Original Article

The Relationship Between Vitamin D and Glucose Indices and Lipid Profile in Obese Diabetic Patients

Duha T. Al-Taie[1], Eman H. Rahi[2] and Zahraa Mahmoud Hussain Al-Hejaj[2]

ABSTRACT

Objective: To examine the relationship between vitamin D levels, glycemic control values, and lipid profiles of obese diabetic patients.

Study Design: Descriptive study

Place and Duration of Study: This study was conducted at the College of Nursing, University of Basrah, Iraq from 1st January 2025 to 31st March 2025.

Methods: Ninety males of 60 diabetic patients and 30 healthy controls were enrolled. Serum levels of vitamin D, HbA1c, glucose, and the elements of the lipid profile (total cholesterol, triglycerides, low-density lipoprotein-cholesterol, high-density lipoprotein-cholesterol, and very low-density lipoprotein-cholesterol) were measured in addition to anthropometric measurements.

Results: The diabetic patients had significantly higher body mass index, Inflammation, immunology, the endocrine system, cardiovascular disease, and dyslipidemia. These are just a few of the numerous areas where vitamin D appears to play a regulatory function. Pearson correlation analysis revealed that the level of vitamin D had a significant negative relation with both HbA1c and glucose concentrations. Non-significant or weak correlations between vitamin D and lipid parameters were, however, found.

Conclusion: The possibility of poor glycemic control in obese diabetics being a factor of deficiency of vitamin D and draw attention to the possible advantages of vitamin Dmonitoring and supplementation in the treatment of diabetes.

Key Words: Vitamin D, Obesity, Diabetes mellitus, Lipid profile, Glycemic control

Citation of article: Al-Taie DT, Rahi EH, Al-Hejaj ZMH. The Relationship Between Vitamin D and Glucose Indices and Lipid Profile in Obese Diabetic Patients. Med Forum 2025;36(12):87-91. doi:10.60110/medforum.361218.

INTRODUCTION

Obesity prevention is one of the most pressing issues in modern medicine, as the rate at which obesity incidence is rising throughout the world suggests a pandemic. Obesity and type 2 diabetes mellitus are significant international health issues affecting a significant proportion of the population with a high rate of prevalence and a high correlation with cardiovascular morbidity and mortality. Obesity leads to insulin resistance, chronic low-grade inflammation, and dyslipidemia, which are core factors in the emergence and progression of T2DM and its complications.[1]

[1.] Ministry of Education, Iraq.
[2.] Department, College of Nursing, University of Basrah, Iraq.

Correspondence: Eman H. Rahi, Department of Basic Sciences, College of Nursing, University of Basrah, Iraq.
Contact No: 09647708735373
Email: eman.rahi@uobasrah.edu.iq

Received: June, 2025
Reviewed: July-August, 2025
Accepted: September, 2025

Vitamin D has long been recognized to perform extra-skeletal functions; however, of late, there has been attention on its non-skeletal extra-bone functions. There is also emerging evidence indicating this vitaminis involved in the process of glucose metabolism, insulin secretion, and insulin sensitivity by affecting pancreatic 0- cell activity and peripheral insulin sensitization and response.[2]

The effect of environmental factors and genetic predisposition on diabetes is enhanced by obesity, which overlaps with each other in its genetic as well as environmental aspects. The consequences of metabolic imbalance due to the accumulation of excess adipose tissue and food are insulin resistance, inability to autophagically break down body cells, and low-grade systemic inflammation. This causes a progressive increase in blood glucose levels and an accelerated loss of functioning β-cells.[3,4]

Recent research has shown a connection between T2DM and a lack of this vitamin.[5-7] It was pointed out that there is a connection between the diagnosis of T2DM and a lack of this vitamin. Hypovitaminosis D may contribute to insulin resistance and Diabetes Mellitus, shedding light on the disease's pathophysiology.[8] An increase in the size of the vitamin D depot and a decrease in the blood's circulating

calcidiol content can result from obesity. A lack of vitamin D is very widespread in the case of obesity and T2DM patients. It is believed that excess adipose tissue stores vitamin D, making it less biologically available, and that little sun exposure and poor diet also contribute to deficiency among this population.[9]

Vitamin D insufficiency and diabetes are both extremely common illnesses across the world.[8] Research points to a connection between the diagnosis of T2DM and a lack of this vitamin. Vitamin D can also affect lipid metabolism besides its impact on glucose metabolism. Experimental and clinical evidence indicate that vitamin D may be able to regulate lipid profiles through its ability to impact the lipoprotein lipase activity, inflammation, and the concentration of parathyroid hormone involved in lipid regulation. Low vitamin D has been linked with increased lipid profile; however, the results of studies are conflicting.[10] In low- and middle-income countries, a portion of the global population is susceptible to vitamin D deficiency because they don't eat enough foods high in vitamin D and don't get enough ultraviolet B radiation from sunshine.[11] Interest has grown significantly in recent years, with new studies concentrating on the function of vitamin D in the body.[12]

There is a need for further clinical and experimental studies on the pathophysiology of these disorders, and an attempt to understand the common mechanisms that work to develop them and increase their complications, and thus try to avoid the damage resulting from obesity and diabetes to increase the safety and efficacy of both existing and recently developed medicines. With the high rate of patients with T2DM having vitamin D deficiency, obesity, and dysmetabolic states, it is of particular clinical significance to learn how vitamin D status is linked to glucose indices and lipid profiles. The clarification of these associations can aid in the creation of prevention and treatment methods that can achieve a better metabolic outcome for obese diabetic patients. Consequently, the proposed study intends to examine the relationship between vitamin D and the glucose indexes and lipid profile among obese diabetic patients.

METHODS

The study was conducted at College of Nursing, University of Basrah, Iraq from 1st January 2025 to 31st March 2025 vide letter No. 199/5/203 dated 2/2/2025. Ninety samples in all, ranging in age from 25 to 50 years were enrolled. Before the sample was collected, the participants were given a brief explanation. The direct interviews were conducted to gather patients' medical histories and other data. Group I (control) included 30 healthy men and Group II included 60 women with diabetes mellitus. Body weight and height were among the anthropometric measurements that were taken. Using the individuals' height and weight, we calculated their BMI. We used a weight scale to determine the weight. The height was measured with a measuring stick on the weight scale.[13,14]

A serum separation tube was filled with roughly 5 milliliters of venous blood. Give it ten minutes or so to develop a clot. To extract the serum, the samples were then centrifuged for ten minutes at room temperature at 3500 rpm. The serum was kept at -20°C in a deep freezer. The COBAS INTEGRA 400 plus was used in an enzymatic colorimetric test to measure the serum. The data were analyzed using SPSS-22 and comparisons between means using least significant differences ($P \leq 0.05$). The correlation among parameters was analyzed by bivariate (Pearson) correlation.

RESULTS

The second group's body mass index (BMI) value was considerably higher than that of the control group (Table 1). The results showed that the second group's HbA1c and glucose concentration levels were noticeably higher than the control group. The findings revealed that the two groups' vitamin D levels had significantly dropped (Table 2).

Lipid profile values were significantly increased, according to the data. On the other hand, the high-density lipoprotein HDL score significantly decreased (Table 3). The associations between body mass index (BMI), glucose, triglycerides, lipid profile, and glycosylated hemoglobin were investigated using a Pearson correlation analysis. Table 4 provides a summary of the findings indicated that increasing levels of these metabolic indicators are linked to higher BMI. They demonstrated a significant positive connection with HbA1c, glucose, TG, LDL, and VLDL. Notably, vitamin D3 and BMI had a negative but non-significant correlation. Concerning glucose, TG, TC, HDL, and VLDL, HbA1c showed strong positive relationships. Moreover, HbA1c and vitamin D3 had a negative association, which means that glycaemic control and vitamin D level could be negatively related. Both TC and TG were positively correlated with glucose levels, and HDL and vitamin D3 were negatively correlated. These results are conducive to a trend of dyslipidemia related to hyperglycaemia. HDL had a high negative correlation with the majority of risk factors, such as BMI, HbA1c, and TG, in line with its cardiovascular protective effect. Vitamin D3 was inversely correlated with HbA1c and glucose, suggesting lower levels in subjects with poorer glycaemic indices, although its correlation with lipids was weak or non-significant (Table 4).

Table No. 1: Frequency of BMI level in patients and healthy individuals

Group	Mean±Standard Error
Group I (Control, N= 30)	29.83±0.85
Group II (Patients, N= 60)	36.35±0.53
P value	0.000

Table No. 2: Glycemic control comparison between groups

Group	HbA1c (%)	Glucose (mg /dL)	Vitamin D (kg/m$^{2)}$
Group I (Control)	5.85±0.08	99.11±4.42	8. 85±1.41
Group II (Patients)	9.99±0.26	255.39±13.86	3.82±0.29
P value	0.000	0.000	0.000

Table No. 3: Concentration of lipid profiles in the patients and healthy individuals

Group	TC (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
Group I (Control)	169.23±6.64	104.20±6.89	45.97±1.76	106.42±4.73	20.84±1.38
Group II (Patients)	196.36±5.18*	205.27±14.29*	24.95±1.56*	123.88±3.76*	41.06±2.86*
P value	0.002	0.000	0.000	0.007	0.000

*Significant (P≤0.05)

Table No. 4: Correlations between BMI, HBA1c, Glucose, TG, TC, HDL, LDL, VLDL, and D3

Variable	Correlation	BMI	HBA1c	Glucose	TG	TC	HDL	LDL	VLDL	D3
BMI	Pearson Correlation	1	.509**	.396**	.269*	.155	.224*	.224*	.269*	-.098
	Sig. (2-tailed)		.000	.000	.010	.146	.035	.035	.010	.357
	N	90	90	90	90	90	89	89	90	90
HBA1c	Pearson Correlation	.509**	1	.618**	.287**	.275**	.291**	.291**	.287**	-.390**
	Sig. (2-tailed)	.000		.000	.006	.009	.006	.006	.006	.000
	N	90	90	90	90	90	89	89	90	90
Glucose	Pearson Correlation	.396**	.618**	1	.269*	.344**	.182	.182	.268*	-.219*
	Sig. (2-tailed)	.000	.000		.010	.001	.087	.087	.011	.038
	N	90	90	90	90	90	89	89	90	90
TG	Pearson Correlation	.269*	.287**	.269*	1	.282**	.268*	.268*	1.000**	-.205
	Sig. (2-tailed)	.010	.006	.010		.007	.011	.011	.000	.052
	N	90	90	90	90	90	89	89	90	90
TC	Pearson Correlation	.155	.275**	.344**	.282**	1	.277**	.277**	.282**	-.186
	Sig. (2-tailed)	.146	.009	.001	.007		.009	.009	.007	.079
	N	90	90	90	90	90	89	89	90	90
DL	Pearson Correlation	-.582**	-.624**	-.336**	-.345**	-.172	-.354**	-.354**	-.345**	.344**
	Sig. (2-tailed)	.000	.000	.001	.001	.105	.001	.001	.001	.001
	N	90	90	90	90	90	89	89	90	90
LDL	Pearson Correlation	.224*	.291**	.182	.268*	.277**	1	1	.268*	-.058
	Sig. (2-tailed)	.035	.006	.087	.011	.009			.011	.591
	N	89	89	89	89	89	89	89	89	89
VLDL	Pearson Correlation	.269*	.287**	.268*	1.000**	.282**	.268*	.268*	1	-.205
	Sig. (2-tailed)	.010	.006	.011	.000	.007	.011	.011		.052
	N	90	90	90	90	90	89	89	90	90
D3	Pearson Correlation	-.098	-.390**	-.219*	-.205	-.186	-.058	-.058	-.205	1
	Sig. (2-tailed)	.357	.000	.038	.052	.079	.591	.591	.052	
	N	90	90	90	90	90	89	89	90	90

**Significance at0.01 level

DISCUSSION

The expression of vitamin D receptors (VDRs) and vitamin D-activating enzymes in pancreatic 8-cells, adipose tissue, and skeletal muscle suggests that vitamin D status can have a mechanistic relationship with metabolic regulation.[15,16] In this study, statistical comparisons demonstrated that the mean BMI of the

second group was notably higher than that of the control group, indicating that the condition diabetes associated with the second group may have influenced body weight significantly.

There are many pathophysiological pathways and complex correlations between type 2 diabetes and obesity.[17,18] This enhances the confirmed correlation between obesity and T2DM, in which adipose tissue hyperplasia supports resistance to insulin, persistent low-grade inflammation, and 8-cell dysfunction. Ruze et al[2] traced the pathophysiological role of obesity in the progression of T2DM, and its mechanisms include metabolic inflammation and ectopic fat deposition.

The findings are aligned with the existing studies that show that there are complex associations between vitamin D, obesity, glycemic control, and lipid metabolism.[19,20]

The research found that the level of glucose and HbA1c is high among diabetic patients and is accompanied by a pronounced decrease in the level of serum vitamin D. They agree with the literature that has proposed that hypovitaminosis D can affect the insulin secretion and sensitivity, worsening the glycemic control.[6] They emphasize that the absence of vitamin D influences systemic inflammation and pancreatic 8 cells and can cause the appearance of diabetes.[21-23]

Further, there were negative relationships among the vitamin D, HbA1c, and glucose levels. These negative correlations support the hypothesis that vitamin D may have some protective effect on metabolism. Wang et al[5] indicates that when vitamin D is taken, it is known to regulate insulin receptor expression and glucose transporter activity, which are known to promote insulin sensitivity and glycemic homeostasis. Also, the patients were found to have a high level of dyslipidemia. BMI and glycemic indices had a strong association with these changes. Interestingly, the correlation of HDL-C with BMI, HbA1c, and TG showed a negative correlation, which proves the inverse correlation of HDL-C with cardiometabolic risk.

Interestingly, it was observed that the weak correlations between vitamin D and lipid markers were still negative; however, the negative association between vitamin D and glycemic markers could mean that vitamin D effects are more directly exerted on glucose metabolism than on lipid profiles. This is in agreement with the findings of Klahold et al[7], who established that vitamin D supplementation was more efficient in enhancing insulin sensitivity than lipid parameters in diabetic patients. All these results are indicative that vitamin D deficiency is not only an outcome of obesity but also a possible contributing factor to the pathogenesis of T2DM and metabolic derangements related to it. This advocates the recommendation to screen and correct vitamin D, particularly in populations at risk of diabetes and obesity, as supported by the world guidelines.

CONCLUSION

A combination of unfavorable glycemic control and lipid profiles in obese diabetic patients indicates that the consideration of vitamin D status in the therapeutic management of diabetes is necessary. Further interventional studies are needed to determine whether correcting hypovitaminosis D can contribute to improved metabolic outcomes in this patient population.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Duha T. Al-Taie, Eman H. Rahi
Drafting or Revising Critically:	Duha T. Al-Taie, Zahraa Mahmoud Hussain Al-Hejaj
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

Conflict of Interest: The study has no conflict of interest to declare by any author.

Source of Funding: None

Ethical Approval: No. 199/5/203 Dated 02.02.2025

REFERENCES

1. Zakharova I, Klimov L, Kuryaninova V, Nikitina I, Malyavskaya S, Dolbnya S, et al. Vitamin D insufficiency in overweight and obese children and adolescents. Front Endocrinol 2019; 10: 103.
2. Ruze R, Liu T, Zou X, Song J, Chen Y, Xu R, et al. Obesity and type 2 diabetes mellitus: connections in epidemiology, pathogenesis, and treatments. Front Endocrinol 2023;14:1161521.
3. WHO Diabetes. Geneva: World Health Organization, 2023; 15.
4. Cho NH, Shaw JE, Karuranga S, Huang Y, da Rocha Fernandes JD, Ohlrogge AW, et al. Global estimates of diabetes prevalence for 2017 and projections for 2045. Diabetes Res Clin Pract 2018;138:271-81.
5. Wang H, Chen W, Li D, Yin X, Zhang X, Olsen N, et al. Vitamin D and chronic diseases. Aging Dis 2017;8;346-53.
6. Berridge MJ. Vitamin D deficiency and diabetes. Biochem J 2017; 474: 1321-32.
7. Klahold E, Penna-Martinez M, Bruns F, Seidl C, Wicker S, Badenhoop K. Vitamin D in type 2 diabetes: genetic susceptibility and the response to supplementation. Horm Metab Res 2020;52: 492-9.
8. deSouza CL, de Sá LBPC, Rocha DRTW, Arbex AK. Vitamin D and diabetes mellitus: a review. J Endocrine Metabol Dis 2016; 6(1): 1-7.

9. Salimova DE, Daminnov AT. A Clinical case based on the experience of treating hypertension in a patient with type 2 diabetes mellitus, obesity, and vitamin d deficiency. Educ Res Univ Sci 2023; 2(12): 150-54.
10. Maia FA, Oliveira LMM, Almeida MTC, Alves MR, De Araújo Saeger VS, Da Silva VB, et al. Autism spectrum disorder and postnatal factors: a case-control study in Brazil. Rev Paul. Pediatr 2019; 37: 398-405.
11. Roth DE, Abrams SA, Aloia J, Bergeron G, Bourassa MW, Brown KH, et al. Global prevalence and disease burden of vitamin D deficiency: a roadmap for action in low- and middle-income countries. Ann NY Acad Sci 2018; 1430: 44-79.
12. Pludowski P, Holick MF, Grant WB, Konstantynowicz J, Mascarenhas MR, Haq A, et al. Vitamin D supplementation Guidelines. J Steroid Biochem Mol Biol 2018; 175: 125-35.
13. Al-Hejaj ZMH, Al-Sudani HSK, Al-Amiri RM, Jasim MH. Evaluation of visfatin hormone level in basrah obese women. J Cardiovasc Dis Res 2021; 12(03): 1529-34.
14. Al-Hejaj ZMH, Al-Amiri RM, Jasim MH. Visfatin hormone concentration in diabetic obese women. Turkish J Physiotherapy Rehab 2019; 32: 3.
15. Lin X, Xu Y, Pan X, Xu J, Ding Y, Sun X, et al. Global, regional, and national burden and trend of diabetes in 195 countries and territories: An analysis from 1990 to 2025. Sci Rep 2020;10: 14790.
16. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med 2006; 3: e442.
17. Malone JI, Hansen BC. Does obesity cause type 2 diabetes mellitus (T2DM), or is it the opposite? Pediatr. Diabetes 2019; 20: 5-9.
18. Amzolini AM, Forţofoiu MC, Barău Abu-Alhija A, Vladu IM, Clenciu D, Mitrea A, et al. Triglyceride and glucose index: A useful tool for non-alcoholic liver disease as-sessed by liver biopsy in patients with metabolic syndrome? Rom J Morphol Embryol 2021; 62: 475-80.
19. Danese VC, Pepe J, Ferrone F, Colangelo L, De Martino V, Nieddu L, et al. The mutual interplay between bone, glucose, and lipid metabolism: The role of vitamin D and PTH. Nutrients 2023; 15(13): 2998.
20. Alzohily B, AlMenhali A, Gariballa S, Munawar N, Yasin J, Shah I. Unraveling the complex interplay between obesity and vitamin D metabolism. Sci Reports 2024; 14(1): 7583.
21. Al Refaie A, Baldassini L, Mondillo C, De Vita M, Giglio E, Tarquini R, et al. Vitamin D and dyslipidemia: is there really a link? A narrative review. Nutrients 2024; 16(8): 1144.
22. Rahi EH, Al-Hejaj ZMH, Al-Taie DT, Almusawi ZAA. The relationship between T3, T4, TSH, and vitamin D3 in obese women from a small population in Basrah city. J Biosci Appl Res 2024; 10(5): 120-26.
23. Al-Hejaj ZMH, Eman H, Tiryag AM. Assessment of mothers' knowledge and attitude about the importance of vitamin d supplements for children in Basra city. Int J Appl Sci Technol 2023; 5(4): 315-25.