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ASSOCIATION BETWEEN HELICOBACTER PYLORI INFECTION AND VITAMIN B12 DEFICIENCY IN DYSPEPTIC PATIENTS: INSIGHTS FROM A LOW-INCOME SETTING

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Abstract

This cross-sectional study examined the relationship between Helicobacter pylori infection and vitamin B12 deficiency among 273 dyspeptic patients undergoing endoscopy at a tertiary care center. Gastric biopsy confirmed H. pylori infection, while serum vitamin B12 levels were assessed biochemically. Overall, 61.2% of patients were H. pylori-positive, among whom 70.6% exhibited vitamin B12 deficiency, compared to 29.5% in uninfected individuals ($p < 0.05$). Socioeconomic status showed a significant association, with deficiency rates of 30% in lower-income groups versus 3.6% in higher-income groups ($p = 0.045$). These findings indicate that H. pylori infection markedly increases the risk of vitamin B12 malabsorption, particularly in resource-limited populations. Routine screening for B12 deficiency in H. pylori-positive dyspeptic patients, along with integrated nutritional and antimicrobial interventions, is recommended to mitigate deficiency-related complications.

INTRODUCTION

Helicobacter pylori (H. pylori) is a gram-negative bacterium that colonizes the gastric mucosa and is a major cause of chronic gastritis, peptic ulcers, and gastric malignancies. Globally, it infects approximately 40% of the population, with higher prevalence in developing countries due to socioeconomic and hygiene-related factors [1]. Beyond its role in gastroduodenal diseases, H. pylori has been linked to micronutrient deficiencies, particularly vitamin B12 (cobalamin), due to its disruption of gastric acid secretion and intrinsic factor production [2,3].

Studies suggest that H. pylori infection can lead to atrophic gastritis, reducing the stomach's ability to absorb vitamin B12, which is essential for DNA synthesis, neurological function, and hematopoiesis [4]. Additionally, certain virulent strains, such as those carrying the cytotoxin-associated gene A (CagA), may exacerbate malabsorption by inducing more severe gastric inflammation [5]. Despite these associations, the prevalence of H. pylori-related B12 deficiency varies across populations, with some studies reporting a strong correlation [6,7], while others find no significant link [8].

Dyspepsia is a common clinical presentation, often requiring endoscopic evaluation to identify underlying causes such as H. pylori infection. However, the extent to which H. pylori contributes to vitamin B12 deficiency in dyspeptic patients remains unclear. Conflicting data exist, some studies report high rates of B12 deficiency in H. pylori-positive individuals [9], while others suggest minimal

impact [7]. In regions like Pakistan, where H. pylori infection and nutritional deficiencies are widespread, understanding this relationship is crucial for clinical management.

Furthermore, socioeconomic factors such as income, dietary habits, and education may influence B12 levels, yet their interaction with H. pylori infection has not been thoroughly examined. Without definitive evidence, clinicians may overlook B12 deficiency in dyspeptic patients, leading to untreated neurological or hematological complications.

This study aims to clarify the association between H. pylori infection and vitamin B12 deficiency in dyspeptic patients by comparing deficiency rates between H. pylori-positive and H. pylori-negative individuals. Given that H. pylori strains and host responses vary geographically, findings from this Pakistani cohort will provide region-specific insights that may differ from Western studies [9].

Additionally, the study evaluates the role of demographic factors such as age, gender, and socioeconomic status in B12 deficiency, offering a comprehensive risk assessment. Since untreated B12 deficiency can lead to irreversible neurological damage and anemia, identifying high-risk groups could inform screening and supplementation guidelines [8].

Ultimately, this research seeks to improve patient care by determining whether H. pylori eradication and B12 assessment should be integrated into dyspepsia management, particularly in resource-limited settings. The findings may also contribute to global discussions on H. pylori's extra-gastric effects, reinforcing the need for a multidisciplinary approach to diagnosis and treatment [5].

Literature Review

The complex relationship between Helicobacter pylori infection and vitamin B12 deficiency continues to generate significant research interest, with recent studies providing deeper insights into this multifaceted interaction. The current understanding of this association has evolved considerably, particularly regarding the mechanisms by which this ubiquitous gastric pathogen may contribute to micronutrient deficiencies [9]. Emerging evidence suggests that H. pylori's impact extends beyond its well-established role in gastroduodenal diseases, potentially influencing systemic nutritional status through both direct and indirect pathways [14].

Recent investigations have reinforced the concept that H. pylori infection may initiate a cascade of gastric mucosal changes that ultimately impair vitamin B12 absorption [4]. The bacterium's ability to induce chronic inflammation and atrophic gastritis appears central to this process, with studies demonstrating that these pathological changes can significantly reduce gastric acid secretion and intrinsic factor production [7]. Particularly compelling are findings showing that the density of bacterial colonization correlates with the degree of vitamin B12 depletion, suggesting a dose-dependent relationship between infection severity and nutritional impact [5]. This observation aligns with clinical data indicating that patients with more extensive gastric involvement tend to exhibit more pronounced deficiencies [6].

The virulence characteristics of H. pylori strains have emerged as another critical factor in this equation. Contemporary research has particularly focused on the cytotoxin-associated gene A (CagA) protein and its potential role in exacerbating nutrient malabsorption [6]. Studies comparing CagA-positive and CagA-negative infections have revealed intriguing differences, with the former associated with more severe mucosal damage and consequently greater impairment of vitamin B12 absorption [15]. These findings have important clinical implications, as they suggest that strain-specific factors may help identify patients at highest risk for developing nutritional deficiencies [9].

The global epidemiology of H. pylori infection adds another layer of complexity to this discussion. Recent meta-analyses have confirmed substantial geographic variation in both infection prevalence and associated complications [11]. In regions with particularly high infection rates, the public health impact of H. pylori-related nutritional deficiencies may be substantial, especially when combined with other risk factors such as limited dietary diversity or compromised socioeconomic conditions [12]. These population-level differences underscore the importance of

context when interpreting the clinical significance of H. pylori-associated vitamin B12 deficiency [10].

Diagnostic advancements have also influenced our understanding of this relationship [16]. Novel detection methods, including sophisticated spectroscopic techniques, have improved our ability to identify and characterize H. pylori infections with greater precision [19]. These technological developments have facilitated more accurate assessment of infection status and bacterial load, enabling researchers to explore more nuanced questions about the infection-nutrition connection [18]. Such methodological improvements are particularly valuable given the challenges of studying this relationship in clinical populations where multiple confounding factors may be present [20].

The therapeutic implications of these findings remain an area of active investigation [8]. While some studies suggest that eradication therapy may lead to improvement in vitamin B12 status, particularly in patients without advanced gastric atrophy, the evidence remains somewhat inconsistent [13]. This variability in treatment response highlights the need for personalized approaches to management, taking into account factors such as infection duration, strain characteristics, and individual host factors [9]. Recent clinical guidelines have begun to incorporate these considerations, though consensus on optimal screening and treatment protocols has yet to emerge [14].

Nutritional science perspectives have contributed additional insights into this complex relationship [4]. Researchers have examined how H. pylori infection might interact with dietary patterns to influence vitamin B12 status, with some studies suggesting that the infection's nutritional impact may be most pronounced in individuals with marginal dietary intake [7]. These findings emphasize the importance of considering both infectious and nutritional factors when evaluating patients with unexplained vitamin B12 deficiency [5].

The immunological aspects of H. pylori infection have also received renewed attention in recent literature [15]. Growing evidence supports the concept that the host immune response to H. pylori may contribute to the development of autoimmune phenomena that further compromise gastric function [9]. This autoimmune component could help explain why some patients develop persistent vitamin B12 deficiency even after successful eradication of the infection, particularly in cases where the infection has triggered irreversible damage to parietal cells [14].

Longitudinal studies have provided valuable data on the natural history of H. pylori-associated gastric changes and their nutritional consequences [11]. These investigations have helped clarify the temporal relationship between infection, gastric pathology, and nutrient deficiencies, supporting the concept that early intervention may be crucial for preventing long-term complications [12]. Such findings have important implications for clinical practice, suggesting that screening for nutritional deficiencies should be considered in patients with chronic H. pylori infection, particularly those with symptoms or risk factors for malabsorption [8].

Recent technological innovations in microbiological assessment have opened new avenues for understanding the mechanisms underlying H. pylori-associated nutrient deficiencies [16]. Advanced genomic and proteomic approaches have provided unprecedented detail about bacterial factors that may directly or indirectly influence nutrient absorption [17]. These molecular insights are helping to bridge the gap between clinical observations and biological mechanisms, offering potential targets for future therapeutic interventions [18].

The public health dimensions of this issue have also come into sharper focus [10]. As researchers have documented the substantial global burden of H. pylori infection, attention has turned to the potential population-level consequences of widespread micronutrient deficiencies associated with this infection [11]. This broader perspective has important implications for health policy, particularly in regions where H. pylori prevalence is high and nutritional resources may be limited [12].

Emerging evidence suggests that the relationship between H. pylori and vitamin B12 status may be influenced by a complex interplay of host genetic factors, environmental exposures, and microbial characteristics [6]. This multifactorial nature helps explain why clinical manifestations can vary so

significantly among infected individuals, and why some patients develop severe deficiencies while others remain unaffected despite long-term infection [7].

The clinical management of H. pylori-associated vitamin B12 deficiency continues to evolve as new evidence accumulates [8]. Current approaches increasingly recognize the importance of comprehensive assessment, including evaluation of gastric function, nutritional status, and potential alternative causes of deficiency [9]. This holistic perspective represents an important advance over earlier, more simplistic conceptualizations of the relationship between infection and nutrition [14].

Future research directions are likely to focus on several key areas, including the development of more precise biomarkers for identifying high-risk patients, optimization of treatment strategies to address both infection and nutritional consequences, and improved understanding of the long-term outcomes of H. pylori-associated nutrient deficiencies [15]. As this field continues to advance, it promises to yield important insights not only about H. pylori specifically, but more broadly about the intricate relationships between chronic infections and systemic nutritional status [19].

Methodology

The methodology employed in this study followed a structured approach to investigate the relationship between Helicobacter pylori infection and vitamin B12 deficiency among dyspeptic patients. A cross-sectional study design was implemented over a six-month period at the Medical Outpatient Department of Civil Hospital Karachi, allowing for the simultaneous assessment of infection status and nutritional markers. Recent advances in diagnostic technologies, including Raman spectroscopy and Fourier transform infrared spectroscopy, have demonstrated improved accuracy in H. pylori detection [18,21], though our study utilized conventional methods to align with standard clinical practice in our setting. The study population consisted of 273 carefully selected adult patients aged 18 to 50 years who presented with persistent epigastric pain or burning sensations lasting more than three months.

Rigorous inclusion and exclusion criteria were applied to ensure a homogeneous sample while minimizing confounding factors. Patients with conditions that could independently affect vitamin B12 levels, such as chronic liver disease, kidney impairment, or those taking medications known to influence absorption, were systematically excluded from participation. Emerging techniques like single-cell identification and whole-genome sequencing [18] offer promising alternatives for future research but were beyond the scope of this clinical study.

Data collection involved a comprehensive multi-step process beginning with detailed patient interviews using a standardized proforma to document demographic characteristics, symptom duration, socioeconomic status, and medication history. Each participant underwent upper gastrointestinal endoscopy performed by experienced physicians with at least five years of endoscopic practice, ensuring procedural consistency. While novel spectroscopic methods for gastric cancer detection have shown excellent sensitivity [19,22], we maintained traditional endoscopic approaches supplemented by histopathological confirmation.

During endoscopy, gastric biopsy specimens were obtained and processed using Giemsa staining, with interpretation conducted by qualified histopathologists to confirm H. pylori presence based on characteristic morphological features. This approach was selected over newer urea breath test technologies [20] due to its direct visualization benefits and established reliability in our clinical laboratory. Concurrently, venous blood samples were collected from all participants for serum vitamin B12 analysis, with deficiency defined as levels below 150 ng/ml to maintain diagnostic consistency.

The analytical approach incorporated both descriptive and inferential statistical methods to examine patterns and relationships within the dataset. Continuous variables like age and symptom duration were expressed as means with standard deviations, while categorical variables including infection status and deficiency rates were presented as frequencies and percentages. Comparative analyses between H. pylori-positive and negative groups employed chi-square testing to assess statistical significance, with stratification by key

demographic variables to control for potential confounding effects. All data processing and statistical computations were performed using SPSS version 20.0, with a predetermined significance threshold of $p<0.05$ for interpreting results. This methodological framework was designed to provide reliable insights while maintaining scientific rigor throughout the investigative process.

Data Analysis

The study analyzed data from 273 dyspeptic patients to assess the relationship between H. pylori infection and vitamin B12 deficiency. The results revealed several key patterns that help understand this association.

Demographic and Clinical Characteristics

The mean age of participants was 46.76 ± 8 years, with nearly equal gender distribution (51.3% male, 48.7% female). Most patients (57.9%) had a monthly income below 25,000 PKR, and 45.8% were illiterate. A high proportion reported NSAID use (76.2%) and smoking (71.4%), both known risk factors for gastric irritation.

Prevalence of H. pylori and B12 Deficiency

The study found that 61.17% of participants tested positive for H. pylori infection. Among these, vitamin B12 deficiency was significantly more common (70.6%) compared to H. pylori-negative patients (29.5%).

Table 1: Vitamin B12 Deficiency by H. pylori Status

H. pylori Status	Vitamin B12 Deficient (%)	Not Deficient (%)	Total
Positive (n=167)	70.6	58.0	167
Negative (n=106)	29.5	42.0	106

This table clearly shows that H. pylori-positive patients had more than double the prevalence of B12 deficiency compared to uninfected individuals.

Stratified Analysis

Table 2: Vitamin B12 Deficiency by Age Group

Age Group	Deficient (%)	Not Deficient (%)	Total
<40 years	27.9	72.1	136
≥40 years	21.9	78.1	137

While younger patients showed slightly higher deficiency rates, the difference was not statistically significant ($p=0.155$).

Table 3: Vitamin B12 Deficiency by Income Level

Monthly Income (PKR)	Deficient (%)	Not Deficient (%)	Total
<25,000	25.9	74.1	158
25,000-50,000	30.0	70.0	80
>50,000	3.6	91.4	35

This table reveals a striking socioeconomic gradient, with the highest deficiency rates among middle-income earners (30%) and the lowest in the highest income bracket (3.6%). The difference was statistically significant ($p=0.045$).

Key Findings and Interpretation

1. **Strong H. pylori Association**

The 70.6% deficiency rate in H. pylori-positive patients suggests the infection substantially impairs B12 absorption, likely through chronic gastritis and reduced intrinsic factor production.

2. **Economic Factors Play a Role**

The much lower deficiency rate (3.6%) in high-income patients (>50,000 PKR/month) implies better nutrition (meat/dairy intake) may protect against deficiency despite infection.

3. **Age and Gender Differences Were Minor**

Neither age nor gender showed significant effects on deficiency rates, suggesting H. pylori's impact transcends these demographic factors.

4. **Clinical Implications**

These results support screening for B12 deficiency in H. pylori-positive dyspeptic patients, particularly those with lower socioeconomic status. The findings also highlight the need to address dietary factors alongside infection treatment.

The tables and analysis demonstrate that while H. pylori infection is a major risk factor for B12 deficiency, economic status modifies this relationship significantly. This underscores the importance of considering both biological and socioeconomic factors in managing dyspeptic patients. Future research should investigate whether B12 levels improve after H. pylori eradication in this population.

Discussion

The findings of this study align closely with emerging research on H. pylori's role in micronutrient deficiencies. The significant association between H. pylori infection and vitamin B12 deficiency (70.6% in positive cases vs. 29.5% in negative cases) supports previous work demonstrating the bacterium's ability to impair gastric function and intrinsic factor production [6,9]. These results are particularly consistent with studies showing that H. pylori-induced chronic gastritis can lead to malabsorption of protein-bound B12, even before overt atrophic changes occur [4,7].

The socioeconomic patterns observed, where higher income correlated with lower deficiency rates (3.6% in top earners vs. 30% in middle-income groups), echo global research on nutrition-inflammation interactions [12,14]. This suggests that while H. pylori infection establishes the pathological framework for deficiency, dietary factors (particularly animal protein intake) may determine whether biochemical deficiency manifests clinically. Interestingly, the lack of significant age or gender differences contrasts with some Western studies [8], but aligns with recent findings in similar low-middle income populations where infection prevalence dominates demographic variables [11].

The high deficiency prevalence among H. pylori-positive patients (70.6%) exceeds rates reported in some studies [5,7], possibly reflecting our cohort's unique characteristics: high NSAID use (76.2%) and smoking rates (71.4%) likely compounded gastric damage. This synergism between infection and lifestyle factors has been noted in recent pathogenesis models [9,15].

These results strengthen calls for integrated management of dyspepsia in endemic regions, where treating H. pylori alone may be insufficient without addressing nutritional status [13,14]. The findings particularly support recent guidelines emphasizing dual assessment of infection and micronutrients in persistent dyspepsia [8,9]. Future studies should explore whether the observed socioeconomic gradient reflects dietary differences, healthcare access, or distinct H. pylori strains, an area where advanced molecular techniques [18,19] could provide valuable insights.

Conclusion

This study confirms a strong association between H. pylori infection and vitamin B12 deficiency in dyspeptic patients, particularly among lower-income groups where dietary factors may exacerbate the risk. Given that 70.6% of H. pylori-positive patients exhibited deficiency, compared to just 29.5% of uninfected individuals, routine B12 screening should be integrated into the management of H. pylori-positive dyspepsia, especially in endemic regions. Eradication therapy alone may not suffice; clinicians should consider concurrent B12 supplementation in

high-risk patients, alongside dietary counseling. Future research should explore whether B12 levels normalize post-eradication and whether strain-specific virulence factors (e.g., CagA) further modulate deficiency risk, potentially guiding more personalized treatment approaches.

For medical practitioners, these findings underscore the need to view H. pylori infection not just as a gastric concern but as a potential driver of systemic micronutrient deficiencies. Gastroenterologists and primary care physicians should adopt a dual approach, testing for B12 deficiency in H. pylori-positive patients, particularly those with lower socioeconomic status or prolonged dyspepsia. For pharmaceutical stakeholders, this presents an opportunity to develop combination therapies targeting both infection and deficiency, especially in regions where H. pylori prevalence exceeds 60%. Point-of-care B12 testing kits could also enhance early detection in resource-limited settings, preventing irreversible neurological complications.

Future studies should prioritize longitudinal cohorts to assess B12 trajectory post-eradication, ideally comparing different therapeutic regimens (e.g., antibiotics with vs. without B12 supplementation). Advanced diagnostics, such as Raman spectroscopy for H. pylori strain typing, could clarify whether certain variants disproportionately impair absorption. Additionally, socioeconomic determinants merit deeper exploration: do dietary interventions (e.g., fortified foods) mitigate deficiency risk even with persistent infection? Multinational collaborations could standardize protocols, ensuring findings are generalizable across diverse populations with varying H. pylori strains and nutritional baselines. Such research would optimize clinical guidelines, moving beyond infection control to holistic patient recovery.

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