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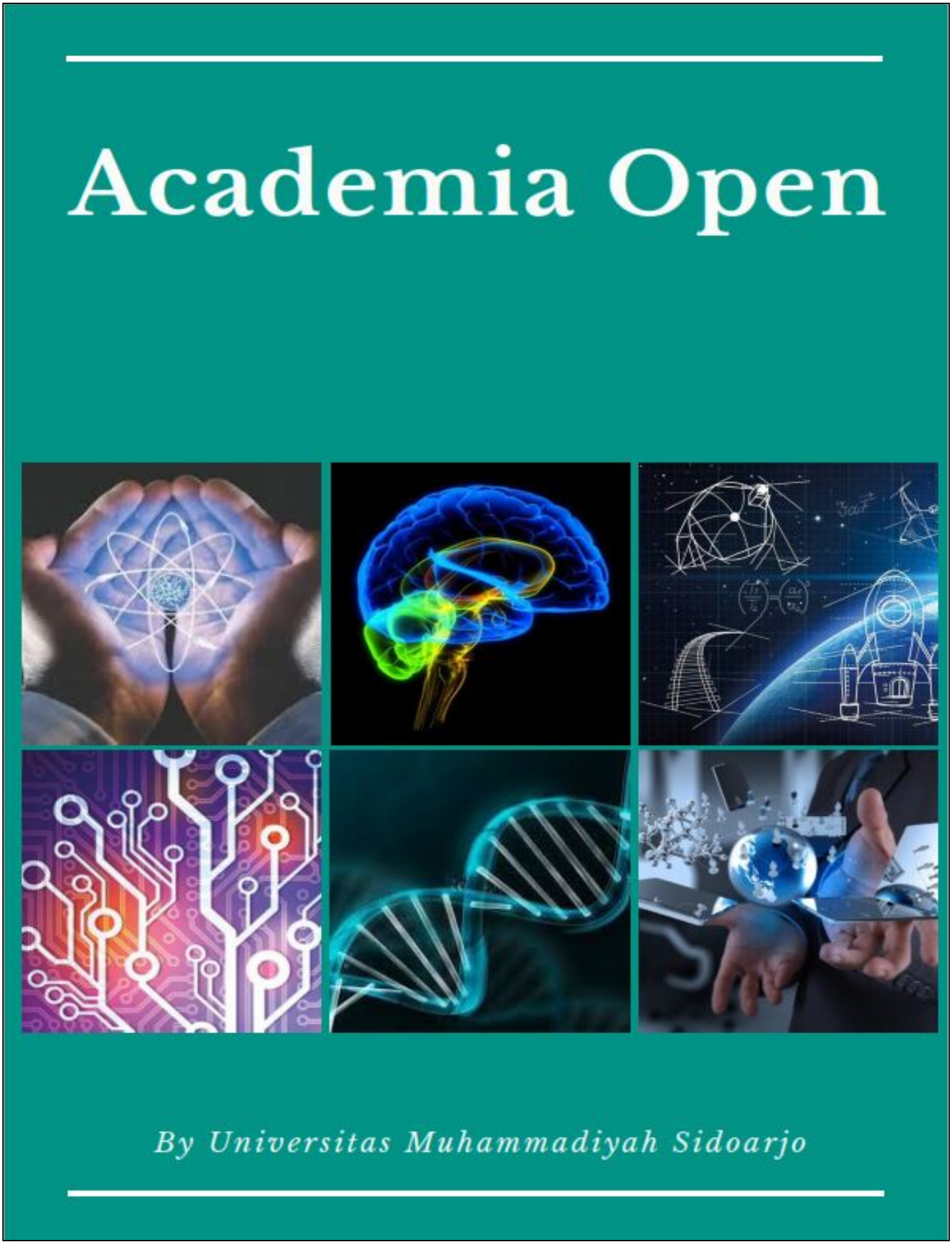

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Evaluation of Serum Vitamin D3, PTH, Calcium and Iron Levels in Patients with H. pylori Infection

Evaluasi Kadar Vitamin D3, PTH, Kalsium dan Zat Besi Serum pada Pasien dengan Infeksi H. pylori

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Abstract

Helicobacter pylori (H. pylori) is a gram negative bacterium, the prevalence of which is associated with chronic gastric infection and nutritional abnormalities. The purpose of this study is to ascertain the relationship between H. pylori infection and serum vitamin D3, PTH, calcium and iron levels in adults. The cross-sectional study was carried out at Al-Nasiriyah teaching hospital 20 patient with H. pylori infection and 20 healthy individuals. Blood samples were also examined for the presence of vitamin D3, PTH, calcium and iron content. It is shown that H. pylori-positive patients had significantly decreased serum vitamin D3, calcium and iron concentration, and increased PTH level comparing with control group. These results provide preliminary evidence to indicate that H. pylori infection may cause disorders in vitamin D3, calcium, iron metabolism that maybe associated with the impaired nutrient utilization that has clinical significance such as osteoporosis and anemia. More studies should be done to discover the causes of these depots and their effects on individuals with H. pylori

Highlights:

1. *H. pylori* causes chronic gastric infections and nutritional issues.
2. Infection reduces vitamin D3, calcium, iron; increases PTH levels.
3. Linked to osteoporosis, anemia; further studies recommended.

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Introduction

Helicobacter pylori is a spiral-shaped bacillus that is Gram-negative. It is one of the primary causes of chronic infection in humans, which in turn affects the gastrointestinal tract and results in micronutrient deficiencies (1). Early infancy fecal-oral HP pollution occurs, and infected populations are disproportionately socioeconomically disadvantaged. It is estimated that around 80% of people living in developing nations globally have HP (2).

The mechanism by which H. pylori spreads is not well known. Since H. pylori appears to have a limited host range, human stomach is the only known reservoir (3). New infections are assumed to result from environmental contamination or direct human-to-human transmission. Vertical and horizontal transmission are the two basic types into which person-to-person transmission may be separated. Within the same family, sickness can transmit vertically occurs from ascendant to descendant, while horizontal transmission transpires through interactions with individuals outside the familial unit or through environmental contamination. (4). An acquired H. pylori infection can spread through vomiting achlorhydric mucus. Gastric fluids may be the mode of transmission, particularly in cases where infantile vomiting occurs (5). Saliva could be a potential source of H. pylori, as this bacterium can inhabit the oral cavity after regurgitation or vomiting, making the gastric microbiome a possible reservoir. (6). Human feces have often been shown to contain H. pylori DNA. However, attempts to cultivate H. pylori from feces have not been very successful since the bacteria mostly survives there in a non-culturable (coccoid) form (7).

H. pylori enter the human stomach through various means, including environmental contamination and poor hygienic conditions. Some authors suggest that water serves as both an environmental reservoir and medium for fecal-oral transmission of the infection (8). Children residing in households with external water sources or ingesting raw vegetables irrigated with untreated sewage exhibit a greater prevalence of H. pylori infection. The correlation between H. pylori antibodies with antibodies targeting water-based pathogens The existence of Hepatitis A virus and Giardia suggests that the infection could be waterborne or linked with inadequate hygiene conditions (9 , 10). Food products, including milk, meat, and vegetables, can be contaminated due to poor handling under hygienic conditions. Studies have identified the role of food in the transmission of H. pylori, with milk being the most studied due to its prevalence during childhood, where the infection is primarily acquired (11).

H. pylori, a bacterium that inhabits the stomach epithelium and has urease activity producing ammonia to defend itself against acidity of the stomach. It also produces enzymes such as glicosulfatase and phospholipase A2 and C, which are responsible for Gastric mucosal damage. H. pylori induces an inflammatory response, resulting in the production of proinflammatory cytokines. Its virulence factors, particularly vacuolating toxin A (Vac-A) and cytotoxin associated gene A (Cag-A) positive strains, contribute to the pathogenesis of infection. Infection reduces gastrointestinal hormones and nutrient absorption, and affects metabolic balance. It also alters ghrelin and leptin levels (12, 13). Ghrelin peptide, a 28 amino acid peptide with a fatty acid chain modification, is a major active or oxygenic molecule that regulates energy balance. The active form regulates growth hormone-releasing and food intake, while the inactive form is effective on cell proliferation and adipogenesis. Acylated ghrelin increases food intake and promotes positive energy balance, while des-acylated ghrelin decreases food intake and lacks endocrine activities. H. pylori infection affects ghrelin and leptin hormone levels, leading to negative effects on appetite and food intake. Leptin concentrations are higher in H. pylori-positive individuals, while ghrelin concentrations and ghrelin/obestatin ratios are lower in H. pylori-negative individuals. After eradication, ghrelin levels and appetite increase, leading to body weight gain. In older ages, ghrelin levels and BMI are lower in H. pylori-positive individuals, suggesting H. pylori infection may be a contributing factor to malnutrition in the elderly (14, 15).

H. pylori infection in the stomach lining can lead to poor absorption of certain vitamins and minerals. Studies have shown that level of vitamin. B12, and folate was lower in patients with H. pylori compared to healthy individuals. Additionally, the reduction of gastric acid secretion, as well as a deficiency of ascorbic acid and blocking of iron binding protein, can result in iron deficiency in H. pylori patients. This can lead to iron deficiency and anemia, especially in children. Hypochlorhydria (is a medical term that refers to a condition in which there is a decrease in the production of hydrochloric acid (HCL) in the stomach. This can lead to a reduction in the absorption of certain nutrients, such as iron, as well as an increased risk of bacterial overgrowth in the digestive system) or reduced stomach acid production, can also result in decrease the absorption of iron by decreasing the availability of ascorbic acid, further impacting the absorption of non-heme iron (16, 17). H. pylori presences in the stomach can result in decreases the conversion of ferric to ferrous iron, and the utilization of iron by H. pylori strains as a growth factor, which may be a primary cause of iron deficiency. This can also result in decreased absorption due to changes in gastric physiology. Additionally, an increase in gastric pH can reduce iron solubility and affect absorption by decreasing the availability of vitamin B12 and folic acid (18).

Decreased intake of vitamin D has been identified as a dietary practice that may be related to H. pylori infection. Studies conducted in Turkey and Lebanon on adult populations have revealed that low blood levels of vitamin D (20 ng/mL) increase the risk of H. pylori infection (19, 20). Additionally, Research indicates an inverse correlation between H. pylori infection and vitamin D insufficiency, suggesting that sufficient levels of vitamin D may provide protection against H. pylori. A study in Bahrain indicated the likelihood of H. pylori infection elevated by about 1.1 units for every unit decrease in blood vitamin D levels. This association was also observed in research conducted on elderly individuals (21,22).

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H. pylori infection may impair calcium absorption by inducing stomach mucosal atrophy and reducing acid production. Therefore, the eradication of H. pylori may enhance calcium absorption and prevent the progression of osteoporosis by reducing inflammatory cytokine levels and improving stomach mucosal atrophy. (23).

The relationship between H pylori infection and hyperparathyroidism has not been well studied. According to one research, individuals with primary hyperparathyroidism were more likely to have a H pylori infection, particularly if they had stomach problems. This suggests that these patients should be evaluated and treated. PHPT was found to have a correlation with H. pylori infection and duodenal ulcer in another case study (24, 25, 26).

Methods

The study was carried out in al-Nasiriyah teaching hospital from 20 Jan 2024 to 35 may 2024. Total of 40(20 patients with H. pylori and 20 healthy). The paints group were Previously diagnosed with H. pylori. The group of patients with H. pylori was divided into (7 men and 13 women). The control group was selected of 20 healthy people (10 men and 10 women). Patients and healthy participants registered in this study accepted and signed informed consents. 3ml of blood sample were collected. Following sampling, blood was processed by centrifugation and serum stored in -20°C for the time of work. We measured serum for PTH, Iron, and vitamin D3 using electrochemiluminescent immunoassay (ECLIA) on an automated instrument (Cobas e400; Roche Diagnostics, GmbH, Mannheim, Germany). Serum Ca+2 was measured by using biochemistry semiauto type biosystem-350.

The statistical calculations of this paper were done by using the Statistical Package for the Social Sciences (SPSS) (version 29.0) program (IBM SPSS Statistics, SPSS Inc., Chicago, Illinois, USA). The test that has been used to perform the adherence of continuous, parametric, variable to the normal distribution was the Anderson-Darling test. Not normally continuous parametric variables were distributed, with significant outlier, presented using their mean and standard deviation (mean±SD) and parametric tests were used; Mann-Whitney t-test was applied for analyzing the differences between the means of two groups. Also, Sperman's correlations were used to find the relationships of different variables.

The discrete, non-parametric variables were presented using their number and percentages. The statistical tests assumed a null hypothesis stating no difference between the means of the variables. A P-value of 0.05 or lower was used to determine statistical significance.

Result and Discussion

The participants of this study included 20 healthy individuals (10 males, and 10 females) classified as (controls) and 20 patient individuals (7 males, and 13 females) were classified as (cases) thus. Table 1 showed distribution of study samples according to gander expressed as percentage with no significantly different (p>0.05).

Gender	Control (%)	Patient (%)	Total
Male	10 (58.82%)	7 (41.18%)	17
Female	10 (43.48%)	13 (56.52%)	23
Total	20	20	40

Table 1. *Gender distribution among the study sample*

The results in table (2) revealed significant differences in several biomarkers between the control and patient with H. pylori, the calcium, Iron, Vitamin D levels were significantly lower in patient group when compared to control group (calcium: p<0.0001, Iron: P<0.001, Vitamin D: P<0.0011), while PTH was higher in patient group (PTH: P<0.0026). The current results illustrated that age is not-significant differences between two groups patients and healthy group, while level of Ca+2 was higher in control group with high statistical significance, p≤ 0.005.

Variables	PATIENT (mean±SD)	CONTROL (mean±SD)	Mann-Whitney T-test (p – value)
Age (years)	39.8±9.43	39.8±9.50	0.9868
S. Ca (mg/dL)	7.31±0.55	8.72±0.484	<0.0001
Iron (µmol/L)	12.66 ± 5.42	22.54 ± 4.75	<0.001
Vitamin D (ng/mL)	18.4±7.70	30.8±17.1	0.0011
PTH (pg/mL)	29.5±20.6	14.4±3.23	0.0026

Table 2. *Comparison of some Biomarkers between Control Group and H. Pylori Patients Group.*

Investigation indicates that reduced stomach acid secretion in patients with atrophic gastritis, induced by H. pylori,

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results in decreased solubility of calcium salts, leading to calcium malabsorption (27). In Finland, it is known that 30-40% of older adults today experience chronic gastritis, typically caused by H. pylori infection, estimated that about half of these individuals will develop atrophic corpus gastritis if the infection remains untreated. This condition can contribute to calcium malabsorption (28). Pan et al., (2018) investigation that H. pylori infection may result in gastric mucosal atrophy, which reduces stomach acid secretion. A Hypochlorhydria (low acid) stomach negatively impacts calcium absorption, disrupting calcium balance (homeostasis) and potentially leading to decreased bone mass (29).

The relationship between H. pylori infection and primary hyperparathyroidism (PHPT). Hypercalcemia, common in PHPT patients, is linked to dyspeptic symptoms and often leads to elevated gastrin levels (hypergastrinemia) (30).

	S.ca	IRON	Vitamin D3	PTH	Age
S.ca	1.00	0.20	0.07	-0.08	-0.31
IRON	0.20	1.00	-0.17	-0.37	0.50
Vitamin D	0.07	-0.17	1.00	0.05	-0.10
PTH	-0.08	-0.37	0.05	1.00	-0.19
Age	-0.31	0.50*	-0.10	-0.19	1.00

Table 3. *correlation of study variables among patients.*

(Independent Samples T Test, P≤0.05)

The iron level is associated with age in patient group, while other parameters didn't show correlation in patient group. H. pylori, a gram-negative bacterium, is recognized for its ability to colonize various organs and its association with numerous extra-gastrointestinal illnesses. The link between H. pylori infection, vitamin D, and iron levels remains unclear; therefore, we examined the relationship between iron and vitamin D in patient infected with H. pylori.

Conclusion

Our results showed that vitamin D levels were higher among control group compared with patients who have H. pylori. This result agrees with study of Asher Shafrir which concluded that the infection with H. pylori was associated with level of vitamin D (31). Additional studies indicate that H. pylori infection is associated with a reduction in serum levels of different vitamins, including vitamin C, vitamin D, and vitamin B12 (32). Study found that low level of vitamin D considered a potential risk factor which may resulted in failure reduction of H. pylori, and supplement of vitamin D may be taken before treated H. pylori (33). The mechanisms that describe the association between H. pylori infection and decreased vitamin d level are unclear. Chronic gastritis is recognized to linked with H. pylori infection. The inflammation of gastritis may affect the absorption of mineral including iron and other micronutrients (32). It was reported that hypochlorhydria, resulting from H. pylori-induced chronic gastritis, causes a decrease in Vitamin B12 and iron absorption (32). The elimination of H. pylori has shown a reduction in stomach inflammation, an enhancement in gastric acid production, and an improvement in the absorption of iron (32). This investigation showed a significant association between decreased vitamin D levels and H. pylori infection.

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