ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

ORIGINAL RESEARCH

Association of Vitamin D deficiency with newly diagnosed Type 2 diabetes in Eastern India

[1]Dr. Prabhakar Telawade, [2]Dr. Kaushik Kumar Bhol

[1]Medical Officer, Panvel Municipal Corporation, Navi Mumbai, Maharashtra, India
[2]Assistant Professor, Department of Medicine, Institute of Naval Medicine, INHS, Asvini, India

Corresponding Author
Dr. Kaushik Kumar Bhol
Assistant Professor, Department of Medicine, Institute of Naval Medicine, INHS, Asvini, India
Email: drkkbhol@gmail.com

Received Date: 16 July, 2024 Accepted Date: 19 August, 2024

Abstract

Background: Vitamin D deficiency has been reported as a rising pandemic. Vitamin D levels have been found to be associated with obesity and insulin resistance. The several variables linked to the correlation between vitamin D levels and diabetes mellitus in the Indian population, however, have not been the subject of significant investigation.

Aims: The objective of this study is to estimate the association of Vitamin D deficiency with newly diagnosed Type 2 diabetes in Eastern India.

Materials and Methods: A hospital based cross-sectional comparative study on newly diagnosed Type 2 Diabetes Mellitus patients in a tertiary care teaching hospital, Raigad District, Maharashtra India. The study commenced after obtaining proper Institutional Ethics Committee approval.Taking into account the study's viability and the patient demographic of the urban health center, all diagnosed T2DM patients who were willing to participate and were at least 18 years old were enrolled. Individuals having renal or parathyroid conditions before to the study were not accepted.

Results: In this study, mean 25(OH)D level among cases and controls were 25.28±15.21 ng/ml and 40.76±24.23 ng/ml, respectively showing significantly lower level in cases ($p < 0.001$). Taking a cut-off level of > 30 ng/ml as normal, the subjects with normal level of 25(OH)D among cases and controls were 53 (26.5%) and 86 (43.0%), respectively. We divided subjects with below normal level of 25(OH) vitamin D into 2 categories: insufficient (> 20 30 ng/ml), and deficient <20 ng/ml). Subjects having insufficient 25(OH)D level among cases and controls were 58 (29.0%) and 63 (31.5%), respectively. On bivariate analysis 25(OH)VitD was negatively and significantly associated with serum insulin and insulin resistance (HOMA-IR) with Pearson correlation coefficient of −0.876 and -0.641, respectively.

Conclusion: Vitamin D deficiency and insufficiency are more prevalent among new onset type 2 diabetes patients compared to healthy controls. Serum 25(OH)D is correlated with both serum insulin and insulin resistance defect, but more with insulin resistance.

Key words: Type 2 Diabetes, 25 hydroxy vitamin-D, Serum insulin, HOMA-IR.

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

Introduction

Type 2 diabetes mellitus (T2DM) has emerged as a global health crisis, characterized by hyperglycemia resulting from impaired insulin sensitivity and secretion.[1]

The increasing incidence of type 2 diabetes, especially in developing nations, has brought attention to the pressing necessity of identifying modifiable risk factors and putting preventative measures into place. The possible function of vitamin D, a fat-soluble prohormone predominantly produced in the skin following exposure to ultraviolet B radiation, in glucose metabolism and diabetes prevention has drawn growing interest.1

There is strong evidence linking vitamin D insufficiency to type 2 diabetes, according to emerging research. The biological effects of vitamin D are mediated by the vitamin D receptor (VDR), which is extensively expressed in a number of organs, such as the liver, skeletal muscle, adipose tissue, and pancreatic beta cells. Studies have demonstrated that vitamin D plays a crucial role in glucose homeostasis by enhancing insulin sensitivity, promoting beta cell function, and reducing inflammation.[2,3]Vitamin D deficiency has also been connected to insulin resistance, a major pathophysiological feature of type 2 diabetes.[4]

Due to a number of variables, including restricted sun exposure from cultural norms, clothing, and skin pigmentation, as well as nutritional deficits, the incidence of vitamin D insufficiency is particularly high in India.[5] Additionally, increasing burden of obesity and sedentary lifestyle, common risk factors for T2DM, can further exacerbate vitamin D insufficiency. Given the high prevalence of both vitamin-D deficiency & T2DM in India, it is imperative to investigate the relationship between these two conditions.

Previous studies have reported varying results regarding the association between vitamin D status and T2DM, with some studies demonstrating a significant association and others showing inconsistent findings.[6,7] More recently, a meta-analysis by Barbarawi et al., In individuals with prediabetes, vitamin D supplementation at moderate to high dosages (≥1000 IU/day) substantially decreased the incidence of occurrence type 2 diabetes, compared with the placebo, according to nine randomized controlled trials (RCTs) including 43,559 participants.[8] These discrepancies may be attributed to differences in study populations, geographical locations, and methodological variations. Therefore, further research is warranted to elucidate the complex interplay between vitamin-D & T2DM in diverse populations.

This study aims to investigate prevalence of vitamin-D deficiency among newly diagnosed T2DM patients in tertiary-care hospital in Eastern India. By examining the association between vitamin-D status & clinical characteristics of T2DM, this study seeks to contribute to the growing body of evidence on the role of vitamin-D in pathogenesis and management of T2DM. The findings of this study may have important implications for public health interventions aimed at preventing and managing T2DM in Indian population.

Material & methods

We conducted this hospital based cross-sectional comparative study on newly diagnosed T2DM patients over period of eighteen months from 01Jan 2022 to 01 Jan 2024 in tertiary care teaching hospital,Raigad District, Maharashtra,India. The study commenced after obtaining proper Institutional Ethics Committee approval.Taking into account the study's viability and the patient demographic of the urban health center, all diagnosed T2DM patients who were willing to participate and were at least 18 years old were enrolled. Patients with pre-existing renal or parathyroid disorders were excluded.

Two hundred individuals who were just diagnosed with type 2 diabetes and were seeking medical attention for the first time were included as cases, while two hundred healthy non-diabetic participants were included as controls. Each person ranged in age from 30 to 80. According to guidelines from the American Diabetic Association (ADA), patients with

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

diabetes were chosen in the following manner:[9]A random plasma glucose of at least 200 mg/dl (11.1 mmol/l) in patients with typical symptoms of hyperglycemia or hyperglycaemic crises, or a HbA1C of at least 6.5%, FPG of at least 126 mg/dl (7.0 mmol/l), or 2-h plasma glucose of at least 200 mg/dl (11.1 mmol/l) while on OGTT.

The definition of fasting was consuming no calories for a minimum of 8 hours. The WHO-recommended oral glucose tolerance test (OGTT) was conducted with a glucose load that was equivalent to 75 g of dissolved anhydrous glucose in water. Each patient had a comprehensive evaluation that included a clinical, biochemical, and history assessment. According to protocol, an anthropometric measurement was made using a standard measuring tape and weighing device. Waist circumference (WC) measurements and body mass index (BMI) calculations were performed using accepted methods and tools. To the closest 0.1 cm, WC was measured at the halfway point between the iliac crest and the lowest rib. A BMI and WC definition of obesity was provided. For both genders, the BMI cutoff point to be considered obese was greater than 25 kg/m2. The definition of obesity used by WC was based on the Adult Treatment Panel III (ATP III) 2002 for the Asian population and the International Diabetes Federation (IDF), 2005. In the context of Asia, the cut-off value of WC to characterize obesity was > 90 cm for men and > 80 cm for women.

Chemiluminescence immunoassay was used to measure the amount of 25(OH)D (CLIA). A serum 25(OH)D level of 30 ng/ml or more was considered normal8. The threshold for vitamin D deficiency was set at 21–29 ng/ml.[10]A condition of vitamin D insufficiency was established at 20 ng/ml or below.[11,12]The threshold for severe vitamin D insufficiency was < 10 ng/ml.3Individuals in the control and new onset diabetes groups were split into four groups according to their vitamin D status: deficiency (<20 ng/ml), insufficiency (20 - 30 ng/ml), and normal (> 30 ng/ml).

Using the following formula, the homeostatic model assessment (HOMA) was utilized to measure β-cell function (HOMA-%B) and insulin resistance (HOMA-IR) respectively:[13]

HOMA-IR =fasting plasma glucose (mmol/l) × fasting plasma insulin (µU/ml) /22.5

HOMA-%B = 20 × fasting plasma insulin/ fasting plasma glucose- 3.5

Statistical analysis

The statistical tools in Microsoft Excel 2007 and SPSS software (Version 16) were used for the statistical analysis. When presenting continuous data, means and standard deviation (SD) with 95% confidence intervals (CIs) were used. When analyzing group differences, Student's t-test was used for unpaired samples, while the chi-square and Fisher exact test were used for dichotomous variables. Using the Pearson correlation coefficient and r2 value, bivariate analysis and linear regression analysis were used to establish correlation. A P value of less than 0.05 was deemed noteworthy.

Result

Out of the 400 patients who were enrolled, 200 individuals with newly diagnosed type 2 diabetes were classified as cases and 200 healthy participants without diabetes were classified as controls. With a mean age of 51.96±11.99 years for patients and 50.64±12.04 years for controls, both study groups were age matched (p = 0.273). There were 117 (58.5%) males and 83 (41.5%) females in cases whereas 105 (52.5%) males and 95 (47.5%) females in controls. The mean BMI of the patients was substantially higher (p < 0.001) than that of the controls, which were 27.07±5.23 kg/m2 and 25.12±4.61 kg/m2, respectively. The mean waist circumference (WC) was found to be substantially greater in cases (p < 0.001), measuring 85.68±6.75 cm in cases and 82.12±6.32 cm in controls.

Mean 25(OH)D levels in patients and controls were 25.28±15.21 ng/ml and 40.76±24.23 ng/ml, respectively, in our investigation, with cases having a substantially lower level (p <

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

0.001). Subjects having normal levels of 25(OH)D among cases and controls were 53 (26.5%) and 86 (43.1%), respectively, assuming a cut-off level of > 30 ng/ml as normal. Subjects with 25(OH) vitamin D levels below normal fell into two groups: those with inadequate levels (> 20–30 ng/ml) and those with deficiencies (<20 ng/ml). Between the cases and controls, there were 58 (29.0%) and 63 (31.5%) subjects with inadequate 25(OH)D levels, respectively. There were 89 (44.5%) and 51 (25.5%) subjects with inadequate 25(OH)D in the case and control groups, respectively.

With a Pearson correlation coefficient of -0.641 and -0.876, respectively, 25(OH)VitD was shown to be adversely and substantially correlated with insulin resistance (HOMA-IR) and serum insulin on bivariate analysis. Our research revealed a substantial and significant correlation between serum insulin and insulin resistance and vitamin D insufficiency.

Table 1: Sex distribution among study groups

	Type 2 Diabetes Subjects (n=200)	Normal Subjects (n=200)	P value
Male	117 (58.5%)	105 (52.5%)	0.482
Female	83 (41.5%)	95 (47.5%)	

***Chi Square test**

Table 2: Demographic and baseline characteristics

	Type 2 Diabetes Subjects (mean ± SD)	Normal Subjects (mean ± SD)	P value
Age (Years)	51.96±11.99	50.64±12.04	0.273
BMI (kg/m2)	27.07±5.23	25.12±4.61	**<0.001**
WC (cm)	85.68±6.75	82.12±6.32	**<0.001**
Hip circumference (cm)	93.14±7.60	86.98±8.31	**<0.001**
WHR	0.93±0.16	0.94±0.14	0.506
25-hydroxy Vit-D (ng/ml)	25.28±15.21	40.76±24.23	**<0.001**
Fasting Blood Glucose (mg/dl)	187.89±57.70	91.98±9.94	**<0.001**
Post-Prandial Blood Glucose mg/dl)	273.16±81.40	129.71±5.96	**<0.001**
HbA1c	9.08±2.12	4.91±0.21	**<0.001**
Serum Insulin(mIU/ml)	23.64±8.76	11.84±7.347	**<0.001**
Insulin Resistance (HOMA IR)	4.42±1.36	2.1±0.32	**<0.001**
Cholesterol (mg/dl)	196.47±57.16	158.70±48.03	**<0.001**
TG (mg/dl)	199.07±105.72	122.47±41.89	**<0.001**
LDL (mg/dl)	111.62±39.88	99.05±26.65	**<0.001**
HDL (mg/dl)	38.83±14.98	51.62±13.20	**<0.001**

***Independent Sample t test**

Table 3: 25(OH) D status among study groups

	Type 2 Diabetes Subjects (n=200)	Normal Subjects (n=200)	P value
Deficient (<20 ng/ml)	89 (44.5%)	51 (25.5%)	**<0.001**
Insufficiency (>20-30 ng/ml)	58 (29.0%)	63 (31.5%)	
Normal (>30 ng/ml)	53 (26.5%)	86 (43.0%)	

***Chi Square test**

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

Figure1: Correlation of 25(OH)D in ng/ml with serum insulin

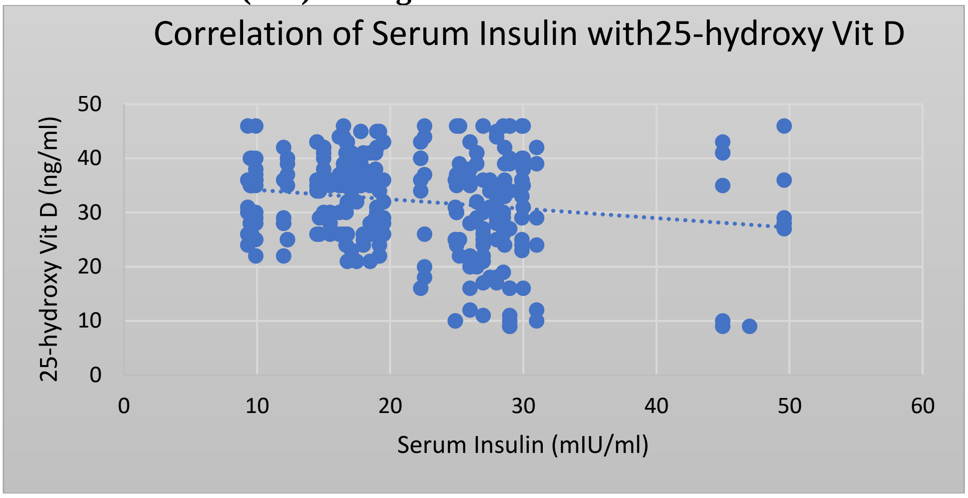
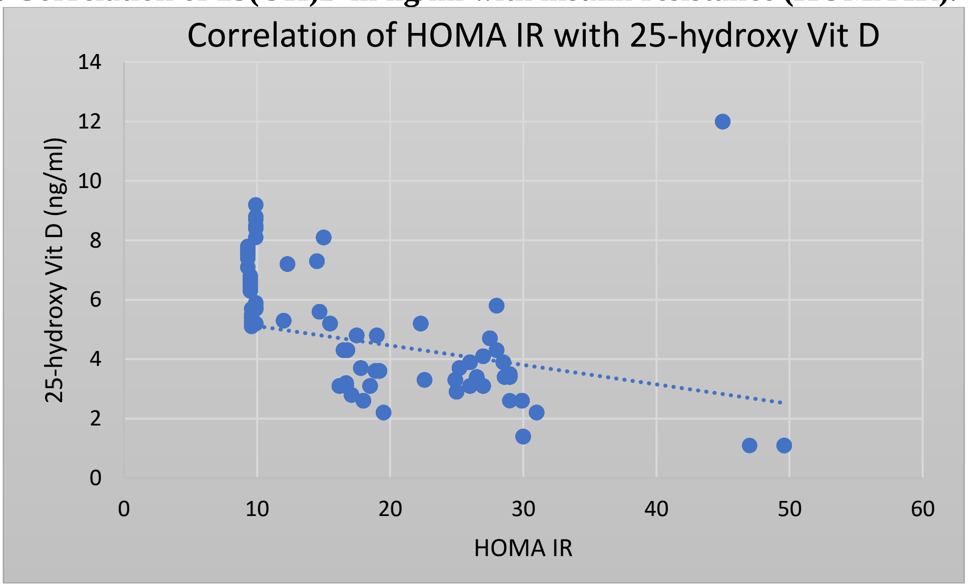


Figure 2: Correlation of 25(OH)D in ng/ml with insulin resistance (HOMA IR).



Discussion

Hyperglycemia is a prevalent endocrine condition known as Type 2 Diabetes Mellitus (T2DM). There are an estimated 69.2 million diabetics in India, according to the International Diabetes Federation (IDF 2015), out of an estimated 415 million diabetics worldwide. Its development appears to be influenced by a number of variables, including genetics, lifestyle, environment, and diet. Vitamin D is perhaps one of the most significant dietary components, either for glycemic management or for reducing the consequences of diabetes. In situations of vitamin D insufficiency, beta cell malfunction and insulin resistance are likely to be the processes that point to the significance of vitamin D in glucose homeostasis.

Vitamin-D deficiency now regarded as pandemic in all age groups in humans. The cross-sectional study involving largest cohort of non-diabetic Americans (n = 6288) reported inverse relationship between serum 25(OH) D concentration and fasting or post glucose load values.[14] In nine of 13 case-control studies reviewed by the authors, patients with T2DM showed a lower mean 25(OH)D concentrations than the non-diabetic controls. Association between vitamin-D intake and incidence type 2 DM was examined in Women's Health Study.[14,15]Subjects with daily vitamin D intake >511 IU had lower risk of incidence of DM when compared to a cohort with daily vitamin D intake of <159 IU per day (2.7% vs. 5.6%).

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

Pittas et alalso examined association between combined vitamin D and calcium intake and incidence of type2 DM among 83,806 women in Nurses' Health Study. After adjusting for BMI, age, & non-dietary covariates,significant inverse association was observed between vitamin-D intake & calcium intake on one hand and risk type2 DM on the other.[16]Few interventional studies have evaluated the impact of vitamin D administration on long-term glycaemic management. Pittas et al studied 92 diabetic subjects and reported decrease in fasting plasma glucose after 3 yr in group receiving daily supplementation of 700 IU of vitamin-D and 500 mg of calcium citrate.[17]

Davidet alhas shown that 63.5% type2 diabetes individuals compared to 36% of type 1 diabetes were deficient in vitamin D.[18]Goswamiet alof AIIMS in 2000 measured vitamin-D levels in apparently healthy subjects in Delhi and showed that significant vitamin-D deficiency was present in 90% of them.[19] Rajesh et al in their pilot study showed that 96.7% of Asian Indian patients with fragility hip fracture were deficient in vitamin D.[20]Harinarayananet alreported vitamin-D deficiency 62% and 72% in urban males and females, 44% and 77% rural males and females in south India.[21]Marwaha et alreported prevalence of vitamin D deficiency in 91.2% of healthy Indians aged above 50 years.[22]

Mean 25(OH)D levels in patients and controls were 25.28±15.21 ng/ml and 40.76±24.23 ng/ml, respectively, in our investigation, with cases having a substantially lower level ($p < 0.001$). Insufficient or lacking vitamin D was found in 44.5% and insufficient in 29.0% of Indians with type 2 diabetes, according to our study. Of the normal West Indian subjects included as controls, 25.5% had vitamin D insufficiency and 31.5% had vitamin D deficiency. In a related research, Sharan A. et al. used a cut-off level of > 30 ng/ml as normal; among the cases and controls, the participants with normal levels of 25(OH)D were 33 (27.5%) and 55 (45.83%), respectively. Subjects with 25(OH) vitamin D levels below normal were split into three groups: severely deficient (< 10 ng/ml), deficient (> 10 - 20 ng/ml), and inadequate (> 20-30 ng/ml). Insufficient levels of 25(OH)D were found in 36 (30%) of cases and 35 (29.16%) of controls, respectively. Between the cases and controls, there were 36 (30%) and 23 (19.16%) subjects with inadequate 25(OH)D, respectively. Comparably, amongst cases and controls, there were 15 (12.5%) and 7 (5.83%) persons with significantly low 25(OH)D levels, respectively.[23]An analogous investigation was conducted in North India with 102 individuals with newly diagnosed type 2 diabetes. The results indicated that patients with T2DM had a lower 25(OH)D level (18.81 ± 15.18) in comparison to healthy controls (28.46 ± 18.89) ($p = 0.001$).[24]In order to ascertain whether serum 25(OH)D was altered in newly diagnosed T2DM and IGT patients, a cross-sectional survey was conducted in a New Zealand workforce consisting of 5,677 Polynesian and Caucasian individuals aged 40-64 years. The results showed that serum 25(OH)D was significantly lower in T2DM and IGT patients compared to controls ($p = 0.0016$).[25]While exposure to sunlight is beneficial, it is restricted to a few months in India, and vitamin D fortification of food is not customary in the nation. Future research must clarify if vitamin D deficiency in diabetic individuals contributes to the pathophysiology of diabetes mellitus.According to Lo et al., because of their darker complexion, Indians need to be in the sun nearly twice as often as Caucasians do in order to get the recommended amount of vitamin D.[26]Working inside or in a small space with little exposure to the sun are examples of lifestyle variables that may contribute to the high incidence of vitamin D insufficiency in our community. In India, the typical workday runs from 10 a.m. to 6 p.m., but the best time to go outside and absorb solar radiation is from 11 a.m. to 2 p.m. A UV index of 7-9 is necessary for the conversion of 7-dehydrocholesterol to pre-vitamin D3.[27]This, however, is implausible given that India is a tropical nation and its summers are extremely hot, requiring the majority of its citizens to spend their time indoors. Because of this, our population has very poor vitamin D status, which is a result of minimal sun exposure.

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

The effects of vitamin D insufficiency on glucose homeostasis and its role in insulin production and sensitivity are most prominent in people who have never taken medication. Therefore, people with pre-diabetes or diabetes with a recent beginning are the best candidates to research the impact of vitamin D insufficiency on insulin secretion defect and insulin resistance. In Kolkata, 157 pre-diabetes patients, 42 diabetic patients, and 28 normal people participated in research.[28]According to our research, 25(OH)VitD had a substantial negative connection (Pearson correlation coefficient of -0.641 and −0.876) with insulin resistance (HOMA-IR) and serum insulin on bivariate analysis. Serum 25(OH)D level and measures of insulin resistance showed a rather significant inverse link, whereas serum 25(OH)D and measures of insulin sensitivity showed a positive correlation, according to a previous study conducted in Kolkata.[28]Rather than an issue with insulin production, the research population's increased frequency of obesity and insulin resistance may account for the relatively weak connection between HOMA-B and vitamin D3 insufficiency.[23]

Limitation of study

Its cross-sectional nature is its limiting factor. The postulated mechanisms for the effects of vitamin D are the presence of vitamin-D receptors on pancreatic cells, activation of 1 α hydroxylase by vitamin D, presence of vitamin-D response element in insulin gene, presence of vitamin-D receptors on skeletal muscle cells, and increase in transcription of insulin receptor genes by vitamin D.

Conclusion

In eastern India, the prevalence of vitamin D deficiency and insufficiency is higher in newly diagnosed type 2 diabetes patients than in healthy controls. Serum 25(OH)D insufficiency is a crucial factor in the development and worsening of glycaemic state in diabetes individuals since it is connected with both serum insulin and insulin resistance defect, but more so with insulin resistance. To determine if vitamin D supplementation and addressing vitamin D insufficiency at the outset of diabetes can prevent early glycaemic deterioration in newly diagnosed type 2 diabetes, prospective trials are required.

References

1. Goyal R, Singhal M, Jialal I. Type 2 Diabetes. [Updated 2023 Jun 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK513253/
2. Pittas AG, Dawson-Hughes B. Vitamin D and diabetes. J Steroid Biochem Mol Biol. 2010;121(1-2):425-9. doi: 10.1016/j.jsbmb.2010.03.042.
3. Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266-281.
4. Scragg R, Sowers M, Bell C. Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the Third National Health and Nutrition Examination Survey. Diabetes Care 2004; 27:2813–2818.
5. Aparna P, Muthathal S, Nongkynrih B, Gupta SK. Vitamin D deficiency in India. J Family Med Prim Care. 2018;7(2):324-330. doi: 10.4103/jfmpc.jfmpc_78_18.
6. Song Y, Wang L, Pittas AG, Del Gobbo LC, Zhang C, Manson JE, Hu FB. Blood 25-hydroxy vitamin D levels and incident type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care. 2013;36(5):1422-8. doi: 10.2337/dc12-0962.
7. Pittas AG, Chung M, Trikalinos T, Mitri J, Brendel M, Patel K, Lichtenstein AH, Lau J, Balk EM. Systematic review: vitamin D and cardiometabolic outcomes. Annals of internal medicine. 2010;152(5):307-14.
8. Barbarawi M, Zayed Y, Barbarawi O, Bala A, Alabdouh A, Gakhal I, Rizk F, Alkasasbeh M, Bachuwa G, Manson JE. Effect of Vitamin D Supplementation on the Incidence of

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

Diabetes Mellitus. J Clin Endocrinol Metab. 2020;105(8): dgaa335. doi: 10.1210/clinem/dgaa335.
9. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diab Care 2014; 37 (Suppl. 1): S81-S90.
10. Dawson-Hughes B, Heaney RP, Holick MF et al. Estimates of optimal vitamin D status. Osteoporos Int 2005; 16: 713-6.
11. Holick MF. High prevalence of vitamin D inadequacy and implications for health. Mayo Clin Proc 2006; 81: 353-73.
12. Bischoff-Ferrari HA, Giovannucci E, Willett WC et al. Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. Am J Clin Nutr 2006; 84: 18-28. (Erratum, Am J Clin Nutr 2006; 84: 1253).
13. Wallace TM, Levy JC, Matthews DR. Use and Abuse of HOMA Modeling. Diab Care 2004; 27: 1487-95.
14. Pittas AG, Lau J, Hu FB, Dawson-Hughes B. Theroleofvitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. J Clin Endocrinol Metab.2007;92(6):2017-29.
15. Liu S, Song Y, Ford ES, Manson JE, Buring JE, Ridker PM. Dietary calcium, vitamin D, and the prevalence of metabolicsyndrome in middle-aged and older U.S. women. Diabetes Care.2005;28(12):2926-32.
16. Pittas AG, Dawson-Hughes B, Li T, Van Dam RM, Willett WC, Manson JE, Hu FB. Vitamin D and calcium intake in relation to type 2 diabetes in women. Diabetes Care. 2006;29(3):650-6.
17. Pittas AG, Harris SS, Stark PC, Dawson-Hughes B. The effects of calcium and vitamin Dsupplementation on blood glucose and markers of inflammation in nondiabeticadults. Diabetes Care.2007;30(4):980-6.
18. Di Cesar DJ, Ploutz-Snyder R, Weinstock RS, Moses AM. Vitamin D deficiency is more common in type 2 than in type 1 diabetes. Diabetes Care. 2006;29(1):174.
19. Goswami R, Gupta N, Goswami D, Marwaha RK, Tandon N, Kochupillai N. Prevalence and significanceoflow25-hydroxyvitamin Dconcentrations in healthy subjects in Delhi.Am J Clin Nutr.2000;72(2):472-5.
20. Khadgawat R, Brar KS, Gahlo M, Yadav CS, Malhotra R, Guptat N, Tandon N. Highprevalence of vitamin D deficiency in Asian-Indianpatients with fragilityhip fracture: a pilot study. J Assoc Physicians India.2010;58:539-42.
21. Harinarayan CV, Ramalakshmi T, Prasad UV, Sudhakar D, Srinivasarao PV, Sarma KV, Kumar EG. High prevalence of low dietary calcium, high phytate consumption, and vitamin D deficiency in healthy south Indians. The American journal of clinical nutrition. 2007;85(4):1062-7.
22. Marwaha RK, Tandon N, Garg MK, Kanwar R, Narang A, Sastry A, Saberwal A, Bandra K. Vitamin D status in healthy Indians aged 50 years and above. J Assoc Physicians India. 2011; 59:706-9.
23. Sharan A, Mukhopadhyay S Majumdar J, Ghosh B, Sengupta S. A Study of Prevalence of Vitamin D Deficiency in New Onset Type 2 Diabetes Mellitus in a Tertiary Care Hospital of Eastern India. JIACM 2018; 19(2): 106-11
24. Laway BA, Kotwal SK, Shah ZA. Pattern of 25 hydroxy vitamin D status in North Indian people with newly detected type 2 diabetes: A prospective case control study. Ind J Endocrinol Metab 2014; 18 (5): 726-30.
25. Scragg R, Holdaway I, Singh V et al. Serum 25-hydroxyvitamin D3 levels decreased in IGT and diabetes. Diab Res Clin Prac 1995; 27: 181-8.
26. Lips P, van Schoor NM, de Jongh RT. Diet, sun, and lifestyle as determinants of vitamin D status. Ann N Y Acad Sci. 2014; 1317:92-8.

ISSN: 0975-3583,0976-2833 VOL15, ISSUE 09, 2024

27. Harinarayan CV, Joshi SR. Vitamin D status in India--its implications and remedial measures. J Assoc Physicians India. 2009; 57:40-8.
28. Dutta D, Maisnam I, Shrivastava A et al. Serum vitamin-D predicts insulin resistance in individuals with prediabetes. Ind J Med Res 2013; 138 (6): 853-60.