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Original Article

RELATIONSHIP OF VITAMIN D STATUS WITH INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background

There is several evidence suggest that altered vitamin D (25Hydroxy (OH) D] and calcium homeostasis may play role in development of Type 2 diabetes mellitus (T2DM) and insulin resistance.

Objectives

The present study was designed to assess vitamin D status (25(OH) D) among patients with T2DM and to investigate the association between serum 25(OH) D levels with insulin resistance.

Methods and Materials

Eighty patient (30 males and 50 females) with T2DM and mean age (50.51± 10.70) were recruited from the diabetic center in Al-Mawane General hospital in Basrah governorate from 20th November 2014 to 25th April 2015. After an overnight fasting blood samples were collected for laboratory measurement of biochemical parameters [25(OH) D, insulin, fasting blood glucose, HbA1c].

Results

The present study revealed that serum 25hydroxy vitamin D had a mean value of (19.94 ± 9.59) ng/ml. This level showed a significant inverse relationship with insulin resistance (HOMA-IR) and HbA1c (p-value < 0.05). While the relationship between serum 25(OH) D with insulin sensitivity (HOMA-IS) revealed a positive significant association, on the other hand statistically significant not independent association was found with FBS and HOMA-%β.

Conclusions

Low 25 (OH) vitamin D level among diabetic subject affects glucose homeostasis. Vitamin D deficiency is strongly associated with insulin resistance and progression of Type 2 diabetes mellitus.

KEYWORD: Vitamin D, T2DM, Insulin Resistance

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INTRODUCTION

Diabetes mellitus is a group of metabolic disorder that caused by absolute or relative insulin deficiency leading to chronic hyperglycemia, which can affect nearly every organ system in the body (ADA, 2014). Diabetes mellitus has been classified into 4 general categories (ADA, 2014): type 1 diabetes (T1DM), type 2 diabetes

(T2DM), other specific types of diabetes mellitus and gestational diabetes mellitus (GDM). Patients with type 2 diabetes have a combination of insulin resistance and dysfunctional β cell (ADA, 2014; WHO, 1999).

Several studies have suggested that vitamin D deficiency may play a role in the pathogenesis of diabetes mellitus (Pittas et al., 2012; Lim et al., 2013; Kayaniyil et al., 2010). Vitamin D is a steroid responsible mainly for enhancing intestinal absorption of calcium and phosphate (Christakos et al., 2011), so it is characterized as a regulator of homeostasis of bone and mineral metabolism. In addition to that it has non – skeletal action (Rosen et al., 2012).

Vitamin D receptors have been found on pancreatic beta cells to express the enzyme 1-α-hydroxylase that responsible for vitamin D formation (Bland et al., 2004). Vitamin D promotes pancreatic β-cell function by stimulating the expression of insulin receptors and facilitates secretion of insulin from pancreatic beta cells (Alvarez & Ashraf, 2010). Also enhances insulin sensitivity by activating peroxisome proliferator-activated receptor-δ (PPAR- δ) (Dunlop et al., 2005) and has indirect effect in insulin secretion by maintain normal flux of serum calcium to intracellular compartment that is required for normal insulin secretion (Pittas et al., 2007; Seshadri et al., 2011); therefore vitamin D plays role in glucose homeostasis (Alvarez & Ashraf, 2010; Cade &Norman, 1986; Zeitz et al., 2003) and vitamin D deficiency may be lead to impaired insulin secretion (Talaei et al., 2013) and increase risk of insulin resistance that may progress to type 2 diabetes mellitus (Talaei et al., 2013). Also it has been found that vitamin D supplement may improve glucose tolerance and insulin resistance (von Hurst et al., 2010).

To the best of our knowledge, no study evaluates the relationship between vitamin D and diabetes in our localities, our aim was therefore to assess vitamin D status (25(OH) D) among patients with T2DM and to investigate the association between serum 25(OH) D levels with insulin resistance.

Subjects and Methods

This cross – sectional study was carried out in Basrah governorate, southern Iraq. The study sample was derived from the diabetic center in Al-Mawane General hospital from 20th November 2014 to 25th April 2015 ; this study was conducted on eighty patients(30 males and 50 females) with T2DM with disease duration of one year or above. The participants were selected by the same physician from those attended Al-Mawane General hospital for regular checkup. All patients were on antihyperglycemic drugs and none of them on insulin therapy. The patients age range from 32 years to 74 years with a mean value of 50.51 ± 10.7 years. Diabetic patients with renal dysfunction, gestational diabetes, hyperthyroidism, hypertension, and chronic liver disease were excluded from this study, as well as those taking vitamin D supplementation or any drugs affecting vitamin D. The Council and the Ethical Committee of the College of Medicine at the University of Basrah approved the protocol for this study.

After an overnight, fasting blood samples (5 ml) were collected and divided into two parts. One portion (2ml) was added to EDTA anticoagulant tube and send for Hb A1c estimation. The rest were transferred into a plain tube without anticoagulant and was left for 30 minutes at room temperature. The blood samples were centrifuged at (3000 rpm) for 5 minutes. Then the serum samples were collected, and stored in plastic tubes to be used for the estimation of Fasting serum glucose, vitamin D, and insulin levels.

Serum Vitamin D is measured by enzyme immunoassay competition method with a final fluorescent detection (ELFA) using kit from BioMerieux, France (Kit Ref No. 30463). Serum insulin was determined by a two-site immune enzymatic assay by kit from Bioscience, USA by using TOSOH instrument. Hemoglobin A1c is measured by ion exchange

high-performance liquid chromatography (HPLC) using kit supplied from Bio-Rad, USA (Kit Ref No. 220-0201). Fasting serum glucose is measured by enzymatic method using kit supplied from Randox Ltd. UK.

Homeostatic Model Assessment

HOMA – IR and HOMA – %β: is a method used to quantify insulin resistance (IR) and β - cell function (% β). Using the fasting serum levels of glucose (mg/dl) and insulin (µU/ml)

(McAuley et al., 2007; Singh & Saxena, 2010).

$$\text{HOMA} - \text{IR} = \frac{\text{Glucose x Insulin}}{405}$$

$$\text{HOMA-\%}\beta = \frac{360 \text{ x Insulin}}{\text{Glucose} - 63} \%$$

Normal value for HOMA-IR = ≤ 2.5 and HOMA-% β = 100% (Muniyappa et al., 2008).

HOMA-IS (QUICKI): is a quantitative insulin sensitivity check index methods to estimate insulin secretion and sensitivity in the human body that provides a reliable and accurate index of insulin sensitivity (Singh & Saxena, 2010; Katz et al., 2000).

$$\text{QUICKI} = \frac{1}{[\text{Log (Fasting Insulin, µU/ml)} + \text{Log (Fasting Glucose, mg/dl)}]}$$

A value of (QUICKI) HOMA-IS < 0. 339 indicate insulin resistance (Singh & Saxena, 2010; Katz et al., 2000).

Statistical Analysis

The data were analyzed in the computer by using SPSS "Statistical Package for Social Sciences programme" version 16. Chi squared test and logistic regression analysis were used to find out the significant differences and associations between different variables. A P value of < 0.05 was considered to be significant.

The results are expressed as [percentage, range, mean ± SD]. Independent t-test was used during comparisons between two different groups. While to find the significance of variables between three or more groups by using one-way ANOVA analysis. Chi-square (x^2 test) has been used in categorical scale to analyse difference proportion between two or more group.

Finally, logistic regression analysis were used to find out the significant differences and associations between different variables and to examine the influence of confounding variables and exclude variables that had little or no influence on the trait under analysis. P value <0.05 was considered the lowest limit for significance.

RESULTS

The eighty patients with type 2 diabetes involved in the study had been allocated into 3 groups based on the cutoff values for vitamin D levels (Al Humaidi et al., 2013) are illustrated in "Figure 1" as follow: vitamin D sufficient [serum 25(OH) D ≥ 30 ng/ml], vitamin D insufficient [serum 25(OH)D = 20 - 29.9 ng/ml], and vitamin D deficient [serum 25(OH)D < 20 ng/ml].

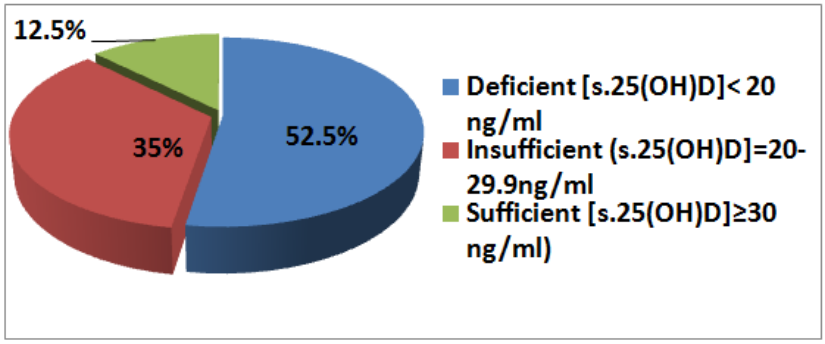

Figure 1: Distribution of Diabetic Patients According to Vitamin D Levels

The distribution of patients with low and normal levels of vitamin D with respects to fasting serum glucose, HbA**1c%,** serum insulin levels, HOMA-IR, HOMA-IS, and HOMA-%β are shown in table (1). The result of the present study shows inverse association between vitamin D levels with fasting serum glucose, HbA**1c** %**,** serum insulin levels, HOMA-IR. While positive association between vitamin D levels with HOMA-IS, and HOMA-%β and all the glycemic indices were statistically significant difference except HOMA-%β. To find out the independent association between the vitamin D status and glycemic indices, multiple logistic regression analysis was carried out as shown in Table (2). Accordingly factors that were found to be independently and significantly association with the vitamin D status were insulin levels, HbA1c, HOMA-IR, and HOMA-IS. While fasting serum glucose and HOMA-%β showed no significant association.

The relationship of 25(OH) vitamin D status in diabetic patients with respect to insulin sensitivity, insulin resistance, and β-cell function of pancreas are shown in "Figures 2, 3 and 4", respectively.

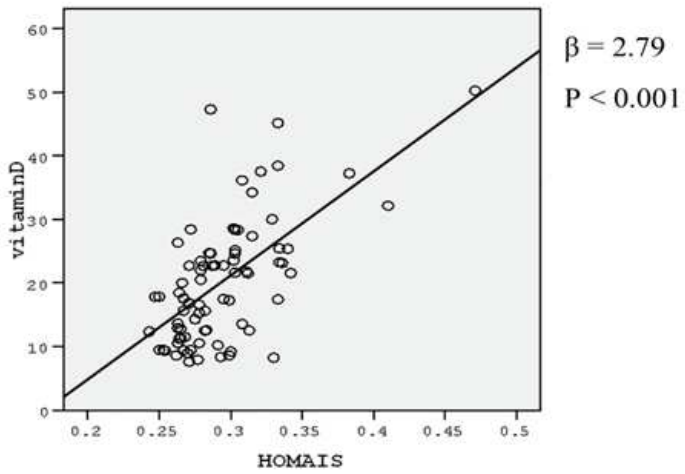

Figure 2: Relationship of 25hydroxy Vitamin D of Diabetic Patients with Respect to Insulin Sensitivity (HOMA-IS)

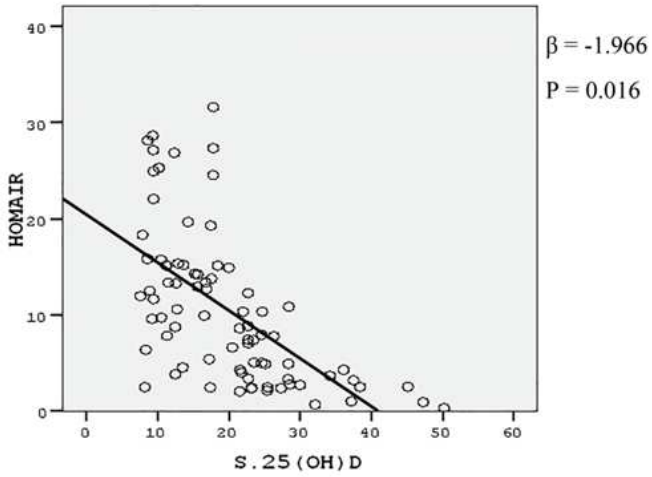

Figure 3: Relationship of 25hydroxy Vitamin D of Diabetic Patients with Respect to Insulin Resistance (HOMA-IR)

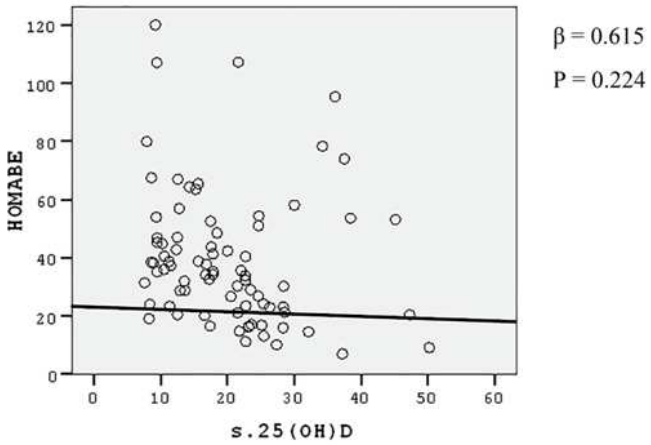

Figure 4: Relationship of 25hydroxy Vitamin D of Diabetic Patients with Respect to B-Cell Function of Pancreas (HOMA-β).

DISCUSSIONS

In recent years several studies observed that altered vitamin D homeostasis may play role in pathogenesis and development of Type 2 diabetes mellitus (Seshadri et al., 2011; Al Humaidi et al., 2013; Valdés-Ramos et al., 2015).

The results in this study revealed that vitamin D deficiency (≤ 19.9 ng/ml) existed in 52.5% of diabetic individuals. Similar results were found in Bahrain being 65% (Saweer 2015). Whereas high prevalence of vitamin D deficiency was reported among diabetic patients in the United Arab Emirates being 83.2% (Sadiya et al., 2014) and 90.4 % in Qatar (Badawi et al., 2012).

Several previous studies showed that low serum 25(OH) D has been associated with glucose intolerance and insulin resistance in type 2 diabetes (Modi et al., 2015; Al-Shoumer &Al-Essa, 2015). The results of the present study showed that serum 25(OH) D was inversely associated with serum insulin levels, insulin resistance (HOMA-IR) , fasting serum glucose and HbA1c. On the other hand a positive association was observed with insulin sensitivity (HOMA-IS) and β-cell function of pancreas (HOMA-%β). In addition to that a statistically significant independent association was observed

between serum 25 (OH) D and serum insulin, IR, IS and HbA1c (p=0.001). However, statistically significant but not independent association was revealed with fasting serum glucose and non-significant association with β - cell function (% β). This finding is consistent with previous studies, which also revealed such relationship (Al-Shoumer &Al-Essa, 2015; Gao et al., 2015). While Erdönmez et al., (2011) reported a controversial result and stated that there was no significant association between vitamin D levels and insulin resistance. Also Sollid et al., (2014) shows that vitamin D supplementation doesn't improve glycemic status. The effect of vitamin D levels on glucose homeostasis is represented by vitamin D receptor (VDR: transcription factor) and vitamin D dependent calcium binding protein. The pancreatic islet cells have both vitamin D receptor (VDR) and vitamin D dependent calcium binding protein, which suggest a role for vitamin D in insulin secretion (Bikle 2003). Vitamin D has direct influence on insulin secretion by stimulating the expression of insulin receptors and increasing insulin responsiveness for glucose transport (Lee et al., 1994). Vitamin D doesn't only improve insulin sensitivity of target cells, but also enhance and improve B-cell function (Kramer et al., 2014). Conversion of pro-insulin to insulin is accelerated by vitamin D (Bourlon et al., 1999). Therefore Vitamin D is essential for improving insulin secretion and sensitivity (Teegarden & Donkin 2009). Moreover, vitamin D links between VDR and peroxisome proliferated activated receptor signaling (PPARS) by identifying PPARS gene as a primary $1\alpha(OH)_2 D_3$ target gene. Peroxisome proliferation activated receptor signaling (PPARS) has been suggested to influence the insulin sensitivity (Henke 2004). In addition to that vitamin D has indirect effects on insulin action through its role in regulating extracellular calcium and maintaining adequate intracellular cytosolic calcium pool (Beaulieu et al., 1993). Therefore alteration in calcium homeostasis may adversely affects on pancreatic islets cell secretory function (Santulli et al., 2015) and may participates to peripheral insulin resistance by impairing phosphorylation and decrease number of glucose transporters (GLUT1 and GLUT4) in cell membrane to promote glucose uptake (Reusch et al 1991). Therefore low vitamin D status considers being a risk factor for the development insulin resistance and pathogenesis of DM type 2 (Xavier et al., 2008; Seshadri et al., 2011; Al Humaidi et al., 2013; Valdés-Ramos et al., 2015).

CONCLUSIONS

In conclusion: Serum 25-hydroxy vitamin D is positively associated with insulin sensitivity (IS), β-cell function of pancreas (β-cell). While serum 25(OH) D inversely associated with insulin resistance (IR). Insulin resistance due to vitamin D deficiency could be consider as risk factor for development of type 2 DM. Whether vitamin D supplementation would improves the health and status of patients with T2DM needs further investigations.

Conflict of Interest: None.

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APPENDIX

Table 1: Distribution of Serum Vitamin D Levels According to Glycemic Profile

Variables		Serum Vit D Deficient-Insufficient		Serum Vit D Normal		Total N No.	OR	95%CI	270 P-Value
		No.	%	No.	%				
FBS(mmol/l)	5.56 - 6.99	0	0	9	100	9	1*	_	0.05 <
	≥ 7.0	42	59.2	29	40.8	71	2.448	1.85-3.23	
HbA1c %	7.5 <	1 1	5.9	16	94.1	17	1*	_	≤ 0.001
	≥ 7.5	41	65.1	22	34.9	63	4.034	1.04-4.270	
S. Insulin (µU/ml)	10 <	3	12.5	21	87.5	24	1*	_	≤ 0.001
	≥ 10	39	69.6	17	30.4	56	14.062	2.02-21.24	
HOMA-IR	≤ 2.5	2	16.7	10	83.3	12	1*	_	0.007
	2.5>	40	58.8	28	41.2	68	3.132	4.03-12.648	
HOMA-IS	≤ 0.34	38	73.1	14	26.9	52	16.28	4.79-55.3	≤0.001
	0.34 >	4	14.3	24	85.7	28	1*	_	
HOMA-β	100 <	40	51.9	37	48.1	77	0.541	0.05-6.212	NS

Table 2: Logistic Regression Analysis for All Variables in Relation to Serum 25 (Oh) Vitamin D

Variables	Coefficient(B)	P-Value	Or	95%Ci
FBS	- 21.573	NS	0.095	0.001-0.294
s. Insulin	- 2.766	0.001<	16.561	4.217-61.153
HbA1c%	- - 0.395	0.001	10.180	3.704-240.026
HOMA-IR	- 1.966	0.016	5.851	1.452-35.137
HOMA-IS	2.790	0.001 <	19.21	0.018-0.209
HOMA-β	0.615	NS	0.244	0.161-21.262