Original Article

Effect of H Pylori on Iron and Serum Ferritin

1. Saqib Sohail 2. Khalid-ur-Rehman 3. Tahir Usman

1. Asstt. Prof. of Physiology, PMC, Faisalabad 2. Asstt. Prof. of Pathology, PMC, Faisalabad
3. Asstt. Prof. of Physiology, AIMC, Lahore

ABSTRACT

Background: Helicobacter pylori produce gastric inflammation and interfere with iron by producing extragastric complications. The deficiency of iron in patients with gastritis or peptic ulcer leads to iron deficiency anemia.
Objective: The objective of this study was to elucidate the effects of Helicobacter pylori infection on red serum iron and ferritin levels.
Study Design: Cross sectional analytical study
Place and Duration of Study: This study was a conducted at the University of Health Sciences, Lahore from March 2009 to September 2009.
Materials and Methods: A total number of 90 subjects were included in the study. They were divided into group A (30 subjects with gastric symptoms and H. pylori infection), group B (30 subjects with gastric symptoms but without H. pylori infection), and group C (30 normal healthy age and sex matched subjects). H. pylori infection was considered positive on the basis of positive serology, rapid urease test and histopathological examination. Serum ferritin was estimated by chemilumisecne technique while serum iron was measured by endpoint colorimetric method.
Results: The results of this study did not show any significant effect on serum ferritin and serum iron (p value > 0.05) within the individual groups nor when compared with each other.
Conclusion: H. pylori infection did not affect the serum ferritin and serum iron levels.
Key Words: Ferritin, Iron, Helicobacter Pylori

INTRODUCTION

Many studies in the late 19th and in the early years of 20th century revealed the presence of Helicobacter pylori in the animal gastric mucosa. Later, it was identified and isolated by doing culture from the human gastric mucosa by Warren and Marshal in 1982 and upon this historic discovery; they were awarded Nobel Prize in Physiology/Medicine in 2005. [1] Helicobacter pylori are a spiral shaped microaerophilic gram negative bacteria. Helicobacter pylori are about 3 µm in length and 0.5 µm in diameter. Helicobacter pylori have the capability that it can change its shape from spiral to non-culturable form, i.e. coccoid. [2] Human stomach is reported to be the primary reservoir for Helicobacter pylori. In the human stomach, Helicobacter pylori are usually found in the gastric antrum. Greatest amount of the bacterium is reported under the mucus layer in gastric pits where it is reported to bind with Lewis b antigen on the surface of gastric epithelial cells with the help of Bab A adhesion molecules. [3,4] After ingestion, Helicobacter pylori first reside in the stomach by neutralizing the gastric acidity. For neutralization, Helicobacter pylori secretes urease enzyme that produces ammonia from urea present in the stomach lumen by the process of hydrolysis. Ammonia being alkaline in nature neutralizes the gastric acidity. [5-7] Helicobacter pylori produces gastric diseases by secreting Cag A and Vac A toxins.[8] Helicobacter pylori associated gastritis is reported to result in many extra gastric complications like iron deficiency and iron deficiency anemia, vitamin B_{12} deficiency and megaloblastic anemia, and ischemic heart disease. [9] The exact mechanism of iron deficiency and anemia due to iron deficiency is not clear. The proposed mechanisms are that H. pylori first produces alkaline media in the stomach by secreting urease enzyme that neutralizes gastric acidity by producing ammonia, thus decreasing the gastric power to dissolve the dietary content of iron.[10, 11] Second, H. pylori decreases the vitamin C content of the gastric juice which is required for the formation of soluble complexes with iron, and also required as a cofactor for the reduction of ferric to ferrous form. [12] Third, it utilizes dietary iron for its own use. [13] The present study is planned to see the effects of H. pylori of iron physiology as literature reveals controversial picture.

MATERIALS AND METHODS

It was a cross sectional analytical study conducted at the University of Health Sciences, Lahore. Subjects with Helicobacter pylori infection and subjects having gastric symptoms only were selected from the Services Hospital, Lahore. Ninety subjects including both male and female were selected for the study. The subjects were between 15-60 years of age. Subjects were divided into three groups. Group 1 was comprised of thirty subjects with Helicobacter pylori infection. Group 2 was composed of thirty subjects with history of gastric symptoms without Helicobacter pylori

infection. In the group 3, age and sex matched healthy subjects without gastric symptoms and Helicobacter pylori infection were included.

An informed consent was taken from all the subjects after explaining the study purpose and procedure. Detailed clinical history was obtained from all the subjects. Diagnosis of Helicobacter pylori infection was made by performing ELISA for H. pylori IgG antibodies, rapid urease test and histopathological examination.

5 milliliter of blood was taken in a plain tube and was centrifuged at 5000 rpm for 10 minutes. Serum was separated and stored at -80 °C for estimation of serum H. pylori IgG antibodies, serum ferritin and serum iron. Serum ferritin was estimated by chemilumisecne technique. Serum ferritin kit by Ortho-Clinical Diagnostics (Lot no. 1070) was used. Serum iron was measured by endpoint colorimetric method. The kit used for the measurement of iron was manufactured by Randox Laboratories Ltd., Ardmore, Diamond Road, Crumlin, Co. Antrim, United Kingdom (Cat No. S1257).

Statistical Analysis: The data was analyzed using SPSS 16.0. Mean ± SD are given for quantitative variables. Frequencies, percentages and graphs are given for qualitative variables. Arithmetic mean and standard deviation of all the quantitative variables like iron and ferritin was determined.

Two –Independent sample t-test was applied to observe group-mean differences between two groups (males and females in each group like A, B and C).

One way ANOVA was applied to determine the difference between groups and association between qualitative variables. The p value of less than 0.05 was considered statistically significant.

RESULTS

The age of subjects was found to be 32.87 ± 12.31 years in group A, 33.27 ± 11.64 years in group B, and 33.60 ± 11.12 years in group C. The difference of Mean ± SD age in three groups was non-significant ($p > 0.05$; Table 1).

There was no significant difference in serum iron concentration between males (116.1± 87.8 µg/dl) and females (95.9 ± 58.8 µg/dl) in group A. The serum ferritin values in males (64.4 ± 55.3 ng/ml) and females (63.8 ± 36.6 ng/ml) of group A were significantly not different ($p > 0.05$; Table 2).

There was no significant difference in serum iron concentration between males (88.0± 44.3 µg/dl) and females (84.4± 51.7 µg/dl) in group B. The serum ferritin values in males (63.6 ± 49.0 ng/ml) and females (59.8 ± 33.9 ng/ml) of group B were significantly not different ($p > 0.05$; Table 3).

No significant difference in serum iron concentration between males (112.1± 38.8 µg/dl) and females (106.9± 62.6 µg/dl) was found in group C. The serum ferritin values in males (62.4 ± 22.7 ng/ml) and females (59.3 ± 33.2 ng/ml) of group C were significantly not different ($p > 0.05$; Table 4).

The difference in serum iron concentration (Fig. 1) between groups A (102.04 ± 87.81 µg/dl), group B (104.91 ± 34.93 µg/dl), and group C (108.93 ± 52.13 µg/dl) was not significant ($p > 0.05$). The serum ferritin values in group A (65.13 ± 48.03 ng/ml), group B (60.55 ± 42.07 ng/ml), and group C (68.96 ± 18.44 ng/ml) were significantly not different ($p > 0.05$; Fig. 2).

Table No.1: Age distribution of subjects in groups A, B and C

Parameter	Group A (n-30) (H.pylori +ve patients)	Group B (n-30) (H.pylori -ve patients)	Group C (n-30) (Healthy control)
Age (Years)	32.9 ± 12.3	33.3 ± 11.6	33.6 ± 11.1

The values are as Mean ± SD and statistically non significant

Table No.2 Comparison of serum ferritin and serum iron between males and females in group A

Parameter	**Male** (n =18)	**Female** (n = 12)	**p value**
Serum ferritin (ng/ml)	64.4 ± 55.3	63.8 ± 36.6	0.92*
Serum iron (µg/dl)	116.1± 87.8	95.9± 58.8	0.27*

The values are as Mean ± SD

* The values are statistically non significant

Table No.3: Comparison of serum ferritin and serum iron between males and females in group B

Parameter	**Male** (n =16)	**Female** (n = 14)	**p value**
Serum ferritin (ng/ml)	63.6 ± 49.0	59.8 ± 33.9	0.67*
Serum iron (µg/dl)	88.0± 44.3	84.4± 51.7	0.71*

The values are as Mean ± SD

* The values are statistically non significant

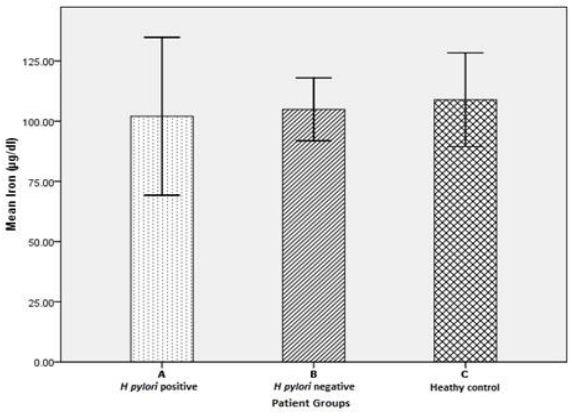

Figure No.1: Serum iron in groups A, B and C

Table No.4: Comparison of serum ferritin and serum iron between males and females in group C

Parameter	Male (n =14)	Female (n = 16)	p value
Serum ferritin (ng/ml)	62.4 ± 22.7	59.3 ± 33.2	0.47*
Serum iron (µg/dl)	112.1± 38.8	106.9± 62.6	0.82*

The values are as Mean ± SD

* The values are statistically non significant

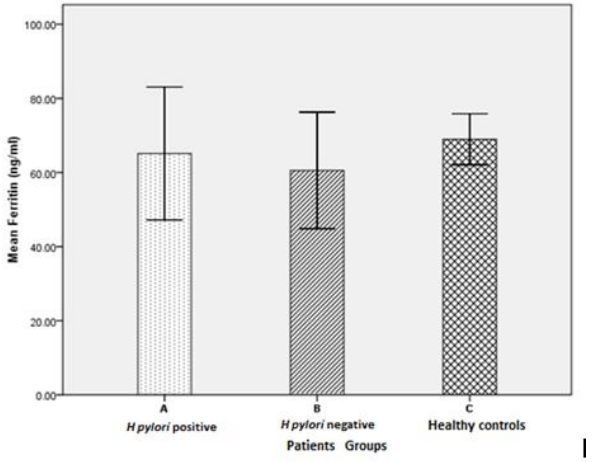

Figure No..2 Serum ferritin in groups A, B and C

DISCUSSION

Literature review shows controversial picture about the association of Helicobacter pylori infection and iron deficiency anemia. A study carried out in the United States has revealed the association of Helicobacter pylori infection with iron deficiency both in the presence or absence of peptic ulcer disease. [14] It has been also reported that iron deficiency anemia in patients with asymptomatic gastritis was corrected successfully when they were given eradication therapy for Helicobacter pylori. [15] A study carried out in Turkish subjects also showed association of iron deficiency with Helicobacter pylori infection. [16] Another study conducted in Korean children had documented decreased serum ferritin levels in patients with Helicobacter pylori infection. [17] Low hemoglobin, ferritin, and B_{12} levels with Helicobacter pylori infection also have been reported in Pakistani population. [18]

While on the other hand, no anemia or iron deficiency was revealed in Helicobacter pylori infected patients. [19] In the same way, a large sample survey conducted in Denmark revealed only iron deficiency but no effect on the hemoglobin; mean corpuscular volume that could indicate iron deficiency anemia.[20] All the parameters of iron deficiency anemia like hemoglobin and mean corpuscular volume showed improvement after eradication therapy for Helicobacter pylori infection except the serum ferritin levels.[21] Asymptomatic Helicobacter pylori infection was not found to be associated with anemia or iron deficiency. [22] No significant difference was observed in hemodialysis patients with or without Helicobacter pylori infection regarding iron deficiency anemia. [23] Our findings coincide with recently published studies. This study did not show any association of Helicobacter pylori infection with ferritin, transferrin receptors, and hemoglobin in these Latin American countries. [24] In a low socioeconomic country like Bangladesh, Helicobacter pylori infection was not proved as a cause of neither iron deficiency anemia nor resulted in failure to iron therapy or supplementation in young children.[25] In older subjects living either at home or in nursing homes, no association was observed between Helicobacter pylori infection and low serum iron, ferritin, transferrin, vitamin B_{12} level.[26] Similarly, no association was observed between Helicobacter pylori infection and hemoglobin level in Helicobacter pylori positive and negative groups.[27] In children of Alaska, no difference was found between the patients and control subjects and eradication therapy for Helicobacter pylori infection failed to resolve iron deficiency anemia and isolated iron deficiency in a subset of 219 children.[28]

CONCLUSION

This study concludes that Helicobacter pylori has no significant effect on serum iron, and serum ferritin levels.

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Address for Corresponding Author:
Dr. Saqib Sohail
Assistant Professor of Physiology
Punjab Medical College, Faisalabad
Cell No. 0300-4357314