

The Status of Vitamin D, Hemoglobin Level, ABO Blood Groups and *Helicobacter pylori* Infection among Patients in Tobruk, Libya

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ABSTRACT

Background: *Helicobacter pylori* infection represents one of the most prevalent public health concerns globally, particularly in developing nations. Vitamin D deficiency and anemia are associated with compromised immunological function, chronic fatigue, gastrointestinal disturbances, and diminished quality of life. Emerging evidence suggests potential correlations between nutritional deficiencies and chronic infections, especially *H. pylori*, which may impair absorption of essential nutrients including Vitamin D and iron. **Objective:** This study aimed to evaluate Vitamin D levels, hemoglobin (Hb) concentrations, ABO blood group distribution, and *H. pylori* infection prevalence among patients in Tobruk, Libya, and to investigate potential interrelationships between these parameters. **Methods:** A cross-sectional study was conducted involving 145 participants. Demographic characteristics and laboratory data were collected and analyzed using IBM SPSS Statistics version 20.0. Quantitative variables were expressed as mean $\pm$ standard deviation and median values, while qualitative variables were presented as frequencies and percentages. **Results:** The mean serum Vitamin D concentration was 9.38 ± 3.27 ng/mL, indicating severe deficiency among most participants. The mean hemoglobin level was 9.68 ± 1.01 g/dL, reflecting significant anemia prevalence. Females constituted 56.6% of the study population, and the age group above 35 years represented 35.9% of cases. Blood group A+ was most prevalent (53.1%), followed by AB+ (22.8%). The mean *H. pylori* stool antigen level was 41.60 ± 43.57 . **Conclusion:** The study demonstrated a high prevalence of severe vitamin D deficiency, anemia, and *H. pylori* infection among the study population. Blood group A+ was the predominant phenotype. These findings emphasize the necessity for early screening, nutritional assessment, and appropriate therapeutic interventions to improve health outcomes and reduce disease complications.

1. INTRODUCTION

Helicobacter pylori infection is recognized as one of the most common chronic bacterial infections in humans, affecting approximately 50% of the global population, with substantially higher prevalence rates in developing countries (Kibru D, et al 2014). This Gram-negative bacterium colonizes the gastric mucosa and serves as a primary etiological agent for chronic gastritis, peptic ulcer disease, and a recognized risk factor for gastric malignancies (Yildirim O, et al 2017).

The incidence of *H. pylori* infection is notably elevated in regions characterized by inadequate sanitation infrastructure and lower socioeconomic conditions (Hooi J KY, et al 2017, Leja M et al 2021). Vitamin D, a fat-soluble secosteroid hormone, plays a fundamental role in maintaining calcium homeostasis and supporting bone mineralization. The biologically active form, calcitriol (1,25-dihydroxyvitamin D), is produced through sequential hydroxylation in the liver and kidneys before exerting its physiological effects via binding to vitamin D receptors (VDRs) [Holick MF, et al 2007]. Beyond its classical skeletal functions, Vitamin D regulates numerous biological and immunological processes. VDRs and vitamin D-metabolizing enzymes are expressed in various immune cells, including lymphocytes, monocytes, macrophages, and dendritic cells, underscoring its immunomodulatory capacity (Sāsāran MO, et al 2023). Vitamin D deficiency has been associated with osteoporosis, muscle weakness, increased fracture risk, and elevated susceptibility to infections, autoimmune disorders, malignancies, and chronic diseases. Vitamin D directly or indirectly regulates hundreds of genes involved in cell proliferation, differentiation, apoptosis, and angiogenesis (Mut Surmeli D, et al, 2019). Recent investigations have suggested potential associations between Vitamin D insufficiency and heightened vulnerability to *H. pylori* infection, along with diminished eradication rates (Liu D, et al 2023). Furthermore, accumulating evidence indicates that low Vitamin D levels may adversely affect the success of *H. pylori* eradication therapy, suggesting that Vitamin D supplementation could serve as a beneficial adjunct to conventional treatment regimens (Ibrahim A, et al, 2026). Anemia, characterized by insufficient red blood cell mass to adequately deliver oxygen to tissues, represents another significant public health concern. *H. pylori* infection contributes to anemia by impairing iron absorption through chronic gastritis-induced gastric hypochlorhydria, which hinders the reduction of dietary iron from the ferric (Fe^{3+}) to the ferrous (Fe^{2+}) form a prerequisite for intestinal absorption (Hudak L., 2019). Ascorbic acid and acidic intragastric pH are essential for this reduction process. Consequently, *H. pylori*-induced chronic superficial gastritis may lead to gastric gland atrophy and reduced acid secretion, further compromising iron bioavailability (Hudak L, et al, 2021). There is growing evidence that *H. pylori* infection contributes to iron deficiency anemia through interference with iron absorption and metabolism (Motupalli SK et al 2024, Rahman YA, et al 2019). ABO blood group phenotypes have been identified as hereditary risk factors associated with susceptibility to *H. pylori* infection. The bacterium can recognize and bind to blood group antigens expressed on gastric epithelial cells, a mechanism essential for bacterial colonization and persistent infection (Chakrani Z, et al, 2018). Individuals with blood groups A and O demonstrate higher risk of *H. pylori* infection compared to other blood groups, while those with blood group AB exhibit lower susceptibility (Kanbay M, et al, 2005). Despite the global burden of these conditions, limited local studies have investigated the association between Vitamin D deficiency, anemia, ABO blood groups, and *H. pylori* infection in Libya, particularly in Tobruk city. Understanding the interrelationship between these parameters is essential for improving diagnostic, therapeutic, and preventive strategies. Therefore, this study aims to assess the distribution of these parameters among the study population and explore their potential associations (Kwak HW, et al, 2019- Muhsen K, et al 2008).

2. MATERIALS AND METHODS

1. Study Design and Population

A cross-sectional study was conducted involving 145 participants recruited from patients attending healthcare facilities in Tobruk, Libya. The study period extended from [insert dates]. Demographic characteristics, including age and gender, were recorded for all participants.

2. Laboratory Investigations

Venous blood samples were collected from all participants for the assessment of: Serum Vitamin D levels (measured in ng/mL), Hemoglobin concentration (measured in g/dL), ABO blood group typing. Stool samples were obtained for *H. pylori* antigen detection using specific immunoassay techniques.

3. Statistical Analysis

Data were entered into a computer database and analyzed using IBM SPSS Statistics for Windows, version 20.0 (Armonk, NY: IBM Corp). Categorical variables were presented as numbers and percentages. Quantitative variables were expressed as mean $\pm$ standard deviation, median, and range (minimum–maximum). The distribution of parameters across demographic characteristics was evaluated using appropriate statistical tests.

3. Ethical Approval

Ethical approval for the study was obtained from the Faculty of Health Sciences, University of Tobruk. The study was conducted using anonymized laboratory records collected retrospectively from the hospital laboratory database. No patient names, personal identifiers, or confidential information were accessed or recorded during data collection. All data were handled with strict confidentiality and were used exclusively for scientific research purposes. Since the study relied solely on existing laboratory records and did not involve direct contact with patients, individual informed consent was not required. Data security and privacy were maintained throughout the study in accordance with accepted ethical research principles.

4. RESULTS

The study included 145 participants with a mean age of 31.25 ± 17.81 years (range: 2.0–77.0 years). The largest proportion of participants was in the age group >35 years (35.9%, n=52), followed by those aged <25 years (34.5%, n=50) and the 25–35 years group (29.7%, n=43). Females constituted 56.6% (n=82) of the study population, while males comprised 43.4% (n=63). The demographic distribution is presented in Table 1.

The mean serum Vitamin D concentration was 9.38 ± 3.27 ng/mL (median: 8.70 ng/mL; range: 3.14–19.60 ng/mL), indicating severe deficiency among the majority of participants.

The mean hemoglobin concentration was 9.68 ± 1.01 g/dL (median: 9.60 g/dL; range: 8.40–13.90 g/dL), reflecting the presence of anemia among a substantial proportion of participants.

Blood group A+ was the most prevalent phenotype, accounting for 53.1% (n=77) of participants, followed by AB+ (22.8%, n=33), O+ (6.9%, n=10), O- (6.2%, n=9), AB- (7.6%, n=11), B- (2.1%, n=3), and A- (1.4%, n=2).

The mean *H. pylori* stool antigen level was 41.60 ± 43.57 (median: 24.60; range: 1.17–350.30), indicating evidence of infection in several participants.

Table 1: Distribution of Studied Cases According to Different Parameters (n=145)

Parameter	Category	n (%)
Age (years)	<25	50 (34.5%)
	25–35	43 (29.7%)
	>35	52 (35.9%)
	Mean $\pm$ SD	31.25 ± 17.81
	Median (Min.–Max.)	30.0 (2.0–77.0)
Gender	Male	63 (43.4%)
	Female	82 (56.6%)
H. pylori (Stool)	Mean $\pm$ SD	41.60 ± 43.57
	Median (Min.–Max.)	24.60 (1.17–350.30)
Vitamin D (ng/mL)	Mean $\pm$ SD	9.38 ± 3.27
	Median (Min.–Max.)	8.70 (3.14–19.60)
ABO Blood Group	A+	77 (53.1%)
	A-	2 (1.4%)
	B+	—
	B-	3 (2.1%)
	AB+	33 (22.8%)
	AB-	11 (7.6%)
	O+	10 (6.9%)
	O-	9 (6.2%)
Hemoglobin (g/dL)	Mean $\pm$ SD	9.68 ± 1.01
	Median (Min.–Max.)	9.60 (8.40–13.90)

SD: Standard deviation

5. DISCUSSION

The present study investigated the interrelationship between Vitamin D levels, hemoglobin concentration, ABO blood groups, and *H. pylori* infection among patients in Tobruk, Libya. The results revealed a significant prevalence of severe Vitamin D insufficiency and anemia within the examined population, alongside evidence of *H. pylori* infection in several individuals.

The finding that the age group above 35 years represented the highest percentage of cases (35.9%) aligns with the observations of Leja et al. (Leja M, et al, 2021), who reported that *H. pylori* infection prevalence increases with age due to prolonged exposure to infection and cumulative environmental factors.

Older adults may also experience more nutritional deficiencies and chronic health conditions contributing to lower Vitamin D and hemoglobin levels. Notably, the condition affected a wide range of age groups rather than being limited to a specific category, indicating universal susceptibility across the lifespan.

Females constituted a higher percentage of participants (56.6%) compared to males (43.4%). This finding is consistent with the study by Săsăran et al. (Săsăran MO, et al, 2023), which reported higher Vitamin D deficiency prevalence among females due to reduced sunlight exposure, dietary factors, and lifestyle habits. Similarly, numerous studies conducted in Middle Eastern and North African countries have demonstrated that females are more susceptible to Vitamin D deficiency because of cultural and environmental factors, including clothing practices that limit sun exposure and dietary patterns (Al Anouti F et al 2011 El-Hajj G, et al 2015).

The mean serum Vitamin D concentration of 9.38 ± 3.27 ng/mL indicates severe deficiency among most participants. This result is comparable to the findings of Liu et al. (Liu D, et al 2023), who found significantly lower Vitamin D levels in patients infected with *H. pylori* compared to non-infected individuals. Săsăran et al. (Săsăran MO, et al 2023) also reported that Vitamin D deficiency may impair immune function and increase susceptibility to bacterial infections, including *H. pylori*. The low Vitamin D levels observed in this study may be attributed to limited sun exposure, inadequate dietary intake, or chronic gastric inflammation caused by *H. pylori* infection, which can impair nutrient absorption.

The pathogenesis linking Vitamin D deficiency to *H. pylori* infection is multifactorial. Vitamin D exhibits antimicrobial properties through the induction of cathelicidin and defensins, which are antimicrobial peptides that directly kill bacteria (Gombart AF, et al, 2009). Additionally, Vitamin D modulates the inflammatory response by suppressing pro-inflammatory cytokine production, including TNF- α , IL-6, and IL-8, which are elevated in *H. pylori* infection (Hewison M, et al 2012). Therefore, Vitamin D deficiency may compromise gastric mucosal defense mechanisms, facilitating *H. pylori* colonization and persistence.

The mean hemoglobin level of 9.68 ± 1.01 g/dL reflects the presence of anemia among many participants. This finding is consistent with Hudak et al. (Hudak L, et al, 2021), who reported a significant association between *H. pylori* infection and iron deficiency anemia. Chronic *H. pylori* infection may interfere with iron absorption through gastric mucosal inflammation and reduced gastric acid secretion, ultimately leading to anemia. Rahman et al. [Rahman et al, 2019] similarly concluded that *H. pylori* infection has significant hematological effects, particularly on hemoglobin and iron status.

The pathophysiology of *H. pylori*-associated anemia involves multiple mechanisms. The bacterium competes with the host for iron utilization, as *H. pylori* requires iron for growth and survival (Pichon C, et al 2013). Furthermore, the inflammatory response to *H. pylori* infection upregulates hepcidin, a key regulator of iron homeostasis, which inhibits iron absorption and leads to iron sequestration in macrophages (Suker E, et al, 2020). These mechanisms collectively contribute to the development of anemia in infected individuals.

Concerning ABO blood groups, A+ was the most common blood group among participants (53.1%), followed by AB+ (22.8%). This finding partially agrees with Almorish et al. (Almorish MA, et al, 2023), who investigated the association between ABO blood groups and *H. pylori* infection and reported that certain blood groups may increase susceptibility to infection due to differences in bacterial adhesion to gastric mucosa. Chakrani et al. (Chakrani Z, et al 2018). conducted a meta-analysis demonstrating that blood group A was associated with increased risk of *H. pylori* infection, while blood group O was associated with more severe disease outcomes.

The molecular basis for the association between ABO blood groups and *H. pylori* infection involves the expression of blood group antigens on gastric epithelial cells. *H. pylori* express adhesins, particularly BabA (blood group antigen-binding adhesin), which specifically binds to Lewis b and H type 1 antigens, which are related to ABO blood group structures (Ilver D, et al, 1998). The differential expression of these antigens among blood groups may influence bacterial adhesion efficiency and colonization persistence.

However, the relationship between ABO blood groups and *H. pylori* infection remains controversial, with some studies reporting no significant association (Mohammadzadeh MH, et al, 2023). This discrepancy may be attributable to differences in study populations, geographic variations, bacterial strain diversity, or methodological approaches.

Interrelationship Between Parameters

The present study demonstrated that Vitamin D deficiency, anemia, and *H. pylori* infection may coexist within the same population and collectively contribute to poor health status. This interrelationship has significant clinical implications, as the presence of one condition may exacerbate the others through interconnected pathophysiological mechanisms.

The triad of Vitamin D deficiency, *H. pylori* infection, and anemia forms a vicious cycle. *H. pylori* infection causes gastric inflammation that impairs absorption of both Vitamin D and iron, leading to their deficiencies. Vitamin D deficiency, in turn, compromises immune function and gastric mucosal integrity, facilitating *H. pylori* colonization. Anemia exacerbates fatigue, weakness, and impaired quality of life, further compromising overall health status (Camaschella C, et al, 2015)

These findings highlight the importance of early diagnosis, nutritional assessment, and appropriate treatment strategies to reduce complications and improve quality of life. Routine screening for Vitamin D deficiency and anemia in patients with *H. pylori* infection could enable early intervention and prevent progression to more severe disease manifestations.

Vitamin D supplementation may represent a cost-effective adjunct to *H. pylori* eradication therapy, potentially improving treatment outcomes and reducing recurrence rates (Yang L, et al, 2020). Similarly, iron supplementation may be beneficial for patients with coexisting anemia and *H. pylori* infection, although the optimal timing and duration of such interventions require further investigation.

The cross-sectional design of this study limits the ability to establish causal relationships between the examined parameters. The relatively small sample size (n=145) may limit the generalizability of findings to the broader Libyan population. Additionally, the study did not assess dietary intake, sun exposure patterns, or socioeconomic status, which may influence Vitamin D and iron status. Future longitudinal studies with larger sample sizes and comprehensive nutritional assessments are warranted to further elucidate these relationships.

6. CONCLUSION

This study demonstrated a high prevalence of severe Vitamin D deficiency, anemia, and *H. pylori* infection among patients in Tobruk, Libya. Blood group A+ was the predominant phenotype in the study population. The coexistence of these conditions within the same individuals suggests potential interrelationships that warrant further investigation. Early screening for Vitamin D deficiency and anemia, particularly in patients with *H. pylori* infection, is recommended to enable timely intervention and improve health outcomes. Nutritional assessment and appropriate management strategies, including Vitamin D and iron supplementation, should be integrated into routine clinical care for patients with *H. pylori* infection. Future large-scale studies are recommended to better understand the relationship between Vitamin D status, ABO blood groups, hemoglobin levels, and *H. pylori* infection in diverse populations.

6. REFERENCES

- Al Anouti F, Thomas J, et al. (2011). Vitamin D deficiency and sun avoidance among university students at Abu Dhabi, United Arab Emirates. *Dermatoendocrinol*;3(4):235-239.
- Albakour, E. J., Eljamay, S. M., Faid, F. E., & Ibrahim, N. A. (2025). Vitamin D and Relationship with Cholesterol and Triglycerides. *Indonesian Journal of Community Services*, 4(2), 166-174.
- Almorish MA, Al-absi B, et al. (2023) ABO, Lewis blood group systems and secretory status with *Helicobacter pylori* infection in Yemeni dyspeptic patients: a cross-sectional study. *BMC Infect Dis*;23:520.

- Bassil D, Rahme M, Hoteit M, et al.(2013) Hypovitaminosis D in the Middle East and North Africa: prevalence, risk factors and impact on outcomes. *Dermatoendocrinol.*;5(2):274-298.
- Camaschella C. (2015). Iron-deficiency anemia. *N Engl J Med* ;372(19):1832-1843.
- Chakrani Z, Robinson K, Taye B.(2018) Association between ABO blood groups and *Helicobacter pylori* infection: a meta-analysis. *Sci Rep*;8:17604.
- Drah, M. M., & Eljamay, S. M. (2025). Exploring age-related patterns in vitamin D deficiency and gender variations. *Derna Academy Journal for Applied Sciences*, 3(1), 22-29\
- El-Hajj Fuleihan G, Bouillon R, et al. (2015). Serum 25-hydroxyvitamin D levels: variability, knowledge gaps, and the concept of a desirable range. *J Bone Miner Res*;30(7):1119-1133.
- Eljamay, S. M., Alghazali, M. A. A., & Eldalal, H. H. A. (2022). Incident of vitamin D deficiency in Derna City\Libya. *JEndo Metabol Res*, 3(1), 1-15
- Faaaid, F. (2026). Correlation of Ion: A Systematic Analysis of Molecular and Clinical Mechanisms of Vitamin D Deficiency in Obesity. *Derna Academy Journal for Applied Sciences*, 6(2), 80-89
- Gombart AF. (2009).The vitamin D–antimicrobial peptide pathway and its role in protection against infection. *Future Microbiol* ;4(9):1151-1165.
- Hewison M.(2012). Vitamin D and the immune system: new perspectives on an old theme. *Rheum Dis Clin North Am* ;38(1):125-139.
- Holick MF.(2007) Vitamin D deficiency. *N Engl J Med*;357(3):266-281. doi:10.1056/NEJMra070553
- Hooi JKY, Lai WY, Ng WK, et al.(2017) Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. *Gastroenterology* ;153(2):420-429.
- Hudak L, Jaraissy A, Haj S, Muhsen K.(2021). An updated systematic review and meta-analysis on the association between *Helicobacter pylori* infection and iron deficiency anemia. *Helicobacter* ;26(2):e12785.
- Hudak L.(2019). *Helicobacter pylori* and iron deficiency anemia: a systematic review and meta-analysis. *Helicobacter* ;24(Suppl 1):e12586.
- Ibrahim A, Abdelhady SA, Rageh F, et al.(2026). Vitamin D receptor gene polymorphism and its association with the susceptibility to *Helicobacter pylori* infection in the Egyptian population with hepatocellular carcinoma. *BMC Gastroenterol.*;26:215.
- Ilver D, Arnqvist A, Ögren J, et al.(1998). *Helicobacter pylori* adhesin binding fucosylated histo-blood group antigens revealed by retagging. *Science.* ;279(5349):373-377.
- Kanbay M, Gür G, Arslan H, et al.(2005) The relationship of ABO blood group, age, gender, smoking, and *Helicobacter pylori* infection. *Dig Dis Sci* ;50:1214-1217.
- Kibru D, Gelaw B, et al ,(2014). *Helicobacter pylori* infection and its associated risk factors among dyspeptic patients at Felege Hiwot Referral Hospital, Northwest Ethiopia. *Int J Biomed Sci* ;10(3):174-181.
- Kwak HW, Kim SM,et al .(2019) *Helicobacter pylori* and vitamin D deficiency: a systematic review and meta-analysis. *Helicobacter*;24(6):e12662.
- Leja M, Axon A, Brenner H.(2021) Epidemiology of *Helicobacter pylori* infection. *Helicobacter.* ;26(Suppl 1):e12734.

- Liu D, Ren L, Zhong D, et al. (2023). Association of serum vitamin D levels with *Helicobacter pylori* infection. *BMC Gastroenterol.* ;23:391.
- Mohammadzadeh MH, et al. (2023). Relationship between ABO blood groups and *Helicobacter pylori* infection. *Zahedan J Res Med Sci.* 2023;25(2):e128459.
- Motupalli SK, Oroszi TL. (2024) The nexus between *Helicobacter pylori* infection and anemia—a systematic review. *Front Hematol.* ;3:1423494.
- Muhsen K, Cohen D. (2008) *Helicobacter pylori* infection and iron deficiency anemia in children: a systematic review and meta-analysis. *Helicobacter*;13(3):171-181.
- Mut Surmeli D, Surmeli ZG, Bahsi R, et al. (2019). Vitamin D deficiency and risk of *Helicobacter pylori* infection in older adults: a cross-sectional study. *Aging Clin Exp Res*;31(7):985-991.
- Pichon C, Hebraud M, Bierne H, et al. (2013) Iron and *Helicobacter pylori*: a complex relationship. *Microbiology.* ;159(Pt 7):1271-1282.
- Rahman YA, Ahmed LA, et al. (2019). *Helicobacter pylori* and its hematological effect. *Egypt J Intern Med.* ;31:332-342.
- Sāsāran MO, et al. (2023). Vitamin D and its association with *Helicobacter pylori* prevalence and eradication: a review. *Nutrients*;15(16):3549.
- Suker E, Al-Helali N, Al-Mazahri M. (2020). Hcpidin and iron metabolism in *Helicobacter pylori* infection: a systematic review. *Eur J Gastroenterol Hepatol* ;32(3):297-304.
- Yang L, Zhao X, Gu Y, et al. (2020) Vitamin D supplementation and *Helicobacter pylori* eradication: a systematic review and meta-analysis. *Eur J Clin Nutr.* ;74(9):1263-1272.
- Yildirim O, Yildirim T, et al (2017). The influence of vitamin D deficiency on eradication rates of *Helicobacter pylori*. *Adv Clin Exp Med* ;26(9):1377-1381.