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Research Article

Serum Iron in Adults with Helicobacter pylori, Entamoeba histolytica, and Co-infection: A Cross-Sectional Study from Iraq

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A B S T R A C T

Introduction: Helicobacter pylori and *Entamoeba histolytica* infections can impair iron status through blood loss, gastric acid–dependent malabsorption, and inflammation; the impact of co-infection is less clear.

Objective: To compare serum iron among adults with *H. pylori*, *E. histolytica*, and co-infection, and test-negative controls

Methods: A cross-sectional study (November 2023–March 2024) was conducted among 100 symptomatic adults grouped by stool antigen/ microscopy.

Results: Mean serum iron was lowest in co-infection, intermediate in single infections, and highest in controls (omnibus $p < 0.001$). Stage-wise analyses showed stepwise declines from acute to chronic (all $p \leq 0.01$)

Conclusion: Co-infection was associated with substantially lower serum iron than single infections or no infection. Screening for iron deficiency should be considered in endemic settings; prospective studies including ferritin, transferrin saturation, haemoglobin, and C-reactive protein (CRP) are warranted.

Keywords: *Helicobacter pylori, Entamoeba histolytica*, Co-Infection, Serum Iron Levels, Iron Deficiency, Gastrointestinal Infections, Anaemia, Iron Metabolism, Chronic Infection, Acute Infection

Introduction

Iron deficiency remains the most prevalent micronutrient disorder worldwide, contributing to fatigue, cognitive dysfunction, reduced work capacity, and adverse pregnancy outcomes. Gastrointestinal infections can exacerbate iron deficiency via occult blood loss, impaired gastric acid–dependent absorption, and inflammatory hepcidin-mediated iron sequestration. *Helicobacter pylori* (*H. pylori*), a gram-negative bacterium infecting nearly half of the global population, is linked to chronic gastritis, peptic ulcer disease, MALT lymphoma, and increased risk of gastric cancer, though many infections remain asymptomatic.[1–3] *Entamoeba histolytica (E. histolytica)*, a protozoan causing amoebiasis, primarily resides in the large intestine, with cysts being the infectious stage. Symptomatic infection may cause abdominal discomfort, bloody diarrhoea, colitis, systemic infection, and iron-deficiency anaemia due to mucosal invasion and tissue destruction.[4–7] While the individual impact of these pathogens on iron metabolism is established, the combined effect of co-infection on circulating iron remains underexplored. This study aimed

to compare serum iron levels among symptomatic adults with *H. pylori*, *E. histolytica*, co-infection, and test-negative controls, hypothesising a stepwise decrease from controls to single infections to co-infection.

Materials and Methods

Patient Selection

Over the duration of the study (November 2023 to March 2024), the researcher was able to include 100 patients, with 50 patients being males and the remaining 50 being females. These patients were aged between 18 and 55 years and showed signs of discomfort in the stomach, fever, vomiting, and diarrhoea due to digestive complications. The various patients involved in this research were selected from numerous private laboratories along with the Al-Yarmouk Teaching Hospital, Iraq. Exclusion criteria were patients with previous gastrointestinal surgery, gastric cancer, severe liver or renal insufficiency, smokers prior to enrolment, patient irregularities in tea or coffee drinking, non-steroidal anti-inflammatory drugs (NSAIDS), anticoagulant/ antiplatelet agents, chemotherapy agents, patients with pregnancy in the last one month.

We conducted a cross-sectional study using consecutive sampling of eligible attendees. After stool testing, participants were allocated into four mutually exclusive groups: *H. pylori* positive/ *E. histolytica* negative; *E. histolytica* positive/ *H. pylori* negative; co-infection (both positive); and controls (both negative). Recruitment continued until approximately 25 participants were included in each group. The sample size was determined for a four-group comparison using one-way ANOVA. Assuming Cohen's f = 0.35, α = 0.05, power = 0.80, and k = 4 groups, the required sample was ~92 participants (~23 per group). We targeted N = 100 (25 per group) to maintain balanced group sizes. For the co-infection versus control contrast, assuming σ = 18 µg/dL and Δ = 15 µg/dL, ~23 participants per group were required. Serum iron was measured on an automated analyser using the ferrozine colourimetric method according to the manufacturer's instructions, with fasting samples, daily two-level quality control, and external quality assessment participation.

Eligibility & Grouping*:

Operational Staging** (acute ≤ 14 days; subacute 15–90; chronic > 90)

Study Groups

This ensured the formation of groups with equal numbers of respondents to enhance the evaluation of the findings by comparing the scores obtained by the four groups.

Stool Sample Collection

Faeces specimens were obtained using sterile, labelled, non-leaking 30 mL wide-mouthed plastic containers with solid lids for preservation and moisture conservation. The samples were split into two portions: one to be used in direct microscopic examination stages of intestinal protozoa, and the other for chromogenic slide agglutination immunoassay for *H. pylori*. According to the immunochromatographic test reagent kits, the sensitivity recorded was 95%.

Stool Sample Examination

Following the preparation that had been done on the slide in the previous step, the stool samples were examined under a microscope using methodology utilising a drop of 0.9% normal saline solution for direct wet smears, 1% local iodine dye, or the ether-formalin concentration technique at either 40X or 100X magnification. In addition, the fast antigen test for *H. pylori* shed was carried out using a cassette in accordance with the directions provided by the manufacturer.

Blood Sampling and Serum Iron Level Measurement

Skin-deep cuts and scratches, minor abrasions and vein punctures were conducted to obtain 4–5 mL of blood samples each from all the participants. Subsequently, the blood was spun in gel tubes and allowed to clot at room temperature for 30 minutes. The obtained samples were centrifuged for 10 minutes at a speed of 3000 rpm, after which the two phases were separated. To prevent interference with the tests, on account of the freeze-thaw cycle, a 5 mL micropipette was used to centrifuge each sample and transfer it into three different sterile Eppendorf tubes. Each blood sample that was collected was processed for analysis in the shortest time possible, and it did not take more than 12 hours for the entire procedure to complete. Determinations of the concentrations of serum iron were accomplished by spectrophotometric analysis on the Hitachi model no. 737 spectrophotometer made in Tokyo, Japan. The determination of serum iron was done with the Cobas 111 Auto-Analyser manufactured by Roche Diagnostics GmbH, Germany. For the purpose of performing this test, an automatic analyser, the Iron Liquicolor photometric, colourimetric test for iron with LCF CAB method analytic test kit was used, manufactured by Human Biochemical and Diagnostic GmbH, Germany.

Assay Specifications

- **Wavelength:** 623 nm
- **Optical Path:** CARTAGE: Incidence of 1 cm
- **Temperature:** 25 °C
- **Pipetting scheme:** Volume of sample/standard: 50 and Reagent (RGT): 1000, and Distilled water: 1000
- **Procedure:** They were mixed well and spun at 1000 g for 30 minutes at 25 °C. The absorbance of the sample was defined as ΔA sample and the absorbance of the

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standard as ΔA standard. They were then compared with the result of the reagent blank.

Calculation of Iron Concentration

However, if a different wavelength range is to be used, usually in the range of 620–640 nm, then the standard provided along with the kit has to be used. Iron concentration calculations are as follows:

C = 100 × ΔA sample/ΔA standard (µL/dL)
C = 17.9 × ΔA sample/ΔA standard (µmol/L)

Ethical Considerations

All the participants had provided written informed consent. The given study was carried out with the approval of the relevant ethics boards and in adherence to the ethical norms to protect the participants' rights and physical and mental well-being.

Statistical Analysis

Normality and variance homogeneity were assessed using the Shapiro–Wilk test and Levene's test, respectively. Serum iron (continuous) was compared across the four independent groups using one-way ANOVA; post-hoc pairwise comparisons were planned with Tukey's HSD when ANOVA assumptions were met, or Kruskal–Wallis with Dunn–Bonferroni when violated. Categorical distributions (stage, age bands, sex, BMI, and serum-iron bands) were compared using Pearson's chi-square test; Fisher's exact test was reserved for sparse cells. All tests were two-sided with $\alpha = 0.05$ and were conducted in SPSS v25.

Results

Table 1 summarises the previous studies that have investigated the prevalence of *H. pylori*, *E. histolytica* or their co-infection on the basis of different age groups, gender and risk factors. Conclusively, it demonstrates that *H. pylori* was present in the early onset of the infection-aggravation phase at 36%, hence could be eradicated more easily than *E. histolytica*, which was often present in subacute and chronic phases, implying that it may linger for longer periods. Analysing the age factor, it was found that there was statistically no variation among various age groups in regard to infection rates (p value = 0.895), hence age may not be a determinant of infection for the subjects included in this study. It was observed that there was no strong correlation between infection and BMI groups, except that higher BMI groups had a slight indication for more co-infections; hence, the p values were statistically insignificant (p value = 0.472). These distributions point out that other physiological or demographic factors could be a moderating influence when it comes to infections. Therefore, there is a need for more research to discover the moderating influence of these parameters on the prevention and treatment of these infections, with a special focus on different demographics. The p value of 0.0001 obtained in the study on acute infection with *H. pylori* points to the areas where specific preventive measures should be employed.

Table 2 shows the levels of serum iron in the various interactive infection groups, as well as various pertinent findings related to *H. pylori*, *E. histolytica* and co-infection on serum iron levels. The findings show that on average, the concentration of iron in the body decreases when *E. histolytica* is present. The results showed that 80% of the patients having *E. histolytica* had serum iron above 60 µg/dL. This decrease in iron levels may be due to parasites affecting its absorption, or intestinal injury, or bleeding. Co-infections worsen the level of iron loss as iron is already decreased, which is evident by the fact that 36% of the patients had iron levels in the lowest range (20–39 µg/dL). This identifies the reductions caused by the infections since the control group retained a higher iron status. The p values stated as 0.001 for the lowest range and 0.0001 for the total test depict that there are differences between the infected group and the control. Applying methods that are directed only to iron-deficient clients and ensuring that dangerous factors are controlled and treated are significant for avoiding further health issues, including anaemia, weak immunity, and impaired intellect. This is especially important in the areas where these diseases are so rampant.

Table 3 represents the mean serum iron profiles in *H. pylori*-positive patients, *E. histolytica*-positive patients, co-infected individuals and normal controls and shows the apparent effect of such infections on the iron status. *H. pylori* patients have a mean serum iron level of 72. In the second group, the mean concentration of C-peptide was equal to 76 µg/dL; this value varied between the participants with moderate dispersion. The iron level was considerably higher in this group than in the co-infected group and the healthy group; thus, *H. pylori* is capable of lowering iron levels by its mere presence. The average blood iron level of *E. histolytica*-infected persons was even less, with a value of 63.10 µg/dL and a variation that occasionally dropped to even 27. This value was as low as 0 µg/dL, which indicates the significantly adverse effects of this pathogen on the processes of iron intake and accumulation. The comparison of the two groups and significant differences in the iron level in the *E. histolytica*-positive group indicated the aggressiveness of the parasites. The co-infection group had the lowest average serum iron level (46). To the current author's knowledge, there have been no prior investigations making comparisons regarding the combined perinatal effects that both *E. histolytica* and *H. pylori* have on the human body's stock of iron at 78 µg/dL. On the other hand, the control group recorded an overall mean iron score of 104. Iron levels decreased to 74 µg/

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dL, which shows the effect of these infections on the iron status. The overall p value of 0.0001 that was observed in all groups suggests that these infections have a major effect on decreasing serum iron levels, further supporting the call for proper consideration of iron levels in infected patients to elucidate severe iron deficiency and related complications. Among infected participants (n = 75), 11 (14.7) were in the acute stage, 31 (41.3) were subacute, and 33 (44.0) were chronic. Group-wise, the *H. pylori* arm had 9 acute, 6 subacute, and 10 chronic cases; the *E. histolytica* arm had 2 acute, 13 subacute, and 10 chronic cases; and the co-infection arm had 0 acute, 12 subacute, and 13 chronic cases. Controls (n = 25) were test-negative and were not staged.

The role of *H. pylori* and *E. histolytica* in causing alterations in iron metabolism has increased over the recent past, as shown by the blood iron patterns among people with infection in the various phases of the two diseases in Table 4. In the case of *H. pylori*, the serum iron level reduced in the chronic phase (59.70 µg/dL) as compared to the acute phase (87.44 µg/dL), and this was highly significant (p = 0.0001). Hence, this chronic state of steady stomach inflammation, together with such malabsorption, underscores the significant impact of the increased presence of *H. pylori* on iron levels. This, perhaps, is rather probable given the fact that there is a certain relation between the two. During the infection caused by *E. histolytica*, there was a reduction of the serum iron from 74 µg/dL in the initial period, 80 µg/dL during the acute state, to 52.26 µg/dL during the chronic stage of amoebiasis infection. This reduction happens in the manner outlined in the above pattern of reduction of total R and D expenditure. Thus, in the chronic stage, the recorded variation (standard deviation = 14.24) indicates that the effect of the disease on the infected individuals differs, wherein some suffer severe iron deficiency. The findings suggest that there is a fairly significant decrease in the serum iron level in the subacute stage in relation to co-infections compared to the chronic stage, with a value of 58.17 µg/dL. Significantly, this decline is noteworthy to the extent that its importance is pronounced in all the evaluated groups (p = 0.0001). These infections contribute collectively towards a reduction in iron storage and thus highlight the importance of interventions to prevent severe anaemia.

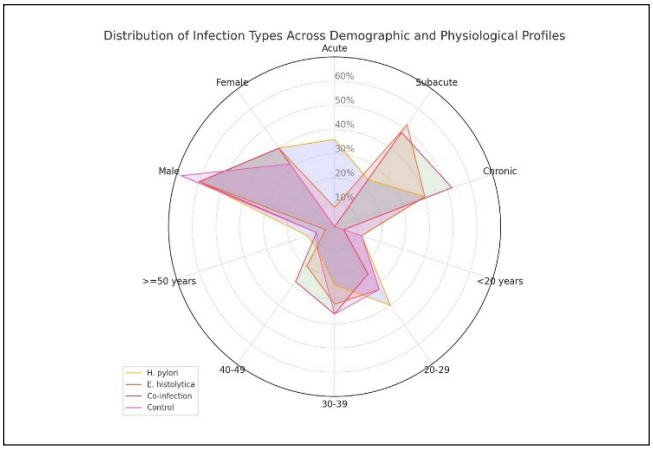

Figure 1.Distribution of Infection Type across Demographic and Physiological Profiles

Table 1.Detailed Statistical Analysis as per Infection Type, Age, Gender, and BMI

Category	H. pylori n (%)	E. histolytica n (%)	Co-Infection n (%)	Control n (%)	p Value
Type of infection					
Acute	9 (36.0)	2 (8.0)	—	—	0.0001*
Subacute	6 (24.0)	13 (52.0)	12 (48.0)	—	
Chronic	10 (40.0)	10 (40.0)	13 (52.0)	—	
Age (years)					
< 20	3 (12.0)	3 (12.0)	1 (4.0)	3 (12.0)	0.895
20–29	10 (40.0)	8 (32.0)	6 (24.0)	8 (32.0)	
30–39	6 (24.0)	8 (32.0)	9 (36.0)	9 (36.0)	
40–49	3 (12.0)	5 (20.0)	7 (28.0)	3 (12.0)	
≥ 50	3 (12.0)	1 (4.0)	2 (8.0)	2 (8.0)	
Gender					
Male	15 (60.0)	15 (60.0)	15 (60.0)	17 (68.0)	0.917
Female	10 (40.0)	10 (40.0)	10 (40.0)	8 (32.0)	
BMI					
Underweight (< 18.5)	2 (8.0)	4 (16.0)	4 (16.0)	—	0.472
Normal (18.5–24.9)	9 (36.0)	7 (28.0)	4 (16.0)	11 (44.0)	
Overweight (25–29.9)	10 (40.0)	10 (40.0)	11 (44.0)	9 (36.0)	
Obese (≥ 30)	4 (16.0)	4 (16.0)	6 (24.0)	5 (20.0)	

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Table 2.Serum Iron Levels across Different Infection Groups

Serum Iron (µg/dL)	*H. pylori* n (%)	*E. histolytica* n (%)	Co-Infection n (%)	Control n (%)	p Value
20–39	-	2 (8.0)	9 (36.0)	-	0.001*
40–59	5 (20.0)	3 (12.0)	7 (28.0)	-	-
60–79	13 (52.0)	20 (80.0)	9 (36.0)	1 (4.0)	-
80–99	6 (24.0)	-	-	11 (44.0)	-
100–119	-	-	-	6 (24.0)	-
120–139	1 (4.0)	-	-	7 (28.0)	-
Mean ± SD (Range)	72.76 ± 16.35 (52.0–131.0)	63.10 ± 13.37 (27.0–78.6)	46.78 ± 16.24 (21.0–69.3)	104.74 ± 18.89 (78.8–138.0)	0.0001*

Table 3.Serum Iron Levels by Infection Stage

Group	Acute (µg/dL) Mean ± SD (Range)	Subacute (µg/dL) Mean ± SD (Range)	Chronic (µg/dL) Mean ± SD (Range)	p Value
H. pylori	87.44 ± 17.11 (71.0–131.0)	72.52 ± 6.25 (63.6–78.4)	59.70 ± 5.11 (52.0–69.7)	0.0001^
E. histolytica	74.80 ± 5.37 (71.0–78.6)	69.65 ± 6.00 (60.5–78.5)	52.26 ± 14.24 (27.0–70.3)	0.001^
Co-infection	-	58.17 ± 10.81 (33.0–69.3)	36.27 ± 13.07 (21.0–62.0)	0.0001#
Overall p value	0.345	0.001^	0.0001^	-

Discussion

In our study of infected participants (n = 75), stages were distributed as acute (14.7%, 11/75), subacute (41.3%, 31/75), and chronic (44.0%, 33/75). Group-wise, 9 *H. pylori* cases were acute, 6 were subacute, and 10 were chronic; 2 *E. histolytica* cases were acute, 13 were subacute, and 10 were chronic; and in the co-infection group, none were acute, 12 were subacute, and 13 were chronic. Stage distribution differed significantly across infection types (p = 0.0001, Table 1). These patterns suggest that *E. histolytica* and co-infection more frequently persist beyond the acute phase, consistent with the reports of prolonged or recurrent intestinal disease and tissue invasion causing sustained morbidity.[8,9] On the other hand, *H. pylori* is classically a chronic, often asymptomatic infection with lifelong persistence if left untreated.[8–10] Our stage snapshot captured a non-trivial acute proportion within the symptomatic population (36% of *H. pylori* being acute within its group), likely reflecting care-seeking during symptomatic flares rather than true onset timing. We observed no significant association between infection category and age (p = 0.895) or gender (p = 0.917) (Table 1). Prior work shows age-linked acquisition in childhood and intrafamilial transmission for *H. pylori*,[11] with broad epidemiologic variation across settings[8–10]. For *E. histolytica*, burden varies with local sanitation, exposures, and travel endemicity rather than sex per se.[12,13] Thus, our null associations may reflect contextual exposures in this symptomatic adult sample and limited cell sizes within strata rather than the absence of demographic effects. BMI categories did not differ significantly across infection groups (p = 0.472), although a modest tilt toward co-infection in higher BMI strata was noted (Table 1). Mechanistically, chronic low-grade inflammation and hepcidin-mediated iron sequestration are enhanced in metabolic dysregulation, potentially compounding infection-associated iron handling defects.[14] Given our exploratory signal and multiple comparisons, these BMI patterns should be interpreted cautiously and confirmed in larger studies. Serum iron differed markedly across groups (overall p = 0.0001, Table 2). The mean ± SD values (range) observed in the controls was 104.74 ± 18.89 µg/dL (78.8–138.0), *H. pylori* group was 72.76 ± 16.35 µg/dL (52.0–131.0), *E. histolytica* group was 63.10 ± 13.37 µg/dL (27.0–78.6), and in the co-infected group, the value was 46.78 ± 16.24 µg/dL (21.0–69.3). Distributional bands underscored this gradient: 36% of co-infected patients were in the 20–39 µg/dL range versus 0% of controls, and 80% of *E. histolytica* clustered at 60–79 µg/dL, while 44%

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of controls were in the 80–99 µg/dL and 28% were in the 120–139 µg/dL group. These findings align with mechanisms whereby *H. pylori* impairs gastric acid–dependent iron absorption and may provoke occult blood loss from gastritis or ulceration,[8–10] and *E. histolytica* causes mucosal invasion, bleeding, and inflammation-driven iron sequestration[14,15]. The co-infection group's lowest mean iron (46.78 µg/dL) supports an additive or synergistic depletion of circulating iron through combined malabsorption, blood loss, and cytokine-induced hepcidin upregulation.[16,17] Stage analyses demonstrated progressive iron decline with chronicity (Table 4). For *H. pylori*, iron fell from 87.44 ± 17.11 µg/dL in acute to 72.52 ± 6.25 µg/dL in subacute and 59.70 ± 5.11 µg/dL in stages (p = 0.0001). For *E. histolytica*, iron fell from 74.80 ± 5.37 µg/dL in acute to 69.65 ± 6.00 µg/dL in subacute and 52.26 ± 14.24 µg/dL in chronic stages (p = 0.001). For co-infection, iron declined from 58.17 ± 10.81 µg/dL in subacute to 36.27 ± 13.07 µg/dL in chronic stages (p = 0.0001). These within-pathogen gradients are coherent with escalating mucosal injury, ongoing inflammatory signalling, and cumulative iron loss over time. The larger variance in chronic *E. histolytica* (SD = 14.24) suggests heterogeneous disease severity, also noted in symptomatic series with variable endoscopic and clinical manifestations.[18] We found the most profound iron depletion in co-infected patients. Work from clinical settings with overlapping enteric pathogens has reported substantial anaemia burdens and parasite-associated iron deficiency.[19,20] Our data extend this by quantifying a stepwise decrement from controls to single infections to co-infection, supporting the hypothesis that concurrent gastric and colonic pathology can compound iron malabsorption and losses.[21,22] The clinical implications are considerable. Routine iron assessment (including ferritin, transferrin saturation, and inflammatory markers) should be considered in symptomatic adults tested for *H. pylori* or *E. histolytica*, given the substantial decrements in serum iron we observed (down to 46.78 µg/dL in co-infection). Pathogen-directed therapy with follow-up iron repletion strategies is biologically justified, given mechanisms of impaired absorption, bleeding, and inflammation-driven sequestration.[23] Screening for co-infection may be warranted in patients with unexpectedly severe or refractory iron deficiency, particularly in endemic or high-risk contexts.[24,25]

The strengths of this study include parallel evaluation of single vs co-infection and stage-wise iron profiles in a single symptomatic study, demonstrating consistent gradients with strong statistical signals (overall p = 0.0001, multiple within-group p ≤ 0.001). Limitations include modest subgroup sizes (e.g., absence of acute co-infection stage), cross-sectional design (no post-eradication iron recovery data), reliance on serum iron alone without ferritin/ C-reactive protein (CRP) or transferrin indices, and potential confounding from unmeasured dietary or helminthic exposures.

Conclusion

Serum iron was significantly lower in infected groups than controls, lowest in the case of *H. pylori–E. histolytica* co-infection, and declined from acute to chronic stages. Co-infection appeared to exacerbate iron depletion; routine iron assessment and prospective studies including ferritin, transferrin saturation, haemoglobin, and CRP are warranted.

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