

Seroprevalence of Anti-*Helicobacter pylori* IgG Antibodies Among Patients with Gastritis in Wasit Province, Iraq

Eman Th. Nadhaif Al-Fatlawy*¹, and Muhamed Ali Al Kabe²

¹Department of Clinical Laboratory Sciences, College of Pharmacy, University of Al-Kufa, Iraq.

²Department of Medical Microbiology and Immunology, College of Medicine, University of Wasit, Iraq.

*Corresponding Author Email: imanth.nadhaif@uokofa.edu.iq, Malkaabi@uowasit.edu.iq

ABSTRACT

Background: *Helicobacter pylori* forms an important part of the epidemiology of chronic gastritis and peptic ulcers and is known to have significant long-term gastric complications. **Objective:** To measure the anti-*Helicobacter pylori* antibody levels and their relationship with some demographic and haematological parameters in patients with gastritis in Wasit Province in Iraq. **Methods:** This is a cross sectional study carried out at Al-Kima and Al-Hiat Laboratories of Al-Kut City, Wasit Province, Iraq from February to June 2021 using 100 cases about gastritis. Anti-*H. pylori* antibodies identified in the serum were performed by rapid immunochromatographic testing. Data on the demographic characteristics and complete blood count (CBC) were gathered and analysed using the SPSS version 25. **Results:** Of the 100 patients enrolled, 39 were male and 61 were female. Anti-*H. pylori* antibodies were detected in 41 patients, giving an overall seroprevalence of 41.0%. Seropositivity was observed in 43.6% of males and 39.3% of females. Most seropositive patients had normal white blood cell counts (90.2%) and normal hemoglobin levels (80.5%). Similarly, most seronegative patients had normal white blood cell counts (81.4%) and normal hemoglobin levels (79.7%). No statistically significant associations were found between seropositivity and gender, white blood cell status, or hemoglobin status. **Conclusion:** The seroprevalence of anti-*Helicobacter pylori* antibodies among patients with gastritis in Wasit Province was 41.0%. Although no significant association was observed with gender or hemoglobin status, the findings highlight the continued relevance of *H. pylori* in gastritis. Larger studies using confirmatory diagnostic methods and broader clinical assessment are recommended.

Keywords: *Helicobacter pylori*; Gastritis; Seroprevalence; Antibodies.

Article Information

Received: April 08, 2026; Revised: June 29, 2026; Online: June 2026

INTRODUCTION

Gastritis is a very frequent gastrointestinal disease in all countries worldwide and still an important cause of morbidities. It is caused by inflammatory disease of the gastric mucosa, caused by infectious and non-infectious factors. The most important infectious diseases include *Helicobacter pylori* infection which is considered as the most important cause of chronic gastroenteritis and is still regarded as a major cause of peptic ulcer, mucosa-associated

lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma (1).

H. pylori is a spiral-shaped, gram-negative, microaerophilic bacterium which resides on the gastric mucosa and adapts by several mechanisms to the acidic gastric milieu. Whilst the bacteria is ubiquitous in the world, an increasing number of strains are becoming resistant to the antibiotics that are used to treat them, and it is linked to a number of serious gastrointestinal diseases, making it a significant public health issue around the

globe. The International Agency for Research on cancer (IARC) has designated it as a group 1 carcinogen, and it has been confirmed to be a cause of gastric cancer (2).

Recent international consensus puts the importance of diagnosis and the right treatment of disease progression on top of the priority list in order to avoid eventual complications such as gastric malignancy. Rising antibiotics resistance is a big challenge to successful eradication, though and continued assessment of treatment plans is needed (3,4).

The *H. pylori* prevalence is different in each area of the world, in socio-economic stratification, in the quality of sanitation and among different populations. Most will occur in childhood and can last for years when left untreated, and can cause chronic gastric inflammation and make them more susceptible to gastrointestinal complications. Generally prevalence is higher in the developing countries than in the developed countries owing to differences in living condition and access to health service in developing countries. Reported prevalence rate in Iraq has ranged among regions and populations indicating the need for locally carried out epidemiological studies to estimate the prevalence of infection and relationship with gastric disease (5,6).

There are multiple methods by which the diagnosis of *H. pylori* infection is made, and these can be broken down into invasive and non invasive methods. The following invasive techniques have been reported: endoscopy and gastric biopsy; histopathological examination; rapid urease testing; culture and molecular techniques. The non-invasive tests encompass the urea breath test, stool antigen test and antibody detection in serum to *H. pylori* antigens (3). Of those, immunochromatographic assays and ELISA are commonly used in Epidemiological studies because they are fast, simple, inexpensive and

can be used for screening large numbers of people (7,8).

The burden of *H. pylori* infection is high in Iraq, but there is still sparse information available from Wasit Province. Thus, the aim of the present study was to measure the seroprevalence of anti-*H. pylori* antibody levels and the possible correlation of these antibody levels to some demographic and hematological data of individuals suffering from gastritis at Wasit Province, Iraq.

MATERIALS AND METHODS

Study Design and Setting: It is a cross sectional study in which patients suffering from gastritis were collected at Al-Kima, Al-Hiat laboratories during February-June 2021 in Wasit Province of the city of Al-Kut in Iraq. A total of 100 patients with gastritis were included in the study. Clinical diagnosis and during the time of the study clinical availability were used to select participants. Structure data collection form was used to collect demographic information such as: age and sex.

All male and female participants as well as informed consent patients who were diagnosed with gastritis were included in the study.

Patients who were previously treated with *H. pylori* eradication and had incomplete data for clinical and laboratory parameters and with any other gastrointestinal condition other than gastritis were excluded.

Venous blood was drawn in aseptically in each case about 5 mL sample draws were made. Blood samples were left to coagulate and centrifuged to extract the serum. The serum samples were frozen at -20°C for later use. All subjects gave informed consent for blood sample collection. The confidentiality and privacy of participants were respected in the study.

Laboratory Investigation: Anti-*H. pylori* IgG antibody was measured in serum with the use of a rapid immunochromatographic One-Step *H. pylori* Test Device (Serum/Plasma), as recommended by the manufacturer. This is a

qualitative test which uses an antigen-antibody reaction to identify anti-*H. pylori* IgG antibodies in serum/plasma. The presence or absence of control and/or test lines determined whether the test result was positive or negative.

The number of leukocytes and hemoglobin concentration were counted by the automatic hematology analyzer following standard laboratory methods and manufacturer's instructions.

Statistical Analysis: Data were subjected to SPSS (version 25) for analysis. Frequencies and percentages were used to summarise categorical variables. The Chi-square test or Fisher's exact test (FET) was used to test for associations with categorical variables, when applicable. Statistically significant P value was defined as < 0.05 .

RESULTS

In this study, 100 patients with gastritis have been included. Women members formed the majority of participants (61.0%) as compared to men (39.0%) (Table 1) (Fig:1). The overall seroprevalence of anti-*Helicobacter pylori* IgG antibodies was 41.0% (41 participants were positive) while 59 participants were anti-*Helicobacter pylori* IgG negative (Table 2) (Fig:2).

Twenty-four of 61 (39.3%) female patients were positive for anti-*H. pylori* antibody whereas 37 (60.7%) were negative (Table 3) (Fig:3). Fifteen out of the 39 male participants (43.6%) were tested positive and 22 male participants (56.4%) were tested negative.

Seropositivity towards *H. pylori* showed no statistically significant difference between the genders (Table 4) (Fig:4).

Only 37/41 seropositive cases (90.2%) had a normal WBC count, and 4 (9.8%) had elevated counts (Table 5) (Fig:5). Of those who were seronegative, 48/59 (81.4%) had normal WBC figures and 11/59 (18.6%) had increased WBC figures. Anti-*H. pylori* seropositivity and being a WBC showed no significant association (Table 6) (Fig:6).

In healthy individuals, 33 of 41 seropositive (80.5%) had normal hemoglobin and 8 (19.5%) had low hemoglobin levels (Table 7) (Fig:7).

Forty-seven individuals with normal hemoglobin levels were found in the seronegative group (79.7% of those seronegative), 11 had low hemoglobin levels (18.6%) and 1 had elevated hemoglobin levels (1.7%). There was no statistically significant relationship between the presence of anti-*H. pylori* antibody against hemoglobin status (Table 8) (Fig:8).

Table 9 shows association between the results of RAT for *H. pylori* with selected demographic and haematological variables. Gender was not associated with the Rapid anti-*H. pylori* antibody positivity ($p = 0.82$). Similarly, there was no difference in the results of rapid anti-*H. pylori* antibody test between the white blood cell statuses ($p = 0.39$). There was also no statistically significant association between the rapid anti-*H. pylori* antibody positivity and hemoglobin status (Fisher's exact test, $p = 0.89$).

Table 1: Distribution of study participants according to gender.

Gender	Frequency (n)	Percent (%)
Males	39	39.0
Females	61	61.0
Total	100	100.0

Table 2: Detection of anti-*Helicobacter pylori* antibodies among study participants.

Rapid anti- <i>H. pylori</i> antibody test result	Frequency (n)	Percent (%)
Positive	41	41.0
Negative	59	59.0
Total	100	100.0

Table 3: Detection of anti-*Helicobacter pylori* antibodies among female participants.

Rapid anti- <i>H. pylori</i> antibody test results	Frequency (n)	Percent (%)
Positive	24	39.3
Negative	37	60.7
Total	61	100.0

Table 4: Detection of anti-*Helicobacter pylori* antibodies among male participants.

Rapid anti- <i>H. pylori</i> antibody test results	Frequency (n)	Percent (%)
Positive	17	43.6
Negative	22	56.4
Total	39	100

Table 5: White blood cell count among participants with positive test results.

WBC status	Frequency (n)	Percent (%)
Normal	37	90.2
High	4	9.8
Total	41	100.0

Table 6: White blood cell count among participants with negative test results.

WBC status	Frequency	Percent (%)
Normal	48	81.4
High	11	18.6
Total	59	100.0

Table 7: Hemoglobin levels among participants with positive test results.

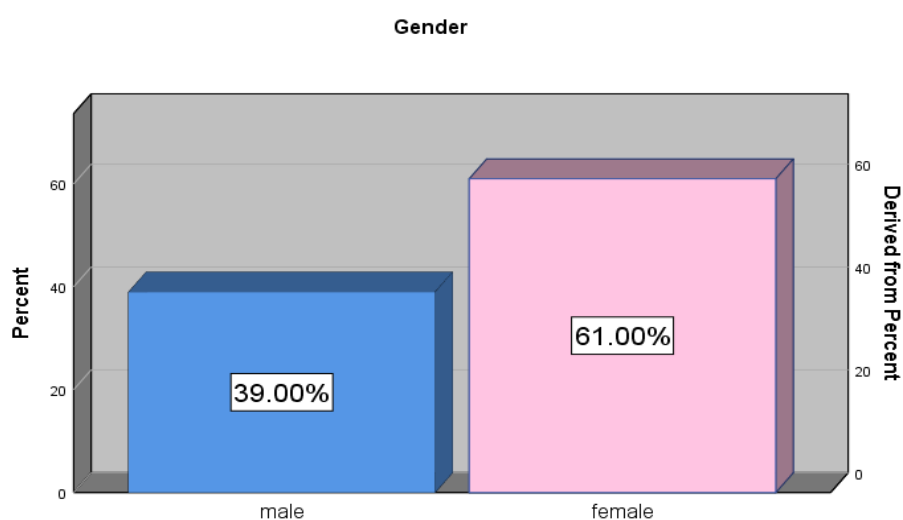
Hemoglobin status	Frequency (n)	Percent (%)
Normal	33	80.5
Low	8	19.5
Total	41	100

Table 8: Hemoglobin levels among participants with negative test results.

Hemoglobin status	Frequency (n)	Percent (%)
Normal	47	79.7
Low	11	18.6
High	1	1.7
Total	59	100.0

Table 9: Association between selected variables and rapid anti-*H. pylori* antibody test results

Variable		Positive n (%)	Negative n (%)	P-value
Gender	Male	(43.6) 17	(56.4) 22	0.82
	Female	(39.3) 24	(60.7) 37	
WBC status	Normal	(90.2) 37	(81.4) 48	0.39
	High	(9.8) 4	(18.6) 11	
Hemoglobin status	Normal	(80.5) 33	(79.7) 47	*0.89
	Low	(19.5) 8	(18.6) 11	
	High	(0.0) 0	(1.7) 1	
* Fisher's Exact Test was used because one cell contained an expected frequency less than 5.				

**Figure 1:** Gender distribution among study participants

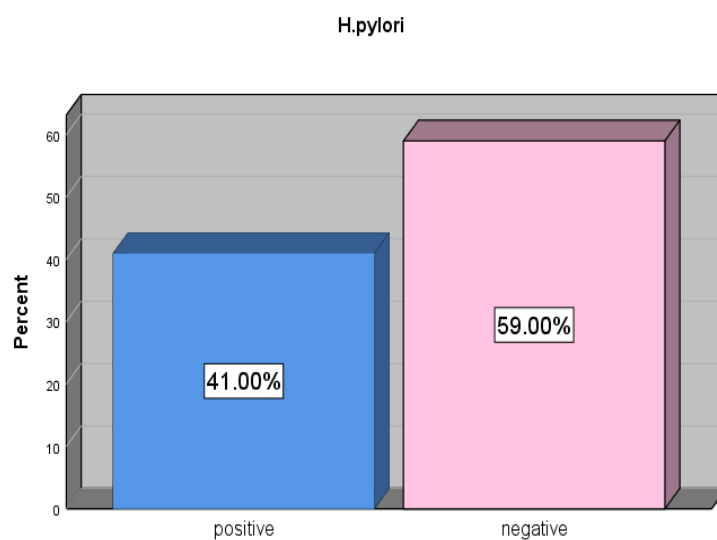


Figure 2: Detection of anti-*H. pylori* antibodies by rapid immunochromatographic assay among study participants.

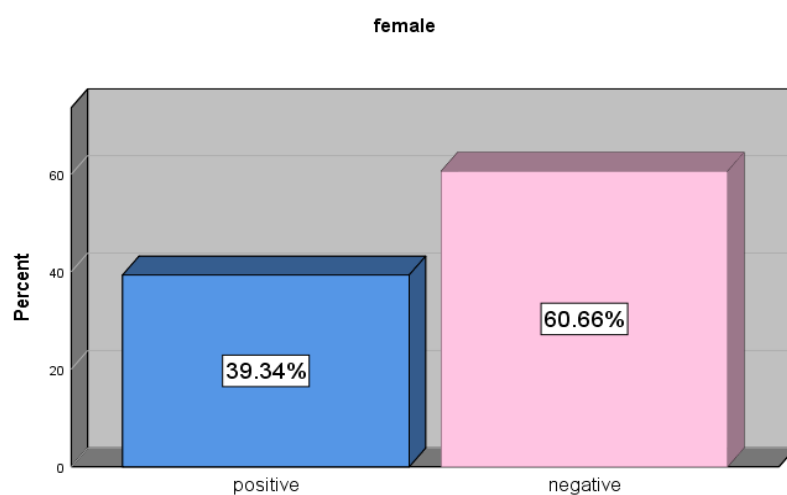


Figure 3: Seroprevalence of anti-*H. pylori* IgG antibodies among female participants.

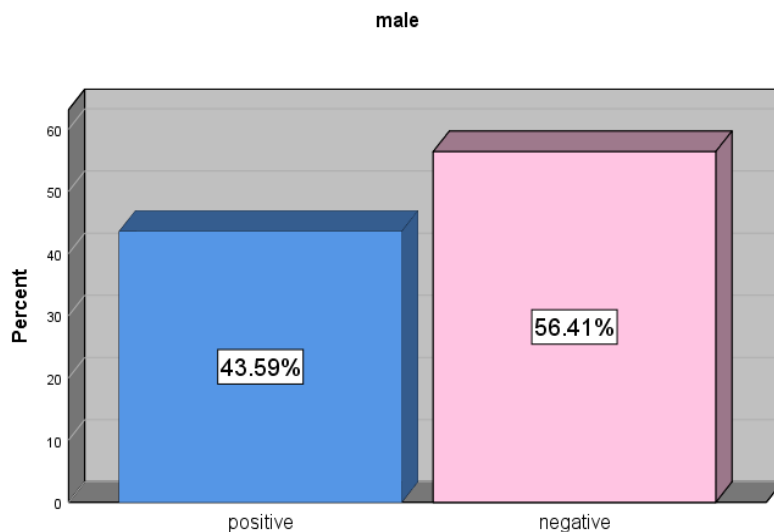


Figure 4: Seroprevalence of anti-*H. pylori* IgG antibodies among male participants.

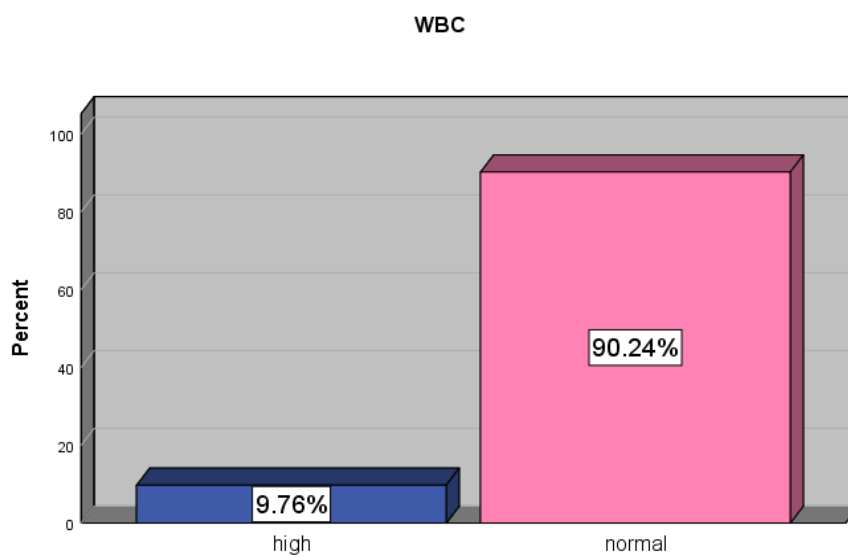


Figure 5: Distribution of white blood cell count among anti-*H. pylori* IgG positive participants.

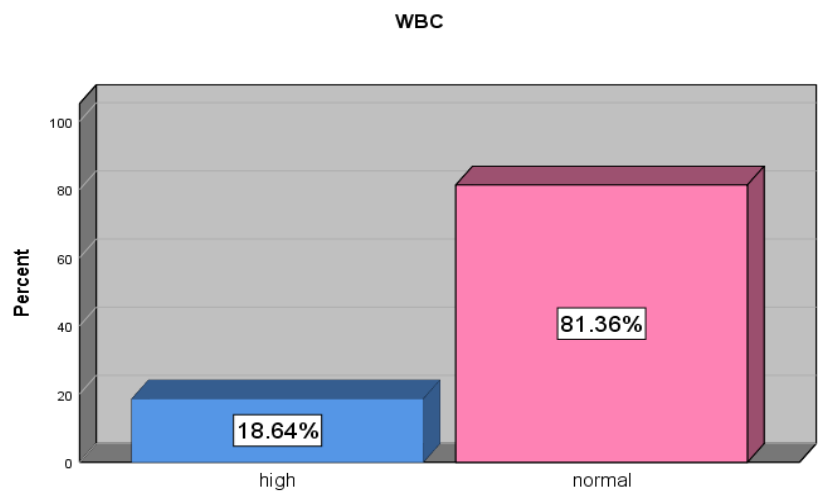


Figure 6: Distribution of white blood cell count among anti-*H. pylori* IgG-negative participants.

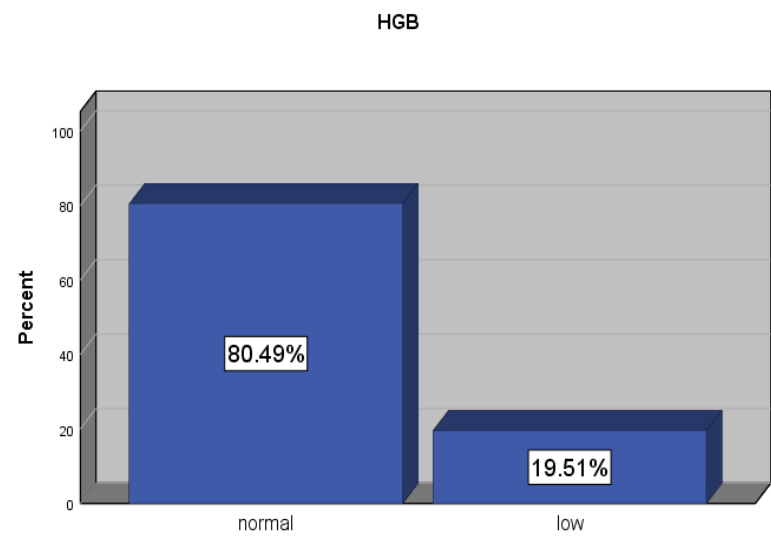


Figure 7: Distribution of hemoglobin levels among anti-*H. pylori* IgG-positive participants.

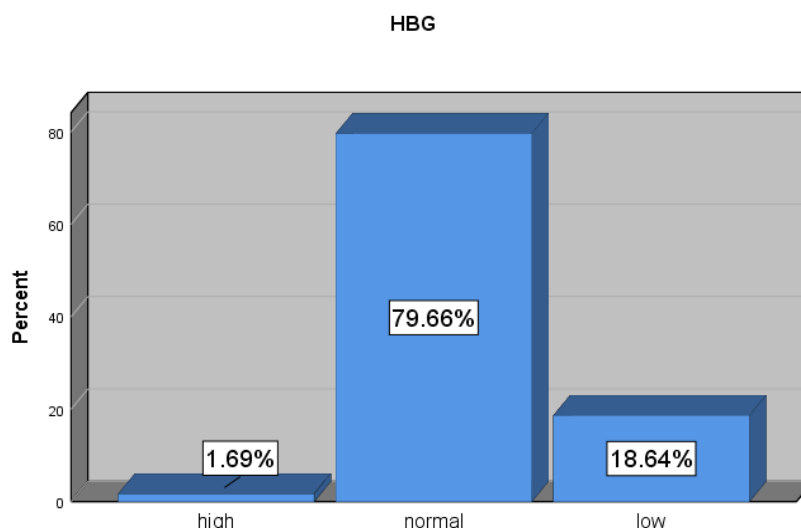


Figure 8: Distribution of hemoglobin levels among anti-*H. pylori* IgG-negative participants.

DISCUSSION

Accordingly, the present study revealed a seroprevalence of 141.0% for anti-*Helicobacter pylori* antibodies related to the presence of gastritis and indicated that *H. pylori* is still a cause of gastric inflammation in the community of Wasit Province. This is in line with the Iraqi results which indicated high burden of infection in various regions. A retrospective study was reported in Duhok Province with a 63% positivity (9) and Naqid *et al.*, had 40.02% of anti-*H. pylori* IgG in adults of the same Province (6). Agreeing with the results of the present study, Saeed *et al.* found the prevalence among symptomatic patients to be 50% in Zakho City, Kurdistan Region, indicating the seroprevalence of the present study to be within acceptable ranges for this Region (10). In the context of Iraqi studies, reported correlates ranged from male gender (6) to age (9) and low hemoglobin level and education (10).

A slight male predominance of seropositive was noticeable in the current study but it was found not to be significant. The Muslim population showed similar findings as there was no significant difference between males and females in Zakho City (10) and the cross-

sectional study by Qiao *et al.* on 81,754 Chinese adults found that males had slightly higher rates of infection, 49.74% versus 49.3%, although there were no significant differences (11). Male gender was a significant risk factor for *H. pylori* infections, as reported by Naqid *et al* (6) and differently in this study was the male gender ($P < 0.043$) in Duhok Province. Likewise, Qiao *et al.*, found sexual differences that were age dependent, with an increased risk of infection in young men compared to women (OR = 1.092, 95% CI: 1.04–1.14) and a decreased risk of infection in older women (OR = 0.908, 95% CI: 0.868–0.950) (11). Differences in these parameters between investigations could explain these discrepancies: sample size, age distribution, geographic background, and type of investigation (6,10,11).

H. pylori infection is clinically significant with regard to hematological abnormalities. There was no significant association observed between seropositivity with hemoglobin status; but the literature shows conflicting results regarding that association. There are a few studies that suggest a link between *H. pylori* infection and anemia, especially iron deficiency anemia. Furthermore, it was

confirmed that this association exists by a systematic review – meta-analysis of Hudak *et al.*, which showed a relationship between *H. pylori* infection and anemia (12), as well as by a systematic review of Motupalli and Oroszi, who indicated the existence of a relationship between *H. pylori* infection and anemia (13).

Previously, in children, Walle *et al.* observed that risk of anemia was associated with *H. pylori* infection (14) and in women, Wang *et al.* observed a correlation with the presence of IDA (15). However, unlike the present study, the correlation between *H. pylori* positivity and low hemoglobin level was significant among symptomatic patients from Zakho City, Iraq (10). The association between *H. pylori* and general anemia, however, isn't always consistent across populations, and may also be due to a small sample size and to the lack of any direct iron indices (like ferritin, serum iron and transferrin saturation) in the present study (10,12,15).

A rapid serological assay was found to be a useful screening technique with some limitations. Based on the presence of antigens, serology is not always reliable for determining whether there was a previous infection or current exposure. In 2024, the authors performed a population-level diagnostic comparison of serology that was found to be very sensitive, but whose positive results had to be confirmed by either stool antigen or urea breath testing to better define active infection (16).

In addition, there were some early reports suggesting that serology is more sensitive than some of the noninvasive tests in some screening scenarios, though its specificity is less than that of urea breath testing; serology primarily provides confirmation of contact with the bacterium rather than evidence of a current infection (17). Thus, the seropositivity observed in this study must be considered as a sign of exposure, and not as being evidence of active infection (16-17).

The results indicated that *H. pylori* was associated with gastritis in a small number of seronegative patients which led to the conclusion that gastritis is multifactorial. The use of non-steroidal anti-inflammatory drugs, bile reflux, autoimmune gastritis, diet, smoking and disadvantageous socio-economic factors may all be able to cause gastric mucosal injury (18,19). Its infection risk might be affected by the education level and/or smoking habits as well as the crowding phenomenon in households, according to local Iraqi data; another Iraqi study revealed significant relationships with age, low hemoglobin and low education (6,10). As such, some patients' lack of disease-related antibodies does not rule out clinically relevant gastritis, but helps to ensure discussion of other possible causes (18,19).

The 2024 guideline from the American College of Gastroenterology (ACG) calls for evidence-based *Helicobacter pylori* (*H. pylori*) eradication using antibiotic susceptibility patterns based on history of treatment. In treatment-naïve patients, optimized bismuth quadruple therapy (3 weeks duration) is the initial treatment of choice and post-treatment test of cure (ToC) is mandatory to document eradication and subsequent reduce persistent infection (3). Interestingly, Saeed *et al.* noted that only 46.7% of patients responded to standard triple therapy, thus underscoring the clinical relevance of variations in treatment options according to local resistance and susceptibility to specific antibiotics within the particular geographical location (10).

The following are some of the limitations of this study. First, the serological rapid test is a test for past exposure and will have limitations in helping to confirm active infection. Secondly, the sample size of this study was small and limited to one specific center that reduces the generalizability of findings to other centers in the study area (Wasit Province). Thirdly, direct iron studies and other

biochemical markers were not performed, dovewhisking a bit of the capacity to find fairly delicate hematological associations. Last, factors that represent important risk factors were not fully analyzed—diet, smoking, and exposure to antibiotics and socioeconomic status (12,18).

The present study revealed that prevalence of *H. pylori* infection was still high in patients suffering from gastritis from Wasit province, Iraq. In this study, no significant sex or hemoglobin association with seropositivity was encountered, but our results confirm that the clinical significance of the interaction of *H. pylori* with gastritis should not be underestimated and indicate the need to use fully updated diagnostic guidelines that rely on the serologic test. A broader assessment of the burden and clinical implications of *H. pylori* infection via confirmation probing in larger longitudinal studies, extending assessments of hematological and environmental consequences, will be required to better characterize the burden and clinical effects of *H. pylori* in this area (3,6,9-19).

CONCLUSION

The seroprevalence of anti-*Helicobacter pylori* antibodies among patients with gastritis in Wasit Province was 41.0%. Although no significant association was observed with gender or hemoglobin status, the findings highlight the continued relevance of *H. pylori* in gastritis. Larger studies using confirmatory diagnostic methods and broader clinical assessment are recommended.

RECOMMENDATIONS

1. Further studies are recommended with larger samples and multicenter based studies in order to get a better estimation of the prevalence of *H. pylori* in different regions of Iraq.
2. Confirmatory techniques for active *H. pylori* infection (e.g. urea breath test,

stool antigen test and endoscopic biopsy-based techniques) should be used in future studies.

3. Dietary and other risk factors, such as socioeconomic status, smoking, home environment and prior antibiotic therapy should be assessed to gain insight into the risk factors for *H. pylori* infection.
4. To optimize treatment, it is advised that antibiotics resistance patterns are monitored continually, and the effectiveness of antimicrobial treatments to eradicate infection be evaluated.
5. Raise public health awareness of *H. pylori* infection risk reduction, early detection and proper treatment of *H. pylori* infection, might be helpful in reducing burden of gastric diseases.

REFERENCES

1. Rokkas T, Graham DY. The unfinished agenda in *Helicobacter pylori* treatment: resistance, microbiome effects, and future directions. *Gastroenterology*. **2026**;171(2):271-284.
2. International Agency for Research on Cancer (IARC). *Helicobacter pylori*. In: IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 100B: Biological Agents. Lyon: International Agency for Research on Cancer; **2012**. p. 363-487.
3. Chey WD, Howden CW, Moss SF, Morgan DR, Greer KB, Grover S, *et al*. ACG clinical guideline: treatment of *Helicobacter pylori* infection. *Am J Gastroenterol*. **2024**;119(9):1730-1753.
4. Malfertheiner P, Megraud F, Rokkas T, Gisbert JP, Liou JM, Schulz C, *et al*. Management of *Helicobacter pylori* infection: the Maastricht VI/Florence Consensus Report. *Gut*. **2022**;71(9):1724-1762.

5. Li Y, Choi H, Leung K, Jiang F, Graham DY, Leung WK. Global prevalence of *Helicobacter pylori* infection between 1980 and 2022: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol*. **2023**;8(6):553-564.
6. Naqid IA, Al-Brefkani AMT, Hussein NR. A study of prevalence and risk factors for *Helicobacter pylori* infection among adults in Duhok Province, Kurdistan Region, Iraq. *Arch Razi Inst*. **2024**;79(2):272-278.
7. Mujtaba A, Ibrahim MS, Parveen S, Sarwar N, Alsagaby SA, Raza MA, *et al*. Comparative analysis of diagnostic techniques for *Helicobacter pylori* infection: insights for effective therapy. *J Cell Mol Med*. **2025**;29(6):e70487.
8. Katelaris P, Hunt R, Bazzoli F, Cohen H, Fock KM, Gemilyan M, *et al*. *Helicobacter pylori* World Gastroenterology Organisation global guideline. *J Clin Gastroenterol*. **2023**;57(2):111-126.
9. Huseyin MY. The prevalence of *Helicobacter pylori* infections in Duhok Province, Iraq: a retrospective study. *Eur J Med Health Res*. **2024**;2(3):71-76.
10. Saeed AY, Rashad BH, Ali BN, Sulaivany AH, Ibrahim KS. *Helicobacter pylori* infection: prevalence, risk factors, and treatment efficacy in symptomatic patients in Zakho City, Kurdistan Region, Iraq. *Cureus*. **2024**;16(11):e73873.
11. Qiao Y, Zhou Y, Zhao L, Yang S, Zhang X, Liu S. Sex differences in *Helicobacter pylori* infection and recurrence rate among 81,754 Chinese adults: a cross-sectional study. *BMC Gastroenterol*. **2024**;24(1):305.
12. Hudak L, Jaraisy A, Haj S, Muhsen K. An updated systematic review and meta-analysis on the association between *Helicobacter pylori* infection and iron deficiency anemia. *Helicobacter*. **2017**;22(1):e12330.
13. Motupalli SK, Oroszi TL. The nexus between *Helicobacter pylori* infection and anemia: a systematic review. *Front Hematol*. **2024**;3:1423494.
14. Walle M, Tesfaye A, Agidew MM, Semaw M, Mekuria S, Getu F. The association of *Helicobacter pylori* infection with the risk of anemia in children: systematic review and meta-analysis. *BMC Infect Dis*. **2025**;25(1):23.
15. Wang ZT, Tan WT, Meng MM, Su H, Li Q, Guo CM, *et al*. The correlation between *Helicobacter pylori* infection and iron deficiency anemia in women. *Eur Rev Med Pharmacol Sci*. **2024**;28(4):1541-1553.
16. Imperial M, Tan A, Fjell C, Chang Y, Krajden M, Kelly MT, *et al*. Diagnosis of *Helicobacter pylori* infection: serology vs. urea breath test. *Microbiol Spectr*. **2024**;12(11):e0108424.
17. Bosch DE, Krumm N, Wener MH, Yeh MM, Truong CD, Reddi DM, *et al*. Serology is more sensitive than urea breath test or stool antigen for the initial diagnosis of *Helicobacter pylori* gastritis when compared with histopathology. *Am J Clin Pathol*. **2020**;154(2):255-265.
18. Kim SH. Gastritis: Pathophysiology, Diagnosis, and Clinical Implications. *Korean J Helicobacter Up Gastrointest Res*. **2026**;26(1):8-14.
19. Genta RM, Lash RH. *Helicobacter pylori*-negative gastritis: seek, yet ye shall not always find. *Am J Surg Pathol*. **2010**;34(8):e25-34.