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Original Research Article

Vitamin B12 Deficiency in Patients with Helicobacter Pylori Infection

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Abstract: *Helicobacter pylori infection is a common gastrointestinal infection and may contribute to micronutrient deficiencies, including vitamin B12 deficiency, through impaired gastric function and reduced nutrient absorption. Evidence regarding the frequency of vitamin B12 deficiency among infected patients remains clinically relevant, particularly in resource-limited settings.* ***Objective:*** *To determine the frequency of vitamin B12 deficiency in patients with Helicobacter pylori infection.* ***Methods:*** *This study was conducted on 185 patients using a non-probability sampling technique in the General Medicine Department, Hayatabad Medical Complex, Peshawar, from 29-06-2023 to 29-12-2023. Patients aged 18 to 70 years, of both genders, with a positive H. pylori stool antigen test were included. Patients with chronic liver disease, renal disease, immunodeficiency disorders, and pregnancy were excluded. Vitamin B12 deficiency was diagnosed using a blood test, with values below 160 pg/mL (118 pmol/L) considered deficient. Data were analysed using IBM SPSS 21. Stratification was performed using the chi-square test, with a p-value ≤ 0.05 considered significant.* ***Results:*** *The mean age of 185 patients was 42.73±15.77 years. Males were 106 (57.3%), and females were 79 (42.7%). Vitamin B12 deficiency was observed in 58 (31.4%) patients. Significant association was found between lower socioeconomic status and vitamin B12 deficiency (p=0.002).* ***Conclusion:*** *Vitamin B12 deficiency was present in 58 (31.4%) of patients with H pylori infection in this study. Lower socioeconomic status was significantly associated with vitamin B12 deficiency.*

Keywords: Helicobacter pylori, vitamin B12 deficiency, socioeconomic status

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Introduction

About half of the global population is affected by the bacterium Helicobacter pylori (H. Pylori). Serious conditions, including chronic gastritis, peptic ulcer, and stomach cancer, are associated with the H. pylori infection. (1) H. pylori is also associated with the onset of conditions such as idiopathic thrombocytopenic purpura, vitamin B12 deficiency, as well as mucosa-associated lymphoid tissue lymphoma. Such H. pylori-associated diseases may respond better to treatment if H. pylori is eliminated. Traditional triple therapy that involves the proton pump inhibitor in addition to clarithromycin and amoxicillin or metronidazole has been the treatment of choice for eradicating the H. pylori for the past two decades. (2)

Invasive and non-invasive techniques can be used to confirm H. pylori infection. Diagnosis of H. pylori antigens in stool, detection of H. pylori antibodies in serum, urine, and oral samples, and detection of urea in breath are all examples of non-invasive diagnostics. The stool antigen test reflects high sensitivity and specificity, and the urea breath test UBT likewise, compared to the invasive procedures. Rapid urease tests (RUT), histopathology, PCR, and fluorescence in situ hybridization are invasive approaches that require stomach tissue for detection. (3,4)

A positive culture from stomach biopsies, or a positive histology and RUT, is the preferred method for making the first diagnosis of H. pylori infection. Existing recommendations for diagnosing H. pylori infection in children involve esophagogastroduodenoscopy and obtaining stomach biopsies for histology and culture. It has been demonstrated that H. pylori UBT and detection of H. pylori antigen in stool are consistent, non-invasive techniques for determining H. pylori eradication. (5-7)

Over half of people with pernicious anemia, caused by untreated vitamin B12 deficiency, have H. pylori. However, medications like lansoprazole and omeprazole can be helpful for PUD, as they also carry the warning that they may lead to serious side effects. (8,9) A study reported that the frequency of vitamin B12 deficiency in patients with H. pylori infection was (86%). (10)

Many gastroenterological disorders have had their treatment strategies modified because H. pylori has been identified as a gastro-duodenal pathogen. This study aims to establish vitamin B12 deficiency in patients with Helicobacter pylori infection. Without conducting costly B12 assays, the findings will aid in developing empirical recommendations for the treatment of vitamin B12 deficiency.

Methodology

The present cross-sectional study was conducted in the General Medicine Department, Hayatabad Medical Complex, Peshawar, from 29-06-2023 to 29-12-2023. Ethical approval (Ref 1266) was obtained before the study began. One hundred eighty-five patients were selected for this study using the WHO sample size calculator, with assumptions including a vitamin B12 deficiency prevalence of 86% (10), a confidence level of 95%, and a margin of error of 5%. A non-probability sampling technique was applied. Inclusion criteria comprised patients aged 18 to 70 years, of both genders, with H. Pylori infection. It was diagnosed after a stool test showed a positive H. pylori antigen. Patients with chronic liver disease, renal disease, immunodeficiency disorders, and pregnancy were excluded.

The purpose, advantages, and risks of this study were explained to the patients, and written informed consent was obtained. Demographics, including gender, age, and socioeconomic status, were recorded for each patient. The patient's medical history was also recorded, and a full physical examination was performed. Patients diagnosed with positive H. Pylori antigens were screened for vitamin B12 deficiency, which was diagnosed by blood test; values <160 pg/mL (118 pmol/L) were categorized as vitamin B12 deficiency. The complete evaluation was performed under the careful supervision of a practitioner with at least 3 years of experience following the fellowship.

Data were analyzed using statistical software (IBM SPSS 21). Frequencies and percentages were reported for qualitative data, such as gender, vitamin B12 deficiency, diabetes, hypertension, and socioeconomic status. Mean and SD were reported for qualitative data, such as age. Vitamin B12 deficiency was stratified by age, gender, diabetes, hypertension, and socioeconomic status to assess effect modifiers. A post-stratification chi-square test was applied, with p-values < 0.05 considered statistically significant.

Results

This study was conducted on 185 patients; the mean age was 42.73±15.77 years. The majority of patients were male (106, 57.3%), while the remaining 79 (42.7%) were women. Regarding the comorbidities, diabetes mellitus was noted in 30 cases (16.2%), hypertension was seen in 51 cases (27.6%), leaving 134 (72.4%). The majority of patients in the present study belonged to a lower socioeconomic background (76, 41.1%) (Table 1).

In the present study, 58 cases (31.4%) were found to have a Vitamin B12 deficiency (Figure 1).

Stratification analysis showed that vitamin B12 deficiency was not associated with age (p = 0.325), gender (p = 0.806), diabetes (p = 0.798), or hypertension (p = 0.726). However, a significant association was observed between vitamin B12 deficiency and lower socioeconomic status (p = 0.002) (Table 2).

Table 1: Demographics

Demographics		n	%
Gender	Male	106	57.3%
	Female	79	42.7%
Diabetes	Yes	30	16.2%
	No	155	83.8%
Hypertension	Yes	51	27.6%
	No	134	72.4%

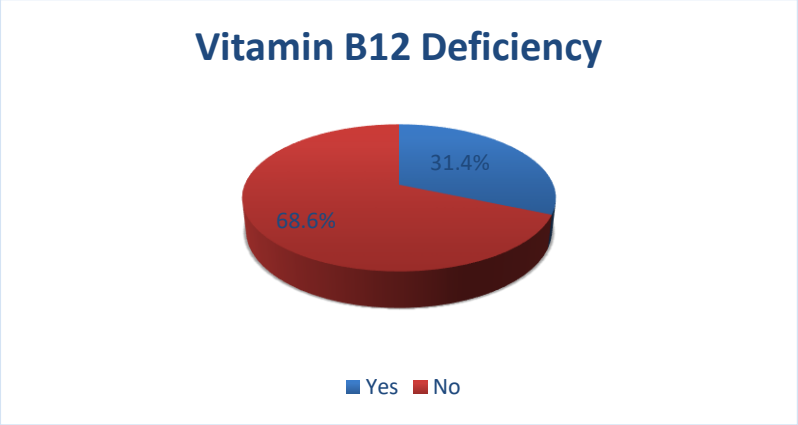

Figure 1: Frequency of vitamin B12 deficiency

Table 2: Stratification of vitamin B12 deficiency with demographics and comorbidities

Demographics and comorbidities		Vitamin B12 deficiency				P value
		Yes		No		
		n	%	n	%	
Age distribution (Years)	18 to 40	29	50.0%	58	45.7%	0.325
	41 to 55	18	31.0%	32	25.2%	
	> 55	11	19.0%	37	29.1%	
Gender	Male	34	58.6%	72	56.7%	0.806
	Female	24	41.4%	55	43.3%	
Socioeconomic status	Lower class	34	58.6%	42	33.1%	0.002
	Middle class	18	31.0%	49	38.6%	
	Higher class	6	10.3%	36	28.3%	
Diabetes	Yes	10	17.2%	20	15.7%	0.798
	No	48	82.8%	107	84.3%	
Hypertension	Yes	15	25.9%	36	28.3%	0.726
	No	43	74.1%	91	71.7%	

Discussion

The literature consistently points toward a connection between chronic Helicobacter pylori colonisation and reduced serum cobalamin levels. Multiple studies have explored this relationship across different populations. In a study of 120 patients presenting with gastritis symptoms, the prevalence of H. pylori positivity was 40.8%, and among those infected, nearly half had vitamin B12 deficiency. (11) This finding

strongly suggested that the bacterium may directly interfere with normal cobalamin absorption mechanisms. Another study reported a lower deficiency rate of 13% among 100 histologically confirmed H pylori gastritis cases, with an additional 5% falling into the borderline category. (12) The mean age in that cohort was 50.8 years, and a male preponderance was noted.

Another study involving 200 H pylori-positive cases revealed that 54.5% had vitamin B12 levels below the normal level, despite only 17% showing macrocytosis on blood films. (13) This observation indicated that biochemical deficiency often precedes haematological manifestations, making it a subclinical entity that could easily be overlooked. The mean age in that study was 41.77 years, and dyspepsia was the most frequent presenting symptom, occurring in 30.5% of cases. Another study reported a 40% prevalence of cobalamin deficiency among 100 H pylori-positive patients, with a mean age of 53.53 years and males in the majority. (14) That study also highlighted that higher monthly income was associated with lower deficiency rates, suggesting a possible protective role of better nutrition or healthcare access.

The pathophysiological link between H pylori infection and impaired cobalamin status has been elaborated through several proposed mechanisms. Chronic infection leads to gastric mucosal inflammation, which over time can progress to atrophic changes in the stomach lining, thereby reducing the secretion of both gastric acid and intrinsic factor. (15) Gastric acid is essential for releasing protein-bound cobalamin from dietary sources, while intrinsic factor is required for its absorption in the terminal ileum. When either of these components is compromised, cobalamin malabsorption from food ensues. Some researchers have suggested that H pylori may trigger an autoimmune response against gastric parietal cells through molecular mimicry, potentially leading to pernicious anaemia in genetically susceptible patients. (15)

The present study's findings add valuable information to the literature. In the present study, the overall frequency of vitamin B12 deficiency was 31.4%. This figure is comparable to the 29.2% observed in the general H pylori positive cohort by Ali UA et al.(11)

Regarding comorbidities, diabetes mellitus was present in 30 (16.2%) patients, and hypertension was noted in 51 (27.6%). When these comorbid conditions were examined in relation to vitamin B12 status, no statistically significant associations were found.

The present study found that most patients with vitamin B12 deficiency were from lower socioeconomic backgrounds. This aligns with the observation of Ali S et al. (14). The likely explanation involves dietary habits, as cobalamin is derived almost exclusively from animal sources such as meat, eggs, and dairy products, which may be less affordable for individuals with limited financial resources. Lower socioeconomic status is often linked to reduced health literacy and delayed healthcare-seeking behaviour, allowing both H pylori infection and its nutritional consequences to persist untreated for longer durations.

Conclusion

In conclusion, this study found that the frequency of vitamin B12 deficiency among patients with Helicobacter pylori infection was 31.4% (58/184). A significant association was observed with lower socioeconomic status. These findings underscore the importance of screening for vitamin B12 deficiency in H pylori-positive patients, especially those from disadvantaged socioeconomic backgrounds. Future research should focus on prospective interventional studies to determine whether successful eradication of H pylori leads to normalisation of B12 levels.

Declarations

Data Availability statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department concerned. (Ref#1266)

Consent for publication

Approved

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Conflict of interest

The authors declared no conflict of interest.

Author Contribution

AW (Postgraduate Resident)
Data collection, Study design and Manuscript drafting
KS (Assistant Professor)
Review of Literature, Critical input.
MR (House Officer)
Literature search
UG (House Officer)
Critical input.
MB (Postgraduate Resident)
Helps in referencing, and interpretation of data

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the study's integrity.

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