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Assessment of vitamin D in erectile dysfunction patients with type 2 diabetes

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ABSTRACT

Aims: Erectile dysfunction (ED) pathophysiology is a multifactorial condition that is primarily characterized by a vascular disorder that is related to a decrease in endothelial function. We aimed to assess the relation among erectile dysfunction (ED) and serum 25-hydroxyvitamin D (Vit D) levels in men with type 2 diabetes mellitus (T2DM).

Methods: This case–control study was directed on 20 male T2DM patients of at least 5 years' duration, aged 20–60 years, and 20 age-matched healthy controls over a period of one year. All participants were obtained written informed consent before enrollment. All participants underwent full history taking, contain marital and sexual history (libido, frequency, morning erection, orgasm, ejaculation), smoking habits, demographic data, medical history, surgical history, and drug history (antihypertensives, lipid-lowering drugs, and prior ED treatment) A complete general and genital examination (penis, scrotum, testes, epididymis, and cord) was performed.

Results: A significant positive correlation was observed among serum Vit D levels and both erectile function (IIEF, p=0.007, r=0.584) and penile hemodynamic parameter PSV (p<0.001, r=0.848). On the contrary, Vit D was inversely correlated with CAIMT (p<0.001, r=−0.719), EDV (p=0.029, r=−0.536), and cavernous IMT (p<0.001, r=−0.904), indicating its protective vascular role. Regarding laboratory findings, Vit D displayed a significant negative correlation with HbA1c (p=0.013, r=−0.546). Utilizing ROC curve for recognition of best cut off point for Vit D in differentiating control groups and cases, 28.64 ng/mL was detected to have best specificity and sensitivity (65.0% and 70.0% separately). Total accuracy was observed to be 67.5%.

Conclusion: A significant correlation among erectile dysfunction and 25(OH)D deficit in T2DM men. This correlation may be because of 25(OH)D impact on endothelial dysfunction and glycemia.

Keywords: Erectile dysfunction, Penile and vascular ultrasound, Type 2 diabetes, Vitamin D

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INTRODUCTION

Men with type 2 diabetes mellitus (T2DM) are more likely to experience erectile dysfunction (ED), which is the constant incapability to sustain and achieve a creation for pleasing sexual intercourse [1]. Diabetic ED incidence differs from 32% to 90%, depending on

the selected population, duration, age, and diabetes type [2]. Additionally, in 12–30% of men, ED is the first sign of diabetes, which is identified at a later time [3]. In diabetic men, ED is considered beneficial for asymptomatic coronary artery disease screening [4]. Erectile dysfunction pathophysiology is multifactorial, but it is primarily characterized by a vascular disorder that is related to a decrease in endothelial function [5]. A higher risk of vascular disease may be linked to 25-hydroxyvitamin D [25(OH)D] deficiency [6]. In fact, 25(OH)D has been well documented. The known consequences of this compound on endothelial cells include the stimulation of NO production [7], the protection in opposition to oxidative stress [8], and the prevention of endothelial apoptosis [8, 9] through various genomic and non-genomic pathways.

The objective of this work was to assess the correlation among the serum 25(OH)D level and erectile dysfunction in T2DM men.

MATERIAL AND METHODS

This case–control study was directed in the Andrology, Dermatology, and STDs Department, Mansoura University Hospital, Egypt. The study contained 20 male patients with T2DM of at least 5 years' duration aged 20–60 years and 20 age-matched healthy controls over a period of one year. Written informed consent was obtained from all participants before the study.

Exclusion criteria were patients with hyperprolactinemia, type 1 diabetes, history of massive pelvic surgery, severe cardiovascular or neurological disease, severe renal or hepatic dysfunction, diabetes severity, organ failure, infections, or major psychiatric disorders.

Patients were separated into 2 identical groups: Case group with T2DM of at least 5 years' duration and control group were healthy controls.

All participants underwent full history taking, containing demographic data, marital and sexual history (frequency, libido, orgasm, morning erection, ejaculation), smoking habits, medical history, drug history (antihypertensives, lipid-lowering drugs, and prior ED treatment), and surgical history. A complete general and genital examination (penis, testes, scrotum, cord, and epididymis) was performed.

Erectile function was evaluated consuming International Index of Erectile Function (IIEF-5) Arabic version. Erectile dysfunction was categorized as mild (22–25), mild-to-moderate (17–21), moderate (11–16), or severe (1–10).

Laboratory Investigations: Blood samples were attained after overnight fasting. The following were assessed: 2-hour postprandial blood glucose and Fasting, glycosylated hemoglobin (HbA1c), serum cholesterol and triglycerides, serum testosterone and prolactin, and urine analysis.

Penile and Vascular Ultrasound (P-CDU)

Following an intravenous administration of 10 mg alprostadil or 30–60 mg papaverine, P-CDU was instituted. The end-diastolic velocity (EDV), cavernous peak systolic velocity (PSV), and intima-media thickness (IMT) were all measured. Cavernous IMT was categorized as normal (<0.3 mm), increased (0.3–0.4 mm), or plaque (>0.4 mm).

Carotid artery IMT was also assessed by B-mode ultrasound, with values lesser than 0.8 mm considered normal and ≥1 mm connected with increased cardiovascular risk and atherosclerosis.

Vitamin D (Vit D) Measurement

25(OH)D levels were detected utilizing a commercially accessible ELISA kit (Calbiotech Inc., USA), according to the manufacturer's instructions. Based on established cutoff points, patients were characterized as deficient (<25 nmol/L), sufficient (>50 nmol/L), or insufficient (>25 to <50 nmol/L).

Test Principle

Based on competitive binding principle, the reagent is a solid phase enzyme-linked immunoassay (ELISA). At room temperature, the anti-Vit D antibody-coated wells were incubated with Vit D controls, standards, samples, and the Vit D-Biotin conjugate for 90 minutes. A predetermined biotin-labeled Vit D quantity participates with the endogenous Vit D in the standard, sample, or quality control serum for a predetermined binding sites number on the anti-Vit D antibody through the incubation process. Once the wash phase is completed, the bound Vit D-Biotin is identified using Streptavidin-HRP (SA-HRP). As Vit D concentration in the specimen increases, the SA-HRP conjugate that is immunologically bound to the well becomes progressively less.

Next, the wells are cleansed, and the unbound SA-HRP couple is deleted. The subsequent step involves the addition of a TMB Reagent solution, which is then incubated at room temperature for 30 minutes. This process results in blue color formation. The color development is halted by stop solution addition and the absorbance is calculated spectrophotometrically at 450 nm. A standard curve is produced by plotting the absorbance against standard concentration. The color intensity was in reverse proportional to 25-OH Vit D quantity present in the sample. The 25-OH Vit D 2 and D3 are both quantified by one assay. A total of 2.5 hours is required to complete the assay procedure.

Specimen Collection and Handling

The assay can be conducted using serum or heparinized plasma samples. Venipuncture is used to capture whole blood for serum, which is then allowed to clot. Gently invert the sample before centrifugation to mix plasma.

Serum or plasma should be centrifuged and separated as soon as practicable following collection. Hemolyzed samples should not be employed. For two weeks, the specimens may be stored in a refrigerator at a temperature between 2 and 8°C. To prevent the recurrence of freeze-thaw cycles, they can be stored at −20°C for an extended period. The samples must be mixed prior to analysis; they must be allowed to equilibrate to ambient temperature for 30 minutes after being refrigerated or frozen-thawed.

Reagent Preparation

Reagents and standards: They are serum-based solutions that remain stable when stored at −2–8°C, kept from light, till the expiration date specified on the label. Prior to utilization, ensure that the necessary quantity of reagents and standards are brought to ambient temperature. 51× Biotin conjugate: prepare 1× work immediately prior to use. A solution at a ratio of 1:51 with assay diluent (e.g., Include 0.1 mL of the 51. Vit D-Biotin conjugates concentrate must be added to 5 mL of assay diluent. Lasting assay Ensure that the diluent is securely capped and stored in a dark location at a temperature between 2 and 8°C. Add bottle fillings (25 mL, 20×) to 475 mL of deionized or distilled water to prepare 1× Wash Buffer. Be sure to store the item at ambient temperature (20–25°C).

Procedure

Prior to using any reagents or specimens, they must be permitted to reach ambient temperature. All reagents must be combined gently to prevent foaming. It is imperative that all stages of the procedure be completed without interruption once it has commenced.

In each well, dispense 10 µL of 25-OH Vit D controls, samples, and Standards as necessary. Each well should be filled with 200 µL of the biotinylated 25 (OH) reagents working solution. Utilizing a plate sharker at 200–400 RPM (or an equivalent motion), meticulously combine the contents of the wells for 20 seconds. Cover the plate with the adhesive plate seal, confirming that the seal is complete over each well, and remove it from the agitator. At ambient temperature, incubate the sealed plate for 90 minutes. Carefully remove the plate closure. Discharge the fillings of the wells into a waste reservoir by vigorously shaking them. Immediately dispense 300 µL of 1× Wash Buffer into each well and vigorously shake the 1× Wash Buffer into a waste reservoir. Detach any lasting droplets by striking the wells with a pointed object on permeable paper. Repeat this process twice more to achieve a total of 3 washing.

It dispenses 200 enzyme units conjugate (Streptavidin-HRP) into each well. At ambient temperature, incubate for 30 minutes. Discharge wells fillings into a waste reservoir by vigorously shaking them. Then, vigorously shake out the 1× Wash Buffer into a waste reservoir after dispensing 300 µL of the buffer into each well. Sharply strike the wells on absorbent paper to eliminate any remaining particles. Repeat this process twice more to achieve a total of three washing. Use a multi-channel pipette to dispense 200 µL of TMB Substrate into each well. Allow the sample to incubate at room temperature for 30 min, preferably in a dark environment [10]. To halt the enzymatic reaction, dispense 50 µL of Stop solution into each well. Proceed with caution when mixing the contents of the plate for a period of 20–30 seconds.

Statistical Analysis

The statistical analysis was directed utilizing SPSS v27 (IBM, Armonk, NY, USA). Histograms and the Shapiro–Wilks test were implemented to assess data distribution normality. The analytical method employed was the unpaired student t-test, and the quantitative parametric data were presented as standard deviation (SD)and mean. Mann Whitney test was performed to analyze quantitative non-parametric data, which were expressed as the interquartile range (IQR) and median. When qualitative variables, applicable were analyzed using the Chi-square test or Fisher's exact test and presented as percentage and frequency. A linear relationship direction and strength between two normally dispersed continuous variables are determined utilizing the Pearson product-moment correlation. The direction and intensity of a linear relationship among two non-normally distributed continuous variables and/or ordinal variables are detected utilizing Spearman's rank-order correlation. ROC curve analysis is employed to evaluate diagnostic test performance or its ability to discriminate between diseased and non-diseased cases. specificity and sensitivity were determined from the curve, and PPV, NPV, and accuracy were computed through cross-tabulation. Data was analyzed utilizing linear regression to expect the independent variable quantity of a continuous parametric outcome. Regression models were implemented to quantify the united variables' influence on the wanted outcome and the prediction equation ($Y = \beta + \alpha^{*}x$) by incorporating significant predictors from the correlation. A two-tailed p value of < 0.05 was regarded as statistically significant.

RESULTS

The assessment between controls and cases revealed no significant difference in age (p=0.89), indicating that the groups were appropriately matched. Vitamin D levels were significantly less in cases than in controls, both when analyzed as continuous data (median 18.57 vs. 30.59, p=0.012) and when categorized at the 25 nm/L cutoff, with cases having a higher proportion of deficiency (65% vs. 10%, p=0.001). Regarding laboratory findings, cases had higher mean HbA1c values (7.29 vs. 5.90), higher cholesterol (179.75 vs. 166.65), and higher triglycerides

(190.30 vs. 160.39) compared with controls, while estradiol and prolactin levels were slightly higher among cases. In contrast, testosterone levels were lower in cases than controls (6.09 vs. 7.12). However, no statistical tests were reported for these laboratory markers, so differences should be interpreted descriptively (Table 1).

Patients with Vit D deficit had significantly lower IIEF scores (p=0.004), lower PSV (p<0.001), higher end diastolic velocity (p=0.017), and increased cavernous intima-media thickness (p<0.001), indicating more pronounced vascular and ED. Furthermore, the deficient group had significantly higher carotid intimal thickness (p=0.008), indicating an association with systemic atherosclerotic changes. HbA1c levels were also significantly higher in the deficient group (p=0.002), indicating poor glycemic control. In contrast, there were no significant changes in serum cholesterol, triglyceride, estradiol, prolactin, or testosterone levels (Table 2).

Serum vitamin D levels showed a significant positive correlation with erectile function (IIEF, p=0.007, r=0.584) and penile hemodynamic parameter (PSV, p<0.001, r=0.848). Vitamin D was found to have a negative correlation with CAIMT (p<0.001, r=–0.719), EDV (p=0.029, r=–0.536), and cavernous IMT (p<0.001, r=–0.904), indicating its protective role in the vascular system. In terms of laboratory findings, vitamin D showed a significant negative correlation with HbA1c (p=0.013, r=–0.546), indicating that higher vitamin D levels lead to better glycemic control. There were no significant associations found with cholesterol, triglycerides, estradiol, prolactin, or testosterone (Table 3).

Men with vitamin D deficiency (≤25 nmol/L) had significantly higher odds of erectile dysfunction than those with sufficient levels (>25 nmol/L), according to an analysis of erectile dysfunction based on vitamin D status. The computed odds ratio was 16.7 (95% CI: 2.9–95.6, p=0.001), suggesting a significant correlation between erectile dysfunction and vitamin D deficiency Table 4.

The optimal cut-off point for Vit D in distinguishing between control and case groups was determined to be 28.64 ng/mL using the ROC curve, with a sensitivity of 70.0% and a specificity of 65.0%, correspondingly. The total accuracy was determined to be 67.5% (Figure 1).

Table 1: Demographic and laboratory data between both groups (n=40)

Variable	Cases (n=20)	Controls (n=20) M	P value
Age (years)	52.12±8.24	52.50±7.33	0.89
Vit D (nm/L)	18.57	30.59	**0.012***
Vit D >25	7 (35%)	18 (90%)	**0.001***
Vit D ≤25, n (%)	13 (65%)	2 (10%)	
HbA1c	7.29±1.26	5.90±0.90	—
Cholesterol	179.75±110.12	166.65±80.12	—
TGS	190.30±50.7	160.39±44.7	—
Estradiol	50.53±24.04	49.53±22.04	—
Prolactin	12.01±5.7	10.11±3.72	—
Testosterone	6.09±3.50	7.12±3.50	—

Data are presented as mean ± SD, median or frequency (%). HbA1c: Glycated hemoglobin, TGS: Triglycerides. *: significant as p value < 0.05

Table 2: Doppler and laboratory findings among studied cases in relation to Vit D status (n=20)

Parameter	Cases (n=20)	Vit D >25 nmol/L	Vit D ≤25 nmol/L	P value
Doppler				
IIEF	13.35 ± 3.13	15.43 ± 3.59	11.46 ± 1.76	**0.004***
Carotid artery IMT	0.07 ± 0.008	0.061 ± 0.003	0.071 ± 0.009	**0.008***
PSV	25.30 ± 8.01	32.44 ± 2.51	19.45 ± 5.79	**<0.001***
EDV (end diastolic velocity)	6.28 ± 2.63	4.43 ± 0.53	7.27 ± 2.78	**0.017***
Cavernous IMT	0.039 ± 0.01	0.029 ± 0.003	0.047 ± 0.01	**<0.001***
laboratory findings				
HbA1c	7.29 ± 1.26	6.42 ± 0.49	8.01 ± 1.27	**0.002***
Cholesterol	179.75 ± 110.12	120.0	164.0	0.73
TGS	190.30 ± 50.7	176.0 ± 39.61	202.0 ± 57.39	0.26
Estradiol	50.53 ± 24.04	46.72 ± 16.89	53.64 ± 29.09	0.54
Prolactin	12.01 ± 5.7	11.68 ± 6.35	12.27 ± 5.36	0.83
Testosterone	6.09 ± 3.50	6.19 ± 3.58	6.02 ± 3.61	0.92

Data are presented as mean ± SD, or median. IIEF: International Index of Erectile Function, IMT: Intima-media thickness, PSV: Peak systolic velocity, EDV: End diastolic velocity, HbA1c: Glycated hemoglobin, TGS: Triglycerides. *: significant as p value < 0.05.

Table 3: Correlation between serum Vit D levels and Doppler/laboratory findings among studied cases

Parameter	Correlation coefficient (r/Rs)	P value
Doppler findings		
IIEF	0.584	**0.007***
CAIMT	−0.719	**<0.001***
PSV	0.848	**<0.001***
EDV	−0.536	**0.029***
Cavernous IMT	−0.904	**<0.001***
Laboratory findings		
HbA1c	−0.546	**0.013***
Cholesterol	−0.216	0.360
TGS	−0.352	0.128
Estradiol	−0.070	0.769
Prolactin	−0.084	0.724
Testosterone	−0.056	0.816

*Rs: Spearman correlation coefficient; r: Pearson correlation coefficient, IMT: Intima-media thickness, CAIMT: Carotid artery intimal medial thickness, PSV: Peak systolic velocity, EDV: End diastolic velocity, HbA1c: Glycated hemoglobin, TGS: Triglycerides, *: significant as p value < 0.05.

Table 4: Odds ratio of erectile dysfunction by vitamin D status:

Vitamin D status	ED cases/Total	Controls/total	Odds ratio (OR)	95% Confidence interval	P value
≤25 nmol/L	13/20	2/20	16.7	2.9 – 95.6	0.001*
>25 nmol/L	7/20	18/20	1 (reference)	–	–

*ED: Erectile dysfunction; OR: Odds ratio; CI: Confidence interval; *P < 0.05.

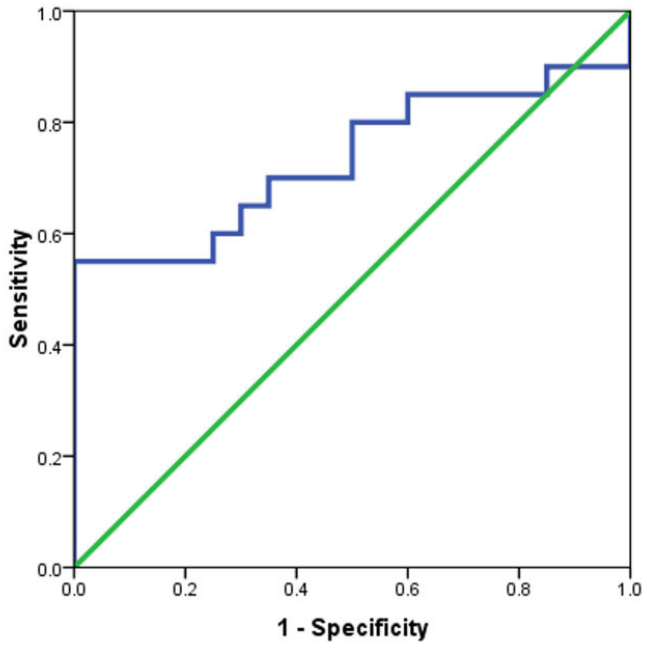

Figure 1: ROC curve for Vit D in differentiating cases and control groups.

DISCUSSION

The inability to attain and/or preserve an erection that is satisfactory to permit satisfactory sexual intercourse is referred to as ED. Many factors, such as vasculogenic, neurogenic, endocrine, and psychogenic factors, can contribute to ED [11].

Diabetes is recognized to induce numerous medical, psychological, and sexual dysfunctions [12, 13]. Impairment of sexual function in men is a well-established diabetes complication. Numerous studies have demonstrated that diabetic men are at an elevated risk for ED, that it manifests at an earlier age [10], and that it is associated with the presence and number of diabetic complications, poor metabolic control, and a prolonged tenure of diabetes [14].

The vascular endothelium is responsible for controlling blood flow and vascular tone in reply to neural, humoral, and mechanical stimuli, in addition to serving as a passive barrier for the venous and arterial blood [15]. A reduction in nitrergic NOS-containing nerve fibres number ,constitutive NOS activity, and decreased neurogenic-mediated smooth and endothelial muscle relaxation are all significant factors related to diabetic ED in human corporal tissue [15, 16].

Numerous pathological conditions are encompassed by endothelial dysfunction, such as dysregulated vascular remodeling, altered anticoagulation, impaired modulation of vascular growth and anti-inflammatory activities [17].

ED is primarily a vascular disorder that is related to a drop in endothelial function, despite the fact that its pathogenesis is multifactorial [5]. 25(OH)D deficiency may be connected to elevated vascular disease risk [6]. Certainly, 25(OH)D has well-established properties on endothelial cells, including the stimulation of NO production [7], the protection of endothelial cells from oxidative stress, and the prevention of endothelial apoptosis [8, 9] through various non-genomic and genomic cellular pathways. Hence, 25(OH)D deficiency could contribute to the development of ED, as endothelial dysfunction has been identified as a critical factor in the conservation and penile erection beginning, as well as atherosclerosis induction [18].

The current investigation was conducted on 20 age-matched cases. The mean age of the cases is 52.5 years, while the control group has a mean age of 52.12 years. A significant difference was detected between the cases and the control group regarding their median Vit D value, with the control group having a higher median value than the cases. The median Vit D level among cases was less than that of the control group, with a 65.5% prevalence of vitamin deficiency compared to a control group of only 10%: 30.59 versus 18.57. Vitamin D deficiency (≤25 nmol/L) was strongly associated with erectile dysfunction, with over a 16-fold increased risk compared to men with higher levels (OR = 16.7, 95% CI: 2.9–95.6, p=0.001).This finding suggests an association between vitamin D status and erectile dysfunction [19].

We determined that the mean ILEF, EDV, carotid artery medial intimal thickness, PSV, and cavernous intimal medial thickness by doppler demonstrate a statistically significant association with vitamin deficit. In contrast to cases with normal Vit D, those with Vit D deficiency exhibited higher mean CAIMT, EDV, and IMT, while those with Vit D deficiency exhibited reduced mean PSV and ILEF. The findings of our investigation were consistent with those of Caretta et al. [20].

In these findings, a significant strong negative correlation was observed between CAIMT by doppler and Vit D level (p<0.001, r=−0.72). This outcome was consistent with Caretta et al findings [20], which indicated a p-value of 0.01 and an r-value of −0.31. In a recent review, it was demonstrated that carotid artery intima-media thickness (CAIMT) is robust future vascular events predictor, involving myocardial infarction and stroke, and is being used more frequently as a surrogate marker of early atherosclerosis [21].

As determined by B mode ultrasound imaging, the typical intima-medial thickness of the common carotid artery was 0.74 ±0.14 mm. According to Mohan et al. [22], there are studies that suggest that a CAIMT value of 0.8 mm or less is associated with healthy individuals, while a CAIMT value of 1 mm or higher is associated with atherosclerosis and a significantly greater cardiovascular risk in any age group.

A strong negative correlation was observed between cavernous intimal media thickness as measured by Doppler and Vit D (p<0.001, r=−0.904). This result was consistent with the study conducted by Caretta et al. [20], which indicated a p-value of 0.009 and a r-value of −0.30. The B mode ultrasound imaging assessment of cavernous intima media thickness (IMT) determined that a cavernous plaque was identified as an IMT 0.4 mm or a two-fold thickening in comparison to the adjacent tracts. Increased normal cavernous IMT (<0.3 mm), cavernous IMT (>0.3 and <0.4 mm), and cavernous plaque (IMT >0.4 mm) were the three categories into which cavernous artery alterations were subdivided [23].

In this study, we observed a statistically significant strong positive correlation between Vit D levels and PSV (p<0.001, r=0.848). EDV exhibited a moderate negative correlation with vitamin D levels (p=0.029, r=−0.49), as demonstrated by the study conducted by Caretta et al. [20] (r=0.23, p=0.042). Caretta et al. [20] reported a p-value of 0.16 and an r-value of −0.16, which was consistent with this outcome.

Our study determined that the mean IIEF exhibited a significant strong positive correlation with Vit D among the cases studied (p=0.007, r=0.584), as demonstrated in the study conducted by Caretta et al. [20]. The study also found that 25(OH)D concentrations reduced as the IIEF-5 score decreased, indicating a significant positive correlation with this parameter (p<0.0001, r=0.39). Additionally, the study conducted by Basat et al. [24] found a moderate positive correlation between the IIEF-5 score and 25(OH)D levels (r=0.21, p=0.038).

The present investigation demonstrated a significant negative moderate correlation between HBA1C and Vit D (p=0.013, r=−0.546).This result was consistent with the findings of the study conducted by Caretta et al. (2016), which detected that the p-value was 0.03, and the correlation coefficient was −0.22 and the investigation conducted by Basat et al. [24], which demonstrated a p-value of 817.

In this study, the mean HBA1C was statistically significantly greater in vitamin deficiency cases than in those with normal Vit D levels (8.01 and 6.42, respectively). Current evidence indicates a correlation between endothelial dysfunction and reduced 25(OH)D plasma levels [6].

According to Al-Tamini and Ali [25], two-thirds of T2DM patients, particularly those with poor glycemic control and prolonged diabetes duration, exhibit a low 25(OH)D status.

Cholesterol, TGS, estradiol, prolactin, and testosterone were not statistically significantly associated with Vit D in our study (p>0.05). This discovery supported the findings of Basat et al. [24], who discovered that LH, testosterone,

prolactin, FSH, and TSH hormone levels were not correlated with 25(OH)D levels and IIEF-5 scores.

Based on the Ludwigshafen Risk and Cardiovascular Health study data, the Wehr group observed a positive correlation between testosterone plasma levels and 25(OH)D, resulting in an agreed-upon model for seasonal variation. Consistent with a previous cross-sectional population-based study that detected a relationship between 25(OH)D deficiency and lower HDL cholesterol (HDL-C) and greater triglycerides levels [26], an association was also observed between 25(OH) D deficiency and known vascular risk factors, including higher triglycerides and lower HDL [27].

Using the ROC curve to determine the optimal cut-off point for Vit D in differentiating control and cases groups, it was determined that 28.64 nm/L had the highest sensitivity and specificity (70.0% and 65.0%, respectively). Total accuracy was determined to be 67.5%.

Our recommendation is that future research is necessary to verify our results and to prospectively assess the impact of 25(OH)D treatment on ED development in T2DM cases. In men with type 2 diabetes, erectile dysfunction was linked to lower vitamin D levels. However, because of the small sample size, multivariable adjustment was not possible, and potential confounders like obesity, diabetes severity, medication use, and lifestyle factors were not fully controlled for. As a result, residual confounding cannot be ruled out, and these results do not suggest that vitamin D plays an independent or causative role. To elucidate these relationships, larger, well-designed studies with suitable control groups and multivariable analyses are required. It is imperative to conduct ED surveillance in all diabetic patients, and screening for DM, which is a primary risk factor for ED, and educational campaigns regarding early treatment advantages and its role in DM complications prevention needed to be implemented widely. Future research should concentrate on the development of simple, non-invasive surveillance tools with an elevated prediction rate for DM-induced ED.

LIMITATIONS

The control group included healthy individuals rather than diabetic men without erectile dysfunction, limiting the ability to distinguish the effects of erectile dysfunction from diabetes on vitamin D levels. Additionally, the small sample size means that correlation and subgroup analyses are exploratory, with a risk of false-positive results.

CONCLUSION

We present a significant correlation between ED and 25(OH)D deficiency in T2DM men. This correlation could be attributed to 25(OH)D impact on endothelial dysfunction and glycemia.

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