Helicobacter pylori infection as a risk factor for changes in some hematological and biochemical parameters among Egyptian population

Mohamed Gamal Abdrabou[1], Emad Rashad badr[2], Hosam Eldin Abouelkheir[1], Abeer Elhadidi[3].

[1]Alexandria University, Faculty of Medicine, Department of Tropical Medicine, Alexandria, Egypt. [2]Damanhour fever hospital, Elbeheira, Egypt. [3]Alexandria University, Faculty of Medicine, Department of chemical and clinical pathology, Alexandria, Egypt.

Corresponding author: Mohamed Gamal Abdrabou, Alexandria University, Faculty of Medicine, Department of Tropical Medicine, Alexandria, Egypt.

Mail address: mg_theking2010@yahoo.com

Telephone number: +201016780167

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Abstract

Background: *Helicobacter pylori* can be an etiology of chronic gastritis, peptic ulcer, and stomach cancer. The aim was to evaluate the relationship between *H. pylori* infection and alterations in some hematological and biochemical parameters among the Egyptian people. **Method:** Ninety individuals with confirmed *H. pylori* infection by the fecal antigen were selected and then followed after a month of treatment with Levofloxacin, amoxicillin, and a proton pump inhibitor; they underwent testing again for hematological and biochemical

parameters after treatment of *H.pylori*. **Results:** both groups, group A (Before treatment) and Group B (After 1 month of therapy), showed a statistically significant relation between the presence of *H. pylori* and the CBC values; patients with pre-existing dyslipidemia exhibited a link between *H. pylori* infection and reduced levels of HDL-C as well as elevated LDL-C and triglycerides. **Conclusion**: *H. pylori* is associated with changes in some hematological and biochemical parameters.

Keywords: *Helicobacter pylori, Hematological, dyslipidemia, Biochemical, parameters, levofloxacin, guidelines, platelets, thrombocytopenia, regimen*

Introduction

H. pylori affects millions of people [1] and may cause gastric cancer [2]. It is known to affect half of the world's population. A gram-negative bacillus causes chronic gastritis and peptic ulcer disease [3]. *Helicobacter pylori* causes 89% of duodenal ulcers in Egypt. Few studies show the effects of *H. pylori* eradication on some hematological and other biochemical parameters in the Egyptian population [4].

Some hematological parameters, such as CBC, have prognostic values regarding Helicobacter pylori infection [5]. Furthermore, some biochemical parameters can be affected: aspartate transaminase, alanine aminotransferase, alkaline phosphatase, total, direct bilirubin, and serum albumin. Some studies proved hemoglobin and hematocrit levels usually decrease due to gastrointestinal blood loss [6,7]. Epidemiological and clinical research estimated that 75% of Egyptians have a high prevalence of *Helicobacter pylori* [8]. In our study, *H. pylori* infection was considered a risk factor for changes in some biochemical and hematological parameters [4].

The present work aimed to assess some biochemical and hematological changes associated with *H.pylori* infection.

Methods

The study was a prospective cohort study that included 90 Egyptian symptomatic individuals positive for *H. pylori* who suffered from dyspepsia, anorexia, nausea, or vomiting. Patients gave informed consent and were divided into groups: Group A (Before treatment) and Group B (the same patients as Group A but after 1 month of therapy). All patients were subjected to a levofloxacin-based treatment regimen.

The study included laboratory investigations: CBC, iron profile, liver enzymes, lipid profile, *H. pylori* fecal antigen, and ultrasound of the abdomen and pelvis. **Exclusion criteria:** We excluded all other causes of disturbed hematological parameters, elevated liver enzymes, dyslipidemia, and hyperbilirubinemia.

Complete blood count was analyzed using Sysmex's XN-Series hematology analyzers (dimension Exl 200 Siemens). Serum iron was measured using 1 (R1) and 2 (R2) reagents. ALT and AST are kinetic assays that were measured by quantifying the rate at which the absorbance at 340 nm decreases due to NADH oxidation. The lipid profile was calculated using this assay. The patients were fasting for 12 hours; Cholesterol esters were hydrolyzed to form cholesterol and free fatty acids, followed by the oxidation reaction; those biochemical reactions were done using dimension Exl 200 Siemens.

Elisa processed *H. pylori* fecal antigen tests and purified H pylori antigens in stool. The *H. pylori* antibody is coated on the surface of microwells. If present, a stool sample is added to wells, and the *H. pylori* antigens bind to the antibody. Using HpSa enzyme immunoassay test kit (**REF 10224 LOT 23-D5-276**)

Results

1. Demographic data of the studied groups:

The study included patients aged 20-65, with a mean of 37 years, 39 males (43.3%) and 51 females (56.7%). (Table 1)

Tab 1. Demographic data of the studied patients

Demographic data	**Patients (n=90)**	
	No.	**%**
Gender		
Males	39	43.3%
Females	51	56.7%

Age (years)	
Min – Max	20 – 65
Median (IQR)	37 (30 – 44)

2**. Comparison of both Groups as regards symptoms:**

There was a statistically significant difference in symptoms between group A and group B, with group A experiencing higher levels of dyspepsia, anorexia, nausea, and vomiting **(**Tab 2).

Tab 2. Comparison of symptoms in both groups.

	Group A (n=90)		**Group B (n=90)**		**P-value**
	No.	**%**	**No.**	**%**	
Dyspepsia	54	60%	14	15.6%	**<0.001***
Anorexia	16	17.8%	4	4.4%	**0.004***
Nausea	11	12.2%	0	0%	**< 0.001***
Vomiting	21	23.3%	4	4.4%	**< 0.001***

*X^2: Chi-square test. *: Statistically significant at p < 0.05. Group A: before treatment. Group B: the same patients of group A but after 1 month of treatment.*

3. Comparison of both groups regarding CBC, iron, lipid profile, liver enzymes, and bilirubin:

There was no statistically significant difference in RBCs and WBCs was found (P>0.05), while there was a significant difference in platelet count between both groups (P 0.001) (Tab 3).

There was a statistically significant increase in serum iron levels in group B compared to group A (P= 0.01). However, the two groups had no significant difference in serum ferritin levels (P>0.05). Both groups' mean serum iron levels ranged from 0.19 - 1.42 mcg/dL and 0.16 - 1.72 mcg/dL, respectively (Tab 3).

The total cholesterol in both groups is group A (116-316 mmol/L) and group B (116-259 mmol/L). Neither group had a statistically significant difference in total cholesterol (P>0.05). The HDL values were comparable between the groups, ranging from 24 to 82 mmol/L and 23

to 81 mmol/L, with a mean of 44 mmol/L. The LDL values were comparable amongst the groups, ranging from 49 to 207 mmol/L and 52 to 173 mmol/L, with a mean of 112 mmol/L. There was no statistically significant disparity in LDL levels in both groups (P>0.05). Group B reduced triglycerides compared to group A (P<0.001) (Tab 3).

There was no statistically significant disparity in the levels of AST, ALT, Total or direct bilirubin between groups A and B. ALT ranged from 8-96 U/L with mean of 21 U/L, while in group B 10-82 U/L with mean of 21 U/L, while the AST ranged from 10-44 U/L in group A and 11-41 U/L in group B with mean of 19, 20 U/L mg/dL respectively. Total bilirubin ranged from 0.25-2.7 mg/dl in group A to 0.46-2.1 mg/dl in group B, whereas the range of direct bilirubin levels varied from 0.09-0.7 mg/dl in group A to 0.11-0.19 mg/dl in group B, there was no notable difference between both groups regarding total and direct bilirubin (Tab 3).

Tab 3. Comparison of CBC, iron profile, lipid profile, liver enzymes, and bilirubin.

Variable	Group A (n=90)	Group B (n=90)	P-value
RBCs			
Min – Max	**3.2 – 6.04**	**3.5 – 6.61**	0.78
Median (IQR)	4.7 (4.3 – 5.2)	4.7 (4.4 – 5.2)	
Hb			
Min – Max	8.5 – 17	9.5 – 16.8	0.65
Median (IQR)	12.9 (11.4 – 14.3)	12.9 (11.3 – 14.4)	
MCV			
Min – Max	59.1 – 103.8	57.9 – 99.5	0.05
Median (IQR)	84 (80 – 88)	85 (79 – 89)	
MCH			
Min – Max	15.9 – 34.6	16.3 – 32.3	0.053
Median (IQR)	27.7 (25.7 – 29.4)	27.8 (25.9 – 29.6)	
Platelets			
Min – Max	90 – 526	150 - 480	**0.001***
Median (IQR)	268 (225 – 317)	254 (224 – 299)	
WBCs			
Min – Max	3.5 – 13.97	3.64 – 11.58	0.05
Median (IQR)	6.9 (5.6 – 8.5)	7.1 (5.5 – 7.9)	
Iron			
Min - Max	0.19 – 1.42	0.16 – 1.72	**0.01***
Median (IQR)	0.57 (0.35 – 0.83)	0.67 (0.46 – 0.9)	
Ferritin			
Min - Max	4.8 - 354	5 - 474	0.13
Median (IQR)	83 (23 – 143)	69 (35 – 145)	
Lipid profile			
Total cholesterol			
Min – Max	116 – 316	116 – 259	0.56
Median (IQR)	180 (156 – 208)	176 (159 – 198)	
HDL			
Min – Max	24 – 82	23 – 81	0.06
Median (IQR)	42 (35 – 49)	44 (37 – 52)	

LDL			
Min – Max	49 – 207	52 – 173	0.45
Median (IQR)	113 (86 – 132)	112 (95 – 133)	
TGs			
Min – Max	46 - 485	46 - 423	**<0.001***
Median (IQR)	135 (96 – 172)	120 (85 – 145)	
ALT			
Min – Max	8 – 96	10 – 82	0.75
Median (IQR)	21 (16 – 27)	21 (15 – 31)	
AST			
Min – Max	10 – 44	11 – 41	0.1
Median (IQR)	19 (16 – 25)	20 (17 – 26)	
Total Bilirubin			
Min – Max	0.25 – 2.7	0.29 – 2.1	0.16
Median (IQR)	0.46 (0.38 – 0.64)	0.51 (0.43 – 0.69)	
Direct Bilirubin			
Min - Max	0.09-0.7	0.11 – 0.19	0.18
Median (IQR)	0.19(0.13-0.23)	0.2 (0.15 – 0.25)	

**: Statistically significant at p < 0.05. Group A: before treatment. Group B: after treatment*.

4. Comparison of both groups as regards *H. pylori* fecal Antigen:

Regarding the *H. pylori* fecal antigen, the values ranged from 21 to 155 in group A, with a median of 54. In group B, the values ranged from 4 to 86, with a median of 13. There was a statistically significant decrease in *H. pylori* fecal antigen in Group B compared to Group A, with a high level of statistical significance; the Eradication rate of H. pylori infection after treatment was 77.8 %, and only 20 cases out of 90 were still positive for H. Pylori fecal antigen (70 cases were cured after treatment) (P<0.001). (Table 4)

Tab 4. Comparison of both groups as regards H. pylori fecal Antigen.

	Group A (n=90)	**Group B (n=90)**	**P-value**
***H. pylori* fecal Ag**			
Min – Max	21 – 155	4 - 86	**<0.001 ***
Median (IQR)	54 (36 – 75)	13 (11 – 14)	

**: Statistically significant at p < 0.05. Group A: before treatment. Group B: after treatment.*

5. Comparison of (subgroups of patients) with abnormalities in CBC, Iron profile, dyslipidemia, and liver enzymes in both groups:

There was no significant difference in RBCs between both groups. However, in terms of hemoglobin levels, group B had a significantly higher Hb level than group A in patients with

anemia. In terms of MCV, group B had a considerably higher MCV than group A in patients with decreased MCV. Regarding (MCH), group B had a significantly higher MCH than group A in patients with decreased MCH. Regarding WBCs, group B had a decreased considerably WBC level compared to group A in patients with leukocytosis (Tab 5).

The study revealed no statistically significant difference in iron levels between groups A and B among patients with iron deficiency (P>0.05). Nevertheless, patients with low ferritin in group B exhibited a significant rise in ferritin compared to group A (P= 0.003). The median iron levels in group A ranged from 0.35 mcg/dL to 1.42 mcg/dL, while in group B, they ranged from 0.31 mcg/dL to 1.72 mcg/dL (Tab 5).

There was a notable reduction in total cholesterol among patients diagnosed with hypercholesterolemia in group B compared to group A (P= 0.001). Group B exhibited a statistically significant elevation in HDL compared to group A (P= 0.006). Group B exhibited a statistically significant reduction in LDL levels compared to group A (P=0.001). Group B significantly reduced TGs compared to group A (P< 0.001). Group A had a median total cholesterol level of 224 mmol/L, whereas Group B had a median total cholesterol level of 201 mmol/L. Regarding LDL levels, group B exhibited a median value of 136 mmol/L, whereas group A displayed a median value of 168 mmol/L. Group B had a notable decrease in median TGs, with a value of 148 mmol/L, compared to group A (Tab 5).

Patients with increased ALT had significantly decreased ALT in group B compared to group A. Total bilirubin was similar between groups A and B, with no significant difference. Direct bilirubin levels were significantly decreased in group B compared to group A in patients with increased direct bilirubin. The study also found no significant difference in total bilirubin levels between groups of patients with hyperbilirubinemia (Tab 5).

Tab 5. Comparison of (subgroups of patients) with abnormalities in CBC, Iron profile, dyslipidemia, liver enzymes, and bilirubin in both groups.

Variable	Group A	Group B	P-value
Erythrocytosis (N=5)			
Min – Max	4.81 – 6.04	4.47 – 6.2	0.3
Median (IQR)	5.5 (4.9 – 5.8)	5.5 (4.9 – 5.7)	
Erythrocytopenia (N=5)			
Min – Max	3.2 – 4.21	3.5 – 4.28	**0.04***
Median (IQR)	3.7 (3.4 – 4)	4.1 (3.8 – 4.3)	
Hb in patients with anemia (N= 29)			
Min – Max	8.5 – 12.7	9.5 – 12.9	**0.014***
Median (IQR)	10.6 (10.1 – 11.4)	10.9 (10.4 – 11.5)	
MCV in patients with decreased MCV(N=35)			**0.007***
Min – Max	59.1 – 82.4	57.9 – 87.4	
Median (IQR)	76.6 (73 – 80.7)	77.9 (74.5 – 81.4)	
MCH in patients with decreased MCH (N=34)			**0.003***
Min – Max	15.9 – 26.9	16.3 – 27.4	
Median (IQR)	25.15 (23.1 – 26.2)	25.2 (22.63 – 26.8)	
WBCs in patients with leukocytosis (N= 10)			
Min – Max	10.09 – 13.97	6.4 – 11.58	**0.005***
Median (IQR)	12.1 (11.1 – 12.8)	9 (7.6 – 9.6)	
Iron in patients with iron deficiency (N= 21)			
Min – Max	0.35 – 1.42	0.31 – 1.72	0.28
Median (IQR)	0.68 (0.52 – 0.89)	0.76 (0.52 – 0.95)	
Ferritin in patients with decreased ferritin(N=19)			
Min – Max	4.8 – 23.4	5 – 46.4	0.014 *
Median (IQR)	8.1 (5.3 – 12)	11 (7.2 – 15)	
T. cholesterol in patients with hypercholesterolemia (N=25)			
Min – Max	205 – 316	133 – 259	0.001 *
Median (IQR)	224 (212 – 247)	201 (190 – 222)	
HDL in patients with decreased HDL (N= 21)			
Min – Max	24 – 34	23 – 43	0.006 *
Median (IQR)	32 (28 – 33)	33 (32 – 39)	
LDL in patients with increased LDL (N= 17)			
Min – Max	141 – 207	87 – 171	0.002 *
Median (IQR)	168 (153 – 193)	136 (118 – 154)	
TGs in patients with increased TGs (N= 35)			
Min - Max	151 – 485	64 - 423	<0.001 *
Median (IQR)	190 (166 – 260)	148 (125 – 188)	
ALT in patients with elevated ALT (N=7)			
Min - Max	**33 – 96**	11 – 82	**0.017 ***
Median (IQR)	**50 (39 – 61)**	41 (21 – 56)	
T. bilirubin in patients with hyperbilirubinemia (N=6)			**0.92**
Min - Max	1.09 – 2.7	0.85 – 2.1	
Median (IQR)	1.67 (1.12 – 2.1)	1.94 (1.07 – 2.1)	
D. bilirubin in patients with D. hyperbilirubinemia (N= 20)			**0.025 ***
Min - Max	0.26 – 0.7	0.11 – 0.8	
Median (IQR)	0.35 (0.29 – 0.48)	0.28 (0.2 – 0.36)	

**: Statistically significant at p < 0.05. Group A: before treatment. Group B: after treatment.*

6. Correlation study between *H. Pylori* fecal Ag and CBC in group B:

No statistically significant correlation between *H. pylori* fecal Ag and CBC, iron profile, lipids profile (except HDL), ALT, AST, total and direct bilirubin in group B (P> 0.05) (Table 6), meanwhile A statistically significant positive correlation (r= 0.579) between *H. pylori* fecal

Ag & HDL in group B, a detrimental association was observed between H. pylori fecal antigen and high-density lipoprotein (HDL) levels in individuals with reduced HDL. (P= 0.011) (Tab 6) (Fig 1).

Tab 6. In group B, a correlation study was conducted between H. Pylori fecal Ag and CBC, iron profile, lipids profile, ALT, AST, and total and direct bilirubin.

Group B	r	P-Value	N
Hb	-0.072	0.499	90
RBCs	-0.124	0.245	90
MCV	0.093	0.384	90
MCH	0.088	0.408	90
PLTs	0.025	0.816	90
WBCs	0.017	0.87	90
Iron	-0.039	0.713	90
Ferritin	0.065	0.54	90
Total cholesterol	0.059	0.579	90
HDL	-0.265	0.011*	90
LDL	0.140	0.188	90
TG	0.180	0.089	90
SGPT	-0.009	0.931	90
SGOT	-0.066	0.535	90
Total bilirubin	-0.119	0.264	90
Direct Bilirubin	-0.148	0.164	90

*r: Pearson correlation coefficient. *: Statistically significant at p < 0.05. Group B: after treatment.*

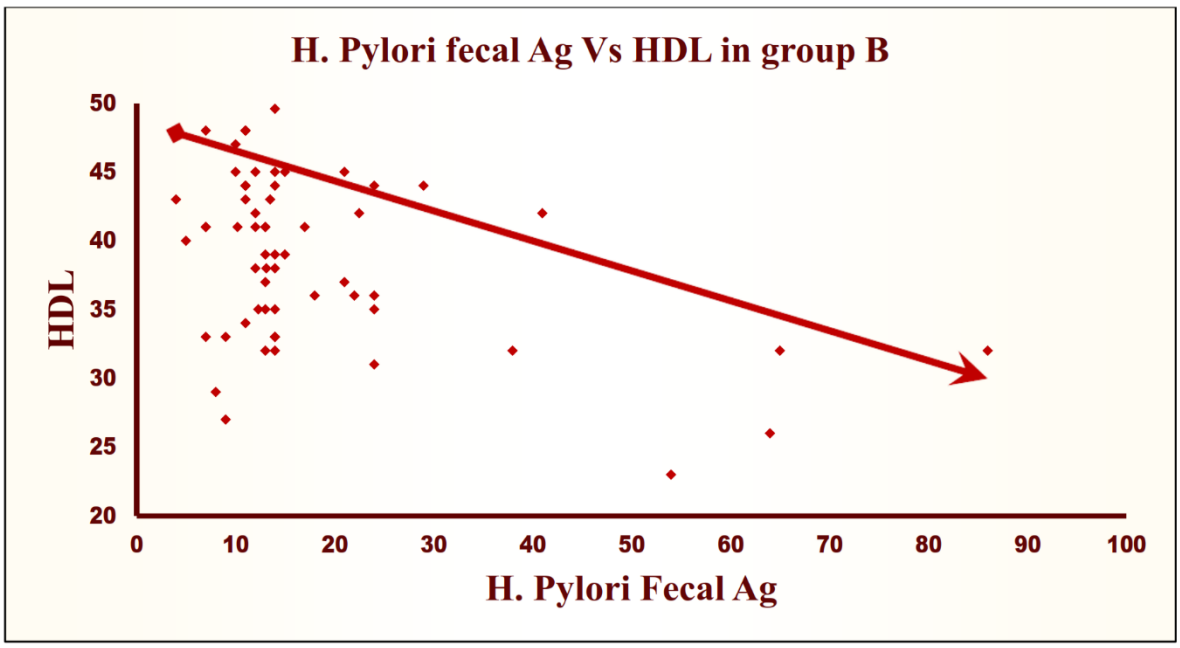

Fig 1. H. Pylori fecal Ag Vs. HDL in group B.

Discussion

Our study was to evaluate the relationship between *H. pylori* infection and alterations in some hematological and biochemical markers among the Egyptian population. During *H. pylori* infection, individuals experience upper abdominal symptoms such as pain, nausea, and vomiting [9.10].

Kim's 2014 study found that symptoms of acute gastroenteritis, nausea, vomiting, epigastric pain, and heartburn occur due to *H. pylori* affection of the stomach. That was consistent with the results of our study as most of the patients included were suffering from those symptoms, as seen in the symptoms table in the results section. [11]

In contrast to our findings, the bulk of guidelines usually recommend triple therapy with levofloxacin as a second-line treatment. A recent study conducted in the United States has shown a significant prevalence of levofloxacin resistance, with a rate of 37.6%. As a result, levofloxacin triple therapy is not recommended as a first treatment option. As an initial therapy,

this regimen showed reasonable eradication rates, ranging from 79.1% to 85.2% [10]. *H. pylori* antibiotic resistance rates were as low as levofloxacin, 20% in Egypt, in another study by Mohamed et al. [12].

Rahmati A. et al. (2022) conducted a study that revealed that eliminating H. pylori has been linked to a rise in platelet count in patients with immune thrombocytopenia. The study included a group of 1907 patients who had either had an *H. pylori* blood antibody test (n=1546) or underwent an upper gastrointestinal endoscopy (n=361). The platelet count, indices, and other blood cell parameters were analyzed in individuals who tested positive and negative for *H. pylori*. There was no significant difference in platelet count and indices between those who tested positive and negative for *H. pylori* ($P > 0.05$) [13].

In a study by Haeri M. et al. (2018), 66.5% of the cases tested positive for *H. pylori.* Male individuals afflicted with *H. pylori* infection exhibited elevated levels of LDL compared to those who did not have *H. pylori*. In addition, those with *H. pylori* infection exhibited elevated triglyceride levels and reduced HDL levels. Nevertheless, there was no statistically significant disparity in HDL and TG between the cases with and without *H. pylori* infection [14].

Limitations of the study: Our limitations were the short follow-up period, small sample size, and single-center study.

Conclusion

Finally, treatment has reduced the high prevalence of gastrointestinal symptoms associated with *H. pylori* infections. There was evidence of a correlation between the *H. pylori* fecal antigen and the CBC values; nonetheless, dyslipidemia may be related to *H. pylori* infection. This correlation necessitates further research to confirm and clarify the processes at

work. Our research indicates that for those who test positive for *H. pylori*, we advise conducting lipid profiles, hematological parameter testing, and long-term community-based studies.

Footnotes.

Mohamed Emara (Professor of gastroenterology, hepatology, and infectious diseases), Marwa Shabana (Assistant professor of clinical pathology), and Amany Mohamed (Assistant professor of family medicine) were the peer reviewers.

E- Editor: Salem Youssef Mohamed, Osama Ahmed Khalil, Amany Mohammed.

Ethical approval:

Written informed consent was obtained from the patient after the studies were well explained before data collection. The hospital's Research Ethics Review Committee approved the study.

In April, the Ethics Committee of the Faculty of Medicine at Alexandria University accepted the current research, with the serial number **0305083**.

Study protocol:

In adherence to the principles outlined in the Helsinki Declaration, the study protocol was implemented with approval from the institutional review board. Before commencing the research, written consent was obtained from the patient to utilize their clinical information.

Data and materials availability: The datasets used or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing interests: The authors declare that they have no competing interests.

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This work was done according to the **STROBE** guidelines.

Authors' contributions

Mohamed Gamal Abdrabou and Emad Rashad Badr collected and followed up on the patients, carrying out the requested investigations. Hosam Eldin Abouelkheir and Abeer Elhadidi followed up with the patients and analyzed the collected data. All authors authorized the manuscript.

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