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Plasma Vitamin D Status in Obese and Non-Obese Individuals: A Comparative Study

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INTRODUCTION

Obesity is a serious health problem in the world. The prevalence of overweight and obesity globally according to WHO estimation was around 1.5 billion and 500 million adults in 2008 Obesity is defined as an excess accumulation of body fat. Being overweight or obese isn't a cosmetic problem. These conditions greatly raise your risk for other health problems. Overweight and Obesity-Related Health Problems in Adults are Coronary Artery Disease, High Blood Pressure, Type II Diabetes mellitus, Stroke, Abnormal Blood Fats, Metabolic Syndrome, Cancer, Osteoarthritis.

Obesity can be measured by many indices which include Body mass index, Fat percentage, Waist Hip Ratio, Waist circumference and Hip circumference. Vitamin D is a secosteroid that plays an essential role in maintaining a healthy mineralized skeleton for most land vertebrates including humans. It maintains the calcium-phosphorous homeostasis. More recently vitamin D deficiency has been related to pathogenesis of such co-morbidities as Insulin resistance, Diabetes Mellitus, Hypertension, Dyslipidemia, Cardiovascular disease, Cancer. Vitamin D deficiency is also an important worldwide public health problem. Risk factors for vitamin D deficiency are low dietary intake, low sun exposure,skin pigmentation, obesity, malabsorption and advanced age.

Although the explanation for the increased risk of Vitamin D deficiency in obesity is unknown, ,low levels of Vitamin D in obese people can be attributed mainly to :-

1. Lower bioavailability of Vitamin D due to sequestration by adipose tissue.
2. The dilution of ingested or cutaneously synthesized vitamin D in enlarged fat mass.
3. Low sun exposure, due to mobility limitations or low sun exposure of large areas of body.

Calcium is tightly regulated by vitamin D and parathormone by their activity in the skeletal

system, kidney and intestine. Serum calcium levels vary according to the vitamin D status. The lipid profile is a blood test done to assess the status of fat metabolism in the body. This includes measuring lipids (fat) and its derivatives known as lipoproteins. Total cholesterol comprises all the cholesterol found in various lipoproteins such as high density lipoprotein [HDL], low density lipoprotein [LDL], and very low density lipoprotein [VLDL]. Triglycerides are neutral fats found in the tissue and blood .Triglycerides containing lipoproteins may also contribute to thedisorder related to coronary heart disease. The main function of HDL is to help soak up excess cholesterol from the walls of blood vessels and carry it to the liver, where it breakdown and is removed from the body in the bile .It is thus called "good cholesterol" .LDL contains the greatest percentage of cholesterol and is responsible for cholesterol deposits on the walls of artery resulting in coronary artery disease (bad cholesterol). As obesity is the accumulation of excess fat, definitely the lipid profile will be deranged among obese individuals9.As reported previously, dyslipidemia associated with vitamin D deficiency. Literature regarding the vitamin D status and the different indices of obesity are conflicting and scarce.

The present study is aimed to find out the level of vitamin D in obese and non-obese individuals. In this study we hypothesized that vitamin D level is low in obese individuals compared to non-obese. We wanted to estimate the levels of vitamin D, calcium and lipid profile (LDL, HDL, Total cholesterol and Triglycerides) in obese and non-obese individuals and to compare the above parameters in two groups.

AIMS AND OBJECTIVES

Primary objective

To measure the Vitamin D levels among obese individuals and compare with vitamin D levels in non-obese individuals.

Secondary Objective

To assess the correlation of vitamin D level with degree of obesity, lipid profile and calcium in obese and non-obese individuals.

REVIEW OF LITERATURE

Vitamin D and Type 1 Diabetes Mellitus

Type 1 diabetes mellitus (T1DM) is characterized by disturbance in the glucose metabolism due to the insulin deficiency caused by the destruction of beta cells of pancreas. It is a chronic autoimmune disease induced by immune cells and cytokines causing insulitis. The exact mechanism still remains unknown.65 1,25-(OH)2D3 inhibit the expression of CD14, Toll-like receptor (TLR4), IL- 1, and TNF-_, IFN-_, IL-17 and IL-2and other proinflammatory cytokines. as well as up-regulate CTLA- 4 (cytotoxic T lymphocyte associated protein -4 which down regulates immune system) and FoxP3(member of FOX family of proteins, which functions as transcription factor in the function of T cells) of T cells. Certain studies show that, it can also reduce the NF-kB-p65 phosphorylation levels of the monocytes in the patients with Type 1 DM.

Vitamin D and Type II Diabetes Mellitus

Several studies suggest relation between vitamin D deficiency and diabetes mellitus type II. Some have also suggested the mechanisms involved. This include

- Evidence of VDR and the 1 alpha hydroxylase enzyme in the beta cells of pancreas and the presence of the vitamin D response element (VDRE) in the human insulin gene promoter sequence
- Activation of transcription of human insulin gene by 1,25 (OH)2 D has been Experimentally proved.
- Researches shown that vitamin D exerts an indirect effect on the secretion on insulin by normalization of calcium level in the blood. Calcium mediated exocytosis is the mechanism of insulin secretion. Thus a deficiency of vitamin D can cause decrease in insulin secretion.

- Angiotensin II inhibits the action of insulin in skeletal muscle and vascular tissue. It causes a decreased insulin uptake by these tissues. It can contribute to the development of insulin resistence. 71 Data support vitamin D-VDR complex regulates renin activity and thus vitamin D deficiency can lead to increased RAAS system.

Vitamin D and obesity

Vitamin D aids weight loss.

1, 25-dihydroxy vitamin D3 [1,25 (OH) 2D3] inhibits adipogenesis in vitro. The calcitriol binds with pre adipocyte 3T3-L1 cell (mouse cells having fibroblast like morphology, but can differentiate into adipocytes) and blocks its differentiation into mature adipocyte. There is evidence of expression of VDR during the early phases of adipogenesis and calcitriol binding to the pre adipocytes rather than mature adipocytes. The inhibition of differentiation is ineffective 24-48 hrs after the differentiation is started. Treatment of 3T3-L1 cells with Vitamin D blocks the expression of C/EBPalpha,), sterol regulatory element-binding protein-1, peroxisome proliferator activated receptor-gamma (PPAR gamma) and other downstream adipocyte markers. Vitamin D down regulates the sterol regulatory element-binding protein-1 expression through its action on of Insulin-induced gene-1 (Insig-1) and its homolog Insig-2. Vitamin D has role in activation of these genes. VDRE has been recognized in the promoter regions of those genes. They encode closely related proteins of the endoplasmic reticulum. These proteins block proteolytic activation of sterol regulatory element binding proteins. SREBP -1 are membrane-bound transcription factors that activate synthesis of cholesterol and fatty acids in animal cells as well as differentiation of preadipocytes into adipocytes. 1,25-(OH)2D3 can directly suppress the expression of peroxisome proliferator-activated receptor gamma protein and inhibits adipocyte differentiation of 3T3-L1 preadipocytes and murine bone marrow stromal cells.

Another study suggests the mechanism of adipogenesis is by the increased intracellular calcium in adipocytes that lead to stimulation of fatty acid synthase complex and thus promote lipogenesis. It has also role in adipolysis. So on calcium and vitamin D supplementation, no increased action of 1_ hydroxylase action in adipocytes, no increased production of intrace-llular calcium in adipocytes and no stimulation of lipogenesis,the study says that calcium and vitamin D supplementation promoted weight loss.

Polymorphisms in the VDR gene are associated with the susceptibility to obesity in subjects with early onset of Type II DM. The pathophysiological mechanisms of these associations remain unexplained, but they could be related to a direct effect of vitamin D in adipocyte differentiation and metabolism, or to an indirect effect by modulation of insulin secretion.

MATERIALS AND METHODS

STUDY DESIGN: Hospital based cross-sectional study.

STUDY SETTING: Individuals attending obesity clinic conducted by Department of Physical medicine and Rehabilitation and applied nutrition as cases and Age and sex matched non-obese individuals attending the department of physical medicine and Rehabilitation as comparative group. Investigations conducted at Department of Biochemistry.

STUDY PERIOD: One year

STUDY POPULATION: The study group consisted of age and sex matched 20 obese and 20 non-obese individuals, satisfying the inclusion and exclusion criteria

INCLUSION CRITERIA: All adults attending obesity clinic with BMI more than 30 were included as cases. Individuals with BMI less than 30 were included as comparative group.

EXCLUSION CRITERIA: Obese patients who have done Bariatric surgery before test have been excluded. Patients who are on supplementation of

Vitamin D or Calcium have been excluded. Pathological conditions that interfere with assessment of obesity like liver failure, renal failure have also been excluded.

METHODOLOGY: After obtaining informed consent, the proforma was filled up which contains patient details, past medical history, dietary history, drug history and sun exposure score.

RESULTS AND DISCUSSION

- Statistical analysis was performed using SPSS for windows version 22
- Quantitative variables were described by mean and standard deviation and qualitative variables were described by frequency distribution.
- Between the groups comparison of qualitative variables were analysed by Chi square test.
- Between the groups comparison of quantitative variables were analysed by independent sample t test.
- Relationship of quantitative variables analysed by Pearson correlation coefficient
- A p value of 0.05 was considered as level of significance

Various study parameters among obese and non –obese individuals

Table-1

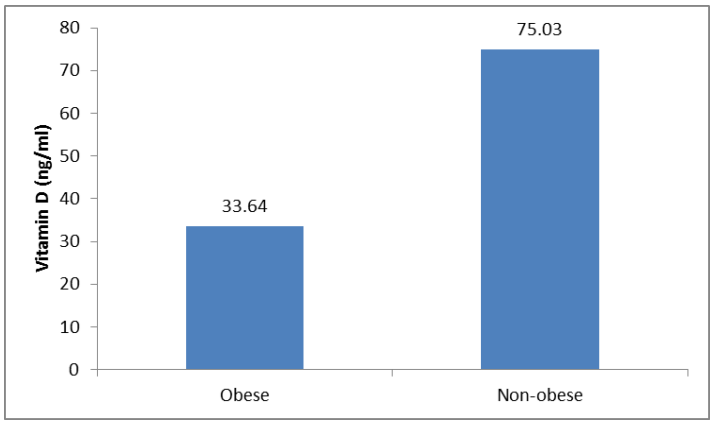
	Obese (N=20)		Non obese (N=20)		t	p
	Mean	SD	Mean	SD		
BMI	35.71	4.77	22.65	2.29	11.032	.000
Fat%	48.75	9.92	27.80	5.90	8.118	.000
WC	113.45	13.86	84.58	6.45	8.447	.000
HC	122.50	10.53	93.05	7.36	10.255	.000
WHR	0.92	0.07	0.91	0.06	.681	.500
Vitamin D	33.64	23.71	75.03	37.84	-4.145	.000
Calcium	8.39	0.97	9.38	0.42	-4.171	.000
TG	137.00	72.65	132.20	41.05	.257	.798
Cholesterol	208.75	40.77	184.50	23.55	2.304	.027
HDL	41.40	11.22	41.15	5.74	.089	.930
LDL	139.82	35.03	114.90	24.36	2.612	.013

Comparison of mean vitamin D values between obese and non-obese individuals

Table-2

BMI	N	Vitamin D		T	P
		Mean	SD		
Obese	20	33.64	23.71	-4.145	<0.001
Non-obese	20	75.03	37.84		
Total	40	54.33	37.56		

Graph.1



The mean vitamin D levels in the obese and non-obese groups were 33.64 and 75.03 respectively. The difference is found to be statistically significant. (p value<0.05

Correlation with Vitamin- D level	Pearson correlation	P
Age	.125	.443
BMI	-.535	.000
Fat%	-.407	.009
WC	-.503	.001
HC	-.533	.000
WHR	-.073	.654
Calcium	.378	.016
TG	-.169	.297
Total Cholesterol	-.261	.104
HDL	-.083	.613
LDL	-.182	.261
Sun Exposure Score	.337	.033

20 obese and 20 non- obese individuals with respect to their BMI are selected for the study.

The mean BMI of the obese group is 35.7 ± 4.7 and that of non- obese is 22.65 ± 2.29.

Of these, 35% fall into the age group less than 30, 25% fall into the age group 31-40 and 40% fall into the age group more than 40. Chi square test was done and this difference in age is not statistically significant. The mean age of the obese group is 36.75 ± 11.62 and of the non- obese individuals is 36.75±10.1 in the present study.

In this study, 15% of the obese individuals and 20% of the non-obese individuals are males. Among the obese patients 85% are females while among the non-obese individuals 80% are females. This difference may be due to their inhibition to attend the clinic. The difference in gender is not found statistically significant on doing chi square test among 2 groups.

The cases and the comparative group selected with similar sun exposure score as sun exposure is a confounding factor that determines the vitamin D status of an individual. The mean Sun exposure score of obese group is 17.08 ±1.7 and that of non- obese group is 17.2 ±1.77. The difference in the 2 groups are statistically insignificant on doing chi square test As vitamin D is rich in non-vegetarian diet, dietary history was taken, and all of them fall into non vegetarian diet category. This study was done in the in the months of April, May when there is enough sunshine. Both the cases and the comparative group sampled in the same season.

We found that mean vitamin D level in study group is lower than the comparative group. The mean plasma vitamin D level among obese patients was 33.64± 23.71 and in non-obese 75.03± 37.84. By doing student t test the difference in mean vitamin D levels are found statistically significant (Table-2, Graph-2). Similar results were obtained in a study conducted by Erlend T Aasheim, Dag Hofso, in morbidly obese patients. Their vitamin D levels were low compared to the non-obese group. (p value <0.01)[87] In overweight or obese women, an increase of 1kg/m^2 in BMI has been associated with decrease of 1.21 nmol/l in 25 (OH) D levels[91]

Some studies observed that intake of calcium and vitamin D can promote weight loss. In a Randomized Control Trial conducted by Weizhu et al, 53 subjects assigned to receive calcium and vitamin D supplementation for 12 weeks or energy restriction only. Significant fat loss observed in the 1st group (p<0.02)[110]

CONCLUSION

Plasma vitamin D levels are significantly low in obese individuals compared to non-obese individuals.

A significant negative correlation is observed for Vitamin D levels with adiposity.

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