Prevalence of *Helicobacter Pylori* Infection in Anemic and Non-anemic Children in Helwan, Egypt: Impact on Blood Cell Parameters

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ABSTRACT

Background: *Helicobacter (H.) pylori* is the most common chronic bacterial infection of humans; affecting ~50% of the world's population. It is the cause of disease states of varying degrees of severity. Anemia is a widespread public health problem; ~50% of cases are diagnosed as iron deficiency anemia (IDA). Recent studies have suggested an association between *H. pylori* infection and IDA in children. **Aims of the work:** this study was conducted to evaluate the prevalence of *H. pylori* infection in children with and without IDA diagnosis and also to determine effects of the bacterium on complete blood count parameters of those children. **Subjects and Methods:** a case-control (retrospective) study design was chosen to conduct this research. The prevalence of *H. pylori* antibody (Ab) seropositivity was compared between 50 children diagnosed with IDA vs. 50 non-anemic control children matching in age and sex. **Results:** a total of 18 (36%) anemic and 10 (20%) non-anemic children were found positive to *H. pylori* Ab (P=0.0013). Also, comparison of the anemic to the control group revealed statistically significant lowering of ferritin, and red blood cell (RBC) parameters (i.e., hemoglobin, packed cell volume, mean corpuscular volume, and mean corpuscular hemoglobin), and also platelet count in the anemic group. Moreover, comparison of *H. pylori* positive and negative anemic children revealed statistically significant lowering of RBC parameters in the *H. pylori* positive anemic children. Also, comparison of *H. pylori* positive and negative children revealed statistically significant lowering of RBC parameters in *H. pylori* positive children. In addition, correlation of *H. pylori* with all other parameters revealed negative significant correlation between *H. pylori* and RBC parameters. **Conclusions:** *H. pylori* infection had a higher prevalence among preschool children with IDA and the hematological impact was more on *H. pylori* positive anemic children. **Recommendations:** both IDA and *H. pylori* are treatable diseases, so children having IDA must be investigated for *H. pylori* infection for early treatment to avoid serious complications of both diseases.

Keywords: Egypt, Helwan, *H. pylori*, IDA, Preschool aged children.

INTRODUCTION

Helicobacter (H.) pylori is a microaerophilic, Gram-negative, spiral-shaped, flagellated organism. It is the most common chronic bacterial infection of humans as it is present in almost half of the world's population [1]. The pathogen has been shown to be the causative agent of disease states of varying degrees of severity, including; chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and gastric mucosa-associated lymphoid tissue lymphoma [2]. The prevalence of *H. pylori* infection varies in different populations, even within the same geographic regions. It has been found that the highest rate of infection is associated with low socioeconomic status during childhood [3]. The rates of infection range from more than 80% in the developing world to less than 40% among industrial countries [4]. In developed countries, widespread use of treatment against *H. pylori* infection has led to dramatic decrease in the prevalence of infection [5].

Iron deficiency (ID) is the most widespread cause of anemia worldwide [6]. The WHO estimated that about two billion people in the world are suffering from anemia, with approximately fifty percent of them are diagnosed as IDA [7]. It develop in three stages; iron depletion, iron deficient erythropoiesis, and IDA [8]. It is estimated to be the most common nutritional deficiency in both developing and developed countries [9]. ID results in impairment of the immune, cognitive, and reproductive functions, as well as lowered work performance. It is also suggested to be related to DNA damage. IDA in children is still considered as a major health problem all over the world. This is because of the long term effects on mental and cognitive skills, immunity, and general physical well being [10].

It was reported that *H. pylori* may influence some extra-gastrointestinal diseases such as idiopathic thrombocytopenic purpura (ITP), anemia, and allergic diseases [5]. The role of *H. pylori* infection in the development of extra-

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gastrointestinal diseases, including IDA, has been the focus of attention during the last decade [11]. Epidemiologic studies have indicated that *H. pylori* seropositivity is associated also with low serum (S.) ferritin and hemoglobin (HB) levels compared with seronegative controls in adults and children [12]. Also, recent studies have suggested an association between *H. Pylori* gastric infection and IDA in children refractory to iron therapy, which is reversed only after bacterial eradication. It has been also reported that eradication of *H. pylori* may result in improvement of anemia even without iron supplementation [13]. This suggests the possible interference of *H. pylori* in iron metabolism that may lead to IDA [14], although the nature of the interactions has not been established [15]. So, it is still a controversial issue as many studies showing negative impact of *H. pylori* on iron status [16].

AIM OF THE WORK

This study was conducted to evaluate the prevalence of *H. pylori* among preschool aged children who are suffering from IDA in comparison to non-anemic children matching in age and sex. The aim of the research was also to evaluate effects of the *H. pylori* on iron status and on blood cell count parameters of infected children in both anemic and non-anemic groups.

SUBJECTS AND METHODS

This is a case-control retrospective study that was conducted between June and October 2015. At first, informed verbal consents were obtained from parents of the participating children. Exclusion criteria included intake of iron supplementation or eating diet rich in iron within one week of sampling. This study compares between two groups of children who were selected from the Cairo Lab Laboratory, Helwan Branch, Egypt. A total number of 100 children were participated in the study (50 in each group; i.e., 1:1). Both groups were matched in age (36-72 months) and sex (25 males and 25 females) in each group. These 2 groups are:

1. Non-anemic (control) group I: formed of 50 selected normal children aged 36-72 [mean (M) ± standard deviation (SD) = 52.38±9.337] months, with no clinical manifestations of anemia, normal or near to high normal S. ferritin level of 12-67 [17] (44.202±22.002) ng/mL, and normal HB Conc of ≥11 [18] (M±SD=11.754±0.6756) g/dL.
2. Anemic (case) group II: formed also of 50 selected anemic children aged 36-72 (M±SD=52.74±13.978) months, with presence of the clinical picture of anemia [19], low or near to baseline S. ferritin level of <12 [17] (M±SD=23.794±13.214) ng/mL, and low HB Conc of <11 [18] (M±SD=9.85±0.856) g/dL.

Three (3) milliliters (mL) of the whole venous blood were drawn from every child cautiously. One (1) mL of them was put in a vacutainer tube containing 1 mg of EDTA (ethylene-diamine-tetra-acetic acid) anticoagulant and mixed gently by hand inversion to avoid hemolysis (for complete blood count "CBC" analysis). The other two (2) mL were left to clot in a vacutainer plain tube (without an anticoagulant) in the incubator at 37^{o}C for 30 minutes, and then centrifuged for another 15 minutes at 3000 revolutions per minute (rpm), and then the supernatant serum was separated carefully into another tube for analysis of S. ferritin and *H. pylori* IgG antibody. All analyses were done soon after sampling without storage or delay).

CBC hematological determination was performed with the autohematology analyzer Mindray (BC-5300) using a flow cytometry, semi-conductor Laser scatter, chemical dye method. This analyzer is calibrated and controlled with standard laboratory quality control methods.

Hematological tests included: RBC (red blood cell count), HB Conc (hemoglobin concentration), PCV (packed cell volume), MCV (mean corpuscular volume), MCH (mean corpuscular hemoglobin), MCHC (mean corpuscular hemoglobin concentration), PLT (platelet count), WBC (total white blood cell count), NE (neutrophil count), LY (lymphocyte count), MO (monocyte count), and EO (eosinophils count). Basophil count was zero.

S. Ferritin Conc had been performed on Maglumi 1000 Snibe fully auto chemiluminescence immunoassay (CLIA) analyzer using a sandwich chemiluminescent immunoassay method (Maglumi Ferritin "CLIA").

H. Pylori IgG antibodies were detected qualitatively by the use of a rapid chromatographic immunoassay one step test device (serum) from Abon Biopharm (Hangzhou) Co., Ltd.

Data entry and statistical analyses were performed using the statistical program [GraphPad InStat, Version 3]. Percentage and proportions were used to determine the prevalence rates of *H. pylori* among control and anemic children. The mean values of the groups were compared using Student's unpaired "t" test and all values were summarized as mean ± standard deviation in tables. Pearson's "r" test

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examined correlation between *H. pylori* and all other analyzed parameters. P-values of <0.05 were considered statistically significant.

RESULTS

Table 1 showed comparison between control (group I) and anemic (group II) children regarding prevalence of *H. pylori* positive cases. Ten (20%) children [5 boys (50%) and 5 girls (50%)] were positive for *H. pylori* IgG Ab test in the control group and 18 (36%) children (5 boys "27.8%" and 13 girls "72.2%") were positive to *H. pylori* IgG Ab test in the case group, with a highly statistical significant difference between the two groups (P=0.0013).

Table 2 showed comparison between case and control groups regarding gender (sex), age, and results of the laboratory analyses that included biochemical (S. ferritin Conc) and hematological (complete blood count) tests. The mean ages of control and case groups were 52.38±9.337 and 52.74±13.978 months, respectively, with no statistical significant difference between them (P=0.8799). Comparison of anemic to the control group revealed highly statistically significant lowering of S. ferritin Conc (P<0.0001), RBC (P=0.003), HB Conc (P<0.0001), PCV (P<0.0001), MCV (P<0.0001), and MCH (P<0.0001), with only a statistically significant lowering of the PLT (P=0.0278) in the anemic group.

Table 3 showed comparison of *H. pylori* positive and negative anemic children. It revealed significant lowering of HB Conc (P=0.0237), PCV (P=0.0103), MCV (P=0.0209), and MCH (P=0.0309) in *H. pylori* positive anemic children.

Table 4 showed comparison of *H. pylori* positive and negative control children and it revealed no statistical significance.

Table 5 showed comparison between *H. pylori* positive and negative children in both groups and it revealed highly statistically significant lowering of HB Conc (P=0.0024) and PCV (P=0.0009), with only statistically significant lowering of MCV (P=0.0426), and MCH (P=0.0289) in the *H. pylori* positive children.

Table 6 showed correlation between *H. pylori* with all other parameters in all children. It revealed highly significant negative correlation between *H. pylori* with HB Conc (P=0.0024) and PCV (P=0.0009), with only significant negative correlation between *H. pylori* with MCV (P=0.0426) and MCH (P=0.0289).

DISCUSSION

Regarding gender, the present study is a case-control retrospective one in which comparison between a total number of 100 children [50 (50%) males and 50 (50%) females] was done. As a whole it showed positive *H. pylori* infection in 28 (28%) children; 10 (~35.7%) males and 18 (~64.3%) females **(table 1)**. An excess of *H. pylori* prevalence in one gender versus the other had been reported; e.g., **Woodward and his colleagues** [20] who observed a higher prevalence of *H. pylori* in men than in women. Also, in Backos (Alexandria, Egypt) boys were more infected with *H. pylori* than girls; however with no statistical significance [21]. Additionally, a more comprehensive meta-analysis of large population-based studies concluded male predominance of *H. pylori* related diseases in adults, but not in children [22]. On the other hand, some studies found no gender-related difference in the prevalence of *H. pylori* infection [23].

Adult males in Egypt are more prone to infection than adult females due to higher exposure; but, in children the chance of exposure is about the same in both genders. However, this is a case-control and not a cross sectional study which may affect the obtained results in either direction. Also, the number of children involved in the present study is not large enough to compare male/female predominance. The strength of the study regarding sex includes the low age of the current participants because some covariates such as menstruation, which might contribute to additional residual confounding factor for ID or the difference between the two sexes, were automatically ruled out.

Regarding age, this study is a case-control retrospective one in which children was selected according to their age i.e., 36-72 weeks. The age of participants was lower than most other similar studies. Because most *H. pylori* infections are acquired during early childhood, particularly in children aged less than 5 years, a continuous contact is required for establishment of a real infection that can last lifelong. Infection rates are lower after this period due to the fact that less contact occurs between mothers and children. This is because children start their school attendance and spend more time outside their homes. Infection with *H. pylori* continues to be acquired by children after that, however at lower rates depending on the mode of transmission. During adult life, also, married couples are at high risk of infection if one spouse is infected [24].

According to *H. pylori* prevalence, 18 (36%) anemic and 10 (20%) non-anemic children were found to be positive for *H. pylori* Ab test with a

total prevalence of 28%. Similar results were also obtained from different areas in Egypt. In Damanhour (Egypt), the prevalence of *H. pylori* was widely age dependent; it was 25.9% among children less than 5 years [25]. In addition, *H. pylori* prevalence among primary school children in Backos (Alexandria, Egypt) was 27.1% [21]. Moreover, one study that was done in central Cairo (Egypt) found *H. pylori* prevalence of 33% in children less than 6 years [26]. These results were more or less similar to the results of the present research.

In the present study, regarding S. ferritin, which is used as a marker for total body iron, and as expected, there was a highly statistically significant lowering of S. ferritin levels in the anemic group compared to the control group (P<0.0001) **(table 2)**. However, when S. ferritin was compared between positive and negative *H. pylori* anemic cases **(table 3)**, positive and negative *H. pylori* controls **(table 4)**, and *H. pylori* positive and negative children **(table 5)**, or when S. ferritin was correlated with *H. pylori* **(table 6)** there were no statistical significances.

Regarding association between H. pylori infection and IDA the results of this study were comparable to another study that was done in Tehran (Iran) among children aging 40-75 months. This study concluded that *H. pylori* infection had a significant high prevalence among preschool children with IDA compared to the controls [27]. However, prevalence of *H. pylori* in the anemic group was very high in the Tehran (Iran) study (81.3%) compared to the present study (36%) which may be due to higher prevalence of *H. pylori* infection in Tehran (Iran) than Helwan (Egypt).

H. Pylori infection affects iron metabolism in humans and several studies have shown a relationship between *H. pylori* and IDA [28]. In addition, it seems that elimination of *H. Pylori* infection induces beneficial effects on ID [29]. Also, S. ferritin levels were found to be reduced in people with increased IgG antibodies to *H. pylori*. Whether this is caused by the increased iron loss or the decreased iron absorption is not clear yet [28]. According to the First World Congress of Pediatric Gastroenterology, Hepatology and Nutrition working group report, children infected with *H. pylori* had lower body iron stores in comparison to age-matched controls [30].

However, in another study, it was concluded that *H. pylori* is not associated with IDA in men with normal gastrointestinal tract endoscopy results, but it may be associated with IDA in patients with impaired gastrointestinal mucosa [31]. According to researchers at the University of Texas Health Science Center at Houston (UTHealth) children without previous ID or anemia who remained infected with *H. pylori* had significantly lower levels of iron compared to children who had the infection eradicated [32]. Also, failure of response to iron supplementation or a recurrence of anemia at puberty may be associated with *H. pylori* infection, thus suggesting possible interference of *H. pylori* in iron metabolism. The eradication of *H. pylori* could resolve the refractory IDA [33].

On the opposite side, another study that was done on children aged 2-14 years reported that there was no association between *H. pylori* infection and IDA [34]. Also, a cross-sectional study that was carried out by **Zamani *et al.*** [35] between children 6 and 12 years old to evaluate the relationship between S. ferritin levels, hence IDA, and *H. pylori* IgG antibody found no association between *H. pylori* infection and low S. ferritin levels or IDA. However, the results of these researches are strange among a lot of researches that relate *H. pylori* infection to IDA.

Although, studies still have controversy about the association between *H. pylori* infection and iron stores, and therefore IDA, some authors believe that variation in *H. pylori* species is one of the possible reasons for disagreement with findings in the literature [36]. Furthermore, studies have compared different parameters to evaluate iron and also included different age groups that may influence their results. For example, since S. ferritin level, a marker of the body iron stores, is an acute phase protein that is elevated during infections and inflammations, its comparison between *H. pylori* infected and non-infected individuals for iron status may have some effects on the results.

The mechanisms responsible for the effect of *H. pylori* on iron status remains unclear, however some theories argue that several pathways may be involved which include consumption of iron by the bacterium, gastrointestinal blood loss due to gastritis or duodenitis, and decrease in iron absorption due to low levels of gastric acid [37].

As expected also in **table 2** when we compared anemic and control groups there were highly significant decreases of RBC count (P=0.003), HB Conc (P<0.0001), PCV (P<0.0001), MCV (P<0.0001), and MCH (P<0.0001) in the anemic group. Also, when *H. pylori* positive and negative anemic children were compared in **table 3** there were statistically significant decreases of HB Conc (P=0.0237), PCV (P=0.0103), MCV (P=0.0209), and MCH (P=0.0309) in the *H. pylori* positive anemic

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children. This indicates more deleterious effects of *H. pylori* infection on anemic children. Moreover, when *H. pylori* positive and negative control children were compared in **table 4** there was no statistical significance difference. However, when *H. pylori* positive and negative children were compared in **table 5** there were highly significant decreases in HB Conc (P=0.0024) and PCV (P=0.0009), with only statistically significant decreases in MCV (P=0.0426) and MCH (P=0.0289) in the *H. pylori* positive children. Also, when *H. pylori* was correlated with all other parameters in **table 6** there was highly significant statistical negative correlation with HB (P=0.0024) and PCV (P=0.0009), with only significant statistical negative correlation with MCV (P=0.0426) and MCH (P=0.0289). From data in **tables 2-6** it was concluded that *H. pylori* is a highly suspected cause of IDA, which is due to the ID state.

In a study that was done to evaluate the effect of *H. pylori* eradication on blood count, the results showed that after three months RBC count, HB Conc, MCV, and MCHC were significantly increased [38]. This confirms results of the present study about RBC parameters decrease in *H. pylori* infected children.

Regarding platelets, when control and anemic children were compared **(table 2)** there was significant decrease in platelet count (P=0.0278) in the anemic group. Also, when *H. pylori* positive and negative anemic cases were compared **(table 3)** there was not quite significant decrease (P=0.0759) in the positive *H. pylori* anemic children. However, when *H. pylori* positive and negative controls were compared **(table 4)** there was non-significant decrease (P=0.6417) in the positive *H. pylori* control children. Moreover, when *H. pylori* positive and negative children were compared **(table 5)** there were also not quite significant decrease (P=0.0594) in the positive *H. pylori* children. In addition, when platelet count was correlated with *H. pylori* **(table 6)** there was not quite significant result (P=0.0595). These results collectively raise a high suspicion that *H. pylori* may lead to decrease in platelet count (with or without thrombocytopenia). However, no child involved in this study had thrombocytopenia. So, *H. pylori* may be the cause of decreased platelet count in those infected children later in life.

These results were in accordance with many investigators. **Yeh *et al.*** [39] document induction of platelet aggregation by *H. pylori* in vitro and showed that this effect is strain-dependent. Also, **Gasbarrini *et al.*** [40] showed a high prevalence of *H. pylori* infection in patients with ITP and reported a good response to the bacterium eradication in most of them. Several studies have also shown that *H. pylori* eradication in infected patients with ITP could lead to a substantial and persistent increase in platelet counts in over half of the patients treated [41] indicating the effect of the organism on the thrombocytes count. In one study the effect of *H. pylori* eradication on blood count results showed that; two weeks after *H. pylori* eradication platelet's count significantly increased [38]. This confirmed the results of the present study about platelet counts decrease in the infected children.

How might *H. pylori* infection contribute to development of thrombocytopenia? *H. pylori* express Lewis (Le) antigens in a strain-specific manner; Le antigens adsorb to platelets and might serve as targets for anti-Le antibodies in patients with an appropriate genetic background [42]. In addition, both *H. pylori* infection and ITP are associated with a T helper 1 type immune response characterized by increased levels of interferon γ and interleukin-2; hence, *H. pylori*-induced alterations in cytokine profiles might promote development of ITP. Additionally, some strains of *H. pylori* bind von Willebrand factor and induce glycoprotein Ib and FcγRIIa-dependent platelet aggregation in the presence of *H. pylori* antibodies [43]. Also, direct antigen mimicry between *H. pylori* and platelet glycoproteins must be considered [44].

Regarding WBC (total and differential counts) when they were compared between groups and subgroups **(tables 2, 3, 4, 5)** and when they were correlated with *H. pylori* **(table 6)** there were no significant results between *H. pylori* positive and negative groups. This indicated that there was no effect of *H. pylori* infection on both total and differential WBC counts.

However, in one study that was done to evaluate the effect of *H. pylori* eradication on blood count, the results showed that total WBC, neutrophil, and lymphocyte counts were significantly reduced [38]. Accordingly, *H. pylori* infection may increase total WBC due to increased neutrophil and lymphocyte counts as they are the dominant two cells of all leucocytes. Thus, neutrophil and lymphocyte counts decrease due to cure from *H. pylori* infection. However, another prospective study confirmed the existence of an association between *H. pylori* infection and chronic idiopathic neutropenia [45]. Another study that compared differential counts of leukocytes in peripheral blood before and after eradication of *H. pylori* found that *H. pylori*

infection of the gastric mucosa increases neutrophil and monocyte counts in the peripheral blood. Also, it was found in the eradicated group that neutrophils and monocytes counts were decreased significantly after eradication, with no significant change in eosinophils, basophils, and lymphocytes. On the other hand, there was no significant change in leucocytes in the non-eradicated group [46]. These results were against the present study and may be due to different strains of the bacterium involved.

It is concluded that; 1. Preschool aged children with IDA had a higher *H. pylori* prevalence, 2. The impact of *H. pylori* infection on RBC parameters is high, with more impact on anemic ones, 3. Platelet count is highly suspected to decrease due to *H. pylori* infection, 4. *H. pylori* infection had no effect on total or differential WBC counts.

It is recommended to; 1. Conduct another study on a larger number of children, 2. Work in the opposite way of this research i.e., searching for the prevalence of anemia among *H. pylori* positive children, 3. Confirm *H. pylori* antigen positivity among infected children e.g., by urea breath or stool antigen tests for estimation of the actual number of diseased children, thus to specify *H. pylori* impact well. 4. Identify the genotype of the *H. pylori* organisms in the area to correlate it well with the present clinical and laboratory settings. 5. Investigate children having IDA for *H. pylori* infection for early treatment to avoid serious complications of both diseases.

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Table 1. Comparison between control and anemic children regarding prevalence of *H. pylori* positive cases

	Control group I (No. = 50)	**Anemic group II (No. = 50)**	**P-value**
***H. pylori* (positive)**	Total = 10	Total = 18	0.0013*
Percent (%)	20%	36%	
	5 M (50%) / 5 F (50%)	5 M (27.8%) / 13 F (72.2%)	---

*; Statistically significant, F; female, M; male, No.; number.

Table 2. Comparison between control and anemic children regarding gender, age, S. ferritin, and CBC

Parameters	**Control children (No. = 50)**	**Anemic children (No. = 50)**	**P-value**
Gender	25 M (50%) / 25 F (50%)	25 M (50%) / 25 F (50%)	---
Age (Months)	52.38±9.337	52.74±13.978	0.8799 (NS)
S. Ferritin (ng/mL)	44.202±22.002	23.794±13.214	<0.0001*
RBC ($\times10^6$/µL)	4.387±0.294	4.601±0.3996	0.003*
HB Conc (g/dL)	11.754±0.6756	9.85±0.856	<0.0001*
PCV (%) (L/L)	35.082±2.021	29.912±3.125	<0.0001*
MCV (fL)	79.608±5.31	65.134±5.556	<0.0001*
MCH (pg)	26.724±1.738	21.688±1.788	<0.0001*
MCHC (g/dL)	33.32±0.6972	33.314±1.23	0.9761 (NS)
PLT ($\times10^3$/µL)	338.36±100.8	297.34±81.938	0.0278*
WBC ($\times10^3$/µL)	8.067±3.085	7.713±2.091	0.5029 (NS)
NE ($\times10^3$/µL)	3.769±2.208	3.411±1.581	0.3535 (NS)
LY ($\times10^3$/µL)	3.614±1.447	3.62±1.082	0.9794 (NS)
MO ($\times10^3$/µL)	0.4266±0.1808	0.4292±0.1705	0.9412 (NS)
EO ($\times10^3$/µL)	0.2514±0.2679	0.2694±0.1548	0.6817 (NS)

*; Significant, No.; Number, NS; Non-significant.

Table 3. Comparison between *H. pylori* positive and negative anemic children regarding gender, age, S. ferritin, and CBC

Parameters	**Negative *H. pylori* anemic children**	**Positive *H. pylori* anemic children**	**P-value**

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	(No. = 32; 64%)	(No. = 18; 36%)	
Gender	20 M (62.5%) / 12 F (37.5%)	5 M (~27.8%) / 13 F (~72.2%)	---
Age (Months)	52.656±13.585	52.889±15.052	0.89556 (NS)
S. Ferritin (ng/mL)	24.594±14.227	22.372±11.44	0.5736 (NS)
RBC ($\times10^6$/µL)	4.637±0.3396	4.538±0.4931	0.4055 (NS)
HB Conc (g/dL)	10.053±0.5553	9.489±1.155	0.0237*
PCV (%) (L/L)	30.747±2.24	28.428±3.918	0.0103*
MCV (fL)	66.478±4.587	62.744±6.415	0.0209*
MCH (pg)	22.094±1.646	20.967±1.848	0.0309*
MCHC (g/dL)	33.231±1.33	33.461±1.051	0.5316 (NS)
PLT ($\times10^3$/µL)	312.75±75.71	269.94±87.507	0.0759 (NS)
WBC ($\times10^3$/µL)	7.786±2.249	7.583±1.831	0.7463 (NS)
NE ($\times10^3$/µL)	3.475±1.68	3.298±1.427	0.7073 (NS)
LY ($\times10^3$/µL)	3.595±1.162	3.666±0.9537	0.8267 (NS)
MO ($\times10^3$/µL)	0.4541±0.1984	0.385±0.09382	0.1717 (NS)
EO ($\times10^3$/µL)	0.2872±0.1558	0.2378±0.152	0.2831 (NS)

* Significant, No.; Number, NS; Non-significant.

Table 4. Comparison between *H. pylori* positive and negative control children regarding gender, age, S. ferritin, and CBC

Parameters	Negative *H. pylori* control children (No. = 40; 80%)	Positive *H. pylori* control children (No. = 10; 20%)	P-value
Gender	20 M (50%) / 20 F (50%)	5 M (50%) / 5 F (50%)	---
Age (Months)	51.425±9.421	56.2±8.351	0.1499 (NS)
S. Ferritin (ng/mL)	44.985±22.079	41.07±22.577	0.6198 (NS)
RBC ($\times10^6$/µL)	4.427±0.2958	4.23±0.2389	0.0575 (NS)
HB Conc (g/dL)	11.813±0.6768	11.52±0.6512	0.2243 (NS)
PCV (%) (L/L)	35.243±2.015	34.44±2.017	0.2657 (NS)
MCV (fL)	79.143±5.618	81.47±3.448	0.2185 (NS)
MCH (pg)	26.648±1.766	27.03±1.671	0.5391 (NS)
MCHC (g/dL)	33.365±0.6762	33.14±0.7877	0.3668 (NS)
PLT ($\times10^3$/µL)	341.73±94.931	324.9±126.55	0.6417 (NS)
WBC ($\times10^3$/µL)	7.916±3.199	8.672±2.637	0.4939 (NS)
NE ($\times10^3$/µL)	3.538±2.256	4.696±1.812	0.1394 (NS)
LY ($\times10^3$/µL)	3.693±1.499	3.295±1.233	0.442 (NS)
MO ($\times10^3$/µL)	0.4333±0.1864	0.4±0.1623	0.608 (NS)
EO ($\times10^3$/µL)	0.2428±0.2438	0.286±0.3626	0.6527 (NS)

* Significant, No.; Number, NS; Non-significant.

Table 5. Comparison between *H. pylori* positive and negative children regarding gender, age, S. ferritin, and CBC

Parameters	*H. pylori* negative group (No. = 72)	*H. pylori* positive group (No. = 28)	P-value

Sex	40 M (~55.6%) / 32 F (~44.4%)	10 M (~35.7%) / 18 F (~64.3%)	---
Age (Months)	51.972±11.389	54.071±12.981	0.4283 (NS)
S. Ferritin (ng/mL)	35.922±21.453	29.05±18.318	0.1381 (NS)
RBC ($\times10^6$/µL)	4.52±0.3309	4.428±0.4412	0.2586 (NS)
HB Conc. (g/dL)	11.031±1.078	10.214±1.401	0.0024*
PCV (%) (L/L)	33.244±3.08	30.575±4.43	0.0009*
MCV (fL)	73.514±8.166	69.432±10.647	0.0426*
MCH (pg)	24.624±2.844	23.132±3.44	0.0289*
MCHC (g/dL)	33.306±1.014	33.346±0.9624	0.8547 (NS)
PLT ($\times10^3$/µL)	328.85±87.539	289.57±104.3	0.0594 (NS)
WBC ($\times10^3$/µL)	7.858±2.799	7.972±2.17	0.8466 (NS)
NE ($\times10^3$/µL)	3.51±2.007	3.797±1.686	0.5044 (NS)
LY ($\times10^3$/µL)	3.649±1.351	3.533±1.055	0.6835 (NS)
MO ($\times10^3$/µL)	0.4425±0.1908	0.3904±0.1199	0.1819 (NS)
EO ($\times10^3$/µL)	0.2625±0.2092	0.255±0.2428	0.8781 (NS)

* Significant, No.; Number, NS; Non-significant.

Table 6. Correlation of *H. pylori* with all other parameters

	Parameters	**Correlation Coefficient (r)**	**P-value**
H. pylori	**Age**	0.08009	0.4283
	S. Ferritin	- 0.1493	0.1381
	RBC	- 0.114	0.2586
	HB	- 0.3004	0.0024*
	PCV	- 0.3266	0.0009*
	MCV	- 0.2032	0.0426*
	MCH	- 0.2186	0.0289*
	MCHC	0.01854	0.8547
	PLT	- 0.1892	0.0594
	WBC	0.01959	0.8466
	NE	0.06753	0.5044
	LY	- 0.04127	0.6835
	MO	- 0.1346	0.1819
	EO	- 0.01554	0.8781

(-) * Significant lowering.

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