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Vitamin B12 Deficiency in *Helicobacter pylori* Infected Patients

Gufran Kadhim, Mashitah Shikh, Hishamuddin Omar, Ahmad Ismail*

Department of Biology, Faculty of Science, University Putra Malaysia, Serdang, Selangor, Malaysia
Email: *aismail@upm.edu.my

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Abstract

Background: *H. pylori* infection is strongly related with chronic gastritis of the antrum of the stomach, which causes impairment in gastric acid and pepsin secretion, and is thus linked to malabsorption of food-vitamin B12. *H. pylori* can cause an individual to have a vitamin B12 deficiency. It is also a known contributor to gastritis ulcers and it can prevent the stomach from being able to absorb the vitamin B12 you consume and leads to a deficiency of vitamin B12. The purpose of this study is to determine the frequency of vitamin B12 deficiency in *Helicobacter pylori* infected patients. **Methods:** All patients aged 17 - 65 years, of either gender were evaluated for urine vitamin B12 level by human vitamin B12 ElSA kit. The data were analyzed in statistical software (SPSS) and the P-value = 0.05 was considered as statistically significant. **Results:** Of one hundred subjects, 65 (65%) were males, while 35 (35%) were females. 31 (36.0%) *H. pylori* infected patients had normal vitamin B12 level and 55 (64%) were Vitamin B12 deficiency in 86 (86%). *H. pylori* infected patients with a significant difference (P value < 0.05). **Conclusions:** Vitamin B12 deficiency is more prevalent in *Helicobacter pylori* infected patients.

Subject Areas

Public Health

Keywords

H. pylori, Vitamin B12, Pernicious Anemia

1. Introduction

Peptic Ulcer Disease (PUD) is a consequence of the imbalance of acid secretion and mucosal defences which resist acid digestion thereby indicating a serious medical problem. Ulcers can manifest in the stomach or duodenum, in the oe-

sophagus or the jejunum and can develop at the margin of a gastroenterostomy or in Zollinger-Ellison syndrome. While the mortality rates are low, peptic ulcer disease is highly prevalent, causing painful suffering and is expensive to treat [1]. Much research has pointed out that *H. pylori* and the deficiency of vitamin B12 are connected and they demonstrate a strong causative correlation. Even in case of those without gastritis or other gastrointestinal issues, scientists have found the *H. pylori* bacteria in over 50% of patients suffering from pernicious anaemia, primarily because of untreated deficiency of vitamin B12 [2]. While the drug such as lansoprazole and omeprazole is useful for PUD, its use does not come without the increased risk of having other complications. Among these are clinically relevant drug-drug interactions; an increased aspiration pneumonia risk among certain patients; and complications related to Vitamin B12 deficiency (e.g. hyperhomocysteinemia, macrocytic anaemia, and/or neuropathies) [3].

2. Methods

2.1. Patients

One hundred randomly selected eligible participants with suggested symptoms of peptic ulcer disease in Al SadarTeching Hospital in Basrah, Iraq. About 0.2 g of stool was collected into a sterile container. All stool samples were frozen at −20˚C until tested for *H. pylori* antigen by HpSAg kit (DRG-Germany). Methods of analysis followed the manufacturer instruction. Patients were further evaluated for urine vitamin B12 level. The first urine of the day (mid-stream) was collected directly into a sterile container. Urine samples were centrifuged to remove particulate matter, frozen at <−20˚C until tested for vitamin B12 by human vitamin B12 ELISA kit.

2.2. Statistical Analysis

The collected data was analyzed in SPSS version (23). The frequency and percentage of gender and vitamin B12 deficiency in patients was calculated. Chi-square was applied to determine the statistical difference in gender and the p-value = 0.05 was considered as statistically significant.

3. Results

One hundred subjects, 65 (65%) were males, while 35 (35%) were females. **Table 1** shows the overall mean age ± SD of individuals, and the mean age ± SD of male and female individuals.

Table 2 shows 31 (36.0%) *H. pylori* infected patients had normal vitamin B12 level and 55 (64%) was Vitamin B12 deficiency in 86 (86%) *H. pylori* infected patients with a significant difference (P value < 0.05).

4. Discussion

Helicobacter pylori have been determined as an etiologic factor in vitamin B 12 deficiency [4]. In populations with a high prevalence of *H. pylori* infection, the

Table 1. Mean age of patients in relation to gender.

Sex	Mean age	N	Std. Deviation
Male	2.63	65	1.069
Female	2.74	35	1.120
Total	2.67	100	1.083

Table 2. Frequency of vitamin B12 deficiency in *H. pylori* infected patients.

			Vitamin B12		Total
			Normal	Deficiency	
H. pylori	Negative	Count	11	3	14
		% within *H. pylori*	78.6%	21.4%	100.0%
	Positive	Count	31	55	86
		% within *H. pylori*	36.0%	64.0%	100.0%
Total		Count	42	58	100
		% within *H. pylori*	42.0%	58.0%	100.0%

P value < 0.05.

frequency of vitamin B12 deficiency and its clinical consequences can be expected to be high. In this study, the high frequency (64%) of vitamin B12 deficiency was found in patients with *Helicobacter pylori* infection leads to immediate development of persistent gastritis, colonization of the stomach by *H. pylori* is almost always accompanied by clinical and histological signs of chronic gastritis associated with both local and systemic immune response. The resolution of gastritis, mucosal immune response to *H. pylori* and normal appearance of gastric epithelium is demonstrated following eradication of the infection with antibiotic therapy [4]. A study by Carmel *et al.*, who investigated the association between *H. pylori* infection and megaloblastic anemia, by examining patients with food-cobalamin malabsorption and the investigators found that patients with low levels of serum cobalamin had a higher seroprevalence of *H. pylori* infection [5]. Infection with *H. pylori* causes a series of changes in gastric function prior to the final stage of gastric atrophy. The initial infection causes transient hypochlorhydria and usually few or no clinical symptoms [6]. In some persons, the infection causes gastric inflammation with elevated serum gastrin and pepsinogen and reduced somatostatin concentrations [7]. The elevated gastrin and reduced somatostatin then cause elevated acid secretion from gastric parietal cells [6], but over time (usually many years) some but not all infected persons develop peptic ulcer disease, gastric cancer, and eventually atrophic gastritis with low gastric acid secretion, and possibly inadequate production of intrinsic factor.

5. Conclusion

Helicobacter pylori have been determined as an etiologic factor in vitamin B12

deficiency. Therefore, vitamin B12 deficiency is more pronounced in *Helicobacter pylori* infected patient.

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