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Research Paper

The role of HbA1c and Vitamin D in Type-2 Diabetes Mellitus

Dr Amit Roushan
Diabetologist, SidhiClinic,Muzaffarpur

ABSTRACT
Background: *Chronic hyperglycemia is a symptom of diabetes mellitus, which is caused by decreased insulin production, peripheral insulin resistance, or both. Vitamin D regulates a variety of hormonal activities in cells that have vitamin D receptors (VDRs). Vitamin D insufficiency has been linked to the development of impaired glucose tolerance, type 2 diabetes, and metabolic syndrome. The major goal of this study is to see if there is a link between vitamin D and HbA1c levels in type 2 diabetic patients.*
Methods and materials: *This cross-sectional study involved 63 Diabetes mellitus type II patients between the ages of 30 and 70. Their serum 25(OH) VITAMIN D3 levels were associated with their HbA1c levels. To examine the relationship between HbA1c levels and serum25 (OH) VITAMIN D3 levels, the Pearson correlation statistical test was used. The Pearson's correlation r-value was -0.3, indicating that the two variables had an inverse relationship. The correlation P-value was 0.013, indicating a strong negative connection.*
Conclusion: *Vitamin D levels in diabetic type II individuals were lower than ideal, according to the findings of the study. Their low vitamin D levels may be contributing to their hyperglycemia. In addition, their poor glycemic management may result in Vitamin D deficiency. Vitamin D therapy to improve glucose control in type 2 diabetes mellitus patients would be biologically correct based on these findings.*
KEYWORDS: *25(OH)VitaminD3,HbA1c(glycosylatedhaemoglobin),DiabetesmellitustypeII*

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I. INTRODUCTION

Diabetes mellitus type II currently contributes to a high burden of morbidity and mortality worldwide, even though it is generally avoidable and manageable with a healthy lifestyle and dietary changes. (1) Type II diabetes mellitus affected more than 500 million people worldwidein 2018, with the rate of occurrence rising at an alarming rate. India now has 32 million diabetics, with the number expected to rise to 80 million by 2030. (2) Diabetes mellitus is a metabolic condition marked by chronic hyperglycemia caused by inadequate insulin production, peripheral insulin resistance, or a combination of the two. (3)Patients with DM must maintain sufficient glycemic control, which is typically tested by testing glycosylated haemoglobin (HbA1c) and fasting blood glucose (FBG) levels, to avoid long-term micro-and macro-vascular problems. (4)

Despite its status as a micronutrient, vitamin D has been linked to several hormonal functions, which are thought to be the result of its interaction with vitamin D receptors (VDRs), which are found on a wide range of cell types. (3) Vitamin D deficiency can exacerbate a variety of ailments, as well as being associated with a diabetes propensity and perhaps playing a role in diabetes development. (5)

Even though we live in a temperate zone, vitamin D insufficiency is very common in India. Around 70% of persons in both rural and urban areas were found to have vitamin D deficient symptoms. (4) Indians have the highest rates of diabetes and Vitamin D deficiency.

Vitamin D levels are inversely connected to glycosylated haemoglobin levels in gestational diabetes mellitus, according to a study. (6) Insulin secretion was reduced in mice lacking functional VDRs in animal trials. (7) Vitamin D treatment was also found to increase insulin production in rat pancreatic islets.(8)

Vitamin D's influence on VDRs expressed on human skeletal muscle and adipose tissue cells may play a role in peripheral insulin sensitivity. (9) Because they are responsible for glucose absorption in response to insulin release, these cells play a role in determining peripheral insulin sensitivity. (9)The goal of this study was to see if there was a link between serum 25(OH) vitamin D3 concentration and glycosylated haemoglobin (HbA1c) levels in type 2 diabetes patients.

II. MATERIALANDMETHODS

This is a cross-sectional study that involved 63 Diabetes mellitus type II patients between the ages of 30 and 70. They were chosen based on the following inclusion criteria: Diabetes mellitus type II for less than 5 years, using only oral hypoglycemic medications as treatment and no vitamin D supplementation.

Diabetics above the age of 5, diabetic patients on insulin, vitamin D supplements, diabetic complications, hypertensive, smoker, alcoholic, autoimmune disorder, thyroid disorders, bone diseases, menopause, and any disease that alters glucose homeostasis in the body are all excluded.

Patients were chosen at random from several diabetes camps and were required to have a fasting blood glucose level of greater than 125 mg/dl to participate in the study.

Before the measurements, the patients were urged to refrain from heavy physical activity for 24 hours and from consuming alcohol or caffeinated beverages for 12 hours. Before blood collection, baseline and anthropometric information were recorded. To assess 25(OH) Vitamin D3 and HbA1c levels, 5ml blood was obtained via venipuncture.

25(OH) Vitamin D3 was measured by Fully Automated Chemi Luminescent Imunno Assay analyzed on Siemens ADVIA centura. Serum 25(OH) D3 level is a better indicator of vitamin D status than 1,25(OH)2D3 since the former has a slower rate of clearance than the latter. [(10)]Glycosylated haemoglobin (HbA1c) was measured using FULLY AUTOMATEDH.P.L.C. using Biorad variant Turbo.

STATISTICAL ANALYSIS

It was performed using the licensed SPSS version 24.The parameters obtained were entered using Microsoft Excel version 10.Pearson correlation statistical test was chosen to see the relation between Glycosylated haemoglobin(HbA1c) and serum 25(OH) vitamin D3 levels.

III. RESULTS

Table 1. Data obtained from Diabetes Mellitus patients

	N	Minimum	Maximum	Mean	Std. Deviation	Skewness		Kurtosis	
	Statistic	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	**Std. Error**
AGE(years)	63	34	59	47.11	6.328	-.010	.285	-.845	**.563**
25(OH)VitaminD3 (ng/ml)	63	6	19	12.27	3.214	.248	.302	-.505	**.595**
HbA1c(%)	**63**	**6.20**	**8.90**	**7.2349**	**.71531**	**.813**	**.302**	**-.040**	**.595**

The average age of the 63 subjects was 47 years, with a mean HbA1c of 7.2349 percent and a mean 25(OH) Vitamin D3 level of 12.27ng/ml among diabetes mellitus type II patients.

TABLE 2. PEARSON CORRELATION(R)BETWEEN 25 (OH)VITAMIN D & HBA1C LEVELS

		25(OH)VITAMIN D3ng/ml	**HBA1C%**
25(OH)VITAMIND3 NG/ML	PearsonCorrelation	1	**-.311***
	Sig.(2-tailed)		**.013**
	N	63	**63**
HBA1C%	PearsonCorrelation	-.311*	**1**
	Sig.(2-tailed)	.013	
	N	63	**63**

***CORRELATIONISSIGNIFICANTATTHE0.05LEVEL(2-TAILED).**

The Pearson's correlation r-value found when analysing the link between HbA1C and 25(OH) vitamin D3 levels among type 2 diabetic patients was r= -0.311, indicating a negative relationship between the two variables. A P value of 0.013 was achieved, with a p-value of less than or equal to 0.05 considered significant.

IV. DISCUSSION

In this study, it was discovered that diabetes mellitus type II patients have lower vitamin D levels, as well as a substantial inverse association between HbA1c and serum 25(OH) vitamin D3 levels, implying a link between glycemic management and vitamin D metabolism.

Vitamin D deficiency could play a role in the development of hyperglycemia in these people. Vitamin D's role in type 2 diabetes is assumed to be mediated not only via regulating plasma calcium levels, which affect insulin synthesis and secretion, but also by a direct effect on pancreatic Beta-cell activity. (11)

The presence of vitamin D receptors (VDR) and vitamin D binding proteins (DBP) in pancreatic tissue, as well as the link between certain allelic polymorphisms in the VDR and DBP genes and glucose tolerance and insulin secretion, has led to the theory that it plays a critical role in glucose metabolism. (12) Mice missing functional VDRs had reduced insulin secretion in animal trials. (7) Vitamin D treatment was also found to increase insulin production in rat pancreatic islets.(8)

V. CONCLUSION

Vitamin D insufficiency causes hyperglycemia, and hyperglycemia causes vitamin D deficiency, resulting in syndemic condition that is linked to each other. The rising incidence and prevalence of diabetes emphasise the need for new approaches to diabetes management and prevention. According to the findings of the study, vitamin D levels in diabetic type II patients should be maintained at optimal levels by combining vitamin D supplementation with their diabetes treatment regimen to achieve euglycemic blood levels. In diabetes mellitus type II patients, excellent glycemic management is also critical for maintaining optimal vitamin D levels.

REFERENCES

[1]. World Health Organization (2013) Global Action Plan for the Prevention and Control of Noncommunicable Diseases 2013-2020.Geneva: WHO

[2]. Kraiser AB, Zhang N, Van Der Pluijm W. Global Prevalence of Type 2 Diabetes over the Next Ten Years. Diabetes. 2018;67(1)

[3]. Wang Y, Zhu J and DeLuca HF: Where is the vitamin D receptor? Arch BiochemBiophys 523: 123-133, 2012

[4]. Qaseem A, Wilt TJ, Kansagara D, Horwitch C, Barry MJ, Forciea MA Clinical Guidelines Committee of the American College of Physicians, corp-author. Hemoglobin A1c targets for glycemic control with pharmacologic therapy for nonpregnant adults with type 2 diabetes mellitus: A guidance statement update from the American college of physicians. Ann Intern Med. 2018;168:569–576

[5]. Hutchinson MS, Figenschau Y, Njolstad I, Schirmer H, Jorde R. Serum 25- hydroxyvitamin D levels are inversely associated with glycatedhaemoglobin (HbA(1c)). The Tromso Study. Scand J Clin Lab Invest. 2011;71(5):399- 406.

[6]. Lau, S., Gunton, J., Athayde, N., Byth, K. and Cheung, N. (2011) Serum 25- hydroxyvitamin D and glycatedhaemoglobin levels in women with gestational diabetes mellitus. Med J Aust 194: 334–337

[7]. Zeitz U, Weber K, Soegiarto DW, Wolf E, Balling R, Erben RG. Impaired insulin secretory capacity in mice lacking a functional vitamin D receptor. FASEB J. 2003;17:509–511.

[8]. Bourlon PM, Billaudel B, Faure-Dussert A. Influence of vitamin D3 deficiency and 1,25

[9]. dihydroxyvitamin D3 on de novo insulin biosynthesis in the islets of the rat endocrine pancreas. J Endocrinol. 1999;160:87–95

[10]. Alvarez JA, Ashraf A. Role of vitamin D in insulin secretion and insulin sensitivity for glucose homeostasis. Int J Endocrinol. 2010:351-385

[11]. Zittermann A. Vitamin D in preventive medicine: are we ignoring the evidence? Br J Nutr 2003;89:552–572.

[12]. Palomer X, González-Clemente JM, Blanco-VacaF,Mauricio D. Role of vitamin D in the pathogenesis of type 2 diabetes mellitus. Diabetes Obes Metab.2008;10(3):185-97

[13]. Lee S, Clark SA, Gill RK, Christakos S. 1, 25-dihydroxyvitamin D3 and pancreatic β- cell function: vitamin D receptors, gene expression, and insulin secretion. Endocrinology 1994; 134: 1602-10

[14]. Dunlop TW, Vaisanen S, Frank C, et al. The human peroxisome proliferator-activated receptor delta gene is a primary target of 1alpha,25-dihydroxyvitamin D3 and its nuclear receptor. J Mol Biol. 2005; 349: 248–260

[15]. Ogunkolade BW, Boucher BJ, PrahlJMet al. Vitamin D receptor (VDR) mRNA and VDR protein levels in relation to vitamin D status, insulin secretory capacity, and VDR genotype in Bangladeshi Asians. Diabetes 2002;51:2294–2300

[16]. Hosseinpour F, Ibranovic I, Tang W, Wikvall K (2003) 25-Hydroxylation of vitamin D3 in primary cultures of pig hepatocytes: evidence for a role of both CYP2D25 and CYP27A1. BiochemBiophys Res Commun 303: 877–83.

[17]. Stefanov MV, Apukhovskaia LI (1996) Features of vitamin D3 metabolism in liver cells in experimental diabetes mellitus. UkrBiokhimZh 68: 66–72.

[18]. MetinAksu N, Aksoy DY, Akkaş M, Yilmaz H, Akman C, et al. (2013) 25-OH- Vitamin D and procalcitonin levels after correction of acute hyperglycemia. Med SciMonit 19: 264–8.