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25-Hydroxycholecalciferol levels and its equation with lipid parameters and Insulin sensitivity in obese and non-obese young adults: A pilot study

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ABSTRACT

Introduction: Vitamin D deficiency and Obesity are the two prevailing health issues of the globe, with India being no exception. The sub optimal Vitamin D levels is associated with an elevated risk of a number of chronic disorders including malignancy, inflammation and autoimmune diseases. Obesity breeds metabolic disharmony. Recent data reports Vitamin D deficiency being more prevalent among the obese.

Objective: This study is undertaken to see the association of Vitamin D levels with Body Mass Index (BMI), Lipid parameters and Insulin resistance in a sample population representing the urban Indian youth.

Materials and Method: Fifty five apparently healthy young adults of 18 to 22 years age were recruited in this study and their serum 25-hydroxycholecalciferol(25OHD), serum insulin, Total Cholesterol, High Density lipoprotein-cholesterol (HDL-c), Low Density lipoprotein- cholesterol (LDL-c) and Triglyceride (TGL) levels were estimated. Insulin resistance(IR) was derived using the Homeostasis Model Assessment equation. Based on BMI, the participants were divided into Obese group with BMI $\geq$ 25 (n=21) and non-obese group with BMI < 25 (n=34).

Result: 25OHD levels were almost same in both Groups (Obese11.1 $\pm$ 4.6 ng/ml and non-obese 11.0 $\pm$ 5.2 ng/ml). Serum total cholesterol, VLDL and insulin levels were significantly increased in the Obese group(p= 0.005, p= 0.015 and p=0.054 respectively) when compared to the non-obese group. We found statistically significant association between 25-hydroxycholecalciferol (25OHD) and TGL/HDL ratio in the Obese group.

Conclusion: In this pilot study we have unravelled the subtle onset of metabolic derangement in Obese individuals with Vitamin D deficiency.

KEYWORDS: Insulin resistance, Metabolic dysfunction, Obesity, Vitamin D

INTRODUCTION:

Vitamin D, a nutritive bio-molecule, has long been associated with bone health. In the past few decades, the focus on the non-skeletal functions of calcitriol has gained momentum. Vitamin D acts via vitamin D receptors (VDR), which are situated on the nucleus of cell and has a wide distribution across different cell types of the body [1]. The non-skeletal functions of Vitamin D like cellular differentiation, immunological modulations and regulation of cell activity are mediated through these VDR [2]. Vitamin D thus plays a vital role in maintaining normal growth, development and well-being of an individual [3]. The sub optimal Vitamin D levels is associated with metabolic, autoimmune and other chronic diseases [4]. Calcitriol is the active form of vitamin D, but because of its shorter half-life of 4 to 6 hours, its precursor form, 25-hydroxy cholecalciferol also called calcidiol, the major circulating form of Vitamin D and one with a longer half-life of 2 to 3 weeks is preferred for measurement and analysis of Vitamin D levels in an individual [5].

Vitamin D qualifies to be a hormone for reasons obvious. Vitamin D is synthesized in the body, on exposure to sunlight. It exerts both autocrine and paracrine effect in the body. Its mechanism of action is mediated through the nuclear receptors substantiating its role as a hormone [6]. A low level of Vitamin D may therefore be the culprit in derangement of the inner harmony and can lead to development of various diseases.

The United States endocrine society defines Vitamin D sufficiency as 25-Hydroxycholecalciferol (25OHD) levels >30 ng/ml, range of 21-29 ng/ml as insufficiency and < 20 ng/ml

is deficiency [7]. Considering these cut-off values, about 1 billion people worldwide have vitamin D deficiency, while 50% of the population has vitamin D insufficiency [8]. The Community based Indian study reports, prevalence of vitamin D deficiency ranging from 50 % to 94 % [9]. The prevalence of vitamin D deficiency was 35% higher in obese subjects irrespective of latitude and age [10].

Obesity definition for South Asian population is a Body mass index (BMI)> 25 [11]. The growing economy all over the world has influenced people to indulge in unhealthy lifestyle and sedentary habits, which has resulted in Obesity epidemic. These changes have led to metabolic derangement in the body causing dyslipidemia and insulin resistance (IR). Vitamin D deficiency is the most commonly encountered feature in obesity. Whether it is a cause or consequence is a debatable subject.

In the current study, we have set out to on a quest to trace this interrelation between, obesity, Vitamin D status, metabolic derangement as measured by the extent of dyslipidemia and insulin resistance in a sample population of young adults in an anticipation that it may pave way for in-depth analysis of the same in the future.

MATERIALS AND METHODS

The study was conducted in Rajarajeswari Medical College and Hospital for a period of 6 months. This is a pilot study involving a sample population of Urban youth, between age group of 18 and 22 years. The subjects in majority are from upper socioeconomic strata of the society. All the individuals are apparently healthy with no history of any co-morbidities, medical or surgical illness. Fifty five MBBS students volunteered to be a part of this study. Their weight and height were measured according to standard protocol and Body Mass Index was calculated. A fasting venous blood sample was drawn from these volunteers and the following Biochemical investigations were conducted. Fasting Blood Glucose was estimated by GOD-POD method, Serum insulin, was estimated by e411 fully automated instrument by Electro-chemiluminescence Immuno-assay method. Lipid profile which included estimation of serum Total Cholesterol, High density lipoprotein–cholesterol, Low density lipoprotein–cholesterol, Triglycerides were measured. Total Cholesterol was estimated by Cholesterol oxidase-peroxidase method, HDL-c and LDL-c were estimated by direct method, Triglyceride was estimated by Glycerol 3-phosphate oxidase method. VLDL-was calculated by the formula VLDL=TGL/5.TG/HDL ratio is another calculated parameter of this study. Insulin resistance(IR) was derived using the Homeostasis Model Assessment equation (HOMA = fasting serum insulin(μunits/ml) $\times$ fasting plasma glucose (mmol/l)/22.5) . Students who had a HOMA score of more than 2.5 were taken as individuals with insulin resistance [12]. Vitamin D the principal bio molecule of interest in this study was measured on Maglumi-1000 Chemiluminescence immuno-assay based instrument. The Institutional Ethics committee approval was taken before beginning the study. A structured questionnaire-based history elicitation about, diet, sun exposure and lifestyle was done. Based on BMI the group of these 55 students were divided into two groups. Group I, Obese group consisted of students with BMI$\geq$ 25, Group II, non-obese group included students with BMI < 25. Accordingly, there were 21 students in group I and 34 students in Group II.

Statistical analysis

Data collected was systematically compiled and analyzed using suitable statistical tool. Mean and Standard deviation (SD) for all Biochemical and anthropometric parameters were calculated. Comparison of these parameters between the two groups were made using Student 't' test. Pearson's correlation was used to look for association between study variables. All statistical analysis was done at 5% level of significance.

RESULTS.

The reference ranges and the values of all the Biochemical and anthropometric parameters we observed in the whole group of 55 students are compiled inTable 1Figure 1 is a graphical representation of Vitamin D levels in Obese and non-obese group.

[Table 1 about here.]

Correlation analysis of 25OHD with various metabolic indices for the whole group is as notified inTable 2Table 3 is a comparative statement of all variables between Obese and non-obese groups. Tables 4 and 5 are correlation analysis of Vitamin D versus metabolic indices in Obese and non-obese group respectively

[Table 2 about here.]

[Table 3 about here.]

[Table 4 about here.]

[Table 5 about here.]

DISCUSSION

The subjects of the current study comprise a sample of apparently healthy young adults. Based on the answers given in the questionnaire, it was evident that all the fifty-five students led an active lifestyle and had adequate exposure to sunlight. The Mean $\pm$ SD values of all the Biochemical parameters assessed in these individuals are well within the normal reference ranges, except Vitamin

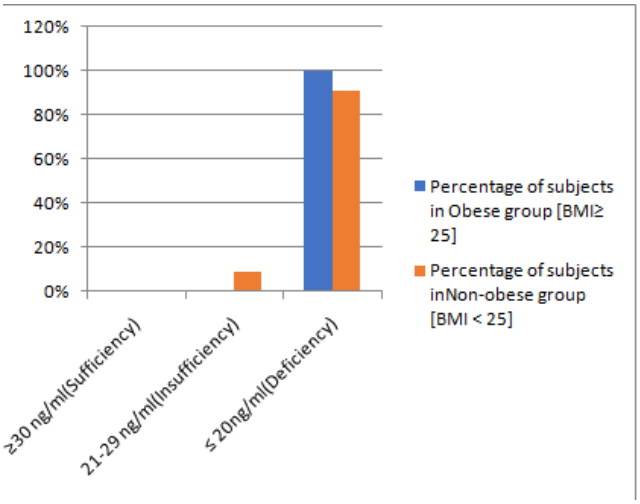

Figure 1: Vitamin D levels in the study groups

D as depicted in Table 1. The Vitamin D deficiency seen in majority of students in the study reinforces the fact about rampantly prevailing Vitamin D deficiency in our population. Of the 55 students, none fall into the range of Vitamin D sufficient levels. Three have levels between 21 and 29 ng/ml and the rest 52 are Vitamin D deficient. Prevalence of Vitamin D insufficiency has been observed in studies conducted on Young adults in different parts of the world, but the severity varies [13]. All 21 Obese subjects have Vitamin D deficiency, whereas in the non-Obese group three fall in insufficiency category and 31 have deficiency. This is graphically represented in Fig 1. Table 2 is correlation of Vitamin D with metabolic dysfunction markers like TGL/HDL ratio, BMI, HOMA-IR and serum insulin levels for the whole group, and we found no significance. The most plausible explanation for this would be the small sample size of this pilot study. After having segregated the subjects based on their BMI, into Obese and Non-Obese group, in Table 3 we can observe that the levels of serum Total Cholesterol, serum very low density lipoprotein and insulin levels are significantly more ($p = 0.005$, $p = 0.015$ and $p = 0.054$ respectively) in Obese group compared to the Non-obese group. A study by Eslami et al on larger medical student population in Iran have reported similar findings of lipid derangement in Obese individuals [14]. The meager sample size of the current study has not deterred to unmask the tyranny caused by Obesity on lipid parameters and Insulin sensitivity. When the indicators of metabolic dysfunction were correlated with vitamin D levels, in Obese group, Vitamin D versus TG/HDL ratio showed a significant correlation ($p = 0.037$). The same kind of correlation was observed by Michos ED et al in a large database study [15]. No such association was noted in the non-obese group of the current study.

CONCLUSION:

In this pilot study we have picked up the subtle onset of metabolic derangement in Obese individuals with Vitamin D deficiency. Further an association between TGL/HDL ratio, an insulin resistance index with Vitamin D levels shows that Vitamin D and Obesity are a dangerous combination which can lead to onset of metabolic dysfunction at an early age. As the debate of Vitamin D being an implication or etiology of Obesity continues, we intend to continue our exploration in this direction in a large prospective study in a similar population.

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Conflict of interest: None

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LIST OF TABLES

Sl no.	Parameter	Reference range	Mean$\pm$ Standard Deviation
1	Fasting Blood Sugar(mg/dl)	70-100	87.18 $\pm$ 10.9
2	Total Cholesterol (mg/dl)	150-200	145.53 $\pm$ 32.24
3	High Density lipoprotein (mg/dl)	>40 in males >50 in females	45.05 $\pm$ 7.29
4	Low Density lipoprotein (mg/dl)	<125	88.16 $\pm$ 27.8
5	Very low Density lipoprotein (mg/dl)	10-50	17.89 $\pm$ 7.37
6	Triglycerides(mg/dl)	< 150	92.2 $\pm$ 41.74
7	TG/HDL ratio	<3	2.11 $\pm$ 1.05
8	Vitamin D(ng/ml)	>30	11.04 $\pm$ 4.96
9	Serum Insulin (μIU/ml)	3-17	7.25 $\pm$ 2.02
10	Body Mass Index (Kg/m^2)	< 25	24.25 $\pm$ 4.35
12	HOMA-IR	$\leq$ 2.5	1.56 $\pm$ 0.48

Table 1: Reference ranges and descriptive statistics of Biochemical and anthropometric parameters of students studied

Sl no	Correlation between	'r' value	'p' value
1	Vitamin D and Serum insulin	-0.0273	0.84
2	Vitamin D and BMI	-0.075	0.58
4	Vitamin D and HOMA-IR	0.028	0.84
5	Vitamin D and TG/HDL ratio	0.215	0.12

Table 2: Pearson correlation between Vitamin D and other Biochemical and anthropometric parameters for the whole group

Sl no.	Parameter	Obese gp[BMI$\geq$ 25] (n = 21) Mean$\pm$SD	Non Obese gp[BMI < 25] (n = 34) Mean$\pm$SD	'p' value
1	Fasting Blood Sugar(mg/dl)	87.3 $\pm$ 11.8	87.1 $\pm$ 10.6	0.948
2	Total Cholesterol(mg/dl)	160.8 $\pm$ 34.9	136.1 $\pm$ 27.0	**0.005**
3	High Density lipoprotein(mg/dl)	45.2 $\pm$ 6.8	45.0 $\pm$ 7.7	0.922
4	Low Density lipoprotein(mg/dl)	96.0 $\pm$ 25.3	83.3 $\pm$ 28.5	0.100
5	VerylowDensitylipoprotein(mg/dl)	20.9 $\pm$ 8.1	16.0 $\pm$ 6.3	**0.015**
6	Triglycerides(mg/dl)	104.7 $\pm$ 40.7	84.5 $\pm$ 41.1	0.081
7	TG/HDL ratio	2.4 $\pm$ 0.9	1.95 $\pm$ 1.1	0.121
8	Vitamin D(ng/ml)	11.1 $\pm$ 4.6	11.0 $\pm$ 5.2	0.942
9	Serum Insulin (μIU/ml)	7.9 $\pm$ 3.1	6.83 $\pm$ 0.6	**0.054**
10	Body Mass Index	28.6 $\pm$ 3.3	21.6 $\pm$ 2.3	**0.0001**
11	HOMA-IR	1.7 $\pm$ 0.7	1.5 $\pm$ 0.2	0.123

Table 3: Comparisons Biochemical and Anthropometric parameters between obese and non obese groups

Sl no	Correlation between	'r' value	'p' value
1	Vitamin D and Serum insulin	-0.134	0.57
2	Vitamin D and BMI	-0.106	0.65
4	Vitamin D and HOMA-IR	-0.043	0.85
5	Vitamin D and TG/HDL ratio	0.457	**0.037**

Table 4: Pearson correlation between Vitamin D and other Biochemical and anthropometricparameters for the Obese group [BMI$\geq$ 25] (n = 21)

Sl no	Correlation between	'r' value	'p' value
1	Vitamin D and Serum insulin	0.214	0.22
2	Vitamin D and BMI	-0.159	0.36
4	Vitamin D and HOMA-IR	0.168	0.33
5	Vitamin D and TG/HDL ratio	0.109	0.54

Table 5: Pearson correlation between Vitamin Dand other Biochemical and anthropometric parameters for the non-obese group[BMI < 25] (n = 34)