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Frequency of Vitamin B12 Deficiency in H Pylori Positive Patients

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ABSTRACT

Introduction: According to epidemiological data, patients who test positive for H pylori have a vitamin B12 deficit, which raises their morbidity. Geographical location affects the frequency of vitamin B12 insufficiency in patients with H pylori. and after looking through the literature, I discovered a lot of research on this topic, but since there aren't many studies in our area, I've chosen to assess the prevalence of vitamin B12 insufficiency in patients with H pylori infection in the community. **Study design:** Descriptive, cross sectional study. **Settings:** Department of Medicine, PAEC General Hospital, Islamabad. **Study duration:** March 2025 to May 2025. **Materials & Methods:** Regardless of gender, 100 patients aged 25 to 65 who reported having a history of upper stomach pain and bitter taste, heartburn, nausea, belching, or nausea at least three times per week and who tested positive for H pylori antigen in their stool were included. Participants were excluded if they had been on steroids for the previous month, already taking vitamin B12 supplements, had a history of H pylori eradication therapy, chronic renal failure, had undergone a gastricectomy, hepatic disease, hemolytic anemia, cancer, aplastic anemia, multiple myeloma, leukemia, or chronic lung disease, were using immunosuppressive or chemotherapeutic drugs, alcoholics. Each patient next had a 5 ml blood sample drawn, which was forwarded to the institution's pathology lab to determine whether or not they had a vitamin B12 deficiency (serum vitamin B12 levels <200 pg/dl). **Results:** The study's participants ranged in age from 25 to 65, with a mean age of 53.53 ± 12.41 years. With a male to female ratio of 1.4:1, 58 (58.0%) of the 100 patients were men and 42 (42.0%) were women. In our study, the average duration of symptoms was 23.11 ± 5.20 weeks. A mean BMI of 27.61 ± 3.04 kg/m2 was recorded. Of the patients in our study who tested positive for H pylori, 40 (40.0%) had vitamin B12 insufficiency. **Conclusion:** Our research supports the link between H. pylori infection and vitamin B12 deficiency, indicating that H. pylori may be a major risk factor for this dietary shortfall.

INTRODUCTION

Peptic Ulcer Disease (PUD) is a serious medical condition caused by an imbalance between the mucosal defenses that prevent acid digestion and the release of acid. The stomach, duodenum, oesophagus, or jejunum can all develop ulcers, either in Zollinger-Ellison syndrome or at the margin of a gastroenterostomy. Peptic ulcer disease is quite common, causes excruciating agony, and is costly to treat, despite the low death rates.[1] A gram-negative, spirally formed microaerophilic bacteria, Helicobacter pylori (H. pylori) only inhabits the stomach and duodenal mucosa. It has developed a special adaptation to survive in the harsh environment of the stomach.[2] It has been acknowledged that H. pylori infection is a global public health issue that is more common in underdeveloped nations than in industrialized ones. Rather than ethnic issues, socioeconomic factors were probably involved.[3]

Numerous micronutrient deficits have a substantial correlation with H. pylori infection. There is a strong positive correlation between H. pylori and the vitamin B12 deficiency.[4] Even in those without gastritis or other digestive issues, researchers have discovered that many patients with pernicious anemia due to poorly managed B12 vitamin insufficiency contain H. pylori bacteria.[5] There is a substantial correlation between chronic gastritis of the abdominal antrum and H. pylori infection. This results in decreased stomach acid and pepsin secretion, which is linked to vitamin B12 malabsorption. Vitamin B12 insufficiency is caused by the H. pylori infection.[6] A study found that 36.0% of patients with H pylori were vitamin B12 deficient.[7] 50.0% of H pylori-positive patients had vitamin B12 insufficiency, according

to another study.[8]

According to epidemiological data, patients who test positive for H pylori have a vitamin B12 deficit, which raises their morbidity. Geographical location affects the frequency of vitamin B12 insufficiency in patients with H pylori.[7,8] and after looking through the literature, I discovered a lot of research on this topic, but since there aren't many studies in our area, I've chosen to assess the prevalence of vitamin B12 insufficiency in patients with H pylori infection in the community. Such a study is necessary locally since our population differs from the global population in terms of social, economic, and food practices, and we anticipate different outcomes from the local population. In addition to determining the problem's local scope, this study will contribute to the body of existing literature. Additionally, my study's findings will assist clinicians in creating a procedure for diagnosing and treating vitamin B12 insufficiency in these specific patients, improving outcomes and lowering our population's morbidity.

MATERIAL AND METHODS

The Department of Medicine of PAEC General Hospital in Islamabad conducted this descriptive cross-sectional study between March and May of 2025. After being approved by the institutional ethical review committee, 100 patients who satisfied the inclusion criteria were selected via non-probability sequential sampling. The informed consent of each patient will be sought. Using a 95% confidence level, 10% margin of error, and a 50.0% rate of vitamin B12 insufficiency in H pylori-positive individuals, the WHO calculator determines a sample size of 100. 8. Regardless of gender, all patients aged 25 to 65 who reported having a history of upper stomach pain and bitter taste, heartburn, nausea, belching, or nausea at least three times per week and who tested positive for H pylori antigen in their stool were included. Participants were excluded if they had been on steroids for the previous month, were already taking vitamin B12 supplements, were pregnant, had a history of H pylori eradication therapy, had chronic renal failure, had undergone a gastricectomy, were anemic with a primary disease, such as hepatic disease, hemolytic anemia, cancer, aplastic anemia, multiple myeloma, leukemia, or chronic lung disease, were using immunosuppressive or chemotherapeutic drugs, were alcoholics, had undergone stomach resection or small bowl surgery, were vegetarians, or had folic acid deficiency.

Each patient next had a 5 ml blood sample drawn, which was forwarded to the institution's pathology lab to determine whether or not they had a vitamin B12 deficiency (serum vitamin B12 levels <200 pg/dl). Every piece of information, including demographic data, was entered into the pre-made proforma.

SPSS version 25.0 was used to enter and analyze the data. The mean ± SD of the patient's age, height, weight, BMI, length of symptoms, and vitamin B12 levels were displayed. The following factors were reported as frequency and percentage: gender, place of residence (rural/urban), smoking status (yes/no), lifestyle (simple/sedentary), occupation (domestic/office/field work), monthly income (<25000/25000-50000/>50000), history of regular NSAID use (yes/no), and vitamin B12 deficiency (present/absent). A chi square/fisher exact test was used after stratification to control effect modifiers such as age, gender, duration of symptoms, BMI, place of residence (rural/urban), smoking status (yes/no), lifestyle (simple/sedentary), occupation (field work/office work/domestic), monthly income (<25000/25000-50000/>50000), and history of regular NSAID use (yes/no). A p-value ≤ 0.05 was deemed significant.

RESULTS

The study's participants ranged in age from 25 to 65, with a mean age of 53.53 ± 12.41 years. According to Table I, the majority of the 56 patients (56.0%) were between the ages of 51 and 70. With a male to female ratio of 1.4:1, 58 (58.0%) of the 100 patients were men and 42 (42.0%) were women. In our study, the average duration of symptoms was 23.11 ± 5.20 weeks. A mean BMI of 27.61 ± 3.04 kg/m2 was recorded. Table 1 displays the patient distribution based on confounding variables.

Of the patients in our study who tested positive for H pylori, 40 (40.0%) had vitamin B12 insufficiency (Fig. 1).

Table 2 displays the stratification of vitamin B12 deficiency by age, gender, length of symptoms, BMI, place of residence, smoking, lifestyle, occupation, monthly income, and history of consistently using NSAIDs.

Table 1

Distribution of Different Variables (N=100)

Confounding variables		Frequency	%age
Age (years)	25-45	44	44.0
	46-65	56	56.0
Gender	Male	58	58.0
	Female	42	42.0
Duration (weeks)	≤24	60	60.0
	25-48	40	40.0
BMI (kg/m²)	≤30	77	77.0
	>30	23	23.0
H/o NSAIDs use	Yes	48	48.0
	No	52	52.0
Lifestyle	Sedentary	64	64.0
	Simple	36	36.0
Smoking	Yes	28	28.0
	No	72	72.0
Occupation	Office	35	35.0
	Field	29	29.0
	Others	36	36.0
Place of living	Rural	38	38.0
	Urban	62	62.0
Monthly income	<25000	23	23.0
	25000-50000	37	37.0
	>50000	40	40.0

Figure 1

Frequency of Vitamin B12 Deficiency in H Pylori Positive Patients (n=100).

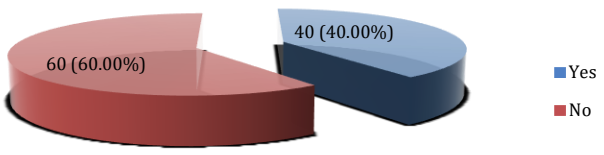

Table 3

Stratification of Vitamin B12 Deficiency with respect to Age, Gender, Duration of Symptoms, BMI, Place of Living, Smoking, Lifestyle, Occupation, Monthly Income and History of Regular Nsaids Use.

Variables		Yes (n=40)	No (n=60)	P-value
Age (years)	25-45	15 (34.09%)	29 (65.91%)	0.285
	46-65	25 (44.64%)	31 (55.36%)	
Gender	Male	22 (37.93%)	36 (62.07%)	0.619
	Female	18 (42.86%)	24 (57.14%)	
Duration (weeks)	≤24	20 (33.33%)	40 (66.67%)	0.096
	25-48	20 (50.0%)	20 (50.0%)	
BMI (kg/m^2)	≤30	33 (42.86%)	44 (57.14%)	0.286
	>30	07 (30.43%)	16 (69.57%)	
H/o NSAIDs use	Yes	20 (41.67%)	28 (58.33%)	0.744
	No	20 (38.46%)	32 (51.54%)	
Lifestyle	Sedentary	25 (39.06%)	39 (60.94%)	0.799
	Simple	15 (41.67%)	21 (58.33%)	
Smoking	Yes	09 (32.14%)	19 (67.86%)	0.317
	No	31 (68.89%)	41 (31.11%)	
Occupation	Office	13 (37.14%)	22 (62.86%)	0.791
	Field	11 (37.93%)	18 (62.07%)	
	Others	16 (44.44%)	20 (55.56%)	
Place of living	Rural	17 (44.74%)	21 (55.26%)	0.449
	Urban	23 (37.70%)	39 (62.30%)	
Monthly income	<25000	09 (39.13%)	14 (60.87%)	0.616
	25000-50000	17 (45.95%)	20 (54.05%)	
	>50000	14 (35.0%)	26 (65.0%)	

DISCUSSION

H. pylori is a major contributor to vitamin B12 insufficiency, especially in less developed countries, according to recent studies.[9] By inhibiting the growth of other bacteria in the gastrointestinal tract, H. pylori may improve the absorption of cobalamin. On the other hand, other bacterial communities may overproliferate in the absence of H. pylori, which would further hinder the absorption of cobalamin. Therefore, regardless of H. pylori status, broad-spectrum antibiotic therapy may enhance cobalamin absorption by lowering the anaerobic bacterial burden.[10]

The prevalence of vitamin B12 deficiency was 40.0% in our study, which comprised 100 patients assessed for both conditions. Males had a higher incidence than females, and the participants' mean age was 53.53 ± 12.41 years. Although only 40.0% of the patients were in a higher socioeconomic group, literacy and a higher socioeconomic level seemed to be associated with decreased infection rates.[11,12] Our findings are corroborated by comparative research from Turkey and Iraq, which indicates that patients with H. pylori positive test results had greater rates of vitamin B12 deficiency.[10,11] These findings highlight H. pylori's potential as a prognostic indicator for vitamin B12 insufficiency.

The observed socioeconomic trends, which showed a correlation between lower insufficiency rates and higher income (3.6% among top earners versus 30% among middle-income groups), are consistent with international research on the relationship between inflammation and nutrition.[13,14] This implies that dietary factors, including animal protein consumption, may influence whether biochemical insufficiency presents clinically, even while H. pylori infection creates the pathological foundation for deficiency. Intriguingly, the absence of notable age or gender disparities is in contrast to certain Western research[15], but it is consistent with new findings in comparable low-middle-income groups where infection prevalence predominates among demographic factors.[16]

With high NSAID use (76.2%) and smoking rates (71.4%) likely exacerbating stomach injury, the high deficiency prevalence among H. pylori-positive individuals (70.6%) surpasses rates reported in several research.[17,18] Recent pathogenesis models have observed this synergy between lifestyle variables and infection.[19,20] In endemic areas, where treating H. pylori alone would not be enough without addressing nutritional status, these findings support demands for combined care of dyspepsia.[14,21]

Researchers have looked at how H. pylori causes vitamin B12 deficiency and found that it may cause stomach autoimmune and make autoantibodies against intrinsic factor or parietal cells. These autoantibodies, or H. pylori itself, stop vitamin B12 from being absorbed by either stopping the creation of IF, stopping IF from working, or stopping vitamin B12 from binding to IF, or by changing the R proteins that IF attaches to in the stomach.[22] H. pylori infection may also affect vitamin B12 absorption by stopping food from releasing vitamin B12, stopping cobalophilins from binding to vitamin B12 and its analogues in the stomach, making it harder for pancreatic enzymes to digest cobalophilins in the gastrointestinal tract, making it harder for cobalomins (other than analogues) to bind to IF, and making H. pylori consume vitamin B12 that is already in the stomach.[23]

According to a study by Carmel et al., 78% of people with severe cobalamin malabsorption, 45% of people with mild cobalamin malabsorption, and 42% of people with normal absorption had H. pylori.[24] About half of patients with low serum cobalamin levels that could not be attributed to conventional causes had food-cobalamin malabsorption, according to a different study by Carnel et al.[25] According to a study by Negrini et al[26], H. pylori may have a mechanism for food-cobalamin malabsorption by exhibiting antigenic cross-reaction with parietal cells as well as the antral mucosa in certain conditions. It is advised that H. pylori be looked into and eliminated in cases of unexplained iron deficiency anemia, ITP, and vitamin B12 deficiency. These conditions were listed as extragastrointestinal manifestations of H. pylori in the 4th Maastricht consensus report, which was created by the EHPSG in 2012.[27]

The results especially corroborate new recommendations that stress the importance of evaluating infection and micronutrients simultaneously in patients with chronic dyspepsia.[28] Future research should investigate whether the observed socioeconomic gradient is due to different H. pylori strains, dietary variations, or access to healthcare; this is an area where sophisticated molecular tools could offer important insights.[29]

Our study does have several limitations, though. The results may not be as broadly applicable as they could be due to the single-center design and sample size. To validate these results and evaluate the impact of eradication therapy on vitamin B12 levels, future research should concentrate on multi-center studies with bigger, more diverse populations.

CONCLUSION

To sum up, our research supports the link between H.

pylori infection and vitamin B12 deficiency, indicating that H. pylori may be a major risk factor for this dietary shortfall. This correlation emphasizes how crucial it is to take H. pylori elimination into account for individuals who have been diagnosed with vitamin B12 insufficiency. Investigating the fundamental processes via which H. pylori affects cobalamin absorption is also essential in order to improve treatment and preventative tactics.

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