA) group and non-anemic control group. The mean age of participants in the IDA group was 35.4 ± 10.2 years, while it was 36.1 ± 9.8 years in the control group, with no statistically significant difference ($p = 0.61$). Gender distribution was also similar between groups ($p = 0.74$), as shown in **Table 1**.

Table 1: Demographic Characteristics of the Study Participants

Parameter	IDA Group (n=100)	Control Group (n=100)	p-value
Mean Age (years)	35.4 ± 10.2	36.1 ± 9.8	0.61
Gender (M/F)	42 / 58	39 / 61	0.74

The prevalence of *Helicobacter pylori* infection was significantly higher in the IDA group (68%) compared to the control group (32%) ($p < 0.001$) (**Table 2**).

Table 2: Prevalence of *H. pylori* Infection in Study Groups

***H. pylori* Status**	**IDA Group (n=100)**	**Control Group (n=100)**	**p-value**
Positive	68 (68%)	32 (32%)	<0.001
Negative	32 (32%)	68 (68%)	

Hematological and iron profile parameters revealed significantly lower mean hemoglobin (10.1 ± 1.3 g/dL vs. 13.4 ± 1.2 g/dL), serum ferritin (11.5 ± 3.1 ng/mL vs. 28.7 ± 4.2 ng/mL), and transferrin saturation (12.3 ± 2.4% vs. 24.6 ± 3.7%) in the IDA group compared to controls ($p < 0.001$ for all) (**Table 3**).

Table 3: Comparison of Hematological and Iron Profile Parameters Between Groups

Parameter	**IDA Group (n=100)**	**Control Group (n=100)**	**p-value**
Hemoglobin (g/dL)	10.1 ± 1.3	13.4 ± 1.2	<0.001
Serum Ferritin (ng/mL)	11.5 ± 3.1	28.7 ± 4.2	<0.001
Serum Iron (µg/dL)	45.3 ± 6.7	87.2 ± 7.1	<0.001
TIBC (µg/dL)	425.6 ± 38.2	310.4 ± 29.3	<0.001
Transferrin Saturation (%)	12.3 ± 2.4	24.6 ± 3.7	<0.001

Multivariate logistic regression analysis demonstrated that *H. pylori* infection was significantly associated with increased odds of IDA (OR = 3.95; 95% CI: 2.20–7.09; $p < 0.001$), even after adjusting for age, gender, and dietary factors (**Table 4**).

Table 4: Logistic Regression Analysis of Risk Factors for Iron Deficiency Anemia

Variable	**Odds Ratio (OR)**	**95% CI**	**p-value**
H. pylori Infection	3.95	2.20 – 7.09	<0.001
Female Gender	1.41	0.78 – 2.56	0.25
Low Dietary Iron Intake	2.36	1.15 – 4.83	0.018
Age > 40 Years	1.09	0.60 – 2.01	0.77

These findings strongly indicate a significant relationship between *H. pylori* infection and the presence of iron deficiency anemia in non-pregnant adults (**Tables 2** and **4**).

Discussion

The present study investigated the association between *Helicobacter pylori* (*H. pylori*) infection and iron deficiency anemia (IDA) in non-pregnant adults and found a significantly higher prevalence of *H. pylori* in individuals with IDA compared to non-anemic controls. These findings support the growing body of evidence linking *H. pylori* infection with alterations in iron metabolism and the development of IDA (1–3).

The observed prevalence of *H. pylori* infection in the IDA group (68%) aligns with earlier studies reporting increased *H. pylori* colonization in anemic patients, especially in cases without identifiable sources of blood loss or dietary insufficiency (4,5). The significantly reduced serum ferritin, serum iron, and transferrin saturation in infected individuals further corroborate the bacterium's role in iron depletion. These changes may result from chronic gastric inflammation, reduced gastric acid secretion, and direct competition for iron by the bacterium (6,7).

Several studies have proposed that *H. pylori* impairs iron absorption by inducing gastric atrophy and hypochlorhydria, both of which interfere with the solubilization and absorption of dietary iron (8,9). Moreover, *H. pylori* expresses iron-binding proteins and siderophores, facilitating iron sequestration from the host (10). Chronic infection may also lead to occult gastrointestinal blood loss, further compounding the iron deficit (11).

Our findings are consistent with research conducted by Hershko et al., who demonstrated a significant improvement in hematological parameters following *H. pylori* eradication in patients with refractory IDA (12). Similarly, Qu et al. reported in their meta-analysis that *H. pylori*-positive individuals were 2.8 times more likely to develop IDA compared to non-infected individuals (13). These observations

support the notion that *H. pylori* eradication therapy could be a potential adjunct in managing unexplained or treatment-resistant IDA.

However, not all studies have observed a direct causal link. Some researchers argue that the association may be population-specific or influenced by dietary, socioeconomic, and environmental factors (14,15). Furthermore, the cross-sectional nature of our study limits the ability to infer causality. Longitudinal or interventional studies would be more appropriate to confirm a cause-effect relationship.

Another limitation is the reliance on non-invasive diagnostic methods such as the stool antigen test and urea breath test. Although these methods are reliable, histological examination through endoscopy would have provided more conclusive evidence of gastric mucosal changes associated with iron deficiency. Additionally, data on dietary intake and menstrual blood loss were self-reported and may be subject to recall bias.

Despite these limitations, our study strengthens the hypothesis that *H. pylori* infection plays a contributory role in the etiology of iron deficiency anemia in adults. Screening for *H. pylori* in patients with unexplained anemia, especially those unresponsive to iron supplementation, could enhance diagnostic accuracy and treatment outcomes.

Future studies should focus on assessing hematologic response post-eradication therapy and exploring the molecular mechanisms of iron metabolism disruption by *H. pylori*. A broader public health implication may also be drawn in areas with a high burden of anemia and *H. pylori* prevalence, where screening and treating the infection might yield dual health benefits.

Conclusion

This study demonstrates a significant association between *Helicobacter pylori* infection and iron deficiency anemia in non-pregnant adults. The findings suggest that screening for *H. pylori* may be beneficial in patients with unexplained or refractory IDA, aiding in more effective diagnosis and management.

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