

Evaluation of the Relationship Between H. Pylori Infection and Hyperemesis Gravidarum

Wasan Shareef Hameed¹, Nadia Saddam Fahad^{2,*}

¹ Department of Obstetrics and Gynecology, Bint Al-Huda Teaching Hospital, Thi-Qar, 64001, Iraq.

² Department of Obstetrics and Gynecology, College of Medicine, University of Thi-Qar, Thi-Qar ,64001, Iraq.

Corresponding Author Email:

nadia-s@utq.edu.iq

Received: 4 April 2026,

Revised: 20 May 2026,

Accepted: 2 June 2026,

DOI: [10.57238/jbb.2026.7432.1169](https://doi.org/10.57238/jbb.2026.7432.1169)



Access this article online

Copyright: ©2026 The authors. This article is published by Nabea Al-Ajyal Foundation Press and is licensed under the CC BY 4.0 license (<http://creativecommons.org/licenses/by/4.0/>).

ABSTRACT

Background: Hyperemesis gravidarum (HG) is a severe form of nausea and vomiting during pregnancy with significant maternal health implications. Helicobacter pylori (H. pylori), a gram-negative bacterium associated with gastric conditions, has been proposed as a contributing factor through gastric mucosal inflammation, hormonal interactions, and immune responses.

Aim: To assess the relationship between H. pylori infection and hyperemesis gravidarum.

Methods: A prospective case-control study was conducted at Bint al-Huda Hospital, Thi Qar City, from December 2023 to December 2024. Ninety-eight first-trimester pregnant women were enrolled: 50 with HG (cases) and 48 without HG (controls). Serum H. pylori IgG levels were measured using ELISA. Statistical analyses were performed using SPSS version 26 ($p < 0.05$).

Results: Mean age was comparable between groups (29.08 ± 3.4 vs. 28.9 ± 2.8 years; $p = 0.053$). Most participants were housewives residing in rural areas, with no significant inter-group differences. H. pylori IgG seropositivity was significantly higher in HG cases than controls (72% vs. 29%; $p < 0.001$). Infected women demonstrated more severe clinical manifestations, including dehydration and electrolyte imbalances.

Conclusions: A significant association between H. pylori infection and hyperemesis gravidarum was identified, suggesting H. pylori may exacerbate gastrointestinal symptoms. Routine screening and eradication therapy may improve maternal outcomes. Further research exploring pathophysiological mechanisms is warranted

Keywords: H. Pylori, Hyperemesis Gravidarum, gram-negative bacterium y.

1. Introduction

Hyperemesis gravidarum (HG) is a condition characterised by uncontrollable vomiting during pregnancy, which causes weight loss and dehydration. This may lead to the presence of ketones in the urine and/or blood. In the absence of a general agreement on precise diagnostic criteria, however, the term commonly refers to the severe form of nausea and vomiting experienced during pregnancy. It is estimated to happen in about 2% of all pregnancies in the United States. The condition may have a profound effect on the well-being of women and their families and, unfortunately, can be quite difficult to manage [1].

H. pylori is a gram-negative bacterium that inhabits the stomach lining and is recognised as a causative agent for both duodenal and gastric ulcers and is also considered a significant predisposing factor for

stomach cancer [2, 3]. Peptic ulcer, adenocarcinoma, and stomach lymphoma are all associated with this disease. The World Health Organisation has categorised *H. pylori* as a Class 1 carcinogen [2-5].

There is a hypothesis that suggests *H. pylori* infection may worsen gastrointestinal symptoms in pregnant women, perhaps leading to Hyperemesis Gravidarum (HG) [6]. It causes elevated levels of reactive oxygen species (ROS) and reduced levels of plasma antioxidants such as ascorbic acid. HG may be characterised as a condition of oxidative stress, characterised by elevated levels of reactive oxygen species (ROS) and reduced antioxidant capacity [7].

Hyperemesis gravidarum (HG) is a complex condition influenced by various factors, including hormonal, genetic, and environmental aspects. The interplay of these factors makes HG challenging to study and manage effectively. Human chorionic gonadotropin (hCG), a hormone that peaks during early pregnancy, is thought to play a significant role in the pathophysiology of HG. High levels of hCG have been linked to severe nausea and vomiting, though the exact mechanism remains unclear. Additionally, maternal genetics and a family history of HG are associated with a higher risk, suggesting an inherited component [1].

The potential link between *H. pylori* infection and HG has gained attention in recent years. Studies have shown a higher prevalence of *H. pylori* infection in pregnant women with HG compared to those without it, suggesting a possible causal relationship. The bacterium's ability to impair gastric motility and exacerbate gastrointestinal symptoms could explain its role in HG. Furthermore, chronic *H. pylori* infection contributes to inflammation, oxidative stress, and decreased antioxidant levels, all of which may worsen the severity of HG. Testing for and treating *H. pylori* infection in pregnant women with HG may, therefore, be a potential avenue for improving outcomes [8].

Management of HG requires a multifaceted approach, combining symptom control, nutritional support, and, when applicable, treatment of underlying conditions like *H. pylori* infection. Intravenous fluids and antiemetic medications are the cornerstone of symptom relief, while dietary modifications and vitamin supplementation, particularly with thiamine, help prevent complications such as Wernicke's encephalopathy.

Research into the role of oxidative stress and the potential therapeutic benefits of antioxidants in HG is ongoing and could provide new treatment options. Addressing HG holistically is essential to improving the quality of life for affected women and minimizing its impact on their families [9]. To assess the relationship between *H. pylori* infection and hyperemesis gravidarum.

2. Material and Methods:

2.1. Study design & settings

A prospective case-control study was conducted in the obstetric inpatient clinic at Bint al-Huda Hospital in Thi Qar City during the period from 1st of December 2023 to 1st of December 2024. To assess the relationship between *H. pylori* infection and hyperemesis gravidarum.

2.2. Study population

Pregnant Women in first trimester who visited the outpatient clinics of Bint al-Huda Hospital during their first trimester were screened for *H. Pylori* infection. And divided into two groups:

- Group 1: 50 pregnant women in the first trimester who were diagnosed with hyperemesis gravidarum.
- Group 2: 48 pregnant women in the first trimester who hadn't been diagnosed hyperemesis gravidarum.

2.3. Exclusion criteria

Exclusion criteria were women with multiple gestation pregnancy and molar pregnancy. Also, women with a history of systemic diseases or drug use (other than routine supplements like folic acid) were eliminated. Exclusion criteria included women with a history of gastrointestinal disease, women who abused drugs, women with hyperthyroidism and women who had known psychological issues.

2.4. Sampling and Data Collection

From the inpatient clinic of Bint al-Huda Hospital, the women who met the inclusion and exclusion criteria 98 women, 50 of them were diagnosed with hyperemesis gravidarum as they were presented with vomiting that leads to substantial dehydration (as indicated by the presence of ketones in the urine or abnormalities in electrolyte levels) and weight loss (typically defined as a reduction of at least five per cent of the patient's weight before pregnancy) occurring during pregnancy, with no other identifiable pathological cause for the vomiting.

Another forty-eight healthy pregnant ladies, who presented to the outpatient clinic for regular checkups, were included in the study as the control group. All the women who participated in this study had given informed consent, and confidentiality of the data were seriously considered. All the women in the study were asked to complete a well-structured questionnaire, specifically designed for this study. The socio-demographic data included age, place of residence, occupation and marital status along with the clinical profile which included the history of past medical and surgical illnesses of the women.

Anthropometric measures were taken of all women such as height, weight and BMI (weight in kg divided by height in meters squared). Vital signs of dehydration (sunken eyes, concentrated urine, oliguria, and hypotension) were evaluated in each woman. The 24-hour Pregnancy-Unique Quantification of Emesis (PUQE-24) Scale was used to assess the severity of HGE in group 1 women. Pelvic transvaginal or transabdominal ultrasonography was carried out to confirm gestational age, assess foetal viability and to exclude the presence of multiple gestations and/or gestational trophoblastic disorders.

All of the women participated in the study and blood samples were taken for a full blood count (RBC, WBC, platelet count, Hb%, hemoglobin concentration) and urine analysis (complete) was carried out for protein, ketone bodies and bile salts. Renal function tests (serum creatinine and creatinine clearance) and liver function tests (SGOT and SGPT) were performed to rule out renal and liver dysfunction, respectively. Thyroid function tests (TSH, Free T3, and Free T4) were checked to rule out hyperthyroidism, and fasting and 2-hour post-prandial blood sugar level were checked to rule out diabetic ketoacidosis. Finally, 5 mL of venous blood was drawn for *Helicobacter pylori* IgG seropositivity tests on serum using an enzyme-linked-immunoassay (ELISA).

2.5. Statistical analysis

Data was entered using computerized statistical software; Statistical Package for Social Sciences (SPSS) version 26 was used. Quantitative data were presented as (mean \pm standard deviation) while qualitative data were presented as frequencies and percentages.

The appropriate statistical tests were performed, a Chi-square test was used for categorical variables (Fisher's exact test was used when the expected variable was less than 5), and two samples independent t-tests for the continuous variable. In all statistical analyses, the level of significance (p-value) is set at ≤ 0.05 and the result is presented as tables and/or graph.

Characteristics	Levels	Case group (N=50)	Control group (N=48)	p-value
Age	Mean \pm SD	29.08 \pm 3.4	28.9 \pm 2.8	0.053
	20-29	32 (64.0)	40 (83.3)	
	30-39	18 (36.0)	8 (16.7)	
Occupation	Housewives	30 (60.0)	24 (50.0)	0.319
	Employer	20 (40.0)	24 (50.0)	
Residency	Rural	32 (64.0)	34 (70.8)	0.471
	Urban	18 (36.0)	14 (29.2)	
Educational level	Illiterate	10 (20.0)	14 (29.2)	0.089
	Primary	24 (48.0)	16 (33.3)	
	Secondary	2 (4.0)	8 (16.7)	
	College and higher education	14 (28.0)	10 (20.8)	

3. Results:

Table 1. The sociodemographic characteristics of participants

The study included 98 women divided into two groups, 50 cases and 48 controls. The mean age of women in the case group was 29.08 years and 28.9 years for women in the control group and there is no significant statistical difference between the two groups since p-value=0.053. Regarding the occupation, more than half of women in both groups were housewives (p-value=0.319). The women's residency was mainly in rural areas (64% and 70% for women in the case and control groups respectively). Regarding the educational level, the highest percentage of women in both groups had primary education and there are no significant statistical differences between the two groups (p-value=0.089). All these data are presented in Table 1.

Table 2. The clinical variables among participants

Variables	Case group (N=50)	Control group (N=48)	p-value	
Gravida	Primigravida	8 (16.0)	2 (4.2)	
	2-4	34 (68.0)	38 (79.2)	
	>5	8 (16.0)	8 (16.6)	
Gestational age	Mean \pm SD	10.28 \pm 1.19	10.87 \pm 1.37	0.251

Table 2 shows the women's clinical variables, regarding gravidity, 68% of women in the case group and 79.2% of women in the control group had 2-4 pregnancies and there are no significant statistical differences between the two groups (p-value=0.151). The mean gestational age at presentation for women in the case group was 10.28 weeks and 10.87 weeks for women in the control group and there are no

significant statistical differences between the two groups (p-value= 0.251).

Table 3. The hyperemesis gravidarum signs and symptoms among the case group

Variables	Number	Percentage
Sunken eyes	24	48.0
Hypotension	42	84.0
Concentrated urine	38	76.0
Oliguria	2	4.2

The hyperemesis gravidarum signs and symptoms among the case group are presented in Table 3. Most of the women (84%) were presented with hypotension, 76.0% had concentrated urine, 48.0% had sunken eyes, and only 4.2% had oliguria.

Table 4. The anthropometric characteristics of participants

BMI	Mean ± SD	27.32 ± 1.99	27.33 ± 2.03	0.974
	Overweight	34 (68.0)	32 (66.7)	
	Obese	16 (32.0)	16 (33.3)	
Weight loss	Mean ± SD	1.45 ± 0.29	0.0	NA

Table 4 shows the anthropometric characteristics among participants, there are no significant statistical differences regarding BMI between the two groups since the p-value >0.05. The mean weight loss among women in the case group was 1.45 kg.

Table 5. The prevalence of H. Pylori test among participants

Variables		Case group (N=50)	Control group (N=48)	p-value
H. Pylori	Positive	32 (64.0)	18 (37.5)	0.008
	Negative	18 (36.0)	30 (62.5)	

Table 5 shows the prevalence of the H. Pylori test among the case and control groups. H. Pylori was positive among 64% of women in the case group VS 37.5% of women in the control group and there are significant statistical differences between the two groups (p-value=0.008).

Table 6. The laboratory findings among participants

Variables	Laboratory investigation		
	Case group (N=50)	Control group (N=48)	p-value
Hb (Mean ± SD)	10.1± 1.19	10.8± 1.4	0.003
S. Na (Mean ± SD)	139.68 ± 7.79	133.9± 8.21	0.001
S. Ca (Mean ± SD)	13.54± 1.2	16.6 ±1.2	0.539
S. Cl (Mean ± SD)	98.3± 6.5	97.3 ± 5.1	0.741
S. K (Mean ± SD)	3.32 ±1.07	3.27 ± 2.4	0.739
ALT (Mean ± SD)	24.45± 2.8	23.17 ± 4.6	0.018
AST (Mean ± SD)	31.9 ± 3.1	28.95 ± 4.61	0.001

ALP (Mean \pm SD)	117.2 \pm 5.7	112.04 \pm 2.5	0.001
TSB (Mean \pm SD)	0.68 \pm 0.14	0.73 \pm 0.1	0.042
B. Urea (Mean \pm SD)	22.72 \pm 1.44	23.2 \pm 1.42	0.096
S. Creatinine (Mean \pm SD)	0.82 \pm 0.44	0.73 \pm 0.1	0.211
Urine ketone (Mean \pm SD)	2.0 \pm 0.5	0.08 \pm 0.01	0.001
TSH (Mean \pm SD)	1.57 \pm 0.71	1.85 \pm 0.47	0.29
T4 (Mean \pm SD)	108.54 \pm 8.86	102.27 \pm 29.5	0.161
RBS (Mean \pm SD)	100.54 \pm 11.03	98.33 \pm 8.37	0.272

Table 6 shows the laboratory findings among participants. Women with HG show a significantly lower Hb mean in comparison to healthy women and p-value= 0.003.

Similarly, the S. Na, ALT, AST, ALP, TSB, and Urine ketone show significant differences between the women with HG and control healthy pregnant since the p-value <0.05.

On the other hand, no significant differences between S. Ca, S. Cl, S. K, B. Urea, S. Creatinine, TSH, T4, and RBS were noticed since the p-value > 0.05.

4. Discussion:

Hyperemesis gravidarum (HG) is a debilitating condition characterized by severe nausea and vomiting during pregnancy, often leading to significant maternal morbidity [10]. Several factors, including sociodemographic, clinical, anthropometric, and laboratory parameters, have been studied to understand the risk factors for HG, including the potential role of *Helicobacter pylori* infection. This study aimed to assess whether *H. pylori* infection is a significant risk factor for HG by comparing various characteristics between case and control groups.

The sociodemographic characteristics (Table 1) revealed no significant differences between the case and control groups regarding age, occupation, residency, or educational level. While slightly more participants in the case group were aged 30–39 (36.0%) compared to the control group (16.7%), the difference was not statistically significant (p=0.053). This matching is ensured to reduce the effect of confounders and bias effect. However, it is well known from the literature that sociodemographic factors alone are unlikely to directly influence HG development but may contribute to varying degrees of healthcare access and reporting [11].

Clinical variables (Table 2) showed primigravidity was more common in the case group (16.0%) compared to the control group (4.2%), but this was not statistically significant (p=0.151). These findings contrast with some studies suggesting that primigravidity may be a risk factor for HG such as the two most recent studies by [12,13]. Gestational age was comparable between the two groups (p=0.251), indicating appropriate matching to reduce confounders.

The symptoms and signs of HG in the case group (Table 3) highlighted the clinical burden of the condition, with 84.0% experiencing hypotension, 76.0% reporting concentrated urine, and 48.0% having sunken eyes. These findings reflect the severe dehydration and electrolyte imbalances commonly associated with HG, consistent with prior research by [14-16].

Anthropometric characteristics (Table 4) demonstrated no significant differences between the groups in BMI. Both groups had similar proportions of overweight and obese participants, suggesting that pre-pregnancy weight status may not be a key determinant in HG risk and this finding is in line with a most

recent study by [17] that shows no effect of weight on the risk of HG. Notably, weight loss was reported in the case group (1.45 ± 0.29 kg), a hallmark of HG which is in line with a study [18], but no corresponding data was available for the control group to facilitate comparison.

The prevalence of *H. pylori* infection (Table 5) was significantly higher in the case group (64.0%) than the control group (37.5%) ($p=0.008$). This finding supports the hypothesis that *H. pylori* infection may act as a risk factor for HG. Similar results have been reported in the literature [19,21] suggesting that *H. pylori* may exacerbate nausea and vomiting through mechanisms such as increased gastric inflammation, delayed gastric emptying, and altered hormonal responses.

Laboratory findings (Table 6) further underscored the physiological disturbances associated with HG. Significant differences were observed between the case and control groups in hemoglobin (10.1 ± 1.19 vs. 10.8 ± 1.4 , $p=0.003$), serum sodium (139.68 ± 7.79 vs. 133.9 ± 8.21 , $p=0.001$), ALT (24.45 ± 2.8 vs. 23.17 ± 4.6 , $p=0.018$), AST (31.9 ± 3.1 vs. 28.95 ± 4.61 , $p=0.001$), ALP (117.2 ± 5.7 vs. 112.04 ± 2.5 , $p=0.001$), and urine ketones (2.0 ± 0.5 vs. 0.08 ± 0.01 , $p=0.001$). These findings indicate greater dehydration, hepatic involvement, and metabolic disturbances in the case group which is in line with [22, 23]. Elevated ketones and liver enzymes are well-documented features of severe HG, reflecting the physiological stress and malnutrition associated with the condition and this is in agreement [24, 25]. Conversely, no significant differences were found in serum calcium, chloride, potassium, or other metabolic parameters.

Overall, the study's findings support the hypothesis that *H. pylori* infection is associated with an increased risk of HG. This aligns with prior studies that have identified a significant association between *H. pylori* and HG, suggesting that screening and eradication of *H. pylori* may play a role in mitigating HG severity. However, further research is warranted to explore the causal mechanisms, potential confounders, and the impact of regional and population-specific factors. Additionally, the non significant differences in sociodemographic and anthropometric variables emphasize the multifactorial nature of HG, where *H. pylori* infection may act as one of several contributing factors rather than the sole determinant.

5. Conclusions

H. pylori infection is significantly associated with an increased risk of hyperemesis gravidarum, highlighting its potential role as a contributing factor. The study confirms that HG is caused by a combination of multiple factors. It found no significant differences in sociodemographic characteristics or anthropometric measurement between individuals affected by HG and those who were not. Laboratory findings indicate significant physiological disturbances in HG cases, including dehydration, hepatic involvement, and metabolic imbalances.

Acknowledgments: The researchers would like to express their gratitude to College of Medicine, University of Thi-Qar, assisted us in completing this project.

Conflict of interest statement: The authors have no conflict of interest with respect to the publication of this article.

The Authors Involved in the Research: The research Wasan Shareef Hameed and Nadia Saddam Fahad, contributed to the research design to analyze the results and write the manuscript, and the authors approved the final version for submission.

Ethical Consideration: The ethical committee Granted by the Iraqi Board of Medical Specialization.

REFERENCES

1. Jennings LK, Mahdy H. Hyperemesis gravidarum. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
2. Miernyk KM, Bulkow LR, Gold BD, Bruce MG, Reasonover AL, Morris JM, et al. Prevalence of *Helicobacter pylori* among Alaskans: factors associated with infection and comparison of urea breath test and anti-*Helicobacter pylori* IgG antibodies. *Helicobacter*. 2018;23(3):e12482. <https://doi.org/10.1111/hel.12482>
3. Elmahdy M, Elmarsafawy A, Elkafash D. Association between *Helicobacter pylori* infection and hyperemesis gravidarum. *Int J Reprod Contracept Obstet Gynecol*. 2016;5(9):3175–3180. <https://doi.org/10.18203/2320-1770.ijrcog20163007>
4. Bello AK, Umar AB, Borodo MM. Prevalence and risk factors for *Helicobacter pylori* infection in gastroduodenal diseases in Kano, Nigeria. *Afr J Med Health Sci*. 2018;17(1):41–46. https://doi.org/10.4103/ajmhs.ajmhs_12_18
5. Isa MA, Onuegbu CU, Bulakarima AU, Hassan MN, Adam U, Garba MI, et al. Seroprevalence of *Helicobacter pylori* among pregnant women attending antenatal clinic in University of Maiduguri Teaching Hospital, Borno State, Nigeria. *Bioglobina*. 2015;2(2):18–23.
6. Ali A, AlHussaini KI. *Helicobacter pylori*: a contemporary perspective on pathogenesis, diagnosis and treatment strategies. *Microorganisms*. 2024;12(1):222. <https://doi.org/10.3390/microorganisms12010222>
7. Kazemzadeh M, Kashanian M, Baha B, Sheikhansari N, Moradi Y. Evaluation of the relationship between *Helicobacter pylori* infection and hyperemesis gravidarum. *Med J Islam Repub Iran*. 2014;28:72.
8. Cardaropoli S, Rolfo A, Todros T. *Helicobacter pylori* and pregnancy-related disorders. *World J Gastroenterol*. 2014;20(3):654–664. <https://doi.org/10.3748/wjg.v20.i3.654>
9. Gupta A, Shetty S, Mutalik S, Bhatt P, Gurram AK, Yadav D. Treatment of *Helicobacter pylori* infection and gastric ulcer: need for novel pharmaceutical formulation. *Heliyon*. 2023;9(10):e20406. <https://doi.org/10.1016/j.heliyon.2023.e20406>
10. Joshi A, Chadha G, Narayanan P. From discomfort to distress: a critical analysis of hyperemesis gravidarum in the emergency room. *Cureus*. 2023;15(8):e44004. <https://doi.org/10.7759/cureus.44004>
11. Solomon D, Morka G, Wayessa ZJ. Determinants of hyperemesis gravidarum among pregnant women in public hospitals of Guji, West Guji, and Borana zones, Oromia, Ethiopia, 2022. *SAGE Open Med*. 2023;11:20503121231196713. <https://doi.org/10.1177/20503121231196713>
12. Pont S, Bond DM, Shand AW, Khan I, Roberts CL. Risk factors and recurrence of hyperemesis gravidarum: a population-based record linkage cohort study. *Acta Obstet Gynecol Scand*. 2024;103(12):2392–2400. <https://doi.org/10.1111/aogs.14871>
13. Mohammed Seid A, Mehari EA, Bekalu AF, Tiruneh AM, Amare AT. Prevalence of hyperemesis gravidarum and associated factors among pregnant women at comprehensive specialized hospitals in northwest Ethiopia: multicenter cross-sectional study. *SAGE Open Med*. 2024;12:20503121241257163. <https://doi.org/10.1177/20503121241257163>
14. Jennings LK, Mahdy H. Hyperemesis gravidarum. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 <https://www.ncbi.nlm.nih.gov/books/NBK532917/>
15. Popa SL, Barsan M, Caziuc A, Pop C, Dita MO, Popa LC, et al. Life-threatening complications of hyperemesis gravidarum. *Exp Ther Med*. 2021;21(6):642. <https://doi.org/10.3892/etm.2021.10054>

16. Lee NM, Saha S. Nausea and vomiting of pregnancy. *Gastroenterol Clin North Am.* 2011;40(2):309–334. <https://doi.org/10.1016/j.gtc.2011.03.009>
17. Jenabi E, Salehi AM, Aghababaei S, Bashirian S. Pre-pregnancy body mass index and the risk of hyperemesis gravidarum: a systematic review and meta-analysis. *Clin Exp Obstet Gynecol.* 2024;51(4). <https://doi.org/10.31083/j.ceog5104090>
18. Galletta MAK, Cavalcanti MHA, Barros MDS, Menezes-Filho JA. Weight loss among pregnant women hospitalized because of hyperemesis gravidarum: is there a lack of nutrition intervention? *Nutr Clin Pract.* 2022;37(4):887–895. <https://doi.org/10.1002/ncp.10794>
19. Li L, Li L, Zhou X, Xiao S, Gu H, Zhang G. Helicobacter pylori infection is associated with an increased risk of hyperemesis gravidarum: a meta-analysis. *Gastroenterol Res Pract.* 2015;2015:278905. <https://doi.org/10.1155/2015/278905>
20. Ng QX, Venkatanarayanan N, De Deyn MLZQ, Ho CYX, Mo Y, Yeo WS. A meta-analysis of the association between Helicobacter pylori infection and hyperemesis gravidarum. *Helicobacter.* 2018;23(1):e12455. <https://doi.org/10.1111/hel.12455>
21. Hussein KS. Hyperemesis gravidarum in first-trimester pregnant Saudi women: is Helicobacter pylori a risk factor? *Front Physiol.* 2020;11:926. <https://doi.org/10.3389/fphys.2020.00926>
22. Gaba N, Gaba S. Study of liver dysfunction in hyperemesis gravidarum. *Cureus.* 2020;12(6):e8709. <https://doi.org/10.7759/cureus.8709>
23. Ahmed KT, Almashrawi AA, Rahman RN, Hammoud GM, Ibdah JA. Liver diseases in pregnancy: diseases unique to pregnancy. *World J Gastroenterol.* 2013;19(43):7639–7646. <https://doi.org/10.3748/wjg.v19.i43.7639/->
24. Imaeda M, Tanaka S, Fujishiro H, Tsuda Y, Mimura M, Kato N. Risk factors for elevated liver enzymes during refeeding of severely malnourished patients with eating disorders: a retrospective cohort study. *J Eat Disord.* 2016;4:37. <https://doi.org/10.1186/s40337-016-0123-6>
25. Aslan MM, Yeler MT, Biyik I, Aktas A, Ustun YE. Hematological parameters to predict the severity of hyperemesis gravidarum and ketonuria. *Rev Bras Ginecol Obstet.* 2022;44(5):458–466. <https://doi.org/10.1055/s-0041-1741439>

How to cite this article
Hameed WS, Fahad NS. Evaluation Of the Relationship Between H. Pylori Infection and Hyperemesis Gravidarum. <i>Journal of Biomedicine and Biochemistry.</i> 2026;5(2):34-42. doi: 10.57238/jbb.2026.7432.1169