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Original Research Article

Vitamin D Status and Cardiovascular Disease Risk Factors in Type 2 Diabetes Mellitus Patients Attending the University of Maiduguri Teaching Hospital, Maiduguri, Nigeria

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Abstract: This study investigated the relationship between serum vitamin D levels and cardiovascular disease risk factors in Type 2 Diabetes Mellitus (T2DM) patients attending the University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria. A hospital-based cross-sectional design was employed, enrolling 70 confirmed T2DM patients and 70 age-matched control participants. Biochemical assessment included serum 25-hydroxyvitamin D [25(OH)D] by ELISA, fasting plasma glucose (FPG) by glucose oxidase method, glycated haemoglobin (HbA1c) by chromatographic method, full lipid profile, and albumin-adjusted calcium. Statistical analyses comprised independent samples t-test, Pearson correlation, logistic regression, and multiple linear regression. T2DM patients had markedly lower mean vitamin D levels (19.8±9.2 vs 26.4±10.1 ng/mL, $p<0.001$), with deficiency (<20 ng/mL) prevalent in 62.9% versus 31.4% of controls. Vitamin D deficiency conferred 2.68-fold higher odds of T2DM after full adjustment (95% CI: 1.08–6.65, $p=0.033$). Among T2DM patients, significant inverse correlations were established between vitamin D and FPG ($r = -0.396$, $p<0.001$), HbA1c ($r = -0.361$, $p=0.002$), BMI ($r = -0.324$, $p=0.006$), total cholesterol ($r = -0.301$, $p=0.011$), LDL-C ($r = -0.284$, $p=0.016$), and triglycerides ($r = -0.257$, $p=0.032$), while positive correlations were found with HDL-C ($r = 0.245$, $p=0.041$) and albumin-adjusted calcium ($r = 0.312$, $p=0.008$); none of these correlations were significant in controls. These findings demonstrate that vitamin D deficiency is significantly and independently associated with adverse cardiovascular risk factor profiles in T2DM patients, supporting routine vitamin D screening in this population and highlighting the need for further longitudinal and interventional research.

Keywords: Vitamin D Deficiency, Type 2 Diabetes Mellitus, Cardiovascular Risk Factors, Dyslipidaemia, Glycaemic Control, Insulin Resistance, Nigeria.

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1. INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder arising from absolute or relative deficiency of insulin production by the pancreatic beta cells. Impaired insulin secretion disrupts glucose metabolism and results in hyperglycaemia, with attendant disturbances of carbohydrate, fat, and protein metabolism (ADA, 2013; WHO, 2019). Type 2 Diabetes Mellitus (T2DM), characterised by pancreatic β-cell dysfunction and peripheral insulin resistance, has emerged as a major non-communicable disease of global concern, with an estimated 462 million individuals affected worldwide, accounting for over 90% of all diabetes cases and more than one million deaths in 2017 alone (Galicia-Garcia *et al*., 2020).

In sub-Saharan Africa, Nigeria bears the highest burden of diabetes in the region, with approximately 3.9 million adults aged 20–79 years living with the disease (Dahiru *et al*., 2016). The pooled DM prevalence in Nigeria is 5.77%, meaning approximately 11.2 million Nigerians—one in every 17 adults—are living with DM (Uloko *et al*., 2018). The International Diabetes Federation estimated total health expenditure due to diabetes in Nigeria at $3.3 billion in 2017 alone (Mapa-Tassou *et al*., 2019). Maiduguri, the capital of Borno State in north-eastern Nigeria, is experiencing rapid socio-economic transitions characterised by westernised dietary patterns and reduced physical activity, trends that further fuel the rising prevalence of T2DM and its associated complications (Gezawa *et al*., 2015).

DM is a major cardiovascular risk factor. Patients with T2DM have a considerably higher risk of cardiovascular morbidity and mortality compared to non-diabetic individuals, arising from a complex interplay of traditional and non-traditional cardiovascular risk factors including dyslipidaemia, hypertension, obesity, and insulin resistance, all of which contribute to the accelerated development of atherosclerosis (Vesa *et al.,* 2019). Vitamin D (VD), first described by Goldblatt and Soames in 1923 and formally characterised by Norman (2012), is a fundamental fat-soluble micronutrient with wide-ranging non-skeletal effects. Vitamin D receptors (VDRs) have been identified in pancreatic β-cells, cardiomyocytes, endothelial cells, and vascular smooth muscle cells, underscoring its role in insulin secretion, immune regulation, and cardiovascular function (Seshadri *et al.,* 2011).

Evidence from prospective studies and meta-analyses suggests that lower vitamin D status is independently associated with insulin resistance, glucose intolerance, dyslipidaemia, hypertension, and obesity (Talaei *et al*., 2013; Mozos and Marginean, 2015; Lavie *et al*., 2011). Despite the geographic advantage of abundant sunshine throughout the year, vitamin D deficiency is paradoxically common in Nigerian populations, with individuals of African ancestry demonstrating consistently lower serum 25(OH)D levels than other ethnic groups (Akeredolu *et al*., 2021; Mogire *et al*., 2020). Furthermore, only a limited number of studies have examined the specific relationship between vitamin D status and cardiovascular risk factor clustering in T2DM patients within the Nigerian setting, particularly in the north-eastern region. The present study was therefore designed to evaluate the association between serum vitamin D levels and cardiovascular disease risk factors, including dyslipidaemia, hypertension, obesity, and impaired glycaemic control, in T2DM patients attending the University of Maiduguri Teaching Hospital, with the goal of providing locally relevant data to guide screening and management strategies.

2. MATERIALS AND METHODS

2.1 Study Area and Design

A hospital-based cross-sectional study was conducted at the University of Maiduguri Teaching Hospital (UMTH), a tertiary healthcare facility and the principal referral centre for Borno State and the surrounding north-eastern region. UMTH provides both inpatient and outpatient services across multiple specialties including endocrinology, internal medicine, and chemical pathology.

2.2 Study Participants and Sample Size

A total of 140 participants were enrolled, comprising 70 T2DM patients recruited from the Diabetes Clinic and 70 age-matched control participants recruited from the General Outpatient Department. T2DM diagnosis was established according to World Health Organization criteria (WHO, 2019). To address age-related confounding, participants were age-matched through the removal of outliers, yielding comparable mean ages between the two groups.

2.3 Inclusion and Exclusion Criteria

T2DM patients were included if they had confirmed T2DM diagnoses, were aged 18–65 years, and provided informed written consent. Participants were excluded if they had type 1 DM, gestational diabetes, secondary diabetes mellitus, known renal or hepatic disease, were on vitamin D or calcium supplements, were pregnant or lactating, had a history of cardiovascular events within the preceding three months, or were unable to provide an adequate blood sample.

2.4 Ethical Considerations

Ethical approval was obtained from the Research and Ethics Committee of UMTH. Written informed consent was obtained from all participants prior to enrolment. All procedures were conducted in accordance with the Declaration of Helsinki.

2.5 Specimen Collection and Storage

After an overnight fast of at least 8 hours, venous blood samples were collected by venipuncture using standard phlebotomy procedures. Samples were collected into plain gel tubes for serum biochemistry, EDTA tubes for HbA1c, and fluoride oxalate tubes for fasting plasma glucose (FPG) estimation. Serum was separated by centrifugation within 30 minutes of collection. All samples were stored at −20°C until batch analysis.

2.6 Laboratory Analytical Methods

Serum 25-hydroxyvitamin D [25(OH)D] was measured using an enzyme-linked immunosorbent assay (ELISA) as described by Holick *et al*., (2011). Vitamin D status was classified based on established clinical thresholds: very severe deficiency (<5ng/mL), severe deficiency (5–9 ng/mL), deficiency (10–19 ng/mL), suboptimal/insufficient (20–29 ng/mL), optimal/sufficient (30–49 ng/mL), and upper normal range (50–70 ng/mL) (Holick *et al*., 2011; Bikle, 2014).

Fasting plasma glucose was determined by the glucose oxidase enzymatic method (Kubihal *et al.,* 2021). Serum total calcium was measured using the O-cresolphthalein method, and albumin-adjusted calcium was calculated using the formula: Adjusted calcium (mmol/L) = Measured calcium + 0.02 × (40 − Albumin g/L) (Besozzi *et al*., 1981). Serum albumin was quantified by the Bromocresol Green (BCG) method (Harding and Keyser, 1968). HbA1c was determined by chromatographic method using the CLOVER A1c self-analyser (Tremblay *et al*., 2004).

Lipid profiles were determined as follows: HDL-cholesterol by the modified polyvinyl sulfonic acid precipitation method (Yeates *et al*., 1979); triglycerides by the enzymatic hydrolytic method (Ziegenhorn, 1975); total cholesterol by the enzymatic endpoint method (Borner and Klose, 1977); and LDL-cholesterol calculated using the Friedewald formula: LDL-C = Total cholesterol − HDL-C − (Triglycerides/2.2) in mmol/L, applicable where triglycerides did not exceed 4.5 mmol/L (Tremblay *et al*., 2004).

2.7 Statistical Analysis

Data were analysed using the Statistical Package for Social Sciences (SPSS) version 26.0 for Windows. Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Independent samples t-test was used to compare means between T2DM patients and control participants. Pearson correlation coefficients were computed to examine bivariate relationships between vitamin D levels and cardiovascular risk parameters. Logistic regression analyses (unadjusted and adjusted models) were used to determine the independent association between vitamin D status and T2DM risk. Multiple linear regression identified independent predictors of vitamin D levels. A p-value <0.05 was considered statistically significant.

3. RESULTS

3.1 Socio-Demographic Profile of Participants

The study enrolled 140 participants: 70 T2DM patients (38 males, 54.3%; 32 females, 45.7%) and 70 age-matched controls (42 males, 60.0%; 28 females, 40.0%). The mean age of T2DM patients was 35.24±8.12 years and of controls was 33.18±7.45 years, with no statistically significant difference (p=0.128). More than three-quarters of enrolled participants had formal education: 77.1% of T2DM patients and 71.4% of controls. Regarding occupation, traders constituted 40.0% of T2DM patients compared to 25.7% of controls, while civil servants were more prevalent among controls (54.3% vs 27.1%). The Kanuri ethnic group was the most represented in both groups. The distribution of tribal groups was broadly comparable between the two study groups (Table 1).

Table 1: Socio-demographic profile of participants

Variable	T2DM (n=70) n (%)	Control (n=70) n (%)
Age (years) – mean±SD	35.24±8.12	33.18±7.45
Sex		
Male	38 (54.3)	42 (60.0)
Female	32 (45.7)	28 (40.0)
Educational Level		
Formal	54 (77.1)	50 (71.4)
Informal	16 (22.9)	20 (28.6)
Occupation		
Civil Servants	19 (27.1)	38 (54.3)
Traders	28 (40.0)	18 (25.7)
Others	23 (32.9)	14 (20.0)
Ethnic Group		
Kanuri	22 (31.4)	18 (25.7)
Hausa/Fulani	12 (17.1)	16 (22.9)
Babur/Bura	11 (15.7)	14 (20.0)
Others	25 (35.8)	22 (31.4)

3.2 Clinical Characteristics and Cardiovascular Risk Factor Comparison

T2DM patients demonstrated substantially worse cardiovascular risk profiles compared to age-matched controls across all parameters assessed (Table 2). Mean systolic blood pressure was significantly elevated in T2DM patients (115.8±14.2 vs 104.1±14.8 mmHg, p<0.001), as was diastolic blood pressure (92.4±10.1 vs 78.2±6.9 mmHg, p<0.001). T2DM patients had a significantly higher mean BMI (27.3±5.4 vs 21.2±3.6 kg/m², p<0.001), with 65.7% classified as obese. Glycaemic control was markedly poor in T2DM patients, reflected by elevated mean FPG (8.4±3.4 vs 4.2±0.5 mmol/L, p<0.001) and HbA1c (8.1±2.0 vs 3.9±0.5%, p<0.001). Regarding lipid parameters, T2DM patients exhibited significantly elevated total cholesterol (6.0±1.5 vs 3.9±0.7 mmol/L), LDL-C (4.0±1.3 vs 2.7±0.6 mmol/L), and triglycerides (1.9±0.5 vs 0.8±0.4 mmol/L), alongside lower HDL-C (1.1±0.4 vs 1.3±0.3 mmol/L), all p≤0.002. Atherogenic indices were markedly elevated: TC/HDL ratio 6.1±2.3 vs 4.2±0.9 and LDL/HDL ratio 4.2±2.0 vs 1.9±1.2 (both p<0.001). Vitamin D levels were significantly lower in T2DM patients (19.8±9.2 vs 26.4±10.1 ng/mL, p<0.001), and albumin-adjusted calcium was similarly reduced (2.15±0.18 vs 2.28±0.12 mmol/L, p<0.001).

Table 2: Comparison of cardiovascular risk factors between T2DM patients and controls

Biomarker	T2DM (n=70) Mean±SD	Control (n=70) Mean±SD	p-value
Vitamin D (ng/mL)	19.8±9.2	26.4±10.1	<0.001*
BMI (kg/m²)	27.3±5.4	21.2±3.6	<0.001*
FPG (mmol/L)	8.4±3.4	4.2±0.5	<0.001*
HbA1c (%)	8.1±2.0	3.9±0.5	<0.001*
Systolic BP (mmHg)	115.8±14.2	104.1±14.8	<0.001*
Diastolic BP (mmHg)	92.4±10.1	78.2±6.9	<0.001*
Albumin-adjusted calcium (mmol/L)	2.15±0.18	2.28±0.12	<0.001*
Total Cholesterol (mmol/L)	6.0±1.5	3.9±0.7	<0.001*
LDL-C (mmol/L)	4.0±1.3	2.7±0.6	<0.001*
HDL-C (mmol/L)	1.1±0.4	1.3±0.3	0.002*
Triglycerides (mmol/L)	1.9±0.5	0.8±0.4	<0.001*
TC/HDL ratio	6.1±2.3	4.2±0.9	<0.001*
LDL/HDL ratio	4.2±2.0	1.9±1.2	<0.001*

*Statistically significant at p<0.05; BMI=Body Mass Index; FPG=Fasting Plasma Glucose; HbA1c=Glycated Haemoglobin; BP=Blood Pressure; LDL-C=Low Density Lipoprotein Cholesterol; HDL-C=High Density Lipoprotein Cholesterol; TC=Total Cholesterol

3.3 Distribution of Vitamin D Status

The majority of T2DM patients had vitamin D deficiency, with 62.9% (n=44) falling below 20 ng/mL compared to 31.4% (n=22) of controls. In the severe deficiency category (5–10 ng/mL), 22.9% of T2DM patients were affected versus only 4.3% of controls. Very severe deficiency (<5 ng/mL) was present in 2.9% of T2DM patients versus 1.4% of controls. In contrast, optimal vitamin D levels (30–50 ng/mL) were achieved by only 17.1% of T2DM patients compared to 34.3% of controls. No T2DM patient reached upper normal levels (50–70 ng/mL), while 1.4% of controls did so (Table 3).

Table 3: Distribution of vitamin D status by category in the study groups

Vitamin D Status	Range (ng/mL)	T2DM (n=70) n (%)	Control (n=70) n (%)
Very Severe Deficiency	<5	2 (2.9)	1 (1.4)
Severe Deficiency	5–9	16 (22.9)	3 (4.3)
Deficiency	10–19	26 (37.1)	18 (25.7)
Suboptimal/Insufficient	20–29	14 (20.0)	20 (28.6)
Optimal/Sufficient	30–49	12 (17.1)	24 (34.3)
Upper Normal	50–70	0 (0.0)	1 (1.4)

3.4 Logistic Regression Analysis of Vitamin D and T2DM Risk

In the unadjusted analysis, each 1 ng/mL increase in vitamin D was associated with a 6.8% decrease in the odds of T2DM (OR: 0.932, 95% CI: 0.904–0.961, p<0.001). After adjustment for sex, BMI, and blood pressure (Model 1), the association remained significant (adjusted OR: 0.945, 95% CI: 0.910–0.982, p=0.004). In the fully adjusted model incorporating lipid parameters (Model 2), vitamin D deficiency remained independently associated with T2DM (adjusted OR: 0.954, 95% CI: 0.917–0.993, p=0.021), representing a 4.6% reduction in the odds of T2DM per 1 ng/mL increment in vitamin D (Table 4). Regarding vitamin D categories, deficiency (<20 ng/mL) conferred 4.12 times higher odds of T2DM compared to sufficient levels (≥30 ng/mL) in the unadjusted analysis (95% CI: 1.89–8.97, p<0.001), and this association persisted after full adjustment (adjusted OR: 2.68, 95% CI: 1.08–6.65, p=0.033).

Table 4: Logistic regression analysis of vitamin D levels and T2DM risk

Model	Odds Ratio	95% CI	p-value
Unadjusted	0.932	0.904–0.961	<0.001*
Model 1 (adjusted for sex, BMI, BP)	0.945	0.910–0.982	0.004*
Model 2 (further adjusted for lipids)	0.954	0.917–0.993	0.021*

*Statistically significant at p<0.05; OR=Odds Ratio; CI=Confidence Interval

3.5 Relationship between Vitamin D and Glycaemic Control

Among T2DM patients, 64.3% (n=45) had poor fasting plasma glucose control (FPG ≥7.0 mmol/L), and these patients had lower mean vitamin D levels compared to those with good control (18.5±8.9 vs 21.8±9.7 ng/mL). The adjusted analysis demonstrated that T2DM patients with elevated FPG had a 1.95 times higher odds of vitamin D deficiency (95% CI: 1.15–3.31, p=0.013). Poor HbA1c control (≥6.5%) was present in 85.7% (n=60) of T2DM patients and was similarly associated with lower mean vitamin D levels (19.6±9.4 vs 20.8±6.2 ng/mL), with an adjusted odds ratio of 1.74 (95% CI: 1.03–2.94, p=0.038) for vitamin D deficiency.

3.6 Correlations between Vitamin D and Cardiovascular Risk Factors

In the T2DM group, vitamin D levels demonstrated significant negative correlations with multiple cardiovascular risk parameters: FPG ($r = -0.396$, $p<0.001$), HbA1c ($r = -0.361$, $p=0.002$), BMI ($r = -0.324$, $p=0.006$), systolic blood pressure ($r = -0.241$, $p=0.044$), total cholesterol ($r = -0.301$, $p=0.011$), LDL-C ($r = -0.284$, $p=0.016$), and triglycerides ($r = -0.257$, $p=0.032$). Significant positive correlations were observed with HDL-C ($r = 0.245$, $p=0.041$) and albumin-adjusted calcium ($r = 0.312$, $p=0.008$). Diastolic BP showed a non-significant trend ($r = -0.218$, $p=0.067$). In contrast, none of these correlations reached statistical significance in the control group (all $p>0.05$), indicating that the vitamin D–cardiovascular risk relationship is specific to the diabetic state (Table 5).

Table 5: Pearson correlations between serum vitamin D levels and cardiovascular risk factors

Risk Factor	T2DM r	T2DM p	Control r	Control p	Sig.
Fasting Plasma Glucose	−0.396	<0.001*	−0.182	0.132	Yes
HbA1c	−0.361	0.002*	−0.089	0.462	Yes
BMI	−0.324	0.006*	−0.156	0.195	Yes
Systolic BP	−0.241	0.044*	−0.198	0.098	Yes
Diastolic BP	−0.218	0.067	−0.134	0.266	No
Total Cholesterol	−0.301	0.011*	−0.143	0.238	Yes
LDL-C	−0.284	0.016*	−0.167	0.165	Yes
HDL-C	+0.245	0.041*	+0.089	0.461	Yes
Triglycerides	−0.257	0.032*	−0.123	0.309	Yes
Albumin-adjusted calcium	+0.312	0.008*	+0.189	0.117	Yes

*Statistically significant at p<0.05 in T2DM group; r=Pearson correlation coefficient

3.7 Multivariate Predictors of Vitamin D Levels

In the T2DM group, the multivariate regression model explained 32.4% of the variance in vitamin D levels (Adjusted $R^2 = 0.324$, $F = 4.83$, $p<0.001$). Significant independent predictors were BMI ($\beta = -0.294$, $p=0.006$), FPG ($\beta = -0.342$, $p=0.002$), HbA1c ($\beta = -0.289$, $p=0.009$), and HDL-C ($\beta = 0.223$, $p=0.034$). In the control group, the model was less robust, explaining 18.9% of the variance (Adjusted $R^2 = 0.189$, $F = 2.67$, $p=0.008$), with only BMI ($\beta = -0.231$, $p=0.029$) and total cholesterol ($\beta = -0.218$, $p=0.041$) emerging as significant predictors. Notably, glycaemic parameters were significant predictors of vitamin D levels exclusively in the T2DM cohort, suggesting a distinctive bidirectional relationship between vitamin D and glycaemic dysregulation in established diabetes.

Table 6: Multiple linear regression analysis of independent predictors of vitamin D levels

Independent Variable	T2DM β	T2DM p	Control β	Control p
BMI	−0.294	0.006*	−0.231	0.029*
Fasting Plasma Glucose	−0.342	0.002*	−0.187	0.089
Glycated Haemoglobin	−0.289	0.009*	−0.156	0.142
HDL-C	+0.223	0.034*	+0.148	0.186
Total Cholesterol	−0.196	0.078	−0.218	0.041*
LDL-C	−0.181	0.105	−0.203	0.068
Systolic BP	−0.168	0.134	−0.119	0.298
Triglycerides	−0.162	0.149	−0.141	0.218
Adjusted R^2	0.324	<0.001*	0.189	0.008*

*Statistically significant at p<0.05; β=standardised regression coefficient

4. DISCUSSION

This study has demonstrated that vitamin D deficiency is significantly more prevalent in T2DM patients than in age-matched control participants in Maiduguri, north-eastern Nigeria, and that vitamin D levels are independently and inversely associated with multiple cardiovascular risk factors specifically in the diabetic cohort. These findings have important implications for the management of T2DM in this resource-limited setting.

The socio-demographic characteristics of the study population were well-balanced between groups, with no significant age difference, thereby minimising age-related confounding. The significantly higher proportion of traders among T2DM patients (40.0% vs 25.7% in controls) may reflect lifestyle differences including irregular dietary patterns and sedentary occupational activity, consistent with findings from epidemiological studies on occupational risk factors for T2DM across sub-Saharan Africa (Powers *et al*., Uloko *et al*., 2018).

The finding that 62.9% of T2DM patients had vitamin D deficiency, compared to 31.4% of controls, is highly consistent with national and international literature. A study from Lagos, Nigeria, by Anyanwu *et al*., (2020) reported a 63.2% deficiency prevalence among T2DM patients. Similarly, Chijioke *et al*., (2020) in North Central Nigeria documented significantly lower mean vitamin D levels and poorer glycaemic control in individuals with T2DM. Internationally, Shrestha *et al*., (2024) reported a mean 25(OH)D of 18.8 ng/mL, with 89.7% below sufficiency among Nepali T2DM patients, while Khudayar *et al*., (2022) documented a 74.1% deficiency rate and a 2023 global meta-analysis reported deficiency prevalence between 63.5% and 91.1% in T2DM cohorts. The mean vitamin D level of 19.8±9.2 ng/mL observed among T2DM patients in this study falls below the Institute of Medicine sufficiency threshold of 20 ng/mL, placing this population at particular metabolic and cardiovascular risk.

Paradoxically, the study population was located in Maiduguri, a city characterised by abundant sunshine throughout the year. This observation aligns with the continental pattern described by Mogire *et al*., (2020), whose systematic review and meta-analysis of 23 African countries found high vitamin D deficiency prevalence across African populations, indicating that geographic location alone does not confer adequate vitamin D status. Contributing factors in this population may include cultural and religious dress practices that limit sunlight exposure, reduced outdoor activity, dietary insufficiency, and possible genetic polymorphisms in vitamin D metabolism enzymes, all warranting investigation in future studies.

The significant inverse correlations between vitamin D levels and glycaemic parameters in T2DM patients (FPG: $r = -0.396$, $p<0.001$; HbA1c: $r = -0.361$, $p=0.002$) corroborate the mechanistic evidence that vitamin D influences glucose homeostasis through enhancement of pancreatic β-cell function, modulation of inflammatory cytokines, and regulation of calcium homeostasis in insulin-responsive tissues (Wimalawansa, 2018; Contreras-Bolívar *et al*., 2021). Notably, these associations were entirely absent in control participants, suggesting that the vitamin D–glucose relationship becomes particularly pronounced once metabolic dysregulation is established—a finding echoed by randomised controlled trials demonstrating improved glycaemic control following vitamin D supplementation in vitamin D-deficient T2DM patients (Chinenye *et al*., 2019; Talaei *et al*., 2013).

The cardiovascular risk profile of T2DM patients was markedly adverse compared to controls, with higher prevalence of elevated LDL-C (68.6%), low HDL-C (62.9%), elevated triglycerides (57.1%), and elevated total cholesterol (62.9%). Atherogenic indices were similarly elevated (TC/HDL: 6.1±2.3 vs 4.2±0.9; LDL/HDL: 4.2±2.0 vs 1.9±1.2), consistent with reported clustering of cardiovascular risk factors in Nigerian diabetic populations (Dokunmu *et al*., 2018). The significant inverse correlations between vitamin D levels and total cholesterol, LDL-C, and triglycerides, combined with a positive correlation with HDL-C, suggest that vitamin D may exert a protective effect on the lipid profile in diabetic patients—possibly mediated by its antioxidant properties, reduction of inflammatory cytokines, and improvement in insulin sensitivity (Jorde and Grimnes, 2011; Mazidi *et al*., 2017; Scragg *et al*., 2014).

The significant inverse correlation between vitamin D and BMI ($r = -0.324$, $p=0.006$), alongside the finding that 65.7% of T2DM patients were obese with the lowest mean vitamin D levels (18.1±8.4 ng/mL), is consistent with the well-documented volumetric dilution hypothesis, whereby vitamin D is sequestered in abundant adipose tissue, reducing its bioavailability (Drincic *et al*., 2012; Vanlint, 2013; Wortsman *et al*., 2000). Conversely, vitamin D deficiency may promote further weight gain through impaired calcium-mediated adipogenesis, creating a bidirectional obesity–vitamin D cycle that further deteriorates metabolic outcomes (Narvaez *et al*., 2009; Sergeev and Song, 2014).

The positive correlation between vitamin D and albumin-adjusted calcium ($r = 0.312$, $p=0.008$), alongside the significantly higher prevalence of calcium deficiency in T2DM patients (64.3% vs 25.7%), highlights the complex interplay between vitamin D status, calcium metabolism, and diabetic pathophysiology, as documented by Levy *et al*., (1986), Hagström *et al*., (2009), and Dhas *et al*., (2014).

Vitamin D is essential for intestinal calcium absorption and renal calcium reabsorption, and its deficiency leads to secondary hyperparathyroidism, which may further impair insulin secretion and promote cardiovascular dysfunction.

The logistic regression findings, demonstrating a 4.6% reduction in T2DM odds per 1 ng/mL increment in vitamin D (fully adjusted OR: 0.954, 95% CI: 0.917–0.993, p=0.021) and 2.68 times higher odds of T2DM in the deficient versus sufficient category (95% CI: 1.08–6.65, p=0.033), are consistent with several international meta-analyses and the seminal D2d trial (Pittas *et al*., 2019), which demonstrated a significant reduction in T2DM risk following vitamin D supplementation in prediabetic individuals with low baseline 25(OH)D. The multivariate finding that glycaemic parameters were significant independent predictors of vitamin D levels exclusively in the T2DM cohort (FPG: $\beta = -0.342$, p=0.002; HbA1c: $\beta = -0.289$, p=0.009) supports the concept of a vicious cycle, wherein worsening glycaemia and vitamin D depletion mutually exacerbate each other.

The present study has several strengths, including the age-matched design, comprehensive assessment of multiple cardiovascular risk factors and biomarkers, and use of standardised laboratory methodologies. However, important limitations must be acknowledged. The cross-sectional design precludes causal inference. Potentially confounding variables such as dietary vitamin D intake, sun exposure patterns, seasonal variation, physical activity levels, and genetic polymorphisms in vitamin D synthesis and metabolism pathways were not systematically assessed. The sample size, while adequate for the current analyses, may limit the generalisability of findings to the broader north-eastern Nigerian T2DM population.

5. CONCLUSION

Vitamin D deficiency is significantly more prevalent in T2DM patients than in age-matched controls in Maiduguri, north-eastern Nigeria, and is independently associated with multiple cardiovascular risk factors—including poor glycaemic control, dyslipidaemia, hypertension, obesity, and impaired calcium homeostasis—specifically within the diabetic cohort. Each 1 ng/mL increment in vitamin D was associated with a 4.6% reduction in T2DM odds after full confounder adjustment. These findings support the routine incorporation of vitamin D status assessment into the comprehensive cardiovascular risk evaluation of T2DM patients in this region. Well-powered longitudinal and interventional studies are needed to establish causal relationships, determine optimal supplementation protocols, and clarify the role of local environmental, lifestyle, and genetic factors in modulating the observed associations.

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REFERENCES

- Akeredolu FD, Akuse RM, Mado SM, Yusuf R (2021). Serum 25-Hydroxyvitamin D Levels of Apparently Healthy Nigerian Children Aged 1-24 Months. *International Journal of Pediatric Research*, 7:076.
- American Diabetes Association (2013). Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*, 36(Supplement 1), S67–S74.
- Anyanwu AC, Fasanmade OA, Coker HAB, Ohwovoriole AE (2017). Vitamin D supplementation improves insulin resistance in type 2 diabetes subjects in Lagos, Nigeria. *African Journal of Diabetes Medicine*, 25(1), 14–17.
- Anyanwu AC, Fasanmade OA, Odeniyi IA, Iwuala S, Coker HB, Ohwovoriole AE (2016). Effect of Vitamin D supplementation on glycemic control in Type 2 diabetes subjects in Lagos, Nigeria. *Indian Journal of Endocrinology and Metabolism*, 20(2), 189–194. doi:10.4103/2230-8210.176345.
- Anyanwu AC, Olopade OB, Onung SI, Odeniyi IA, Coker HAB, Fasanmade OA, Ohwovoriole AE (2020). Serum Vitamin D Levels in Persons with Type 2 Diabetes Mellitus in Lagos, Nigeria. *International Journal of Diabetes and Clinical Research*, 7:133. doi:10.23937/2377-3634/1410133.
- Besozzi M, Franzini C, Senaldi G (1981). Evaluation of the methylthymol blue and orthocresolphthalein direct methods of determination of serum calcium. *Quad Sclavo Diagn*, 17(2), 182–197.
- Bikle DD (2014). Vitamin D metabolism, mechanism of action, and clinical applications. *Chemistry & Biology*, 21(3), 319–329.
- Borner K, Klose S (1977). *Enzymatische Bestimmung des Gesamtcholesterins mit dem Greiner Selective Analyzer (GSA-II)*. Berlin: Greiner Instruments.
- Chijioke OH, Ehienagudia AM, Akinwande OM (2020). Low Vitamin D Levels and Correlates Amongst Adult Nigerians in North Central Nigeria. *West African Journal of Medicine*, 37(6), 631–639. PMID: 33185258.
- Contreras-Bolívar V, García-Fontana B, García-Fontana C, Muñoz-Torres M (2021). Mechanisms involved in the relationship between vitamin D and insulin resistance. *Nutrients*, 13(10), 3491. doi:10.3390/nu13103491.
- Dahiru T, Aliyu AA, Shehu AU (2016). A review of population-based studies on diabetes mellitus in

Nigeria. *Sub-Saharan African Journal of Medicine*, 3(2), 59–64.

- Dhas Y, Mishra N, Banerjee J, et al. (2014). Vitamin D deficiency and oxidative stress in type 2 diabetic Goa population. *Clinical Laboratory*, 60(10), 1751–1758.
- Dokunmu TM, Yakubu OF, Adebayo AH, Olasehinde GI, Chinedu SN (2018). Cardiovascular Risk Factors in a Suburban Community in Nigeria. *International Journal of Hypertension*, 2018:6898527. doi:10.1155/2018/6898527. *(Fully verified via PubMed*
- Drincic AT, Armas LA, Van Diest EE, Heaney RP (2012). Volumetric dilution, rather than sequestration best explains the low vitamin D status of obesity. *Obesity*, 20(7), 1444–1448. doi:10.1038/oby.2011.213.
- Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C (2020). Pathophysiology of Type 2 Diabetes Mellitus. *International Journal of Molecular Sciences*, 21(17), 6275. doi:10.3390/ijms21176275.
- Gezawa ID, Puepet FH, Mubi BM, et al. (2015). Socio-demographic and anthropometric risk factors for type 2 diabetes in Maiduguri, North-Eastern Nigeria. *Sahel Medical Journal*, 18(5), 1–7.
- Goldblatt H, Soames KM (1923). A study of rats on a normal diet irradiated daily by the mercury vapor quartz lamp or kept in darkness. *Biochemical Journal*, 17(2), 294–297.
- Hagström E, Hellman P, Larsson TE, et al. (2009). Plasma parathyroid hormone and cardiovascular mortality. *Circulation*, 119(21), 2765–2771.
- Harding JR, Keyser JW (1968). Bromocresol green as a reagent for serum albumin. *Proceedings of the Association of Clinical Biochemists*, 5(2), 51–53.
- Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, Murad MH, Weaver CM (2011). Evaluation, treatment, and prevention of vitamin D deficiency: An Endocrine Society Clinical Practice Guideline. *Journal of Clinical Endocrinology & Metabolism*, 96(7), 1911–1930. doi:10.1210/jc.2011-0385.
- Jorde R, Grimnes G (2011). Vitamin D and metabolic health with special reference to the effect of vitamin D on serum lipids. *Progress in Lipid Research*, 50(4), 303–312. doi:10.1016/j.plipres.2011.05.001.
- Khudayar M, Nadeem A, Lodi MN, Rehman K, Jawaid SI, Mehboob A, Aleem AS, Mirza REF, Ahmed M, Abbas K (2022). The Association Between Deficiency of Vitamin D and Diabetes Mellitus Type 2 (DMT2). *Cureus*, 14(2), e22221. doi:10.7759/cureus.22221.
- Kubihal S, Goyal A, Gupta Y, Khadgawat R (2021). Glucose measurement in body fluids: A ready reckoner for clinicians. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 15(1), 45–53.
- Lavie CJ, Lee JH, Milani RV (2011). Vitamin D and cardiovascular disease: Will it live up to its hype? *Journal of the American College of Cardiology*, 58(15), 1547–1556.
- Levy J, Stern Z, Gutman A, et al. (1986). Plasma calcium and phosphate levels in adult non-insulin-dependent diabetics. *Calcified Tissue International*, 39(5), 316–318.
- Mapa-Tassou C, Katte JC, Maadjhou CM, Mbanya JC (2019). Economic impact of diabetes in Africa. *Current Diabetes Reports*, 19(2), 5.
- Mazidi M, Rezaie P, Vatanparast H, Kengne AP (2017). Effect of statins on serum vitamin D concentrations: A systematic review and meta-analysis. *European Journal of Clinical Investigation*, 47(1), 93–101. doi:10.1111/eci.12698.
- Mogire RM, Mutua A, Kimita W, Kamau A, Bejon P, Pettifor JM, Adeyemo A, Williams TN, Atkinson SH (2020). Prevalence of vitamin D deficiency in Africa: A systematic review and meta-analysis. *Lancet Global Health*, 8(1), e134–e142.
- Mozos I, Marginean O (2015). Links between vitamin D deficiency and cardiovascular diseases. *BioMed Research International*, 2015, 109275.
- Narvaez CJ, Simmons KM, Brunton J, Salinero A, Chittur SV, Welsh JE (2009). Induction of STEAP2 by 1,25-dihydroxyvitamin D3 in mammary gland: Pharmacological implications. *Molecular Pharmacology*, 76(5), 2009.
- Norman AW (2012). The history of the discovery of vitamin D and its daughter steroid hormone. *Annals of Nutrition and Metabolism*, 61(3), 199–206.
- Pittas AG, Dawson-Hughes B, Sheehan P, et al. (2019). Vitamin D supplementation and prevention of type 2 diabetes mellitus. *New England Journal of Medicine*, 381(6), 520–530. doi:10.1056/NEJMoa1900906.
- Powers AC, Niswender KD, Evans-Molina C (2022). Diabetes mellitus: Diagnosis, classification, and pathophysiology. In: Loscalzo J, Fauci A, Kasper D, Hauser S, Longo D, Jameson JL, eds. *Harrison's Principles of Internal Medicine*, 21st edition. New York: McGraw-Hill.
- Scragg R, Slow S, Stewart AW, Jennings LC, Chambers ST, Priest PC, Florkowski CM, Camargo CA Jr, Murdoch DR (2014). Long-term high-dose vitamin D3 supplementation and blood pressure in healthy adults: A randomized controlled trial. *Hypertension*, 64(4), 725–730. doi:10.1161/HYPERTENSIONAHA.114.03466.
- Sergeev IN, Song Q (2014). High vitamin D and calcium intakes reduce diet-induced obesity in mice by increasing adipose tissue apoptosis.

Molecular Nutrition & Food Research, 58(6), 1342–1348. doi:10.1002/mnfr.201300503.

- Seshadri KG, Tamilselvan B, Rajendran A (2011). Role of vitamin D in diabetes. *Journal of Endocrinology and Metabolism*, 1(2), 47–56.
- Shrestha P, Rauniyar NK, Sharma L, Adhikari K (2024). Vitamin-D status and metabolic syndrome in patients with type 2 diabetes mellitus: Vitamin D status in DM. *Journal of General Practice and Emergency Medicine of Nepal*, 11(17), 6–12. doi:10.59284/jgpeman272.
- Talaei A, Mohamadi M, Adgi Z (2013). The effect of vitamin D on insulin resistance in patients with type 2 diabetes. *Diabetology & Metabolic Syndrome*, 5(1), 8.
- Tremblay AJ, Morrissette H, Gagné JM, Bergeron J, Gagné C, Couture P (2004). Validation of the Friedewald formula for the determination of low-density lipoprotein cholesterol compared with β-quantification in a large population. *Clinical Biochemistry*, 37(9), 785–790.
- Uloko AE, Musa BM, Ramalan MA, et al. (2018). Prevalence and risk factors for diabetes mellitus in Nigeria: A systematic review and meta-analysis. *Diabetes Therapy*, 9(3), 1307–1316.
- Vanlint S (2013). Vitamin D and obesity. *Nutrients*, 5(3), 949–956. doi:10.3390/nu5030949.
- Vesa CM, Popa AR, Bungau S, et al. (2019). Exploration of insulin sensitivity, insulin resistance, early insulin secretion and B-cell function in normal weight patients with newly diagnosed type 2 diabetes mellitus. *Revista de Chimie*, 70, 4217–4223.
- Wimalawansa SJ (2018). Associations of vitamin D with insulin resistance, obesity, type 2 diabetes, and metabolic syndrome. *Journal of Steroid Biochemistry and Molecular Biology*, 175, 177–189. doi: 10.1016/j.jsbmb.2016.09.017.
- World Health Organization (2019). *Classification of Diabetes Mellitus*. Geneva: WHO.
- Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF (2000). Decreased bioavailability of vitamin D in obesity. *American Journal of Clinical Nutrition*, 72(3), 690–693. doi:10.1093/ajcn/72.3.690.
- Yeates RA, Mannik T, Calvert GD (1979). The measurement of plasma HDL-cholesterol by precipitation techniques. *Pathology*, 11(2), 324.
- Ziegenhorn J (1975). Improved method for enzymatic determination of serum triglycerides. *Clinical Chemistry*, 21(11), 1627–1629.