Biomedical & Pharmacology Journal, June 2026. Vol. **19**(2), p. 1853-1864

Prevalence of *Helicobacter pylori* Infection in a sample of Egyptian Children with Refractory Iron Deficiency Anemia

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https://dx.doi.org/10.13005/bpj/3462

(Received: 27 January 2026; accepted: 04 May 2026)

***Helicobacter pylori* (*H. pylori*) infection has been implicated in refractory iron deficiency anemia (IDA) in children, raising concern due to its potential impact on growth and development. We aimed to determine the occurrence of H. pylori infection in children with IDA, its association with refractoriness to oral iron therapy, and its impact on growth.100 children participated in this cross-sectional study aged 4–12 years with IDA who failed to respond to 6 weeks of therapeutic iron supplementation. Participants, recruited from El Galaa Teaching Hospital, underwent full clinical assessment, anthropometry, and laboratory evaluation for IDA indices, *H. pylori* infection, liver and kidney functions. *H. pylori* infection was detected in 77% of patients. Children who were infected had much lower BMI Z-scores, hematocrit ($P = 0.001$), hemoglobin ($P < 0.001$), serum iron ($P < 0.001$), and ferritin ($P = 0.003$), and significantly higher TIBC ($P < 0.001$) than uninfected children. *H. pylori* infection was negatively correlated with BMI Z-score, Hb, Ht, MCV, serum iron, and ferritin levels ($P < 0.05$), and positively correlated with TIBC ($P < 0.001$). No significant differences were observed in height-for-age or weight-for-age Z-scores. *H. pylori* infection was strongly associated with refractory IDA and should be investigated in all affected cases. Although no association with linear growth was identified, *H. pylori* infection was negatively associated with nutritional status, as reflected by lower BMI Z-scores.**

Keywords: Growth impairment; *Helicobacter pylori*; Interleukin- 6; Iron deficiency anemia; Refractory iron deficiency anemia.

Reduced red blood cell count, hematocrit or hemoglobin concentration that is more than two standard deviations below the mean for age and sex is referred to as anemia. [1] The most prevalent form of nutritional anemia is iron deficiency anemia (IDA), The pathogenesis of iron deficiency anemia (IDA) results from insufficient iron intake, poor bioavailability, or depleted iron stores, which ultimately lead to inadequate hemoglobin synthesis. These deficiencies contribute to various adverse

health outcomes, including immune dysfunction, impaired growth, and cognitive deficits. [2]

The World Health Organization (WHO) estimates that anemia affects about 25% of the global population about half of the cases caused by iron deficiency anemia (IDA).[3] IDA is particularly common among high-risk groups such as infants, young children, and women of reproductive age.[4]

The burden is greater in developing countries, where prevalence among preschool children reaches about 39%, compared to 20% in developed regions. This problem persists into later childhood, with high rates reported among school-aged children.[5]

Helicobacter pylori (H. pylori) is a spiral-shaped bacterium that colonizes the human stomach lining. It was first isolated in 1982 by Barry Marshall and Robin Warren.[6] The prevalence of Helicobacter pylori infection in children variable across different countries. In Egypt, a population-based cross-sectional study conducted among asymptomatic schoolchildren reported an overall prevalence of 72.38%. The main risk factors identified were living in overcrowded households and in socially deprived areas. [7]

Most infected children are asymptomatic, but other H. pylori infected children complaining of many gastrointestinal manifestations such as gastric dyspepsia, nausea and/or vomiting, recurrent unexplained abdominal pain.[8] In addition to its well-known gastrointestinal effects, H. pylori infection has been associated with a wide range of extra-gastric diseases. These include lymphoma, cardiovascular and dermatological diseases, liver and gallbladder disorders, Iron deficiency anemia, diabetes mellitus, autoimmune diseases, atopy, asthma, neurological and bone disorders, micronutrient deficiencies, growth retardation, and idiopathic thrombocytopenic purpura.[9]

These associations are thought to arise from systemic effects of the infection mediated through hormonal, immunological, and inflammatory pathways, including the release of cytokines and chemokines that extend beyond the gastrointestinal system.[10]

Several studies have suggested a link between IDA and infection with H. pylori. This association may be explained by the development of chronic gastritis, which can lead to increased iron loss due to hemorrhagic gastritis, atrophic changes, or even Gastric Cancer. In addition, reduced gastric acidity caused by the infection can impair iron absorption, further contributing to iron deficiency, as well as improvements in unexplained IDA have been observed following successful eradication of the bacterium in both pediatric and adult populations.[11,12]

The current work aimed to investigate the occurrence of *H. pylori* infection and its relation to IDA refractoriness to treatment in a sample of Egyptian children. The present study also assessed the probable relationships among growth retardation, malnutrition and *H. pylori* infection in children.

MATERIALS AND METHODS

Calculation of sample size

Using the PASS 11th release, set the power = 0.80 and á = 0.05 and based on preceding studies, a sample size of 100 cases was included in this study. [13]

Participants and Study design

This study (cross-sectional) was carried out from July 2024 to September 2024 and included 100 children diagnosed with IDA who received iron therapy at the proper therapeutic dose for 6 weeks without any response to oral iron therapy. They were selected from patients followed in the Pediatric Outpatient Clinic of El Galaa Teaching Hospital.

Inclusion criteria

1. Age: 4–12 years.
2. Sex: both sexes.
3. Patients who were diagnosed with IDA and received regular proper therapeutic oral iron therapy for 6 weeks without any improvement in hemoglobin level.
4. The absence of intestinal parasites, including hookworms, was confirmed via examination of three consecutive samples of stool.

Exclusion criteria

1. Patients with renal impairment, chronic liver disease or malabsorption.
2. Patients receiving blood transfusion or intake of anticoagulants, salicylates, steroid therapy or non-steroidal anti-inflammatory (NSAID) drugs.
3. Patients with apparent loss of blood (melena, hematochezia, hematuria, repeated epistaxis or hemoptysis).

4. Patients with conditions that cause anemia including hematological disorders, malignancy, and connective tissue disease.
5. Pubertal female patients who had menstruated, had irregular menstruation or prolonged or heavy menstrual periods of > 5 days with a large quantity of bleeding (changes > 2 napkins/day).
6. Patients consumed antibiotics or proton-pump inhibitors during the last two weeks prior to joining the study.

Every patient was exposed to the following

Complete history taking including:

1- The demographic data included residence, age and sex.
2- Family history: A History of *H. pylori* infection and IDA, GIT ulcers and chronic gastrointestinal diseases (e.g. ulcerative Colitis). Crohn's disease and inflammatory bowel disease occur in family members.
3- Past history: Parasitic infection, recurrent post-prandial abdominal pain (location, frequency, duration, character, severity, bleeding, relieving and aggravating factors), drugs, previous diagnostic testing and specific therapy for gastrointestinal diseases, bowel habits, history of nausea or vomiting, appetite nature and any changes in weight.

Clinical examination

General examination including the following:

1- General appearance: lips, skin, and conjunctiva for pallor.
2- Vital signs: pulse, temperature and blood pressure.
3- Anthropometric measurements:

Anthropometric evaluation was performed, including height and weight, which were measured via standardized equipment. The body mass index (BMI) is calculated by dividing height (in square meters) by weight (in kilograms). The Z scores (standard deviation scores) of weight, height and BMI were calculated via the WHO ANTHRO Plus software. [14]

Systemic examination including: the chest, heart, abdomen, etc.

Laboratory Investigations:

A DYNACOUNT 3D fully automatic hematology analyzer was used to produce a complete blood count (CBC).

A Hitachi 704 auto-analyzer and colorimetric enzymatic methods were used to measure serum creatinine, urea, AST, and ALT (Roche Diagnostics, Switzerland).

Iron profile

Iron in the serum was determined via atomic absorption spectrophotometer (Perkin Elmer, Singapore).

Total iron binding capacity (TIBC) was assessed using an enzyme-linked immunosorbent assay (ELISA) (R&D Systems, Minneapolis, MN, USA).

To assess ferritin, the enzyme-linked immunosorbent assay (ELISA) was used (R&D Systems, Minneapolis, MN, USA).

Guaiac fecal occult blood test: This type, sometimes called gFOBT, uses cards to test samples from multiple bowel movements.

Stool sample for *H. pylori* antigen: The test was performed on both fresh and frozen samples of stool. When the test could not be performed for one day, the sample was stored at –20°C.

A commercially available enzyme-linked immunosorbent assay (ELISA) from Pishtaz Teb Diagnostics was used to screen for *H. pylori* antigen in feces. The manufacturer's instructions were followed when doing ELISA.

Ethical approval

The study was conducted in accordance with the Helsinki Declaration and was authorized by the General Organization of Teaching Hospital and Institutes' ethical committee (No. HG000103). All patients or their legal guardians gave their informed permission.

Statistical analysis

By using IBM SPSS version 27, the data was analyzed using the Statistical Package used for comprehensive statistical analysis, data management, and predictive modeling in the social sciences. For normally distributed data (Gaussian (normal) distribution), the quantitative data are shown as means ± SDs, ranges, and parametric tests; for non-normally distributed data, they are shown as medians and interquartile ranges (IQRs) with non-parametric tests. Qualitative attributes are presented using percentages and numbers. For qualitative data, the Fisher's exact test and/or chi-square test were used to compare two groups; for quantitative data, the Mann-Whitney test was used as a non-parametric test and an independent t test was used as a parametric test. Correlations were

determined via the Spearman correlation test. The p value < 0.05 was considered significant and the confidence interval was set to 95%,.

RESULTS

One hundred children who were diagnosed with IDA and received regular proper therapeutic oral iron therapy for 6 weeks without any improvement in the hemoglobin levels were involved in this work.

The mean age of the studied patients was 7.57 ± 2.47 (4.6–12) y. Among them 54 were females (54%), while 46 were males (46%).

Regarding the growth assessment of all the studied cases, the median and IQR of the weight Z-scores of our cases was 0.95 (-1.1–1.2), and that of the height Z-scores was 0.95 (0.1 – 1.6), while the nutritional assessment revealed that the mean ± SD of the BMI Z-scores was (- 0.48 ± 0.81), with a range of (- 2.67 – 1.20). Table (1).

With respect to the Clinical features of the total studied patients, pallor with observed in 32% of patients without any family history to *H. pylori* infection and IDA or Family history of chronic GIT disease such as Crohn's disease and inflammatory bowel disease. (Table 2).

With respect to the laboratory findings of the total studied patients, the findings of CBC and iron studies, including low serum ferritin and serum iron and high total iron binding capacity, indicated the persistence of IDA. Liver and renal function tests were within normal values. *Helicobacter pylori* antigen was positive in 77 % of the patients. (Table 3).

Patients were divided into two groups based on whether or not they had the H. pylori antigen, and they were then compared. There was no discernible difference in age between patients with and without H. pylori infection (p > 0.05). However, a comparison between the two groups according to sex distribution revealed a significant difference (p < 0.001), where the majority of patients with *H. pylori* infection were females (70.1%). Females are far more likely than males to develop H. pylori infection, suggesting a clear correlation between sex and the infection. In terms of the physical growth of *H. pylori* infected

Table 1. Anthropometric characteristics and demographics of all the patients studied

Variable		Patients (n =100)
Age (years)	Mean ± SDRange	7.57 ± 2.47 4.6 – 12
Sex	Females (n, %)Males (n, %)	54 (54%) 46 (46%)
Weight (Z-score)	Median (IQR)Range	0.95 (-1.1 – 1.2) -1.5 – 1.7
Height (Z-score)	Median (IQR)Range	0.95 (0.1 – 1.6) -2 – 2.3
BMI (Z-score)	Mean ± SDRange	- 0.48 ± 0.81 - 2.67 – 1.20

SD: standard deviation, IQR: interquartile range, BMI: body mass index.

Table 2. Clinical features of all the patients studied

Variables	Value
General appearance (n, %)	
Pallor	32 (32%)
Jaundice	0 (100.0%)
Family history similar condition (n, %)	0 (100.0%)
Family history of chronic GIT disease	0 (100.0%)

children, the mean weight and height Z-scores did not differ significantly from those of *H. pylori* non-infected patients (p = 0.954 & 0.698, respectively). However, regarding the nutritional status of our patients, a comparison of *H. pylori*-positive and *H. pylori*-negative patients' BMI Z scores revealed that there was a significant difference in the infected group (p < 0.001). (Table 4).

Means ± SDs were compared using the student's t test; medians (IQRs) were compared using the Kruskal-Wallis test; numbers (%) were compared using the Chi-square test; p < 0.05 =

Table 3. Laboratory results of all the patients studied

Parameter		Patients (n =100)
Hemoglobin (g/dL)	Mean ± SD	8.23 ± 1.37
	Range	5.8 – 10.4
Hematocrit (%)	Mean ± SD	25.12 ± 2.56
	Range	18 – 28
Mean corpuscular volume (fl)	Mean ± SD	66.92 ± 5.95
	Range	49 – 73
Red cell count ($x10^6/cm^3$)	Mean ± SD	3.91 ± 0.87
	Range	2.4 – 5.4
Serum iron (mg/dL)	Mean ± SD	34.3 ± 11.24
	Range	18 – 46
Total iron binding capacity (TIBC) (ig/dL)	Mean ± SD	418.69 ± 67.01
	Range	301 – 504
Serum ferritin (ng/mL)	Mean ± SD	14.92 ± 5.77
	Range	4 – 25
AST (IU/L)	Mean ± SD	29.92 ± 4.76
	Range	23 – 44
ALT (IU/L)	Mean ± SD	35.36 ± 3.81
	Range	28 – 42
Urea (mg/dl)	Mean ± SD	27.38 ± 7.13
	Range	18 – 43
Cr (mg/dl)	Mean ± SD	0.7 ± 0.16
	Range	0.5 – 1.1
H. pylori stool antigen	Positive	77 (77%)
	Negative	23 (23%)

Table 4. Comparing patients with and without *H. pylori* infection based on anthropometric and demographic information

Variable		*H. pylori* stool antigen -ve N = 23	*H. pylori* stool antigen +ve N = 77	P-value
Age (years)	Mean ± SD	7.47 ± 2.88	7.61 ± 2.36	0.819
	Range	4.6 – 12	4.6 – 12	
Sex	Females	0 (0%)	54 (70.1%)	< 0.001
	Males	23 (100%)	23 (29.9%)	
Weight (Z-score)	Median (IQR)	-0.9(-1.2 – 1.4)	1(-1.1 – 1.1)	0.954
	Range	-1.2 – 1.4	-1.5 – 1.7	
Height (Z-score)	Median (IQR)	0.9(-0.5 – 1.6)	1(0.1 – 1.2)	0.698
	Range	-0.5 – 2.3	-2 – 2.3	
BMI	Mean ± SD	0.45 ± 0.54	- 0.75 ± 0.66	< 0.001
Z scores	Range	-1.7	-2.67 – 0.73	

significant; IQR stands for interquartile range; and BMI stands for body mass index.

With respect to laboratory markers, there were significant differences between *H. pylori* infected patients and non-infected patients in terms of hemoglobin levels, hematocrit values, MCVs, serum iron, TIBC, and ferritin serum concentrations ($p < 0.05$). Moreover, no significant differences were detected in red cell count or serum alanine transaminase, aspartate transaminase, urea or creatinine levels ($p > 0.05$) (Table 5).

Table 5. Comparison of laboratory findings in patients with and without H. pylori infection

Variable		*H. pylori* stool antigen Negative N = 23	Positive N = 77	P
Hb	Mean ± SD	9.68 ± 0.68	7.8 ± 1.22	<0.001
	Range	8.9 – 10.4	5.8 – 9.3	
Ht	Mean ± SD	26.7 ± 1.22	24.65 ± 2.67	0.001
	Range	25 – 28	18 – 28	
MCV	Mean ± SD	71.17 ± 1.67	65.65 ± 6.18	<0.001
	Range	69 – 73	49 – 73	
RBCs	Mean ± SD	3.67 ± 0.82	3.98 ± 0.88	0.145
	Range	2.4 – 5.1	2.6 – 5.4	
Serum iron	Mean ± SD	44.78 ± 1.28	31.17 ± 11.00	<0.001
	Range	43 – 46	18 – 46	
TIBC	Mean ± SD	366 ± 60.6	434.43±60.78	<0.001
	Range	301 – 467	301 – 504	
Serum Ferritin	Mean ± SD	18 ± 3.91	14 ± 5.94	0.003
	Range	14 – 25	4 – 25	
AST	Mean ± SD	29.43 ± 3.6	30.06 ± 5.07	0.58
	Range	23 – 33	23 – 44	
ALT	Mean ± SD	34.78 ± 3.92	35.53 ± 3.79	0.411
	Range	28 – 42	28 – 42	
Urea	Mean ± SD	28.09 ± 8.02	27.17 ± 6.89	0.591
	Range	18 – 43	18 – 43	
Cr	Mean ± SD	0.76 ± 0.17	0.69 ± 0.16	0.069
	Range	0.5 – 1.1	0.5 – 1.1	

Student's t test, $p < 0.05$ = significant.

Table 6. Correlation of H. pylori infection with anthropometric measurements and laboratory findings

Variable	R	P
Age	0.058	0.568
Z Weight	- 0.006	0.954
Z Height	- 0.039	0.700
Z BMI	- 0.617**	< 0.001
Hb	- 0.561**	< 0.001
Ht	- 0.338**	0.001
MCV	- 0.421**	< 0.001
RBCs	0.148	0.141
Serum Iron	- 0.482**	< 0.001
TIBC	0.410**	< 0.001
Serum Ferritin	- 0.297**	0.003

Moreover, the findings of our study revealed a significant negative correlation between *H. pylori* infection and BMI, Z scores, Hb, Ht, MCV, serum ferritin levels and serum iron ($p < 0.05$), whereas TIBC was significantly positively correlated with *H. pylori* infection ($p < 0.001$) (Table 6).

DISCUSSION

In cases of refractory IDA, it is recommended to search for *H. pylori* infection in the pediatric population since low iron status has been associated with *H. pylori* infection in multiple studies. Harris et al. [15] revealed that

hypochlorhydria, inflammation and *H. pylori* infection, are correlated with IDA in a prospective trial with 123 children.

In this study, we assessed the prevalence of *H. pylori* infection among Egyptian pediatric patients diagnosed with IDA refractory to treatment. The major finding of our study was the high pervasiveness of *H. pylori* infection, which was 77% among patients with IDA. This finding is higher than the findings of researches carried out by Galal et al[16] , Deeb et al [17] , Mohammad et al [18] and Abdulqawi et al [19] which revealed the prevalence of *H. pylori* infection among Egyptian anemic patients (44%, 64.6%, 72.4%, and 68%, respectively).

Moreover, in Saudi Arabia, a study conducted by Hasosah et al [20] reported that the frequency of *H. pylori* infection in symptomatic pediatric patients was 49.8%. In another research, Iwañczak et al [21] described a maximal pervasiveness of *H. pylori* infection in Nigerian (82%), Ethiopian (48%), Bulgarian (61.7%) and Mexican patients (43%).

In addition, Bille and Mabeku [22] revealed a high predominance of *H. pylori* infection (80.88%) among a pediatric populace with IDA in a sub-Saharan setting.

In contrast to our findings, a low pervasiveness of *H. pylori* infection has been described in researches performed by Abdelaziz et al [23,] Zamani et al [24] and Naous et al [25] (25%, 26%, and 21% respectively). Furthermore, a prospective study conducted on children aged 2–18 years in Turkey reported that the occurrence ratio of *H. pylori* infection was 30.7%. [26] Another Polish study estimated that *H. pylori* sero-positivity was 32.0% in children less than 18 years of age. [27]

The difference in *H. pylori* infection prevalence among various studies may be caused by variations in the design of the study, methods of *H. pylori* diagnosis, sample size, exclusion and inclusion criteria, and socioeconomic classes of the target population. [28]

The studies that described a comparatively decreased incidence of *H. pylori* infection may be attributed to methods of diagnosis of *H. pylori*, since testing *H. pylori* stool antigen is an effective, accurate and noninvasive technique, whereas serum antibody testing procedures for *H. pylori* detection may be concomitant with high false positive results, as they are unable to differentiate between active and past infections. [29] This could also be elucidated by the spontaneous curability of *H. pylori* infection due to the use of antibiotics to treat infections other than *H. pylori* infection in childhood, as a study carried out by Zhou et al [30] reported a yearly spontaneous curability rate of 2.9% in children infected with *H. pylori.* However, the elevated prevalence of *H. pylori* infection is usually accompanied by numerous risk factors such as a decreased socioeconomic level, overcrowded living environments, low sanitation and an absence of sanitary water and reinfection between family members. [31]

In 64% to 75% of individuals with both *H. pylori* infections and IDA, IDA totally disappeared once H. pylori was eliminated, according to Hershko and Camaschella. [32]

No significant difference between the two groups in terms of age was revealed ($p = 0.819$) with respect to the relation between *H. pylori* infection and age in the current study, comparing children with *H. pylori* infection to those without infection Moreover, an insignificant relationship between *H. pylori* infection and age was found in our cases ($p = 0.568$).

Galal et al [16] revealed that *H. pylori* was more prevalent with increasing age; pediatric patients over ten years had the greatest rates of *H. pylori* infection (32.9%), whereas children younger than three years had the lowest (13.8%) which in contrast to our results.

Similarly, Hasosah et al [20] described that the *H. pylori* prevalence was 10.5% in the pediatric population aged less than three years; nevertheless, it was 57.7% in patients older than ten years. This may be attributed to the greater exposure of the pediatric population to the public and outside contact, resulting in poor nutritional habits such as having street food. In contrast, other researchers detected an increase in infection during the infantile period because of sharing beds with brothers or sisters with the infection. [33]

In the present study, comparisons between *H. pylori* infected and non-infected children according to sex distribution revealed significant changes ($p < 0.01$), with the majority of *H. pylori* infected children being females (70.1%). Thus, there appears to be a very strong relationship between sex and *H. pylori* infection, with females

being significantly more likely to have *H. pylori* than males. This could be attributed to sex discrimination and extremely inherent customs, habits and beliefs, as more care is directed towards boys, mainly in a developing nation such as Egypt.

In contrast, sex was not detected as a risk factor for infection with *H. pylori* in children in the study by Galal et al. among Egyptian children.[16] This was also reported in Saudi Arabia by Hasosah et al.[20]

Studies have yielded controversial results concerning the effects of infection with *H. pylori* on children's growth, since a number of them have demonstrated a relationship, [34] whereas others have denied this relationship. [35-38]

However, a cohort study by Mera et al [39] confirmed the long-lasting value of eliminating *H. pylori* in the catch-up of growth in a pediatric population monitored for nearly four years.

Yang et al [40]stated that effective elimination of *H. pylori* in children accelerates growth and returns acylated ghrelin serum concentrations to normal.

Moreover, a previous study revealed a greater proportion of growth retardation among pediatric patients with *H. pylori* infection than among healthy children. [41]

In the current study, we also assessed the potential association of growth retardation with *H. pylori* infection in children. No significant difference in the anthropometric measurements (weight and height Z scores) between the *H. pylori* positive patients and *H. pylori* negative patients ($p = 0.954$ & 0.698).

Incompatible with our results, research carried out in Egypt examining the impacts of *H. pylori* infection on growth in the pediatric population, stunted growth was highly prevalent among the pediatric population with *H. pylori* infection compared with non-infected individuals; the weight-for-age Z scores were significantly lower among the pediatric populace with *H. pylori* infection than among those without infection. [19]

Moreover, a meta-analysis performed by Wei et al. revealed a significant increase in growth retardation in children with *H. pylori* infection compared with those without infection, as they analyzed 15 observational studies comprising 4199 subjects. A greater rate of growth retardation was detected among the *H. pylori* infected pediatric population than among those without infection, especially for height. In addition, there was a relationship between *H. pylori* infection and retarded growth among the pediatric population in studies that included *H. pylori* with a prevalence equal to or less than thirty percent or more than thirty percent but not with a prevalence greater than fifty percent. This agrees with our results as the pervasiveness of *H. pylori* infection was very high in our patients (77%), which was > 50%; thus, we did not detect any correlation of Weight for Age Z score or hight for Age Z score with *H. pylori* infection in our patients ($p = 0.954$ & 0.700).[42]

Additionally, Wei et al [42] in their meta-analysis, performed a subgroup analysis and illustrated the associated growth of children with *H. pylori* infection in Europe, America, and Asia but not in Africa. This finding is also compatible with our results, as we detected non-significant difference in weight or height Z scores amongst *H. pylori* non- infected and infected children who were African.

A possible justification for those variations could be varying *H. pylori* prevalence rates, dissimilar environments and nutrition in diverse nations. Additional influences associated with an augmented prevalence of infection *with H. pylori* and growth retardation among the pediatric population are connected to low socio-economic levels, which are accompanied by inadequate health care, nutritional deficiency and deprived living circumstances, which can affect outcomes. [43-46]

Additionally, the difference between these studies and our findings may be attributed to the fact that the impaired growth of children in these studies was determined by varying guidelines, leading to wide percentile ranges or 1-3 SDs. Furthermore, the differences in age, rate of growth and hormonal concentrations of every sample in the pediatric population might have been important factors in every research. Other possible explanations may be associated with IDA, malnutrition, environmental factors and inter-individual differences. Hence, managing the growth and development of *H. pylori* infected pediatric populations must consider these wide-ranging influences.

Notably, our results revealed an insignificant association of *H. pylori* infection with height ($p = 0.700$). The findings of the current

work are inconsistent with those described by preceding cross-sectional studies, confirming the negative association of *H. pylori* infection with linear growth. Large community-based cross-sectional research (n = 3315) in rural Germany among the pediatric population aged five to seven years revealed a significant decrease in height in children with *H. pylori* infection compared with those without infection.[47]

An additional study in Egypt revealed a significantly greater percentage of children with *H. pylori* infection than of those free of infection who were under the fifth height-for-age percentile. [19]

In parallel with our results, height was not related to *H. pylori* infection in research among Alaskan, [36] Iranian,[37] and Australian[38] pediatric populations.

Regular assessment of anthropometric measurements during childhood and adolescence should be performed. Thus, approaching and evaluating nutritional status non-invasively can assist in monitoring physical growth. Hence, adjusting the nutritional status entirely, not only height and weight separately, is essential. During a child's examination, body mass index (BMI) represents one of the most commonly utilized measures to assess obesity and malnutrition. [48]

With respect to the laboratory data, *H. pylori* positive patients had significantly lower Hb, hematocrit, and MCV values (p = 0.000, 0.001 and 0.000, respectively) than *H. pylori* negative patients did. We also noted a negative correlation between *H. pylori* infection and Hb, Ht, MCV, ferritin, and iron serum levels ($p < 0.05$).

The conclusions of our work also coincide with the findings performed by Bille & Mabeku that described a significantly lower Hb concentration (P = 0.01), Ht value (P = 0.04), insignificant MCV (P = 0.13) and low borderline RBC count (P = 0.06) in *H. pylori* infected patients than in non- infected patients, as well as a negative relationship among *H. pylori* infection and Hb level. [22] Another study performed by Muhsen et al. reported significantly lower Hb concentrations in *H. pylori* positive pediatric patients than in the uninfected individuals. [49] However, a meta-analysis of randomized controlled trials of *H. pylori* elimination by Yuan et al [50] revealed that elimination of *H. pylori* can increase Hb concentrations.

Consistent with our findings, an Egyptian study to Demerdash et al. to assess the causative role of *H. pylori* infection in unexplained or refractory IDA revealed that the MCV was significantly negatively correlated with *H. pylori* infection (P = 0.046), confirming the association of *H. pylori* infection with refractory or IDA of unknown cause among Egyptian adult subjects. IDA may be managed by *H. pylori* elimination combined with iron administration in patients with IDA associated with *H. pylori* infection. It may be impossible to identify a treatable cause of anemia if *H. pylori* infection is not looked into. [3]

In the current work, we detected a significant decrease in serum ferritin ($p=0.003$) and serum iron ($p < 0.001$) and a significant increase in total iron binding capacity ($p < 0.001$) among *H. pylori* positive patients compared with *H. pylori* negative patients.

These findings agree with the study that reported low serum iron and serum ferritin levels and increased serum TIBC levels in *H. pylori* positive patients with IDA. [22]

Study limitations

First, it did not include a normal control group but this could be compensated with that all the parameters included in the laboratory assessment of our cases had normal standards for comparison, and the measures used for the anthropometric assessment were converted into Z scores.

Second, the cross-sectional design of our study prevented us from following up with patients to confirm improvements in IDA and child growth after they were treated with *H. pylori* therapy in addition to iron administration. Moreover, improving nutrition and the living environment throughout the long-term observation of these children can influence these outcomes.

Third, the sociodemographic factors that are strongly linked to H. pylori infection were not examined. An additional limitation of this study is the restricted generalizability of its findings, as the results and conclusions are based solely on an Egyptian cohort and may not be representative of populations in other geographic or demographic contexts.

Fourth, although we did not perform a dietary analysis of our patients to explore whether there was dietary deficiency in the necessary constituents needed for hematopoiesis and whether

our patients were receiving their recommended daily allowances, we assessed the nutritional status of our patients via BMI-Z scores.

CONCLUSION

The current study's findings point to a substantial correlation between refractory IDA in children and H. pylori infection. Therefore, we recommend the use of *H. pylori* detection in routine work-up protocols for refractory or unexplained IDA management.

When *H. pylori* infection is being diagnosed, immediate eradication of the infection should be started to prevent consequent strong harmful influences. Moreover, it is essential to create a constant standard classification for growth retardation in children. We recommend that strategies for *H. pylori* diagnosis and therapy should be prioritized as a public health issue, particularly in developing countries such as Egypt. Future studies in children without symptoms are required to address this concern, as successful elimination of *H. pylori* during childhood is essential.

To further explore the effects of H, additional high-quality, well planned controlled clinical trials should be carried out. pylori elimination on IDA and these children's development.

ACKNOWLEDGEMENTS

The author would like to thank the General Organization of Teaching Hospital and Institutes for their guidance during the Satellite data procurement.

Funding Sources

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Conflict of interest

The authors do not have any conflict of interest.

Data Availability Statement

This statement does not apply to this article.

Ethics Statement

The study was conducted in accordance with the Helsinki Declaration and was authorized by the General Organization of Teaching Hospital and Institutes' ethical committee (No. HG000103). All patients or their legal guardians gave their informed permission..

Informed Consent Statement

This study did not involve human participants, and therefore, informed consent was not required.

Clinical Trial Registration

This research does not involve any clinical trials.

Permission to reproduce material from other sources

Not Applicable

Author contributions

Nahla A. Mohamed: Conceptualization, writing the original draft and approved final draft; Eman Refaat Youness and Marwa A. Mahmoud: laboratory procedures, data analysis and approved final draft; Mina Wassef Girgiss: collect the data; Omaima Mostafa Badran: Clinical investigations; Hanaa Reyad Abdallah writing the original draft and approved final draft.

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