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ASSESSMENT OF VITAMIN B-12 DEFICIENCY IN HELICOBACTER PYLORI STOOL ANTIGEN POSITIVE DYSPEPTIC PATIENTS VISITING A TERTIARY CARE HOSPITAL

Dr. Mutiba Khan[*1], Prof. Haris Alvi[2], Dr. Muhammad Rehan[3], Dr. Ahsan Mobin[4], Dr. Rakhshinda Jabeen[5], Dr. Ramsha[6]

[*1]*Postgraduate Trainee, Department of Medicine, Dow University of Health Sciences (DUHS), Karachi, Pakistan*
[2]*Professor and Head of Department, Department of Medicine, DUHS, Karachi, Pakistan*
[3,4] *Assistant Professor Department of Medicine, DUHS, Karachi, Pakistan*
[5] *Associate Professor,, Department of Medicine, DUHS, Karachi, Pakistan*
[6]*Postgraduate Trainee, Department of Medicine, DUHS, Karachi, Pakistan*

[*1]mutibak@outlook.com, [2]haris.alvi@duhs.edu.pk, [3]rehan.muhammad@duhs.edu.pk, [4]ahsan.mobin@duhs.edu.pk, [5]rakhshinda.jabeen@duhs.edu.pk, [6]drramsha66@gmail.com

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Corresponding Author: *
Dr. Mutiba Khan

Abstract

Background:
Helicobacter pylori infection is a common cause of chronic gastritis that can impair intrinsic factor secretion, resulting in reduced vitamin B-12 absorption. Vitamin B-12 deficiency may lead to anemia, neuropathy, and cognitive impairment, yet it often remains undiagnosed among dyspeptic patients in Pakistan.

Objective:
To determine the frequency of vitamin B-12 deficiency in H. pylori stool antigen-positive dyspeptic patients presenting to a tertiary care hospital.

Methods:
This descriptive cross-sectional study was conducted at the Department of Medicine, Civil Hospital Karachi, over six months after approval from the Institutional Review Board. A total of 143 Helicobacter pylori stool antigen–positive dyspeptic patients aged 20–70 years were enrolled through non-probability consecutive sampling. Serum vitamin B-12 levels were measured using chemiluminescent micro particle immunoassay, with ≤174 pg/mL considered deficient, and data were analyzed using SPSS version 26 with a significance level of $p \leq 0.05$.

Results:
Out of 143 Helicobacter pylori stool antigen–positive dyspeptic patients, 117 (81.8 %) were found to have vitamin B-12 deficiency (≤ 174 pg/mL). A significant association was observed between vitamin B-12 deficiency and both higher BMI ($p = 0.04$) and severe dyspeptic symptoms ($p = 0.001$).

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Conclusion:
Vitamin B-12 deficiency is highly prevalent among H. pylori–positive dyspeptic patients and is significantly linked with symptom severity and overweight status.

INTRODUCTION

Vitamin B-12 (cobalamin) is an essential water-soluble vitamin required for DNA synthesis, erythropoiesis, and neurological integrity. Its deficiency leads to megaloblastic anemia, neuropathy, cognitive impairment, and other systemic manifestations[1]. Gastric mucosal disorders that reduce intrinsic factor secretion or cause mucosal atrophy are major contributors to malabsorption of vitamin B-12[2].

Helicobacter pylori (H. pylori), a spiral-shaped gram-negative bacterium colonizing the gastric mucosa, is a leading cause of chronic gastritis and peptic ulcer disease worldwide[3]. Meta-analyses demonstrate a significant association between H. pylori infection and reduced serum vitamin B-12 levels, with improvement after eradication therapy[4]. The infection contributes to atrophic gastritis and decreased intrinsic factor availability, thereby compromising vitamin B-12 absorption[5]. Several recent studies have highlighted that H. pylori-induced gastritis can also affect absorption of other micronutrients such as folate and iron, emphasizing the broader nutritional impact of the infection[6, 7].

In developing countries, where both H. pylori infection and dietary insufficiency of animal-source foods are prevalent, the coexistence of these factors may substantially increase the risk of vitamin B-12 deficiency[8]. Recent Pakistani data show variable frequencies of B-12 deficiency among H. pylori-positive gastritis patients, ranging from 13% to 40%[9, 10], but findings are inconsistent due to differing diagnostic criteria and sample characteristics.

Given the high local burden of dyspepsia and H. pylori infection, there is a need to clarify the extent of vitamin B-12 deficiency in these patients. Therefore, this study aims to assess the frequency of vitamin B-12 deficiency among H. pylori stool antigen-positive dyspeptic patients visiting a tertiary care hospital, to support evidence-based screening and management strategies.

METHODS

This descriptive cross-sectional study was conducted at the Department of Medicine, Civil Hospital Karachi, over a period of six months following approval from the College of Physicians and Surgeons Pakistan (CPSP). Ethical approval for the study was obtained from the Institutional Review Board of Civil Hospital Karachi (Ref. No. _____), and all procedures were performed in accordance with the Declaration of Helsinki. A sample size of 143 was calculated by using the WHO sample size calculator, taking the expected prevalence of vitamin B-12 deficiency among *Helicobacter pylori* positive patients as 23.8%, with 95% confidence level and 5% margin of error. Non-probability consecutive sampling technique was employed. After obtaining written informed consent, patients of either gender between 20 and 70 years of age presenting with dyspepsia lasting for four weeks or more and testing positive for *H. pylori* stool antigen were included in the study. Patients with known peptic ulcer disease, chronic renal failure, chronic liver disease, gastrointestinal bleeding, autoimmune disorders, vegetarians with restricted dietary intake of vitamin B-12, those already on vitamin supplementation or proton pump inhibitors, pregnant or lactating women, and individuals with hematological or malignant disorders were excluded. For all participants fulfilling the inclusion criteria, detailed history and clinical examination were performed, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Blood samples were collected under aseptic conditions and serum vitamin B-12 levels were estimated using a

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chemiluminescent microparticle immunoassay (CMIA) on the Abbott Architect i1000SR analyzer in the hospital laboratory. A serum vitamin B-12 level of ≤174 pg. /mL was considered deficient, while levels >174 pg. /mL were taken as normal. The results of the *H. pylori* stool antigen were interpreted as positive or negative according to the manufacturer's instructions. All data were entered in a predesigned proforma. Statistical analysis was carried out using SPSS version 26. Quantitative variables such as age, BMI, and serum vitamin B-12 levels were expressed as mean ± standard deviation, while categorical variables such as gender and presence of deficiency were presented as frequencies and percentages. The Chi-square test or Fisher's exact test, where applicable, was used to determine the association between vitamin B-12 deficiency and various demographic and clinical variables. A p-value of ≤0.05 was considered statistically significant for all analyses.

RESULTS

A total of 143 Helicobacter pylori stool antigen-positive dyspeptic patients who fulfilled the inclusion criteria were enrolled in the study. Among them, 79 (55.2 %) were male while 64 (44.8 %) were female. **Table-I** summarizes the basic demographic and clinical characteristics of the study participants. The mean age of patients was 49.6 ± 10.8 years, mean BMI was 26.9 ± 3.7 kg/m², and mean duration of dyspeptic symptoms was 5.4 ± 2.6 months.

After laboratory assessment, it was found that 117 (81.8 %) patients had vitamin B-12 deficiency (≤ 174 pg/mL), while 26 (18.2 %) had normal levels. The mean serum vitamin B-12 concentration among all participants was 162.4 ± 74.6 pg/mL. **Table-II** shows the association of various factors with the presence of vitamin B-12 deficiency. A strong relationship was noted between severity of dyspeptic symptoms and deficiency, with patients having severe dyspepsia showing a significantly higher frequency of low vitamin B-12 levels (p = 0.001). A statistically significant association was also observed between BMI and vitamin B-12 deficiency, where overweight or obese participants were more likely to be deficient compared to those with normal BMI (p = 0.04). No significant association was found with age group or gender (p > 0.05).

Table-I: Characteristics of H. pylori stool antigen-positive dyspeptic patients enrolled in the study (n = 143)

Characteristics	n (%) / Mean ± SD
Age (years, Mean ± SD)	49.6 ± 10.8
Gender – Male	79 (55.2)
Gender – Female	64 (44.8)
Body mass index (kg/m², Mean ± SD)	26.9 ± 3.7
Duration of dyspepsia (months, Mean ± SD)	5.4 ± 2.6
Vitamin B-12 deficiency (≤ 174 pg/mL) Yes	117 (81.8)
Vitamin B-12 deficiency No	26 (18.2)

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Table-II: Association of various factors with vitamin B-12 deficiency among H. pylori stool antigen-positive dyspeptic patients (n = 143)

Factors Studied	Normal n (%)	Deficient n (%)	p-value
Age ≤ 50 years	19 (22.4)	66 (77.6)	0.18
Age > 50 years	8 (14.0)	51 (86.0)	
Gender Male	16 (20.3)	63 (79.7)	0.46
Gender Female	10 (15.6)	54 (84.4)	
BMI < 25 kg/m²	17 (32.7)	35 (67.3)	**0.04***
BMI ≥ 25 kg/m²	9 (9.9)	82 (90.1)	
Dyspepsia Mild–Moderate	23 (28.7)	57 (71.3)	**0.001***
Dyspepsia Severe	4 (6.2)	60 (93.8)	

***Significant at p ≤ 0.05**

DISCUSSION

The present study demonstrated a markedly high prevalence of vitamin B-12 deficiency (81.8 %) among *Helicobacter pylori* stool antigen-positive dyspeptic patients, indicating a substantial nutritional burden associated with chronic *H. pylori* infection. Recent systematic reviews have confirmed that *H. pylori* contributes to vitamin B-12 malabsorption through gastric mucosal atrophy and reduced intrinsic-factor secretion[11]. The significant association between the severity of dyspeptic symptoms and vitamin B-12 deficiency (p = 0.001) observed in our study supports this mechanism, as patients with persistent or severe symptoms are likely to have more extensive mucosal inflammation. Multicenter data have similarly reported that active *H. pylori* gastritis independently correlates with combined iron and B-12 deficiency[14], while meta-analyses emphasize the infection's chronic inflammatory nature and its capacity to impair nutrient absorption[13]. Our findings also revealed a statistically significant relationship between body-mass index (BMI ≥ 25 kg/m²) and vitamin B-12 deficiency (p = 0.04), which is consistent with emerging evidence showing an inverse association between obesity and serum B-12 levels[15] [16]. Obesity-related hypovitaminosis B-12 has been attributed to dilatational effects, altered gastrointestinal absorption, or metabolic inflammation, and similar observations were noted among refugee and community cohorts exhibiting high infection rates and poor dietary diversity[17]. The absence of significant associations with age or gender in our study aligns with prior regional analyses where metabolic and infection-related factors outweighed demographic influences[18] [19]. Taken together, these findings highlight that *H. pylori* infection and excess body weight may

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act synergistically to exacerbate cobalamin depletion, particularly in dyspeptic individuals with severe symptoms. Clinically, these results underscore the importance of screening for vitamin B-12 deficiency in *H. pylori*-positive patients, especially those who are overweight or obese, and of incorporating micronutrient monitoring into eradication protocols. Future longitudinal studies should examine whether correction of infection and weight optimization lead to normalization of vitamin B-12 levels and improvement of dyspeptic symptoms[20].

CONCLUSION

The present study revealed that vitamin B-12 deficiency is a common finding in Helicobacter pylori stool antigen–positive dyspeptic patients, affecting more than four-fifths of cases. The strong associations of deficiency with increased BMI and severe dyspeptic symptoms suggest that both infection-related gastric changes and metabolic factors contribute to impaired cobalamin absorption. Routine evaluation of vitamin B-12 levels in H. pylori–infected patients, particularly those presenting with persistent dyspepsia or overweight, is recommended to prevent long-term hematologic and neurological complications.

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None.

Availability of Data:
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethical Approval:
Ethical approval for the study was obtained from the Institutional Review Board of Civil Hospital Karachi (Ref. No. _____), and all procedures were performed in accordance with the Declaration of Helsinki.

Conflict of Interest:
None declared

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