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Assessment of vitamin D deficiency and hyperparathyroidism in metabolically healthy and unhealthy obese patients

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ABSTRACT

Aims: This study aimed to compare the levels of vitamin D in metabolically healthy (MHO) and metabolically unhealthy obese (MUO) individuals and determine if there are differences between these two groups concerning vitamin D deficiency and hyperparathyroidism.

Methods: A total of 263 obese female patients were included in the study and divided into two groups based on metabolic syndrome diagnostic criteria. Biochemical and anthropometric data obtained after a 12-hour fasting period were analyzed.

Results: Among the patients, the average 25-OH vitamin D level was 10.9±6.5 ng/ml. A total of 242 patients (92%) had vitamin D deficiency, and 132 patients (50.2%) were diagnosed with hyperparathyroidism. Significant differences were found in vitamin D ($p=0.003$) and uric acid ($p<0.001$) levels between the MHO and MUO groups. Additionally, the groups with vitamin D deficiency showed significantly different glucose ($p=0.026$) and homeostatic model assessment for insulin resistance ($p=0.042$) values. Patients with hyperparathyroidism had higher waist circumference ($p<0.001$), waist-to-hip ratio ($p=0.018$), BMI ($p=0.006$), and systolic ($p=0.001$) and diastolic ($p<0.001$) blood pressure values compared to those with normal parathyroid hormone levels.

Conclusion: The study emphasizes the importance of monitoring vitamin D deficiency and hyperparathyroidism in obese patients, as these conditions are more prevalent in this population and might be associated with metabolic syndrome parameters, increasing cardiometabolic risk.

Keywords: Vitamin D deficiency, obesity, hyperparathyroidism, metabolic syndrome, cardiovascular disease

INTRODUCTION

Obesity, a chronic condition, has experienced a rise in prevalence in both developed and developing nations. Recent global assessments indicate that nearly 108 million children (approximately 5% prevalence) and 604 million adults (around 12% prevalence) are affected by obesity.[1] Generally, obesity rates are higher among women compared to men, and the risk tends to increase with age after 14 years.[1,2] In recent decades, there have been findings indicating that certain obese individuals have a notably reduced risk of developing cardio metabolic diseases. This has resulted in the introduction of the term "metabolically healthy obesity" (MHO) in scientific literature.[3] While the exact definition of MHO remains unclear, it is suggested that it can be identified by the absence of metabolic syndrome (MS) criteria.[4] Moreover, individuals with MHO are reported to exhibit lower levels of visceral fat, higher insulin sensitivity, and better-preserved beta cell reserve.[3,4]

Vitamin D deficiency is recognized as a significant global public health problem, impacting various bodily systems beyond musculoskeletal health.[5,6] Vitamin D deficiency in obese individuals is a well-documented observation; however, a definitive cause-and-effect relationship has not been established.[7,8] Recently, studies have suggested that vitamin D deficiency may be linked to the pathogenesis of MS components, including obesity, insulin sensitivity, hypertension and carbohydrate metabolism disorders.[5,7,9,10] Moreover, there are reports suggesting that secondary hyperparathyroidism, arising from vitamin D deficiency, is linked to obesity and its associated comorbidities.[11-13] It has even been suggested that secondary hyperparathyroidism may ameliorate with obesity treatment.[13]

Based on all the available data, the objective of this study is to examine whether there exists a disparity in

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vitamin D levels, secondary hyperparathyroidism and the incidence of vitamin D deficiency between patients with MHO and those with "metabolically unhealthy obesity" (MUO).

METHODS

Ethical Approval

The study was carried out with the permission of Erzincan Binali Yıldırım University Clinical Researches Ethics Committee (Date: 11.05.2023, Decision No: 2023-10/2). All procedures adhered to ethical guidelines and the principles outlined in the Declaration of Helsinki. Additionally, all participants provided informed written consent.

Study Population

Our study included 263 obese (body mass index, (BMI) ≥30 kg/m²) female patients, aged 18-65, who newly applied for follow-up at our hospital's obesity center. Patients who were pregnant or suspected to be pregnant, patients with chronic kidney insufficiency, those who had received vitamin D replacement therapy within the last 6 months, and patients using agents that could alter MS parameters were excluded from the study after screening.

Anthropometric Measures and Laboratory Analyses

Biochemical data and anthropometric measurement data obtained after 12 hours of fasting were obtained using the files of the obesity center. The recorded parameters included age (in years), waist and hip circumference (in cm), systolic and diastolic blood pressure (in mmHg), C-Reactive Protein (CRP), total cholesterol, HDL-cholesterol, LDL-cholesterol, triglyceride, glucose, calcium, phosphate, creatinine, and uric acid levels (in mg/dl), HbA1c (in %), thyroid-stimulating hormone level (in U/ml), parathyroid hormone level (in pg/ml), and 25-OH-vitamin D level (in ng/ml).

Based on the baseline data provided above, BMI (in kg/m²), waist-to-hip ratio, and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) values were calculated appropriately using the relevant formulas.

The presence of MS was assessed using the revised NCEP-ATP ATP III MS criteria.[14] According to this, the diagnosis of MS includes having three or more of the following criteria: elevated fasting blood sugar (≥100 mg/dl) or a diagnosis of type 2 diabetes (T2DM), high blood pressure (≥130/85 mm Hg) or a diagnosis of hypertension, low HDL cholesterol levels (less than 50 mg/dl for women), elevated triglyceride levels (≥150 mg/dl), and increased waist circumference specific to the population (≥90 cm as recommended by national guidelines for women in our country).[15]

Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences 15.0 (SPSS Inc., Chicago, IL, USA) for Windows. Normality was tested by Kolmogorov-Smirnov test. Normally distributed data were expressed as mean ± standard deviation. Inter Quartile ranges were used for variables without a normal distribution. Data with a normal distribution were compared by Student's t test and comparisons of continuous variables with an asymmetric distribution were made by using the Mann-Whitney U test. Statistically significant differences between the groups were determined by the chi-square test for categorical variables. A p-value less than 0.05 was considered significant.

RESULTS

We included 263 obese female patients with an average age of 42.9±10.4 years and an average BMI of 37.5±4.5 kg/m² in our study. Among the patients, 49 were diagnosed with T2DM. Additionally, 5 new cases of T2DM were identified based on HbA1c and fasting glucose levels. During the assessment, 18 patients met all 5 criteria for MS, 40 patients met 4 MS criteria, and 85 patients had 3 positive MS criteria, totaling 143 cases of MS and MUO patients. Moreover, 85 patients had 2 MS criteria, 32 had 1 criterion, and 3 had no MS criteria, making a total of 120 MHO patients.

The average 25-OH vitamin D level among the patients was 10.9±6.5 ng/ml, while the average PTH (parathyroid hormone) level was 92.5±42.4 pg/ml. Out of the patients, 242 (92%) had a deficiency in 25-OH vitamin D, while 21 (8%) had a vitamin D level ≥20 ng/ml. Furthermore, 131 patients (49.8%) had PTH values within the normal range, and 132 patients (50.2%) were diagnosed with hyperparathyroidism.

As seen in **Table 1**, between the MHO and MUO patient groups, in addition to the MS parameters used for the definition and differentiation of the groups, 25-OH-D vitamin (p=0.003) and uric acid (p<0.001) were found to be statistically different. Among the groups with and without vitamin D deficiency, glucose (p=0.026) and HOMA-IR (p=0.042) values were found to be statistically different (**Table 2**). Moreover, in the patient group with hyperparathyroidism, waist circumference (p<0.001), waist-to-hip ratio (p=0.018), BMI (p=0.006), systolic (p=0.001), and diastolic blood pressure (p<0.001) values were found to be significantly higher compared to the group with normal parathyroid hormone levels (**Table 3**).

Table 1. Demographic and clinical parameters of metabolically healthy and unhealthy obese patients

Parameters	Metabolically healthy obese patients n=120	Metabolically unhealthy obese patients n=143	p value
Age (years)	42±10	44±11	0.098
Waist circumference (cm)*	102±10	109±8	<0.001
Hip circumference (cm)	125 (120-133)	127 (122-135)	0.484
Waist to hip ratio	0.81±0.1	0.86±0.1	<0.001
Body mass index (kg/m²)	36.3 (32.9-39.6)	37.8 (35.3-40.4)	0.075
Systolic blood tension (mmHg)*	110 (110-120)	130 (120-140)	<0.001
Diastolic blood tension (mmHg)*	70 (60-80)	80 (70-90)	<0.001
C-reactive protein (mg/L)	4.6 (3.3-8.4)	4.45 (3.2-8.6)	0.943
Total cholesterol (mg/dl)	198±41	203±39	0.298
HDL- cholesterol (mg/dl)*	53 (45-61)	47 (42-52)	<0.001
LDL- cholesterol (mg/dl)	129±30	134±28	0.182
Triglyceride (mg/dl)*	110 (83-135)	168 (125-226)	<0.001
Glucose (mg/dl)*	91 (85-95)	101 (91-114)	<0.001
HbA1c (%)*	5.5 (5.3-5.7)	5.7 (5.5-6.3)	<0.001
Calcium (mg/dl)	9.4 (9.2-9.5)	9.3 (9.1-9.6)	0.830
Phosphate (mg/dl)	3.6 (3.2-3.9)	3.7 (3.3-4)	0.093
Creatinine (mg/dl)	0.76 (0.71-0.82)	0.75 (0.7-0.83)	0.167
Uric acid (mg/dl)	4.6±1.1	5.2±1.2	<0.001
TSH (U/ml)	2.4 (1.5-3.4)	2.3 (1.4-3.9)	0.860
PTH (pg/ml)	77.7 (60.4-110.6)	87.5 (69.5-115.6)	0.051
25- OH-Vit-D (ng/ml)	11.3 (7.7-16.7)	9.3 (5.6-13.9)	0.003
25- OH-Vit D deficiency [n (%)]	14 (11.4%)	7 (4.9%)	0.044
HOMA-IR	2.33 (1.47-3.58)	3.99 (2.6-6.27)	<0.001

HDL; High-Density Lipoprotein, LDL; Low-Density Lipoprotein, HbA1c; Glycolyzed Hemoglobin, TSH; Thyroid Stimulating Hormone, PTH; Parathormone, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance. Parameters with normal distribution are presented as the Mean±Standard deviation (Student t test). Parameters with abnormal distribution presented as median and interquartile range (Mann-Whitney U test). Chi-square test was used to compare categorical data. * Parameters used in the diagnosis of metabolic syndrome.

Table 2. Demographic and clinical parameters in patients with and without vitamin D deficiency

Parameters	Patients with a vitamin D value <20 ng/ml n=242	Patients with a vitamin D value ≥20 ng/ml n=21	p value
Age (years)	44 (36-50)	42 (37 -50)	0.625
Waist circumference (cm)*	106 100-113)	99 (95-109)	0.007
Hip circumference (cm)	126 (121-135)	124 (116.75-128)	0.053
Waist to hip ratio	0.83 (0.8-0.88)	0.82 (0.79-0.87)	0.269
Body mass index (kg/m²)	37.4 (35.1-40.1)	35.8 (32.6-39.3)	0.095
Systolic blood tension (mmHg)*	120 (110-133)	120 (110-130)	0.809
Diastolic blood tension (mmHg)*	80 (70-80)	70 (63-80)	0.470
C-reactive protein (mg/L)	4.6 (3.3-8.8)	3.6 (2.9-6.6)	0.073
Total cholesterol (mg/dl)	197 (174-224)	209 (183-236)	0.251
HDL- cholesterol (mg/dl)*	49 (43-59)	50 (46-55)	0.994
LDL- cholesterol (mg/dl)	131 (114-151)	135 (120-157)	0.207
Triglyceride (mg/dl)*	131 (97-189)	135 (79-174)	0.190
Glucose (mg/dl)*	95 (88-105)	92 (83-95)	0.026
HbA1c (%)*	5.6 (5.4-6)	5.6 (5.2-5.8)	0.076
Calcium (mg/dl)	9.3 (9.1-9.6)	9.4 (9.1-9.5)	0.911
Phosphate (mg/dl)	3.6 (3.3-3.9)	3.8 (3.3-3.9)	0.570
Creatinine (mg/dl)	0.75 (0.7-0.82)	0.76 (0.71-0.85)	0.727
Uric acid (mg/dl)	4.9 (4-5.7)	4.9 (4.5-5.9)	0.926
TSH (U/ml)	2.4 (1.5-3.6)	2.1 (1.02-3.1)	0.136
PTH (pg/ml)	87.3 (65.5-113.8)	64.3 (41.6-95.1)	0.006
25- OH-Vit-D (ng/ml)	9.5 (6.1-13.5)	24.7 (21.3-26.3)	<0.001
HOMA-IR	3.32 (1.98-4.99)	2.33 (1.21-3.30)	0.042

HDL; High-Density Lipoprotein, LDL; Low-Density Lipoprotein, HbA1c; Glycolyzed Hemoglobin, TSH; Thyroid Stimulating Hormone, PTH; Parathormone, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance. Parameters with normal distribution are presented as the Mean±Standard deviation (Student t test). Parameters with abnormal distribution presented as median and interquartile range (Mann-Whitney U test). * Parameters used in the diagnosis of metabolic syndrome.

Table 3. Demographic and clinical parameters in obese patients with and without hyperparathyroidism

Parameters	Patients with Normal PTH value PTH<85 pg/ml n=131	Patients With Hyperparathyroidism PTH≥85 pg/ml n=132	p value
Age (years)	42.5 (36-48)	44 (35-52)	0.234
Waist circumference (cm)*	104±10	108±9	<0.001
Hip circumference (cm)	125 (119-134)	127 (122-135.75)	0.051
Waist to hip ratio	0.82 (0.79-0.86)	0.84 (0.81-0.88)	0.018
Body mass index (kg/m²)	36.8±4.8	38.3±4.1	0.006
Systolic blood tension (mmHg)*	120 (110-130)	120 (110-140)	0.001
Diastolic blood tension (mmHg)*	70 (60-80)	80 (70-90)	<0.001
C-reactive protein (mg/L)	4.6 (3.2-8.7)	4.3 (3.2-8.3)	0.584
Total cholesterol (mg/dl)	194 (172-227)	200 (176-223)	0.826
HDL- cholesterol (mg/dl)*	49 (43-57)	50 (43-58)	0.676
LDL- cholesterol (mg/dl)	132±31	131±27	0.712
Triglyceride (mg/dl)*	137 (100-192)	129 (93-184)	0.756
Glucose (mg/dl)*	95 (88-103)	95 (88-107)	0.644
HbA1c (%)*	5.6 (5.3-5.9)	5.6 (5.3-6)	0.955
Calcium (mg/dl)	9.4 (9.1-9.5)	9.3 (9.1-9.6)	0.928
Phosphate (mg/dl)	3.7 (3.3-4)	3.5 (3.3-3.9)	0.128
Creatinine (mg/dl)	0.76 (0.71-0.83)	0.75 (0.70-0.81)	0.837
Uric acid (mg/dl)	4.9±1.2	5±1.2	0.281
TSH (U/ml)	2.1 (1.4-3.1)	2.7 (1.7-3.9)	0.074
PTH (pg/ml)	64.3 (55.5-73.5)	112.5 (97.1-139.9)	<0.001
25- OH-Vit-D (ng/ml)	11.8 (8.65-17.1)	8.7 (5.1-11.8)	<0.001
HOMA-IR	3.15 (1.69-4.87)	3.30 (2.26-4.85)	0.089

HDL; High-Density Lipoprotein, LDL; Low-Density Lipoprotein, HbA1c; Glycolyzed Hemoglobin, TSH; Thyroid Stimulating Hormone, PTH; Parathormone, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance. Parameters with normal distribution are presented as the Mean±Standard deviation (Student t test). Parameters with abnormal distribution presented as median and interquartile range (Mann-Whitney U test). * Parameters used in the diagnosis of metabolic syndrome.

DISCUSSION

The present study has several important findings. Firstly, the prevalence of vitamin D deficiency is 92% among obese patients. Secondly, lower levels of vitamin D and vitamin D sufficiency rates have been observed in patients with MUO. Lastly, vitamin D deficiency is associated with higher levels of glucose and insulin resistance, while elevated PTH levels are correlated with increased waist circumference, BMI, and blood pressure.

In our country, where patients receiving vitamin D replacement are not excluded, the rate of vitamin D deficiency can reach up to 71.5% in patient groups reflecting the general population.[16] Moreover, during winter months, it has been reported that the rate of vitamin D deficiency may increase up to 83.9% in individuals whose vitamin D use is excluded.[17] Additionally, higher rates of vitamin D deficiency have been reported in female patients.[18] In studies conducted outside our region, the rates of vitamin D deficiency in obese patients vary between 40% and 80%.[19] Therefore, our data from the known obese female patient group with higher rates of vitamin D deficiency have been appropriately evaluated according to the literature.

Multiple pathophysiological mechanisms have been proposed to explain the relationship between vitamin D and MS. One of these mechanisms suggests that vitamin D deficiency affects the capacity of beta cells to convert pro-insulin to insulin, leading to decreased insulin secretion and sensitivity.[7,19] Another mechanism points to increased visceral fat mass in MS patients, which could lead to an increase in the distribution volume of vitamin D and subsequently lower serum vitamin D levels.[7,8,19] Additionally, it is claimed that individuals with both MS and obesity may have low vitamin D intake due to their dietary habits.[7,19] Other possible mechanisms include reduced exposure to sunlight, variations in gene expression of enzymes involved in vitamin D metabolism, and impaired hepatic 25-hydroxylation.[7,8,19,20] Furthermore, Osmancevic and colleagues[21] discovered that there was a negative correlation between BMI and the rise in serum vitamin D3 following UVB exposure, even after considering other variables. Lastly, another significant mechanism is the suppressor effect that active vitamin D may have on its precursor, 25-OH-D levels. Studies have shown that obese individuals tend to have higher levels of active vitamin D compared to non-obese individuals.[22] It has been suggested that the inhibitory effect of this active metabolite on the liver's synthesis of its precursor could be a contributing factor to the lower vitamin D levels observed in obesity[8]. These mechanisms are among the potential factors that may play a role in the relationship between vitamin D and MS.

Many studies that did not include all obese patients have provided inspiration for these potential pathophysiological mechanisms concerning the relationship between MS and vitamin D deficiency. In a study conducted on postmenopausal women, the rate of MS was found to be 57.8% in patients with hypovitaminosis D, whereas it was 39.8% in patients without hypovitaminosis D.[23] Another study revealed that in individuals meeting the criteria for obese MS, vitamin D deficiency was significantly higher compared to those without vitamin D deficiency (60.9% vs. 33.3% respectively).[24] Similarly, Lee et al.[25] identified that low 25(OH)D levels were associated with an increased risk of MS. Researchers, by categorizing patients into quartiles based on their vitamin D levels, demonstrated a relationship between vitamin D levels and the prevalence of MS. The lower the vitamin D levels, the higher the prevalence of increased waist circumference, hypertriglyceridemia, and elevated low-density lipoprotein cholesterol (LDL) concentrations.[25] Zhu and Heil[26] reported an association between 25(OH)D concentrations and glucose and blood lipid concentrations, indicating that low vitamin D levels were related to the presence of MS. The authors of this study revealed that every 1 ng/ml increase in vitamin D was associated with a 54% decrease in MS risk.[26]

While the relationship between vitamin D and MS syndrome has been primarily emphasized, many studies have also given importance to the association between MS and PTH, stating that individuals with MS have higher PTH levels or a higher rate of secondary hyperparathyroidism.[11-13] However, in some studies, hyperparathyroidism in obese individuals has been attributed to factors other than vitamin D deficiency, such as high body weight and obesity.[12,27-29] This is supported by findings where normal vitamin D levels or even vitamin D supplementation did not result in a significant decrease in PTH levels.[30,31] Moreover, a recent article reported that the serum vitamin D level needed to suppress PTH levels was lower in patients before and after bariatric surgery compared to non-obese patients.[32] Therefore, it can be hypothesized that the inverse relationship between PTH and vitamin D in obesity is not causal, but both biochemical abnormalities may directly result from obesity itself. In our study, while there were only statistical differences in terms of glucose and HOMA-IR between the groups with and without vitamin D deficiency, significant differences were observed in waist circumference, waist-to-hip ratio, BMI, systolic and diastolic blood pressure between the groups with and without hyperparathyroidism. These findings suggest that hyperparathyroidism may be more closely related to the criteria of MS and may be closely associated with MS and obesity in addition to vitamin D deficiency.

Our study has certain limitations. Firstly, it is a cross-sectional study, and therefore, causal relationships cannot be established. Additionally, the study only included female patients and was conducted at a single center, which may limit its generalizability to the entire population. Furthermore, the relatively small size of the population without vitamin D deficiency in our study could be considered a weakness of the study.

CONCLUSION

High rates of vitamin D deficiency were found in obese patients. In people with MUO, vitamin D levels are lower and the prevalence of vitamin D deficiency is higher. Vitamin D deficiency is associated with glucose and insulin resistance, while hyperparathyroidism is associated with a greater number of parameters associated with the MS, such as waist circumference, waist-to-hip ratio, BMI, and systolic and diastolic blood pressure. Clinicians should be alert to the presence of vitamin D deficiency and hyperparathyroidism in obese patients, as these are more common in this population. In addition, clinicians should be aware that obese patients with vitamin D deficiency and hyperparathyroidism may be at increased cardiometabolic risk due to the association of vitamin D deficiency and hyperparathyroidism with MS parameters.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Erzincan Binali Yıldırım University Clinical Researches Ethics Committee (Date: 11.05.2023, Decision No: 2023-10/2).

Informed consent: Written consent was obtained from the patient participating in this study.

Referee Evaluation Process: Externally peer reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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