ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Research Article DOI: 10.5281/zenodo.11036774

Are 25-hydroxyvitamin D deficiency and *Helicobacter pylori* infection more common in obese people?

Adil Furkan KILIÇ[1]
Muharrem BAYRAK[1]
Kenan ÇADIRCI[1]

[1]Health Sciences University, Erzurum Faculty of Medicine, Department of Internal Medicine, Erzurum, Turkey

ABSTRACT

Objective: Introduction: Our study aimed to show the significant relationship between *Helicobacter pylori (Hp)* positivity and 25-hydroxy vitamin D deficiency in obese individuals.

Materials and Methods: Patients over the age of 20 who applied to the internal medicine department with dyspeptic complaints between January 1, 2019, and December 31, 2019, were divided into three groups as 18-24.9 (normal weight), 25-29.9 (overweight), 30-39.9 (obese) according to their body mass indexes (BMI). Urea breath test for *Hp* infection, 25-hydroxyvitamin D and other biochemical parameters, anthropometric measurements, education levels, systemic diseases, smoking history of patients who did not use proton pump inhibitor, and 25-hydroxyvitamin D for the last six months were retrospectively analyzed from the patient file archive.

Results: The study was carried out with 632 cases, 51.6% (n = 326) of the patients were male, and 48.4% (n = 306) were female.The ages of the cases ranged from 21 to 65, and the mean age was 43.97 ± 12.87 years.Body mass index measurements of the cases included in the study ranged between 18.8 and 39.9 kg/m^2, with a mean of 28.02 ± 4.98 kg/m^2; %31.3% (n = 198) were normal weight, 35.5% (n = 224) were overweight and 33.2% (n = 210) were obese.

Conclusion: We think that vitamin D deficiency should be eliminated for eradication treatment in *Hp* positive individuals, and *Hp* should be investigated closely in obese people.We want to state that the study will contribute to studies on the relationship between *Hp* and vitamin D deficiency in obese people in the literature.

Keywords: 25-Hydroxyvitamin deficiency, *Helicobacter pylori*, obesity.

INTRODUCTION

The worldwide prevalence of obesity has been increasing. The prevalence of obesity in the United States (US) is over 20%, and the prevalence in 30 states of the US is over 25%. Of the people over 18 years of age in Europe,35.9% were of overweight (BMI between 25-29.9 kg/m^2), and 17.2% were obese.[1] The relationship of obesity with Helicobacter pylori *(Hp)* infection, as its relationship to the increase in the prevalence of type-2 diabetes mellitus and cardiovascular diseases, has been addressed in several studies, some of which indicated a positive association.[2,3] This relationship could not be demonstrated in the NHANES III study.[4] Kamada et al. have shown that the patients undergoing *Hp* eradication gained more weight than the patients without eradication.[5] The *Hp* is a microorganism that has infected more than half of the world population, with the prevalence varying with age and countries' level of development.[6] The *Hp* is a bacterium that resides in the gastric mucosa and has been associated with peptic ulcer, chronic gastritis, and gastric cancer.[7] Polymorphonuclear leukocyte infiltration is observed in gastric mucosa with acute infection, the effects of which are not clear. The immune response to acute infection leads to mononuclear cell infiltration an increase in pro-inflammatory cytokine is observed with inflammation.[8] In recent years studies, the risk of urinary tract infections, respiratory tract infections, tuberculosis has been increased with 25-hydroxyvitamin-D vitamin deficiency.[9-11] Vitamin D receptor (VDR) has been shown to play a crucial antimicrobial role against *Hp*.[12] Pro-inflammatory cytokines in the Hp infection have been shown in various studies to produce extragastric effects such as obesity, metabolic syndrome, and 25-hydroxyvitamin D (vitamin D) deficiency.[13-15]

Received 18.01.2024
Accepted 10.03.2024
Publication Date 23.04.2024

Corresponding author:
Muharrem BAYRAK
E-mail: muharem.bayrak@sbu.edu.tr

Cite this article: Kılıç AF, Bayrak M, Çadırcı K. Are 25-hydroxyvitamin D deficiency and Helicobacter pylori infection more common in obese people?. *Pharmata*. 2024;4(2):31-38

Adipose tissue is one of the main locations of storage for vitamin D.[16] Adipogenic gene expression plays an important role in the regulation of vitamin D. Vitamin D reduces the release of cytokines and inflammation of visceral adipose tissue by the inhibition of the nuclear factor kappa-light-chain (NF-kB).[16] Recent studies have indicated that the inflammation accompanying insulin resistance and the increased release of pro-inflammatory cytokines affected the synthesis of vitamin D negatively.[17,18] Recent studies have shown that insulin resistance in obese individuals and type-2 diabetes mellitus influence vitamin D receptors, leading to vitamin D deficiency.[19,20]

Various studies have investigated the relationship between obesity and *Hp* infection. We aimed to investigate the relationship between *Hp* infection and vitamin D deficiency in obese people.

METHODS

The study included the patients who applied to the Department of Internal Medicine at our hospital due to distension, epigastric pain, and dyspeptic complaints between 1 January and 31 December 2019. This study was approved by the Ethical Commitee (Date: 06.01.2020, Decision No: 01/03).

All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Written informed consent was obtained from patients who participated in this study.

Exclusion Criteria

The study excluded the patients who had undergone *Hp* eradication, had received other drug therapies in the past six months (oral anti-diabetics, anti-hyperlipidemics, proton pump inhibitors, vitamin D supplements, anti-inflammatory drugs, antibiotics); had acute and chronic infections, autoimmune diseases, refractory anemia, thrombocytopenia, solid tumors or hematological malignancies, chronic liver disease, chronic kidney disease, or inflammatory bowel disease; were positive for hepatitis B surface antigen (HBsAg) or anti-HCV,;had had a gastric operation; or were under the age of 20 years.

A total of 632 patients were divided into three groups based on their body-mass indices (BMIs): normal weight (18-24.9 kg/m^2), overweight (25-29.9 kg/m^2), and obese (30-39.9 kg/m^2). The patient files were examined for socioeconomic indicators and smoking history.

Laboratory Tests and Anthropometric Measurements

The patient records were examined for the laboratory tests and anthropometric measurements done in the last three months. The patients' fasting glucose, postprandial glucose, glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), aspartate aminotransferase (AST), triglyceride (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), total cholesterol, albumin, uric acid, erythrocyte sedimentation rate (ESR), vitamin D, and serum reactive protein (CRP) were investigated.

Vitamin D

Blood samples were collected in tubes containing ethylenediaminetetraacetic acid (EDTA), centrifuged, and collected into microcentrifuge tubes. Serum 25-hydroxyvitamin D was determined with liquid chromatography-tandem mass spectrometer (LC-MS/MS), and by using the ARCHITECT 5P02 kit and (Abbot ARCHITECT analyzer). Vitamin D levels were measured in December, January, or February. Serum 25-hydroxy D vitamin levels were considered as vitamin deficiency of less than 20 nmol/L.

Urea Breath Test

For the 13C urea breath test, the patients ingested a capsule containing 75 mg 13C-urea (which decomposes into carbon dioxide and ammonia) with 200 mL of liquids (orange juice, grapefruit juice, etc.) after at least 12 hours of fasting. Before the ingestion of the capsule, a baseline breath sample was collected into a breath collection bag with a mouthpiece (Helibacter Test INFAI). A second breath sample was collected after the ingestion. The baseline value at minute 0, and the value at 30th minute were recorded, and delta over baseline value was determined with HeliFan plus® (Fisher Analysen Instrumente GmgH, Leipzig, Germany). A delta over baseline value (DOB,δ ‰) at above or 3.0 δ ‰. was considered a positive test result. This test has a sensitivity of 95% and a specificity of 100%.

Waist Circumference

Waist circumference was measured between the arcus costarium and spina ilica anterior superior when the person was standing, with normal expiration and an empty stomach.

- Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) score
- HOMA-IR = fasting glucose (mg/dL) x fasting insulin (uU/mL)/405
- Body mass index (BMI)
- BMI = weight (kg)/height^2 (m^2)

Metabolic Syndrome Criteria

Metabolic syndrome diagnostic criteria were based on that of the International Diabetes Foundation (IDF)-2005: Abdominal obesity (Waist circumference: ≥94 cm in men, ≥80 cm in women) with at least two of the following:

- Triglyceride ≥150 mg/dL,
- HDL: <40 mg/dL in men, <50 mg/dL in women,
- Blood pressure ≥130/85 mmHg,
- Fasting blood glucose ≥100 mg/dL, or Type 2 DM.

Statistical Analysis

The statistical analyses were performed with the SPSS program (version 20, SPSS Inc., Chicago, IL). The descriptive statistics were presented as mean and standard deviation, median and minimum-maximum, or frequency and percentages). The normal distribution of numerical data was checked with the Kolmogorov-Smirnov test, Shapiro-Wilk test, and graphical evaluations. One-way analysis of variance (ANOVA) was used for multiple-group comparisons for data with normal distribution; the Bonferroni test was used for two-group comparisons. Kruskal-Wallis test was used for multiple-group comparisons for data without normal distribution; Bonferroni Dunntest test was used for two-group comparisons. Pearson Chi-Square test and Fisher-Freeman-Halton Exact test were also used for comparisons. The significance level was set at $p<0.05$ for all analyses.

RESULTS

The study included a total of 632 patients: 326 males (51.6%) and 306 females (48.4%). Average age was 43.97 ± 12.87 years (range 21 - 65 years). Of the patients, 9.2% (n=58) were illiterate, 34.7% (n=219) were elementary school graduates, 39.2% (n=248) were high school graduates, and 16.9% (n=107) were college graduates (Table 1).

The mean BMI was 28.02 ± 4.98 kg/m2 (range 18.8-39.9 kg/m^2). Of the patients, 31.3% (n=198) were of normal weight, 35.5% (n = 224) were of overweight, and 33.2% (n = 210) were obese. Of the patients, 24.7% (n=156) had hypertension, 15.0% (n=95) had diabetes, and 39.1% (n=247) had metabolic syndrome. The result of the 13C-urea test for *Hp* infection was positive in 45.4% (n=287) of the patients (Table 2).

When the patients were grouped by their age, gender, marital status, or history of smoking, no significant differences were found among the groups regarding their BMIs (*P*>.05). When the patients were grouped according to their educational status, a significant difference was found among the groups in terms of their BMIs (*P*=.001).

Table 1. Demographic characteristics

		n (%)
Age (years)	Min-Max (Median)	21-65 (44)
	Mean±SD	43.97±12.87
	< 30	109 (17.2)
	30- 39	165 (26.1)
	40- 49	124 (19.6)
	50- 59	134 (21.2)
	≥ 60	100 (15.8)
Gender	**Male**	326 (51.6)
	Female	306 (48.4)
Education	**Illiterate**	58 (9.2)
	Elementary	219 (34.7)
	High school	248 (39.2)
	College	107 (16.9)
Marital status	**Single**	139 (22.0)
	Married	442 (69.9)
	Widowed	51 (8.1)
Smoking	**No**	376 (59.5)
	Yes	256 (40.5)

SD: standard deviation

Table 2. Anthropometric measurements and chronic diseases

		n (%)
BMI (kg/m^2)	Min-Max (Median)	18.8-39.9 (27.2)
	Mean±SD	28.02±4.98
	Normal weight	198 (31.3)
	Overweight	224 (35.5)
	Obese	210 (33.2)
Waist circumference (cm)	Min-Max (Median)	69-128 (97)
	Mean±SD	95.84±8.93
Hypertension	**No**	476 (75.3)
	Yes	156 (24.7)
Diabetes mellitus	**No**	537 (85.0)
	Yes	95 (15.0)
Metabolic syndrome	**No**	385 (60.9)
	Yes	247 (39.1)
Helicobacter pylori	**No**	345 (54.6)
	Yes	287 (45.4)
Systolic pressure (mmHg)	Min-Max (Median)	103-178 (124)
	Mean±SD	125,12±8.21
Diastolic pressure (mmHg)	Min-Max (Median)	52-120 (83)
	Mean±SD	82.95±5.51

BMI: body-mass index, SD: standard deviation

The patients who were illiterate or elementary school graduates were more likely to be of overweight and obese (Table 3). Significant differences were found among the frequencies of hypertension in different BMI groups (*P*=.001). In two-groups comparisons, the frequency of hypertension in obese patients was higher than those in the normal or overweighted patients.

Table 3. Comparison of demographic characteristics of BMI groups

		BMI			
		Normal weight (n=198)	**Overweight (n=224)**	**Obese (n=210)**	
		n (%)	**n (%)**	**n (%)**	**p**
Age (years)	Min-Max (Median)	21-65 (44.5)	21-65 (43)	21-65 (44)	[a] **0.385**
	Mean±SD	44.69±13.08	43.04±12.65	44.29±12.91	
	< 30	33 (30.3)	41 (37.6)	35 (32.1)	
	30- 39	50 (30.3)	60 (36.4)	55 (33.3)	
	40- 49	37 (29.8)	46 (37.1)	41 (33.1)	
	50- 59	42 (31.3)	48 (35.8)	44 (32.8)	
	≥ 60	36 (36.0)	29 (29.0)	35 (35.0)	
Gender	**Male**	103 (31,6)	116 (35,6)	107 (32,8)	[b] **0.974**
	Female	95 (31.0)	108 (35.3)	103 (33.7)	
Education	**Illiterate**	8 (13.8)	25 (43.1)	25 (43.1)	[b] **0.001****
	Elementary	46 (21.0)	80 (36.5)	93 (42.5)	
	High school	97 (39.1)	78 (31.5)	73 (29.4)	
	College	47 (43.9)	41 (38.3)	19 (17.8)	
Marital status	**Single**	51 (36.7)	51 (36.7)	37 (26.6)	[b] **0.283**
	Married	134 (30.3)	152 (34.4)	156 (35.3)	
	Widowed	13 (25.5)	21 (41.2)	17 (33.3)	
Smoking	**No**	113 (30.1)	142 (37.8)	121 (32.2)	[b] **0.333**
	Yes	85 (33.2)	82 (32.0)	89 (34.8)	

BMI: body-mass index, SD: standard deviation [a]Oneway ANOVA Test [b]Pearson Ki-kare Test **p<0.01

Table 4. Comparison of anthropometric measurements and chronic diseases of BMI groups

		BMI			
		Normal weight (n=198)	**Overweight (n=224)**	**Obese (n=210)**	
		n (%)	**n (%)**	**n (%)**	**p**
Waist circumference (cm)	Min-Max (Median)	69-102 (91)	82-109 (98)	88-128 (102)	[a] **0.001****
	Mean±SD	87.49±8.37	97.16±4.98	102.30±6.22	
Hypertension	**No**	166 (83.8)	179 (79.9)	131 (62.4)	[b] **0.001****
	Yes	32 (16.2)	45 (20.1)	79 (37.6)	
Diabetes mellitus	**No**	186 (93.9)	197 (87.9)	154 (73.3)	[b] **0.001****
	Yes	12 (6.1)	27 (12.1)	56 (26.7)	
Metabolic syndrome	**No**	163 (82.3)	134 (59.8)	88 (41.9)	[b] **0.001****
	Yes	35 (17.7)	90 (40.2)	122 (58.1)	
Helicobacter pylori	**No**	132 (66.7)	124 (55.4)	89 (42.4)	[b] **0.001****
	Yes	66 (33,3)	100 (44,6)	121 (57,6)	
Systolic Pressure (mmHg)	Min-Max (Median)	103-137 (121)	110-145 (124)	109-178 (128)	[a] **0.001****
	Mean±SD	121.81±7.74	124.62±7.02	128.77±8.39	
Diastolic Pressure (mmHg)	Min-Max (Median)	70-98 (81)	52-96 (82)	71-120 (85)	[a] **0.001****
	Mean±SD	81.54±5.53	82.90±5.54	84.35±5.12	

BMI: body-mass index, SD: standard deviation [a]Oneway ANOVA Test [b]Pearson Ki-kare Test
**p<0.01

Significant differences were also found among the frequencies of diabetes in different BMI groups (*P*=.001). In two-groups comparisons, the frequency of diabetes in the obese patients was higher than those in the normal or over-weighted patients. The frequency in over-weighted patients was higher than that in the patients with normal weight. Significant differences were found among the frequencies of *Hp* infection in different BMI groups (*P*=.001). In two-groups comparisons, the frequency of *Hp* infection in the obese patients was higher than those in the normal or over-weighted patients.

The frequency in overweight patients was higher than that in the patients with normal weight. The systolic blood pressures of the obese patients were higher than those of normal and over-weighted patients (*P*=.001; *P*=.001, respectively). The diastolic blood pressures of the obese patients were higher than the normal and over-weighted patients (*P*=.001; *P*=.016, respectively) (Table 4). Significant differences were also found among the vitamin D levels of the groups by BMI (*P*=.001). The vitamin D level of the obese group was significantly lower than those of the overweight and normal-weight groups (*P*=.001, *P*=.001, respectively). The vitamin D level of the overweight group was significantly lower than that in the normal-weight group (*P*=.001). Significant differences were found among the HOMA-IR scores of the groups by BMI (*P*=.001) (Table 5). A significant relationship was found between the BMIs and vitamin D levels of patients who had HP infection (*P*=.001). The rate of patients with a vitamin D level below 10 nmol/L in the obese group was higher than those in the normal and overweight groups. The rate of patients with a vitamin D level of 10-20 nmol/L in the overweight group was higher than those in the normal-weight or obese groups. The rate of patients with a vitamin D level of 21-30 nmol/L in the normal and overweight groups were higher than that in the obese group. Moreover, the rate of patients with a vitamin D level of 21-30 nmol/L in the normal group was higher than that in the overweight group (Table 6).

Table 5. Comparison of laboratory test results of BMI groups

		BMI			
		Normal weight (n=198)	**Overweight (n=224)**	**Obese (n=210)**	*P*
LDL(mg/dL)	Min-Max (Median)	58-184 (116)	56-189 (125.5)	63-232 (127.5)	[a].001**
	Mean±SD	116.64±21.84	123.01±27.32	130.88±30.92	
HDL(mg/dL)	Min-Max (Median)	22-95.5 (46.8)	24.9-84.3 (45,9)	21.3-100.9 (42.3)	[a].001**
	Mean±SD	47.80±9.95	44.96±9.60	44.08±11.68	
Triglyceride(mg/dL)	Min-Max (Median)	52-369 (126)	46-556 (146)	45-1044 (163)	[c].001**
	Mean±SD	130.22±45.16	159.79±74.20	192.00±122.10	
Total Cholesterol(mg/dL	Min-Max (Median)	106-219 (184)	96-306 (195)	97-334 (201.5)	[a].001**
	Mean±SD	177,34±25.51	192,20±36.70	203.50±40.15	
ALT(U/L)	Min-Max (Median)	4-42 (12)	5.1-65 (15)	6-140 (18)	[c].001**
	Mean±SD	14.49±6.13	17.38±8.94	22.93±17.08	
AST(U/L)	Min-Max (Median)	5-42 (16)	6-56 (16)	8-72 (17.5)	[c].002**
	Mean±SD	16.99±6.93	18.82±8.27	19.81±9.38	
Uric acid (mg/dL)	Min-Max (Median)	1.3-7 (4,2)	2-18 (4.4)	2.3-13 (5.7)	[a].001**
	Mean±SD	4.24±1.09	4.55±1.59	5.78±1.62	
Albumin(g/dL)	Min-Max (Median)	2.1-4.7 (4.1)	2.9-5 (4.1)	3.2-5.2 (4,3)	[c].001**
	Mean±SD	4.07±0.25	4.09±0.25	4.32±0.34	
CRP(mg/dL)	Min-Max (Median)	0.1-9.1 (1.7)	0.1-18 (2.2)	0.3-20.1 (2.8)	[c].001**
	Mean±SD	1,77±1,55	2,41±2,45	3,14±3,29	
Vitamin D(nmol/L)	Min-Max (Median)	10-38 (21.2)	6.8-36.5 (18.6)	4.6-34.9 (14.9)	[a].001**
	Mean±SD	21.09±5,58	18.77±5.67	15.45±6.44	
Fasting glucose (mg/dL)	Min-Max (Median)	70-104 (78)	70-185 (78)	71-325 (84)	[c].001**
	Mean±SD	81.14±8.73	82.45±12.79	92.39±26.91	
Postprandial glucose (mg/dL)	Min-Max (Median)	106-202 (124)	81-269 (129)	86-540 (138)	[c].001**
	Mean±SD	129.39±20.78	139.07±27.42	160.52±52.26	
HOMA-IR	Min-Max (Median)	0.2-9 (1.9)	1.3-16.1 (4.2)	2.9-33.7 (6.9)	[c].001**
	Mean±SD	2.39±1.43	4.46±1.73	7.57±3.59	
HbA1c(%)	Min-Max (Median)	4.8-7.2 (5.2)	4.8-8.1 (5.4)	4.6-11.4 (5.6)	[c].001**
	Mean±SD	5.33±0,42	5.58±0,50	5.98±1,07	

BMI: body-mass index, SD: standard deviation,LDL:low-density lipoprotein,HDL:high-density lipoprotein, ALT: alanine aminotransferase, AST: aspartate aminotransferase, CRP:C-reactiveprotein,HOMA-IR:Homeostatic Model Assessment for Insulin Resistance,HbA1c:glycated hemoglobin. [a]Oneway ANOVA Test ,[c]Kruskal Wallis Test ***P*<.01

Table 6. The relationship between BMI and vitamin D levels in individuals with or without Helicobacter pylori infection

		BMI			
		Normal weight	**Overweight**	**Obese**	
		n (%)	**n (%)**	**n (%)**	*P*
Helicobacterpylori (-) [n=345]		132	124	89	
Vitamin D level	< 10 nmol/L	0 (0)	6 (4,8)	5 (5,6)	.001**
	10-20 nmol/L	59 (44,7)	64 (51,6)	51 (57,3)	
	21-30 nmol/L	67 (50,8)	51 (41,1)	25 (28,1)	
	>30 nmol/L	6 (4,5)	3 (2,4)	8 (9,0)	
Helicobacterpylori (+) [n=287]		66	100	121	
VitaminD level	< 10 nmol/L	1 (1,5)	8 (8,0)	45 (37,2)	.001**
	10-20 nmol/L	37 (56,1)	74 (74,0)	71 (58,7)	
	21-30 nmol/L	27 (40,9)	18 (18,0)	5 (4,1)	
	>30 nmol/L	1 (1,5)	0 (0)	0 (0)	

BMI: body-mass index Fisher-Freeman-Halton Exact test ***P*<0.01

DISCUSSION

Obesity is a very complex condition that causes many pathological changes, leading to endocrine, metabolic, and cardiovascular diseases and cancer.[21] With the increasing prevalence of obesity, its relationship with other diseases has become an important health concern. Several recent studies have indicated a relationship between the *Hp* infection, insulin resistance, and obesity.[15,22-24] In our study, we investigated whether *Hp* infection and vitamin D deficiency were more common in obese people compared to those with normal weight.In our study, the average age was 43.97 ± 12.87 years; the study was similar to other studies in the literature in terms of the age distribution.[25,26] No significant differences were found between the two genders. In the study by Li-Wei Chen et al.[27], there was a higher number of obese female patients. We found that obesity decreased as the level of education increased. Cynthia L. et al.[28] found that obesity was less common among university graduates in the United States. Obesity was also less common among university graduates in our study. We think that socioeconomic factors contribute to this result. In a meta-analysis that mostly involved European countries, Lender N et al.[29] found a negative correlation between obesity and *Hp* seropositivity. In a retrospective study involving 3,039 people in China, Mei-Yan Xu et al.[30] found that although the *Hp* seropositivity was more frequent (54.6%) in the obese group, the difference was not statistically significant. In our study, the rate of *Hp* seropositivity was 57.6% in the obese group, which was significantly higher than that in the other groups. In a study of 214 patients in Turkey, Aslan et al.[31] found an *Hp* seropositivity rate of 57.2% among obese individuals, which was significantly higher than that in the non-obese group. In a study involving 2,050 people in China, Yan Zhang et al.[32] found that the rate of *Hp* seropositivity was significantly higher among obese people.In a cohort study of 235,107 people, Suki et al.[33] found a higher rate of *Hp* seropositivity. Al-Zubaidi et al.[34] found that *Hp* infection was observed more frequently in the gastroscopic biopsies from obese patients. We think that the differences among the studies may be due to the differences in the socioeconomic, genetic, and environmental factors.

We found a mean vitamin D level of 15.45 ± 6.44 nmol/L in the obese group, which was significantly lower than those in the normal and overweight groups. In a study investigating the results of vitamin D supplementation in 16,540 people with vitamin D deficiency, Saliba et al.[35] found that the response to the supplementation therapy was lower in the obese group compared to that in the normal-weight group. We think that the lower vitamin D levels in the obese group in our study might be due to their sedentary lifestyle and excessive immobility. The higher HOMA-IR scores and CRP values in the obese group also indicated that insulin resistance and chronic inflammation might have decreased the vitamin D levels in obese individuals.

There is limited data that is showing the relationship between vitamin D and helicobacter pylori. It is confirmed that vitamin D regulates the expression of antimicrobial peptides cathelicidin and β-defensin.[36] The peptide β-defensin, which secreted from the gastric mucosal surface constitutes a defence against after *Hp* infection. In a vitamin D deficiency situation, macrophages cannot perform the synthesis of vitamin D necessary for the production of cathelicidin and β-defensin unable to be effective against *Hp.*[37] In our study, the average vitamin D levels were 15.79 ± 5.61 ng/mL in the Hp-positive group and 20.56 ± 6.0 ng/mL in the Hp-negative group. The results were similar to those from a previous study, which demonstrated that the infections by H. pylori, gram-negative bacteria, and other microorganisms decreased the vitamin D levels.[38] In a study of 150 patients, El Shahawy et al.[39] demonstrated that they had increased vitamin D levels after H. pylori eradication, which supported the results of our study. Consistent with the literature, we found that insulin resistance, type-2 diabetes mellitus, hyperlipidemia, and metabolic syndrome were more common in the obese group.[40,41]

The limitations of this study included the fact that the study included a small number of patients, it was a retrospective study, and tissue biopsy was not performed in the diagnosis of *Hp* infection.

In conclusion, we investigated whether *Hp* infection and vitamin D deficiency were more common in obese people compared to those in people with normal or overweight. We want to emphasize that vitamin D deficiency in obese people should be more at attention, and diet treatments should be adjusted accordingly. We think that *Hp* infection is more common in people with vitamin D deficiency and that should supplied vitamin D deficiency for eradication treatment in Hp-positive individuals and study contributes to the discussion in the literature, and there is a need for prospective studies with a larger group of patients.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Clinical Resarch Ethics Committee of Erzurum Regional Training and Research Hospital (Date: January 06, 2020, Number: 2020/01-03).
Informed Consent: Written informed consent was obtained from patients who participated in this study.
Peer-review: Externally peer-reviewed.
Author Contributions: Concept – A.F.K.; Design – A.F.K.; Materials – A.F.K., M.B., K.Ç.; Data Collection and/or Processing – A.F.K., M.B., K.Ç.; Analysis and/or Interpretation – A.F.K., M.B., K.Ç.; Literature Search – A.F.K., M.B., K.Ç.; Writing Manuscript – A.F.K., M.B., K.Ç.; Critical Review – A.F.K.
Conflict of Interest: The authors have no conflicts of interest to declare.
Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Grethen E, McClintock R, Gupta CE, et al. Vitamin D and hyperparathyroidism in obesity. *The Journal of Clinical Endocrinology & Metabolism*. 2011;96(5):1320-1326. [CrossRef]
2. Wu M-S, Lee W-J, Wang H-H, Huang S-P, Lin J-T. A case-control study of association of Helicobacter pylori infection with morbid obesity in Taiwan. *Archives of Internal Medicine*. 2005;165(13):1552-1555. [CrossRef]
3. DiBaise JK, Zhang H, Crowell MD, Krajmalnik-Brown R, Decker GA, Rittmann BE. Gut microbiota and its possible relationship with obesity. Elsevier; 2008:460-469. [CrossRef]
4. Cho I, Blaser MJ, François F, et al. Helicobacter pylori and overweight status in the United States: data from the Third National Health and Nutrition Examination Survey. *American Journal of Epidemiology*. 2005;162(6):579-584. [CrossRef]
5. Kamada T, Sugiu K, Haruma K. Development of obesity and hyperlipidemia after eradication of Helicobacter pylori. *Nihon rinsho Japanese Journal of Clinical Medicine*. 2005;63:536-538.
6. Sayılar EI, Çelik B, Dumlu Ş. Relationship between Helicobacter pylori infection and metabolic syndrome. *Turk J Gastroenterol*. 2015;26(6):468-473. [CrossRef]
7. Parsonnet J. Helicobacter pylori and gastric cancer. *Gastroenterology Clinics of North America*. 1993;22(1):89-104. [CrossRef]
8. Larussa T, Leone I, Suraci E, Imeneo M, Luzza F. Helicobacter pylori and T helper cells: mechanisms of immune escape and tolerance. *Journal of Immunology Research*. 2015;2015 [CrossRef]
9. Shalaby SA, Handoka NM, Amin RE. Vitamin D deficiency is associated with urinary tract infection in children. *Archives of Medical Science*. 2018;14(1):115-121. [CrossRef]
10. Wang H, Chen W, Li D, et al. Vitamin D and chronic diseases. *Aging and Disease*. 2017;8(3):346. [CrossRef]
11. Coussens AK, Martineau AR, Wilkinson RJ. Anti-inflammatory and antimicrobial actions of vitamin D in combating TB/HIV. *Scientifica*. 2014;2014 [CrossRef]
12. Martins DdJ, Matos GC, Loiola RS, D'Annibale V, Corvelo T. Relationship of vitamin D receptor gene polymorphisms in Helicobacter pylori gastric patients. *Clinical and Experimental Gastroenterology*. 2018:19-27. [CrossRef]
13. Lim SH, Kim N, Kwon JW, et al. Positive association between Helicobacter pylori infection and metabolic syndrome in a Korean population: a multicenter nationwide study. *Digestive Diseases and Sciences*. 2019;64:2219-2230. [CrossRef]
14. Shin DW, Kwon HT, Kang JM, et al. Association between metabolic syndrome and Helicobacter pylori infection diagnosed by histologic status and serological status. *Journal of Clinical Gastroenterology*. 2012;46(10):840-845. [CrossRef]
15. Chen L-W, Chien C-Y, Hsieh C-W, et al. The associations between Helicobacter pylori infection, serum vitamin D, and metabolic syndrome: a community-based study. *Medicine*. 2016;95(18):e3616. [CrossRef]
16. Abbas MA. Physiological functions of Vitamin D in adipose tissue. *The Journal of Steroid Biochemistry and Molecular Biology*. 2017;165:369-381. [CrossRef]
17. Wimalawansa SJ. Associations of vitamin D with insulin resistance, obesity, type 2 diabetes, and metabolic syndrome. *The Journal of Steroid Biochemistry and Molecular Biology*. 2018;175:177-189. [CrossRef]
18. Garbossa SG, Folli F. Vitamin D, sub-inflammation and insulin resistance. A window on a potential role for the interaction between bone and glucose metabolism. *Reviews in Endocrine and Metabolic Disorders*. 2017;18(2):243-258. [CrossRef]
19. Peterson CA, Tosh AK, Belenchia AM. Vitamin D insufficiency and insulin resistance in obese adolescents. *Therapeutic Advances in Endocrinology and Metabolism*. 2014;5(6):166-189. [CrossRef]
20. Ju SY, Jeong HS, Kim DH. Blood vitamin D status and metabolic syndrome in the general adult population: a dose-response meta-analysis. *J Clin Endocrinol Metab*. 2014;99(3):1053-1063. [CrossRef]
21. An J, Yoon SR, Lee JH, Kim H, Kim OY. Importance of adherence to personalized diet intervention in obesity related metabolic improvement in overweight and obese Korean adults. *Clinical Nutrition Research*. 2019;8(3):171. [CrossRef]
22. Kumari M, Kozyrskyj A. Gut microbial metabolism defines host metabolism: an emerging perspective in obesity and allergic inflammation. *Obesity Reviews*. 2017;18(1):18-31. [CrossRef]
23. Xu X, Li W, Qin L, Yang W, Yu G, Wei Q. Relationship between Helicobacter pylori infection and obesity in Chinese adults: a systematic review with meta-analysis. *PloS one*. 2019;14(9):e0221076. [CrossRef]
24. Tang DM, Kumar S. The association between Helicobacter pylori infection and nonalcoholic fatty liver disease. *Current Gastroenterology Reports*. 2017;19:1-5. [CrossRef]
25. Xu C, Yan M, Sun Y, et al. Prevalence of Helicobacter pylori infection and its relation with body mass index in a Chinese population. *Helicobacter*. 2014;19(6):437-442. [CrossRef]
26. Wan Z, Hu L, Hu M, Lei X, Huang Y, Lv Y. Helicobacter pylori infection and prevalence of high blood pressure among Chinese adults. *Journal of Human Hypertension*. 2018;32(2):158-164. [CrossRef]
27. Chen L-W, Kuo S-F, Chen C-H, Chien C-H, Lin C-L, Chien R-N. A community-based study on the association between Helicobacter pylori Infection and obesity. *Scientific Reports*. 2018;8(1):10746. [CrossRef]
28. Ogden CL. Prevalence of obesity among youths by household income and education level of head of household—United States 2011–2014. *MMWR Morbidity and Mortality Weekly Report*. 2018;67. [CrossRef]
29. Lender N, Talley N, Enck P, et al. associations between H elicobacter pylori and obesity-an ecological study. *Alimentary Pharmacology & Therapeutics*. 2014;40(1):24-31. [CrossRef]
30. Xu M-Y, Liu L, Yuan B-S, Yin J, Lu Q-B. Association of obesity with Helicobacter pylori infection: a retrospective study. *World Journal of Gastroenterology*. 2017;23(15):2750. [CrossRef]
31. Arslan E, Atılgan H, Yavaşoğlu İ. The prevalence of Helicobacter pylori in obese subjects. *European Journal of Internal Medicine*. 2009;20(7):695-697. [CrossRef]

32. Zhang Y, Du T, Chen X, Yu X, Tu L, Zhang C. Association between Helicobacter pylori infection and overweight or obesity in a Chinese population. *The Journal of Infection in Developing Countries*. 2015;9(09):945-953. [CrossRef]
33. Suki M, Weissman YL, Boltin D, et al. Helicobacter pylori infection is positively associated with an increased BMI, irrespective of socioeconomic status and other confounders: a cohort study. *European Journal of Gastroenterology & Hepatology*. 2018;30(2):143-148. [CrossRef]
34. Al-Zubaidi AM, Alzobydi AH, Alsareii SA, Al-Shahrani A, Alzaman N, Kassim S. Body mass index and Helicobacter pylori among obese and non-obese patients in Najran, Saudi Arabia: a case-control study. *International Journal of Environmental Research and Public Health*. 2018;15(11):2586.
35. Saliba W, Barnett-Griness O, Rennert G. The relationship between obesity and the increase in serum 25 (OH) D levels in response to vitamin D supplementation. *Osteoporosis International*. 2013;24:1447-1454. [CrossRef]
36. Wang T-T, Nestel FP, Bourdeau V, et al. Cutting edge: 1, 25-dihydroxyvitamin D3 is a direct inducer of antimicrobial peptide gene expression. *The Journal of Immunology*. 2004;173(5):2909-2912. [CrossRef]
37. Neshani A, Zare H, Akbari Eidgahi MR, Hooshyar Chichaklu A, Movaqar A, Ghazvini K. Review of antimicrobial peptides with anti-Helicobacter pylori activity. *Helicobacter*. 2019;24(1):e12555. [CrossRef]
38. Ramanathan B, Davis EG, Ross CR, Blecha F. Cathelicidins: microbicidal activity, mechanisms of action, and roles in innate immunity. *Microbes and Infection*. 2002;4(3):361-372. [CrossRef]
39. El Shahawy MS, Hemida MH, El Metwaly I, Shady ZM. The effect of vitamin D deficiency on eradication rates of Helicobacter pylori infection. *JGH Open*. 2018;2(6):270-275. [CrossRef]
40. Saltiel AR, Olefsky JM. Inflammatory mechanisms linking obesity and metabolic disease. *The Journal of Clinical Investigation*. 2017;127(1):1-4. [CrossRef]
41. Hall ME, Wang Z, do Carmo J, Kamimura D, Hall JE. Obesity and metabolic syndrome hypertension. *Disorders of Blood Pressure Regulation: Phenotypes, Mechanisms, Therapeutic Options*. 2018:705-722. [CrossRef]