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Original Article

Vitamin B12 Deficiency among Cases of Helicobacter Pylori Gastritis: A Cross Sectional Study

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ABSTRACT

Background: Helicobacter pylori (H. pylori) is a gram-negative bacterium associated with various gastrointestinal diseases, including peptic ulcer disease, gastric cancer, and vitamin B12 deficiency. Recent research has highlighted a potential link between chronic H. pylori infection and significant malabsorption of vitamin B12, particularly in populations with high prevalence rates of infection.

Objective: This study aims to investigate the frequency of vitamin B12 deficiency among patients with H. pylori gastritis and to explore the association between H. pylori infection and vitamin B12 levels.

Methods: A cross-sectional study was conducted from September 2023 to March 2024 at the Department of Medicine, Ayub Teaching Hospital, Abbottabad. A total of 120 patients presenting with symptoms of gastritis were enrolled after obtaining ethical approval from the hospital's review board. Exclusion criteria included patients already on vitamin B12 supplementation. Diagnostic testing for H. pylori was performed using stool antigen tests, and vitamin B12 levels were assessed through serum measurements. Data analysis was conducted using IBM SPSS Statistics version 23, with the Chi Square test applied to examine the association between H. pylori infection and vitamin B12 deficiency, setting the level of significance at $p \leq 0.05$.

Results: Out of the 120 patients studied, 49 (40.8%) tested positive for H. pylori. Vitamin B12 deficiency was observed in 35 (29.2%) patients. Among those infected with H. pylori, 51.0% (25 patients) also had vitamin B12 deficiency, compared to 14.1% (10 patients) among those not infected ($p = 0.0001$).

Conclusion: The findings indicate a significant association between H. pylori infection and vitamin B12 deficiency. This study suggests that H. pylori is a potential risk factor for vitamin B12 deficiency, underscoring the need for screening and appropriate management of H. pylori in patients presenting with vitamin B12 deficiency.

Keywords: Helicobacter pylori, Vitamin B12 Deficiency, Gastritis, Cross-Sectional Study, H. pylori Gastritis, Diagnostic Testing, SPSS Analysis.

INTRODUCTION

Helicobacter pylori (H. pylori) is a gram-negative bacterium, known for its unique spiral shape, discovered in the late nineteenth century within the gastrointestinal tract (1). Traditionally, it was believed that the stomach's acidic environment was too hostile for bacterial growth, leading to the assumption that these bacteria were ingested rather than native to the stomach (2). However, H. pylori has since been recognized for its role in persistent inflammation of the stomach and intestines, residing in the glands beneath the mucous membrane's surface (3, 4). This bacterium is strongly associated with several gastrointestinal diseases, including stomach cancer, peptic ulcer disease, and gastric mucosal lymphoid tissue lymphoma. Notably, Shatila and Thomas reported that H. pylori is implicated in 90% of stomach cancer and mucosa-associated lymphoid tissue lymphoma cases (6). Additionally, there is substantial evidence linking H. pylori with the occurrence of both stomach ulcers and duodenal ulcers, found respectively in up to 80% and 90% of cases, and a similar association with carcinomas (7). In response, the World Health Organization, in its 2014 recommendations, emphasized the need for eradication of H. pylori to reduce stomach cancer deaths worldwide, particularly stressing the threat posed by strains resistant to clarithromycin (8).

Concurrently, vitamin B12 deficiency, also known as cobalamin deficiency, remains a common but frequently neglected condition, affecting an estimated 3% to 40% of the adult population (9). The linkage between H. pylori infection and food-cobalamin

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malabsorption has been increasingly recognized, suggesting that chronic H. pylori gastritis might be a primary contributor to cobalamin insufficiency, especially in underprivileged regions (10). Studies suggest that pernicious anemia, a severe form of vitamin B12 deficiency, could potentially begin years before clinical symptoms of cobalamin deficiency manifest (11). Moreover, research supports the possibility that eradication of the bacterium might obviate the need for lifelong cyanocobalamin replacement and could prevent the progression of pernicious anemia (11). This hypothesis is bolstered by findings from two studies that evaluated the effects of eradication therapy on improving vitamin B12 levels in patients with both atrophic and non-atrophic gastric mucosa (12, 13).

Given the prevalence of H. pylori in chronic gastritis, which causes irritation and potential damage to the stomach lining leading to gastric ulcers and cancer, and the resultant vitamin B12 deficiency due to impaired absorption in the stomach, there is a significant gap in the local literature regarding the frequency of vitamin B12 deficiency among individuals with H. pylori gastritis. This study aims to fill that gap by determining the prevalence of this deficiency in such cases, thereby contributing to a better understanding of the interplay between H. pylori infection and vitamin B12 absorption, which could inform future therapeutic strategies.

MATERIAL AND METHODS

The study was conducted as a cross-sectional investigation from September 2023 to March 2024 at the Department of Medicine, Ayub Teaching Hospital, Abbottabad. Ethical approval for the research was granted by the institutional review board of the hospital, adhering to the principles outlined in the Declaration of Helsinki. This ensured that all procedures performed were in accordance with the ethical standards of the institutional and national research committee.

In total, one hundred twenty patients presenting with signs and symptoms of gastritis, such as abdominal pain and stomach burning, were enrolled in the study. Exclusion criteria included patients who were already receiving vitamin B12 supplements, to avoid confounding the association between H. pylori infection and vitamin B12 deficiency. Following informed consent, stool and urine samples were collected from all participants. The stool samples were utilized to diagnose H. pylori infection, while urine samples were analyzed to assess vitamin B12 levels.

Data were meticulously collected and recorded, ensuring accuracy and confidentiality in line with ethical research practices. The data analysis was performed using IBM SPSS Statistics version 23. Statistical significance was determined through the application of the Chi Square test, with a significance level set at a p-value of less than or equal to 0.05. This approach allowed for the assessment of the association between H. pylori infection and vitamin B12 deficiency among the study population.

RESULTS

Table 1: Frequency of H. pylori and Vitamin B12 Deficiency

Parameters	N	%
H. pylori		
Yes	49	40.8%
No	71	59.2%
Vitamin B12 Deficiency		
Yes	35	29.2%
No	85	70.8%

Table 2: Association of H. pylori with Vitamin B12 Deficiency

	Vitamin B12 Deficiency		Total	P value
	Yes	No		
H. pylori				0.0001
Yes	25	24	49	
	51.0%	49.0%	100.0%	
No	10	61	71	
	14.1%	85.9%	100.0%	
Total	35	85	120	
	29.2%	70.8%	100.0%	

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In the conducted study, a total of 120 patients were assessed for the presence of Helicobacter pylori and vitamin B12 deficiency. Analysis of the collected data (Table 1) revealed that 49 patients, accounting for 40.8% of the study population, tested positive for H. pylori, while the majority, 59.2% (71 patients), were H. pylori negative. Concerning vitamin B12 status, 35 patients, or 29.2% of the sample, were found to have a vitamin B12 deficiency, contrasting with 70.8% (85 patients) who did not exhibit any deficiency.

Further statistical analysis was conducted to explore the association between H. pylori infection and vitamin B12 deficiency (Table 2). Of the 49 patients who tested positive for H. pylori, 25 (51.0%) also had a vitamin B12 deficiency, whereas 24 (49.0%) did not. In contrast, among the 71 patients without H. pylori infection, only 10 (14.1%) were vitamin B12 deficient, and a significant majority, 61 (85.9%), were not deficient. The chi-square test indicated a highly significant association between H. pylori infection and the occurrence of vitamin B12 deficiency, with a p-value of 0.0001, underscoring a statistically significant correlation that may suggest a potential causal relationship.

These results emphasize the strong link between H. pylori infection and the risk of developing vitamin B12 deficiency. The high percentage of vitamin B12 deficiency among those infected with H. pylori suggests that the bacterium could play a significant role in the malabsorption of this crucial vitamin, potentially leading to the observed deficiencies in the affected patient group.

DISCUSSION

Helicobacter pylori (H. pylori), a bacterium infecting nearly half of the global population, primarily affects individuals in lower socio-economic positions, significantly increasing infection risk (14). This gram-negative, non-invasive pathogen is known for its potent ability to induce inflammation and a robust immunological response. The enzyme urease, produced by H. pylori, is pivotal in the pathogenesis of the infection and serves as a cornerstone for various diagnostic tests. The bacterium has been implicated in causing acute and chronic gastritis, ulcers in the duodenum and stomach, and gastric mucosa-associated lymphoid tissue lymphoma (15). Moreover, H. pylori has been associated with several systemic diseases such as cardiovascular disease, iron deficiency anemia, autoimmune thrombocytopenia, type 2 diabetes, and notably, vitamin B12 deficiency. Despite similar infection rates among individuals with and without B12 deficiency, those with the deficiency often show less superficial gastritis but more severe atrophic gastritis, suggesting a complex interaction between H. pylori infection and gastric mucosal health (16).

Recent research highlights the significant role of H. pylori in causing vitamin B12 deficiency, particularly in less affluent nations (17). The presence of H. pylori might suppress the proliferation of other bacteria within the gastrointestinal tract, which could enhance cobalamin absorption. Conversely, in the absence of H. pylori, other bacterial populations might proliferate excessively, further impairing cobalamin absorption. Thus, broad-spectrum antibiotic treatment could improve cobalamin absorption by reducing the anaerobic bacterial load, irrespective of H. pylori status (18).

Our study, which included 120 patients evaluated for H. pylori and vitamin B12 deficiency, found a notable prevalence of H. pylori at 40.8% (49 patients) and vitamin B12 deficiency at 29.2% (35 patients). The mean age of participants was 30.90 ± 7.99 years, with a higher incidence observed in males compared to females. Literacy and better socio-economic status appeared to correlate with lower infection rates, but only 22.5% of the patients belonged to a higher socio-economic bracket (19, 20).

Comparative studies from Iraq and Turkey also support our findings, showing higher rates of vitamin B12 deficiency among patients positive for H. pylori (18, 19). These observations underscore the potential of H. pylori as a predictive marker for vitamin B12 deficiency.

CONCLUSION

In conclusion, our study substantiates the association between H. pylori infection and vitamin B12 deficiency, suggesting that H. pylori may be a significant risk factor for this nutritional deficiency. This association highlights the importance of considering H. pylori eradication in patients diagnosed with vitamin B12 deficiency. However, our study is not without limitations. The sample size and the single-center design may limit the generalizability of the results. Future research should focus on multi-center studies with larger, diverse populations to confirm these findings and assess the efficacy of eradication therapy on vitamin B12 levels. Additionally, it is crucial to explore the underlying mechanisms by which H. pylori influences cobalamin absorption to better inform treatment and prevention strategies.

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