Crimson Publishers
Wings to the Research

Research Article

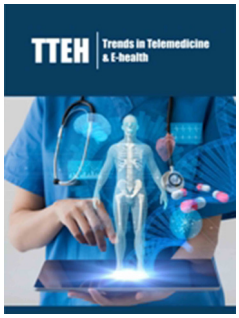
Haematological Indices and Serum Ferritin Among Patients with *Helicobacter Pylori* Infection at Federal Teaching Hospital Owerri Nigeria

Wealthy-Emmanuel Enajite[1]*, Onyeshinwa Jacklyn[2] and Aloy-Amadi Oluchi[1]

[1]Department of Haematology and Blood Transfusion Science, Federal University Otuoke, Nigeria

[2]Department of Haematology and Blood Transfusion Science, Imo State University, Nigeria

ISSN: 2689-2707



***Corresponding author:** Wealthy-Emmanuel Enajite, Department of Haematology and Blood Transfusion Science, Federal University Otuoke, Nigeria

Submission: September 17, 2026
Published: November 12, 2026

Volume 6 - Issue 5

How to cite this article: Wealthy-Emmanuel Enajite*, Onyeshinwa Jacklyn and Aloy-Amadi Oluchi. Haematological Indices and Serum Ferritin Among Patients with *Helicobacter Pylori* Infection at Federal Teaching Hospital Owerri Nigeria. Trends Telemed E-Health. 6(5). TTEH. 000650. 2026.
DOI: 10.31031/TTEH.2026.06.000650

Copyright@ Wealthy-Emmanuel Enajite, This article is distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use and redistribution provided that the original author and source are credited.

Abstract

Background: *Helicobacter pylori* infection is common in low- and middle-income settings and has been linked with iron-restricted erythropoiesis and anaemia. Local evidence describing red-cell indices and iron stores among infected adults in southeastern Nigeria remains limited.

Objective: To compare haemoglobin concentration, mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration, red cell distribution width and serum ferritin between adults with *H. Pylori* infection and apparently healthy *H. Pylori* -negative controls, examine sex-related differences among infected participants, and assess correlations between ferritin and red-cell indices.

Methods: This comparative cross-sectional study included 60 adults recruited at Federal Teaching Hospital, Owerri: 30 patients with confirmed *H. Pylori* infection and 30 age- and sex-matched controls. EDTA-anticoagulated blood was analysed for haemoglobin and red-cell indices, while serum ferritin was measured by sandwich enzyme-linked immunosorbent assay. Group means were compared with independent-samples t tests, and Pearson correlation was used to assess relationships between ferritin and haematological variables. Statistical significance was set at $p < 0.05$.

Result: Compared with controls, infected participants had lower haemoglobin (11.49 ± 0.45 versus 13.84 ± 0.24g/dL), MCV (87.25 ± 1.49 versus 90.79 ± 1.24fL), MCH (27.70 ± 0.50 versus 29.30 ± 0.37pg), MCHC (33.04 ± 0.25 versus 33.66 ± 0.19g/dL) and ferritin (40.20 ± 2.64 versus 115.77 ± 5.16ng/mL), but higher RDW (14.53 ± 0.44 versus 12.51 ± 0.20%); all $p < 0.001$. No parameter differed significantly between infected males and females. Ferritin showed no significant correlation with haemoglobin, MCV, MCH, MCHC or RDW (all $p > 0.05$).

Conclusion: *H. Pylori* infection was associated with lower haemoglobin, lower average red-cell indices, greater red-cell size variability and markedly lower ferritin than in controls. Because mean MCV remained within a normocytic range and ferritin was interpreted without inflammatory markers or a prespecified deficiency threshold, the findings support anaemia with comparatively reduced iron stores rather than proving iron-deficiency anaemia in every infected participant. Larger studies incorporating C-reactive protein, serum iron, transferrin saturation and post-eradication follow-up are warranted.

Keywords: *Helicobacter pylori*; Anaemia; Serum ferritin; Haemoglobin; Red-cell indices; Red cell distribution width and Nigeria

Introduction

Helicobacter pylori is a curved, Gram-negative, microaerophilic organism adapted to persistent colonisation of the human gastric mucosa. Its urease activity, flagellar motility, adhesins and immune-evasion mechanisms allow it to survive close to the epithelium despite

gastric acidity and a sustained inflammatory response. Infection is usually acquired in childhood and, without treatment, may persist for decades. Although many infected people remain asymptomatic, *H. Pylori* is the principal cause of chronic active gastritis and an important determinant of peptic ulcer disease, gastric mucosa-associated lymphoid tissue lymphoma and non-cardia gastric adenocarcinoma [1,2]. The distribution of *H. Pylori* is unequal. Global syntheses show a heavier burden in Africa and other regions where household crowding, unsafe water, limited sanitation and lower socioeconomic status facilitate transmission. Nigeria therefore carries a clinically relevant intersection of frequent *H. Pylori* exposure, nutritional vulnerability, parasitic disease and anaemia. These overlapping conditions complicate attribution: a low haemoglobin concentration in an infected adult may reflect iron loss, inadequate intake, malaria, haemoglobinopathy, chronic inflammation, renal disease, menstrual loss or several mechanisms acting together [3,4].

Interest in the extra gastric effects of *H. Pylori* has grown because eradication may improve selected disorders in some patients. The best-established haematological associations involve otherwise unexplained iron deficiency and immune thrombocytopenia. Proposed pathways linking infection to impaired iron status include microscopic gastric blood loss, reduced gastric acidity and ascorbic-acid availability, bacterial acquisition of host iron, reduced intake during dyspeptic illness, and inflammation-mediated changes in iron trafficking. These mechanisms are biologically plausible but do not operate uniformly; bacterial strain, extent of gastritis, diet, host response and duration of infection may modify the phenotype [5-7]. Ferritin is central to the laboratory assessment of iron stores, yet its interpretation is not straightforward in an infectious or inflammatory setting. Low ferritin is highly specific for depleted stores, whereas a value within the conventional reference interval cannot reliably exclude deficiency when inflammation increases ferritin synthesis. The World Health Organization consequently recommends that ferritin be interpreted alongside indicators of infection or inflammation when these are prevalent. C-reactive protein, alpha-1-acid glycoprotein, serum iron, transferrin or total iron-binding capacity and transferrin saturation can help distinguish absolute deficiency from iron sequestration [8,9].

Red-cell indices add morphological context. Mean corpuscular volume reflects average erythrocyte size; MCH and MCHC describe haemoglobin content and concentration; and RDW expresses heterogeneity in cell size. Early iron restriction may first increase RDW as newly produced cells diverge from older circulating erythrocytes. MCV and MCH may fall later, and a group mean can remain normocytic even when some individuals are iron deficient. Interpretation should therefore integrate indices rather than assigning a diagnosis from a single mean value [10,11].

Evidence from meta-analyses supports an association between *H. Pylori* and iron deficiency or iron-deficiency anaemia, but heterogeneity is considerable. Differences in age, sex, diagnostic tests, ferritin thresholds, inflammatory adjustment, background nutrition and study design limit direct transfer of findings across populations. African data are especially important because the high prevalence of infection coexists with multiple competing causes of anaemia. Locally generated evidence can identify whether infected patients present a recognisable laboratory pattern and can guide more focused investigations [5,12]. At Federal Teaching Hospital, Owerri, patients investigated for dyspepsia and *H. Pylori* infection are frequently assessed with routine haematology, yet the combined pattern of red-cell indices and ferritin has not been sufficiently described. Establishing whether infected patients differ from comparable *H. Pylori* -negative adults may support earlier recognition of anaemia and prompt appropriate evaluation. It may also discourage empiric iron therapy without identifying continuing gastric disease, while avoiding the opposite error of assuming that infection explains every low haemoglobin result [1,2].

H. Pylori occupies the mucus layer overlying gastric epithelial cells. Urease converts urea to ammonia and carbon dioxide, buffering the organism's immediate environment, while flagella permit movement through mucus. Adhesins maintain attachment and virulence determinants such as cytotoxin-associated gene A and vacuolating cytotoxin A alter epithelial signalling, barrier function and immune responses. The resulting chronic gastritis may remain superficial or progress toward atrophy and altered acid secretion, thereby influencing nutrient absorption [1,2]. Clinical outcome reflects an interaction between organism, host and environment. Antral-predominant gastritis can increase acid output and predispose to duodenal ulceration, whereas corpus-predominant or atrophic gastritis can reduce acid secretion and impair non-haem iron absorption. This distinction matters in haematology because dietary ferric iron requires an acidic environment for solubilisation and reduction to the more absorbable ferrous form. Long-standing gastritis may therefore affect iron availability even without overt gastrointestinal bleeding [2,6].

Current consensus guidance treats *H. Pylori* as an infectious disease and recommends reliable test-and-treat strategies in appropriate clinical groups. Non-invasive options include urea breath testing and stool antigen testing, whereas biopsy-based tests may be used during endoscopy. Serology has limitations because antibodies may remain after clearance and cannot consistently distinguish active from past infection. Diagnostic heterogeneity can therefore introduce misclassification when participants are enrolled using different tests [2,13]. Body iron is tightly conserved because there is no regulated pathway for excretion. Dietary absorption in the duodenum balances basal losses, while macrophages recycle iron from senescent erythrocytes. Hepcidin, produced mainly by the liver, reduces iron export by binding ferroprotein. Inflammation increases hepcidin through interleukin-6-related signalling, sequestering iron within macrophages and reducing intestinal absorption. The marrow may consequently experience restricted iron supply even when storage iron is not entirely exhausted [7,9].

Ferritin is an intracellular iron-storage protein that appears in small amounts in serum. In otherwise healthy people, serum ferritin broadly tracks storage iron and a low value strongly supports absolute deficiency. During inflammation, liver disease, malignancy or cellular injury, however, ferritin may rise independently of usable iron. A mean ferritin value should not be labelled deficient unless

Copyright © Wealthy-Emmanuel Enajite

the assay-specific and population-appropriate threshold is defined and inflammatory context is considered [8,9]. Comprehensive iron assessment typically combines ferritin with transferrin saturation or serum iron and total iron-binding capacity. Soluble transferrin receptor, reticulocyte haemoglobin content and hepcidin may provide additional information where available. In resource-limited settings, these tests may not be routinely accessible, making the careful interpretation of full blood count indices particularly valuable. Even so, normal MCV does not exclude early iron deficiency, and reduced ferritin relative to controls does not establish deficiency in each participant [8,10].

Several mechanisms may act simultaneously. Inflamed gastric mucosa can bleed microscopically; erosions or ulcers may add overt or occult loss. Hypochlorhydria can reduce absorption of non-haem iron, while reduced gastric ascorbic acid may impair iron reduction. *H. Pylori* also expresses iron-acquisition systems that support bacterial persistence. Over time, these processes may create a negative iron balance, especially when dietary intake is marginal or physiological demand is high [5-7]. Inflammation adds a second pathway. Cytokine-driven hepcidin elevation reduces ferroprotein-mediated iron release, lowering circulating iron available for erythropoiesis. This functional restriction may coexist with absolute depletion. In such mixed states, ferritin may appear normal or only modestly reduced despite limited marrow iron. The laboratory phenotype may therefore be normocytic initially, with raised RDW or falling MCH preceding clear microcytosis [7,9,10].

Eradication studies provide indirect support for causality, particularly among patients with otherwise unexplained or refractory iron deficiency. Nevertheless, response is not universal, and study quality varies. The strength of association does not justify assuming that every infected adult has iron deficiency. A defensible clinical approach is to test for *H. Pylori* when indicated, document the type and cause of anaemia, eradicate confirmed infection using guideline-based therapy, and reassess response [5,12,13].

Empirical Evidence and Knowledge Gap

Systematic reviews have reported higher odds of iron deficiency and iron-deficiency anaemia among infected individuals, although pooled estimates show substantial between-study variability. The association appears stronger in selected groups such as children, adolescents and patients with unexplained or refractory deficiency. Differences in laboratory definitions and failure to adjust ferritin for inflammation remain recurring limitations [5,12].

Nigeria's epidemiological setting requires cautious interpretation. Malaria, intestinal parasites, nutritional insufficiency, menstrual loss and inherited haemoglobin disorders can alter haemoglobin and red-cell indices independently of *H. Pylori*. Hospital controls may also differ from community controls in unmeasured ways. Matching for age and sex improves comparability but cannot remove residual confounding from socioeconomic status, diet, medication exposure or occult disease [4,14]. The present study addresses part of this gap by reporting six related laboratory variables in infected adults and matched controls from one tertiary hospital. Its contribution lies in the internally consistent group comparison and the inclusion of sex-specific and correlation analyses. Its limitations, including small sample size, varied diagnostic methods and absence of inflammatory and expanded iron markers, define priorities for subsequent research [8,15].

Materials and Methods

Study area and design

A comparative cross-sectional study was conducted at Federal Teaching Hospital, Owerri, Imo State, Nigeria. The hospital is a tertiary referral centre serving Owerri and surrounding communities in southeastern Nigeria. The design compared contemporaneously measured laboratory variables in adults with confirmed *H. Pylori* infection and apparently healthy *H. Pylori* -negative controls. Although the source project used the term case-control, the absence of retrospective exposure ascertainment makes "comparative cross-sectional" the more precise epidemiological description [15,16].

Study population and sampling

Sixty adults aged 18 years or older were enrolled: 30 *H. Pylori* -positive patients attending gastroenterology or internal medicine services and 30 apparently healthy, age- and sex-matched controls without gastrointestinal complaints who tested negative for *H. Pylori*. The infected group included 11 males and 19 females. Participants were recruited after study information was provided and written informed consent was obtained. Matching was intended to reduce confounding by age and sex [15,16].

Eligibility criteria

Eligible cases were adults with *H. Pylori* infection confirmed by a standard diagnostic method, including rapid urease, stool antigen or urea breath testing, who had not received eradication therapy during the preceding three months. Controls were adults without gastrointestinal symptoms and with a negative *H. Pylori* test. Exclusion criteria for both groups included known haematological disorders, recent transfusion, chronic kidney or liver disease, malignancy or chemotherapy, pregnancy, acute infection or inflammatory disease, and recent iron or vitamin B12 therapy. Refusal of consent also excluded participation [2,13].

Sample size

The source project estimated the minimum per-group sample using the formula for comparing two independent means, with a two-sided 5% significance level, 80% power, an expected haemoglobin difference of 1.7g/dL and pooled standard deviation of 1.10g/dL. The calculation yielded approximately seven participants per group. The number was increased to 28 to improve precision and allow for losses, and 30 participants were ultimately recruited into each group. The inflation improved stability but was not a substitute for a prespecified allowance based on an anticipated non-response rate [15].

Copyright © Wealthy-Emmanuel Enajite

Sample collection and processing

Five millilitres of venous blood was collected aseptically from each participant. Two millilitres was dispensed into dipotassium EDTA, mixed gently and analysed within four hours for haematological variables. Three millilitres was placed in a plain tube, allowed to clot for 30-45 minutes and centrifuged at 3,000rpm for 10 minutes. Separated serum was analysed immediately or stored at -20 °C until ferritin measurement. Unique study codes linked specimens to records while limiting use of personal identifiers [17].

Laboratory analysis

Haemoglobin was determined by the cyanmethemoglobin method described in the source protocol. Blood was diluted in Drabkin reagent, allowed to form stable cyanmethemoglobin and read spectrophotometrically at 540nm against a reagent blank and haemoglobin standard. MCV, MCH, MCHC and RDW were obtained using an automated haematology analyser operating on electrical impedance and photometric principles. The indices were calculated from primary red-cell measurements by the analyser [10,17]. Serum ferritin was measured by sandwich ELISA. Standards and diluted sera were added to antibody-coated wells, incubated and washed before addition of enzyme-labelled detection antibody. Following further incubation and washing, tetramethylbenzidine substrate was added, the reaction was stopped with acid, and absorbance was read at 450nm. Concentrations were interpolated from the calibration curve and adjusted for dilution. Samples and standards were assayed in duplicate according to the kit procedure [8,17].

Quality assurance

Equipment was calibrated according to manufacturers' instructions. Background and start-up checks were completed before haematology analysis, and low, normal and high commercial control materials were examined daily; patient testing proceeded only when control results fell within acceptable limits. ELISA controls and duplicate determinations were included, reagent storage and expiry were monitored, and discrepant or implausible results were repeated. Pre-analytical control included correct tube selection, adequate mixing, timely separation and temperature-controlled storage [17,18].

Ethical considerations

Ethical approval was obtained from the Research Ethics Committee of Federal Teaching Hospital, Owerri. Participation was voluntary and based on written informed consent. Participants could withdraw without affecting their care. Records and specimens were coded, access was restricted to the research team, and clinically relevant abnormal results were made available for counselling and referral. The study followed the ethical principles of the Declaration of Helsinki [19].

Statistical analysis

Data were analysed with SPSS version 25. Continuous variables were summarised as mean ± standard deviation. Normality was assessed with the Shapiro-Wilk test and histogram inspection. Independent-samples t tests compared infected participants with controls and compared males with females in the infected group. Pearson correlation assessed linear relationships between ferritin and haemoglobin, MCV, MCH, MCHC and RDW. Tests were two-sided and $p < 0.05$ denoted statistical significance [15].

Results

All six measured variables differed significantly between groups. Infected participants had a mean haemoglobin concentration of 11.49 ± 0.45g/dL compared with 13.84 ± 0.24g/dL among controls ($t = -25.14$, $p < 0.001$). MCV, MCH and MCHC were also lower in the infected group. RDW was higher by approximately 2 percentage points, indicating greater heterogeneity of erythrocyte size. Mean ferritin was 40.20 ± 2.64ng/mL in infected participants and 115.77 ± 5.16ng/mL in controls ($t = -71.38$, $p < 0.001$) (Table 1).

Table 1: Haematological indices and serum ferritin in *H. pylori*-positive participants and controls. Values are mean ± SD.

Parameter	*H. pylori* positive n=30	Control n=30	t	p
Haemoglobin (g/dL)	11.49 ± 0.45	13.84 ± 0.24	-25.14	<0.001
MCV (fL)	87.25 ± 1.49	90.79 ± 1.24	-10.01	<0.001
MCH (pg)	27.70 ± 0.50	29.30 ± 0.37	-13.97	<0.001
MCHC (g/dL)	33.04 ± 0.25	33.66 ± 0.19	-10.94	<0.001
RDW (%)	14.53 ± 0.44	12.51 ± 0.20	22.95	<0.001
Serum ferritin (ng/mL)	40.20 ± 2.64	115.77 ± 5.16	-71.38	<0.001

Abbreviations: MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; MCHC: Mean Corpuscular Haemoglobin Concentration; RDW: Red Cell Distribution Width

None of the measured variables differed significantly between infected males and females. Male participants had marginally higher mean haemoglobin and ferritin, but the differences were small and compatible with sampling variation. The closest group means were observed for MCHC, while all p values exceeded 0.48. These results do not demonstrate equivalence between sexes; they show only that this sample did not provide evidence of a difference (Table 2).

Ferritin did not correlate significantly with any haematological variable among infected participants. The correlation with haemoglobin was weak and negative ($r = -0.099$), while correlations with MCV, MCH and RDW were very weak and positive. The largest coefficient was observed for MCHC ($r = 0.235$), but it remained weak and non-significant ($p = 0.2116$) (Table 3).

Copyright © Wealthy-Emmanuel Enajite

Table 2: Haematological indices and serum ferritin in H. Pylori-Positive participants by sex.

Parameter	Male n=11	Female n=19	t	p
Haemoglobin (g/dL)	11.53 ± 0.38	11.47 ± 0.50	0.34	0.738
MCV (fL)	87.34 ± 1.30	87.21 ± 1.62	0.23	0.82
MCH (pg)	27.78 ± 0.49	27.65 ± 0.52	0.7	0.491
MCHC (g/dL)	33.05 ± 0.25	33.03 ± 0.25	0.24	0.811
RDW (%)	14.58 ± 0.33	14.50 ± 0.50	0.48	0.632
Serum ferritin (ng/mL)	40.57 ± 2.70	39.99 ± 2.66	0.58	0.57

Values are mean ± SD. No comparison reached $p < 0.05$.

Table 3: Correlation of serum ferritin with haematological variables among *H. Pylori*-positive participants.

Variable	n	Pearson r	p
Haemoglobin	30	-0.099	0.604
MCV	30	0.013	0.945
MCH	30	0.073	0.7
MCHC	30	0.235	0.212
RDW	30	0.083	0.663

All correlations were statistically non-significant at $p < 0.05$.

Discussion

The principal finding was a consistent shift in erythrocyte and iron-storage measures among *H. Pylori* -positive adults. Compared with matched controls, infected participants had lower haemoglobin, MCV, MCH, MCHC and ferritin, together with higher RDW. This pattern supports an association between infection and anaemia accompanied by comparatively reduced iron stores. Because all measurements were made at one time point, the study cannot establish that infection preceded or caused the laboratory abnormalities [5,12,15]. The 2.35g/dL difference in mean haemoglobin was large and statistically persuasive within this dataset. A mean value of 11.49g/dL indicates that anaemia was common at group level, although individual classification requires sex-specific thresholds and information about smoking, altitude and pregnancy status.

The narrow standard deviations and extremely large t statistic warrant careful verification against raw data, because unusually low within-group variability can amplify test statistics. Reporting confidence intervals in future analyses would improve appreciation of precision [15,20]. Several pathways could explain the lower haemoglobin. Chronic gastritis may reduce iron absorption through altered acid secretion, reduce intake because of dyspepsia, or contribute to microscopic blood loss. Bacterial iron acquisition and inflammation-related hepcidin activity may further restrict iron availability. These mechanisms align with systematic-review evidence linking infection with iron deficiency and anaemia, particularly when no other cause is found. They nevertheless remain explanatory hypotheses here because gastric histology, blood loss, dietary intake and hepcidin were not measured [5-7].

The red-cell indices require nuanced interpretation. MCV was significantly lower in infected participants, but the mean of 87.25fL remained within the conventional adult normocytic range. It is therefore inaccurate to describe the group as uniformly microcytic. MCH was also lower, while MCHC showed a smaller absolute difference. These changes may represent early or mixed iron-restricted erythropoiesis, but individual frequency distributions and the proportion below validated cut-offs would be needed to identify microcytosis or hypochromia [10,11]. RDW was appreciably higher in infected participants. Increased RDW indicates broader variation in erythrocyte volume and can emerge early when newly produced iron-restricted cells circulate alongside older normocytic cells. It may also rise with mixed nutritional deficiencies, haemolysis, recent bleeding or recovery from anaemia, so it is not specific to iron deficiency. In this study, RDW strengthens the evidence for altered erythropoiesis but does not identify its cause on its own [10,11].

Ferritin showed the largest relative separation between groups: the infected mean was roughly one third of the control mean. This supports lower storage iron among infected participants and is biologically compatible with impaired iron balance. However, a mean of 40.20ng/mL cannot be called deficient without a prespecified diagnostic threshold, participant-level results and knowledge of inflammation. Ferritin may be raised during infection; consequently, some individuals with apparently acceptable values could still have iron deficiency, while others may have genuinely adequate stores [8,9]. The absence of ferritin correlations with haemoglobin and red-cell indices does not contradict the between-group findings. Ferritin reflects storage iron, whereas circulating erythrocytes integrate marrow conditions over approximately 120 days. Timing, inflammation and mixed causes of anaemia can weaken a cross-sectional linear relationship. Restriction of range within the infected group also reduces correlation: when participants have similarly clustered ferritin and index values, a meaningful association may be difficult to detect even if groups differ substantially [7,15].

Sample size is another explanation for the non-significant correlations. With only 30 infected participants, the study had limited power to detect weak or moderate coefficients, and confidence intervals around r would be wide. The point estimate for MCHC was larger than those for other indices but remained imprecise. Future work should calculate sample size specifically for the correlation objective rather than relying only on a calculation designed for a two-group haemoglobin comparison [15]. No significant sex difference was detected among infected participants.

Copyright © Wealthy-Emmanuel Enajite

This result should not be interpreted as proof that sex has no influence. Only 11 infected males were included, and the analysis did not adjust for age, menstruation, menopausal status, diet or other causes of iron loss. Matching was applied between cases and controls, not necessarily within the infected sex comparison. Larger stratified samples are required before concluding that the haematological effect of infection is identical in men and women [8,15].

The findings broadly agree with evidence that *H. Pylori* infection is associated with reduced iron status. Meta-analytic work has found increased odds of iron deficiency and iron-deficiency anaemia, while eradication plus iron therapy can improve outcomes in some affected patients. Variation across studies remains important. Populations differ in nutritional background, age distribution, diagnostic methods and prevalence of competing causes, and publication bias cannot be excluded. The present data add a Nigerian hospital-based observation rather than a definitive causal estimate [5,12]. Recent individual studies provide useful comparisons. Singh et al. [21] found *H. Pylori* positivity in 87.5% of 24 patients with iron-deficiency anaemia, compared with 29.2% of 24 healthy controls. This supports an association between infection and iron-deficiency anaemia, but their groups were selected by anaemia status, whereas the present study compared participants by infection status. Their findings therefore complement the lower haemoglobin and ferritin observed in Owerri without establishing that infection caused these changes [21].

Khan et al. [22] studied 236 adults who already had iron-deficiency anaemia and found lower haemoglobin, ferritin and MCV, together with higher total iron-binding capacity, in *H. Pylori*-positive patients. The direction of the haemoglobin and ferritin differences agrees with the present findings. However, their restriction to patients with established iron-deficiency anaemia differs from our comparison with apparently healthy controls. Their results cannot be used to classify every infected participant in the present study as iron deficient [22].

Not all studies report lower ferritin during infection. Kishore et al. [23], comparing 300 infected participants with 300 controls, found lower serum iron and total iron-binding capacity but higher ferritin in the infected group. This contrasts with the lower ferritin observed here and shows why ferritin must be interpreted in its inflammatory context. Differences in patient selection and the balance between depleted stores and inflammation-related iron sequestration may contribute to the contrasting patterns. Without inflammatory markers and a complete iron profile, the present study cannot determine the relative contribution of these mechanisms [8,9,23]. Lipopolysaccharide (LPS), an endotoxin component of the outer membrane of Gram-negative bacteria, is another possible contributor to host inflammatory responses. Its biological activity varies between organisms: experimental work by Muotiala et al. [24] showed substantially lower activity of *H. Pylori* LPS than enterobacterial LPS. Inflammation can restrict the iron available for erythropoiesis through hepcidin-mediated changes in iron handling, but this general pathway does not demonstrate an LPS-specific effect in our participants. Plasma LPS, inflammatory cytokines and hepcidin were not measured, so endotoxin-related interference with erythropoiesis remains a hypothesis for further investigation [9,24].

The broader literature also discusses LPS in relation to neurotoxicity and chronic disease, including the editorial by Martins [25]. That discussion concerns neurological mechanisms and does not directly establish impaired erythropoiesis in *H. Pylori* infection. Its relevance here is to encourage investigation of systemic bacterial products while keeping those proposed effects separate from the haematological associations actually measured in this study [25]. A clinically useful implication is that unexplained anaemia should prompt a structured assessment rather than isolated treatment. In a patient with dyspeptic symptoms or another indication for *H. Pylori* testing, confirmation and guideline-concordant eradication may remove a contributor to iron restriction. At the same time, clinicians should investigate other sources of blood loss, nutrition, renal disease, inflammation, malaria and haemoglobinopathy as appropriate. Eradication success should be confirmed with an active-infection test after the recommended interval [2,13].

Laboratory assessment should extend beyond ferritin when resources permit. Transferrin saturation, serum iron, transferrin or total iron-binding capacity, CRP and possibly soluble transferrin receptor would permit better classification of absolute deficiency, functional restriction and mixed anaemia. Reticulocyte haemoglobin can provide a more immediate measure of iron available for erythropoiesis. Peripheral blood film review would also show whether anisocytosis, microcytosis or another morphology supports the analyser results [8-10]. The study has several strengths. Cases and controls were equal in number and matched by age and sex, related erythrocyte variables were assessed together, and laboratory quality-control procedures were described. The inclusion of sex and correlation analyses broadened interpretation beyond a simple two-group comparison. Presenting exact means, standard deviations, t statistics and p values allows readers to judge both magnitude and statistical evidence [15,18].

Important limitations remain. Recruitment at one hospital and the small sample restrict generalisability. A cross-sectional comparison cannot determine temporality or evaluate response after eradication. Infection was accepted from more than one diagnostic method, and the study did not report test distribution, medication exposure such as proton-pump inhibitors, endoscopic findings, menstrual history, diet, socioeconomic status, malaria or stool-parasite assessment. Each could influence the observed association [2,13,15]. Laboratory limitations are equally relevant. Ferritin was not paired with CRP or alpha-1-acid glycoprotein, and expanded iron indices were unavailable. The manuscript reports group means but not individual diagnostic categories, confidence intervals or effect sizes. Multiple t tests were conducted without a stated multiplicity plan. The very small standard deviations and correspondingly extreme t statistics should be checked against

Copyright © Wealthy-Emmanuel Enajite

original analyser printouts and the raw spreadsheet before journal submission [8,15].

The title and interpretation should therefore remain descriptive. The data show lower ferritin and altered red-cell indices among infected participants; they do not demonstrate that *H. Pylori* caused iron-deficiency anaemia. A longitudinal study measuring inflammation and complete iron profiles before treatment and after confirmed eradication would provide stronger evidence. Multivariable analysis could then adjust for age, sex, diet, menstruation, malaria and other confounders [15,16].

Clinical and laboratory implications

For medical laboratory practice, the central lesson is that an apparently simple request for a full blood count and ferritin requires interpretive context. A result pattern of low haemoglobin, falling MCH, increased RDW and ferritin that is low or lower than expected should prompt review of the patient's symptoms, inflammatory state and potential blood loss. Analytical flags, histograms and the peripheral film should be examined rather than relying solely on printed numerical values. If results are discordant, repeat analysis and assessment of sample integrity are preferable to forcing the pattern into a single diagnostic label [8,10,18]. Ferritin reporting can be strengthened by providing method-specific reference intervals and an interpretive comment where inflammation is known or suspected. A laboratory should not equate every value inside the population reference interval with adequate iron stores. Reference intervals describe the distribution in a selected population, whereas clinical decision limits address the probability of deficiency. This distinction is especially relevant in settings where infection and inflammation are common. When CRP and transferrin saturation are available, reflex or coordinated testing may reduce both missed deficiency and unnecessary iron treatment [8,9].

From the clinician's perspective, *H. Pylori* should be considered one possible contributor to anaemia, not a complete explanation. The patient's age, sex, menstrual and obstetric history, gastrointestinal warning symptoms, diet, medication use and family history remain important. Men and postmenopausal women with confirmed iron deficiency may require evaluation for gastrointestinal blood loss according to clinical guidance. Premenopausal women may also have substantial gastrointestinal or nutritional contributors. Treating infection does not remove the obligation to investigate serious alternative causes when the presentation warrants it [2,7,13]. The findings also have implications for antimicrobial stewardship. A haematological association does not justify treatment without reliable evidence of active infection. When treatment is indicated, regimen choice should reflect current recommendations, previous macrolide or fluoroquinolone exposure and local resistance patterns. Completion, adverse effects and adherence should be addressed, and eradication should be confirmed. These steps are important because persistent infection could continue to influence gastric physiology, while repeated empirical therapy may select resistance and expose patients to avoidable adverse effects [2,13].

Research implications

A stronger follow-up study would use a prospective cohort or before-and-after eradication design. Participants could undergo a uniform active-infection test, full blood count, reticulocyte indices, ferritin, CRP, serum iron, transferrin or total iron-binding capacity and transferrin saturation at baseline. Repeating the panel after confirmed eradication would show whether haematological improvement tracks clearance. Including an untreated comparison group may be ethically or clinically inappropriate when eradication is indicated, but carefully timed observational follow-up can still provide useful evidence [2,8,13].

Future mechanistic studies could include plasma LPS alongside CRP, hepcidin, transferrin saturation and reticulocyte haemoglobin before and after confirmed eradication. The LPS assay would need validation for the sample matrix and the low biological activity of *H. Pylori* endotoxin. A plasma LPS result alone would not identify its bacterial source or prove marrow suppression; interpretation would require assessment of other infections and inflammatory conditions. These measurements could help test whether endotoxin exposure is associated with iron restriction and changes in erythropoiesis [9,24]. Participant-level classification would add more information than group means alone. Investigators should report the prevalence of anaemia using current sex-specific criteria, the proportion with low ferritin under explicitly stated thresholds, the frequency of microcytosis and raised RDW, and overlap between these categories. Regression models could estimate the independent association of infection with haemoglobin or iron deficiency after adjustment for age, sex, socioeconomic circumstances, diet, menstruation, malaria and parasite infection. Effect estimates with 95% confidence intervals should accompany p values [15,20].

Finally, qualitative and health-system questions deserve attention. Delayed presentation, cost of active-infection tests, access to endoscopy, availability of ferritin and CRP, and inconsistent post-treatment confirmation may determine whether the biological association translates into preventable morbidity. A multicentre Nigerian study that combines robust laboratory phenotyping with information on diagnostic access and treatment response would be more directly applicable to policy than a larger numerical comparison alone [3,4,8].

Conclusion

Adults with *H. Pylori* infection at Federal Teaching Hospital, Owerri, had significantly lower haemoglobin, MCV, MCH, MCHC and serum ferritin, and higher RDW, than matched *H. Pylori*-negative controls. No significant sex differences or ferritin correlations were demonstrated within the infected group. The results are compatible with anaemia, altered erythropoiesis and reduced iron stores associated with infection, but they do not establish individual iron-deficiency diagnoses or causation. Interpretation should be tempered by the normocytic mean MCV, the absence of inflammatory markers and expanded iron studies, and the small cross-sectional sample [5,8,15].

Copyright © Wealthy-Emmanuel Enajite

Recommendations

1. Patients with confirmed *H. Pylori* infection who have symptoms or risk factors for anaemia should receive a full blood count and a clinically appropriate iron assessment rather than haemoglobin testing alone [8-10].
2. Low or borderline ferritin in infected patients should be interpreted alongside CRP or another inflammatory marker and, where possible, transferrin saturation, serum iron and total iron-binding capacity [8,9].
3. Clinicians should investigate alternative and coexisting causes of anaemia and confirm successful *H. Pylori* eradication with an appropriate active-infection test [2,13].
4. Future multicentre studies should recruit larger, sex-balanced samples, use a uniform test for active infection, report individual deficiency categories and confidence intervals, and follow participants before and after eradication [15,16].
5. Before publication, the raw dataset and analyser records should be audited to confirm the unusually small standard deviations and large t statistics reported for the primary comparisons [15,18].

Declarations

1. Ethical approval and consent to participate: Ethical approval was obtained from the Research Ethics Committee of Federal Teaching Hospital, Owerri. All participants provided written informed consent.
2. Competing interests: The authors declare no competing interests.
3. Funding: No external funding was reported for this study.

References

1. Kusters JG, Van Vliet AHM, Kuipers EJ (2006) Pathogenesis of *Helicobacter pylori* infection. Clin Microbiol Rev 19(3): 449-490.
2. Malfertheiner P, Megraud F, Rokkas T, Javier Gisbert P, Jyh-Ming Liouet, et al. (2022) Management of *Helicobacter pylori* infection: The Maastricht VI/Florence consensus report. Gut 71(9): 1724-1762.
3. Hooi JKY, Lai WY, Ng WK, Michael Suen MY, Fox Underwood E, et al. (2017) Global prevalence of *Helicobacter pylori* infection: Systematic review and meta-analysis. Gastroenterology 153(2): 420-429.
4. Zamani M, Ebrahimtabar F, Zamani V, Miller WH, Alizadeh-Navaei R, et al. (2018) Systematic review with meta-analysis: The worldwide prevalence of *Helicobacter pylori* infection. Aliment Pharmacol Ther 47(7): 868-876.
5. Hudak L, Jaraisy A, Haj S, Muhsen K (2017) An updated systematic review and meta-analysis on the association between *Helicobacter pylori* infection and iron deficiency anemia. Helicobacter 22(1): e12330.
6. Franceschi F, Zuccalà G, Roccarina D, Gasbarrini A (2014) Clinical effects of *Helicobacter pylori* outside the stomach. Nat Rev Gastroenterol Hepatol 11(4): 234-242.
7. Camaschella C (2019) Iron deficiency. Blood 133(1): 30-39.
8. World Health Organization (2020) WHO guideline on use of ferritin concentrations to assess iron status in individuals and populations. World Health Organization, Geneva, Switzerland.
9. Ganz T, Nemeth E (2015) Iron homeostasis in host defence and inflammation. Nat Rev Immunol 15(8): 500-510.
10. Bain BJ, Bates I, Laffan MA (2017) Dacie and Lewis practical haematology. (12th edn), Elsevier, Amsterdam, Netherlands
11. Salvagno GL, Sanchis-Gomar F, Picanza A, Lippi G (2015) Red blood cell distribution width: A simple parameter with multiple clinical applications. Crit Rev Clin Lab Sci 52(2): 86-105.
12. Qu XH, Huang XL, Xiong P, Zhu CY, Huang YL, et al. (2010) Does *Helicobacter pylori* infection play a role in iron deficiency anemia? A meta-analysis. World J Gastroenterol 16(7): 886-896.
13. Chey WD, Leontiadis GI, Howden CW, Moss SF (2017) ACG clinical guideline: Treatment of *Helicobacter pylori* infection. American Journal of Gastroenterology 112(2): 212-239.
14. Olokoba AB, Gashau W, Bwala S, Adamu A, Salawu FK (2013) *Helicobacter pylori* infection in Nigerians with dyspepsia. Ghana Med J 47(2): 79-81.
15. Charan J, Biswas T (2013) How to calculate sample size for different study designs in medical research. Indian J Psychol Med 35(2): 121-126.
16. Setia MS (2016) Methodology series module 3: Cross-sectional studies. Indian Journal of Dermatology 61(3): 261-264.
17. Clinical and Laboratory Standards Institute (2017) Collection of diagnostic venous blood specimens (7th edn), CLSI standard GP41. CLSI, Wayne, Pennsylvania, USA.
18. Westgard JO (2016) Basic QC practices: Training in statistical quality control for medical laboratories (4th edn), Westgard QC, Madison, Wisconsin, USA.
19. World Medical Association (2013) World Medical Association declaration of Helsinki: Ethical principles for medical research involving human subjects. JAMA 310(20): 2191-2194.
20. World Health Organization (2024) Guideline on hemoglobin cutoffs to define Anaemia in individuals and populations. World Health Organization.
21. Singh D, Upadhyay V, Singh SB, Rimal D, Rawal OK, et al. (2025) Evaluating *Helicobacter pylori* infection as a risk factor for iron deficiency anemia: A case-control approach. Cureus 17(5): e84340.
22. Khan A, Fatima SMN, Ahmad Vohra SMM, Ahmad J, Ashfaq H, et al. (2026) Association between *Helicobacter pylori* infection and iron deficiency anemia in adult patients attending a tertiary care hospital. Cureus 18(6): e111431.
23. Kishore G, Ejaz M, Kumar J, Lal A, Tahir H, et al. (2021) Association between *Helicobacter pylori* infection and serum iron profile. Cureus 13(9): e17925.
24. Muotiala A, Helander IM, Pyhälä L, Kosunen TU, Moran AP (1992) Low biological activity of *Helicobacter pylori* lipopolysaccharide. Infect Immun 60(4): 1714-1716.
25. Martins IJ (2018) Bacterial lipopolysaccharides and neuron toxicity in neurodegenerative diseases. Neurology Research & Surgery 1(1): 1-3.

Copyright © Wealthy-Emmanuel Enajite