

Association of serum vitamin D₃ with newly diagnosed type 2 diabetes mellitus

Taznuva Anwar, Md. Matiur Rahman, Forhadul Hoque Mollah and Subrata Kumar Biswas

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Department of Biochemistry and Molecular Biology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh

For Correspondence:

Md. Matiur Rahman
dr.matiur57@yahoo.com

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Abstract

Vitamin D has an important role and supposed to be a risk factor in the development of pathogenesis of type 2 diabetes mellitus by affecting either insulin sensitivity or β -cell function, or both. The present study was conducted to evaluate the relation of serum vitamin D₃ and type 2 diabetes mellitus. Total 80 individuals were enrolled in this study. Forty newly diagnosed type 2 diabetic individual were enrolled as cases and 40 healthy glucose tolerant subjects were enrolled as controls. Serum vitamin D₃ was measured by chemiluminescence microparticle immunoassay. Fasting and 2 hours plasma glucose level were measured by hexokinase method using the Dimension clinical chemistry system. The mean serum vitamin D₃ level was significantly low in type 2 diabetes mellitus than in controls ($p=0.007$). The individual with vitamin D₃ deficiency showed 3.4 times higher chances to develop type 2 diabetes mellitus compare to the individual with normal vitamin D status. Correlation test showed significant ($r=-0.351$) negative correlation ($p=0.026$) between serum vitamin D₃ and type 2 diabetes mellitus. It can be concluded that vitamin D₃ deficiency is related to type 2 diabetes mellitus.

Introduction

Type 2 diabetes mellitus is a metabolic disorder that has become increasingly prevalent in the developing world.¹ The exponential increase is a great cause for concern as it is no longer a disease found solely in adults, but has become increasingly evident in children as well.² Vitamin D deficiency has been related to multiple types of health problems including different cancer, autoimmune and metabolic diseases like type 1 and 2 diabetes mellitus.³

In addition to genetics which predisposes individuals to develop type 2 diabetes mellitus, there are also many environmental factors which contribute greatly to its development like physical inactivity, poor nutrition and obesity. Recent evidence suggests that vitamin D deficiency (as measured by serum 25-hydroxy-vitamin D₃ concentration) may also contribute to the pathogenesis of type 2 diabetes mellitus.⁴

The pathogenesis of type 2 diabetes mellitus is quite complex. It involves many different pathways, organs, tissues and hormones.⁵

In keeping with the notion that type 2 diabetes mellitus cannot manifest without β -cell failure. It is important to examine the role of vitamin D metabolites (1,25(OH)₂D₃) in pancreatic β -cell function. The 1 α (OH)ase enzyme, although

originally thought to be present only in the renal tissue, has been identified also in the extra-renal tissues like pancreatic β -cells.⁶ The identification of the 1 α (OH)ase in the β -cells suggests that 1,25(OH)₂D₃ may play a role in the overall β -cell function. *In vitro* and *in vivo* studies have ascertained that 1, 25(OH)₂D₃ is necessary for insulin secretion and glucose homeostasis.⁷ These reports suggest that vitamin D deficiency affects the β -cell function and that vitamin D supplementation improves the β -cell function.⁸

Currently, the incidence of hypovitaminosis D is high. It has long been suspected as a risk factor for glucose intolerance.⁹ So, this study was conducted to know the association of serum vitamin D₃ deficiency and type 2 diabetes mellitus.

Materials and Methods

This case-control study was conducted from January to December 2013. It was a convenient and purposive sampling. Forty newly diagnosed type 2 diabetic individuals of both sexes, age between 30 to 60 years, attending in the sample collection booth of the department were enrolled and 40 healthy glucose tolerant subjects were selected from the university



Table I

Comparison of vitamin D₃ status between case and control

Parameters	Group		p value
	Case (Mean ± SD)	Control (Mean ± SD)	
Fasting blood glucose (mmol/L)	9.3 ± 3.4	5.0 ± 0.6	0.001
2 hours blood glucose (mmol/L)	14.8 ± 4.5	7.1 ± 1.9	0.001
Vitamin D (ng/mL)	17.7 ± 4.5	21.6 ± 7.7	0.007

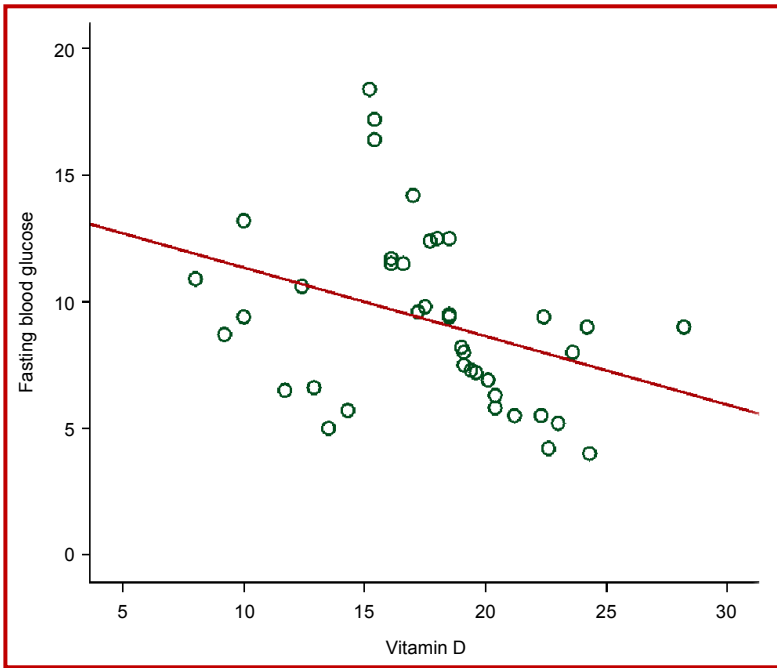


Figure 1: Correlation of vitamin D with fasting blood glucose in type 2 diabetes mellitus

employees, colleagues and patients' attendants of both sex, age between 30 to 60 years were enrolled as controls. The blood sample was collected from each study subject with all aseptic precaution. Fasting plasma glucose and 2 hours plasma glucose were measured by hexokinase method using the Dimension clinical chemistry system.¹⁰ Serum vitamin D₃ was estimated by chemiluminescence microparticle immunoassay (Ci 4100 ARCHITECT).¹¹ Hypovitaminosis D was defined as a serum 25(OH)D level below 20 ng/mL.

Statistical analysis

The statistical analysis was done using the software SPSS. The results were expressed as mean ± SD (Standard Deviation) and median (range). Comparison was done by unpaired t-test, X² test and Pearson's correlation test. The p value of <0.05 was considered statistically significant.

Results

The mean ± SD of serum vitamin D concentration in the newly diagnosed type 2 diabetes mellitus was 17.7 ± 4.5 ng/mL with the range of 8–28.2 ng/mL and in non-diabetic controls were 21.6 ± 7.7 ng/mL with the range of 13.6–49.2 ng/mL. Serum vitamin D concentration was found to be significantly low (p=0.007) in type 2 diabetic cases as compared to non-diabetic controls (Table I).

The prevalence of hypovitaminosis D in newly diagnosed type 2 diabetes mellitus was found to be more (27.5%) as compared to non-diabetic controls (10%). Hypovitaminosis D was significantly high (p=0.045) in type 2 diabetes mellitus than the normal glucose tolerant individuals.

Vitamin D showed significant negative (r=0.351) correlation (p=0.026) with fasting blood glucose in newly diagnosed type 2 diabetic cases (Figure 1).

Discussion

Vitamin D₃ deficiency is an increasingly recognized health concern related to skeletal and non-skeletal outcomes. Recent studies have found a relationship between vitamin D₃ status and diabetes and may be a risk factor in the pathogenesis of type 2 diabetes mellitus by affecting either insulin sensitivity or β-cell function, or both.¹²

In this study, the mean serum vitamin D level was significantly lower in type 2 diabetes than in controls. Tracy and Mazen (2010)¹³ found that Vitamin D₃ deficiency has been linked to type 2 diabetes mellitus, with circulating levels of 25(OH)D₃ significantly (p<0.008) lower in type 2 diabetics than in controls, although both groups had mean values well below the level considered ideal. This finding conforms to other similar studies done by Targher et al. (2006),¹⁴ Sung et al. (2012),¹² Chiu et al. (2004).⁸

Hypovitaminosis D was found to be higher in diabetic patients (27.5%) than in controls (10%). Our study finding is consistent with the observation of Targher et al. (2006).¹⁴

Vitamin D₃ deficiency has been suspected as a risk factor for impaired glucose tolerance and diabetes among adults. In the current study, we found that plasma 25(OH)D₃ concentrations were inversely associated with fasting glucose. The inverse association between plasma 25(OH)D₃ and fasting glucose is consistent with previous epidemiologic studies done by Targher et al. (2006),¹⁴ Sung et al. (2012).¹² Chiu et al. (2004).⁸

Further, consistent with data from non-Hispanic Whites in the NHANES III and Liu et al. (2006),¹⁵ plasma 25(OH)D₃ was not associated with 2 hours

blood glucose in our study. Chiu et al. (2004)⁸ found that circulating 25(OH)D₃ concentrations were inversely related to 2 hours plasma glucose in oral glucose tolerance test which is inconsistent with the current study. However, inconsistent results between the studies may be attributed to different populations studied and different time points of glucose measurement during oral glucose tolerance test.

Conclusion

Vitamin D₃ deficiency is inversely correlated with fasting blood glucose thus associated with type 2 diabetes mellitus. The individual with vitamin D₃ deficiency showed 3.4 times high-risk to develop type 2 diabetes mellitus compare to the individual with normal vitamin D status.

Ethical Issue

Approval received from Institutional Review Board of Bangabandhu Sheikh Mujib Medical University for the study (Reg. No. 746; date 30.6.13). Written consent of all the study subjects were taken free of duress and without exploiting any weakness of subjects.

Conflict of interest

There is no conflict of interest to declare from any of the authors.

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