Original Article

Iraqi National Journal of Medicine. January 2026, Volume 8, Issue 1

Serum Iron Profile in Sudanese Patients Infected by *Helicobacter Pylori*

Rimaz Gurashi[1], Abuagla M. Dafalla[2], Suhair A. Ahmed[1], Shereen Faisal[1], AbdElkarim A. Abdrabo[1], Amira S. Khalafalla[2], GadAllah Modawe[3]

[1] Al-Neelain University, Faculty of Medical Laboratory Sciences, Department of Clinical chemistry, Khartoum, Sudan. [2] University of Gezira, Faculty of Medical Laboratory Sciences, Clinical Chemistry Department, Wad Medani, Sudan. [3] Omdurman Islamic University, Faculty of Medicine and Health Sciences, Department of Biochemistry, Omdurman, Sudan.

ABSTRACT

Background: *Helicobacter pylori* is a Gram-negative, spiral-shaped, motile bacterium that represents one of the most widespread chronic infections in humans, affecting nearly half of the global population. Iron is a vital micronutrient required for almost all living organisms, including *H. pylori*, which relies on iron to establish colonization. Although several studies have indicated an association between iron deficiency (ID) or iron deficiency anemia (IDA) and *H. pylori* infection, the precise mechanisms remain incompletely understood. **Aim**: To assess the serum iron profile among Sudanese patients with *H. pylori* infection. **Methods**: Analytical case-control hospital-based study was conducted at Bahri Hospital, Khartoum state during the period from December 2018 to April 2019. Eighty subjects were involved; forty patients with *H. pylori* and forty healthy individuals as a control group. Serum total iron and Total Iron Binding Capacity (TIBC) concentrations were estimated using the colorimetric method with a Mindray Analyzer. Serum total ferritin concentrations were estimated using an ELISA kit method with a TOSOH Analyzer. **Results**: The mean ± SD of serum iron, TIBC, and ferritin in the case groups were 92.80±34.38, 304.65±50.19, and 159.73±80.58, respectively. In the controls, the corresponding values were 109.00±22.51, 323.75±32.14, and 200.95±45.09, respectively. These parameters were significantly lower in cases compared with controls ($p<0.05$). **Conclusions**: Sudanese patients with *H. pylori* infection exhibited significantly lower serum iron, TIBC, and ferritin levels compared with controls. No significant association was detected between iron profile parameters and patient age.

Keywords: Serum iron, *H. pylori* infection, ferritin, TIBC

Corresponding author: GadAllah Modawe. E-mail: gadobio77@gmail.com
Disclaimer: The authors have no conflicts of interest.

DOI: https://doi.org/10.37319/iqnjm.8.1.7

Received: 4 OCT 2025 Accepted: 20 DEC 2025 Published online: 15 JAN 2026

INTRODUCTION

Helicobacter pylori is among the most prevalent chronic bacterial infections worldwide, affecting an estimated 4.4 billion people, nearly 59% of the global population.[1] The bacterium colonizes the gastric mucosa by traversing the mucus barrier and adhering to epithelial cells, with a preference for areas of mucosal injury. In Africa, prevalence rates surpass 80%, and infection is often acquired in childhood, persisting into adulthood unless eradicated.[2-4] Iron deficiency anemia (IDA) is the most common nutritional deficiency globally, affecting about

30% of the population.[5] Because the human body lacks the capacity to synthesize iron, its homeostasis depends on dietary intake, gastrointestinal absorption, and recycling processes.[6,7] *H. pylori* infection alters gastric physiology through mechanisms such as hypochlorhydria and mucosal inflammation, thereby reducing iron absorption and increasing susceptibility to IDA.[8-11] Moreover, chronic gastritis and ulceration linked to infection can contribute to blood loss and further depletion of iron stores. Considering the high prevalence of both *H. pylori* infection and iron deficiency, especially in low-resource settings, investigating their interrelationship is crucial for effective clinical management and public health strategies. This study was therefore designed to assess the serum iron profile in Sudanese patients infected with *H. pylori*.

MATERIALS AND METHODS

Study population: This study was a case-control hospital-based study conducted at Bahri Hospital, Khartoum State, during the period from December 2018 to April 2019. Eighty subjects were involved: forty Patients with H. pylori and forty healthy individuals in the control group were included in this study. Inclusion and exclusion criteria: Eligible participants included adults with confirmed *H. pylori* infection (Stool Antigen Test) and healthy controls. Exclusion criteria included pregnancy, smoking, patients with laboratory evidence of malaria in the previous month, chronic illnesses such as chronic liver disease, liver cirrhosis, chronic renal failure, tuberculosis, blood disorders like thalassemia, acute leukemia, hemoglobinopathies, clotting disorders, gastric carcinoma or other cancers, patients who had undergone gastrostomy, ileostomy, or colostomy, patients receiving radiotherapy or chemotherapy, and patients taking medicine causing folic acid deficiency, such as Gabapentin, Methotrexate, Trimethoprim, and Pyrimethamine were excluded from this study. Data collection: Demographic and clinical data were collected through structured questionnaires. Venous blood specimens were drawn between 9:00 and 12:00 a.m. under aseptic conditions. Samples were allowed to clot, centrifuged at 3500 rpm for 10 minutes, and processed according to Clinical and Laboratory Standards Institute guidelines. Serum iron and TIBC were determined via a colorimetric method (Mindray Analyzer), while ferritin was measured using an ELISA technique (TOSOH Biochemistry Analyzer). Ethical consideration: This study was approved by the ethical committee of the Faculty of Medical Laboratory Science of Al-neelain University.

Data analysis**:** Data were expressed as mean ± standard deviation (SD). Statistical comparisons between groups were conducted using independent t-tests, while Pearson correlation analysis assessed associations between study parameters and age. A p-value ≤0.05 was considered statistically significant.

.

RESULTS

The demographic characteristics of the study population are shown in Figures 1–3. As illustrated in Figure 1, both sexes were represented in nearly equal proportions, and no significant sex-related differences were observed in iron profile parameters. Figure 2 demonstrates the distribution of patients according to the duration of symptoms, where individuals with long-standing symptoms formed a noticeable proportion of the sample. This subgroup showed significantly reduced serum iron, TIBC, and ferritin levels, indicating that prolonged infection has a stronger impact on iron depletion. Figure 3 depicts the pattern of presenting symptoms, primarily gastrointestinal complaints consistent with *H. pylori* infection. The presence and chronicity of these symptoms correlate with the observed reductions in iron indices, supporting the role of persistent gastric inflammation in impairing iron absorption. The mean ± SD of serum iron, TIBC, and ferritin in the case groups were 92.80±34.38, 304.65±50.19, and 159.73±80.58, respectively. The mean ± SD of serum iron, TIBC, and ferritin in the control groups were 109.00±22.51, 323.75±32.14, and 200.95±45.09 Table1. The Mean **±**SD of serum iron, TIBC, and ferritin in male groups were 90.96±34.37, 303.52±51.57, and 160.08±98.26, respectively, while the mean ± SD of serum iron, TIBC, and ferritin in female groups were 95.87±35.38, 306.53±49.53, and 159.13±95.85 (Table 2), respectively. There were no significant differences between study parameters. The mean **±**SD of serum iron, TIBC, and ferritin in patients with long-standing symptoms were 87.82±33.65, 295.27±47.32, and 155.91±98.05, respectively. The mean ± SD of serum iron, TIBC, and ferritin in patients with occasional symptoms were 94.69±30.86, 308.20±42.92, and 161.17±89.15 (Table 3), respectively. The study parameters were significantly decreased in patients with long-standing symptoms compared to those with short-term symptoms. The scatter distribution shows no significant relationship between

the two variables, indicating that iron concentrations were not affected by age among the study participants. This finding aligns with the overall statistical analysis, which confirmed the absence of age-related differences in iron, TIBC, and ferritin levels. Therefore, the decline in iron indices observed in infected patients is attributable to *H. pylori* infection rather than age-dependent factors. Figure 5 shows the correlation between age and Serum TIBC. The scattered distribution of data points demonstrates the absence of any meaningful relationship between the two variables. This agrees with the study's statistical findings, which showed no significant age-related differences in iron indices. Accordingly, the reduction in TIBC observed in infected patients appears to be driven by *H. pylori* infection itself rather than by age.

The correlation between age and serum ferritin, shown in the scatter plot (Figure 6), indicates no significant relationship between the two variables, as data points are irregularly distributed with no clear pattern. This aligns with the statistical results showing no age-related differences in ferritin, confirming that fluctuations in ferritin among the study population arise from *H. pylori* infection rather than age.

Table 1: Comparison of serum iron profile among study groups

Parameters	Case (Mean ± SD)	Control (Mean ± SD)	P-value
Total Iron (µg/dl)	92.80±34.38	109.00±22.51	0.015
TIBC (µg/dl)	304.65±50.19	323.75±32.14	0.044
Ferritin (ng/dl)	159.73±80.58	200.95±45.09	0.020

Table 2: Serum iron profile among patients according to sex

Parameters	Male (Mean ± SD)	Female (Mean ± SD)	P-value
Total Iron (µg/dl)	90.96±34.37	95.87±35.38	0.668
TIBC (µg/dl)	303.52±51.57	306.53±49.53	0.857
Ferritin (ng/dl)	160.08±98.26	159.13±95.85	0.977

Table 3: Serum iron profile among patients according to duration of symptoms

Parameters	Long Time (Mean ± SD)	Some Time (Mean ± SD)	P-value
Total Iron (µg/dl)	87.82±33.65	94.69±30.86	0.030
TIBC (µg/dl)	295.27±47.32	308.20±42.92	0.013
Ferritin (ng/dl)	155.91±98.05	161.17±89.15	0.038

Table 4: Serum iron profile among patients according to family current infection

Parameters	Yes (Mean ± SD)	No (Mean ± SD)	P-value
Total Iron (µg/dl)	75.00±37.78	98.73±31.64	0.043
TIBC (µg/dl)	279.00±55.08	313.20±46.29	0.034
Ferritin (ng/dl)	111.10±97.54	175.93±94.49	0.031

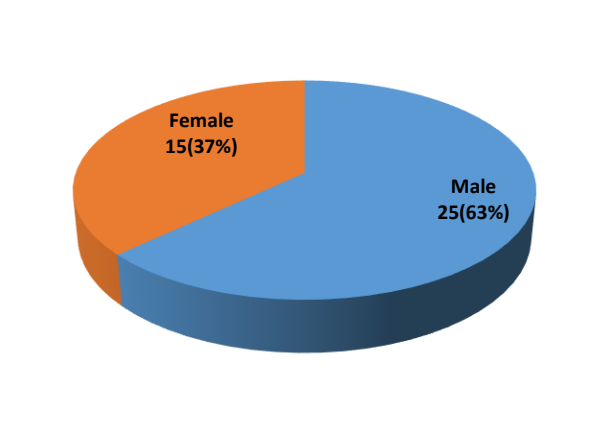

Figure 1: Distribution of patients according to sex

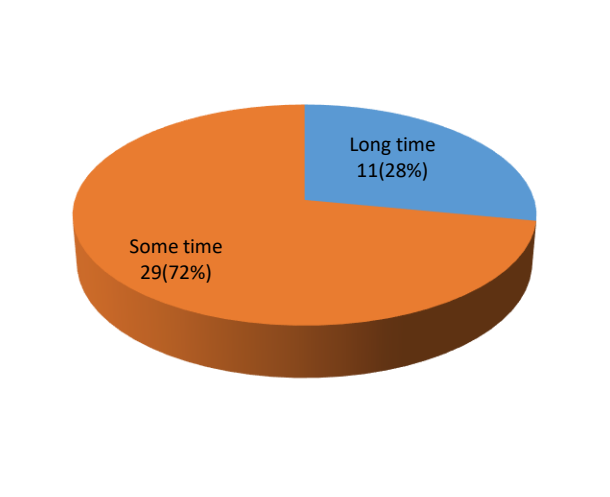

Figure 2: Distribution of patients according to duration of the disease

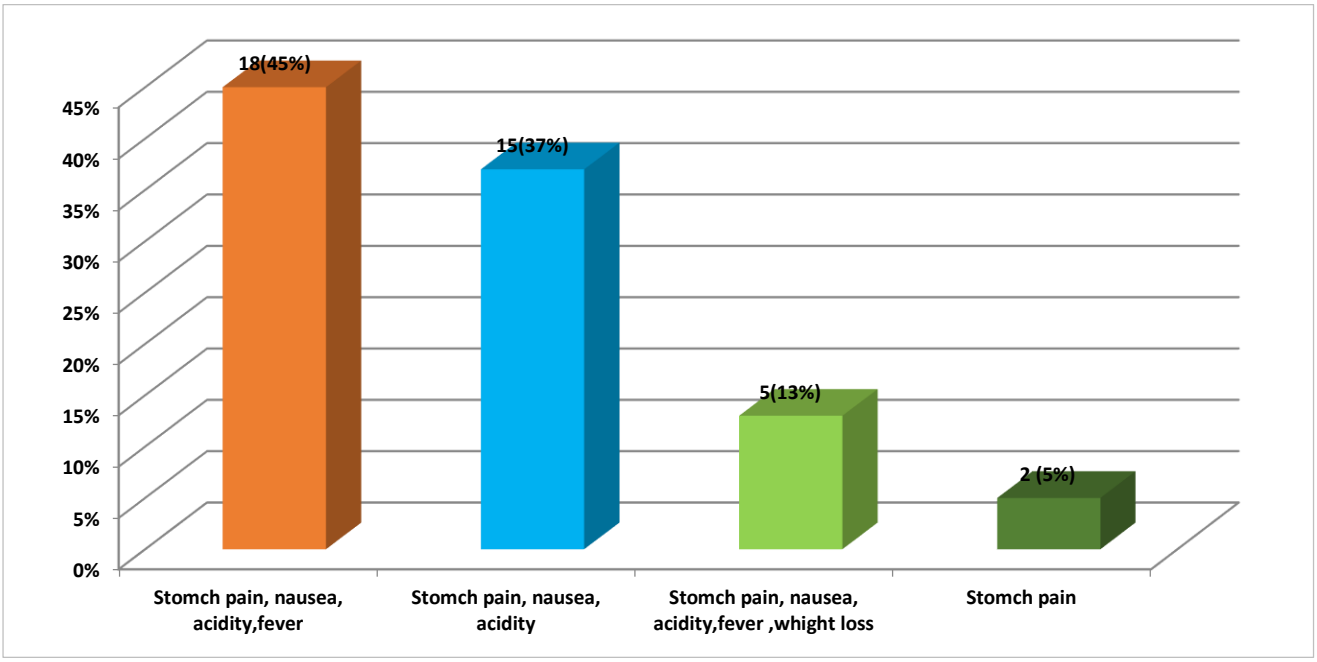

Figure 3: Distribution of patients according to symptoms

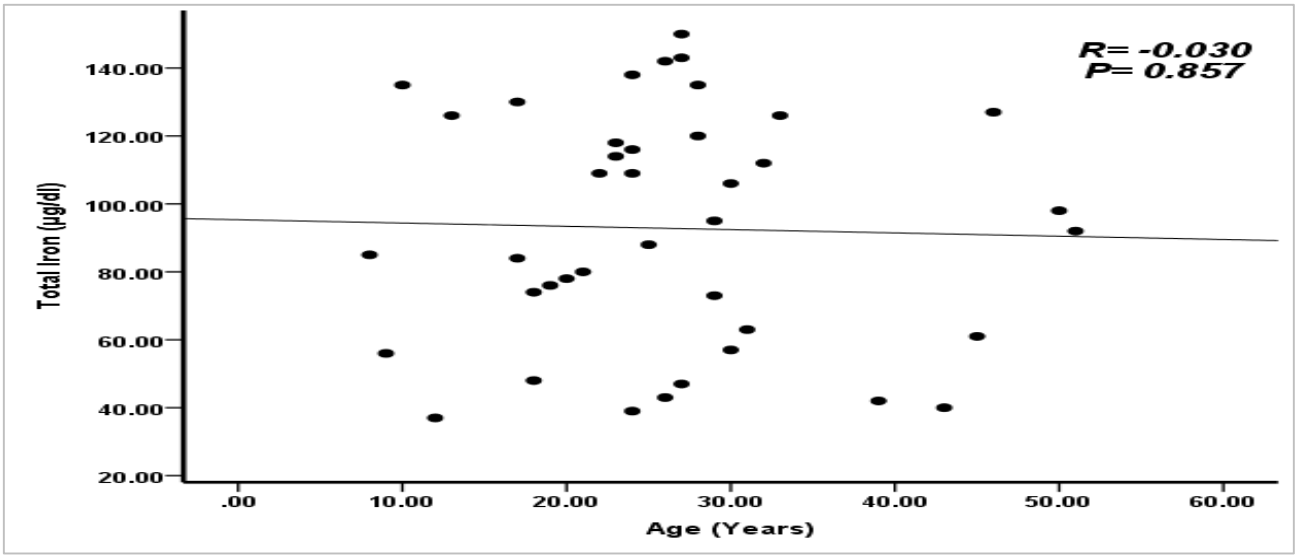

Figure 4: Correlation between age and serum total iron

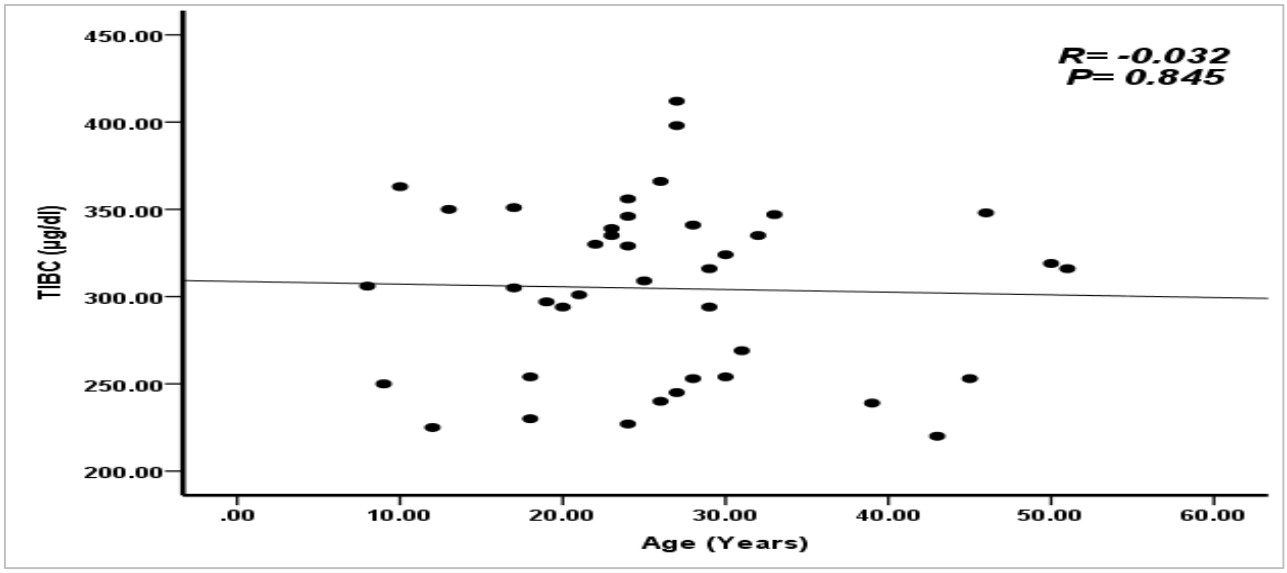

Figure 5: Correlation between age and serum TIBC

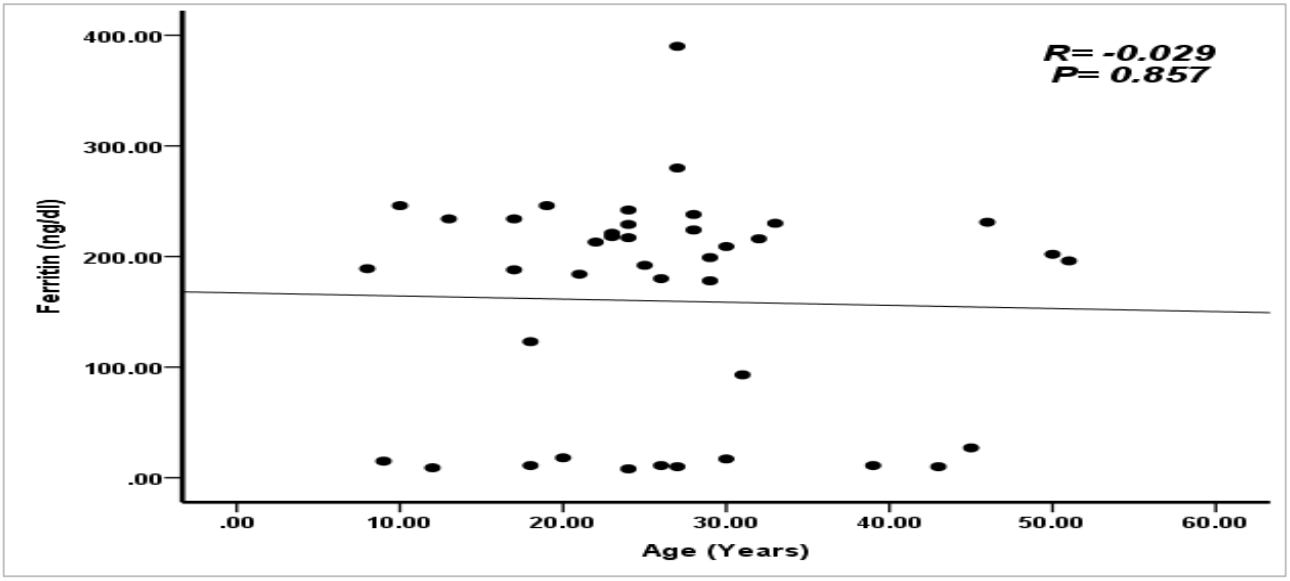

Figure 6: Correlation between age and serum ferritin

DISCUSSION

This study demonstrates that Sudanese patients infected with Helicobacter pylori exhibit significantly lower serum iron, TIBC, and ferritin levels compared with healthy controls. These findings provide strong evidence that *H. pylori* infection disrupts iron metabolism through multiple biological pathways. One of the primary mechanisms involves *H. pylori*-induced gastritis, which alters gastric acidity and interferes with the conversion of dietary non-heme iron into its absorbable ferrous form. Reduced gastric acid directly lowers iron absorption in the duodenum. Moreover, *H. pylori* is an iron-dependent organism that expresses several outer-membrane receptors capable of binding iron, transferrin, and lactoferrin, thereby competing with the host for available iron stores. This bacterial competition further contributes to the depletion of circulating iron. In addition, chronic gastric inflammation caused by persistent *H. pylori* infection stimulates the release of pro-inflammatory cytokines such as interleukin-6 (IL-6), which enhances hepatic production of hepcidin. Elevated hepcidin levels inhibit intestinal iron absorption and trap iron within macrophages, resulting in functional iron

deficiency even when iron stores are not fully exhausted. Long-standing infection may also lead to mucosal erosions and microscopic gastrointestinal blood loss, which can progressively reduce ferritin, the primary marker of iron storage. These proposed mechanisms are strongly supported by previous studies. John et al. (2018) reported that *H. pylori* infection was significantly associated with unexplained iron deficiency anemia, attributing the pattern to reduced absorption and inflammation-mediated hepcidin elevation. Hassan et al. (2020) similarly found marked decreases in ferritin among infected children, linking the findings to both impaired absorption and chronic blood loss. Studies from Egypt, Ethiopia, and other regions of sub-Saharan Africa have consistently shown significantly lower iron indices in *H. pylori*-positive individuals compared with non-infected controls, reinforcing the international relevance of our findings. In the present study, no significant sex-related differences were observed in iron parameters, confirming that the effect of *H. pylori* on iron homeostasis is similar among males and females. Likewise, the lack of correlation between age and iron profile indices suggests that the infection affects different age groups in a relatively uniform manner. However, patients with longer symptom duration demonstrated significantly lower iron, ferritin, and TIBC levels, highlighting the cumulative impact of chronic infection on nutrient depletion and iron storage. Overall, these results underscore the role of *H. pylori* as an important contributor to reduced iron stores and disturbed iron metabolism. The consistency between our findings and previous regional and international studies supports the need for routine evaluation of iron status in patients with chronic *H. pylori* infection, as well as the consideration of eradication therapy as a potential intervention to prevent or correct iron deficiency.

This study demonstrates that Sudanese patients infected with H. pylori had significantly lower serum iron, TIBC, and ferritin levels compared with healthy controls. These findings provide compelling evidence that *H. pylori* may interfere with iron metabolism through several mechanisms, including impaired gastric acid production, damage to the mucosal lining, and chronic gastrointestinal blood loss. Consistent with earlier work, no substantial sex-based differences were observed in iron indices, suggesting that sex has little influence on the effect of *H. pylori* infection on iron status.[12] In contrast, longer symptom duration was strongly associated with progressively lower serum iron and ferritin levels, supporting the notion that persistent infection exacerbates nutritional deficits. Patients reporting concurrent family infection also exhibited diminished iron parameters, which may reflect shared environmental exposures or genetic predispositions. Our results align with studies from Egypt and other regions that have identified comparable reductions in iron status among *H. pylori*-infected populations.[13,14] Although the correlation between age and iron indices was weak in this study, prior experimental and clinical research indicates that age could modulate host susceptibility to infection and related outcomes.[15] Collectively, the data reinforce the perspective that *H. pylori* constitutes a significant risk factor for iron deficiency and anemia in endemic regions. Further longitudinal and interventional research is warranted to clarify causality and to determine whether eradication of *H. pylori* can restore iron balance and reduce the prevalence of anemia. Several biological mechanisms may explain the reduction in serum iron and ferritin observed in *H. pylori*-infected patients in the present study. Chronic active gastritis and *H. pylori*-induced hypochlorhydria impair the solubilization and reduction of dietary non-heme iron, thereby limiting its absorption in the proximal small intestine. In addition, *H. pylori* is a highly iron-dependent organism that expresses multiple outer-membrane receptors capable of capturing iron, transferrin, and lactoferrin from the gastric lumen, creating direct competition with the host for available iron stores. The persistent mucosal inflammation triggered by the infection also promotes the release of pro-inflammatory cytokines such as interleukin-6, which stimulates hepatic hepcidin production and leads to sequestration of iron within macrophages and reduced availability of circulating iron. Over time, chronic gastritis and peptic ulceration may cause occult gastrointestinal blood loss, further exacerbating iron depletion and progressively lowering ferritin as an indicator of iron stores. These mechanisms, which have been described in both clinical and experimental studies, are consistent with our finding of significantly decreased serum iron, TIBC, and ferritin levels in *H. pylori*-infected Sudanese patients, particularly among those with longer symptom duration.

CONCLUSIONS

The study concluded that there are significantly reduced serum iron, TIBC, and ferritin levels in *H. pylori*-infected individuals compared with healthy individuals. These findings indicate that the infection negatively affects iron

absorption and storage. No correlation was found between iron profile parameters and age or sex, while patients with longer symptom duration exhibited greater reductions in iron status. Overall, *H. pylori* may contribute to iron deficiency, emphasizing the need for early detection and treatment.

Funding information: Nothing to declare.

REFERENCES

1. Denic M, Touati E, De Reuse H. Pathogenesis of *Helicobacter pylori* infection. *Helicobacter*. 2020 Sep;25:e12736.
2. Eyoum Bille BB, Kouitcheu Mabeku LB. Relationship between active *Helicobacter pylori* infection and anemia, iron deficiency, iron deficiency anemia: A cross-sectional study in a sub-Saharan setting. *JGH Open*. 2022 Aug;6(8):554-68.
3. John S, Baltodano JD, Mehta N, Mark K, Murthy U. Unexplained iron deficiency anemia: does *Helicobacter pylori* have a role to play? *Gastroenterology Report*. 2018 Aug;6(3):215-20.
4. Karkhah A, Ebrahimpour S, Rostamtabar M, Koppolu V, Darvish S, Vasigala VK, et al. *Helicobacter pylori* evasion strategies of the host innate and adaptive immune responses to survive and develop gastrointestinal diseases. *Microbiol Res*. 2019 Jan 1;218:49-57.
5. Kumar S, Hingorjo B, Maheshwari N, Fatima S. Serum ferritin and iron profile in *Helicobacter pylori* infected young adult males reporting at a tertiary care hospital. *Prof Med J*. 2019 Jun 10;26(06):896-901.
6. Geta TG, Gebremedhin S, Omigbodun AO. Prevalence and predictors of anemia among pregnant women in Ethiopia: Systematic review and meta-analysis. *PLoS One*. 2022 Jul 27;17(7):e0267005.
7. Paul P, Maiti D, Adhikary T. Iron deficiency anemia—An updated overview. *Int J Res Pharm Pharm Sci*. 2021;6(4):28-33.
8. Ren S, Cai P, Liu Y, Wang T, Zhang Y, Li Q, et al. Prevalence of *Helicobacter pylori* infection in China: A systematic review and meta-analysis. *J Gastroenterol Hepatol*. 2022 Mar;37(3):464-70.
9. Sabbagh P, Mohammadnia-Afrouzi M, Javanian M, Babazadeh A, Koppolu V, Vasigala VR, et al. Diagnostic methods for *Helicobacter pylori* infection: ideals, options, and limitations. *Eur J Clin Microbiol Infect Dis*. 2019 Jan;38(1):55-66.
10. Resál T, Farkas K, Molnár T. Iron Deficiency Anemia in Inflammatory Bowel Disease: What Do We Know? *Front Med*. 2021.
11. Smith S, Fowora M, Pellicano R. Infections with *Helicobacter pylori* and challenges encountered in Africa. *World J Gastroenterol*. 2019 Jul 7;25(25):3183.
12. Kishore G, Ejaz M, Kumar J, Lal A, Tahir H, Anjum Z, et al. Association between *Helicobacter pylori* infection and serum iron profile. *Cureus J Med Sci*. 2021 Sep 13;13(9).
13. Hassan MA, Kasem IA, Ali AS. Correlation between *Helicobacter pylori* infection and iron deficiency anemia in school-aged children. *Egypt J Hosp Med*. 2020 Oct 1;81(2):1489-98.
14. Walle M, et al. The association of *Helicobacter pylori* infection with the risk of anemia: A population-based study. *BMC Infect Dis*. 2024.
15. Ghobashi EA, Abd-Ellatieffb HA, Goda WM, Hamada R, Abou-Rawash AR, Hassan SM. Clinico-Pathological Studies on *Helicobacter pylori* Infection in Albino Rats. *Damanhour J Vet Sci*. 2022 Dec 1;9(1):1-6.