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Association between *Helicobacter Pylori* Infection and Iron Deficiency Anemia: A Cross Sectional Study

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Summary:

Background: **Helicobacter pylori (H. pylori)** ***remains one of the most common worldwide human infections and is associated with a number of important upper gastrointestinal (GI) conditions including chronic gastritis, peptic ulcer disease, gastric carcinoma and special type of lymphoma. Anaemia is a common disorder in developing countries and the commonest cause is iron deficiency. There are many causes for high prevalence of this disorder in our society, many are well-known and investigated while some are new and not well established. Although*** **H. pylori** ***associates peptic ulcers and gastric malignancy can cause bleeding, resulting in iron deficiency, but majority of patients infected with*** **H. pylori** ***does not have ulcer or malignancy. They usually have chronic gastritis that is not associated with GI bleeding. About 35% of iron deficiency anaemia cases remain unexplained after a gastrointestinal evaluation. Recently investigation focused on the role of*** **H. pylori,** ***in the development of extra-gastrointestinal diseases including Iron deficiency anaemia.***

Methodology: This observational, cross sectional study was carried out at BSMMU, Dhaka from July 2010 to October 2011 to find out the association between **H. pylori** ***infection and iron deficiency anemia in Bangladeshi adults. Patients with dyspepsia and indication of upper GI endoscopy were initially enrolled in the study and finally a total of 168 subjects were included based on inclusion and exclusion criteria.***

Result: Among 168 patients, 105 (62.5%) were male and 63(37.5%) were female with the mean age of 33.25 years. On endoscopy, 147(87.5%) of the patients had normal findings and 21(12.5 %) had erosive gastritis. None of them had hemorrhagic erosive gastritis. Among 168 patients, 115(68.45%) were positive **and 53(31.55%)** ***were negative for*** **H. pylori** ***by rapid urease test.***

In our study, we have found a significant low mean serum ferritin level (P<0.001) in **H. pylori** ***infected patients. This result is consistent with most of previous data concerning the effect*** **of H. pylori** ***infection on iron metabolism. This study has also revealed a significant (P<0.001) lower value of mean MCV and MCH in*** **H. pylori** ***positive patients. Regarding hemoglobin value, our study has showed no significant (P>0.05) different in hemoglobin level according to*** **H. pylori** ***status.***

Conclusion: Though there was no significant low hemoglobin level in our study, mean hemoglobin level was low in **H. pylori** ***positive patient. In our study*** **H. pylori** ***was not significantly associated with iron deficiency anemia but it was significantly associated with iron deficiency.***

Key words: Helicobacter pyloin (HP), iron deficiency anaemia (IDA), Proton pump inhibitor (PPI).

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Introduction:

Helicobacter pylori (H. pylori) remains one of the most common worldwide human infections and is associated with a number of important upper gastrointestinal (GI) conditions including chronic gastritis, peptic ulcer disease, gastric carcinoma and special type of lymphoma. The prevalence of *H. pylori* is closely tied to socioeconomic conditions and accordingly, this infection is more common in developing countries than in developed countries.[1] In developing countries, 70% to 90% of the population carries *H. pylori,* almost all of these acquire the infection before the age of 10 years.[2] In developed countries, the prevalence of infection is lower, ranging from 25% to 50%. The data from developed countries also suggest that most infections are acquired in childhood.[2]

The prevalence of *H. pylori* infection is very high in Bangladesh. A study conducted on Bangladeshi children by ICDDRB scientists have shown that, 60% are infected by the age of three months and 80% are infected by three years of age.[3] In another study , carried out on adult male by Ahmed et al. about 92% have been found to be seropositive for *H. pylori* antibody.[4]

Helicobacter pylori is a gram-negative, micro-aerophilic curved bacterium which persistently colonizes the human stomach because of its capacity to establish close contact with the gastric mucosal cells.[5] It is an obligate parasite, specific for primates, including humans[6]. *Helicobacter pylori* colonizes almost half of world's population[7] and amongst these only a minority of them develop associated clinical disease.[8]

The infection is typically acquired in childhood and has a long latent period.[6] In most patients, it does not cause symptoms and the infection often persists without any clinically evident disease. However, only 10–20% of infected patients develop severe diseases during their lifetime.[7] In fact, according to some epidemiological studies it is a class 1 carcinogen for gastric adenocarcinoma.[9] It is the most common proven risk factor for human gastric cancer (those not arising from cardiac end of the stomach).[4,10]

The virulence factors for *H. pylori* include cagA, vacA, urease, and outer membrane proteins including babA, sabA, oipA, alpA as well as iceA.[11] It is proposed that the clinical outcome of *H. pylori* infection is linked to certain strains expressing vacA and cagA[12,13,14,15,] and these factors have been extensively studied.

Recent studies suggest that in most patients Chronic gastritis (CG) is a bacterial (microbial) disease, and that a successful treatment of the infection results in improvement of gastritis.[16] This has important therapeutic implications, particularly with respect to peptic ulcer disease.

Numerous studies are being carried out for extra-gastric manifestations of *H. pylori* infection including cardio-vascular, hematological, neurological and hepatobilliary diseases.[17,18] The Maastricht III consensus report, 2005 recommended that presence of *H. pylori* infection should be sought for and treated in unexplained iron deficiency anemia and ITP.[15]

Anemia is a common disorder in developing countries and the commonest cause is iron deficiency[19-21]. According to WHO, anemia is estimated to affect more than 2 billion people worldwide. Half of all reason for anemia is iron deficiency[22]. There are many causes for high prevalence of this disorder in our society, many are well-known and investigated while some are new and not well established. Although *H. pylori* associates peptic ulcers and gastric malignancy can cause bleeding, resulting in iron deficiency, but majority of patients infected with *H. pylori* does not have ulcer or malignancy. They usually have chronic gastritis, that is not associated with GI bleeding[23]. About 35% of iron deficiency anemia cases remain unexplained after a gastrointestinal evaluation[24]. Recently investigation focused on the role of *H. pylori*, in the development of extra-gastrointestinal diseases including Iron deficiency anemia.

Recently several sero epidemiologic studies have suggested a link between iron deficiency anemia and *H. pylori* infection.[25,26] Epidemiological studies have shown that persons seropositive for *H. pylori* infection have a significantly lower s. ferritin level.[27,28] In a Danish population based study, persons who were seropositive for *H. pylori* infection had a 40% increased risk of having a reduced serum ferritin level as compared with seronegative individuals.[23] However, a few epidemiologic studies do not support an association between *H. pylori* infection and iron deficiency anemia. Collett et al. found no significant differences in serum ferritin levels between *H. pylori* seropositive or seronegative males or females in a study of 1060 adults in New Zealand.[29] Further studies are required to confirm a causal relationship and explore mechanisms of disease. In Bangladesh, no study has been carried out to find the association between *H. pylori* infection and iron deficiency anemia in adult.

Possible pathogenic mechanisms for iron deficiency anemia (IDA) in *H. pylori* infected patient are bleeding from PUD or gastric malignancy, Occult blood loss secondary to chronic erosive gastritis, increased iron uptake and utilization by *H. pylori* bacteria and decreased iron absorption secondary to chronic gastritis and hypo or achlorhydria. Most dietary iron is in the non-haemic ferric form, and an acidic intragastric P^H is needed to reduce it to the ferrous form for absorption. This reaction is promoted by gastric acidity and ascorbic acid (AA), which is thus considered the most potent regulator of iron absorption.[30] Patients with *H. pylori*

gastritis showed an increased in intra gastric P^H with a median of > 3, a P^H that is known to be critical in the process of iron absorption. Moreover, ascorbic acid is actively secreted from plasma to the gastric juice,[31] but the concentration of ascorbic acid in the gastric juice of patients with *H. pylori* gastritis and IDA is clearly reduced in comparison with both healthy and non-anemic *H. pylori* controls.[32-34]

The level of ferritin in serum reflects the magnitude of the mobilizable iron stores in the body[35] and is a sensitive marker for iron status. Hemoglobin levels are maintained with in normal range until the body iron stores are exhausted.[23]Thus hemoglobin levels could be within normal ranges with very low or absent iron stores.[23,36]

H. pylori may have a protective role in Barrett's esophagus, and esophageal adenocarcinoma, as an inverse association have been observed between the presence of it (especially cagA positive strains) and these disorders.[37] The absence of *H. pylori* is associated with an increased risk of allergies and childhood onset asthma.[38] Multiple studies have revealed that it may also be involved in physiologic regulation of leptin and ghrelin the multifunctional hormones that help to regulate the body weight.[39]

The status of iron was defined as:

Adequate iron stores: ferritin >30 µg /L,

low iron stores: ferritin 15-30 µg /L,

depleted iron stores: ferritin <15 µg /L,

iron deficiency anaemia males: ferritin <15 µg /L + haemoglobin <13.0 g/dl and

iron deficiency anaemia females: ferritin <15 µg /L + haemoglobin <11.50 g/dl

Objectives:

To find out the association between *H. pylori* infection and iron deficiency anemia.

Specific objectives: 1. To see any significant change in blood iron parameters in *H. pylori* positive patients and to compare blood iron parameters between *H. pylori* positive and *H. pylori* negative patients.

Materials and Methods:

Type of study: An observational, cross sectional study.

Place of study: Department of Gastroenterology, Biochemistry, Clinical pathology of BSMMU, Dhaka, Bangladesh.

Period of study: From July 2010 to October 2011.

Study population: Patients attending the Department of Gastroenterology for dyspeptic symptoms like upper abdominal pain or discomfort, anorexia, nausea, vomiting, bloating, belching, early satiety etc were initially enrolled for the study. Patients who were found to be normal or found to have endoscopic atrophic gastritis or erosive gastritis were finally included for the study. Informed written consents were obtained from the all patients.

Sample size: Minimum sample size was calculated at 5% level of significance and 95% confidence level. According to this formula, the minimum sample size was 384. But due to time constraint sample size was compromised. However, one hundred and sixty eight patients based on inclusion and exclusion was included in the study.

Ethical clearance was obtained from ethical review committee of BSMMU

Inclusion criteria:

1. Patients having indication for upper GIT endoscopy.
2. Age: 18-60 years.
3. Ambulatory patients of both sexes.
4. Patients willing to undergo upper GI endoscopy and biopsy.

Exclusion criteria:

1. Patients having peptic ulcers, erosive hemorrhagic gastritis, gastric malignancy and varices.
2. Patients having menorrhagia, upper or lower GI bleeding, or recent blood donation.
3. Patients with obvious feature of malabsorption.
4. Patients having Intestinal worm infestation.
5. Pregnancy and Lactation.
6. Concomitant other severe systemic diseases.
7. Regular user of NSAIDS.
8. Diagnosed case of hematological disease.
9. Unwilling or contraindication for upper GI endoscopy.
10. Cases with false positive RUT

Methods:

Patients attending the department of gastroenterology with dyspepsia and indication for upper GI endoscopy were initially enrolled based on the above mentioned inclusion and exclusion criteria. Their clinical history and physical findings were noted in the standard data sheet. Patients who received proton pump inhibitors, antibiotics or bismuth compounds were advised to come for endoscopy after stopping the above mentioned drugs for at least two weeks. After proper explanation written informed consent were obtained from the selected patients for upper GI endoscopy and biopsy. Patients underwent endoscopy in the department of Gastroenterology, BSMMU in presence of an experienced endoscopist using an Olympus forward viewing video endoscope under topical lignocaine anesthesia. Patients who were found to be normal or found to have endoscopic atrophic gastritis or erosive gastritis were finally included for the study During endoscopy two biopsy samples were taken, one from the body and another from the antrum of the stomach. Rapid urease test (CLO) of biopsy specimens for detection of *H. pylori* was done immediately and was observed for up to 24 hours to see the color change of test medium from straw to pink or reddish. Patients who tested positive for *H. pylori* were placed under group A wing and those who tested negative for *H. pylori* were placed under group B wing. Different variables (e.g age, sex, symptoms etc) and means of iron parameters (Hb, S. ferritin, MCV, MCH) of two groups were compared. Prior to the procedure endoscope and biopsy forceps were disinfected by immersing in the gluteraldehyde solution 2% (CIDEX^{R}) for 10 minutes. Side channels were rinsed with normal saline. All patients underwent CBC, S. Ferritin estimation and Stool R/M/E for detection of ova in stool.

A gel matrix media embedded with urea and a pH-sensitive indicator was used for the rapid urease test (RUTs).

Results:

A total of 168 patients were finally included for the study. Among them 115 patients were RUT positive for *H. pylori* and 53 patients were RUT negative for *H. pylori*. *H. pylori* positive patients were placed in group A and *H. pylor*i negative patients were placed in group B.

A total of 168 patients were finally included in this study. They were divided into five groups according to age. Majority of the patients were found in the age group of 21-30 years, which was 51(44.3%) in Group A and 24(45.3%) patients in Group B. The mean age was found 34.1±11.8 years in Group A and 32.5 ±8.1 years in Group B. The value of unpaired t-test was 0.372 and it was not statistically significant (p>0.05). (Table I)

Table-I

Age distribution of the study patients (n=168).

Age (in year)	Group A (n=115)		Group B (n=53)		P Value
	n	%	n	%	
≤20	9	7.8	2	3.8	
21-30	51	44.3	24	45.3	
31-40	27	23.5	20	37.7	
41-50	14	12.2	6	11.3	
51-60	14	12.2	1	1.9	
Mean ±SD	34.1±11.8		32.5±8.1		0.372^{ns}
Range (min-max)	(18-60)		(19-52)		

Group A= Positive for *H. pylori*, Group B=Negative for *H. pylori*, NS=Not significant
P value reached from unpaired t-test.

Gender distribution of study patients (n=168).

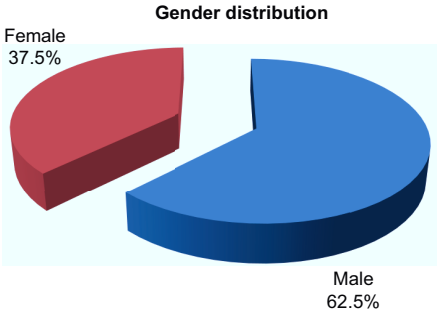

Fig.-I: *Pie chart showing male and female proportion in the study patients (n=168).*

Table-II

Clinical feature of the study patients (n=168)

Age (in year)	Group A (n=115)		Group B (n=53)		P Value
	n	%	n	%	
Upper abdominal pain	92	80.0	28	52.8	0.001[s]
Anorexia	47	40.9	10	18.9	0.005[s]
Belching	27	23.5	22	41.5	0.016[s]
Abdominal discomfort	30	26.1	20	37.7	0.124[ns]
Burning sensation of abdomen	57	49.6	32	60.4	0.191[ns]
Nausea	59	51.3	26	49.1	0.786[ns]
Vomiting	21	18.3	4	7.5	0.069[ns]
Bloating	87	75.7	34	64.2	0.122[ns]
Early satiety	41	35.7	14	26.4	0.235[ns]

Table-III

Endoscopic diagnosis of the study patients (n=168).

Endoscopic diagnosis	Group A (n=115)		Group B (n=53)		P Value
	n	%	n	%	
Normal	100	87.0	47	88.7	0.754[ns]
Erosive gastritis	15	13.0	6	11.3	

This study finally involved 168 patients. Among them 105 patients (62.5%) were male and 63 patients (37.5%) were female. (Figure I)

The above table IV shown the clinical feature of the study patients. Maximum patients had abdominal pain in group A, which was 92(80.0%), however in group B, most of the patients had bloating, which was 34(64.2). Upper abdominal pain, anorexia and belching were statistically significant ($p<0.05$) but abdominal discomfort, burning sensation of abdomen, nausea, bloating, and early satiety was not significant ($p>0.05$) between two groups in chi square test.

The above table V shown the endoscopic diagnosis of the study patients. Normal finding was found 100(87.0%) in group A and 47(88.7%) in group B. Erosive gastritis was found 15(13.0%) and 6(11.3%) in group A and group B respectively. The difference was not statistically significant ($p>0.05$) between two groups in Chi square test.

H. pylori status of the study patients (n=168).

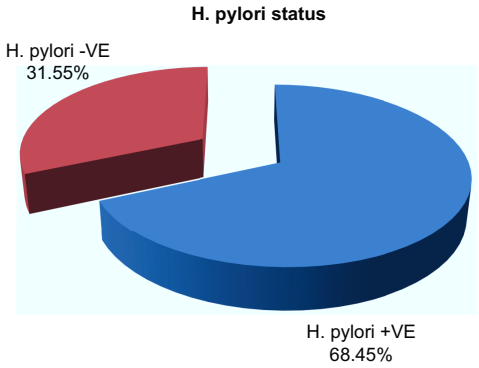

Fig.-IV: *Pie chart showing H. pylori status of the study patients (n=168).*

Table-IV

Mean serum ferritin of the study patients (n=168).

S. ferritin (mg/L)	Group A (n=115)		Group B (n=53)		P Value
	n	%	n	%	
>30 mg/L	78	67.8	51	96.2	
15-30 mg/L	21	18.3	2	3.8	
<15 mg/L	14	12.2	0	0.0	
Mean ±SD	64.6±61.6		120.5±77.9		0.001[s]
Range (min-max)	(0.01-402.8)		(25-370.2)		

P value reached from unpaired t-test.

Table-V

Association between H. pylori infection and log transformed serum ferritin levels in multiple linear regression model.

	Adjusted for age and sex		Adjusted for multiple covariates*	
H pylori infection	Geometric mean of S. ferritin level		% change (95% CI)	% change (95% CI)
Not infected	53	99.1	Reference	Reference
Infected	115	39.9	-28.3% (-4.83 to-8.4)	-29.0%(-4.9 to -9.0)

* Adjusted for sex, age, residence, profession, income and family member

This study was finally involved 168 patients. Among them 115 patients (68.45%) were CLO positive for *H. pylori* and 53 patients (31.55%) were CLO negative for *H. pylori*. (Figure IV)

The above table VI shown the S. ferritin of the study patients. Maximum S. ferritin level was found >30 mg/L in both groups, which was 78(67.8%) in group A and 51(96.2%) in group B. S. ferritin level 15 - 30 g/L was found 21(18.3%) in group A and 2(3.8%) in group B. S. ferritin level <15 mg/L was found 14(12.2%) in group A but not found in group B. The mean S. ferritin was found 64.6±61.6 mg/L with range from 0.01 to 402.8 mg/L in group A and 120.5±77.9 mg/L with range from 25 to 370.2 mg/L in group B. The mean difference was statistically significant ($p<0.05$) between two groups in unpaired t-test.

Table-VI

Mean MCV of the study patients (n=168).

	Group A (n=115)		Group B (n=53)		P Value
	Mean	±SD	Mean	±SD	
MCV (in fL)	85.4	±6.9	89.3	±3.9	0.001[s]
Range (min-max)	(56	-103)	(82	-98)	

P value reached from unpaired t-test.

Table-VII

Mean MCH of the study patients (n=168)

	Group A (n=115)		Group B (n=53)		P Value
	Mean	±SD	Mean	±SD	
MCH (in pg)	27.5	±2.4	29.0	±1.2	0.001[s]
Range (min-max)	(19	-32)	(26	-31)	

P value reached from unpaired t-test.

Table-VIII

Hb of the study patients according to male and female (n=168)

Hb (gm/dl)	Group A (n=115)		Group B (n=53)		P Value
	n	%	n	%	
Male	(n=62)		(n=43)		
≥13 (normal) gm/dl	51	82.3	38	88.4	
<13 (Low) gm/dl	11	17.7	5	11.6	
Mean ±SD	13.9	±1.2	14.3	±0.8	0.059[ns]
Range (min-max)	(9.3	-16.2)	(12.6	-15.8)	
Female	(n=53)	(n=10)			
eH11.5 (normal) gm/dl	38	71.7	9	90.0	
<11.5 (Low) gm/dl	15	28.3	1	10.0	
Mean ±SD	12.0	±1.1	12.6	±1.0	0.114[ns]
Range (min-max)	(9.0	-14.3)	(10.6	-14.1)	

P value reached from unpaired t-test.

The percentage of change in *serum* ferritin concentration according to the presence of the infection was calculated by exp (*b*), where *b* is the corresponding regression coefficient.

The above table VII presents the results of linear regression models. The log transformation of serum ferritin concentration was necessary due to highly skewed distribution. The log transformation values serum ferritin concentration were close to normally distributed. Age and sex adjusted geometric mean of S. ferritin concentration was 99.1 µg/L for noninfected and 39.9 µg/L for *H. pylori* infected persons. In the multiple regression model *H. pylori* infection *was associated with a 29.0% decrease in serum* ferritin (95% CI = 4.9 to – 9.0); r^2=0.26).

The above table VIII shown the MCV of the study patients. The mean MCV was found 85.4±6.9 fL with range from 56 to 103 fL in group A and 89.3±3.9 fL with range from 82 to 98 fL in group B. The mean difference was statistically significant (p<0.05) between two groups in unpaired t-test.

The above table IX shown the MCH of the study patients. The mean MCH was found 27.5±2.4 pg with

Table-IX

Iron deficiency Anaemia status according to Hb and Serum ferritin level of the study patients (n=168).

Iron deficiency Anaemia	Group A(n=115)		Group B(n=53)		P value
	n	%	n	%	
Male	(n=62)		(n=43)		
Iron deficiency anaemia	2	3.2	0	0.0	0.346^{ns}
No iron deficiency anaemia	60	96.8	43	100.0	
Female	(n=53)	(n=10)			
Iron deficiency anaemia	7	13.2	0	0.0	0.278^{ns}
No iron deficiency anaemia	46	86.8	10	100.0	

Iron deficiency anaemia:
Male: Hb <13 gm/dl and S. ferritin <15 µg/L
Female: Hb <11.5 gm/dl and S. ferritin <15 µg/L
P value reached from chi square test.

Table-X

Frequency of iron deficiency, iron deficiency anemia according to H. pylori status.

	Hemoglobin					
	Low Hb Depleted iron stores			Normal Hb Low iron stores		
	Depleted iron stores n	Low iron stores iron stores N	Adequate iron stores n	Depleted iron stores N	Low iron stores N	Adequate iron stores N
Male						
Group A	2	0	8	2	10	40
Group B	0	0	5	0	2	36
Female						
Group A	7	1	7	5	10	23
Group B	0	0	1	0	0	9
Total						
Group A	9	1	15	7	20	63
Group B	0	0	6	0	2	45

range from 19 to 32 pg in group A and 29±1.2 pg with range from 26 to 31 pg in group B. The mean difference was statistically significant (p<0.05) between two groups in unpaired t-test.

The above table X shown the Hb of the study patients according to male and female patients. Among the male, Hb level e"13 (normal) gm/dl was found 38(82.3%) in group A and 38(88.4%) in group B. Hb level <13 (Low) gm/dl was found 11(17.7%) and 5(11.6%) in group A and group B respectively in male patients. The mean Hb level was 13.9±1.2 gm/dl in group A and 14.3±0.8 in group B in male patients. Among the female patients, Hb level was e"11.5 (normal) gm/dl found 38(71.7%) in group A and 9(90.0%) in group B. Hb level was <11.5 (Low) gm/dl found 15(28.3%) and 1(10.0%) in group A and group B respectively in female patients. The mean Hb level was 12.0±1.1 gm/dl in group A and 12.6±1.0 in group B in female patients. The mean difference was not statistically significant (p>0.05) between two groups of both male and female in unpaired t-test.

The above table XI showed the iron deficiency anaemia status according to Hb and S. ferritin level of the study patients. In male patients, iron deficiency anaemia was found 2(3.2%) in group A and not found in group B. In the female patients, it was found 7(3.2%) in group A and not found in group B. The difference was not statistically

significant (p>0.05) between the two groups in chi square test. Other results are depicted in the table XI.

Frequency of iron deficiency anemia was 9 and low iron stores 1 in low hemoglobin level in group A but not found in group B. Frequency of depleted iron stores was 7 and low iron stores 20 in normal hemoglobin level in group A. In group B with normal hemoglobin level, depleted iron stores was not found but low iron stores found in 2 patients.(Table XII)

Discussion:

Anemia is a common disorder in the developing countries and the commonest cause is iron deficiency.[19-21] there are many causes for the high prevalence of this disorder in our society, many are well-known and investigated while some are new and not well established. Although *H. pylori* associated peptic ulcers and gastric malignancies can cause bleeding, resulting in iron deficiency, but majority of patients infected with *H. pylori* does not have ulcer or malignancy. They usually have chronic gastritis which is not associated with GI bleeding.[23] However in many cases despite thorough investigations causes of IDA remain unexplained. Recently investigations are more focused on the role of *H. pylori*, in the development of extra-gastrointestinal diseases including Iron deficiency anemia.

The present study is the first of its kind in Bangladesh where we have seen association between *Helicobacter pylori* infection and iron deficiency anaemia in adult. Our study has revealed 68.45% of patients were positive for *H. pylori* who underwent endoscopic biopsy and rapid urease test. This is similar to previous study conducted in Karachi, Pakistan.[40] In Faisalabad, India 92% of duodenal ulcer patients found H. pylori positive by rapid urease test.[41] Previous study conducted in Korean Children where only 23.6% of patients are positive for *H. pylori* by rapid urease test.[42] This variation may be due to different prevalence of *H. pylori* in developed and developing countries, and change of prevalence with age.

In our study has found significant low mean serum ferritin level (P<.001) in *H. pylori* infected patients. This result is consistent with most of previous data concerning the effect of *H. pylori* infection on iron metabolism. Most previous study had found significant inverse relationship between serum ferritin and *H. pylori* infection. Low serum ferritin level were found in *H. pylori* infected patients in Pakistan[40], China[43], Korea[44,45], German[27], Denmark[23], Australia[46], America[47] and Alaska[48]. There was no significant differences of serum ferritin level according to *H. pylori* status found in New Zealand [29], and Korea[49], Iran[50], Egypt.[51]

In the multiple linear regression model (adjusted for sex, age, residence, income, family member) our study showed, *H. pylori* was associated with a 29% decrease of serum ferritin (95% CI= 4.9 to – 9.0; $r^2 = 0.26$). Previous study in Denmark[23], Germany[27], and America[47], in the multiple linear regression model *H. Pylori* were associated with a 40%, 17% and 40% decrease of serum ferritin respectively.

This study has also revealed a significant (<.001) lower value of mean MCV and MCH in *H. pylori* positive patients. This was similar to that in China[43], Korea[42], and Egypt[51]. These findings did not correlate with that of Pakistan where there was no difference in MCV according to *H. pylori* status.[40]

Regarding Hemoglobin value, our study has showed no significant (>.05) different in haemoglobin level according to *H. pylori* status. This is similar to study in Denmark[23], Germany[27], and USA.[47] Low haemoglobin values were found in *H. pylori* infected patient in Pakistan[40], China[43] and Egypt.[51] Though there was no significant low haemoglobin level in our study but mean haemoglobin level was low in *H. pylori* positive patients.

Our study has showed significant low level of serum ferritin, MCV, MCH in *H. pylori* infected patients but haemoglobin value is not significantly decrease. According to literature serum ferritin relates to mobilizable iron stores of the body and can be used as a marker for iron stores.[23] Haemoglobin levels are maintained within normal range until the body iron stores are exhausted.[36] Thus haemoglobin levels could be within normal ranges with very low / absent iron stores.[23,36]

Conclusion:

After exclusion of the most of the possible causes of iron deficiency or iron deficiency anemia by clinical and investigational evaluation our study was able to show that *H. pylori* infection was significantly associated with decrease in serum ferritin, MCV, MCH. Though in our study, *H. pylori* were not significantly associated with iron deficiency anemia, it was significantly associated

with iron deficiency. Based on the above findings, *H. pylori* infection may be sought for and treated in patients with unexplained iron deficiency anemia. Further study with larger sample size may be carried out to establish *H. pylori* infection as a communicable cause of iron deficiency or iron deficiency anemia next to helminthiasis.

Conflict of Interest: None

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