ORIGINAL RESEARCH

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Does Vitamin D Level Affect Beta Cell Activity?

D Vitamini Seviyesi Beta Hücre Aktivitesini Etkiler mi?

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Abstract

Objective: The prevalence of vitamin D deficiency is ever-increasing throughout the world. Vitamin D deficiency is associated with disorders such as diabetes, insulin resistance, obesity, dyslipidemia, and hypertension. This research study was carried out to assess serum vitamin D levels of patients with type 2 diabetes, insulin resistance, obesity, and impaired fasting glucose.

Method: This study was carried out on 504 patients, who were newly diagnosed and did not receive medication before, aged 18 to 80 years, and who had type 2 diabetes, insulin resistance, obesity, and impaired fasting glucose. Their fasting glucose, fasting insulin, total cholesterol, high-density lipoprotein (HDL)-cholesterol, low-density lipoprotein-cholesterol, triglyceride, 25(OH)D concentration, height and body weight measurements were analyzed retrospectively. Body mass index, homeostatic model assessment (HOMA) for insulin resistance and HOMA beta (B) values of the patients were calculated.

Results: It was found that 89.9% of the patients with type 2 diabetes, 90.6% of the insulin resistance patients, 91.7% of the obesity patients and 94.1% of the impaired fast glucose patients had less than 30 ng/mL of serum 25(OH)D concentration. It was seen that the serum 25(OH)D concentrations of the patients were negatively correlated with their fasting insulin concentrations and HOMA B values, and positively correlated with their HDL-cholesterol levels.

Conclusion: Based on this study, vitamin D deficiency was found to be common in the patients with type 2 diabetes, insulin resistance, obesity, and impaired fasting glucose. Serum 25(OH)D concentrations were correlated with the levels of fasting insulin, HDL-cholesterol, and beta-cell function.

Keywords: HOMA B, HOMA-IR, insulin resistance, obesity, type 2 diabetes, vitamin D

Öz

Amaç: D vitamini yetersizliği tüm dünyada giderek artmaktadır. D vitamini yetersizliği diyabet, insülin direnci, obezite, dislipidemi ve hipertansiyon gibi düzensizlikler ile ilişkilidir. Bu araştırma tip 2 diyabet, insülin direnci, obezite ve bozulmuş açlık glikozu olan hastaların serum D vitamini düzeylerini değerlendirmek amacıyla yapılmıştır.

Yöntem: Bu araştırma, 18-80 yaş arası yeni tanı konulmuş ve daha önce ilaç tedavisi almamış, tip 2 diyabet, insülin direnci, obezite ve bozulmuş açlık glikozu olan 504 bireyin bulguları incelenerek yapılmıştır. Hastaların açlık glikoz, açlık insülin, total kolesterol, yüksek yoğunluklu lipoprotein (HDL)-kolesterol, düşük yoğunluklu lipoprotein-kolesterol, trigliserit, 25(OH)D konsantrasyonları, boy ve vücut ağırlığı ölçümleri retrospektif olarak analiz edilmiştir. Hastaların vücut kütle indeksi, insülin direncinin homeostatik modeli değerlendirmesi (HOMA) ve HOMA beta (B) değerleri hesaplanmıştır.

Bulgular: Tip 2 diyabetli hastaların %89,9'unun, insülin direnci hastalarının %90,6'sının, obezite hastalarının %91,7'sinin ve bozulmuş açlık glikozu hastalarının %94,1'inin serum 25(OH)D konsantrasyonlarının 30 mg/mL'nin altında olduğu bulunmuştur. Serum 25(OH)D konsantrasyonu ile açlık insülin konsantrasyonu ve HOMA B değerleri arasında negatif korelasyon, HDL-kolesterol seviyesi ile pozitif korelasyon olduğu görülmüştür.

Sonuç: Bu çalışmada tip 2 diyabet, insülin direnci, obezite ve bozulmuş glikoz toleransı hastalarında D vitamini yetersizliği sık görülmüştür. Serum 25(OH)D konsantrasyonu açlık insülin, HDL-kolesterol ve beta hücre fonksiyonu ile ilişkili bulunmuştur.

Anahtar kelimeler: HOMA B, HOMA-IR, insülin direnci, obezite, tip 2 diyabet, vitamin D

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Introduction

Vitamin D is a fat-soluble vitamin. It is stored in adipose tissue and released when needed (1). Vitamin D deficiency is quite common across the world (2). It is thought that vitamin D deficiency is linked to cardiometabolic risk factors including low levels of chronic inflammation, dyslipidemia, hypertension, insulin resistance, and obesity (3).

Serum 25(OH)D concentration is checked in order to assess the status of vitamin D in the body. How it is classified is given in Table 1 (4).

Obesity is negatively correlated with the status of vitamin D in the body (1). Possible causes of low vitamin D levels in obesity include the fact that obese people do not sufficiently take vitamin D, they get less sunlight, and thus their vitamin D synthesis through the skin is decreased (3). Vitamin D storage increases and the bioavailability of the vitamin decreases in the adipose tissue, which increases with obesity. This mechanism is effective in reducing serum vitamin D levels in obesity. Vitamin D deficiency leads to an increase in the release of the parathyroid hormone. The parathyroid hormone stimulates the transport of calcium into adipocytes and increases lipogenesis (2).

Obesity and low vitamin D levels seem to synergistically affect the risk of insulin resistance (1). There is a relationship between vitamin D deficiency and insulin resistance (2,3). There is a negative correlation between serum vitamin D concentrations and fasting insulin levels (3).

Vitamin D deficiency is associated with metabolic syndrome and type 2 diabetes (4). A change in vitamin D balance plays an important role in the development of type 2 diabetes and insulin resistance (5). Vitamin D deficiency is common in diabetes. Low serum 25(OH)D levels, which are indicative of vitamin D status in general, are negatively correlated with type 2 diabetes and impaired fasting glucose (6). The effect of vitamin D on insulin resistance can mediate the relationship between type 2 diabetes and serum vitamin D concentrations (7). People with a low serum 25(OH)D level are more prone to develop diabetes than those with a high serum 25(OH)D level (8). Among the reasons for this close relationship are the facts that vitamin D affects peripheral insulin sensitivity, that it provides cytokine activation associated with insulin resistance and that it plays an important role in maintaining pancreatic beta-cell function (9,10).

Vitamin D has an important role in glucose and insulin metabolism. Vitamin D affects pancreatic islet cells via receptors and increases insulin secretion. Vitamin D also has anti-inflammatory and immune regulatory effects. It can lead to an increase in insulin release and a decrease in insulin resistance by regulating the immune system (11).

Table 1. Classification of serum 25(OH)D concentration levels

Serum 25(OH)D concentration	
0-9.9 ng/mL	Severe insufficiency
10-19.9 ng/mL	Insufficiency
20-29.9 ng/mL	Deficiency
30-100 ng/mL	Optimal concentration
>100 ng/mL	Risk of toxicity

Homeostatic model assessment (HOMA) is a method used to evaluate insulin resistance and beta cell function. While calculating HOMA, serum fasting glucose and insulin values are used (12). HOMA of insulin resistance (HOMA-IR) is the main method used in the assessment of insulin resistance (13). HOMA-IR is calculated using HOMA-IR= [glucose (fasting) (mg/dL) × insulin (fasting) (μIU/mL)]/405 (14). HOMA beta (HOMA B) is useful for determining beta cell function (12). HOMA B is calculated using HOMA B= [insulin (fasting) (μIU/mL) × 360]/[(glucose (fasting) (mg/dL)-63] (14).

This research study was carried out to assess serum vitamin D levels of patients with type 2 diabetes, insulin resistance, obesity, and impaired fasting glucose.

Materials and Methods

This study was carried out on 504 patients aged 18 to 80 years, who had type 2 diabetes, insulin resistance, obesity and impaired fasting glucose, and who were referred by the internal medicine clinic of a hospital to the diet polyclinic. The patients included in the study were newly diagnosed and did not receive medication for their diagnosis before. Certain biochemical findings and anthropometric measurements of the patients were examined retrospectively.

The study included the patients who did not have any systemic diseases other than type 2 diabetes, insulin resistance and obesity, whose body length and weight were measured, and whose serum 25(OH)D fasting glucose, fasting insulin, total cholesterol, HDL-cholesterol, LDL-cholesterol, and triglyceride concentrations were checked.

A total of 3.220 patients' hospital files were examined during the research period. Among these patients, the number

of those who had type 2 diabetes or insulin resistance or obesity and also serum 25(OH)D concentrations recorded in files were 504 and they were included in the study. The remaining patients were not included in the study because they did not have the diseases that constituted the subject of the study, or because they also had diseases that did not constitute the subject of the study, or because they did not undergo the necessary biochemical measurements.

Body mass indexes (BMIs) of the patients referred from the internal medicine polyclinic to the diet polyclinic were calculated using their body length and weight measurements. Body mass indices were calculated using the following formula (15).

BMI=Body weight (kg)/[height (m)]^2

All biochemical measurements were made at the Central Laboratory of İstanbul Anatolian North Public Hospitals Association.

Blood glucose levels were measured by using a Roche Hitachi Modular DPP device and determined by enzymatic colorimetric measurements. Blood insulin levels were measured by using a Beckman Coulter Unicel DXI 800 device. Determination of insulin is an immunoenzymatic measurement.

Blood total cholesterol, triglyceride, and HDL-cholesterol levels were measured using a Roche Hitachi Modular DPP device. Total cholesterol and triglyceride concentrations were measured by using an enzymatic colorimetric test. HDL-cholesterol levels were measured by using a homogeneous enzymatic colorimetric assay. Finally, LDL-cholesterol levels were calculated by using the formula below (16).

LDL-cholesterol=Cholesterol-(HDL-cholesterol+triglycerides/5)

Serum 25(OH)D levels of the patients were measured by drawing blood samples on an empty stomach. Serum 25(OH)D concentrations were measured by using an Abbott/architect device and the chemiluminescent microparticle immunoassay method at the Central Laboratory of İstanbul Anatolian North Public Hospitals Association.

HOMA-IR and HOMA B values were calculated by using the fasting glucose and fasting insulin values of the patients. HOMA-IR and HOMA B were calculated by using the following formulas (14).

HOMA-IR=[glucose (fasting) (mg/dL) × insulin (fasting) (µIU/mL)]/405

HOMA B=[insulin (fasting) (µIU/mL) × 360]/[(glucose (fasting) (mg/dL)-63]

Statistical Analysis

IBM SPSS Statistics Version 22 package program was used in order to analyze the data. The mean and standard deviation values of age, height, body weight, BMI, and serum 25(OH)D concentration levels were calculated. The means and standard deviations of serum fasting glucose, fasting insulin, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, 25(OH)D concentrations, and HOMA-IR and HOMA B values were also calculated separately for the patient groups. One-Way analyses of variance were carried out to determine whether there were notable differences between the groups. Distributions of the patients according to the disease groups and serum 25(OH)D levels were determined by using calculations based on percentages. Pearson correlation analyses were carried out to determine whether the fasting glucose, fasting insulin, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglyceride, HOMA-IR, and HOMA B values of the patients were related to their ages, body weights, BMIs, and serum 25(OH)D values.

This research was evaluated and approved by the İstanbul Medipol University Non-Interventional Clinical Research Ethics Committee. Moreover, it is registered in the Chinese Clinical Trial system, which is one of the international clinical trial registry systems. Chinese Clinical Trial Registration Number: ChiCTR1900027133.

Results

This study included 504 patients with type 2 diabetes, insulin resistance or obesity. Of the patients, 385 (76.4%) were female, and 119 (23.6%) were male. The mean age of the patients was 46.02±13.7 years, their height was 162.19±8.51 cm, body weight was 88.25±16.74 kg, the average BMI was 33.65±6.22 kg/m^2, and serum 25(OH)D concentration was 16.7±10.1 ng/mL.

Table 2. Distribution of patients according to serum vitamin D levels

Level of vitamin D		N	%
0-9.9 ng/mL	Severe insufficiency	143	28.4
10-19.9 ng/mL	Insufficiency	204	40.5
20-29.9 ng/mL	Deficiency	112	22.2
≥30 ng/mL	Normal	45	8.9
Total		504	100.0

Distribution of the patients according to serum vitamin D levels is given in Table 2. Of the patients, 143 (28.4%) were found to have 0-9.9 ng/mL of serum 25(OH)D concentration, 204 (40.5%) had 10-19.9 ng/mL, 112 (22.2%) had 20-29.9 ng/mL, and 45 (8.9%) had 30 ng/mL or more serum 25(OH)D concentration.

In Table 3, the patients' mean biochemical findings are given according to their diagnoses. The mean serum 25(OH) D concentrations of all groups were at the level of 10-19.9 ng/mL, which is the insufficient level. The mean HOMA-IR value in obese patients was 1.7±0.4. The mean HOMA B value was 168.8±83.8 in patients with insulin resistance, 113.8±73.5 among obese patients. The differences between the mean fasting glucose (p=0.000), fasting insulin (p=0.000), total cholesterol (p=0.022), HDL-cholesterol (p=0.002), triglyceride (p=0.000), HOMA-IR (p=0.000) and HOMA B (p=0.000) were found to be extremely significant among the patients with type 2 diabetes, insulin resistance, obesity and impaired fasting glucose. Nevertheless, no major difference was observed between the groups in terms of the mean LDL-cholesterol (p=0.166) and mean 25(OH)D concentration (p=0.965).

Table 4 shows the distribution of the patients according to the disease groups and serum 25(OH)D levels. Serum 25(OH)D concentration of 89.9% (n=133) of patients with type 2 diabetes mellitus, 90.6% (n=135) of patients with insulin resistance, 91.7% (n=143) of obese patients and 94.1% (n=48) of patients with impaired fasting glucose were below 30 ng/mL.

Table 5 shows the results on the relationship between the biochemical findings of the patients and their age, body weight, BMI and serum 25(OH)D values. It was found that the patients' ages were positively but poorly correlated with their fasting glucose and triglyceride concentration, positively and moderately correlated with their total cholesterol and LDL-cholesterol, and negatively but moderately correlated with their fasting insulin concentration. The patients' body weights were found to be positively but poorly correlated with their HOMA-IR (Graph 1) and HOMA B values (Graph 2), moderately correlated with their fasting insulin, and negatively but poorly correlated with their total cholesterol, HDL-cholesterol and LDL cholesterol. The patients' BMIs were found to be positively but poorly correlated with their fasting insulin and HOMA-IR values (Graph 3). No

Table 3. Patients' average biochemical findings according to their diagnoses

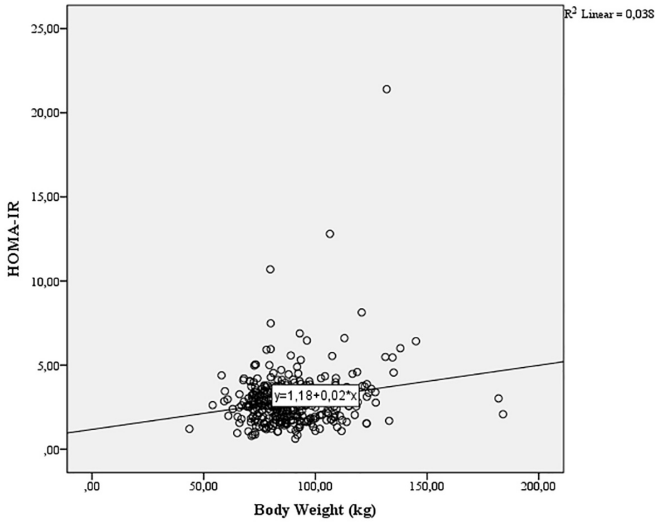
Biochemical findings	Diagnosis				
	Type 2 diabetes Mean ± SD	Insulin resistance Mean ± SD	Obesity Mean ± SD	Impaired fasting glucose Mean ± SD	p
Fasting glucose (mg/dL)	161.0±65.4	96.3±6.9	95.3±64.0	110.0±5.3	0.000*
Fasting insulin (μIU/mL)	10.7±5.3	14.4±4.3	7.9±2.7	12.6±7.1	0.000*
Total cholesterol (mg/dL)	215.0±40.9	208.7±43.3	208.9±41.0	229.0±47.9	0.022*
HDL-cholesterol (mg/dL)	44.6±11.0	47.0±12.0	49.9±11.3	46.0±11.9	0.002*
LDL-cholesterol (mg/dL)	140.8±34.5	135.6±37.0	135.6±36.7	148.0±43.4	0.166
Triglycerides (mg/dL)	162.5±80.8	138.5±97.5	121.1±62.9	165.4±66.2	0.000*
25(OH)D (ng/mL)	16.9±11.2	16.7±10.2	16.7±9.7	16.0±8.2	0.965
HOMA-IR	4.0±3.5	3.4±0.9	1.7±0.4	3.4±2.0	0.000*
HOMA B	63.5±45.4	168.8±83.8	113.8±73.5	92.8±53.8	0.000*

*One-Way ANOVA (p<0.05), SD: Standard deviation, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HOMA-IR: Homeostatic model assessment for insulin resistance, Homa B: Homeostatic model assessment beta

Table 4. Diagnosis and distribution of patients according to serum 25(OH)D levels

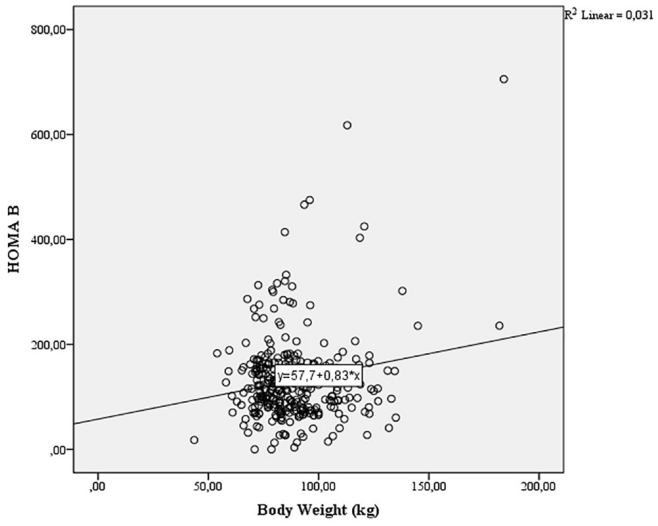
	Serum 25(OH)D								TOTAL	
	0-9.9 ng/mL		10-19.9 ng/mL		20-29.9 ng/mL		≥30 ng/mL			
Diagnosis	N	%	N	%	N	%	N	%	N	%
Type 2 diabetes mellitus	42	28.4	61	41.2	30	20.3	15	10.1	148	100.0
Insulin resistance	41	27.5	63	42.3	31	20.8	14	9.4	149	100.0
Obesity	46	29.5	58	37.2	39	25.0	13	8.3	156	100.0
Impaired fasting glucose	14	27.5	22	43.1	12	23.5	3	5.9	51	100.0

statistically significant correlation was found between the body mass indexes of the patients and their HOMA B values (Table 5) (Graph 4). A positive but weak correlation was found between the patients' serum 25(OH)D values and their HDL-cholesterol values, and a negative but weak correlation was found between their serum 25(OH)D values and their fasting insulin and HOMA B values. Serum 25(OH)D concentrations were found to be not correlated with fasting glucose concentrations (Graph 5).



Graph 1. Scatterplot for the relationship between patients' body weights and HOMA-IR values

HOMA-IR: Homeostatic model assessment for insulin resistance



Graph 2. Scatterplot for the relationship between patients' body weights and HOMA B values

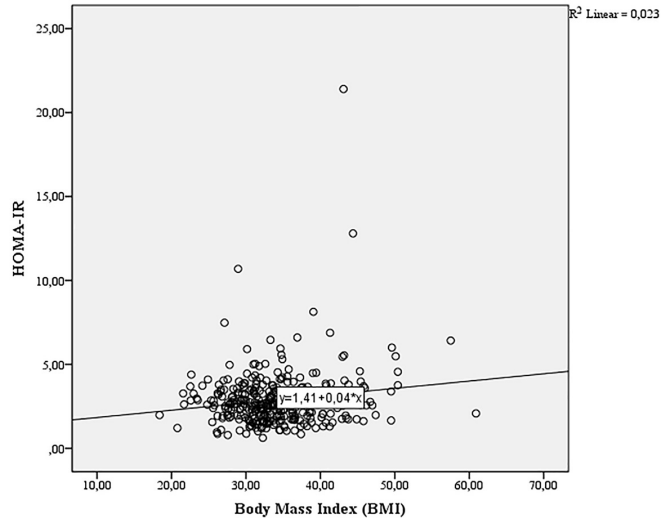
HOMA B: Homeostatic model assessment beta

Discussion

Vitamin D is thought to be a complex steroid hormonal system that regulates calcium balance and gets involved in autocrine, paracrine and endocrine processes (17). Vitamin D levels in the body are determined by measuring plasma 25(OH)D levels (3).

Vitamin D deficiency is a global social health problem and is mainly associated with various chronic diseases (3,17). In a study carried out by Schmitt et al. (18), 463 post-menopausal women were divided into 3 groups according to their serum vitamin D levels. It was found that 151 of the women (32.6%) suffered deficiency, and 164 (35.4%) were included in the group of insufficiency. In a study carried out by Diaz et al. (19), serum 25(OH)D levels of 712 people with metabolic syndrome were categorized. In this study, vitamin D deficiency was found in the majority of patients with type 2 diabetes mellitus, insulin resistance, obesity, and impaired fasting glucose. This result clearly supports previous studies in which vitamin D deficiency is highly common among such patients (4).

It is indisputable that obesity is closely related to vitamin D deficiency (3,20). Serum 25(OH)D levels of 89 overweight and obese people were analyzed in a study carried out by Kaseb et al. (21). Based on their findings, 93.2% of the overweight and obese people participating in the study were found to have serum 25(OH)D concentrations below 30 ng/mL. The mean serum 25(OH)D concentration of the people was found to be 13.8±11.36 ng/mL. In this



Graph 3. Scatterplot for the relationship between patients' body mass indexes and HOMA-IR values

HOMA-IR: Homeostatic model assessment for insulin resistance

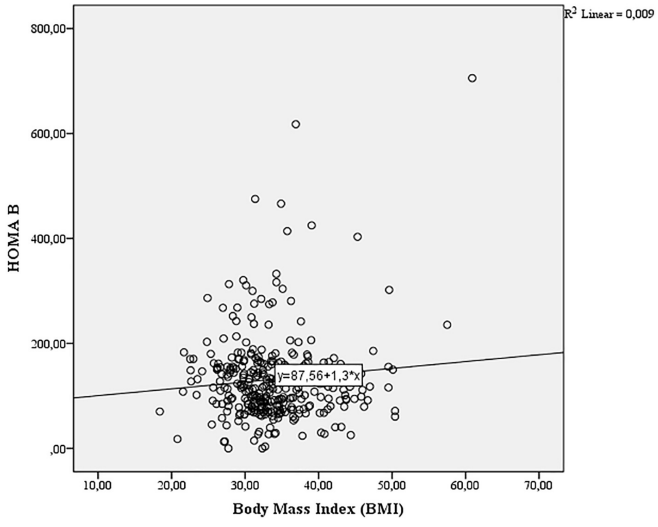
present study, the mean serum 25(OH)D concentration of the obese people was found to be 16.7±9.7 ng/mL (Table 3). Moreover, 91.7% (n=143) of the obese people had serum 25(OH)D concentration below 30 ng/mL (Table 4). Less exposure to the sun light and more covered clothing of obese individuals reduce vitamin D synthesis (3). In addition, vitamin D is stored in adipose tissue and as a result, its amount in circulation decreases (2). The causes of the negative correlation between the status of vitamin D and obesity include volumetric dilution, storage, and inactivation (1). For this reason, it is important to note that vitamin D supplementation has positive effects on body weight management (22).

There is a negative correlation between vitamin D levels in the body and insulin resistance. Low vitamin D levels increase insulin resistance (23,24). In the present study, the mean serum 25(OH)D concentration of the patients with insulin resistance was 16.7±10.2 ng/mL (Table 3). Of the patients, 27.5% (n=41) had 0-9.9 ng/mL of serum 25(OH)D concentration, 42.3% (n=63) had 10-19.9 ng/mL, 20.8% (n=31) had 20-29.9 ng/mL, and 9.4% (n=14) had 30 ng/mL or more serum 25(OH)D concentration (Table 4). In this study, it was concluded that 90.6% (n=135) of the patients with insulin resistance had vitamin D deficiency. This result supports the relationship between vitamin D deficiency and insulin resistance. Low serum 25(OH)D concentration contributes to insulin resistance and the development of diabetes through death of beta cells. Vitamin D plays an important role in reducing inflammation, which is

Table 5. Results on the relationship between the biochemical findings of the patients and their age, body weight, BMI and serum 25(OH)D values

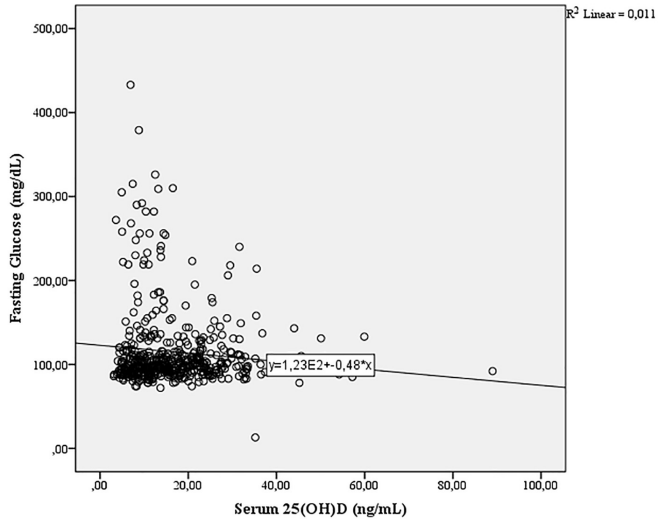
Biochemical findings	Age		Body weight		BMI		Serum 25(OH)D	
	r	p	r	p	r	p	r	p
Fasting glucose	0.153**	0.001	-0.035	0.431	-0.059	0.185	-0.080	0.073
Fasting insulin	-0.211**	0.000	0.210**	0.000	0.147**	0.008	-0.146**	0.008
Total cholesterol	0.336**	0.000	-0.133**	0.005	-0.035	0.458	0.037	0.435
HDL-cholesterol	0.066	0.169	-0.171**	0.000	-0.064	0.179	0.097*	0.042
LDL-cholesterol	0.320**	0.000	-0.110*	0.022	-0.025	0.600	0.016	0.738
Triglycerides	0.131**	0.005	0.069	0.145	-0.039	0.410	-0.026	0.585
HOMA-IR	-0.065	0.241	0.196**	0.000	0.152**	0.006	-0.101	0.068
HOMA B	-0.342	0.000	0.177**	0.001	0.094	0.089	-0.155**	0.005

BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HOMA-IR: Homeostatic model assessment for insulin resistance, HOMA B: Homeostatic model assessment beta, *Pearson correlation analysis correlation significant at the 0.05 level, ** Correlation significant at the 0.01 level



Graph 4. Scatterplot for the relationship between patients' body mass indexes and HOMA B values

HOMA B: Homeostatic model assessment beta

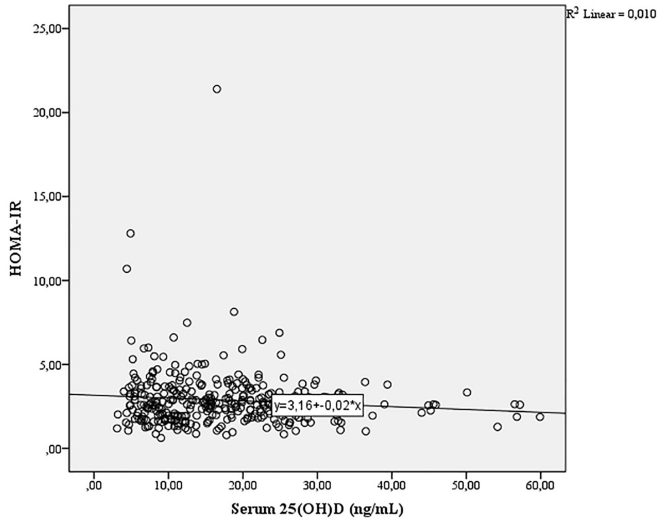


Graph 5. Scatterplot for the relationship between serum 25(OH)D values and fasting glucose values of patients

an important process in the development of insulin resistance (25). Research has associated low plasma concentration of 25(OH)D with increased serum fasting glucose concentration, increased insulin resistance and increased insulin release (23,26). A study by Sun et al. (5) has shown that vitamin D supplementation may reduce insulin resistance. In a study carried out by Gupta et al. (27), 25 patients with polycystic ovary were supplemented with 60,000 IU/week vitamin D for 12 weeks. It was found that fasting insulin, fasting glucose and insulin resistance decreased and insulin sensitivity increased at the end of the supplementation period. In a study carried out by Wang et al. (24), it was found that serum 25(OH)D concentration was negatively correlated with serum insulin levels (r=-0.209, p=0.012) and HOMA-IR (r=-0.273, p=0.001). Serum 25(OH)D concentrations of 92 healthy and overweight people were analyzed in a study carried out by Wallace et al. (28). It was found that the difference between fasting insulin values of the people with sufficient and insufficient serum vitamin D levels was not statistically significant (p>0.05). It was concluded that there was no correlation between serum 25(OH)D levels and insulin resistance. In a study of 1,514 adult people without diabetes carried out by Li et al. (29), it was determined that there was a negative correlation between serum 25(OH)D concentrations and HOMA-IR. A study conducted by Schmitt et al. (18) showed that women with low serum 25(OH)D levels had high levels of insulin and HOMA-IR (p<0.05). In a study carried out by Han et al. (30), although there was a negative correlation between serum 25(OH)D levels and HOMA-IR in men (r=-0.055, p=0.028), there was no significant correlation between these two parameters in women (r=-0.005, p=0.798). In a study carried out by Yoon et al. (31), serum vitamin D concentrations of non-diabetic people were found not to have a significant correlation with their HOMA-IR values, but to have a negative correlation with their HOMA B values. It was concluded that vitamin D was not correlated with insulin resistance, but with beta-cell function. In a study conducted, the difference between HOMA-IR and HOMA B values in diabetic patients was found to be significant in terms of serum 25(OH)D levels (p<0.05), and it was seen that there was a positive correlation between serum 25(OH) D levels and beta-cell function (32). In this study, serum 25(OH)D concentration was found not to be correlated with HOMA-IR (r=-0.101, p=0.068) (Graph 6), but to be negatively correlated with HOMA B (r=-0.155, p=0.005) (Table 5) (Graph 7).

Vitamin D deficiency is associated with insulin resistance and decreased beta-cell function (33). In a study conducted by Pan et al. (34), patients with metabolic syndrome and people without metabolic syndrome were compared in terms of serum 25(OH)D concentration levels. It was found that the serum 25(OH)D concentration of the patients with metabolic syndrome was 21.74±7.43 ng/mL, and that of the people without metabolic syndrome was 24.96±8.76 ng/mL. It was concluded that the concentration of vitamin D in serum was lower in the patients with metabolic syndrome. In a study carried out by Haidari et al. (35) the mean serum 25(OH)D concentration of the type 2 diabetes patients was found to be 11.01±5.55 ng/mL. In this present study, the mean serum 25(OH)D concentration of the patients with type 2 diabetes was found to be 16.9±11.2 ng/mL. This study supports previous research. Vitamin D deficiency or insufficiency was found in almost all of the patients with Type 2 diabetes mellitus included in this study. It was concluded that vitamin D deficiency was widespread in the patients with type 2 diabetes. Vitamin D has anti-inflammatory and insulin sensitivity support effects. Vitamin D deficiency promotes inflammation and insulin resistance, which contributes to the formation of type 2 diabetes mellitus, and as a result, the risk of developing type 2 diabetes mellitus increases (9,10).

In a study conducted by Haidari et al. (35), the relationship between serum 25(OD)D levels of patients with type 2

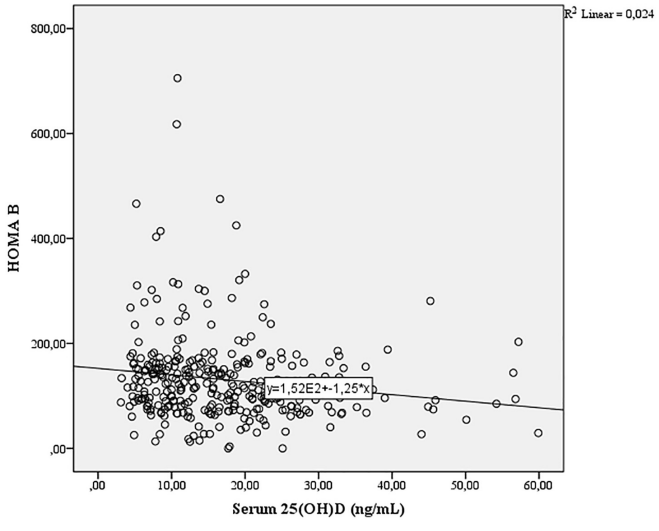


Graph 6. Scatterplot for the relationship between serum 25(OH)D values and HOMA-IR values of patients

HOMA-IR: Homeostatic model assessment for insulin resistance

diabetes and their fasting glucose, fasting insulin and HOMA-IR was examined. It was determined that vitamin D levels were negatively correlated with fasting glucose concentrations, but not significantly with HOMA-IR. In their study, Kwon and Lim (36) grouped people with prediabetes according to their serum vitamin D concentrations. No correlation was found between the mean serum fasting glucose, fasting insulin and HOMA-IR levels of the people and their serum 25(OH)D levels. In a study carried out by Srimani et al. (37), serum 25(OH)D levels and blood glucose levels were found to be negatively and significantly correlated. In this present study, the relationship between fasting glucose, fasting insulin concentrations and serum 25(OH)D concentrations was examined. Serum 25(OH)D concentrations were found to be not correlated with fasting glucose concentrations (r=-0.080, p=0.073), but negatively correlated with fasting insulin concentrations (r=-0.146, p=0.008) (Table 5). This study supports some of the results found in previous research.

Vitamin D deficiency is correlated with decreased high-density lipoprotein (HDL) levels, increased triglyceride levels, and hypercholesterolemia. Vitamin D levels can be low in people with hypercholesterolemia (3). It has been concluded in a study carried out by de Courten et al. (23) that decreased plasma 25(OH)D concentration is associated with increased triglyceride levels and decreased HDL-cholesterol levels. In a study conducted by Yang et al. (32), 97 patients with type 2 diabetes were assigned into two groups: Serum 25(OH)D concentrations below 37.5 nmol/L (n=61) and above (n=36). As a result of the study, the difference between the groups was significant in total cholesterol and HDL-cholesterol concentrations (p<0.05). In this study, a positive correlation was found between serum 25(OH)D concentration and HDL-cholesterol concentration. This may be due to the effect of vitamin D on increasing lipoprotein lipase activity, regulating calcium metabolism and decreasing parathyroid hormone activity (38).



Graph 7. Scatterplot for the relationship between serum 25(OH)D values and HOMA B values of patients

HOMA B: Homeostatic model assessment beta

Conclusion

Vitamin D is a fat-soluble vitamin and has certain functions other than regulating calcium metabolism. Vitamin D deficiency is common in society. There is a strong correlation between vitamin D deficiency and metabolic syndrome risk factors. In this study, it was seen that vitamin D deficiency was found in almost all patients with type 2 diabetes, insulin resistance, obesity and impaired fasting glucose. There was no significant difference between serum 25(OH)D concentrations of the patients with type 2 diabetes, insulin resistance, obesity and impaired fasting glucose. Serum 25(OH)D concentrations were found to be negatively correlated with serum fasting insulin concentrations and positively correlated with serum HDL cholesterol concentrations. It was determined that HOMA B, which shows beta-cell function, was negatively correlated with serum 25(OH)D concentrations. In conclusion, vitamin D deficiency was found to be common in the patients with type 2 diabetes, insulin resistance, obesity and impaired fasting glucose. Examining serum 25(OH) D concentrations of patients with type 2 diabetes, insulin resistance, obesity and impaired fasting glucose, and giving them supplementations may positively contribute to the treatment process.

Ethics

Ethics Committee Approval: This research was evaluated and approved by the İstanbul Medipol University Non-Interventional Clinical Research Ethics Committee. Moreover, it is registered in the Chinese Clinical Trial system, which is one of the international clinical trial registry systems. Chinese Clinical Trial Registration Number: ChiCTR1900027133.

Informed Consent: This study was conducted retrospectively by using the routine values of the patients.

Peer-review: Externally peer-reviewed.

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