



Association of Vitamin D Status with Renal Function Markers in Elderly Women with Diabetes Mellitus

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Abstract

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Background: Diabetes is a significant cause of mortality among older adults and is often misunderstood as a chronic disease. Poor management of diabetes can lead to complications like renal dysfunction. Key renal markers, such as urea and creatinine, can be affected by these complications. Additionally, Vitamin D deficiency has been linked to various health issues in diabetic patients.

Objective: To explore the correlation between Vitamin D levels and renal biochemical markers in elderly diabetic women, an area that has been under-researched. The primary goal is to measure serum levels of urea, calcium, creatinine, and Vitamin D, and assess how Vitamin D levels relate to renal function in this population.

Materials and methods: A case-control study was conducted at Nasiriyah Teaching Hospital in Thi-Qar Governorate, Iraq, from November 2024 to March 2025. The study involved 100 women aged 60 to 75, divided into two groups: 50 healthy individuals and 50 diabetic patients. Researchers measured serum levels of urea, creatinine, total calcium, and vitamin D through biochemical and immunoassay methods after collecting venous blood samples. Data analysis was performed using SPSS version 23, employing one-way ANOVA for group comparisons and Pearson's correlation coefficient to assess associations between variables. The diagnostic utility of vitamin D was evaluated using receiver operating characteristic (ROC) curve analysis.

Results: compared to the control group, diabetic patients exhibited considerably greater serum urea [(47.8 ± 11.5 mg/dL)] and creatinine [(1.48 ± 0.33 mg/dL)] levels than the control group [(31.5 ± 7.2 mg/dL and 0.96 ± 0.17 mg/dL, respectively; p < 0.001)] ; p < 0.001]. Diabetic patients had lower serum vitamin D levels [(16.2 ± 5.8 ng/mL)] than the control group [(28.1 ± 6.6 ng/mL; p < 0.00001)]. Serum calcium levels were statistically similar in both groups. Vitamin D had statistically significant negative correlations with serum creatinine [(r = -0.42, p = 0.004)] and urea [(r = -0.32, p = 0.012)], and a positive correlation with total calcium [(r = +0.36, p = 0.007)]. Vitamin D had a good diagnostic accuracy (AUC = 0.82) at a cut-off value of 20 ng/mL based on the ROC analysis.

Conclusions: Elderly women with diabetes mellitus show notable renal biochemical disturbances and significant vitamin D deficiency. There is a close relationship between vitamin D status and renal impairment, evidenced by a negative correlation between kidney performance markers and vitamin D levels. These findings suggest that measuring vitamin D alongside traditional renal biomarkers could improve early detection and treatment of renal issues in diabetic patients.

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1. Introduction

Type 1 diabetes mellitus (DM) is described as an autoimmune destruction of the pancreatic b-cell islets, causing a complete deficiency of insulin. Islet cell destruction is indeed autoimmune, as they are the target for T cells, B cells, and macrophage infiltration, and autoantibodies against the islet b-cells have been documented [1]. Numerous immune-mediated disorders, such

as rheumatoid arthritis, multiple sclerosis, systemic lupus erythematosus, and bowel inflammation, have been connected to vitamin D deficiency.[2,3]. It is asserted that vitamin D insufficiency and Type 1 diabetes are related. Considering that VDR are in human T and B lymphocytes, it is believed that Vitamin D influences the Th1/Th2 cell cytokine response. Additionally, Vitamin D is known to alter immune response by preventing lymphocyte proliferation. Vitamin D deficiency was shown to increase the frequency and severity of diabetes in the NOD (non-obese diabetic) mice [4]. Furthermore, studies showed that by lowering the quantity of effector T cells, 1,25(OH)₂D reduced the prevalence of diabetes in NOD mice. According to one study, 1,25(OH)₂D may modify the expression of Fas, which controls the cytokine-induced death of human islet cells. [1-4].

Sunlight levels and the risk of developing type 1 DM have been studied extensively. Giving newborns in Northern Europe vitamin D drops (which receives fewer daylight hours than the rest of Europe) lowered the risk of developing type 1 DM. Children from this study who were suspected of having rickets had a type 1 DM relative risk (RR) of 3.0 (1.0-9.0); however, the children who ingested 2000 IU vitamin D daily had an RR of 0.22 (0.05-0.89). Some of the studies have been directed to find out and understand the role vitamin D plays in preserving beta (β) cell action following type 1 diabetes[5, 6]. Out of the four studies, two did not show any significant result as far as the administration of vitamin D was concerned in preserving β cell function. One study, however, documented the positive administration effect of vitamin D in preserving β cell function after the onset of type 1 DM. In a randomized control study, 38 patients with recently diagnosed type 1 DM were split into two equal groups and given either a daily dose of cholecalciferol 2000 IU or a placebo. Cholecalciferol intervention group had a significantly enhanced incidence (in comparison to the placebo group) progressive development to fasted and triggered C-peptide levels that are invisible (≤ 0.1 ng/mL).

In a different research, alfalcidol (0.25 μg/d) maintained β-cell activity in kids suffering from recently diagnosed type 1 DM [7,8]. Additional research is required to establish if 25(OH)D or 1,25(OH)₂D can delay the development of type 1 DM [9,10].

The condition known as diabetes mellitus can occur due to various reasons. There can be problems with the secretion of insulin, or problems with the body's response to insulin. Either way, the body will suffer from what is known as hyperglycemia, or the presence of excess glucose in the bloodstream. There is also damage to and chronic dysfunction of various bodily systems. These systems may consist of the heart, blood arteries, nerves, kidneys, and eyes. This damage happens as a result of chronic hyperglycemia, and is often long-term and irreversible. With the rapid increase in population and rates of aging around the world, needless to say, urbanization, and the rise in obesity, and dwindling levels of physical activity, Type 2 diabetes has swiftly emerged as a major worldwide health concern. It is crucial to stop the rising trends of diabetes and manage the complications that come with it. Due to diabetes, chronic kidney disease or End Stage Renal Disease (ESRD) has become the leading cause of death in the United States[11, 12], and in countless other countries, the situation only continues to worsen. It also has severe implications that can be deemed medical, social or economic. There is a progressive mechanism with which diabetes affects the kidneys. Diabetes causes the kidneys to enlarge and disrupt the glomerular filtration rate (GFR) [13].

Recent research in both, the basic and clinical areas has brought attention to the intersections of sclerosis and failure of the kidneys. Regular screening, early detection, and prompt and adequate management of the long-term problems are essential to effectively lessen the consequences of diabetes mellitus on morbidity and death.

During the early stages of developing kidney disease, some of the blood protein known as albumin, begins to seep into the urine, triggering a condition known as microalbuminuria. During this time, the kidneys' filtration functionality usually remains intact. As the disease further advances, progressively greater quantities of albumin spill into the urine, a condition termed macroalbuminuria, or proteinuria[12-14]. With the further increase of protein albumin in the urine, the filtering ability of the kidneys begins to further decline, leading to the bodily retention of a variety of waste byproducts. As this kidney damage rises, blood pressure rises as well, or hypertension may worsen the kidney injury cycle. Pioneering the detection of kidney damage may aid in delaying the continuum of damage. Because of the protracted nature of these processes, damage to the kidneys very

infrequently occurs during the first decade of diabetes, with a time frame of 15 to 25 years usually applying to kidney failure. For patients with diabetes who have lived for more than 25 years without any evidence of kidney failure, the likelihood of developing kidney failure appears to decline [14, 15]. This study aimed to analyze the levels of serum urea, creatinine, total calcium, and vitamin D in older women with diabetes mellitus, as well as to evaluate the connections among certain markers of kidney functioning and vitamin D levels.

2. Materials and Methods

This case-control study was carried out in the Thi-Qar Governorate of Iraq at the Nasiriyah Teaching Hospital, between November 2025 and March 2026. A total of 100 female participants aged 60–75 years were enrolled and divided equally into two groups: 50 patients diagnosed with diabetes mellitus (Group A) and 50 apparently healthy age-matched controls (Group D). Sample size was calculated initially for an infinite population using standard statistical formulas based on a 95% confidence level, a population proportion of 0.5, and an acceptable margin of error, followed by adjustment to the target population.

Diabetes mellitus with elderly women documented in central Thi-Qar was inclusion, and lack of documented hypertension, malignant tumors, or acute infections was exclusion. An ethics committee authorized the study.

In total, 6 mL of each participant's venous blood was collected in an aseptic manner and placed in plain blood tubes. After allowing blood samples to coagulate, they were centrifuged for ten minutes at 3000 revolutions per minute. Serum was separated and kept for analysis at -20 degrees Celsius. Each test was performed two times to achieve repeatability.

In almost all studies, the biochemical parameters included urea, creatinine, total calcium, and vitamin D. Colorimetric methods were used to measure serum urea and the Jaffe procedure and some enzymatic methods were used to find out about creatinine. The o-cresolphthalein complexone (CPC) method was used to find out about the total calcium concentration and competitive ELISA was used to find out about the serum vitamin D. The manufacturer's instructions for the devices measuring the different parameters were followed with the various UV/VIS spectrophotometers and the ELISA readers. SPSS version 23 was used for statistical analysis. The results' mean and standard deviations (SD) were computed. One-way ANOVA was utilized to ascertain group differences, and the coefficients of correlation of Pearson were computed to ascertain the connections among biochemical factors. A significant p-value was one that was less than 0.05.

3. Results

Table 1 and Figures 1-4 summarize the differences in biochemical variables between the diabetic participants and the healthy controls. Diabetic patients had higher serum urea levels (47.8 ± 11.5 mg/dL) than the control group participants (31.5 ± 7.2 mg/dL) with a significant statistical difference ($p = 0.0001$) (Figure 1). Similarly, serum creatinine levels were higher in insulin patients (1.48 ± 0.33 mg/dL) than in controls (0.96 ± 0.17 mg/dL) with a significant statistical difference ($p = 0.0003$) (Table 1, and Figure 2).

Diabetic patients had marginally lower serum calcium levels (8.75 ± 0.48 mg/dL), than healthy individuals (9.02 ± 0.41 mg/dL). Despite this, it was deemed not of statistical significance ($p = 0.071$) (Figure 3, Table 1). The blood vitamin D levels of the ill group (16.2 ± 5.8 ng/mL) and the controls (28.1 ± 6.6 ng/mL) differed significantly ($p = 0.00001$) (Figure 4).

Table 2 and Figures 6-8 displays the results of the Pearson correlation analysis performed on the patients suffering from diabetes. One of the important outcomes of these figures is the high and negative correlation of serum vitamin D and creatinine levels ($r = -0.48$, $p = 0.002$) (Figure 6). Additionally, vitamin D levels and serum urea are also negatively correlated, albeit to a lesser extent, at ($r = -0.32$, $p = 0.012$) (Figure 7). Finally, the correlation of vitamin D and total calcium is positive and significant ($r = 0.48$, $p = 0.037$) (Figure 8).

The analysis of the receiver operating characteristic (ROC) curve, presented in Table 3 and Figure 5, was utilized to differentiate between diabetic patients and healthy controls according to their vitamin D levels. The vitamin D measurement showed a commendable area under the curve (AUC) of 0.82. The optimal cutoff point was determined to be 20 ng/mL, achieving a response rate of 78% and a specificity of 80%.

4. Discussion

The alteration of renal function and disturbed vitamin D status in elderly women with diabetes mellitus compared to healthy age-matched controls, however, is biochemically confirmable. Principal findings are elevated serum urea and creatinine, decreased vitamin D, correlations with vitamin D and renal function parameters, and via ROC vitamin D diagnostic capability, among other findings.

The deficit in renal function, which is a recognized consequence of enduring diabetes mellitus, is unrefuted in the greater than average serum urea and creatinine levels in the subject diabetic individuals [16, 17]. Urea is a reflection of the renal clearance level of nitrogenous waste, while creatinine is an indicator of the glomerular filtration rate (GFR) which is often used to monitor renal function. These findings are in accordance to multiple studies which demonstrate an increase in the urea and creatinine levels of diabetic individuals as a consequence of the involvement of the kidneys, even before any signs of diabetic nephropathy are present. These findings strongly confirm the existence of subclinical renal dysfunction in the elderly diabetic women [18, 19].

The substantial disparity in blood vitamin D levels between the diabetic and control groups is one of the study's main conclusions. Vitamin D insufficiency is frequently seen in people with diabetes and chronic kidney disease, and it is known to be a contributing cause to metabolic and renal problems rather than only a consequence of compromised kidney function. [20, 21, 22]. The vitamin D receptor (VDR) mediates the actions of vitamin D, found in renal tubular cells, podocytes, as well as smooth muscle and immune cells, highlighting its importance in both kidney function and disease [23, 24]. The loss of vitamin D may exacerbate kidney damage through increased inflammation. Diabetic kidney disease is mostly caused by excessive oxidative stress and aberrant renin-angiotensin-aldosterone system (RAAS) activation [25, 26].

The relationship among vitamin D insufficiency and kidney impairment is further supported by the correlation among blood vitamin D levels and serum creatinine as well as urea levels in diabetic individuals. Vitamin D deficiency, defined as low serum 25-hydroxy vitamin D levels, has been linked to declining estimated glomerular filtration rates (eGFR), elevated serum creatinine levels, and an increased risk of chronic kidney disease among diabetics, according to numerous recent large-scale observational and cross-sectional studies [16, 27, 28]. Indeed, It has been shown that a lack of vitamin D contributes to tubular and glomerular damage. In contrast, the activation of vitamin D receptors has been shown to be renoprotective by Down regulating certain inflammatory cytokines, renal interstitial fibrosis, and preserving podocyte mechanical function [20, 22].

On the other hand, diabetic patients and controls exhibited no significant difference in serum calcium levels. This implies that the systemic calcium homeostasis might still be relatively intact, in spite of the significant deficiency in Vitamin D. However, the noted significant positive correlation in the this study between Vitamin D and total serum calcium illustrates the physiological state of dependence Vitamin D status must be in, for calcium absorption and metabolism. Previous studies have reported comparable findings suggesting that the subclinical phase of crudely defined mineral (Calcium) metabolism disruption commences long before the presence of hypocalcemia [17, 24]. This insight reiterates the necessity of monitoring Vitamin D levels status irrespective of serum calcium levels being in the reference range.

The serum vitamin D ROC curve analysis reveals that serum vitamin D proved to have the best diagnostic accuracy in identifying diabetic patients from the healthy controls, with an area under the curve of 0.82 with the sensitivity and specificity being best at the value of 20 ng/ml. This value is aligned with the standard values for vitamin D deficiencies or insufficiencies

[29,30]. Vitamin D is not customarily a diagnostic indicator for diabetes/ renal disease. However, from the current study, the correlation of Vitamin D to renal indicator/ biomarkers does offer the hypothesis that Vitamin D may be a residual indicator of the diabetes/ renal disease and an indicator of the reiterative metabolic/ renal dysfunction.

The relationship among vitamin D insufficiency and the severity and course of diabetic kidney disease is also acknowledged by systematic reviews and meta-analyses [19, 22, 31]. The effects of vitamin D supplementation on kidney health vary widely in the research. Following the injection of vitamin D or vitamin D analogs, several studies observed a decreasing trend in kidney damage and inflammatory markers, which indicated the possibility of a protective effect on the kidneys [21, 32, 33]. The variability that is present in the literature can be attributed to study design, baseline vitamin D status, disease stage, dosage, duration of supplementation, and several other factors.

The current study also has its limitations. From a cross-sectional standpoint, determination of the direction of association will be a purely speculative endeavor. The study is also exclusively based on a female elderly cohort, which will impact the external validity of the findings to other populations and other age and sex combinations. The effects of food habits, UV exposure, levels of parathyroid hormone, vitamin D, and vitamin D supplementation were not examined. The study is nevertheless one of the few to investigate the connection between kidney functioning and vitamin D levels in older women with diabetes.

5. Conclusions

The present study shows that elderly diabetic women demonstrate significant changes to renal biochemical parameters and vitamin D status when compared to healthy age-matched controls. Diabetic patients presented with markedly increased serum urea and creatinine levels and a significant deficiency in serum vitamin D levels. The substantial inverse relationship between serum vitamin D levels and both urea and creatinine indicates that renal dysfunction may be a consequence of vitamin D deficiency in diabetes. The positive correlation between vitamin D and total serum calcium demonstrates that despite the absence of hypocalcemia, vitamin D performs its physiological functions. Moreover, the receiver operating characteristic analysis shows that serum vitamin D has the potential to be an adjunct biomarker to evaluate the diabetic state, and its added usefulness to differentiate presence and absence of diabetes proves the metabolic and renal alterations that accompany diabetes. The findings revealed that vitamin D deficiency is associated with biochemical evidence of renal impairment in elderly women with diabetes. The addition of vitamin D status with typical parameters of renal function should allow for more sensitive and timely detection of diabetes complications.

More longitudinal and intervention type studies need to be done to establish the cause and effect and to determine if treating vitamin D deficiency improves kidney outcomes in individuals with diabetes.

Table 1. Comparison of Biochemical Parameters between Patients and Controls

Parameter	Patients (Mean ± SD)	Controls (Mean ± SD)	P-value
Urea (mg/dL)	47.8 ± 11.5	31.5 ± 7.2	0.0001
Creatinine (mg/dL)	1.48 ± 0.33	0.96 ± 0.17	0.0003
Calcium (mg/dL)	8.75 ± 0.48	9.02 ± 0.41	0.071
Vitamin D (ng/mL)	16.2 ± 5.8	28.1 ± 6.6	0.00001

Table 2. Pearson Correlation Among Parameters in Diabetic Patients

Parameters	Correlation (r)	P-value	Interpretation
Vitamin D vs Creatinine	-0.42	0.004	Significant negative correlation
Vitamin D vs Urea	-0.32	0.012	Significant negative correlation
Vitamin D vs Calcium	+0.36	0.007	Significant positive correlation

Table 3. ROC Curve Analysis

Biomarker	AUC	Cut-off Value	Sensitivity	Specificity	Interpretation
Vitamin D	0.82	20 ng/mL	78%	80%	Good diagnostic accuracy

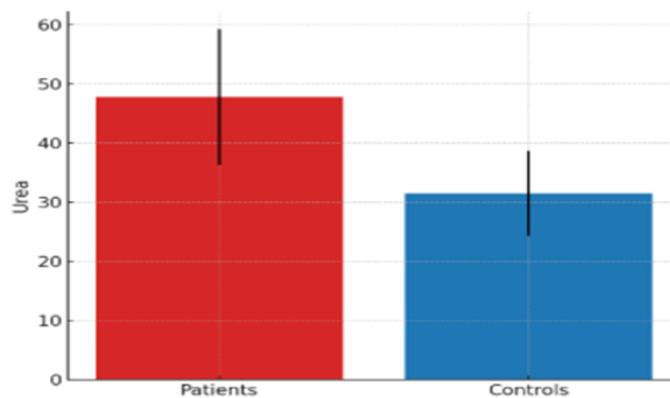


Figure 1. The Urea (mg/dL) in Patients vs Controls

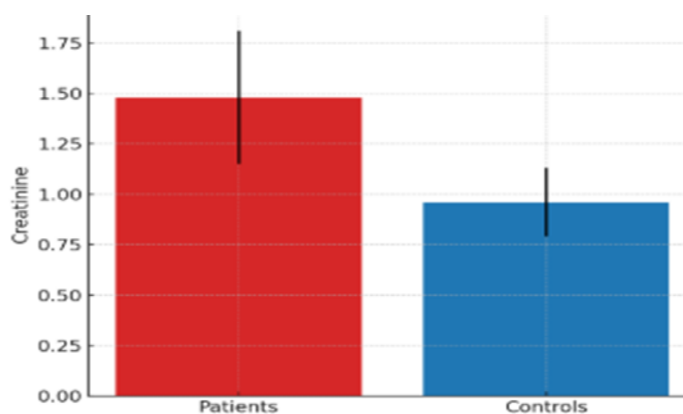


Figure 2. The Creatinine (mg/dL) in Patients vs Controls

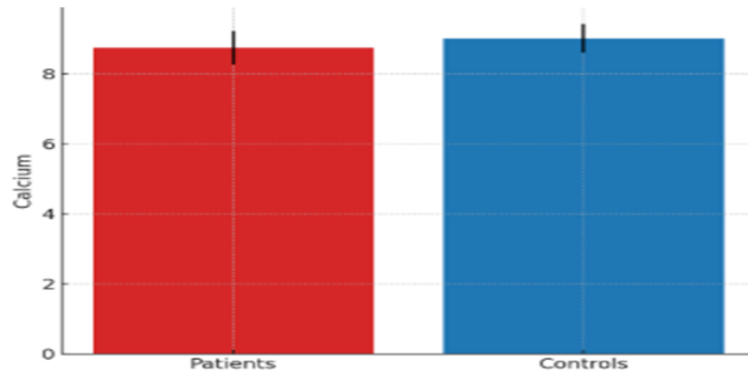


Figure 3. The Calcium (mg/dL) in Patients vs Controls

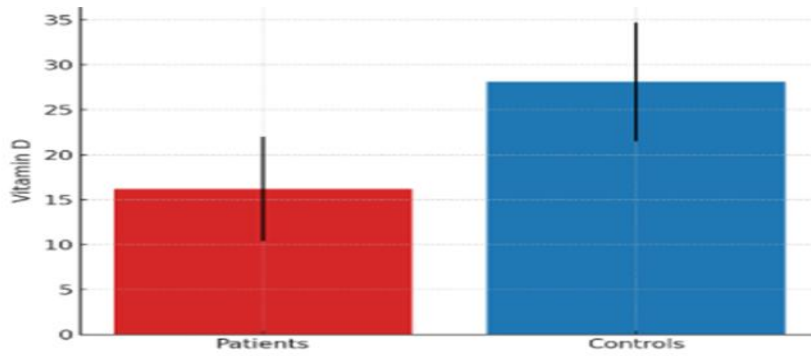


Figure 4. The Vitamin D (ng/mL) in Patients vs Controls.

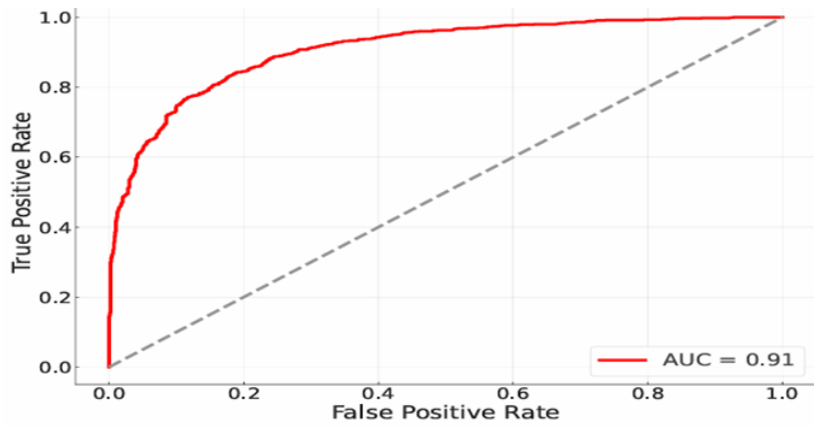


Figure 5. ROC Curve for Vitamin D as a Diagnostic Marker Distinguishing Diabetic Patients from Healthy Controls.

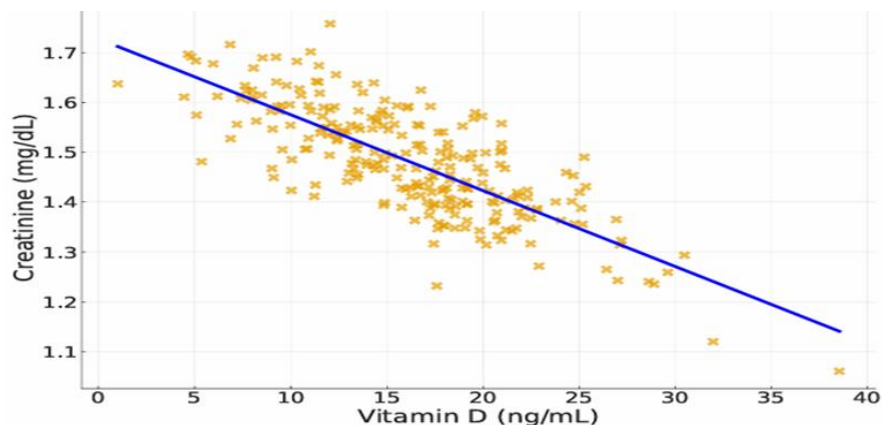


Figure 6. Scatter Plot Showing the Negative Correlation Between Serum Vitamin D and Creatinine Levels in Elderly Diabetic Patients.

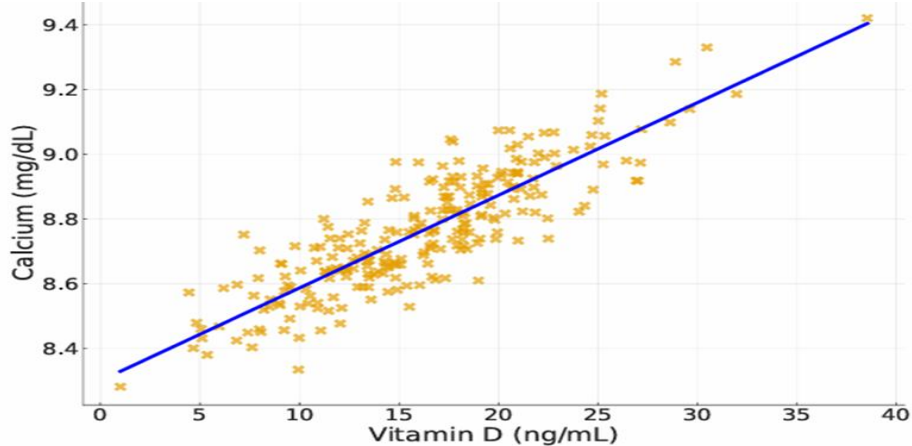


Figure 8. Scatter Plot Illustrating the Positive Correlation Between Vitamin D and Total Serum Calcium in Diabetic Patients.

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