

Frequency of Iron Deficiency Anemia in patients with Helicobacter Pylori Gastritis

TARIQUE ALI¹, LAIQ SAID BACHA², FAZLI RABBI³, SHAISTA FAHEEM⁴, SAFIA BIBI⁵, NOWSHERAWAN⁶

¹Medical Officer, Department of General Medicine, Federal Govt Polyclinic Hospital, Islamabad

²District Medical Specialist, Department of General Medicine, THQ Hospital Chakdara Lower Dir

³Assistant Professor, Department of Medicine, Mardan Medical Complex, Mardan

⁴Medical Specialist, PNS Hafeez Naval Hospital, Islamabad

⁵Assistant Professor Physiology, Department of Physiology, Bannu Medical College, Bannu

⁶Associate Professor, Department of Internal Medicine, Pak International Medical College Hayatabad, Peshawar

Correspondence to: Fazli Rabbi, Email: drfazalrabbi8@gmail.com

ABSTRACT

Objective: Helicobacter pylori (H. pylori) causes worldwide infection that leads to chronic gastritis alongside multiple gastrointestinal disorders. The research shows that H. pylori increases the risk of developing iron deficiency anemia (IDA). Scientific research will determine the frequency of iron deficiency anemia (IDA) among patients suffering from H. pylori gastritis.

Methods: This study was done at the Department of Medicine at Federal Govt Polyclinic Hospital Islamabad over a period of six months. Material included 228 confirmed H. pylori infection patients via stool antigen testing. Hemoglobin levels (men < 13 g/dL, women < 12 g/dL); serum ferritin concentrations (men < 20 ng/mL, women < 10 ng/mL); diagnosis IDA. We performed statistical analyses, consisting of chi-square tests, to determine the association of IDA with patient demographics.

Results: We identified 56 (24.6%) of 228 H. pylori infected patients with IDA. Hemoglobin mean 13.2 ± 1.9 g/dL and serum ferritin mean 24.2 ± 10.4 ng/dL. There was no statistically significant difference in IDA prevalence between males (26.6%) and females (22.1%; $p = 0.432$). IDA prevalence showed no significant effect by age stratification ($p = 0.910$).

Conclusion: In fact, around one-fourth of patients with H. pylori infection had been observed to have IDA, which hinted at an association of the infection with iron metabolism disturbances. H. pylori infected individuals should receive routine screening for IDA to enable early intervention and better clinical outcomes. Work needs to be done to advance causal understanding and explore mechanisms.

Keywords: Helicobacter pylori infection, Iron deficiency anemia, Gastritis, Hemoglobin, Serum ferritin.

INTRODUCTION

H. pylori exists as a Gram-negative spiral bacterium that inhabits human gastric tissue leading to gastritis and peptic ulcer disease while potentially causing gastric malignant growths (Thakur 2017; Ali et al. 2024; Sykes & J.E. 2021). This bacterial pathogen affects half of the worldwide human population (Muhammad et al. 2012; Sethi et al. 2013). H. pylori infection prevalence exhibits major differences which stem from geographic locations together with socioeconomic status and ethnic background and dietary patterns (Merryweather & J. A. 2023). The bacterium presents a significant public health issue even though it often causes no symptoms yet (Kato et al., 2022; Kady et al., 2020).

Iron deficiency anemia remains a visiting public health challenge especially within developing nations (Mawani et al., 2016). The medical condition results in diminished blood hemoglobin levels creating symptoms such as fatigue and pale complexion and diminished mental abilities (Jbireal et al., 2020). The development of IDA emerges from dietary deficiencies caused by malnutrition combined with chronic blood loss through trauma and infections with parasitic organisms along with persistent inflammatory diseases according to Stein et al. (2016). The medical research community has recently shown increasing interest in the possible link between H. pylori infection and IDA based on newly available evidence (Tseng et al. 2019).

The mechanisms whereby H. pylori causes iron deficiency anemia remain unclear yet scientists believe multiple mechanisms play a role (Kato et al., 2022). H. pylori directly results in the development of chronic gastritis along with gastric atrophy and hypochlorhydria. This medical condition decreases the iron absorption potential of digestive systems and thus reduces blood iron levels (Basyigit et al., 2016). Studies show H. pylori uses iron as a nutrient source which leads to competition between the bacterium and human metabolism (Bürkli & S., 2021). H. pylori leads to elevated gastric blood loss through peptic ulcers and mucosal inflammation and microbleeding which results in greater iron deficiency (Hojsak & I., 2016).

Hepcidin functions as a hormone produced by hepatocytes

to control iron metabolism through its degenerative activity against ferroportin which prevents iron uptake (Nemeth et al., 2021). The H. pylori infection triggers inflammatory responses that result in elevated hepcidin levels yet infection without hepcidin elevation occurs normally (Burns et al., 2015; Duque et al., 2023) indicating that H. pylori infection impairs iron homeostasis thus causing anemia. Medical studies confirm patients with H. pylori infection show heightened serum hepcidin and prohepcidin concentrations that decrease after successful H. pylori elimination (Enko et al., 2019). Systemic iron absorption and utilization process seems to be regulated by H. pylori infections according to research by Hudak et al. 2017.

Multiple researchers have thoroughly analyzed the link between H. pylori infection and IDA across various populations (Hudak et al., 2017). Epidemiological and clinical studies demonstrate that individuals affected by H. pylori infection show decreased levels of hemoglobin and serum ferritin than those without H. pylori (Haile, 2021). Medical research indicates that H. pylori elimination leads to better iron status outcomes together with treatment resolution of anemia in infected patients (Tseng et al., 2019). Research data supports conflicting results about H. pylori infection as a source of IDA particularly when anemia develops from other causes including poor diet and parasitic diseases (Kato et al., 2022). Experts found anemia across 30.9% of H. pylori infected persons with dyspepsia according to Haile et al. (2021). Research from Hooi et al. (2017) found that H. pylori infects New Zealand adolescent women which leads to iron deficiency end points. This demonstrates how H. pylori impacts different populations. A study based in Haiti did not link H. pylori seropositivity tests to anemia prevalence rates although research is needed to uncover this observed discrepancy.

Physicians recommend H. pylori infection testing for screening IDA among patients with gastritis originating from H. pylori infection (Sadiq et al., 2022). Early diagnosis and treatment of H. pylori stands as the solution to control anemia and enhance your healthcare results (Elbehiry et al., 2023). The effectiveness of eradication therapy to resolve anemia differs among individuals because it depends on the existence of additional conditions leading to anemia (Kumar et al., 2022) and initial iron storage levels combined with dietary consumption (Kumar et al., 2022).

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The current research evaluated the connection between H. pylori infection and iron deficiency anemia prevalence among local residents. Determining the prevalence rate of IDA in patients with H. pylori gastritis was the main goal since clinicians heavily rely on these findings to manage their patients' anemia cases. Knowledge about the regularity of these conditions and their ability to reverse allows healthcare providers to establish appropriate early prevention and therapy plans.

METHODOLOGY

This research employed a cross sectional design which studied the patients in the Department of Medicine at Federal Govt Polyclinic Hospital Islamabad during six months from July 2020 to December 2020. Stool antigen testing diagnosed 228 subjects as having H. pylori infection. Healthy levels of hemoglobin together with serum ferritin readings served as indicators for iron deficiency anemia assessment.

The research studied patients who had H. pylori infection and dyspepsia under the inclusion criteria of participants aged between 18 and 70 years. The research excluded pregnant woman patients and those who received H. pylori eradication therapy within the prior three months and individuals with recent blood transfusion or a history of hematological malignancies.

The hospital ethical review body sanctioned the study through official approval. A structured questionnaire obtained data from patients regarding demographic information combined with medical histories and laboratory test results. The results from stool antigen tests confirmed H. pylori infection along with blood test complete picture (CP), and serum ferritin values helped determine iron deficiency anemia. Male patients had reduced hemoglobin to <13 g/dL and females to <12 g/dL but both genders had normal ferritin below 20 ng/ml for men and 10 ng/ml for women.

Data analysis required the use of SPSS version 21 for processing. The descriptive statistics calculated mean value and standard deviation points for quantitative data including age and hemoglobin and serum ferritin level measurements. The researchers presented quantitative gender distribution and anemia prevalence through frequency tables and percentages. Post stratification chi square tests provided results and statistical significance occurred when p value remained below 0.05.

RESULTS

This study included a total of 228 patients with diagnosed Helicobacter pylori (H. pylori) infection. Participants' ages ranged from 18 to 70 years of age with a mean of 42.8 ± 12.1 years. Table 1 shows that the gender distribution was 124 males (54.4%) and 104 females (45.6%). Finally, the study population was further stratified into two age groups of 18–50 years (64.9%) and 51–70 years (35.1%) (Table 2).

Out of 228 patients, 56 were diagnosed with iron deficiency anemia (IDA), 24.6% of the study population (Table 3). Mean Hb was 13.2 ± 1.9 g/dL and mean serum ferritin was 24.2 ± 10.4 ng/dL (Table 4 and Table 5). The mean Hb was 13.1 ± 2.1 g/dL and serum ferritin 25.3 ± 10.2 ng/dL in the male patients. Mean Hb was 13.2 ± 1.9 g/dL and mean serum ferritin level 22.9 ± 10.6 ng/dL in female patients.

To assess the impact of age and gender on the prevalence of iron deficiency anemia (IDA), data were stratified into subgroups.

IDA was found in 33 out of 124 patients (26.6%) among male participants. On the other hand, among female patients, 23 of 104 (22.1%) were found to be suffering from IDA. No statistically significant difference was found between IDA prevalence in males compared to females using a chi square test ($p = 0.432$) suggesting that gender was not a significant factor in the development of IDA in the current study population (Table 6).

Of 148 patients who were stratified by age, 36 (24.3%) 18 to 50 years old were found to have IDA. For example, among the patients aged 51 to 70 years, 20 out of 80 subjects (25.0 percent) had IDA. No statistically significant difference in IDA prevalence

was observed between these two age groups in a chi square test ($p = 0.910$), which indicated that age did not contribute significantly to the development of IDA in H. pylori infected patients (Table 7). These findings are consistent with past work that has shown H. pylori infection to be associated with iron deficiency anemia. IDA has reportedly been found in amounts from 2% to 15% of H. pylori infected patients and up to 30.9% have been reported. The prevalence of IDA of 24.6% found in the present study is in this range.

Research by Kibru D et al studied dyspeptic patients with H. pylori and showed anemia prevalence of 30.9% slightly above the present study findings. Fraser et al. discovered that H. pylori infection causes iron deficiency through decreased iron saturation levels in female university students who tested positive for H. pylori serology.

Scientific research conducted by Cardenas VM et al discovered that H. pylori infection resulted in lower serum ferritin measurements and higher prevalence of iron deficiency anemia. The H. pylori-IDA connection appears differently among various populations due to socioeconomic factors and nutritional deficiencies alongside genetic predispositions between H. pylori infection and the disease.

The research data shows that health professionals should conduct regular H. pylori examinations on patients suffering from chronic gastritis because these patients might also manifest iron deficiency anemia. Timely diagnosis of IDA in this patient group leads to better outcomes because treatment prevents negative impacts of long-term anemia like tiredness and mental decline and diminished well-being. The treatment of H. pylori provides additional benefits to iron status for patients suffering from this disease thus promoting early medical interventions.

This study contained various confounders yet its accomplished findings remain vital. The cross sectional research method fails to determine cause and effect relationships for H. pylori infection in IDA thus we eliminate H. pylori as an IDA origin. The research failed to examine dietary habits together with socioeconomic indicators or comorbidities which would influence iron status measurements. Research should proceed with more controlled studies that use large sample populations to study H. pylori's connection with iron deficiency anemia by evaluating H. pylori infected subjects versus healthy subjects.

Table 1: Gender Distribution in Study Sample

Gender	Frequency	Percentage (%)
Male	124	54.4
Female	104	45.6
Total	228	100.0

Table 2: Age Group Distribution

Age Groups (Years)	Frequency	Percentage (%)
18-50	148	64.9
51-70	80	35.1
Total	228	100.0

Table 3: Frequency of Iron Deficiency Anemia in Study Sample

Iron Deficiency Anemia	Frequency	Percentage (%)
Present	56	24.6
Absent	172	75.4
Total	228	100.0

Table 4: Mean Hb% in Study Sample

Gender	Mean Hb (%)	Standard Deviation
Male	13.1	2.1
Female	13.2	1.9
Total	13.2	1.9

Table 5: Mean Serum Ferritin (ng/dL) in Study Sample

Gender	Mean Serum Ferritin (ng/dL)	Standard Deviation
Male	25.3	10.2
Female	22.9	10.6
Total	24.2	10.4

Table 6: Stratification for Effect Modifiers (Gender-wise IDA Distribution)

Gender	Iron Deficiency Anemia Present	Iron Deficiency Anemia Absent	Total	p-Value (Chi-square)
Male	33	91	124	0.432
Female	23	81	104	
Total	56	172	228	

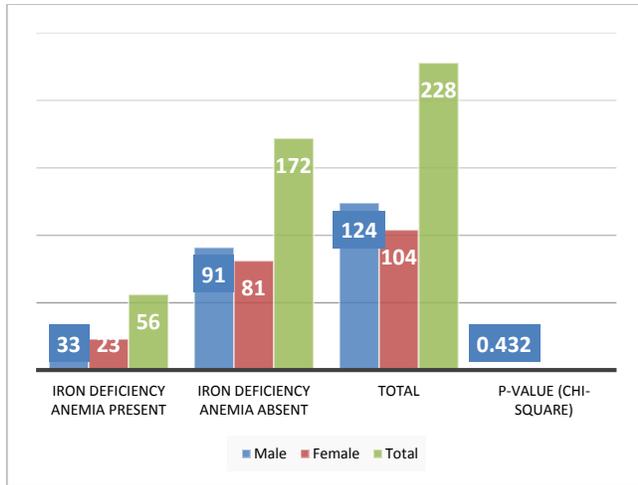


Figure 1: Stratification for Effect Modifiers (Gender-wise IDA Distribution)

Table 7: Stratification for Effect Modifiers (Age-wise IDA Distribution)

Age Groups (Years)	Iron Deficiency Anemia Present	Iron Deficiency Anemia Absent	Total	p-Value (Chi-square)
18-50	36	112	148	0.910
51-70	20	60	80	
Total	56	172	228	

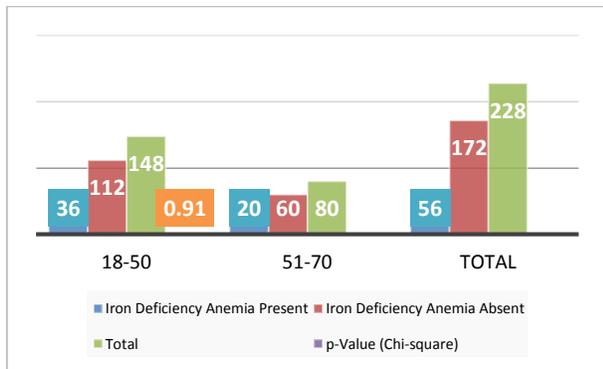


Figure 2: Stratification for Effect Modifiers (Age-wise IDA Distribution)

The research determined that iron deficiency anemia existed in two out of every ten H. pylori positive patients. The results from the age group and gender-specific stratified data analysis showed no significant statistical differences emerged. The evidence from these findings indicates that regular screening should be performed for iron deficiency anemia in H. pylori infected patients. The study findings are reinforced by additional studies conducting expanded measurements with diverse participant groups combined with control subjects.

DISCUSSION

Various research teams worldwide continue to study the connection between H. pylori infection and iron deficiency anemia (IDA) yet they present inconsistent findings. Several scientific studies indicate that one of the most common bacterial infections globally affects about 50% of worldwide populations. Scientists conducted recent research to explore how OM could cause

hematological disturbances while specifically focusing on producing IDA. This research evaluated the frequency of IDA in patients diagnosed with H. pylori gastritis while establishing their position in current evidence supporting this connection.

This research demonstrated that anemia caused by iron deficiency affected 24.6% of patients diagnosed with H. pylori gastritis. Studies presented a wide spectrum of anemia incidence in H. pylori infected patients with adult populations showing between 2% and 15% diagnostic rates. Several studies have detected slightly elevated H. pylori prevalence rates but this research contradicts other findings. The reported anemia prevalence of 30.9 percent among patients with dyspeptic H. pylori infection exceeds the 27.6 percent rate our study determined.

The analysis by Fraser et al. explored H. pylori infection and IDA relationships among female New Zealand student inhabitants. Research data indicated that people who tested positive for H. pylori bacteria had a 1.20 times greater chance of developing iron deficiency yet showed a clear positive connection between low iron and H. pylori seropositivity. The research demonstrated a linkage between ethnic differences in H. pylori infection rates and genetic and socio-economic elements affecting iron metabolism.

Ordion Study conducted by Cardenas et al using the National Health and Nutrition Examination Survey (NHANES) data confirmed that H. pylori infection leads to diminished serum ferritin values and elevated prevalence of IDA. The presence of H. pylori bacteria in the body leads to a 40 percent increase in IDA risk according to their research which demonstrates microbial interference with iron control mechanisms.

Several possible explanations exist for the IDA relationship with H. pylori infection. Continuous H. pylori infection results in sustained gastric inflammation which produces changes to gastric acid production and iron uptake. Higher gastric acidity is needed for the conversion of Fe³⁺ into Fe²⁺ which becomes easier to absorb. The presence of H. pylori gastritis particularly when causing atrophic gastritis or hypochlorhydria leads to reduced iron absorption rates from the duodenum.

As a growth factor H. pylori maintains repression of iron usage while it fights to obtain iron stores from the host. Scientific research indicates H. pylori infection elevates hepcidin hormone concentrations therefore reducing macrophage iron absorption and release. Clinical research demonstrates that destroying H. pylori bacteria restores regular hepcidin hormone levels which indicates that the microorganism disrupts iron homeostasis directly.

Chronic bleeding through the gastrointestinal tract functions as a major factor in developing IDA. The presence of Helicobacter pylori serves as a proven infection trigger for Peptic ulcer disease and Erosive gastritis. Slow iron depletion usually creates small bleeding episodes before leading to anemia.

Many research findings indicate that H. pylori infection leads to IDA yet some studies show conflicting results. Saler et al found that male patients with intact gastric mucosa did not show a connection between H. pylori infection and IDA while factors affecting this relationship include individual gastric pathologies.

An evaluation by Santos et al. of six cross sectional studies in Latin America produced weak evidence presentation about H. pylori infection associations with anemia in children, adolescents, adults and pregnant women. These differences provide useful information regarding how H. pylori infection modifies iron status because they show that different areas may exhibit different levels of dietary iron consumption together with distinct genetic and anemia-related factors.

A study conducted by Shak et al. in Haiti demonstrated that H. pylori infection did not correlate with anemia status in children at 80.1% rate or adults at 63.6% even though infection rates were extremely elevated. Anemia related to nutritional deficiencies or parasitic infections results in higher baseline amounts of anemia which may overshadow the unique contributions of H. pylori to the development of IDA.

Study findings alongside others demonstrate a substantial proportion of anemia cases in H. pylori positive subjects making

screening for anemia among patients with H. pylori gastritis an appropriate recommendation. Early diagnosis coupled with medical treatment of anemia in these patients helps prevent complications while improving overall health in populations who lack proper health care access.

Multiple research findings demonstrate that eliminating H. pylori through therapy successfully enhances iron metabolic levels in the body. Medical research indicates that H. pylori eradication leads to complete recovery from anemia in more than 60 percent of cases. Mulayim et al. conducted an investigation among pregnant women which demonstrated that H. pylori-positive mothers exhibited diminished iron and lower hemoglobin thus showing beneficial hematological results following effective H. pylori treatment. Medical experts have identified that evaluating and treating H. pylori infection represents a beneficial approach for managing anemia in susceptible populations including children and adolescents along with pregnant women.

Healthcare providers must exercise careful consideration before giving proton pump inhibitors (PPIs) to H. pylori infected patients because PPIs inhibit iron absorption and protracted PPI therapy leads to additional detriment in iron uptake. Physicians should provide iron supplementation when prescribing PPIs in order to prevent possible deficiencies from occurring.

Research findings from this study reinforce that H. pylori infection leads to IDA yet additional understanding requires more studies to address known gaps in knowledge. A larger prospective study involving well-defined H. pylori negative controls should establish the direct link between infection rates and anemia development.

Future studies need to determine if genetic or environmental conditions explain the correlation observed between H. pylori infection and IDA. Researchers must conduct additional studies to understand how dietary iron consumption interacts with coinciding parasitic infections alongside genetic variations that control iron metabolic processes.

Randomized controlled trials which evaluate the long-term impact of H. pylori eradication therapy on iron status for anemic patients would generate important clinical data about treating this infection for patients who are anemic. Research studies will help improve clinical guidelines by developing recommendations which use aligned evidence and address individual patient groups.

CONCLUSION

Results indicated that Helicobacter pylori (H. pylori) gastritis patients showed a 24.6% prevalence of IDA. Research findings support the idea that H. pylori infection causes disturbances to iron metabolism while contributing to this evidence base. H. Pylori infected patients require early intervention and testing to prevent anemia complications because the observed IDA prevalence rate does not differ based on patient age or gender.

Scientists agree that H. pylori accelerates iron deficiency through four mechanisms including chronic gastric inflammation, hypochlorhydria which hinders iron absorption and direct bacterium iron consumption and gastrointestinal bleeding. Numerous studies exist showing various levels of H. pylori relation to IDA yet our investigation matches reports indicating infection involvement in iron deficiency pathogenesis.

Healthcare professionals need to perform IDA diagnosis screenings on patients with H. pylori gastritis who exhibit anemic symptoms. Current research findings demonstrate that H. pylori elimination treatment results in better iron levels thus indicating its potential role in treating IDA comprehensively. The success rate of these interventions depends on multiple factors including patient diets alongside their original iron storage levels and other existing health issues.

The research design as a cross-sectional study fails to demonstrate that H. pylori infection directly leads to IDA development. Further research must conduct both longitudinal observation studies alongside intervention studies of large patient populations with controlled groups to identify the biological

pathways through which H. pylori influences iron metabolism disturbances.

This research indicates that physicians should consider H. pylori infection status when evaluating patients at risk of iron deficiency anemia. Systematic screening and treatment combination can benefit patient health results and reduce the IDA burden from chronic gastritis. Studies need to progress to quantify the causal effects of H. pylori elimination on long-term IDA prevention and management.

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