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Association of Vitamin D with oxidative stress in patients with Type 2 diabetes mellitus

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is a non-communicable disease, manifesting hyperinsulinemia, insulin resistance, hyperglycemia, and low-grade chronic inflammation associated with various micro and macrovascular complications. The present study aimed to estimate vitamin D (Vit D) levels, total antioxidant capacity, and malondialdehyde (MDA) levels in T2DM patients compared with healthy individuals. In addition, we assessed Vit D, total antioxidant capacity, and MDA levels in patients with T2DM and their association with HbA1c, insulin resistance and lipid profile parameters.

Methods: Seventy patients with T2DM aged 35 to 50 years were selected and 70 healthy age-matched subjects were selected as controls. Serum Vit D and insulin were estimated by the enzyme-linked immunosorbent assay (ELISA). Glycosylated hemoglobin (HbA1C) was assessed by high-performance liquid chromatography (HPLC) method and other routine lipid profile investigations were carried out using a Beckman Coulter fully automated analyzer.

Results: Vitamin D levels significantly decreased in T2DM patients. HbA1C and insulin resistance values are significantly increased in type 2 diabetic patients. Vitamin D levels negatively correlated with MDA, insulin resistance, and HbA1c, while positively correlated with total antioxidant capacity. Nevertheless, there is no significant correlation between lipid profile parameters.

Conclusion: Vitamin D deficiency may be one of the vital risk factors responsible for increased oxidative stress in patients with T2DM. Regular monitoring and supplementation of Vit D are beneficial for the reduction of oxidative stress and vascular complications in these patients.

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Introduction

Diabetes mellitus is a non-communicable disease that is a global health burden. Further, it was estimated in 2019 as the 9th largest cause of mortality and prevalent in all income levels (1-3). Vitamin D (Vit D) has been reported to regulate cellular proliferation, differentiation, and immune modulation (4). Several studies demonstrated that vit D is recognized for its anti-rachitic properties and protective effects against various diseases, including diabetes, hypertension, cardiovascular, autoimmune and dermatological diseases and cancer (5,6). Vit D and 25 dihydroxy vit D [25(OH)2D] active hormonal form, 1,25-dihydroxyvit D [1,25(OH)2D] are crucial for human biological and physiological functions, such as slow down inflammation and to reduce the intracellular oxidative stress (7,8). Vit D is one of the key controllers of systemic inflammation, oxidative stress and mitochondrial respiratory function, and thus, the aging process in humans. In turn, molecular and cellular actions form 1,25(OH)2D slow down oxidative stress, cell and tissue damage, and the aging process. On the other hand, hypovitaminosis D impairs mitochondrial functions, and enhances oxidative stress and systemic inflammation. Sufficient or adequate Vit D is beneficial to suppress the peroxides, Peroxyacyl nitrates and improves mitochondrial and endocrine functions, reducing the risks of vascular disorders, such as autoimmunity, infections, metabolic derangements, and impairment of DNA repair; all of this aids a healthy, graceful aging process (9-11).

Vit D further increases the expression of glutathione peroxidase and converts the ROS molecule H2O2 to water. Vit D also effect the formation of glutathione through activation of the enzyme glucose-6-phosphate dehydrogenase which down regulates nitrogen oxide (NOx), a potent precursor for generating ROS that converts O2- to H2O2 and upregulating superoxide dismutase (SOD). These vit D-related actions collectively reduce the burden of intracellular ROS (12-14).

Research studies suggest that higher Vit D levels are associated with a lower risk of insulin resistance, a condition in which the body is unable to respond to or effectively use the insulin it produces. Vit D metabolites stimulate the immune response in children and adults, thereby having a crucial role in defense against pathogens (15).

Oxidative stress occurs when the rate of free radical generation exceeds the capacity of antioxidant defense systems, leading to the toxic effects of free radicals (16,17). Oxidative stress is a potent risk factor for vascular complications in diabetes, and insulin resistance induces pathophysiologic changes in diabetes mellitus (18-20). So, the aim of this study was to investigate Vit D, total antioxidant capacity, malondialdehyde (MDA) levels in patients with T2DM and their association with HbA1c, insulin resistance and lipid profile parameters.

Methods

In this case-control study, seventy patients with T2DM, aged 35-50 years of age, were enrolled from August 2022 to April 2023. These patients, who were on oral hypoglycemic drugs, attended Government General Hospital attached to Siddhartha Medical College, Vijayawada, Andhra Pradesh, India. Patients on insulin, thyroid disorders, smokers, alcoholics, tobacco chewers, other active infective diseases, neoplastic disorders, liver dysfunction, history of myocardial infarction, stroke, and occlusive peripheral vascular disease were excluded from the study. Seventy healthy age- and sex-matched subjects were selected as control. Written informed consent was obtained from all subjects, and the study was approved by the Institutional Human Ethics Committee (IHEC) (IEC-SMCGGH/2024/AP/018). Experiments were conducted in accordance with the Helsinki Declaration.

Fasting blood samples were obtained from the subjects and centrifuged at 2,000 g for 10 min. Samples were analyzed for glucose, lipid profile (Total Cholesterol, HDL-C, LDL-C, and triglycerides) using a Beckman Coulter fully automated analyzer. Vit D and insulin were assessed by Enzyme-linked immunosorbent assay (ELISA). Moreover, HbA1c was assayed with HPLC method. The total antioxidant capacity was estimated by Benzie et al. method (21), and lipid peroxidation using spectrophotometry for MDA quantification (22). Homeostasis model assessment for insulin resistance evaluation (HOMA-IR) was calculated by fasting plasma insulin × glucose/22.5 (23).

Statistical analyses were carried out by SPSS software version 25.0. The values were expressed as mean ± standard deviation (SD) using T-test. The Pearson correlation test was used for correlation analysis. P-value <0.05 was considered statistically significant.

Results

Table 1 presents the comparison of baseline parameters between the control group and patients with type 2 diabetes mellitus (T2DM). The mean age did not differ significantly between the two groups. However, body mass index (BMI), systolic blood pressure (BP), and diastolic BP were significantly higher in the T2DM group compared to controls ($p < 0.05$ or $p < 0.001$). Waist-to-hip ratio showed no significant difference between the groups. The average duration of diabetes among T2DM patients was 6.1 ± 2.0 years.

Table 2 compares fasting and postprandial glucose levels, lipid profile, HbA1c, insulin levels, HOMA-IR, vit D, total antioxidant capacity, and MDA between the control and diabetic groups. Fasting plasma glucose (FPG), postprandial plasma glucose (PPG), HbA1c, insulin, HOMA-IR, total cholesterol, serum triglycerides, LDL cholesterol, and MDA levels were significantly increased in T2DM patients ($p < 0.05$ or $p < 0.001$).

In contrast, HDL cholesterol, vit D levels, and total antioxidant capacity were significantly decreased in diabetic patients compared to controls ($p < 0.001$). The mean vit D levels in the control group and patients with T2DM were 35 ng/ml and 20.8 ng/ml, respectively (Figure 1).

Table 1. Comparison of baseline parameters in the control and patients with T2DM

Parameters	Controls (n=70)	T2DM patients (n=70)	P-value
Age	41.7±3.9	43.1±4.7	0.07
Body mass index (BMI)	24.5±1.3	26.9±3.6 [a*]	0.001*
Waist/Hip ratio	0.92±0.03	0.92±0.06	0.08
Systolic BP (mmHg)	112.9±7.1	125.5±15.5 [a#]	0.04 #
Diastolic BP (mm Hg)	75.1±3.4	81.7±7.5 [a#]	0.05 #
Duration of T2DM (years)	-	6.1±2.0	NA

Data are expressed as mean ± SD, * $p < 0.001$, # $p < 0.05$ was considered statistically significant. BP: Blood Pressure. T2DM: Type 2 Diabetes Mellitus.

Table 2. Comparison between fasting plasma glucose, and postprandial glucose, lipid profile, HbA1c, insulin, HOMA-IR, Vitamin D, total antioxidant capacity, and MDA levels in the controls and patients with T2DM

Parameters	Controls (n=70)	T2 DM patients (n=70)	P-value
Fasting plasma glucose (mg/dl)	89.3±10.2	178.7±7.9	0.001*
Post prandial plasma glucose (mg/dl)	110.3±9.4	210.1±23.6	0.001*
Total cholesterol	181.5±8.9	223.5±16.9	0.04#
Serum triglycerides (mg/dl)	112.6±15.7	201.8±12.8	0.03#
HDL cholesterol (mg/dl)	43.1±8.5	35.6±5.8	0.03#
LDL cholesterol (mg/dl)	122.6±16.2	172.8±16.7	0.001*
HbA1c %	5.3±0.8	9.6±1.9	0.001*
Insulin (µ IU/ml)	8.0±1.6	17.8±3.2	0.001*
HOMA-IR	1.8±0.15	6.1±2.1	0.001*
Vitamin D (ng/ml)	35±2.9	20.8±4.8	0.001*
Total antioxidant capacity (µmol/l)	436.4±25.7	348.4±25.7	0.001*
MDA (µmol /l)	2.8±0.5	5.3±1.7	0.001*

Data are expressed as mean ± SD, * $P < 0.001$, # $P < 0.05$ was considered statistically significant. HD: High-Density Lipoprotein; HOMA-IR: Homeostasis Model Assessment for Insulin Resistance; LDL: Low-Density Lipoprotein; MDA: Malondialdehyde

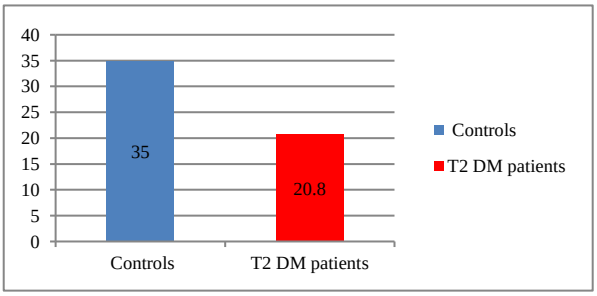

Figure 1. Vitamin D levels in the controls and patients with T2DM

Table 3 illustrates the correlation between vit D levels and various biochemical parameters in both T2DM patients and controls. Vit D showed a significant positive correlation with total antioxidant capacity and a significant negative correlation with MDA, HbA1c, and HOMA-IR in both groups ($p < 0.05$ or $p < 0.01$). However, no significant correlations were observed between vit D and lipid profile parameters, including total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol.

Table 3. Correlation between Vitamin D and measured parameters in the patients with T2DM and controls

Parameters	Correlation Coefficient-r -T2 DM group	P-value	Correlation Coefficient-r -Control group	P-value
Total antioxidant capacity	0.378	0.04*	0.342	0.04*
MDA	- 0.413	0.01*	- 0.378	0.01*
HbA1c	- 0.378	0.02*	- 0.321	0.02*
HOMA-IR	- 0.407	0.01**	- 0.413	0.01**
Cholesterol	0.227	0.07	0.187	0.07
TG	0.178	0.06	0.121	0.06
HDL-C	- 0.215	0.07	- 0.201	0.07
LDL-C	0.300	0.06	0.282	0.06

*Correlation is significant at the 0.05 level (2-tailed).
**Correlation is significant at the 0.01 level (2-tailed). TG: Triglyceride.

Discussion

T2DM is associated with insulin resistance, a defective response to physiological or increased exogenous or endogenous insulin concentration, leading to hyperglycemia and hyperinsulinism. Insulin not only regulates the metabolism but also acts as a growth factor. Hyperinsulinism stimulates abnormal activation of multiple cellular signaling cascades and strengthens growth factor-dependent cell proliferation (24,25). In the present study, we observed significant reduction of Vit D levels in patients with T2DM compared with healthy controls. Studies have reported that Vit D may have a pivotal role in insulin sensitivity through a different mechanism, including an increase in the transcriptional activation and expression of insulin receptor genes, which promotes basal and insulin-stimulated glucose oxidation, thereby improving insulin sensitivity (26,27). Nazarian et al. reported that Vit D3 supplementation is beneficial to improve insulin sensitivity in subjects with impaired fasting glucose levels (28).

The present study also demonstrates that Vit D levels negatively correlate with insulin resistance and HbA1c. One of the experimental studies revealed Vit D levels could promote the synthesis and secretion of insulin in the pancreas of mice (29). Vit D restores glucose-stimulated insulin secretion by promoting β-cell survival due to modulation of cytokines (30,31). Further insulin secretion is also influenced by calcium concentration and flux through β cells (32). Vit D regulates the function of calbindin, a systolic calcium-binding protein found in pancreatic β-cells, and acts as a modulator of depolarization-stimulated insulin secretion via regulation of intracellular calcium. Parathyroid Hormone (PTH), which is regulated by vit D, is associated with insulin synthesis and secretion in the pancreas (33). It means that decreased Vit D level is one the confounding factors for increased insulin resistance and elevated HbA1c levels in patients with T2DM.

The present reveals significant oxidative stress in patients with T2DM, as assessed through the evaluation of total antioxidant capacity and lipid peroxidation (MDA) assessment. Vit D levels positively correlated with total antioxidant capacity, whereas negatively correlated with MDA levels. A previous study demonstrated that Vit D deficiency caused insulin resistance by causing oxidative stress in hepatocytes (34). Vit D could play a role in increasing some anti-inflammatory cytokines while decreasing the production of some pro-inflammatory cytokines. Depletion of Vit D and stable silencing of 1α (OH)ase in L02 hepatocytes led to a significant production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), along with subsequent p53-p21 activation and DNA damage (35-38). Therefore, decreased Vit D levels are crucial to promoting oxidative stress in patients with T2DM.

Conclusion

Vit D deficiency may be one of the vital risk factors responsible for increased oxidative stress in patients with T2DM. Regular monitoring and supplementation of Vit D are beneficial for the reduction of oxidative stress and vascular complications in these patients.

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Ethical statement

Institutional Human Ethics Committee (IHEC) (IEC- SMCGGH/2024/AP/018) approved this study.

Conflicts of interest

The Multi-Disciplinary Research Unit (MRU), Siddhartha Medical College, Vijayawada, supported this study. Further thankful to everyone who has helped in this study.

Author contributions

Balu Mahendran Kanumuru: Data collection and processing, Manuscript preparation. Sridevi Nutakki: Study design, validation, resources and formal analysis, and statistical analysis.

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