

Relationship Between Insulin Resistance and Serum Vitamin D Levels in Type 2 Diabetes Mellitus Patients

Shashank Tyagi¹, Pankaj Singh Chauhan², Chandrashekhar Waghmare³

¹Professor & Head, Department of Biochemistry, SRVS Medical College, Shivpuri, Madhya Pradesh, India. ²Associate Professor, Department of Neurosurgery, Superspeciality Hospital, Shyam Shah Medical College, Rewa, Madhya Pradesh, India. ³Associate Professor, Department of Forensic Medicine and Toxicology, Government Medical College, Satna, Madhya Pradesh, India

Abstract

Background: Vitamin D deficiency is common- it has been linked with insulin resistance and inability to glucose metabolism. This study aims at exploring the relationship between serum 25-hydroxyvitamin D [25(OH)D] and HOMA-IR, or insulin resistance, in individuals with type 2 diabetes. **Material and Methods:** A cross-sectional study was designed on 200 individuals with type 2 diabetes in a hospital. Serum 25(OH)D levels were classified as deficient (<20 ng/mL), insufficient (20-29.9 ng/mL), and sufficient (\geq 30 ng/mL) according to clinical recommendations. HOMA-IR was calculated using fasting plasma glucose and insulin levels. Pearson/Spearman correlation, ANOVA across vitamin D categories, and multivariable linear regression were used to control for age, sex, BMI, diabetes duration, and HbA1c. **Results:** The average age was approximately 52 years, and vitamin D insufficiency was widespread. Serum 25(OH)D had a substantial inverse connection with HOMA-IR (Pearson $r = -0.231$, $p = 0.001$; Spearman $\rho = -0.239$, $p < 0.001$). In adjusted regression, 25(OH)D remained independently linked with decreased HOMA-IR ($\beta = -0.051$, $p = 0.041$). **Conclusion:** Lower levels of 25(OH)D were substantially related with increased insulin resistance in T2DM patients. Vitamin D insufficiency screening and correction may be considered as part of comprehensive metabolic care, however interventional trial data is equivocal.

Keywords: Vitamin D, 25(OH)D, insulin resistance, HOMA-IR, type 2 diabetes mellitus.

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INTRODUCTION

Chronic hyperglycemia and a higher risk of microvascular and macrovascular complications are the outcomes of type 2 diabetes mellitus (T2DM), which is characterized by insulin resistance and β -cell dysfunction.^[1] Worldwide, vitamin D deficiency is widespread, and research on its extra skeletal effects—such as insulin sensitivity and glucose metabolism—is growing.^[1,2]

Skeletal muscle, adipose tissue, and pancreatic β -cells all contain vitamin D receptors, suggesting a possible role in the release and action of insulin. Insulin resistance and blood 25-hydroxyvitamin D [25(OH) D] levels are inversely correlated, according to observational research. Reduced β -cell function and elevated insulin resistance are two consequences of hypovitaminosis D.^[3]

This association has been further supported by prospective cohort studies. Baseline serum 25(OH) D concentrations were found to predict future insulin resistance and glycemic status by Forouhi et al.^[4] Although causality is unclear, systematic reviews and meta-analyses have also discovered an inverse connection between circulating 25(OH)D levels and the incidence of type 2 diabetes mellitus.^[5,6] In diabetic populations, lower vitamin D levels have been associated with β -cell dysfunction and impaired insulin sensitivity.^[7] The outcome of intervention studies on vitamin D supplementation has been conflicting, as has the outcome of a number of geographical regions indicating a continuing

relationship between glucose regulation issues and insufficiency of vitamin D.^[8] Some studies revealed no difference in high-risk individuals' glycemic indices and insulin resistance, whereas others reported slight improvements.^[9] Given the significant incidence of vitamin D deficiency in adults, especially T2DM patients, more research into its association with insulin resistance is clinically important.^[10]

As a result, the current study was conducted to assess the relationship between serum 25-hydroxyvitamin D [25(OH)D] levels and insulin resistance, as measured by the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), among patients with type 2 diabetes mellitus at a tertiary care hospital.^[11]

Aim:

To evaluate the connection between insulin resistance (HOMA-IR) and serum 25(OH) D in diabetes mellitus patients admitted to a tertiary care facility.

Address for correspondence: Dr. Chandra Shekhar Waghmare, Associate Professor, Department of Forensic Medicine and Toxicology, Government Medical College, Satna, Madhya Pradesh, India.
E-mail: drskeharr05@gmail.com

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MATERIALS AND METHODS

This is a Hospital-based cross-sectional observational study conducted in a tertiary care center. A total of 200 adults with confirmed T2DM were included using consecutive sampling.

Inclusion criteria

- Age ≥ 18 years
- Diagnosed T2DM on treatment as per standard criteria
- Patients who provide written informed consent for the study

Exclusion criteria

- Age < 18 years
- Chronic kidney disease stage ≥ 3 , chronic liver disease, malabsorption
- Current high-dose vitamin D therapy (therapeutic doses)
- Patients who not provide written informed consent for the study

Data collection: Data were collected using a structured proforma. Demographic details including age and sex was recorded, and body mass index (BMI) was measured. Clinical information regarding the duration of diabetes mellitus was noted. Biochemical parameters assessed included HbA1c, fasting plasma glucose, fasting insulin, and serum 25-hydroxyvitamin D [25(OH)D]. Vitamin D status was classified as deficient (< 20 ng/mL), insufficient (20–29.9 ng/mL), and sufficient (≥ 30 ng/mL). HOMA-IR, or the

Homeostatic Model Assessment of Insulin Resistance, was used to calculate insulin resistance.

Statistical analysis: Statistical analysis was performed using appropriate statistical software. Continuous variables were measured in terms of mean and standard deviation and frequencies and percentages were used to measure the categorical variables. Pearson and Spearman tests of correlation were used to investigate the correlation between serum 25(OH)D and HOMA-IR. One-way ANOVA was applied in comparing the vitamin D categories. Multivariable linear regression analysis was performed with HOMA-IR as the dependent variable. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 200 diabetes mellitus patients were enrolled and analysed in this study. The majority of patients belonged to the 50–59 years age group (32%), followed by 40–49 years (29%). Males constituted 52% of the participants, while females accounted for 48%. Most subjects were married (86%) and resided in urban areas (59%). With respect to education, 31% had secondary education and 22% were graduates or above. More than half of the participants belonged to the middle socioeconomic class (54%), indicating a predominance of middle-income urban patients in the study cohort [Table 1].

Table 1: Socio-demographic characteristics of study participants (n = 200)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	<40	32	16%
	40–49	58	29%
	50–59	64	32%
	≥ 60	46	23%
Gender	Male	104	52%
	Female	96	48%
Marital status	Married	172	86%
	Unmarried	28	14%
Residence	Urban	118	59%
	Rural	82	41%
Educational status	Illiterate	38	19%
	Primary	56	28%
	Secondary	62	31%
	Graduate & above	44	22%
Occupation	Homemaker	54	27%
	Unskilled worker	48	24%
	Skilled worker	42	21%
	Professional	28	14%
	Retired/Unemployed	28	14%
Socioeconomic status	Lower	46	23%
	Middle	108	54%
	Upper	46	23%

Based on serum 25-hydroxyvitamin D levels, the majority of participants were vitamin D deficient (61%), followed by insufficient (32.5%), while only 6.5% had sufficient vitamin D levels. Patients with vitamin D deficiency had higher mean BMI (27.6 ± 3.9 kg/m²), higher HbA1c levels ($8.4 \pm 1.4\%$), and greater mean values for HOMA-IR (5.57 ± 2.80) in

comparison to individuals with adequate and inadequate vitamin D levels. Although HOMA-IR values showed a decreasing trend with improving vitamin D status, the difference across vitamin D categories did not reach statistical significance on one-way ANOVA ($p = 0.085$).

Table 2: Baseline characteristics correlated with the vitamin D status (n=200)

Vitamin D status	n	Age (years)	Male (%)	BMI (kg/m ²)	HbA1c (%)	25(OH)D (ng/mL)	HOMA-IR
Deficient (< 20)	122	52.1 ± 10.3	54.9	27.6 ± 3.9	8.4 ± 1.4	13.3 ± 4.8	5.57 ± 2.80
Insufficient (20–29.9)	65	51.6 ± 9.1	53.8	26.0 ± 4.0	8.2 ± 1.5	23.9 ± 2.6	4.99 ± 3.03
Sufficient (≥ 30)	13	49.1 ± 11.0	38.5	24.5 ± 4.4	8.6 ± 1.1	33.9 ± 3.0	3.92 ± 1.68

Correlation analysis demonstrated a significant inverse relationship between serum 25(OH)D levels and insulin resistance. Pearson’s correlation showed a moderate negative correlation between vitamin D levels and HOMA-IR ($r =$

$-0.231, p = 0.001$). This finding was further supported by correlation analysis ($\rho = -0.239, p < 0.001$), indicating that lower vitamin D levels were associated with higher insulin resistance.

Table 3: Correlation between serum 25(OH) D and insulin resistance

Correlation	Vitamin D vs. HOMA-IR	P-Value
Pearson (r)	-0.231	0.0010
Spearman (ρ)	-0.239	0.0006

Serum 25(OH)D levels remained independently associated with HOMA-IR after adjusting for age, sex, BMI, duration of diabetes, and HbA1c ($\beta = -0.051, p = 0.041$). Body mass index emerged as the strongest predictor of insulin resistance

($\beta = 0.274, p < 0.0001$). The overall regression model explained approximately 21% of the variability in HOMA-IR ($R^2 \approx 0.21$).

Table 4: Multivariable linear regression for HOMA-IR (dependent variable = HOMA-IR)

Predictor	Beta (SE)	95% CI	P-Value
25(OH)D (ng/mL)	-0.051 (0.025)	-0.101 to -0.002	0.0413
Age (years)	0.000 (0.019)	-0.036 to 0.037	0.9800
Sex (male)	0.307 (0.377)	-0.436 to 1.050	0.4161
BMI (kg/m ²)	0.274 (0.047)	0.180 to 0.368	<0.0001
Duration of DM (years)	-0.111 (0.064)	-0.238 to 0.016	0.0859
HbA1c (%)	0.259 (0.133)	-0.003 to 0.522	0.0525

A scatter plot demonstrating the inverse relationship between insulin resistance (HOMA-IR) and serum 25-hydroxyvitamin D levels [Figure 1].

documented to increase the expression of the insulin receptors and insulin responsiveness, increasing insulin sensitivity.^[15] Chronic low-grade inflammation is a symptom of insulin resistance and type 2 diabetes mellitus. Vitamin D has anti-inflammatory effects, and it has been reported to inhibit the production of pro-inflammatory cytokines and promote the actions of the anti-inflammatory pathways.^[16] Hypovitaminism of 25 (OH) D could thus independent of age, sex, body mass index, diabetes duration, and HbA1c, be relevant in increasing the inflammatory processes that cause insulin resistance. In the current study, multivariate linear regression revealed that serum levels of 25 (OH) D were independent of insulin resistance even after controlling age, sex, body mass index, diabetes duration, and HbA1c. The body mass index proved to be a powerful predictor of the insulin resistance and this has been in line with the known facts attributing adiposity to poor insulin sensitivity.^[17] This indicates that T2DM in the context of vitamin D deficiency may be synergistic with obesity in increasing insulin resistance in the patients. Interventional studies which evaluated the effects of vitamin D supplementation in increasing insulin resistance in T2DM patients have produced differing results. There are randomized controlled trials which showed some modest improvements in insulin sensitivity especially with insulin deficient in the baseline.^[18] Other trials have however not reported any notable metabolic advantages particularly because of differences in dose of supplementation, period of supplementation, pre-testing vitamin D positioning and features of the population.^[19] These contradictions suggest that vitamin D supplementation does not have a consistent effect on all T2DM patients and indicates the need to assess them individually. Nonetheless, the current study contributes to the existing range of literature regarding the role of vitamin D deficiency in insulin resistance in type 2 diabetes mellitus. The cross-sectional design does not allow the causal inference, but the results highlight the necessity of longitudinal and interventional research directed at the groups of diabetic patients having vitamin-D-deficiency to

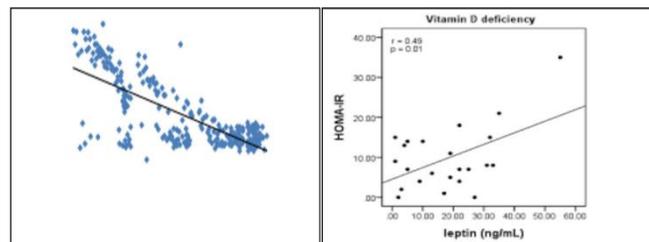


Figure 1: Scatter plot: Serum Vitamin D vs. HOMA-IR

DISCUSSION

In the current cross-sectional analysis, negative relation was found between 25-hydroxyvitamin D [25(OH)D] serum level and insulin resistance, which was determined through HOMA-IR in individuals with diabetes mellitus type 2. The subjects were found to have increased HOMA-IR among subjects with reduced vitamin D status, meaning that insulin resistance aggravates as regards lower vitamin D levels. This observation corroborates the increasing body of evidence that is associated with vitamin D deficiency and poor metabolic profiles in diabetic groups. A number of epidemiological studies have reported the similarity in relation to low insulin resistance. The results of the population-based studies suggest that vitamin D deficiency might play a role in insufficient insulin-signaling and decreased glucose uptake in peripheral tissues.^[12,13] These results indicate the possible metabolic undoing of vitamin D to its traditional bone-related impacts. The biological process of such relationship is multifactorial. Vitamin D is also known to have effect on intracellular calcium levels, which are also required to carry out insulin-induced glucose uptake in muscle and adipose tissue.^[14] Also, vitamins D have been

establish whether the deficiency correction can positively modify insulin resistance and glycemic results.^[20]

CONCLUSION

The measured report had discovered that there was a great inverse correlation between the level of serum vitamin 25-hydroxy and insulin resistance in patients with type 2 diabetes mellitus with level. Vitamin D deficiency was common and it was correlated with increased scores on HOMA-IR. The findings suggest that in the general metabolic assessment of T2DM patients, measuring vitamin D status could be useful. Vitamin D deficiency screening and treatment can be included in the overall diabetes treatment, but more prospective and interventional research after that is required to confirm causality and treatment efficacy.

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Conflicts of interest

There are no conflicts of interest.

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