Scientific Journal for Publishing in Health Research and Technology

Volume 2, Issue 2, 2026, Pages: 122-132

Journal homepage: https://sjphrt.com.ly/index.php/sjphrt/en/index

Impact of vitamin B12 deficiency on Hematological parameters and Its relationship with Helicobacter pylori infection in Derna City, Libya

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تأثير نقص فيتامين ب12 على المؤشرات الدموية وعلاقته بعدوى جرثومة المعدة في مدينة درنة، ليبيا

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Received: March 25, 2026	Accepted: April 25, 2026	Published: May 16, 2026

Abstract:

This study was conducted to evaluate the alterations in hematological parameters and investigate the pathophysiological relationship between *Helicobacter pylori* (*H. pylori*) infection and vitamin B12 deficiency in clinical cases presenting with symptoms associated with cobalamin depletion. A total of 120 adult patients were enrolled and stratified into two distinct groups based on their vitamin B12 status. The first group (vitamin B12-deficient) comprised 36 men (30.0%) and 55 women (45.8%), while the second group (normocobalaminemic controls) consisted of 14 men (11.6%) and 15 women (12.5%). Supervised blood samples were collected from all participants to perform comprehensive laboratory analyses, including a complete blood count (CBC), serum vitamin B12 quantification, and active *H. pylori* screening.The laboratory findings revealed a profound and statistically significant correlation between active *H. pylori* infection and systemic vitamin B12 deficiency. Specifically, 45 female patients (81.8%) and 25 male patients (69.4%) who tested positive for vitamin B12 deficiency were concurrently infected with *H. pylori*. Regarding erythrocyte profiles, hemoglobin (HGB) concentrations and mean corpuscular volume (MCV) were significantly altered in vitamin B12-deficient patients of both sexes compared to the healthy control group. Additionally, a statistically significant decrease in mean corpuscular hemoglobin (MCH) was observed exclusively among female patients, whereas male subjects demonstrated only a slight, non-significant variance in this parameter. Regarding other blood components, total white blood cell (WBC) counts showed a marginal, non-significant elevation in the deficient cohorts, while mean platelet (PLT) counts and associated platelet indices were lower in both deficient males and females compared to their healthy counterparts. In conclusion, vitamin B12 deficiency represents a highly prevalent yet frequently silent or asymptomatic micronutrient disorder worldwide. Within the evaluated Derna community, this deficiency was found to be widespread across various age groups and

both genders. While a systemic shortage of cobalamin exerts negligible effects on total red blood cell, white blood cell, and platelet counts, it induces major diagnostic variations in hemoglobin concentrations, MCV values, and female-specific MCH profiles. These variations are heavily driven by the concurrent high prevalence of *H. pylori* infections, which chronically compromise gastric absorption pathways.

Keywords: Vitamin B12 deficiency, Hematological parameters, *H.pylori* and infection.

الملخص

أُجريت هذه الدراسة لتقييم التغيرات في المؤشرات الدموية(Hematological parameters) ، واستقصاء العلاقة المرضية الفسيولوجية (Pathophysiological relationship) بين عدوى جرثومة المعدة (*Helicobacter pylori*) ونقص فيتامين ب12 (VB12) في الحالات السريرية التي تعاني من أعراض مرتبطة بنفاذ الكوبالامين .(Cobalamin depletion) وقد شملت الدراسة 120 مريضاً من البالغين، تم تقسيمهم إلى مجموعتين منفصلتين بناءً على مستويات فيتامين ب12 لديهم؛ حيث ضمت المجموعة الأولى (المصابة بنقص فيتامين ب12) 36 رجلاً (بنسبة 30.0%) و55 امرأة (بنسبة 45.8%)، في حين تألفت المجموعة الثانية (المجموعة الضابطة ذات المستويات الطبيعية من الكوبالامين) من 14 رجلاً (بنسبة 11.6%) و15 امرأة (بنسبة 12.5%). وجُمعت عينات الدم من جميع المشاركين تحت إشراف طبي لإجراء تحاليل مخبرية شاملة، تضمنت فحص صورة الدم الكاملة(CBC) ، وتقدير كمية فيتامين ب12 في المصل، والكشف عن العدوى النشطة لجرثومة المعدة. وأظهرت النتائج المخبرية وجود ارتباط وثيق وذو دلالة إحصائية (Statistically significant) بين عدوى جرثومة المعدة النشطة والنقص القياسي في فيتامين ب12؛ وتحديداً، تبيّن أن 45 مريضة من الإناث (بنسبة 81.8%) و25 مريضاً من الذكور (بنسبة 69.4%) ممن ثبتت إصابتهم بنقص فيتامين ب12، كانوا مصابين تزامنياً بجرثومة المعدة. وفيما يتعلق بمؤشرات خلايا الدم الحمراء(Erythrocyte profiles) ، فقد طرأ تغير ملحوظ ومعنوي في تركيزات الهيموجلوبين (HGB) وحجم الكريات المتوسط (MCV) لدى المرضى الذين يعانون من نقص فيتامين ب12 من كلا الجنسين مقارنة بالمجموعة الضابطة السليمة. بالإضافة إلى ذلك، لوحظ انخفاض ذو دلالة إحصائية في معدل هيموجلوبين الكرية المتوسط (MCH) بين المريضات من الإناث حصرياً، في حين أظهر الذكور تبايناً طفيفاً وغير معنوي إحصائياً في هذا المؤشر. وعن مكونات الدم الأخرى، أظهر إجمالي عدد خلايا الدم البيضاء (WBC) ارتفاعاً هامشياً غير معنوي في المجموعات التي تعاني من النقص، بينما انخفض متوسط عدد الصفائح الدموية (PLT) والمؤشرات المرتبطة بها لدى الذكور والإناث المصابين بالنقص مقارنة بنظرائهم الأصحاء. وفي الختام، يمثل نقص فيتامين ب12 أحد أكثر اضطرابات المغذيات الدقيقة (Micronutrient disorders) انتشاراً في جميع أنحاء العالم، وغالباً ما يكون صامتاً أو خاوياً من الأعراض السريرية الواضحة. وضمن مجتمع الدراسة الذي تم تقييمه في مدينة درنة، وُجد أن هذا النقص واسع الانتشار عبر مختلف الفئات العمرية ولدى كلا الجنسين. وبينما يسفر النقص القياسي في الكوبالامين عن تأثيرات طفيفة يمكن إهمالها على إجمالي أعداد خلايا الدم الحمراء، وخلايا الدم البيضاء، والصفائح الدموية، فإنه يتسبب في تغيرات تشخيصية جوهرية في تركيزات الهيموجلوبين، وقيم حجم الكريات المتوسط(MCV) ، ومؤشرات هيموجلوبين الكرية المتوسط (MCH) الخاصة بالإناث؛ وهي تغيرات تُعزى بشكل وثيق إلى الانتشار المرتفع المتزامن لعدوى جرثومة المعدة، والتي تؤدي مزمنّاً إلى إضعاف مسارات الامتصاص المعدي.

الكلمات المفتاحية: نقص فيتامين ب12، المؤشرات الدموية، جرثومة المعدة والعدوى.

INTRODUCTION

Vitamin B12 (VB12), alternatively referred to as cobalamin (CBL), stands as a critical water-soluble micronutrient indispensable for the functional integrity of every cell within the human

body (Gherasim et al., 2013; Yamada, 2013). Physiologically, it exerts its biological effects primarily through its two active coenzyme forms: methylcobalamin and adenosylcobalamin (Gherasim et al., 2013). Given that the human body lacks the metabolic pathways required for de novo synthesis of cobalamin, its maintenance relies strictly on dietary acquisition (Hannibal et al., 2016). In a standard human diet, sustainable concentrations of vitamin B12 are exclusively derived from animal-source foods, including meat, fish, poultry, eggs, and dairy products, which accumulate the vitamin via symbiotic microbial synthesis (Cozzolino, 2016).

At the cellular level, vitamin B12 serves as a mandatory cofactor in DNA synthesis, regulation, and cellular metabolism. It operates in tandem with folate to facilitate nucleotide production, making it crucial for high-turnover tissues, most notably the bone marrow during erythropoiesis (Green et al., 2013; Koury & Ponka, 2004). Consequently, a clinical deficiency in this micronutrient hinders purine and thymidylate synthesis, impairs proper DNA replication, and triggers erythroblast apoptosis, leading to ineffective erythropoiesis and macrocytic anemia (Koury & Ponka, 2004; Krzywański et al., 2020). Beyond its hematological significance, cobalamin is vital for neurological health, where it plays a definitive role in maintaining the myelin sheaths of nerve cells and supporting healthy brain development (Green et al., 2013). Furthermore, altered cobalamin levels present a highly complex relationship with dermatological health, as both systemic deficiency and excess can manifest through diverse cutaneous symptoms (Green et al., 2013).

In recent clinical research, the gastroduodenal pathogen *Helicobacter pylori* (*H. pylori*) has emerged as a major etiologic agent responsible for inducing vitamin B12 deficiency (Akçam, 2010). Chronically colonizing the gastric mucosa, *H. pylori* induces architectural changes in the stomach lining, leading to chronic gastritis and peptic ulcer disease (Kadhim et al., 2018). This persistent inflammatory state compromises gastric physiology, resulting in the malabsorption of dietary nutrients (Kadhim et al., 2018). Specifically, the infection diminishes the secretion of gastric acid and pepsin, while concurrently impairing the mucosal capacity to secrete intrinsic factor—a transport glycoprotein essential for cobalamin absorption in the terminal ileum (Haddad & Abdulrahman, 2020; Kadhim et al., 2018). Interestingly, studies have shown that *H. pylori* can drive the depletion of serum vitamin B12 levels even in the early stages of infection prior to the development of severe gastric atrophy, with the severity of the deficiency often correlating with the overall bacterial load in the antral mucosa (Gümürdülü et al., 2003; Serin et al., 2002).

As a direct consequence of this micronutrient depletion and localized chronic inflammation, *H. pylori* infection exerts a profound impact on systemic hematological parameters (Haile & Timerga, 2021; Sağlam & Civan, 2023). Patients suffering from chronic *H. pylori* infections frequently present with significant alterations across multiple blood cell indices, including reductions in hemoglobin concentration, mean corpuscular volume (MCV), serum iron levels, and total erythrocyte counts (Haile & Timerga, 2021; Mwafy & Afana, 2018). Moreover, the systemic inflammatory response triggered by the bacterium can lead to distinct fluctuations in cellular defense profiles, notably causing an elevation in inflammatory markers and white blood cell counts, alongside structural variations in platelet indices (Rahman et al., 2019; Sağlam & Civan, 2023). While the epidemiological links between *H. pylori* infection, vitamin B12 status, and altered hematological profiles have been thoroughly documented in various global populations, comprehensive baseline research detailing these specific clinical interactions remains significantly scarce within the Libyan demographic, particularly within the Northeast region.

Aim of the Study

The primary objective of this study was to estimate the prevalence rate of vitamin B12 (VB12) deficiency within a sample of the population in Derna City, Northeast Libya, and to analyze its

association with key hematological parameters and *Helicobacter pylori* (*H. pylori*) infection. To date, there is limited empirical evidence regarding the determinants of vitamin B12 deficiency and appropriate representative population sampling methods in this region. Consequently, no comprehensive study investigating the dynamics of cobalamin insufficiency and its pathophysiological correlations has been previously recorded in Derna City, making this research a vital baseline contribution to the regional medical literature.

MATERIALS AND METHODS

Study Design and Participants

This cross-sectional study enrolled 120 adult individuals suspected of having vitamin B12 deficiency who were attending various private diagnostic laboratories in Derna City. Under strict medical supervision, whole blood and serum samples were collected from each participant to perform a complete blood count (CBC), measure serum vitamin B12 levels, and conduct *H. pylori* testing. Based on established laboratory reference ranges, the participants were stratified into two distinct groups: the vitamin B12-deficient group and the normocobalaminemic (normal control) group. Additionally, a structured questionnaire was administered to gather essential clinical and demographic data from each participant, including age, sex, presence of clinical symptoms (such as fatigue and lethargy), and relevant medical history regarding other comorbidities.

Statistical Analysis

Statistical analysis was performed using Minitab and GraphPad Prism software packages. The normality of the collected data was verified prior to analysis, and a one-way analysis of variance (ANOVA) was applied to determine statistical variances between the groups. In all analyses, a *p*-value of less than 0.05 ($p < 0.05$) was considered statistically significant (Paulson, 2008).

RESULTS

During the designated study period, 120 adult subjects presenting with clinical symptoms clinically associated with vitamin B12 deficiency were recruited for evaluation. The study cohort comprised 50 (41.7%) male and 70 (58.3%) female participants, as illustrated in Figure 1.

Biochemical and laboratory assessments revealed a high burden of cobalamin depletion among the symptomatic individuals. Specifically, the prevalence of confirmed vitamin B12 deficiency was 30.0% (n = 36) among male subjects and 45.8% (n = 55) among female subjects. In contrast, the proportion of participants who presented with matching symptoms but tested negative for vitamin B12 deficiency (normal controls) was 11.6% (n = 14) for males and 12.5% (n = 15) for females, as detailed in Figure 2.

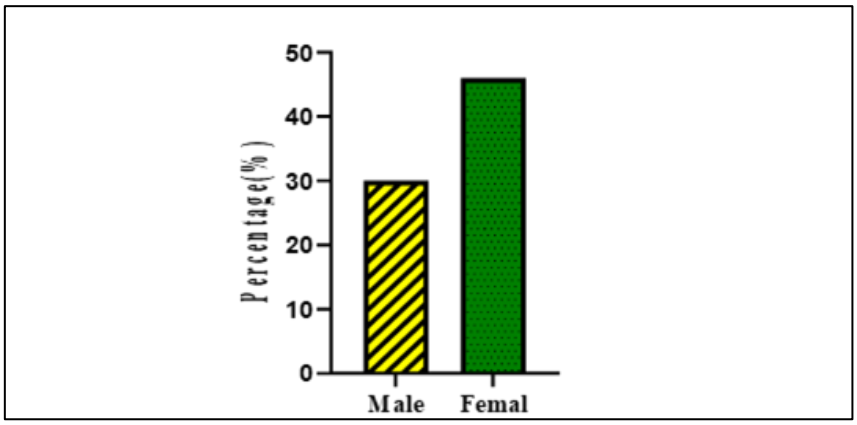

Figure 1: Percentage of adult patients with vitamin B12 deficiency

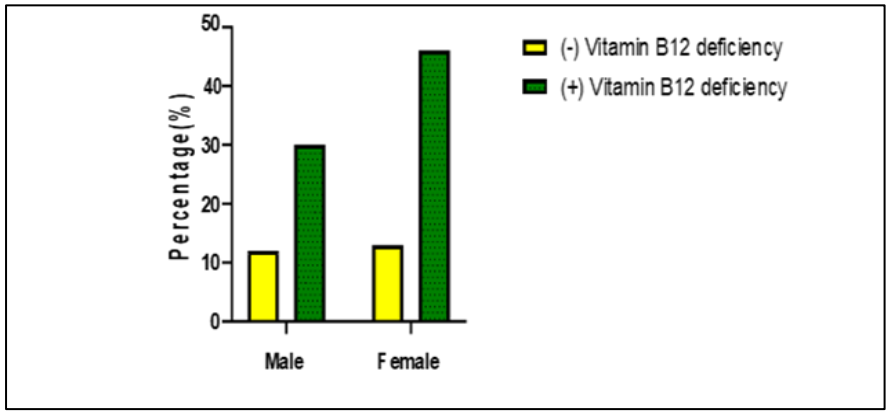

Figure 2: Percentage number of cases upon positive/negative vitamin B12 deficiency in each adult gender

Age distribution of male and female subjects

A total of 120 adult cases, comprising 50 males and 70 females, participated in this study. Table 1 displays the detailed distribution of these cases stratified by specific age groups for each gender individually, utilizing standardized 10-year cohort intervals.

Among the female subjects, the age group of 31–40 years exhibited the highest prevalence of positive vitamin B12 deficiency at 10.83%, closely followed by the age groups of 41–50 years (9.16%), 21–30 years (8.33%), over 60 years (>60) at 6.66%, and 51–60 years (5.83%). Conversely, for the male individuals, the highest prevalence of positive vitamin B12 deficiency was observed in the 21–30 years age cohort (10.83%), followed sequentially by the age groups of 41–50 years (5.83%), 31–40 years (4.16%), and 51–60 years (3.33%). Notably, the adolescent and young adult cohort aged 15–20 years demonstrated the lowest overall frequency of vitamin B12 deficiency across both genders.

Table 1: Age distribution of male and female adult subjects

Age (Years)	Female No of cases (%)		Male No of cases (%)	
	Negative with *Vitamin B12 deficiency*	Positive with *Vitamin B12 deficiency*	Negative with *Vitamin B12 deficiency*	Positive With *Vitamin B12 deficiency*
15-20	1 (0.83%)	6 (5%)	1(0.83%)	2 (1.66%)
21-30	3 (2.5%)	10(8.33%)	3 (2.5%)	13 (10.8%)
31-40	3 (2.5%)	13(10.8%)	3 (2.5%)	5 (4.16%)
41-50	2 (1.66%)	11(9.16%)	2(1.66%)	7 (5.83%)

51-60	3 (2.5%)	7 (5.83%)	3 (2.5%)	4(3.33%)
>60	3 (2.5%)	8 (6.66%)	2(1.66%)	5 (4.16%)
Total	15 (12.5%)	55(45.8%)	14(11.6%)	36(30%)

Data are expressed as mean ± SEM of each gender. Within each column for male or female separately, means with different superscript (a, b or c) were significantly different at $p<0.05$. Where means without superscripts mean that there is no significant difference ($p>0.05$).

Results of hematological parameters

For every patient diagnosed with vitamin B12 deficiency, complete blood count (CBC) values, including differential cell counts, were recorded and compared to those of the negative control subjects. In general, the data indicated that the hematological responses between both genders followed a similar trend. Table 2 displays the comprehensive findings of the analysis for RBC, WBC, and PLT counts across the study groups.

The mean RBC count for female and male adult subjects in the deficient group was $4.26 \pm 0.06 \times 10^6/\mu l$ and $4.65 \pm 0.10 \times 10^6/\mu l$, respectively; these values showed no statistically significant difference compared to the vitamin B12-negative (control) subjects. In contrast, hemoglobin (HGB) concentrations were significantly lower in the B12-deficient group than in the negative controls, dropping to 10.36 ± 0.22 g/dl in females and 10.95 ± 0.24 g/dl in males ($p < 0.05$).

Regarding red blood cell indices, mean corpuscular volume (MCV) varied significantly between patients and controls ($p < 0.05$), averaging 76.62 ± 0.88 fl in females and 80.59 ± 0.97 fl in males. Additionally, mean corpuscular hemoglobin (MCH) levels were found to be 24.41 ± 0.45 pg for females (representing a statistically significant decrease) and 27.86 ± 0.46 pg for males, which showed a non-significant difference when compared to the control group.

Regarding total leukocyte and platelet profiles, the white blood cell (WBC) counts in B12-deficient subjects increased to $8.49 \pm 0.28 \times 10^3/\mu l$ in females and $7.54 \pm 0.27 \times 10^3/\mu l$ in males, revealing no statistically significant differences between genders or against controls. Meanwhile, the mean platelet (PLT) counts in patients with vitamin B12 deficiency decreased to $263.1 \pm 8.22 \times 10^3/\mu l$ in females and $254.27 \pm 8.80 \times 10^3/\mu l$ in males, which also remained within a non-significant statistical variance.

Table2: Values of RBC, Hb, MCV, MCH, WBC and PLT in positive/negative vitaminB12 deficiency in adults' subjects

Parameters	**Female**		**Male**	
	Negative Mean± SEM	Positive Mean± SEM	Negative Mean± SEM	Positive Mean± SEM
RBC ($\times 10^6/\mu l$)	4.40±0.07 a	4.26± 0.06 a	4.60± 0.10 a	4.65±0.10 a
Hb (g/dl)	11.73± 0.20 a	10.36±0.22 b	13.14±0.39 a	10.95±0.24 b
MCH (pg)	28.30± 0.28a	24.41± 0.45 b	28.67±0.28 a	27.86±0.46 a
MCV (fL)	82.66 ±0.59 a	76.62± 0.88 b	85.28±0.65 a	80.59±0.97 b

WBC ($\times 10^3/\mu l$)	7.50± 0.40a	8.49± 0.28a	6.89± 0.30 a	7.54± 0.27 a
PLT ($\times 10^3/\mu l$)	265.1± 8.08a	263.1± 8.22a	263.6± 8.51 a	254.27±8.80a

Data are expressed as mean ± SEM of each gender. Within each column for male or female separately, means with different superscript (a, b or c) were significantly different at $p<0.05$. Where means without superscripts mean that there is no significant difference ($p>0.05$).

Result of vitamin B12 deficiency and relationship with *H.pylori*

The findings demonstrated a link between vitamin B12 insufficiency and *H. pylori*, with women more vulnerable than healthy individuals (45 female by 81.8%) and 25 males by 69.4%. In comparison to healthy individuals, it was discovered that women are more prone to this (45 females by 81.8%) and 25 males by 69.4%.

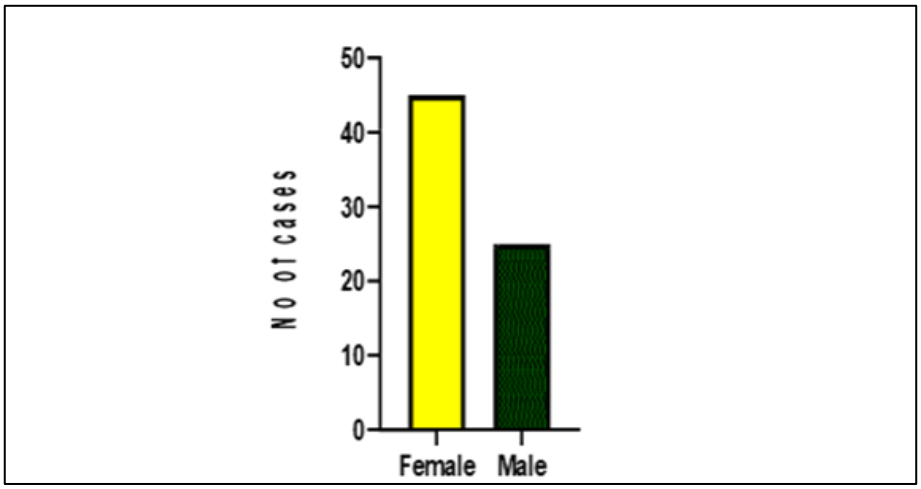

Figure 3: Shows the number of cases of *H.pylori* and vitamin B12 deficiency in males and females.

DISCUSSION

The epidemiological profile and prevalence of vitamin B12 (VB12) deficiency exhibit profound variations across different global populations, socio-economic strata, and geographic regions. Prior to this investigation, epidemiological data characterizing the exact baseline prevalence of cobalamin insufficiency within the Libyan population remained remarkably scarce and largely undocumented. The empirical findings generated from the current survey establish that VB12 deficiency is extensively widespread within the examined population of Derna City. Notably, a high accumulation of positive deficiency cases was observed among individuals spanning the 15 to 50 age brackets, a trend that manifested consistently across both genders. This widespread distribution among young and middle-aged adults underscores the clinical significance of screening for cobalamin depletion even in non-elderly cohorts. This is particularly critical because initial cobalamin depletion often progresses as a "silent" or asymptomatic condition before presenting with severe neurological or hematological manifestations.

Furthermore, recent regional data from the Libyan pediatric and adolescent demographics indicate that gastrointestinal factors frequently impair the long-term bioavailability and absorption of this vital micronutrient, proving that age-related susceptibility is heavily modulated by localized medical conditions rather than nutritional intake alone (Faris et al.,

2026). This perspective is corroborated by broader clinical evidence indicating that low initial serum levels of vitamin B12 can remain hidden for long periods across diverse patient populations, emphasizing the need for targeted biochemical screening protocols in clinical settings (Trimarchi et al., 2004).

The outcomes of this study clearly demonstrate that a deficiency in vitamin B12 exerts a statistically significant and detrimental impact on critical hematological parameters—specifically hemoglobin concentration (HGB), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH). The pathophysiology linking cobalamin depletion to altered red blood cell indices is deeply rooted in molecular erythropoiesis. At the cellular level, vitamin B12 operates as a mandatory metabolic cofactor; its systemic deficiency directly inhibits both purine and thymidylate syntheses, thereby severely disrupting cellular DNA synthesis and replication within high-turnover bone marrow cells. This cellular disruption induces a state of megaloblastic maturation arrest, driving erythroblast apoptosis and halting normal cell division, which ultimately results in clinical anemia driven by ineffective erythropoiesis.

Our findings regarding these altered erythrocyte profiles align closely with established international clinical data, including investigations conducted in Poland and other ambulatory care environments, which underscore the structural dependence of hemoglobin formation on adequate cobalamin status. However, it is clinically vital to recognize that utilizing some traditional hematological parameters alone can sometimes yield unreliable results or delay diagnosis, as macrocytosis may be masked by coexisting iron deficiency, highlighting the diagnostic necessity of pairing complete blood counts with more sensitive biochemical or metabolic biomarkers (Asma et al., 2010).

From a physiological standpoint, because mature erythrocytes are structurally responsible for the systemic transport of oxygen to peripheral tissues, vital organs, and muscles, any functional reduction in total hemoglobin or corpuscular efficiency drastically compromises cellular oxygenation. This induced state of chronic tissue hypoxia accounts for the primary physical complaints reported by the patients in this study, namely generalized physical weakness, persistent lethargy, compromised cognitive performance, and a diminished sense of overall wellbeing.

A major focus of this investigation was to elucidate the close relationship between *Helicobacter pylori* (*H. pylori*) infection and the development of vitamin B12 deficiency. The current study revealed a remarkably high co-prevalence of active *H. pylori* infections among cobalamin-deficient patients in Derna City, with females showing an infection rate of 81.8% and males presenting 69.4%. The underlying mechanism driving this correlation is the biological capacity of *H. pylori* to cause chronic gastritis and localized mucosal ulceration. The chronic inflammatory cell infiltration triggered by the bacterium degrades the functional architecture of the gastric mucosa, resulting in a profound downregulation of gastric acid (hypochlorhydria or achlorhydria) and pepsin secretion. Under normal physiological conditions, gastric acid and pepsin are essential to liberate dietary vitamin B12 from its bound animal protein matrix. Furthermore, the progression of *H. pylori*-induced gastritis impairs the capacity of gastric parietal cells to synthesize and secrete intrinsic factor—the primary transport glycoprotein required for stable cobalamin binding and its subsequent receptor-mediated endocytosis in the terminal ileum. Consequently, this multi-stage malabsorption cascade prevents the gastrointestinal tract from absorbing dietary cobalamin, precipitating systemic deficiency over time.

Our observations mirror clinical investigations performed in Iraq by Kadhim and associates, which similarly identified *H. pylori* as a definitive, primary etiological driver of secondary cobalamin deficiency. This pathological model is further supported by prospective multicenter studies confirming that chronic *H. pylori* gastritis regularly induces dual micro-nutritional stress, primarily depleting both iron and vitamin B12 storage reserves before transitioning into

severe autoimmune gastric phenotypes (Osmola et al., 2024). Additionally, clinical research conducted across pediatric and younger demographics demonstrates that even in childhood, *H. pylori* colonization significantly alters serum levels of ferritin and vitamin B12, establishing a life-long trajectory toward nutrient depletion if left untreated (Akcam et al., 2007).

The clinical reality of *H. pylori*-mediated micronutrient depletion is also highlighted by cross-sectional data from various developing and sub-Saharan environments, which heavily link active infection states with systemic anemia, iron deficiency, and full-scale iron deficiency anemia (Eyoum Bille & Kouitcheu Mabeku, 2022). This systemic impact becomes even more profound when compounding medical variables are present; for instance, *H. pylori* infection has been shown to exacerbate or accelerate vitamin B12 deficiency in patients concurrently utilizing medications like metformin, which independently interfere with ileal cobalamin absorption (Gökışık & Uyar, 2020). Finally, the neurological implications of this connection are profound, as the micro-nutritional malabsorption induced by *H. pylori* frequently results in systemic cobalamin drops severe enough to trigger or worsen complex neurological and neuropsychiatric symptoms secondary to peripheral nerve demyelination (Özcan et al., 2013). Collectively, these findings underscore that eradicating *H. pylori* and closely monitoring hematological profiles are vital clinical steps to prevent and manage the systemic complications of vitamin B12 deficiency.

CONCLUSION

In summary, this investigation highlights that vitamin B12 (VB12) deficiency remains one of the most prevalent yet frequently "silent" micronutrient disorders globally, characterized by an insidious onset that often evades early clinical detection, with the empirical data gathered from this study demonstrating its widespread prevalence across various age groups and both genders within the population of Derna City, Libya. Regarding the hematological manifestations, the statistical analysis revealed distinct, parameter-specific impacts induced by cobalamin depletion; specifically, while VB12 deficiency exhibited no statistically significant effect on the total red blood cell (RBC) count in either gender, it exerted a profound and significant impact on hemoglobin (HGB) concentrations and mean corpuscular volume (MCV) across both sexes, alongside a significant effect on mean corpuscular hemoglobin (MCH) observed exclusively in female patients, whereas no significant variance was recorded among males. Furthermore, this systemic shortage of cobalamin demonstrated no discernible or statistically significant influence on total white blood cell (WBC) counts or platelet (PLT) parameters in either gender. Beyond these isolated hematological variations, this study establishes a strong epidemiological and pathophysiological connection between VB12 deficiency and *Helicobacter pylori* infection, with both conditions being concurrently prevalent across the demographic strata of Derna, driven by the fact that the persistent mucosal inflammation and subsequent gastric hypochlorhydria induced by *H. pylori* serve as a key etiological driver of dietary cobalamin malabsorption. Ultimately, these findings underscore the clinical necessity of establishing routine, comprehensive screening protocols that pair sensitive biochemical markers with complete blood counts, alongside targeted *H. pylori* eradication strategies, as such proactive measures are vital to mitigate the long-term risk of secondary hematological disorders and hidden micronutrient deficiencies within the Libyan community.

ACKNOWLEDGEMENT

The authors extend their sincere thanks and appreciation to the private analytical laboratories (Tabib Laboratory, Derna Specialized Laboratory, Al-Rashid Laboratory, and Al-Razi Laboratory) in Derna, Libya. We would also like to express our gratitude to the individuals who participated in this study.

ETHICS

Participants provided their informed consent after being fully informed about the objectives and methods of the study.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

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