THE RELATION BETWEEN HELICOBACTER PYLORI SEROPOSITIVITY AND IRON DEFICIENCY ANEMIA IN IRAQ-KURDISTAN-SULAIMANI CITY

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ABSTRACT

Background

Iron deficiency is the most common cause of anaemia worldwide and is seen in general practice. The cause of iron deficiency anemia changes in different age groups, whether due to blood loss, gastrointestinal causes or increase in body demand for iron.

Objectives

This study was designed to determine any relation between *Helicobacter pylori* positive subjects serologically in Sulaymaniyah city/Iraqi Kurdistan with Iron deficiency anemia.

Patients and Methods

The current study enrolled one hundred twenty female volunteers (eighty female with positive H. *pylori* serology, either both or any one of IgG & IgM positive) and forty subjects with negative serology. Then, contributors divided to four groups according to the age. *H. pylori* was detected serologically by using anti-*H. pylori* IgG and IgM antibodies ELISA kit.

Results

Hemoglobin level, mean corpuscular volume, white blood cells, platelets counts, serum ferritin and total iron binding capacity, were measured. Hemoglobin level and mean corpuscular volume in all patients considerably decreased ($P < 0.01$) compared with all control groups. While, the outcome of white blood cells and platelets counts demonstrated no considerable changes in all patient groups compare with all control groups. Serum ferritin in all patient categories significantly reduced ($P < 0.01$) compared with all control groups. While, TIBC levels significantly raised in patient groups compare with control groups.

Conclusion

There is a relation between *Helicobacter pylori* positive serology and iron deficiency anemia in Kurdistan-Sulaimania city.

Keywords: *Iron deficiency anemia, H.pylori, Hiwa Hematology Hospital, Sulaimani.*

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INTRODUCTION

Iron deficiency is the most common cause of anemia globally and is seen in medical practice. Iron deficiency anemia (IDA) is caused by deranged synthesis of hemoglobin, resulting in red cells that are include reduced amounts of hemoglobin (hypochromic) and smaller than normal (microcytic) [1].

The health effects of reduced body iron include anemia, underactive physical activity and reduced intellectual wellbeing as well as functional alterations of the small bowel [2]. Iron deficiency evolves through three stages: iron depletion, IDA and iron deficient erythropoiesis. Although the mechanisms leftover unclear, epidemiologic and clinical researches suggest that infection with *Helicobacter pylor*i (*H. pylori*) is associated with defect of iron and IDA [3].

Helicobacter pylori were first separated in 1983, a gram-negative bacterium discovered on the luminal surface of the gastric epithelium [4]. *H. pylori* infection is in charge for many gut disorders, including atrophic gastritis, gastro duodenal ulcer, intestinal metaplasia, gastric adenocarcinoma and gastric mucosa associated lymphoid tissue lymphoma (MALT lymphoma) [5]. *H. pylori* have been responsible in some extra-gastric diseases, eg. IDA and vitamin B12 deficiency [6-7]. This study was designed to found any connection between *H. pylori* positive serology and IDA in Slulaimani city/ Iraqi Kurdistan.

MATERIALS AND METHODS

One hundred twenty female subjects were recruited in this study, eighty female patients randomly who referred to hematology outpatient clinic in Hiwa Hemato-Oncology Hospital in Sulaimani between July, 2015 to December, 2015 with forty control Subjects. Inclusion criteria includes new iron deficient patients, between 10 -50 years of age while exclusion criteria are those already on iron tablet, heavy menstruating, pregnant and lactating ladies. The informed consent was taken from all patients. *H. pylori* test was performed in all patients. *H. pylori* was diagnosed serologically by using anti-*H. pylori* IgG and anti-*H. pylori* IgM antibodies ELISA kit (CHROMATE, Germany).

Hematological analysis

Two ml whole blood was collected into Ethylene diaminetetraacetic acid (EDTA) tubes and analyzed on using an automated hematological analyzer (Cell Dyn 1800, Abbott, USA). The analyzer aspirates the blood, dilutes and counts white blood cells, red blood cells and platelets, measures Mean Cell Volume (MCV) and Haemoglobin (Hb) .

Serological analysis

Five milliliters (ml) venous blood was obtained from the participants. All blood samples were dispensed into dry glass test tubes for clotting and retraction to happen. Sera were obtained after samples were centrifuged at 2000 g for five minutes and stored at -20°C until assayed for laboratory investigations. Levels of serum total iron binding capacity (TIBC) were measured using Accent 200(Germany) and serum ferritin were measured using Cobas device E411 (Germany).

Statistical analysis

Data were analyzed using a Minitab program under SPSS and Microsoft Excel program. The data were presented in simple measure of mean ± SD (standard deviation), minimum and maximum values. Results were analyzed statistically using Analysis of Variance (ANOVA) test, in order to evaluate the significance of variability between treated and control groups. Means of data were compared using Duncan's Multiple Range test. Probability levels of more than 0.01 were regarded as non-significant, while values less than 0.01 were considered as significant.

RESULTS

Hematological analysis

One hundred twenty female subjects recruited for this study, eighty of them with *H. pylori* positive serology with forty control group, all participants were divided to four groups according their age Table 1. The results of Hb-levels in all patient groups significantly decreased compared with all control groups as show in Table 2. Also, the results of MCV-levels in all patient groups significantly reduced compared with all control groups as in Table 2. The Hb and MCV levels in IgG groups significantly reduced in comparsion with IgM groups in all ages categories as in Table 2. While, the results of white blood cells and platelets showed no significant variation in all patient groups compare with all control groups as show in Table 3.

Serological analysis

The outcome of S. ferritin levels in all patient groups

significantly reduced compared with all control groups as in Table 4. On the other hand, the TIBC-levels in all patient groups significantly increased compared with all control groups as in Table 4. There are non-significant changes in the levels of S. ferritin and TIBC between IgG and IgM patient groups as in Table 4.

Table 1. The tests of anti-*H. pylori* in all cases.

Age	H. pylori (+ve) %(No.)	H. pylori (-ve) %(No.)
10-19	13.3 (16)	8.3 (10)
20-29	16.7 (20)	8.3 (10)
30-39	20.8 (25)	8.3 (10)
40-50	15.8 (19)	8.3 (10)

Table 2. The relation between *H. pylori* seropositivity and Hb g/l and MCV.

Age	Hb-levels			MCV-levels		
	H. pylori (IgM)	H. pylori (IgG)	Control	H. pylori (IgM)	H. pylori (IgG)	Control
10-19	11.87 ± 0.15	11.1 ± 0.36	12.85 ± 0.29	64 ± 5	57.7 ± 2.5	76 ± 2.5
20-29	10.1 ± 0.27	9.3 ± 0.21	13.76 ± 0.37	69 ± 6	58.3 ± 1.5	80.8 ± 3
30-39	11.3 ± 0.3	10.67 ± 0.15	13.74 ± 0.44	65.5 ± 3.1	55.8 ± 3.8	80.7 ± 3
40-50	9.8 ± 0.65	9.4 ± 0.6	12.58 ± 0.4	60.5 ± 5.3	57 ± 3.1	74.4 ± 3.7

Table 3. The relation between *H-pylori* seropositivity and WBC ($X10^3$) and platelets ($X10^4$).

Age (year	WBC-levels ($X10^3$)			Platelets levels ($X10^4$)		
	H. pylori (IgM)	H. pylori (IgG)	Control	H. pylori (IgM)	H. pylori (IgG)	Control
10-19	7.1 ± 0.78	6.8 ± 0.46	6.93 ± 0.5	24.7 ± 2.2	25.3 ± 2.1	24.9 ± 3.6
20-29	8.3 ± 0.64	7.7 ± 0.31	7.8 ± 0.55	26.6 ± 2.6	24.7 ± 2.3	26.4 ± 0.87
30-39	7 ± 0.4	6.7 ± 0.41	7.08 ± 0.28	30.8 ± 3.7	29.6 ± 6	29.9 ± 3.8
40-50	7.3 ± 0.43	7.6 ± 0.17	73.58 ± 0.5	27.5 ± 5.2	26.3 ± 5	27.4 ± 2.1

Table 4. The relation between *H. pylori* seropositivity and S. ferritin and TIBC.

Age	S.ferritin levels			TIBC-levels		
	H. pylori (IgM)	H. pylori (IgG)	Control	H. Pylori (IgM)	H. pylori (IgG)	Control
10-19	2.6 ± 0.67	2.3 ± 0.3	11.8 ± 1.71	442 ± 29.7	488 ± 45.7	319.7 ± 14.4
20-29	3.4 ± 0.45	3.5 ± 0.8	11.4 ± 0.93	493.8 ± 41.5	518 ± 21.4	415.4 ± 11.1
30-39	5.7 ± 0.58	6.1 ± 0.35	13.3 ± 0.96	482.8 ± 60	470.8 ± 34.5	402.5 ± 45.7
40-50	3.3 ± 0.57	3.1 ± 0.55	11.5 ± 1.29	460.3 ± 39.6	471.3 ± 52.2	427.5 ± 9.6

DISCUSSION

Helicobacter pylori nfection is a repeated cause of iron-refractory or iron-dependent anaemia in patients [8]. In the current study, the levels of Hb and MCV significantly reduced in patient classes compare with control groups in all ages. While, the levels of white blood cells and platelets counts showed no significant changes in patient and control groups. While, the levels of S. ferritin and TIBC showed significant decrease and increase, respectively in all patient groups compare with control groups in all ages. As In Jasem et al. study at 2011, they discovered significant decrease in the levels of S. ferritin in patients who infected with *H. pylori*, they suggest the reason of decrease were due to the gastric ulcer that caused by *H. pylori* [9]. Also, Annibale et al. 1999 referred that *H. pylori* causes iron-deficiency anemia, they suggest *H. pylori* could lead to IDA by decreasing iron uptake or increasing the demand for iron [10] that is in parallel with the results of our study.

In study of Darvishi et al. 2015, they saw significant reduction in the levels of S.ferritin and hemoglobin in patients who infected with *H. pylori* and significant increase in TIBC-levels compare with control groups [11]. Takezako et al. 2013 referred that *H. pylori* lead to IDA; they found the levels of white blood cells and platelets showed no significant changes between patients and control groups that are in agreement with results of the current study [12]. On other hand, they saw the levels of Hb and MCV showed no significant changes between patients and control groups, that are contrary with the results of our study. In the study of AL-Kazazz 2013, did not find any link between *H. pylori* infection and serum ferritin and TIBC levels. Indeed, there was no significant connection between *H. pylori* infection and patients with iron deficiency and control group. The mechanism by which *H. pylori* induces the change in the iron stores is not understood, but it seems to involve several pathways, including gastrointestinal blood loss, enhanced uptake of the iron by the bacterium and decrease absorption of dietary iron. IDA is often caused by gastrointestinal bleeding due to peptic ulcer.

Helicobacter pylori infection, which has been obvious to play the role in peptic ulcer, has been blamed as a cause of IDA refractory to oral iron treatment [13]. Tari et al. 2016 referred that *H. pylori* infection cause a reduction in serum ferritin and rise in TIBC levels, they stated that significant changes in levels of serum ferritin (decreased) and TIBC (rise) in IgG groups and IgM groups. They suggest the relationship; therefore it appears important to assess the digestive tract for the treatment of patients with IDA [14], although serology is not an excellent way for detection of real *H. pylori* infection of GI-tract if compared to culture, histopathology, urea breath test, or stool *H. pylori* antigen test, but our paper showed that still a large number of *H. pylori* serology positive patients have IDA whom they need treatment of their anemia with possible eradication of their *H. pylori* infection, a part from effect of this bacterial infection on GIT tract.

In conclusion, there is a connection between *H. pylori* infection and IDA in Kurdistan- Sulaymaniyah city. Which probably makes concomitant treatment of iron deficiency anemia and *H. pylori* eradication a possible suitable option especially in patients without other sources of obvious blood loss?

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