RESEARCH ARTICLE

ASSESSMENT OF VITAMIN D DEFICIENCY IN TYPE 2 DIABETES MELLITUS PATIENTS

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ABSTRACT

Aim: To assess vitamin D deficiency in type 2 diabetes mellitus patients. **Materials & Methods:** Eighty- two type 2 diabetes mellitus patients of either gender and equal number of healthy controls were also enrolled. 25(OH)D level was estimated by chemiluminescence immunoassay (CLIA). Normal serum 25 (OH)D level was defined as 30 ng/ml or more. Vitamin D insufficiency state was defined at a level of 21 - 29 ng/ml. Vitamin D deficiency state was defined at a level of 20 ng/ml or less. Severe vitamin D deficiency was defined as < 10 ng/ml. **Results:** There were 35 severely deficient, 20 deficient, 12 insufficient and 15 normal patients in group 1 and 2 severely deficient, 4 deficient, 8 insufficient and 8 normal subjects in group 2. **Conclusion:** Type II DM patients had severe vitamin D deficiency as compared to healthy control subjects.

KEYWORDS

diabetes mellitus patients, vitamin D deficiency, Health, Insulin.

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder, the prevalence of which is increasing steadily all over the world (Anjana et al., 2011). It has been estimated that, by 2030, the global population of diabetes would have been 562 million (Shetty, 2012). Although the number of people with T2DM is increasing in every country, its major contribution is from developing countries, where it is fast becoming an epidemic (Kahn et al., 2013; Campbell et al., 1975). It is widely known that the pathophysiology of type 2 diabetes involves progressive impairment of insulin secretion associated with a coexisting insulin resistance. Along with the classic role of 1,25 (OH)2 D in calcium homeostasis and bone metabolism (Unnikrishnan et al., 2007).

Vitamin D deficiency (VDD) is presently a major health problem in both adults and children across the globe, and it is more so in the Indian subcontinent (Ford et al., 2005). Many studies that focused on pediatric age have demonstrated high prevalence of VDD in normal children and adolescents. In the past decade, interest in diabetes mellitus and vitamin D metabolism has grown (Baynes et al., 1997). 1 billion people worldwide are suffering from vitamin D deficiency or insufficiency. Even in the sunniest countries including India, vitamin D deficiency is very common as most of the body surface is shielded from the sun (Afzal et al., 2013).

Vitamin D, originally described merely as a vitamin, is indeed a misnomer as it is now well-established that its active form is a hormone which is not only involved in bone metabolism but also in a plethora of non-skeletal physiological processes (Song et al., 2013). Several mechanisms have been proposed, indicating a positive effect of vitamin D on insulin secretion and sensitivity, which include its direct effect via activation of vitamin D

receptor on pancreatic β -cells and insulin sensitive organs and indirect effect via regulation of calcium homeostasis (Palomer et al., 2008; Norman et al., 1980). We attempted present study to assess vitamin D deficiency in type 2 diabetes mellitus patients.

2. METHODOLOGY

Eighty- two type 2 diabetes mellitus patients of either gender was included (Group 1). Equal number of healthy controls were also enrolled (Group 2). The selection was based on the basis of American Diabetic Association (ADA) guideline as follows: $HbA1C \geq 6.5\%$ or $FPG \geq 126$ mg/dl (7.0 mmol/l) or 2-h plasma glucose ≥ 200 mg/dl (11.1 mmol/l) during on OGTT or in patients with classical symptoms of hyperglycaemia or hyperglycaemic crisis, a random plasma glucose ≥ 200 mg/dl (11.1 mmol/l). Inclusion was done after obtaining written consent. Ethical approval was sought from ethical and review committee.

All patients underwent oral glucose tolerance test (OGTT) using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water. Obesity was defined in terms of BMI and WC. Obesity was defined with $BMI > 25$ kg/m² both for males and females. 25(OH)D level was estimated by chemiluminescence immunoassay (CLIA). Normal serum 25 (OH)D level was defined as 30 ng/ml or more. Vitamin D insufficiency state was defined at a level of 21 - 29 ng/ml. Vitamin D deficiency state was defined at a level of 20 ng/ml or less. Severe vitamin D deficiency was defined as < 10 ng/ml. Results were subjected for statistical inferences using chi- square test. The level of significance was set below 0.05.

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3. RESULTS

Table 1: Baseline parameters

Parameters	Group 1	Group 2	P value
BMI (Kg/m ²)	26.2	24.7	<0.05
Waist circumference (cm)	86.3	82.5	<0.05
Hip circumference (cm)	92.1	87.5	<0.05
WHR	0.93	0.90	<0.05
25-hydroxy Vit D (ng/ml)	23.7	40.5	<0.05
HbA1C (%)	9.8	5.1	<0.05

The mean BMI was 26.2 kg/m² in group 1 and 24.7 kg/m² in group 2, waist circumference was 86.3 cm in group 1 and 82.5 in group 2, hip circumference was 92.1 in group 1 and 87.5 in group 2, WHR was 0.93 in group 1 and 0.90 in group 2. The mean 25-hydroxy Vit D level was 23.7 ng/ml in group 1 and 40.5 ng/ml in group 2 and HbA1C level was 9.8% in group 1 and 5.1% in group 2. A significant difference was observed ($P < 0.05$) (Table 1, Graph 1).

Graph 1:

Table 2: Assessment of 25(OH)D status among subjects

Groups	Severely deficient (<10)	Deficient (10-20)	Insufficient (20-30)	Normal (>30)
Group 1	35	20	12	15
Group 2	2	4	8	68

There were 35 severely deficient, 20 deficient, 12 insufficient and 15 normal patients in group 1 and 2 severely deficient, 4 deficient, 8 insufficient and 68 normal subjects in group 2 (Table 2, Graph 2).

Graph 2:

4. DISCUSSION

In this study, 82 type II DM patients and equal number of controls were taken. The level of vitamin D was assessed in both groups (Laway et al., 2014). Dietary vitamin D is a fat-soluble vitamin which is absorbed in the small intestine and incorporated into chylomicrons (Chiu et al., 2004). Dietary vitamin D travels to the liver, bound to vitamin D-binding protein and in continued association with chylomicrons and lipoproteins, where it and endogenously synthesized cholecalciferol are metabolized (Scragg et al., 2004). The association of oral vitamin D with chylomicrons and lipoproteins permits a more rapid hepatic delivery when compared to endogenously synthesized or parenterally administered hormones, which circulate exclusively on vitamin D-binding protein (Holick et al., 2005).

This difference results in a rapid but less sustained increase in plasma 25-hydroxyvitamin D (25OHD) levels obtained with oral as opposed to parenteral administration or endogenous synthesis (Lips et al., 2006). In various tissues cholecalciferol undergoes a hydroxylation reaction with the formation of 25-hydroxycholecalciferol [25(OH)D], which in turn enters the general circulation bound to a specific protein carrier (American Diabetes Association. 2014).

The mean BMI was 26.2 kg/m² in group 1 and 24.7 kg/m² in group 2, waist circumference was 86.3 cm in group 1 and 82.5 in group 2, hip circumference was 92.1 in group 1 and 87.5 in group 2, WHR was 0.93 in group 1 and 0.90 in group 2. (Sharan et al., 2018) explored the prevalence of vitamin D deficiency in new onset type 2 diabetes mellitus as well as the relationship of blood level of 25 hydroxy vitamin D [25(OH)D] with insulin resistance and insulin secretion. Based on the vitamin D status, cases and controls were divided into 4 groups: normal (> 30 ng/ml), insufficiency (20 - 30 ng/ml), deficiency (10 - 20 ng/ml) and severe deficiency (< 10 ng/ml). Insulin resistance and insulin secretion defect were calculated using HOMA-IR and HOMA-B, respectively. In this study, mean 25(OH)D level among cases and controls were 24.91 ± 14.58 ng/ml and 41 ± 28.33 ng/ml, respectively showing significantly lower level in cases ($p < 0.001$). 25(OH)D level was normal in 33 (27.5%), insufficient in 36 (30%), deficient in 36 (30%) and severely deficient in 15 (12.5%) cases. 25(OH)D level was normal in 55 (45.83%), insufficient in 35 (29.16%), deficient in 23 (19.16%) and severely deficient in 7 (5.83%) controls. On bivariate and linear regression analysis 25(OH)D had significant negative association with HOMA-IR and HOMA-B, more strongly with HOMA-B.

The mean 25-hydroxy Vit D level was 23.7 ng/ml in group 1 and 40.5 ng/ml in group 2 and HbA1C level was 9.8% in group 1 and 5.1% in group 2. In their study 72 newly detected youth-onset diabetes subjects (age < 25 years), and 41 age- and gender-matched healthy controls were studied. In addition to basic information and management regarding their diabetes, metabolic parameters and serum 25(OH)D were measured in both the groups (Daga et al., 2012). Vitamin D deficiency was seen in 91.1% of the subjects with diabetes, and 58.5% of the healthy controls. Mean ±SD 25(OH)D was significantly low, 7.88 ± 1.20 ng/mL in subjects with diabetes against 16.64 ± 7.83 ng/mL in controls. Sixty percent of cases had severe Vitamin D deficiency compared with 8.3% in controls. Levels of vitamin D did not correlate with clinical parameters, such as gender, body mass index; or with biochemical parameters, such as serum calcium, phosphorus, alkaline phosphatase, fasting plasma glucose, and HbA1C.

In their study a moderately strong inverse correlation was observed between serum 25(OH)D level and measures of insulin resistance and a positive correlation was observed between serum 25(OH)D and measures of insulin sensitivity (Dutta et al., 2013).

5. CONCLUSION

Type II DM patients had severe vitamin D deficiency as compared to healthy control subjects.

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