ABSTRACT

The present study highlights the importance of Vitamin D3 and its significance in Diabetes. We have recruited 34 type 2 diabetes patients cross-sectionally who were on regular treatment between 0-5 years from a Community Health Care Center in Chennai, after obtaining the informed consent in vernacular language. Blood investigations on Fasting, Post Prandial & Glycosylated Haemoglobin, Vit D3 levels were measured. Our main aim of this study was to elicit the relationship between Vit D3 and HBA1C. Among those participants 15(44.1%) were females and 19(55.9%) males. Data was analysed using SPSS 16.0, and the mean±SE (mean) of 8 patients who had “Good control” (i.e. HBA1C < 7%) was 16.5±0.82. Among them 3(37.5%) were females & 5(62.5%) were males. For 26 patients who were under ‘Bad Control’ (HBA1C ≥ 7%), Pearson's coefficient of correlation between HBA1C and Vit D3 was observed to be negative ‘r = - 0.369’ (P = 0.044), which is yet to be perused with larger sample size.

Keywords: Vit D3, HBA1C, type2 diabetes, Correlation

INTRODUCTION

Diabetes is a major threat to global public health that is rapidly getting worse and the biggest impact is on adults of working age. In developing countries at least one in ten deaths among adults aged 35 to 64 is attributable to diabetes, and in some, the figure is as high as one in five. Simple lifestyle adjustments such as a healthy diet and physical activity, often combined with medication, have been shown to be effective in promoting a full and healthy life with diabetes. Various factors play a role in the etiopathogenesis and the glycemic control among type 2 diabetes mellitus (t2DM) patients. Vitamin D is derived from 7 dehydro cholesterol or ergosterol by UV radiation. Cholecalciferol is hydroxylated at the 25\textsuperscript{th} position in the liver to form 25 hydroxy cholecalciferol. This is the major transport form of the vitamin. It then gets hydroxylated at the first position to form calcitriol which is the active form of vitamin D.

Interestingly, many studies reveal that Vitamin D3, (calcitriol) has a role in the synthesis and secretion of insulin. Previous studies reveal that Vitamin D3 receptors are found in ß cells of pancreatic islets promoting insulin secretion and decreasing insulin resistance by receptor mediated molecular mechanisms. Various definitions for vitamin D insufficiency have appeared in the literature; the best established one pertains to serum levels below 30 ng/mL. A recent meta-analysis has demonstrated that low vitamin D levels in middle-aged and elderly populations represent a risk factor for t2DM, cardiovascular disease and metabolic syndrome. Deficiency of Vitamin D is associated with impairment of insulin synthesis and secretion and increase in insulin resistance. This study is done to find out the correlation between vitamin D3 levels and blood glucose levels as well as glycemic control in already diagnosed t2DM patients.

In many cases, t2DM – accounting for over 90% of all cases of diabetes – can be prevented through lifestyle interventions alone. The aim of this study was to examine the correlation between HBA1C and vit D3 and to test for clinical and statistical significance. This study was ethically approved by the institutional research committee (IEC/54/2011-12).

MATERIALS & METHODOLOGY

Study Design: Cross-Sectional Study

Study Area: A Community Health Care Center in a metropolitan City, Chennai
Study Period: July 2011 to Feb 2012
Study Population: Type2 diabetes patients who were on regular treatment between 0-5 years who didn’t have any other comorbid illness and who had given informed consent.
Study Material: The structured form with informations on Age, Sex, FBS,PPBS,HBA1C & Vit D3 along with the duration of regular treatment collected on first hand.

RESULTS

The data was analysed using SPSS 16.0 and were presented as descriptive statistics like frequency, percentage, mean, standard error, and the inferential statistics - student-t-test for two independent samples, 95% confidence interval, ANOVA and correlation coefficient. The data were segregated according to the glycaemic control and tested for its significance at 5% level. Values within parantheses represent percentage.

Table 1 describes the distribution of patients according to the glycemic control of t2dM patients over a period of 3 months. 8(23.5) of them had a good control with a Mean±SE of 16.5±0.82, 7(20.6) had a fair control with 20.1±1.03 , majority 16(47.1) had unsatisfactory control with 20.0±0.91 and 3(8.8) were on poor control over HBA1C with 20.6±0.66 respectively. The 95% CI for the ‘good control’(HBA1C ≤ 7.0) group was observed to be narrow when compared with the ‘bad control’ (HBA1C > 7.0) group.

![Table 1](data:image)
when compared for 16 ‘Unsatisfactory control’ patients with 20.0±0.91 issued a t-value = 2.45 (P= 0.02) and for the 3 ‘Poor control’ patients with 20.6±0.66 issued a statistically Significant t-value = 2.89 (P= 0.01). This is presented in Fig 1. The ANOVA of Vit D3 for glycemic control groups was observed to be F=2.84(P=0.055) statistically significant.

DISCUSSION

Type 2 diabetes mellitus is recognized as a worldwide public health problem due to the high medical and socioeconomic costs that result from complications associated with the disease. In our study there existed a statistically insignificant correlation between HBA1C and Vit D3 for all 34 patients. But when we removed 8 patients under ‘good control’ we observed a negative correlation between HBA1C and Vit D3 with a magnitude of ‘r = - 0.369’ (P = 0.044) but gender didn’t show any significant association with all glycemic parameters along with Vitamin D3.

Since this is a time bound (8 months) study and the limited cost involved for Vit D3 investigation, we couldn’t arrive at large number of patients without comorbid illness, but we could find a minimal negative correlation which is of Clinical Significance. The Chi-Square = 0.766 (P=0.858) showed an insignificant association between sex and HBA1C.

The present study observed that type 2 diabetic individuals with vitamin D insufficiency showed poor glycemic control. According to various references, vitamin D has a potential influence on glucose homeostasis as suggested by the following factors.

Effect of vitamin D on insulin secretion: Pancreatic β cells express 1 alpha hydroxylase which is responsible for an active vitamin D3 synthesis and also by increasing the insulin response to the glucose stimulation, but not affecting the basal insulin secretion. In some populations, type 1 diabetes is associated with certain polymorphisms within the VDR gene which is present in the human insulin gene promoter and also in the skeletal muscle and in adipose tissues. The insulin secretion and sensitivity is influenced by Vitamin D mediated intracellular calcium secretion that enhances the binding of the calcium binding protein to the IRS – 1 (Insulin receptor substrate 1), stimulating tyrosine phosphorylation and PI3 kinase activation and thus promoting insulin secretion.

Vitamin D deficiency may also impair the insulin secretion through its associated increase in the PTH levels which may impair the calcium signal which is needed for glucose-induced insulin secretion. Of significance is the finding, that the vitamin D potentiation of glucose-induced insulin secretion is seen in normal individuals but not in patients with established type-2 DM. Whether the insulin secretion is influenced by the direct action of Vitamin D or through its receptor or through changes in calcium, or PTH, is a matter of ongoing studies. It is also possible that the insulin secretion may be influenced by a combination of different mechanisms.

Effect of vitamin D on the insulin sensitivity: Type-2 DM, a state of chronic systemic inflammation has been found to increase the insulin resistance. Type-2 DM was found to be associated with an increase in the levels of the tumour necrosis factor-a and b, the C reactive protein, the plasminogen activator inhibitor-1 (PAI-1), and interleukin-6 (IL-6). The increase in these inflammatory mediators may precede and even predict the development of type-2 DM. In support of this concept, is the finding that VDR has been found on almost all the cells of the immune system and that vitamin D can repress the type 1 cytokines, inhibit dendritic cell maturation, and upregulate the regulatory T cells. Vitamin D also
suppresses the antigen-presenting capacity of the macrophages, it modulates the development of the CD4 lymphocytes and it inhibits the production of IFNc (interferon c) and IL-2 (interleukin 2) among other cytokines. These cytokines are known to activate the macrophages and the cytotoxic T cells, which in turn can lead to the destruction of the pancreatic islets. By the modulation of the immune and the inflammatory processes, vitamin D may also decrease insulin resistance and increase the insulin secretion in type-2 DM, which are the two characteristic defects in this condition. From the above discussion, it is clear that vitamin D has a significant role to play in the molecular mechanisms of the synthesis, secretion and the peripheral sensitivity of insulin. Hence, hypovitamnosis D may be associated with insulin resistance and beta cell dysfunction. A variety of limitations of this study need however to be addressed. The small sample size did not allow a multivariate approach incorporating additional, potentially meaningful factors modifying the levels of serum 25(OH) D but it should be declared that from the evidence provided that improving vitamin D status will help establishing better glycemic control in people with DM type 2. Nevertheless, it seems that routine screening for vitamin D insufficiency may provide meaningful information and could be considered for diabetic care. Interventional studies are needed to evaluate whether vitamin D long-term supplementation could reduce morbidity in diabetic population with awareness of side effects.

REFERENCES


9. Hennekens CH, Burings JE, Epidemiology in Medicine, LittleBrown and Company, Boston/toronto


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