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Original Research Article

Relationship between Vitamin D Status, Inflammation and Hyperglycemia in Type 2 Diabetic Patients Attending the Laquintinie Hospital of Douala, Cameroon

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Abstract

Background

Vitamin D has been increasingly linked to type 2 diabetes mellitus (T2DM) pathogenesis through its role in β-cell function, insulin secretion, and sensitivity. Reviews suggest that due to its anti-inflammatory properties, inadequate levels may exacerbate hyperglycaemia and low-grade chronic inflammation associated with T2DM. This study investigated the relationship between vitamin D status, inflammation, and hyperglycaemia in T2DM patients at Laquintinie Hospital, Douala.

Methods

A cross-sectional study was conducted involving 125 participants with T2DM aged ≥ 25 years, recruited via convenience sampling. Data was collected using structured questionnaires, and venous blood samples were analysed for serum vitamin D, fasting plasma glucose (FPG), glycated haemoglobin (HbA1c), creatinine, and high-sensitivity C-reactive protein (hs-CRP). Statistical significance was set at p-value ≤ 0.05.

Results

Among 125 participants, 66.4% had hypovitaminosis D, including 28.8% with deficiency. Vitamin D significantly correlated inversely with FPG ($r = -0.214$, $p = 0.016$) and hs-CRP ($r = -0.394$, $p < 0.001$). No significant associations were seen between vitamin D status, HbA1c and diabetic complications.

Conclusion

Results of this study suggest that hypovitaminosis D is strongly associated with inflammation and hyperglycaemia in T2DM patients.

Keywords: *Type 2 diabetes mellitus, vitamin D deficiency, hyperglycaemia and inflammation.*

1. Introduction

Diabetes mellitus (DM) is known to be one of the world's most rapidly growing chronic non-communicable diseases, imposing a substantial burden on public health and socio-economic development [1]. In 1980, 108 million people globally had diabetes, a figure that rose to 463 million in 2019, and by 2021, 537 million people aged between 20 and 75 were living with the disease, with 1 in 10 cases undiagnosed. Projections from the International Diabetes Federation (IDF) Diabetes Atlas estimate that this figure will rise to 784 million by 2045 [2]. The rising burden is particularly concerning in sub-Saharan Africa, which has been experiencing a rapid rise in the prevalence of this disease, with an estimate of 24 million adults living with diabetes, 54% of people living with undiagnosed diabetes, and 416,000 deaths in the year 2021. About 615,000 adults in Cameroon have diabetes, which is 6% of the population. Diabetes is the fifth leading cause of death in Cameroon, responsible for 2% of annual mortality [2]. This study focuses on T2DM, which makes up over 90% of diabetes cases. Its numerous complications, such as cardiovascular disease, neuropathy, and kidney failure, significantly reduce the quality of life and functional capabilities of the patients and are considered the third highest risk factor of worldwide premature mortality due to hyperglycaemia [3].

Vitamin D, an essential fat-soluble nutrient well known for its classical function in calcium and phosphorus homeostasis and bone health, has gained increasing attention for its extra-skeletal functions. Initially identified during efforts to combat rickets in the 1700s [4], vitamin D is now known to exert effects on a wide range of tissues that have no direct role in calcium and bone metabolism, such as pancreatic β-cells, adipose tissues, brain cells, skeletal muscles, immune cells, cardiovascular tissues, and breast, colon, and bronchial epithelial cells [5, 6]. The discovery of vitamin D receptors (VDRs) in these tissues has broadened our understanding of their physiological role in regulating a wide range of cellular processes, such as proliferation and differentiation, apoptosis, neurogenesis, immune modulation, nerve conduction, oxidative stress, and inflammation [7]. Despite its biological importance, vitamin D deficiency has become a global health problem, with major contributors such as inadequate dietary intake, malabsorption, age, a sedentary lifestyle, and limited sunlight exposure. Approximately 1 billion people are vitamin D deficient, and about 50% of the global population is insufficient [8]. Prevalence estimates of vitamin D deficiency range from 20% to 90% across Europe, the USA and the Middle East, and similar trends have been reported in Australia, Africa, and South America [9]. Beyond musculoskeletal consequences, vitamin D deficiency has been linked to a spectrum of chronic diseases, including cardiovascular diseases, autoimmune disorders, cancers, and infections, as well as metabolic diseases such as diabetes mellitus [10, 11].

Evidence supporting the role of vitamin D in T2DM pathophysiology has grown in recent decades. Experimental and clinical studies have shown that vitamin D potentially regulates insulin secretion and signalling; enhances insulin receptor exposure to target cells; and regulates pancreatic β-cell function and survival [12]. Mechanistically, insulin secretion is a calcium-dependent process, and vitamin D influences the intracellular flux and concentration of calcium. Furthermore, vitamin D has immunomodulatory effects by preventing inflammation and β-cell dysfunction via VDR expressed on antigen-presenting cells, activated T-cells and islet β-cells. These effects have been demonstrated in a study of non-obese diabetic mice using 1,25(OH)D, where 1,25(OH)D-deficient mice showed a tendency to develop a more aggressive form of T2DM [6]. Vitamin D thus protects against chronic low-grade inflammation, a major process involved in the development of insulin resistance and T2DM progression [12, 13].

Nevertheless, despite mounting evidence, gaps remain in understanding how vitamin D deficiency interacts with hyperglycaemia and inflammation in real-world clinical populations, particularly in sub-Saharan Africa, where the diabetes burden is escalating and vitamin D deficiency is prevalent but under-researched. This study was therefore designed to investigate the relationship between vitamin D deficiency, inflammation, and hyperglycaemia in patients with T2DM attending the Laquintinie Hospital of Douala. By investigating these interactions, the findings may provide insights and inform strategies for using potential models that target these pathways to address this disease condition, such as developing new treatment protocols or public health initiatives aimed at managing vitamin D levels and inflammation in T2DM patients.

2. Materials and Methods

2.1 Study area

This study was conducted at Laquintinie Hospital, located in the Akwa district of Douala, the economic capital of Cameroon. Douala is a cosmopolitan city with over 3.7 million inhabitants, situated on the Atlantic coast (3°48' N 10°08' E), 19 m above sea level. The city experiences a tropical monsoon climate with a mean annual temperature of 27°C and relative humidity of 87%. The Laquintinie Hospital is a second-category referral and teaching hospital covering over nine hectares. It is a particularly important health structure for the population due to the affordability of its wide range of services and specialised care, including a diabetes and endocrinology unit that served as the recruitment site for this study.

2.2 Study Design and Study Population

This study was a hospital-based cross-sectional study carried out from the month of March 2022 to May 2022. A total of 125 consenting T2DM participants (determined using Lorentz's formula) aged 25 years and above were enrolled by convenience sampling. Participants were recruited from the diabetic consultation unit and inpatient wards of the Laquintinie Hospital. However, the study excluded pregnant women, patients taking vitamin D supplements or medications that affect vitamin D metabolism, individuals with chronic metabolic diseases, those with chronic liver disease, and overweight or obese patients.

2.3 Sample Collection and Storage

After obtaining informed consent, venous blood samples (4 mL) were collected from each participant by venipuncture and placed into appropriately labelled ethylenediaminetetraacetic acid (EDTA), sodium fluoride, and dry (red top) test tubes. Blood samples in the dry tubes and sodium fluoride tubes were centrifuged (BIOBASE model BCK-TL511) at

3000 rpm for 5 minutes. Serum was transferred into appropriately labelled Eppendorf tubes using sterile micropipettes and stored at -80°C for subsequent batch analysis of vitamin D, hs-CRP, and creatinine. Plasma from sodium fluoride tubes was used for the measurement of fasting plasma glucose levels post centrifugation, while whole blood samples from EDTA tubes were used for analysis of HbA1c shortly after collection.

Fasting plasma glucose levels were measured by colorimetry using the Biolabo Glucose GOD-PAP reagent kit, and results were read at an absorbance of 500 nm using a spectrophotometer (Genrui WP21A). Normal glucose levels (good glycaemic control) were considered for patients with FBS values from 70 to 130 mg/dL, whereas those with FBS >130 mg/dL were recorded as elevated glucose levels (poor glycaemic control).

The measurement of the HbA1c fraction of haemoglobin was done by capillary electrophoresis using the MINICAP HbA1c commercial kit on the MINICAP FLEX-PIERCING analyser, and readings were done at 415 nm. HbA1c levels < 6.5% were considered normal HbA1c levels (acceptable glycaemic control), whereas levels ≥ 6.5% were considered elevated HbA1c levels (poor glycaemic control).

The measurement of total serum vitamin D concentration was done by fluorescence immunoassay technique using the Finecare™ vitamin D rapid quantitative test commercial kit, which measures total serum vitamin D (25(OH)D_2/D_3), with the following interpretation criteria: (i) (i) (i) (i) Deficient (≤ 20 ng/mL); (ii) Insufficient (20 - 30 ng/mL); (iii) Sufficient (30 - 100 ng/mL); (iv) Toxicity (> 100 ng/mL).

Serum hs-CRP levels were quantitatively measured by the nephelometry technique using the Genrui hs-CRP detection kit, and readings were done using the Genrui PA54 analyser. Levels ≤ 3 mg/L were considered "normal", while those > 3 mg/L were interpreted as indicative of inflammation.

Serum creatinine concentration was measured to evaluate kidney function. Analysis was done kinetically using the Biolabo creatinine reagent kit on the Genrui WP21A semi-automated spectrophotometer. Normal ranges of 0.9 to 1.3 mg/dL were considered for men and 0.6 to 1.1 mg/dL for women. The values of eGFR for each participant were then calculated using the 2021 Chronic Kidney Disease Epidemiology Collaboration's (CKD-EPI) creatinine equation based on their serum creatinine levels, age, and sex to assess nephropathy. An eGFR > 60 mL/min/1.73 m^2 was interpreted as "normal kidney function".

2.4 Data Management and Analysis

Questionnaire data and laboratory results were coded, logged, and stored in a secure database. Statistical analyses were performed using the Statistical Package for Social Science (SPSS) version 28.0. Continuous variables were expressed as mean ± standard deviation (mean ± SD), and categorical variables as frequencies and percentages. The student's *t*-test was used to assess the difference between means for parametric variables. The chi-squared (X^2) was used to measure the statistical difference between the frequencies of categorical variables. The Pearson's correlation test was used to measure statistical relationships between continuous variables. A multivariate analysis was done for variables with multiple outcomes. All tests were considered significant at a *p*-value < 0.05.

2.5 Ethical Considerations

Ethical clearance was obtained from the Faculty of Health Sciences Institutional Review Board, University of Buea (IRB/FHS-UB) on 16th March 2022. Administrative authorisation was granted by Laquintinie Hospital, Douala, on March 24, 2022. All participants who voluntarily agreed to take part in the study provided written consent prior to enrolment. Confidentiality was ensured through unique participant codes, with identifiers stored securely.

3. Results

3.1 Sociodemographic data of study participants

A total of 125 participants were enrolled in this study. The majority were females (57.6%, n=72), while males accounted for 42.4% (n=53) of the study population. Participants' ages ranged from 25 to 89 years, with a mean ± SD age of 56.15 ± 13.76. The largest proportion were within the 45-64 years age group (52.8%, n = 66). Most participants were married (67.2%, n=84), and the highest educational level attainment was secondary education (38.4%, n=48), while 18.4% (n=23) had attained tertiary education. Participants represented 9 out of the 10 regions of the country, with the majority from the Littoral region (36.8%, n=46).

3.2 Vitamin D Status in Study Participants

The mean serum concentration of vitamin D among participants was 28.46 ± 12.96 ng/mL. None of the participants enrolled had vitamin D toxicity (>100 ng/mL). Of the total study population, 33.6% (n=42) had sufficient levels of vitamin D, while 66.4% (n=83) presented with hypovitaminosis D. The prevalence of vitamin D deficiency in participants was 28.8% (n=36).

Hypovitaminosis D was more prevalent among females (69.4%, n=50) than males (62.3%, n=33). Hypovitaminosis D was seen to be most prevalent in participants aged ≥ 65 years (70%, n = 21) and in participants with diabetes duration > 15 years (80%, n = n=12). Participants who had attained tertiary education had the highest prevalence of hypovitaminosis D (69.6%, n=16). However, these associations were not statistically significant, with all p-values > 0.05.

3.3 Glycemic Status in Study Participants

3.3.1 Fasting Plasma Glucose Status in Study Participants

The majority of the participants had normal levels of FPG (n=76, 60.8%), while 38.4% (n=48) had elevated FPG levels. Females accounted for most participants with normal levels of FPG (66.7%, n=48). A greater proportion of males (46.2%) than females (33.3%) presented with elevated FPG levels. However, there was no significant association between FPG status and sex in participants (p-value = 0.077). Participants aged 25-44 years had the highest proportion of elevated FPG levels (48.3%, n=14). There was also no significant association observed between FPG levels and the ages of participants (p-value = 0.148).

3.3.2 Glycated Haemoglobin Status in Study Participants

The majority of the study participants (81.6%, n=102) had elevated HbA1c levels, while 18.4% (n=23) had normal HbA1c levels. Females (56.9%, n=58) accounted for more cases of elevated HbA1c compared to males (43.1%, n=44). Participants aged 25-44 years had the highest proportion (86.2%, n=25) of elevated HbA1c levels. Neither sex (p-value = 0.506) nor age (p-value of 0.685) was significantly associated with HbA1c status. Results of analysis showed a significant positive correlation (r=0.268, p=0.003) between fasting plasma glucose levels and glycated haemoglobin levels.

3.4 Relationship between Vitamin D Status and Glycaemic Markers

3.4.1 Relationship between Vitamin D Levels and Fasting Plasma Glucose

The mean serum vitamin D concentration was higher among participants with normal FPG levels (30.19 ± 13.01 ng/mL) compared to those with elevated FPG concentrations (25.85 ± 12.66 ng/mL). However, this difference was not statistically significant (p-value = 0.07). Among the 76 participants who had normal FPG levels, 61.8% (n=61) had hypovitaminosis D, while 38.2% (n=29) had sufficient serum vitamin D levels. In participants with elevated FPG levels (38.4, n=48), 72.9% (n=35) had hypovitaminosis D, while 27.1% of participants (n=13) had sufficient vitamin D levels. A significant inverse linear relationship (r=-0.214*,* p=0.016) was observed between FPG and serum vitamin D levels in participants (Figure 1).

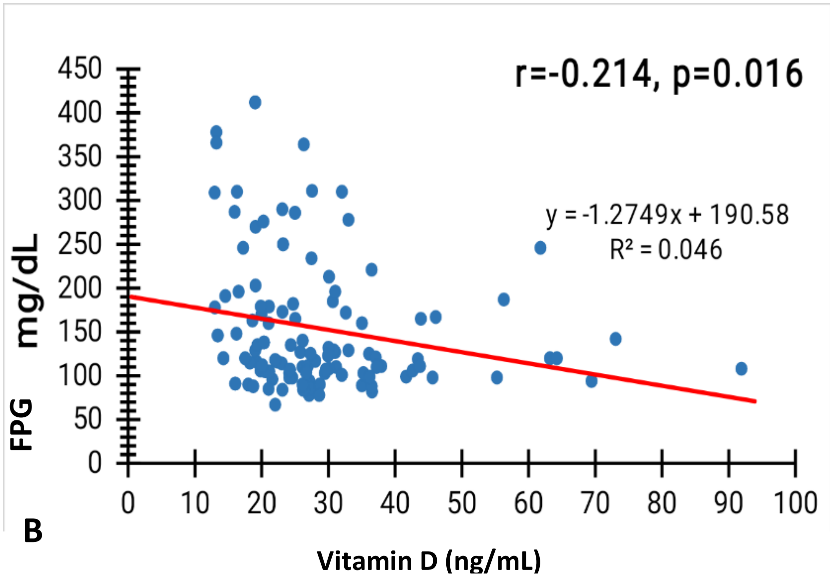

Figure 1: Correlation between vitamin D and fasting plasma glucose

3.4.2 Relationship between Vitamin D Levels and Glycated Haemoglobin

There was no statistically significant difference (p-value = 0.752) in the mean serum vitamin D concentration between participants with normal HbA1c levels (28.29 ± 1.30 ng/mL) when compared to those with high HbA1c levels (29.24 ± 2.61 ng/mL). Among the 102 (81.6%) participants with elevated HbA1c, 67.6% (n=69) had hypovitaminosis D. Whereas, among the 18.4% (n=23) of participants with normal HbA1c levels, 60.9% (n=14) had hypovitaminosis D, while 39.1%

(n=9) had sufficient vitamin D levels. Figure 2 shows a nonsignificant inverse linear relationship (*r*=-0.91, *p*=3.11 and R^{2}=0.0186) between HbA1c levels and serum vitamin D levels.

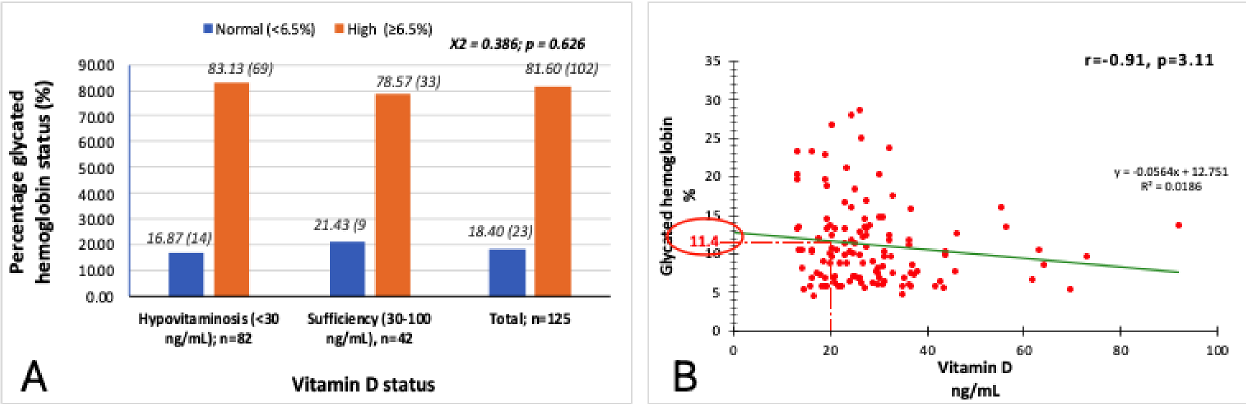

Figure 2: Relationship between vitamin D and glycated haemoglobin levels.
A: Association between vitamin D status and glycated haemoglobin levels; B: Vitamin D and glycated haemoglobin levels are correlated.

3.5 Relationship between vitamin D levels and high-sensitivity CRP levels

The mean serum vitamin D concentration was significantly higher (*p*-value < 0.001) in participants with normal hs-CRP levels (42.47 ± 17.17 mg/L) compared to those with elevated hs-CRP levels (25.30 ± 9.33 mg/L). A statistically significant association (p-value < 0.001) existed between hypovitaminosis D and elevated hs-CRP levels. Among the 102 participants with elevated hs-CRP levels, 78.4% (n=80) had hypovitaminosis D, while among participants with normal hs-CRP levels, 13% (n=3) had hypovitaminosis D.

A strong inverse relationship (*r* = -0.394, p-value < 0.001) was observed between hs-CRP and vitamin D levels, indicating hs-CRP levels tend to increase with decreasing levels of serum vitamin D (Figure 3).

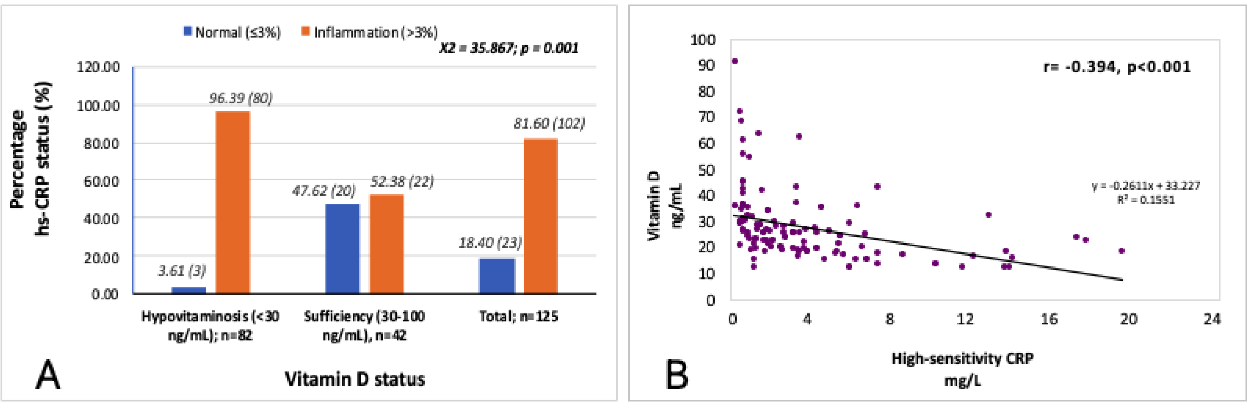

Figure 3: Relationship between serum vitamin D levels and hs-CRP levels.
A: Association between vitamin D status and hs-CRP levels; B: Correlation between vitamin D levels and hs-CRP levels.

3.6 Relationship between Vitamin D and Diabetic Complications

Among the participants, 31.2% (n=39) had hypertension, 17.6% (n=22) had neuropathy, 24% (n=30) had retinopathy, 15.2% (n=19) had diabetic foot ulcers (DFU), 20.8% (n=26) had cardiovascular disease (CVD) and 29.6% (n=37) had nephropathy. In each case, the prevalence of hypovitaminosis D was higher among participants with complications compared to those without.
Despite these trends, none of the associations between vitamin D status and complications was statistically significant. A multivariate analysis confirmed the absence of significant relationships between serum vitamin D levels and diabetic complications (Table 1).

Table 1: Multivariate analysis of the association between vitamin D and diabetic complications

Diabetic complications	Category	Vitamin D Status (mg/L)		
		Deficiency <20 n (%)	Insufficiency 21-29 n (%)	Sufficiency 30-100 n (%)
Hypertension	No	23 (26.7)	36 (41.9)	27 (31.4)
	Yes	14 (35.9)	16 (41.0)	9 (23.1)
P-value = 0.593; X² = 1.046				
Neuropathy	No	29 (28.2)	42 (40.8)	32 (31.1)
	Yes	8 (36.4)	10 (45.4)	4(18.2)
P-value = 0.542, X² = 1224				
Retinopathy	No	27 (28.4)	44 (46.3)	24 (25.3)
	Yes	10 (33.3)	8 (26.7)	12 (40)
P-value = 0.142; X² = 3.898				
Diabetic Foot Ulcers	No	31 (29.2)	43 (40.6)	32 (30.2)
	Yes	5 (26.3)	10 (52.6)	4 (21.1)
P-value = 0.714; X² = 0.674				
Cardiovascular disease	No	27 (27.3)	42 (42.4)	30 (30.3)
	Yes	9 (34.6)	11 (42.3)	6 (23.1)
P-value = 0.662, X² = 0.825				
Nephropathy eGFR < 60 ml/min/1.73 m²	No	24 (66.7)	36 (69.2)	27 (75.0)
	Yes	13 (34.2)	16 (42.1)	9 (23.7)
P-value = 0.728, X² = 0.635				

4. Discussion

Several studies have reported high prevalences of vitamin D deficiency in T2DM patients. From this present work, the prevalence of hypovitaminosis D recorded was 66.4%, with vitamin D insufficiency and deficiency accounting for 40.8% and 25.6%, respectively. This result aligns with findings from a similar study in Kenya by Karau *et al.* (2019), which recorded a 60.3% prevalence in hypovitaminosis (21.9% insufficiency and 38.4% deficiency) [14]. However, the prevalence of hypovitaminosis D recorded in our study was considerably lower than reports from Europe, North America and Asia. For instance, in a study conducted in Saudi Arabia, the prevalence of hypovitaminosis D was 98% (38.6% insufficiency and 59.8% deficiency) [15]. The wide differences in prevalence may be explained by differences in genetics (skin pigmentation), dietary habits, cultural practices, and climate, which can all influence vitamin D synthesis and absorption in the body.

There is currently limited data reporting the prevalence of vitamin D deficiency in Cameroon. However, the prevalence of hypovitaminosis D recorded in this hospital-based study is about twofold the 25.8% previously obtained in 2018 by Tangoh *et al.* on the general population of the Southwest region of Cameroon within the same age group as our study participants [11]. This finding might be a reflection of the relationship between T2DM and vitamin D levels, as highlighted by Mezza *et al.* [13].

A significant inverse relationship was observed between serum vitamin D and FPG levels, consistent with results reported in similar studies [14, 16]. A study by Hurskainen *et al. in* Finland (2012) showed an inverse association between vitamin D levels and FPG, fasting plasma insulin and the 2-hour glucose tolerance test [17]. Although HbA1c did not significantly correlate with serum vitamin D levels in our study, similar null findings have been reported. A similar study in 2021 by Tran *et al.* [18] showed no significant association between HbA1c and serum vitamin D levels in T2DM patients. These same authors also reported that patients with better glucose-related parameters, such as FPG and insulin resistance index, had a better vitamin D status and glycaemic control-related factors, such as physical exercise. The 2018 community-based longitudinal study conducted by Alkhatatbeh and Abdul-Razzak [19] showed that participants with normal glucose levels but hypovitaminosis D faced a higher risk of developing prediabetes or T2DM by the end of the study. These findings provide supportive evidence that vitamin D deficiency is associated with impaired glucose metabolism [20]. In 2013, Salehpour *et al.* went further to investigate the effects of vitamin D supplementation in healthy overweight and obese women [21]. It was observed that increasing serum 25(OH)D concentrations by vitamin D3 supplementation led to body fat mass reduction, suggesting a potential role of vitamin D in lean body mass development and inhibition of the development of adipocytes.

While the importance of vitamin D in diabetes pathology and outcome management is becoming evident, scientists are getting more interested in the potential role of inflammation in both type 1 and type 2 diabetes (T1D and T2D) pathophysiology, as well as associated metabolic disorders. Several anti-inflammatory drugs are actively being explored for their potential in the prevention and management of diseases. However, such data is limited in Cameroon. The present study showed a strong significant inverse association between hypovitaminosis D and hs-CRP, which is consistent with other studies in T2DM, reflecting the interplay between vitamin D status and systemic inflammation [22, 23]. A cross-sectional study carried out by Haidari *et al.* on non-obese T2DM patients showed that the inflammatory cytokine TNF-α plays a significant role in inducing insulin resistance through increased systemic inflammation and was significantly elevated in participants with low serum vitamin D levels [24]. This agrees with experimental studies, which showed that vitamin D is capable of suppressing TNF-α production [25]. Our findings also underscore a positive linear correlation between FPG levels and hs-CRP levels. This finding is in line with a study carried out by Babu *et al*., 2017, which indicated that FPG levels increase with increased levels of hs-CRP [22]. Another study investigating the relationship between glycated haemoglobin and hs-CRP in T2DM found that HbA1c was found to increase significantly as hs-CRP increases [23]. Recent studies provide evidence that supports the link between hyperglycaemia and inflammation. A case-control study carried out by Yang *et al. (2017)* on T2DM showed a higher fasting plasma insulin in the group with increased levels of hs-CRP compared to subjects with lower hs-CRP levels [26]. A previous study reported that increased hs-CRP levels were associated with increased insulin resistance [27]. It is currently hypothesised that low-grade chronic inflammation induces insulin resistance by triggering synthesis of hs-CRP along with other inflammatory cytokines. Although the findings from numerous studies show the relationship between increased levels of CRP and diabetes, the molecular mechanism of the pathways involved is not fully clear yet.

There was no association seen between vitamin D and any of the diabetic complications recorded in this study (neuropathy, nephropathy, retinopathy, diabetic foot ulcer, and cardiovascular disease). This result was not in line with the NHANES study and several other studies, which reported that vitamin D deficiency is associated with an increased risk of micro- and macrovascular complications in diabetic patients [28]. Our results, however, are consistent with a cross-sectional study carried out in China investigating the association between vitamin D status and diabetic complications, which reported no significant association between vitamin D deficiency and retinopathy and nephropathy, though an association with diabetic foot ulcer was observed [29]. This finding contrasts with a similar study that demonstrated a relationship between vitamin D levels and the occurrence and severity of diabetic nephropathy and neuropathy, while showing no significant relationship between vitamin D levels and retinopathy [30]. This is in line with several other studies that speculated that, although VDR is present in the vascular endothelial cells of the retina, low vitamin D levels have little effect on retinopathy [30]. A positive association was seen between vitamin D levels and nephropathy in a study carried out on T2DM patients in Korea; however, there was no association with diabetic retinopathy and neuropathy [31]. Limited studies have investigated the association between vitamin D levels and CVD in diabetic patients. Li *et al.* (2025) demonstrated a non-linear correlation between cardiovascular disease risk and vitamin D levels [32]. A meta-analysis carried out by Iannuzzo *et al.* (2018) recorded that patients with peripheral arterial disease (PAD) had lower vitamin D levels than controls, and both vitamin D deficiency and vitamin D insufficiency are significantly associated with PAD [33]. Berghout *et al.* (2019) found that serum vitamin D levels were associated with the prevalence of stroke but not with the incidence; only severe deficiency was associated with stroke [34]. Several research studies indicated that decreased vitamin D levels are associated with increased risk of hypertension and T2DM. Al-Tu'ma *et al.* found that low vitamin D levels were associated with diabetic hypertensives compared to non-diabetic hypertensives. This could be due to the inhibitory effects of vitamin D on renin secretion and activity, thereby acting as a negative regulator of the renin-angiotensin-aldosterone system (RAAS) and its antiproliferative effects on vascular smooth muscle cells [35].

5. Conclusion

Overall, our findings demonstrate a high prevalence of hypovitaminosis D among T2DM patients in Cameroon, with significant associations with high FPG levels and hs-CRP but not with diabetic complications. These results call attention to the need for more longitudinal and interventional studies to clarify the role of vitamin D in diabetes pathophysiology and to explore the potential benefits of vitamin D supplementation as an adjunct in the management of T2DM patients.

Authors' Contributions

Delphine Anye Tangoh and Denis Zofou conceived the work, oversaw the laboratory work, and drafted the manuscript; Nkwa Brian Esong conducted the bench work and took part in drafting the manuscript. All the authors approved the last version of the work and its submission to the journal.

Conflict of Interest Statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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