Burden of *Helicobacter pylori* Infections and Associated Risk Factors among Cases of Iron Deficiency Anaemia in Egypt

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**Authors’ contributions**

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

**Article Information**

DOI: 10.9734/MRJI/2020/v30i730241

Editor(s):
(1) Dr. Kai Zhang, State University of New York at Buffalo, USA.
(2) Dr. Lachhman Das Singla, Guru Angad Dev Veterinary and Animal Sciences University, India.

Reviewers:
(1) Josue Jesus Aliaga Ramos, Cayetano Heredia University, Peru.
(2) P. N. Remya, SRM Institute of Science and Technology, India.

Complete Peer review History: [http://www.sdiarticle4.com/review-history/58186](http://www.sdiarticle4.com/review-history/58186)

**ABSTRACT**

**Introduction**: Iron deficiency anaemia (IDA) is a worldwide nutritional problem; it accounts for about half of the world's anaemia burden. Globally, *Helicobacter pylori* (*H. pylori*) is becoming an increasingly troublesome economic and public health problem. The colonization of the organism in gastric mucosa may impair iron uptake and increase iron loss, potentially leading to iron deficiency anaemia. The mechanisms by which *H. pylori* is postulated to cause IDA are *H. pylori*-associated chronic gastritis resulting in hypo/or achlorhydria, reduced ascorbic acid secretion and reduced intestinal iron absorption, occult blood loss due to chronic erosive gastritis, and sequestration and utilization of iron by *Helicobacter pylori*.

**Aims**: To detect *H. pylori*-related IDA prevalence among asymptomatic cases of anaemia and to address the possibility that such infection may play a detrimental role in their blood picture, serum iron and ferritin levels and total iron binding capacity (TIBC)

**Study Design & Methods**: Facility based cross-sectional study was conducted in the period from December 2018 to May 2019. Screening was done for asymptomatic attendants of a number of private laboratories in Beheira, Alexandria and Gharbiya governorates. Three
hundreds of whom were proved to be cases of IDA and were further tested for H. pylori antigen in stool.

**Results:** *Helicobacter pylori* Ag test in stool was positive in 180 out of 300 cases of iron deficiency anaemia. The infection significantly affected the haemoglobin level, MCV, MCH, and RDW in studied cases (p<0.05). Infection with *H. pylori* also significantly affected the serum iron, serum ferritin and TIBC in the studied cases of IDA (p<0.05).

**Conclusion:** A significant association between *H. pylori* infection and IDA. Screening for *H. pylori* among unexplained cases of IDA is recommended.

**Keywords:** H. pylori; H. pylori stool AG test; Iron deficiency anaemia; serum iron; TIBC.

1. **INTRODUCTION**

Anemia is a medical condition associated with increased or decreased RBCs characterized by inadequate oxygen-carrying capacity to meet physiological needs [1]. Iron deficiency is the most common cause of anemia globally. Iron deficiency anemia (IDA) is caused by deranged synthesis of haemoglobin, resulting in red cells that are include reduced amounts of hemoglobin (hypochromic) and smaller than normal (microcytic) [2]. Iron deficiency evolves through three stages: iron depletion is the earliest stage of iron deficiency in which storage iron is decreased or absent but serum iron concentration and blood hemoglobin levels are normal, the second stage is iron deficiency without anemia then iron deficiency anemia [3].

*Helicobacter pylori* (*H. pylori*) infection is the most common chronic infection involving half of the population worldwide [4]. *H. pylori* is a microaerophilic, Gram-negative, spiral-shaped bacterium [5]. Numerous researchers have focused on the role of *H. pylori* infection on a wide range of gastrointestinal disorders that vary from asymptomatic gastritis to peptic ulcer, and also gastric carcinoma and lymphoma [6]. It has been reported that *H. pylori* may influence some extra-gastrointestinal diseases including iron deficiency (ID), and iron deficiency anemia [4].

The major cause of iron deficiency (ID) in developed countries is overt or occult gastrointestinal blood loss [3]. The mechanisms by which Hp infection is postulated to cause IDA are Hp-associated chronic gastritis resulting in hypo or achlorhydria, reduced ascorbic acid secretion and reduced intestinal iron absorption, occult blood loss due to chronic erosive gastritis, and sequestration and utilization of iron by *Helicobacter pylori* [4].

This study aimed to detect *H. pylori*–related IDA prevalence among cases of anaemia who are asymptomatic regards GIT complaints and to address the possibility that such infection may play a detrimental role in their blood picture, serum iron and ferritin levels and total iron binding capacity (TIBC).

2. **MATERIALS AND METHODS**

A facility based descriptive cross-sectional study design was conducted in the period from December 2018 to May 2019 in some private laboratories in Beheira, Alexandria and Gharbiya governorates. Screened subjects included all the attendance seeking a routine check up blood picture and those indicated for follow up of cases of anaemia. Selected participants in the study were those 300 cases proved to be cases of IDA; 200 subjects were newly diagnosed as IDA and the remaining were on iron supplements. All the 300 subjects were asymptomatic regards GIT manifestations of *H. pylori* infection.

Patients with malignancies, chronic diseases, dimorphic anemia, obvious causes of IDA, obvious non-GI causes of blood loss, chronic renal failure and acute infections were excluded from the study. Also patients who were on non-steroidal anti-inflammatory drugs for long periods were excluded. None of the patients enrolled mentioned previous diagnosis, endoscopic examination or receiving treatment for *H. pylori* infection.

2.1 **Questionnaire**

Participants were asked to fill in a questionnaire covering personal and demographic data and dietary habits; personally or by the parents.

2.2 **Blood Sample**

2.2.1 **Complete Blood Count (CBC)**

Blood sampling was done for all screened subjects throughout the duration of the study and were examined for complete blood count (CBC) done by (Sysmex-XS 500i).
2.2.2 Ferritin

It was measured using the Elecsys 2010 system using a Roche diagnostics kit by the electrochemiluminescence immunoassay (ELISA) method.

2.2.3 Serum levels of iron

Serum levels of iron were measured by the colorimetric method with a Roche modular analyzer.

2.2.4 TIBC

TIBC was measured with the Roche modular analyzer.

2.3 Stool Samples

2.3.1 Helicobacter pylori stool antigen

Stool samples were provided by participants and were tested for H. pylori stool antigen (H. pylori Antigen ELISA Diagnostic Kit; CTK Biotech HpSA kit, San Diego, CA 92121 Inc., USA). The test was done on fresh stool samples. A random stool specimen in a clean, dry receptacle was collected. The stool collection device with the specimen’s ID number (patient ID sticker) was labeled. The stool collection device was opened by unscrewing the top and using the collection stick to randomly pierce in 2-5 different sites, twisting the collection stick into the stool specimens to help collection if necessary. All inner grooves of the collection stick were filled with the stool specimen. However, excess stool specimen on the outside of grooves was scraped off the collection stick and the stool collection device was tightened securely to close. According to the manufacturer leaflet provided with the H. pylori test kits the relative sensitivity, relative specificity, and overall agreement were 96.7%, 93.8%, and 94.9%, respectively. Its analytical sensitivity was 100% positive detection rate at 1 ng/mL of pylori lysate antigen in fecal specimens.

3. RESULTS

Three hundred participants suffering from IDA were recruited in this study. The age of the participants ranged from 3 to 68 years old with a mean of 31.2 ± SD10.3. H. pylori Ag test was positive in 180/300 (60%) of the participants.

In this study although the prevalence was highest among participants > 50 years old (62%) compared to all other younger participants; yet no statistically significant difference between age groups was reported (p=0.077) [Table 1].

In this work the prevalence rate of H. pylori was significantly higher among females (62.2%) than among males (57%). (p=0.001) [Table 1].

One hundred and thirty four out of the 300 cases of IDA studied in this study (44.7%) were residents of urban areas while 166 (55.3%) were residents of rural areas. Prevalence rate of H. pylori infection among urban dwellers was 57.5% (77/134) compared to 62% (103/166) in rural dwellers. Residence was not significantly associated with prevalence of H. pylori [Table 1].

Family history of H. pylori infection significantly affected the prevalence rate of H. pylori among the participants. (p=0.037). Prevalence rate among those with positive family history was 71.4% compared to 57% among those with no family history. It is worth mentioning that in the 63 cases with positive family history; it was the mother who had H. pylori infection [Table 1].

An inverse association between the level of education and H. pylori infection among the studied cases was reported in this study. H. pylori prevalence recorded among those who were illiterate or had only primary school education was 73.55 and 78.4%, respectively compared to 51.8% among those who had high school education and to only 30.8% among those who achieved university education or higher. This variance was highly statistically significant (p=0.001) [Table 1].

Smoking and eating spicy food had no statistically significant effect on the prevalence of H. pylori among the participants. (p >0.05) [Table 2]. Intake of high protein diet and skipping meals were significantly implicated to increase the risk of H. pylori infection among the enrolled cases of IDA. (p<0.05) [Table 2].

In addition, drinking coffee and tea significantly increased the prevalence rate of H. pylori among participants compared to those who didn’t have such dietary habits. (p <0.05) [Table 2].

Helicobacter pylori infection significantly affected the haemoglobin level, MCV, MCH and RDW in the studied cases (p<0.05). [Table 3]. On the other hand no statistically significant
Table 1. Sociodemographic data of the studied patients with IDA in relation to Helicobacter pylori infection

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Helicobacter Pylori (Stool antigen test)</th>
<th>Total (n = 300)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive (n = 180)</td>
<td>Negative (n = 120)</td>
<td>No</td>
</tr>
<tr>
<td>Sex:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>73</td>
<td>55</td>
<td>128</td>
</tr>
<tr>
<td>Female</td>
<td>107</td>
<td>65</td>
<td>172</td>
</tr>
<tr>
<td>Mean age ± SD (years): (Min.-Max.)</td>
<td>30.2 ± 9.6</td>
<td>32.4 ± 11.8</td>
<td>31.2 ± 10.3</td>
</tr>
<tr>
<td>Residence:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>77</td>
<td>57</td>
<td>134</td>
</tr>
<tr>
<td>Rural</td>
<td>103</td>
<td>63</td>
<td>166</td>
</tr>
<tr>
<td>Family history of H.pylori infection:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>45</td>
<td>18</td>
<td>63</td>
</tr>
<tr>
<td>No</td>
<td>135</td>
<td>102</td>
<td>237</td>
</tr>
<tr>
<td>Education: **</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>10</td>
<td>8</td>
<td>18</td>
</tr>
<tr>
<td>Primary</td>
<td>71</td>
<td>42</td>
<td>113</td>
</tr>
<tr>
<td>Secondary</td>
<td>63</td>
<td>25</td>
<td>88</td>
</tr>
<tr>
<td>University</td>
<td>36</td>
<td>45</td>
<td>81</td>
</tr>
</tbody>
</table>

*Percentages were calculated from the total sample size, while other by the total of each row.
*For children, the educational level of mother was considered.
*p-value <0.05 is statistically significant

Table 2. Smoking and dietary habits of the studied patients with IDA in relation to Helicobacter pylori infection

<table>
<thead>
<tr>
<th>Habits</th>
<th>Helicobacter pylori (Stool antigen test)</th>
<th>Total (n = 300)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive (n = 180)</td>
<td>Negative (n = 120)</td>
<td>No</td>
</tr>
<tr>
<td>Smoking:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>64</td>
<td>31</td>
<td>95</td>
</tr>
<tr>
<td>No</td>
<td>116</td>
<td>89</td>
<td>205</td>
</tr>
<tr>
<td>Protein rich diets:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>168</td>
<td>96</td>
<td>264</td>
</tr>
<tr>
<td>No</td>
<td>12</td>
<td>24</td>
<td>36</td>
</tr>
<tr>
<td>Skipping meals:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>174</td>
<td>105</td>
<td>279</td>
</tr>
<tr>
<td>No</td>
<td>6</td>
<td>15</td>
<td>21</td>
</tr>
<tr>
<td>Eating spicy food</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>76</td>
<td>41</td>
<td>117</td>
</tr>
<tr>
<td>No</td>
<td>104</td>
<td>79</td>
<td>183</td>
</tr>
<tr>
<td>Drinking coffee:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>175</td>
<td>92</td>
<td>267</td>
</tr>
<tr>
<td>No</td>
<td>5</td>
<td>28</td>
<td>33</td>
</tr>
<tr>
<td>Drinking Tea:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>178</td>
<td>114</td>
<td>292</td>
</tr>
<tr>
<td>No</td>
<td>2</td>
<td>6</td>
<td>8</td>
</tr>
</tbody>
</table>

*Percentages were calculated from the total sample size, while other by the total of each row.
*p-value <0.05 is statistically significant
Table 3. The main hematological parameters for the studied patients with iron deficiency anemia in relation to *Helicobacter pylori*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Helicobacter pylori</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Positive (n=180)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Mean ± SD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Negative (n=120)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Mean ± SD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total (n=300)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Mean ± SD)</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>g/dl</td>
<td>10.1 ± 1.6</td>
<td>10.6 ± 1.5</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>%</td>
<td>33.4 ± 4.1</td>
<td>33.1 ± 4.0</td>
</tr>
<tr>
<td>MCV</td>
<td>Fl</td>
<td>72.3 ± 5.9</td>
<td>74.1 ± 3.8</td>
</tr>
<tr>
<td>MCH</td>
<td>Pg</td>
<td>22.3 ± 2.8</td>
<td>23.1 ± 2.6</td>
</tr>
<tr>
<td>MCHC</td>
<td>g/dl</td>
<td>29.9 ± 1.2</td>
<td>30.1 ± 1.3</td>
</tr>
<tr>
<td>RDW</td>
<td>%</td>
<td>16.0 ± 1.3</td>
<td>15.7 ± 1.2</td>
</tr>
<tr>
<td>RBC's count</td>
<td>x10^{12}/L</td>
<td>3.7 ± 0.45</td>
<td>3.8 ± 0.51</td>
</tr>
<tr>
<td>WBC's count</td>
<td>x10^{9}/L</td>
<td>6.3 ± 2.4</td>
<td>6.0 ± 2.3</td>
</tr>
<tr>
<td>Platelets count</td>
<td>x10^{9}/L</td>
<td>306.9 ± 78.4</td>
<td>305.1 ± 79.5</td>
</tr>
<tr>
<td>ESR</td>
<td>mm/hr</td>
<td>19.9 ± 17.4</td>
<td>18.7 ± 16.6</td>
</tr>
</tbody>
</table>

*MCV: Mean corpuscular volume    MCH: Mean corpuscular hemoglobin    RDW: Red cells distribution width    MCH: Mean corpuscular hemoglobin concentration    RBC: Red blood cells    ESR: Erythrocytes sedimentation rate    WBC: white blood cells    p-values> 0.05 are statistically insignificant*

The difference between both positive and negative groups for *H. pylori* regards their hematocrit level, MCHC, RBCs count, WBCs count, platelets count and ESR was recorded (p>0.05).

Meanwhile, *H. pylori* infection significantly affected the serum iron, serum ferritin and TIBC in studied cases of IDA (p<0.05). [Table 4].

4. DISCUSSION

Iron (Fe) is an essential element for hemoglobin synthesis, oxidation–reduction reactions, and cellular proliferation. The term iron deficiency (ID) describes a deficit in total body iron, resulting in reduction of serum ferritin levels below normal limit [7]. Iron deficiency may occur due to dietary deficiency or chronic blood loss [8].

Based on the WHO estimation, iron deficiency is responsible for 50 percent of all anemias. The prevalence of anemia during infancy and early childhood is higher than at any other time in the life cycle such as pregnancy [9].

**Table 4. Iron profile for the studied patients with IDA in relation to *H. pylori***

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Positive (n=180)</th>
<th>H. pylori</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td></td>
</tr>
<tr>
<td>Serum iron</td>
<td>µmol/L</td>
<td>12.4 ± 7.3</td>
<td>18.3 ± 6.4</td>
<td>0.001*</td>
</tr>
<tr>
<td>TIBC</td>
<td>µmol/L</td>
<td>75.1 ± 11.5</td>
<td>71.0 ± 11.0</td>
<td>0.002*</td>
</tr>
<tr>
<td>Serum ferritin</td>
<td>ng/ml</td>
<td>19.2 ± 10.1</td>
<td>24.6 ± 12.3</td>
<td>0.001*</td>
</tr>
</tbody>
</table>

*Serum iron : Reference range: 13 – 36 µmol/L; TIBC: Total iron binding capacity Reference range: 45-78 µmol/L; Serum ferritin : Reference range: M : 30 – 400 ng/ml , F : 15 – 150 ng/mL  
*p-values< 0.05 are statistically significant*
gastritis and decreased iron stores comes also from case reports, epidemiologic studies and clinical trials [14].

The Maastricht IV Consensus on the management of Hp infection recommends testing and treatment of Hp infection in patients with unexplained IDA [3]. These guidelines may be applicable in countries with high prevalence for Hp infection.

Results of the present study revealed a prevalence rate of 60% for H. pylori infection among asymptomatic cases of IDA. Different results were reported among Egyptians as those reported by Hassanein et al., 2017: 24% [15] and by Sabah et al., 2015: 69.4% [16]. The present prevalence rate was in line with results reported in previous studies carried out in African countries as that in Nigeria: 52.5% [17] in Libya: 54.4% [18] and 56.5% [19]. Higher percentages were reported in Ethiopia: 70% [20], Libya: 76% [21], Nigeria: 80% [22], Tunis: 83% [23], Morocco: 92.65% [24] and in a public survey carried out also in Nigeria 93.6% [25].

The recorded percentages in studies carried out in Asian countries, for screening for H. pylori infection among asymptomatic subjects, varied from as low as 13.1% in Iran [26], Lebanon (21%) [27], India (46%) [28], Saudi Arabia (51%) [29], Korea (54.4%) [30], China (63.4%) [31], Oman (69.5%) [32] to as high as 82.5% in Turkey [33].

Even in developed countries variable rates of H. pylori infection were reported among asymptomatic subjects as in Portugal: 84.2% [34], Mexico: 52.2% [35], Brazil: 41.1% [36], Canada: 37.9% [37], Netherlands: 32% [38], USA: 25.4% [39] and Belgium: 11% [40].

It is actually difficult to compare the prevalence rates in different studies due to variations in age and the sector of population studied. This difference in reported rates could be attributed to the different methodology, prevalence of common risk factors, criteria of the sample, as well as the specificities of techniques employed in studies.

A number of studies have suggested H. pylori infection as a reason of refractory IDA (IDA that does not respond properly to oral iron supplementation) in patients with no obvious other cause of anemia. [41,42] In one study, 58% of cases with IDA were H. pylori positive at endoscopy. [43] Another study showed that 61.9% of patients with IDA had H. pylori infection [44].

A different study showed that 62% of patients with IDA were H. pylori positive. [45] It has been obviously demonstrated in earlier studies that Hp eradication can reverse the negative influence of H. pylori infection on iron absorption and lead to improvement of IDA in case series and in clinical trials in both children and adults. [11,43]

In this research, age of the participants didn’t significantly influence the prevalence rate of H. pylori among them. On the other hand, other studies as that carried out in 2019 in Egypt by Youssef et al., highlighted a very high significant statistical association of age with H. pylori positive antibody, the highest proportion was at the age more than 65 years old, then the prevalence shows decrease gradually with deceasing the age groups. [46] This result was in line with the results in our region (EMRO) and most of developing countries, which regarded the increasing in the age as a risk factor for H. pylori infection. [47]

In this study the prevalence rate of H. pylori was significantly higher among females (62.2%) than among males (57%). Significantly higher prevalence rates among females were also previously reported in many studies worldwide. [17,19,38] The relationship between gender and H. pylori infection has been controversial in other studies [46,48,49]. The role of sex to put males at significantly higher risk of H. pylori infection compared to females was observed in many previous studies [17,50,51]. Nevertheless, such trend contradicted other studies; where gender was not significantly associated with H. pylori infection [52-54]. In addition, a systematic review with meta-analysis carried out in 2018 reported that no significant difference was observed between the two genders in worldwide H. pylori prevalence [55].

In this piece of work, prevalence rate of H. pylori infection among urban dwellers was 57.5% compared to 62% in rural dwellers. Residence was not significantly associated with prevalence of H. pylori. This finding is in line with previous studies carried out in Egypt, Mexico and Libya [19,56,57]. On the other hand, several researchers reported a positive correlation between rural life and H. pylori infection [30,58,59]. This could be attributed to inadequate sanitary conditions and to absence or poor personal hygiene [60]. Urbanization and educational level are in fact major determinants of H. pylori prevalence [27,61,62].

In the present study, H. pylori prevalence among those with family history of H. pylori (especially
infection of the mother) was 71.4% compared to 57% among others. The difference between both groups was statistically significant.

Fathers tend to have less contact with their siblings than mothers, so they are less involved in the transmission. It was reported that the relative risk of a person becoming infected with \textit{H. pylori} is approximately four or eight times greater; when the father or the mother is infected, respectively [63]. Molecular studies carried out to trace intrafamilial transmission confirmed the mother-to-child transmission in most cases and further reported a grandmother-to-child transmission. It seems that mothers could transmit the infection through mouth secretions; using common spoons or tasting the food. [64]

Furthermore, interfamilial transmission may be also responsible for re-infection with \textit{H. pylori} as its presence among asymptomatic family members may facilitate the transmission among households. Several previous studies consistently supported infected siblings as a risk factor for \textit{H. pylori} infection among families. [64-66]

An inverse association between the level of education and \textit{H. pylori} infection among the studied students was reported in the current work. The same association was reported in other similar studies [34,40]. Unlikely, Youssef et al., (2019) in Egypt reported no significant association between level of education and prevalence of \textit{H. pylori} infection. [46]

Smoking showed no significant association with \textit{H. pylori} infection among the cases screened in this research. This is in line with several previous reports [19,31,67]. The absence of association in such studies may be due to less number of smokers screened, besides the type of tobacco and the frequency of smoking. On the other hand, other researchers reported that smokers were at higher risk of acquiring \textit{H. pylori} infection. [20,33,46,60]

As regards the dietary habits of the participants in the current work; drinking coffee and tea, intake of high protein diet and skipping meals were significantly implicated to increase the risk of \textit{H. pylori} infection. (p<0.05) This finding coincided with the results of a study carried out in Ethiopia [20] and another in Egypt. [68]

As for protein rich food stuffs, it was postulated that \textit{H. pylori} could survive in some animal products rich in protein, including meat and dairy products at temperature below 30°C. Moreover, such foods could serve as source of amino acids which support the growth of this bacterium in the stomach [69] Drinking coffee supports the growth of \textit{H. pylori} by suppressing acid production in the stomach. Coffee drinking was also claimed to be involved in hyper stimulation and increased levels of stress related hormones such as cortisol, adrenaline and norepinephrine [18]; which in turn could negatively influence the activity of the immune system supposed to combat \textit{H. pylori}. On the other hand, Rana (2007) reported tea consumption as a protective factor against \textit{H. pylori} infection. [70]

On the other hand, eating spicy food was not significantly associated with a higher \textit{H. pylori} prevalence in the present work. This is in line with the findings of a Libyan study [19].

\textit{H. pylori} infection remains the most frequent and persistent bacterial infection worldwide; thus the need for an accurate diagnosis of infection is imperative. The ideal test for detection of \textit{H. pylori} infection should be non-invasive, highly accurate, widely available and inexpensive [71].

The invasive techniques for diagnosis of \textit{H. pylori} are difficult, expensive and not preferred by the patients, therefore; a rapid and cost-effective detection method for diagnosis of \textit{H. pylori} infection is required. Therefore, non-invasive testing for \textit{H. pylori} has been strongly recommended as it is cheaper, more patient friendly than invasive methods and does not require very complicated laboratory facilities [72].

In the current study, \textit{H. pylori} infection significantly affected the haemoglobin level, MCV, MCH and RDW in studied cases (p<0.05). On the other hand no statistically significant difference between both positive and negative groups for \textit{H. pylori} regards their hematocrit level, MCHC, RBCs count, WBCs count, platelets count and ESR was recorded. Meanwhile, \textit{H. pylori} infection significantly affected the serum iron, serum ferritin and TIBC in studied cases of IDA. Several studies highlighted that after confirmation of eradication of \textit{H. pylori}, the mean values of hemoglobin and iron indices including ferritin have improved significantly without the use of iron supplementation which indicates improved absorption of dietary iron with subsequent improvement of IDA [72,73].

Stool Ag test is one of the non-invasive methods that is broadly used in the diagnosis of \textit{H. pylori} infection and had been known for the accuracy of its results and comparability to invasive methods. In the current research, \textit{H. pylori} stool Ag test was
considered the gold standard method for diagnosis of *H. pylori* infection. This is attributed to its previously reported high sensitivity and specificity (up to 97%) [74,75] and its excellent positive and negative predictive values regardless of *H. pylori* prevalence [74].

Compared to UBT, stool Ag test was reported by Frenck et al., [76] at Cairo University to be equivalent to its sensitivity and specificity. They concluded that UBT and stool Ag test had comparable high sensitivity (98 and 94%, respectively) and specificity (89% and 81%, respectively) and thus the stool Ag test has been evaluated as equivalent to the UBT.

*H. pylori* can be tested for using stool antigen test (HPSA) which is an enzymatic immunoassay to detect bacterial antigen of actual ongoing infection in stool is a reliable noninvasive marker in the primary diagnosis and in the monitoring of post treatment outcome. [77]

5. CONCLUSION

- *Helicobacter pylori* is highly associated risk factor with cases of iron deficiency anaemia
- Family history of *H. pylori* infection, limited level of education and some dietary habits as eating high protein diet, skipping meals, drinking coffee and tea are considered as risk factors for acquiring *H. pylori* infection.
- *Helicobacter pylori* infection significantly affects haemoglobin level, blood indices, serum iron and ferritin and TIBC.

6. RECOMMENDATIONS

- This study is small and did not exclude other causes of IDA by extensive laboratory work and interventions including upper and lower endoscopy. Therefore, further large scale case control studies are warranted among study participants to evaluate the relationship between *H. pylori* infection and IDA and to set successful management regimens for treatment of cases.
- Effective treatment strategies must be applied for treatment of *H. pylori* infection to guard against development of IDA.

CONSENT

A written consent was signed for approval to be part of this research work.

ETHICAL APPROVAL

This study was fully funded by the authors only. Author identifying information are present on the title page that is separate from the manuscript.

This study received ethical approval from the High Institute of Public Health (HIPH) Ethics Committee.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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