Influence of Vitamin D Level on Diabetic Dyslipidemia

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Authors’ contributions

This work was carried out in collaboration between all authors. Author MM designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author AE managed the analyses of the study and author HE managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

\textbf{Background:} Vitamin D deficiency is greater than expected all over the world and it is linked to many health and disease conditions.

\textbf{Aim of the Work:} Our work aimed to study the relation between serum level of vitamin D 25( OH ) and lipid parameters.

\textbf{Patients and Methods:} This study included 176 participants 88 patients with type 2 diabetes mellitus and 88 clinically healthy volunteers age and sex matched persons with normal glycated hemoglobin as a control group and their age ranged from 30 to 60 years old. A full history and clinical examination were done for both patients and control group. Fasting samples (12hrs) for lipid parameters including total cholesterol, triglycerides, (HDL) and (LDH), serum 25 (OH) vitamin D level ,fasting blood sugar and 2 hour postprandial and (HbA1c).

\textbf{Results:} In this study 71 patients had vitamin D deficiency (80.7%) and 17(19.3%) patients had either vitamin D insufficiency or sufficiency. Compared to the control group which showed that 38 persons had deficient vitamin D (43.2%) and 50 (56.8%) had either vitamin D insufficiency or

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1. INTRODUCTION

Type 2 diabetes mellitus is a metabolic disorder characterized by hyperglycemia in the context of insulin resistance and relative lack of insulin [1]. Long-term complications of diabetes include heart disease, strokes, diabetic retinopathy where eyesight is affected, kidney failure which may require dialysis, and poor blood flow in the limbs leading to amputation, it has also been associated with an increased risk of cognitive dysfunction and dementia through disease processes such as Alzheimer’s disease and vascular dementia [2]. Vitamin D receptor and 1α-hydroxylase, which is necessary for the production of the active form of the hormone 1,25(OH)2D (1,25-dihydroxy vitamin D), are present in pancreatic β-cells [3].

Vitamin D stimulates insulin receptor expression and insulin-induced glucose transport in vitro, it also directly regulates fatty acid metabolism in skeletal muscle and adipose tissue, and low concentrations of vitamin D are associated with impaired insulin sensitivity, whereas substitution with vitamin D in the deficient state improves insulin sensitivity [4]. Type 2 diabetes is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities, including reduced high density lipoprotein (HDL) cholesterol, a predominance of small dense lipoproteins particles (LDL) and elevated triglycerides each of these dyslipidemia features is associated with an increased risk of CVD [5]. It was suggested that vitamin D has both direct and indirect effects on modifying the lipid profile and that is one of the proposed mechanisms for the relationship between vitamin D deficiency and CVD [6].

2. PATIENTS AND METHODS

This study included 176 Egyptian subjects, 88 patients with type 2 diabetes mellitus, and 88 clinically healthy age and sex matched volunteers as a control group. Inclusion criteria, Type 2 diabetic patients receiving oral drugs or insulin of any disease duration who are not receiving any lipid lowering drugs, and the exclusion criteria were type 1 diabetic s, patients under 18 years, patients known to have hepatic, renal or other endocrinical disorders that may affect lipid parameters. Serum 25-hydroxy vitamin D level was measured in these patients and in the control to study the relation between vitamin D and lipid parameters in type 2 diabetes mellitus patients compared to the control group. A full medical history and clinical examination were done for both diabetic and control groups including age, sex measuring blood pressure, waist circumference, body weight, height and body mass index, duration of diabetes for diabetics, fasting samples (12 hrs) for lipid parameters including total cholesterol (TC), triglycerides (TG) high density lipoprotein (HDL) and low density lipoprotein (LDL), serum 25(OH) vitamin D level, fasting blood sugar and 2 hour postprandial and glyceded hemoglobin (HbA1c).

3. STATISTICAL ANALYSIS

Collected data were computerized and analyzed using Statistical Package for Social Science (SPSS) version 16. Descriptive statistics were used to describe variables; percent, proportion for qualitative variables. Mean ± SD and range for Quantitative variables. Student’s t-test was used to compare measures of two independent groups of quantitative data.
Chi square test was used to compare two of more than two qualitative groups. P values with significance of less than 5% were considered statistically significant.

4. RESULTS

This study was conducted in Fayoum university hospitals from December 2014 to December 2015 included 176 participants 88 patients with type 2 diabetes mellitus and 88 normal subjects as a control group, and their age ranged from 30 to 60 years old. Diabetic patients were 61 females (69.3%) and 27 males (30.7%) with mean age (47.2 ± 8.5) years old and the control group were 42 females (47.7%) and 46 males (52.3%) with mean age (47.9 ± 10.0) years old.

The study showed that there was a statistically significant difference between diabetic patients and the control subjects as regards the mean values of vitamin D blood level (12.7 ± 10.8 vs. 20.7 ± 8.9) (P < 0.0001) as shown in Fig. 1, and the proportion of participants with vitamin D deficiency was higher among diabetic than the control group (80.7% vs. 43.2%) and the difference was statistically significant as shown in Table 1 and Fig. 2.

![Fig. 1. Comparison of 25(OH) D levels among the study groups](image)

![Fig. 2. Illustrates vitamin D status in the study groups](image)
Table 1. Distribution of study participants regarding vitamin D level

<table>
<thead>
<tr>
<th>Variable</th>
<th>Diabetic(N=88)</th>
<th>Control(N=88)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D blood level (ng/ml)</td>
<td>12.7 ± 10.8</td>
<td>20.7 ± 8.9</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Variable</td>
<td>N (%)</td>
<td>P-value</td>
<td></td>
</tr>
<tr>
<td>Vitamin D deficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficient (&lt;20 ng/ml)</td>
<td>71 (80.7)</td>
<td>38 (43.2)</td>
<td>&lt;0.0001*</td>
</tr>
<tr>
<td>Insufficient &amp; sufficient (≥20 ng /ml)</td>
<td>17 (19.3)</td>
<td>50 (56.8)</td>
<td></td>
</tr>
</tbody>
</table>

ng/ml: nanogram per milliliter

Table 2 and Fig. 3. showed that, among control group, there was a statistically significant difference between participants with vitamin D deficiency compared to subjects with insufficient or sufficient vitamin D as regards the mean values of BMI (34.5 ± 4.8 vs. 25.0 ± 4.4), triglycerides (257.4 ± 106.2 vs. 155.8 ± 108.9), and HDL (30.7 ± 10.1 vs. 44.3 ± 15.5), (P<0.0001). While in diabetic there was a statistically significant difference between patients with vitamin D deficiency compared to those without regarding the mean values of triglycerides (237.3 ± 120.9 vs. 186.3 ± 77.0), and HDL (33.5 ± 6.4 vs. 40.4 ± 13.2) , (P=0.037 and 0.003, respectively).

This study also revealed that there was a negative statistically significant correlation between vitamin D level and HbA1C (P=0.035) in diabetic group.

5. DISCUSSION

Vitamin D is a lipophilic molecule essential for calcium and phosphate balance and osteo-metabolic system regulation. It is produced onto the skin through an UV mediated reaction, then it is metabolized to its active 1,25(OH) D form
through two consecutive hydroxylation exerted by liver and kidney, respectively. In adult population, the prevalence of hypo-vitaminosis D is from 5% to 30%, but it reaches a peak of 75% in patients with metabolic syndrome [7]. Vitamin D is a multi-factorial hormone that affects aspects of metabolism in the human body from immune function to bone metabolism [8]. Vitamin D insufficiency and deficiency are considered serious public health problems. The studies demonstrate that vitamin D deficiency may predispose an individual to metabolic bone diseases and to cancer, insulin resistance, metabolic syndrome, type 2 diabetes and cardiovascular diseases [9].

Vitamin D receptors (VDRs) are present on a large variety of cell types, including myocytes, cardiomyocytes, pancreatic beta-cells, vascular endothelial cells, neurons, immune cells, and osteoblasts [10].

The potential mechanisms for the effects of vitamin D on type 2 DM includes, defects in pancreatic β-cell function, insulin sensitivity, and systemic inflammation. Vitamin D may have a beneficial effect on insulin action either directly, by stimulating the expression of insulin receptor and thereby enhancing insulin responsiveness for glucose transport, or indirectly via its role in regulating extracellular calcium and ensuring normal calcium influx through cell membranes and adequate intracellular cytosolic calcium [Ca2+] pool [11]. It is recognized that type 2 DM is associated with systemic inflammation. Systemic inflammation has been linked primarily to insulin resistance, but elevated cytokines may also play a role in β-cell dysfunction by triggering β-cell apoptosis. Vitamin D may improve insulin sensitivity and promote β-cell survival by directly modulating the generation and effects of cytokines [12].

Lipoprotein lipase (LPL) and serum 25-hydroxyvitamin D (25(OH)D) play important roles in the regulation of lipid metabolism. LPL is also directly or indirectly implicated in some pathophysiological conditions such as insulin resistance and type 2 diabetes. Reduction of LPL is observed in patients with type 2 diabetes and individuals with insulin resistance. Low LPL activity accompanied by high TG was observed in diabetic dyslipidemia [13]. Several mechanisms are recommended for the impact of vitamin D on the serum lipids. In theory, vitamin D could affect the serum lipid levels directly, but also indirectly through its effect on serum parathyroid hormone (PTH) and/or on the calcium balance [13]. Individuals with low serum 25(OH)D concentration are at increasing risk for dyslipidemia, insulin resistance and type 2 diabetes [14].

This study aimed to assess relation between serum level of vitamin D 25 (OH) and lipid profile in type 2 diabetic patients.

Fig. 4. Relation of vitamin D status and dyslipidemias in the study groups

HDL: high density lipoprotein
Table 2. Relation between vitamin D status and lipid parameters

<table>
<thead>
<tr>
<th></th>
<th>Diabetic Deficient (N=71)</th>
<th>Insufficient &amp; sufficient (N=17)</th>
<th>Control Deficient (N=38)</th>
<th>Insufficient &amp; sufficient (N=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P-value</td>
<td>Mean ± SD</td>
<td>P-value</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
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<tr>
<td></td>
<td></td>
<td>34.8 ± 6.3</td>
<td>3.39 ± 5.5</td>
<td>0.623</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>187.3 ± 58.9</td>
<td>170.2 ± 62.2</td>
<td>0.264</td>
</tr>
<tr>
<td><strong>Triglycerides</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>237.3 ± 120.9</td>
<td>186.3 ± 77.0</td>
<td>0.037*</td>
</tr>
<tr>
<td><strong>HDL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>33.5 ± 6.4</td>
<td>40.4 ± 13.2</td>
<td>0.003*</td>
</tr>
<tr>
<td><strong>LDL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>107.7 ± 56.3</td>
<td>101.9 ± 36.1</td>
<td>0.685</td>
</tr>
</tbody>
</table>

*BMI: body mass index, HDL: high density lipoprotein, LDL: low density lipoprotein

Table 3. Relation of vitamin D status and dyslipidemias

<table>
<thead>
<tr>
<th></th>
<th>Diabetic Deficient (N=71)</th>
<th>Insufficient &amp; sufficient (N=17)</th>
<th>Control Deficient (N=38)</th>
<th>Insufficient &amp; sufficient (N=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p-value</td>
<td>N (%)</td>
<td>p-value</td>
<td>N (%)</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>&gt; 200 mg/dl</td>
<td>0.243</td>
<td>26 (36.6)</td>
<td>5 (29.4)</td>
<td>9 (23.7)</td>
</tr>
<tr>
<td>≤ 200 mg/dl</td>
<td>0.576</td>
<td>45 (63.4)</td>
<td>12 (70.6)</td>
<td>29 (76.3)</td>
</tr>
<tr>
<td><strong>Triglycerides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 150 mg/dl</td>
<td>0.563</td>
<td>51 (71.8)</td>
<td>11 (64.7)</td>
<td>33 (86.8)</td>
</tr>
<tr>
<td>≤ 150 mg/dl</td>
<td>0.563</td>
<td>20 (28.2)</td>
<td>6 (35.3)</td>
<td>5 (13.2)</td>
</tr>
<tr>
<td><strong>HDL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 40 mg/dl</td>
<td>0.009*</td>
<td>15 (88.2)</td>
<td>38 (53.3)</td>
<td>35 (92.1)</td>
</tr>
<tr>
<td>≥ 40 mg/dl</td>
<td>0.381</td>
<td>2 (11.8)</td>
<td>33 (46.5)</td>
<td>3 (7.9)</td>
</tr>
<tr>
<td><strong>LDL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 130 mg/dl</td>
<td>1.000</td>
<td>21 (29.6)</td>
<td>3 (17.6)</td>
<td>1 (2.6)</td>
</tr>
<tr>
<td>≤ 130 mg/dl</td>
<td>0.381</td>
<td>50 (70.4)</td>
<td>14 (82.4)</td>
<td>37 (97.4)</td>
</tr>
</tbody>
</table>

HDL: high density lipoprotein, LDL: low density lipoprotein, mg/dl: milligrams per deciliter.
This study included 176 Egyptian subjects, 88 type II diabetic patients recruited from Fayoum university hospitals their age ranged from 30-60 years they included 61 females (69.3%) and 27 males (30.7%).

The control group included 88 clinically healthy volunteers their age ranged from 30-60, 42 of them were females (47.7%) and 46 of them were males (52.3%).

A full medical history and clinical examination were done for both diabetic and control groups including age, sex, measuring weight and height, duration of diabetes for diabetics, blood pressure, body mass index, waist circumference, serum level of 25(OH) vitamin D by ELIZA, fasting plasmas glucose, 2 hour post prandial blood sugar, HbA1c and lipid profile (cholesterol triglycerides, HDL and LDL level).

The results illustrated that, 71 patients (80.7%) had vitamin D deficiency and 17 patients (19.7%) had either vitamin D insufficiency or sufficiency, while the control group showed that 38 persons (43.2%) had vitamin D deficiency and 50 persons (56.8%) had either vitamin D insufficiency or sufficiency.

These results demonstrate that there were a statistically significant difference between diabetic patients compared to the control group as regard to vitamin D level.

The mean value of vitamin D in diabetic group was (12.7±10.8) and in the control group was (20.7±8.9) and the difference was statistically significant as (80.7%) of the diabetic patients have vitamin D deficiency versus (43.2%) of the control group.

This finding was in agreement with Majumder and colleagues [15] who demonstrated the high prevalence (66.34%) of vitamin D deficiency in Gujarati population from India, this was more common in type II diabetic patients (71%) Vitamin D3 level was 17.77±7.19 nmol/L compared to controls (63%) Vitamin D3 level was 24.38±9.30 nmol/L (p<0.05).

In our study it was found that there is a significant negative correlation between vitamin D and HbA1C in diabetic patients.

These findings were also consistent with Majumder and colleagues in 2014 [15] who stated that increase in mean HbA1C level was observed as vitamin D3 level reduced from its normal level in type II diabetic patients, no such changes in mean HbA1C level were observed in controls.

Also, our results agree with Zhang et al. [16] in 2016 who found high prevalence of vitamin D deficiency in Kuwaiti adults, and low vitamin D status was associated with a high prevalence of diabetes as their results suggested that nearly half of the Kuwaiti adults had vitamin D insufficiency and more than one-third had vitamin D deficiency. These together suggest more than 80% of the Kuwaiti adults are at risk for inadequate vitamin D. Also, Zhang et al. [16] in 2016 found that the serum concentration of vitamin D was significantly lower in pre-diabetic and diabetic participants (39.6 and 39.8 ng/ml) than non-diabetic participants (42.9 ng/ml, p = 0.01). When the prevalence odds of pre-diabetes and diabetes were evaluated by vitamin D status in multinominal logistic regression models adjusting for age, vitamin D insufficiency was associated with 70% increased odds of pre-diabetes (OR = 1.9, 95% CI: 1.2–3.0), and vitamin D deficiency was associated with two-fold increased odds of pre-diabetes (OR = 2.1, 95% CI: 1.3–3.5), and these associations were dose-dependent (P_trend = 0.008) [16].

The present study demonstrated that, among the control group hypertriglyceridemia and decrease HDL were significantly associated with vitamin D deficiency (P <0.0001) and the proportion of participants with dyslipidemia was higher among participants with vitamin D deficiency than others (86.8% vs 36.0) and (92.1% vs 32.0%) for hypertriglyceridemia and decreased HDL respectively.

This results agree with Lupton et al. in 2016 who used the very Large Database of Lipids, which includes US adults their study focused on 20,360 subjects who had data for lipids, 25(OH) D, age, gender, hemoglobin A1c, insulin, creatinine, and blood urea nitrogen. Subjects were split into groups based on serum 25(OH)D: deficient (<20 ng/mL), intermediate (≥20-30 ng/mL), and optimal (≥30 ng/mL). The deficient group was compared to the optimal group using multivariable linear regression that denoted that, deficient serum 25(OH)D was associated with significantly lower serum HDL-C (-5.1%) and higher total cholesterol (+9.4%), non-HDL-C (+15.4%), directly measured LDL-C (+13.5%), intermediate-density lipoprotein cholesterol (+23.7%), very low-density lipoprotein cholesterol
oxidative stress in hyperlipidemic patients with type II diabetes mellitus. A double-blind randomized placebo-controlled trial was carried out in 70 participants with type 2 diabetes, aged 30-75 years of age. The participants were randomly assigned to two groups. One group received two capsules of calcitriol (0.25 µg 1,25-dihydroxycholecalciferol per capsule) per day. The second group received placebo tablets. All participants received their oral hypoglycemic drugs as prescribed by the endocrinologist. At the beginning, after 6 weeks, and at the end of the 12-week supplementation trial, serum total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglyceride (TG), and serum malon-di-aldehyde (MDA) levels were measured. They found a significant reduction in total cholesterol, LDL-cholesterol, TG, and MDA levels in both treatment and placebo groups (P < 0.05). Serum HDL-cholesterol level decreased significantly in the placebo group (P < 0.05), while it remained unchanged in the treatment group. However, the P values related to the between group's comparisons were not significant for any variables. They suggested that active vitamin D reduced lipid profile and oxidative stress markers in diabetic patients compared to the control group, but these alterations were not statistically significant [21].

In contrast to this study Jorde et al. [22] showing that 1-year supplementation with 20,000 or 40,000 IU/week vitamin D3 did not improve glucose metabolism or serum lipids in 330 overweight or obese Caucasian subjects. Also, Islam et al. [23] in 2014 studied the effect of vitamin D, calcium and multiple micronutrients supplementation on lipid profile in premenopausal Bangladeshi garment factory workers with hypovitaminosis D. Through placebo-controlled intervention trial conducted over a period of one year randomly assigned a total of 200 apparently healthy subjects aged 16-36 years to 4 groups. The subjects received daily supplements of 400 IU of vitamin D (VD group) or 400 IU of vitamin D+600 mg of calcium lactate (VD-Ca group), or multiple micronutrients with 400 IU of vitamin-D+600 mg of calcium lactate (MMN-VD-Ca group), or the group consuming placebo (PL group). Serum concentrations of lipid and lipoprotein, 25-hydroxyvitamin D (25OHD) and intact parathyroid hormone (iPTH) were measured at baseline and after one year of follow-up. No significant changes in the serum levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), LDL-C/HDL-C

In contrast, Chiu et al. [18] showed no relationship between serum levels of 25(OH) D and TG or HDL cholesterol in healthy subjects, also Garry John and colleagues (2005) conducted a study on 170 UK Bangladeshi adults (69 men and 101 women) with no history of diabetes or other chronic disease. Their data showed that the serum level of 25(OH) D is an independent predictor of fasting apolipoprotein A1. However, no relationship was observed between 25(OH) D and TG or HDL cholesterol [19].

In our study the diabietic group showed a statistically significant difference between patients with vitamin D deficiency and those without regarding the mean values of triglycerides (237.3 ± 120.9 vs 186.3 ± 77.0) and HDL (33.5 ± 6.4 vs 40.4 ± 13.2) with (P = 0.037 and 0.003) respectively. The percentage of patients with decreased HDL was higher among diabetic patients with vitamin D deficiency than those without (88.2% vs 53.3%).

Our findings are similar to the results of Saedisomeilia et al. [20] in 2014 who found that there was a negative, but non-significant, relationship between serum levels of 25(OH) D and that of TG in diabetic patients through a cross-sectional study that was conducted on 108 type 2 diabetics among members of the Iranian Diabetes Association according to study criteria. Fasting concentration of 25(OH) D, calcium, phosphorus, parathyroid hormone (PTH) and lipid profiles (including triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol) were measured. Also they found that the mean serum levels of 25-hydroxyvitamin D (25(OH) D) and PTH were 53.41 ± 33.25 nmol/l and 40.24 ± 18.24 pmol/l, respectively, in type 2 diabetic patients. Prevalence of vitamin D deficiency was 58.34% and vitamin D sufficiency and insufficiency combined was 41.66%. Although in diabetic patients with vitamin D deficiency, serum levels of total cholesterol, TG, and LDL were higher and HDL was lower compared to patients with vitamin D sufficiency, this association was statistically significant only for serum level of TG (145.91 ± 79.00 vs. 122.95 ± 55.82 mg/dl) [20]. Also, Eftekhar et al. [21] in 2014 studied the effect of calcitriol (vitamin D3) on lipid profile and oxidative stress in hyperlipidemic patients with
ratio were observed in the supplemented groups compared to the placebo group. Supplementation had a positive effect (p<0.05) on very low-density-lipoprotein cholesterol (VLDL-C) and triacylglycerol (TAG). A negative correlation between changes in serum iPTH and HDL-C was observed, which indicated that subjects with the greatest decline in S-iPTH had the greatest increase in HDL-C. The results suggest that consumption of adequate vitamin D with calcium or MMN for one-year may have no impact on serum lipid profile in the subjects studied [23].

Our study revealed that there was a negative statistically significant correlation between vitamin D level and HbA1c (P = 0.035) in diabetic group and it was found to be a significant predictor for vitamin D (P=0.018).

Similarly Manickam et al. [24] in 2013 who studied the relationship between glycated hemoglobin (HbA1c) and circulating 25-hydroxyvitamin D concentration in African American (AAM) and Caucasian American (CAM) men. They examined whether (1) serum 25(OH)D level is a risk factor for hyperglycemia, as assessed by glycated hemoglobin (HbA1c), in African American men (AAM) and (2) 25(OH)D is a predictor of HbA1c in AAM and Caucasian American men (CAM).prospectively assessed 25(OH)D and HbA1c in 1,074 men, outpatients with and without diabetes, at an urban Veteran Administration Medical Center (66.8% AAM, 26.4% CAM, 6% Hispanic, 0.4% Asian, and 0.4% Native American men). Multivariate regression analyzed the determinants of HbA1c after accounting for potential confounders.

They found high prevalence of low (< 30 ng/mL) 25(OH)D (81%) and elevated (≥5.7%) HbA1c (53.5%). The 25(OH) D was inversely associated with HbA1c in all men (r = -0.12, P<.001), in AAM (r = -0.11, P = .003), and in CAM (r = -0.15, P = .01). In the entire group the independent determinants of HbA1c included body mass index (BMI), age, 25(OH)D levels, systolic blood pressure (BP), triglycerides, high-density lipoprotein (HDL), and current alcohol use (P<.0001, .01, .02, and .03, respectively), while age had borderline significance (P = .06). In CAM, these included BMI, age, and triglycerides (P = .01, .03, and .004, respectively) but not 25(OH)D levels (P =.50). They found that circulating low 25(OH) D is a risk factor for hyperglycemia, as assessed by HbA1c, in AAM and this agree with our result [24].

6. CONCLUSIONS

This study showed that, the distribution of participants as regards dyslipidemia, the dyslipidemias was higher among diabetic than the control group for hypercholesterolemia, hypertriglyceridemia, and increased LDL.

It was found that the proportion of participants with vitamin D deficiency was higher among diabetic than the control group (80.7% vs. 43.2%) and the difference was statistically significant (P < 0.0001). Also among the control group subjects with vitamin D deficiency had higher triglycerides and lower HDL compared to subjects with deficient or sufficient vitamin D. On the other hand, in diabetics only decreased HDL was higher among diabetic patients with vitamin D deficiency compared to diabetics with insufficient or sufficient.

This study also revealed that there was a negative statistically significant correlation between vitamin D level and HbA1C in diabetic group.

CONSENT

As per international standard or university standard, patient's written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES


22. Islam MZ, Shamim AA, Akhtaruzzaman M, et al. Effect of vitamin D, calcium and multiple micronutrients supplementation on lipid profile in pre-menopausal...


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