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CORRELATION BETWEEN LEVELS OF SERUM 25(OH)D AND LIPID PROFILE COMPONENTS IN STUDY POPULATION

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ABSTRACT

Background: Since the prevalence of vitamin D deficiency is high in some countries and the trend of hypertension and obesity is increasing, this study aimed to investigate the serum level of vitamin D and explore its correlation and association with obesity, blood pressure, and lipid profile. Our hypothesis is that serum vitamin D levels are associated with risk factors for cardiometabolic disease such as hypertension. **Objective**: To find out the correlation between levels of serum 25(OH)D and lipid profile components in study population. **Methods:** This cross-sectional analytical study was carried out in the Department of Biochemistry of Sir Salimullah Medical College & Mitford Hospital, during July 20- June 21. 100 female subjects aged belong to 19-29 yrs were selected by purposive sampling. Among them 50 women were obese (BMI ≥25 kg/m²) and 50 women were non-obese (BMI< 25 kg/m²). Initial evaluation was done by history taking and anthropometric indices were measured. **Results:** Vitamin D had no significant (p>0.05) relationship (r=0.032,p=0.755) with age. Vitamin D had a significant inverse relationship (r=-0.264,p=<0.01) with BMI. It was also inversely related to WC (r=-0.209, p=<0.05) which was significant (p<0.05).Vitamin D had a significant (p<0.05) inverse relationship with WHR, TC, TAG and LDL-C (r=-0.246, -0.299, -0.276, -0.257, p= 0.014, 0.003, 0.005, 0.010 respectively). **Conclusion:** Obese young women have low serum vitamin D level. There is a significant correlation between levels of serum 25(OH)D and lipid profile components in young women.

KEYWORDS: Obesity, Young adults, Vitamin D deficiency.

INTRODUCTION

According to the guidelines of US Endocrine Society, the serum levels of 25(OH)D below 20 ng/mL (50 nmol/L) are stated as vitamin D deficiency while 25(OH)D serum levels between 21–29 ng/mL (52.5–72.5 nmol/L) are defined as vitamin D insufficiency.[1] For subjects with serum 25(OH)D levels between 20 and 21 ng/mL, it is generally accepted that values of serum 25(OH)D levels below 20.5 ng/mL are taken as 20 ng/mL and, therefore, considered vitamin D deficiency while serum 25(OH)D levels ≥ 20.5 ng/mL are taken as 21 ng/mL and considered to be vitamin D insufficient.[2]

When serum calcium levels decrease, PTH-dependent calcitriol activation prompts the formation and VDR-mediated differentiation of osteoclasts. This activation induces the mobilization of calcium from the bone by stimulating the secretion of the receptor activator for nuclear factor kappa-B ligand, which, in turn, is responsible for osteoclastogenesis and bone resorption.[3] At the same time, vitamin D inhibits mineralization through the increase of pyrophosphate levels and osteopontin.[4] Calcitriol promotes bone formation and growth, by activating chondrocyte differentiation, and increasing serum calcium and phosphate levels. Thus, vitamin D deficiency results in inadequate mineralization of the skeleton, and when low vitamin D levels are maintained, bone growth plates cannot be mineralized due to calcium and phosphate depletion.[5,6]

Obesity is associated with several comorbidities such as cardiovascular (CV) disease, hypertension (HTN), stroke, type 2 diabetes mellitus (T2DM), dyslipidemia, osteoarthritis and some cancers. According to WHO

(2020) around 2.8 million people are dying each year as a result of being overweight or obese.

As with obesity, vitamin D deficiency is reaching epidemic proportions worldwide, in both pediatric as well as adult populations.[7] Evidence is accumulating to suggest that there is a potential link between obesity and vitamin D deficiency among global populations.[8] Several studies reported low serum level of Vitamin D in obese individuals.[9,10]
Vitamin D can be supplied via food or supplements and can also be synthesized in the skin by the action of ultraviolet light; however, the prevalence of vitamin D insufficiency or deficiency has been increasing and has become an emerging public health issue, particularly among young Asian women.[1,11,12]

It remains unclear whether low vitamin D status is responsible for the development of obesity or whether obesity results in vitamin D insufficiency or deficiency. Low serum 25(OH)D is more likely a consequence of obesity, rather than the cause of obesity (Paul et al. 2020). In a meta-analysis, the prevalence of vitamin D deficiency was 35% higher in obese subjects compared to the eutrophic group and 24% higher than in the overweight group (Pereira-Santos et al., 2015). One "superfluous" BMI unit is known to induce a 1.15% reduction in the 25(OH)D concentration.[13] Evidence also supports the inverse association of fat mass with 25(OH)D levels.[14]

In Bangladesh, Kamrul-Hasan et al. (2018) have reported a high prevalence of vitamin D deficiency in various subsets of the population.[15] According to a meta-analysis of studies on vitamin D deficiencies, there are racial or regional differences in vitamin D status. For example, the level of 25-hydroxyvitamin D[25(OH)D], a proxy for vitamin D status, was generally higher in North America than in other countries, such as Europe, the Asia Pacific region, the Middle East, Africa, or South America.[16]

OBJECTIVES

General objectives

To find out the correlation between levels of serum 25(OH)D and lipid profile components in study population.

Specific objectives

1. To measure anthropometric indices (BMI ,WHR) of the study subjects.
2. To estimate the components of lipid profiles (TAG,TC, HDL-C, LDL-C) of the study subjects.
3. To determine serum 25(OH)D level of the study subjects.

METHODOLOGY

Type of study:	Cross sectional analytical study.
Place of study:	Department of Biochemistry of Sir Salimullah Medical College, Dhaka, Bangladesh.
Study period:	1st July 2020 to 30th June 2021
Study population:	100 apparently healthy young women aged 19-29 years were included in the study
Sampling technique:	Purposive convenient sampling.
Sample Size:	50 subjects were included from each of non-obese and obese group.

Inclusion criteria

Apparently healthy young women from 19 yrs to 29 yrs of age.

Exclusion criteria

- Underweight and morbid obese.
- Pregnancy.
- Breastfeeding.
- Diabetes mellitus.
- Hypertension.
- Renal disorder.
- Thyroid disease.

Grouping of study subjects

Group A: Non-obese young women.
Group B: Obese young women.

Variables of the study

- Age
- Body Mass Index (BMI)
- Waist Circumference (WC)
- Serum 25(OH)D level
- Serum fasting lipid profile includes
- Total cholesterol (TC)
- Triacylglycerol (TAG)
- Low density lipoprotein (LDL-C)
- High density lipoprotein (HDL-C)

Study procedure

Female subjects were selected from Matlab-North, Chandpur. Ethical permission was taken from the Ethical Review Committee of Sir Salimullah Medical College. After proper counseling aim, objectives, risk and procedure of the study were explained in details to all participants. Only voluntary candidates were recruited as participants of the study. They had the freedom to withdraw themselves from the study at any stage. Written informed consent was taken from all participants. Socio-demographic as well as other relevant data were taken and recorded in the data collection sheet

with a prefixed questionnaire. Age and anthropometric measurements of each subjects was recorded following standard procedure. Blood sample was collected for biochemical variables to be measured.

Blood sample collection

Fasting blood samples was collected from all participants. They were asked to fast overnight (10 – 12 hours). Blood was collected from the antecubital vein after all aseptic precautions. 4 ml venous blood was taken in a plane tube by sterile disposable syringe. Then serum was separated after centrifuging at 3000 rpm for 10 minutes and was collected into two eppendorff tubes with labeled properly.

Data Collection and Processing

Before collecting specimen, each patient were interviewed and relevant information were recorded systematically in a pre-designed standard data sheet. Then data were checked, edited and compiled.

Data analysis

Data were analyzed with the help of software SPSS (Statistical Package for Social Sciences) version 22.0 for Windows (SPSS Inc., Chicago, IL, USA). Qualitative data were expressed as frequency and percentage (%). Quantitative data were expressed as mean ± standard deviation and tested by unpaired t-test. Data were normal distribution. Pearson linear correlation was used to observe the correlation between parameters. Chi-square test was done to observe association between qualitative variables. p value of <0.05 was considered statistically significant.

RESULT

Table I: Correlation of vitamin D with different variables in study subjects (n=100).

Biochemical parameters	r value	p value
AGE	0.032	0.755
BMI (kg/m^2)	-0.264	<0.01
WC	-0.209	<0.05
WHR	-0.246	0.014
TC (mg/dl)	-0.299	0.003
TAG (mg/dl)	-0.276	0.005
LDL-C (mg/dl)	-0.257	0.010
HDL-C (mg/dl)	0.110	0.274

Table I shows analysis was done by Pearson`s correlation. It was evident that vitamin D had no significant (p>0.05) relationship (r=0.032,p=0.755) with age. Vitamin D had a significant inverse relationship (r=-0.264,p=<0.01) with BMI. It was also inversely related to WC (r=-0.209, p=<0.05) which was significant (p<0.05).Vitamin D had a significant (p<0.05) inverse relationship with WHR, TC, TAG and LDL-C (r=-0.246, -0.299, -0.276, -0.257, p= 0.014, 0.003, 0.005, 0.010 respectively). It had no significant(p>0.05) relationship (r=0.110,p=0.274) with HDL-C.

Table II: Distribution of study subjects accordingly to vitamin D status (n=100).

Vitamin D status	Group A(n=50) number(%)	Group B(n=50) number(%)
Normal	27(54%)	18(36%)
Insufficient	23(46%)	32(64%)

Vitamin D levels

≥30 ng/ml= Normal.
<30 ng/ml= Insufficient.

Table II shows vitamin D distribution of the study subjects. Among 50 non-obese young women 27(54%) had normal vitamin D and 23(46%) had vitamin D insufficiency. However, 32(64%) of the 50 obese young women had vitamin D insufficiency and 18(36%) had normal vitamin D.

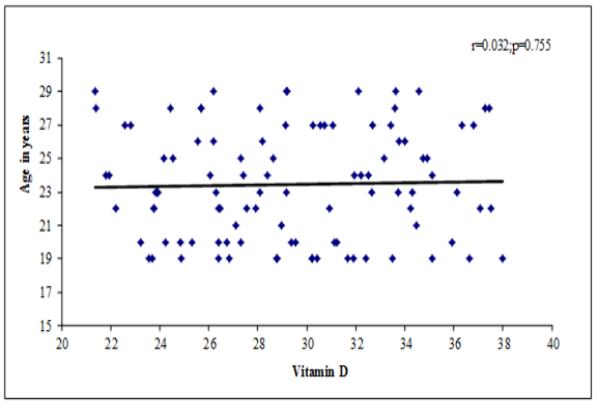

Figure 1: Scatter diagram showing correlation (r= 0.032; p= 0.755) between vitamin D and age.

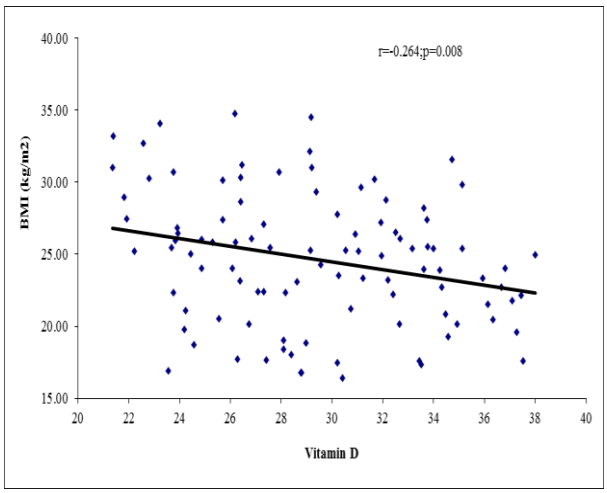

Figure 2: Scatter diagram showing negative correlation (r= -0.264; p<0.01) between vitamin D and BMI.

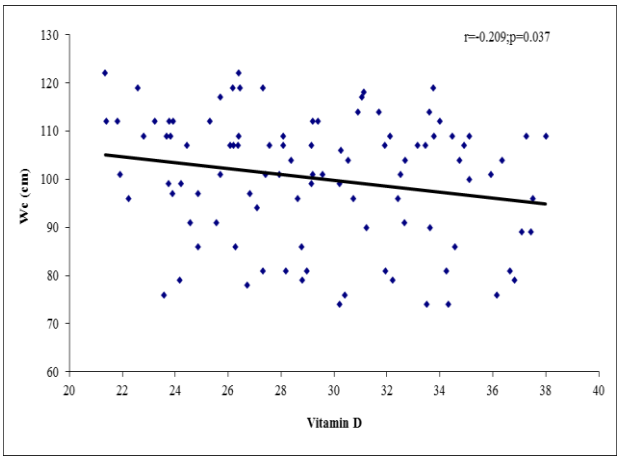

Figure 3: Scatter diagram showing negative correlation (r= -0.209; p<0.05) between vitamin D and WC.

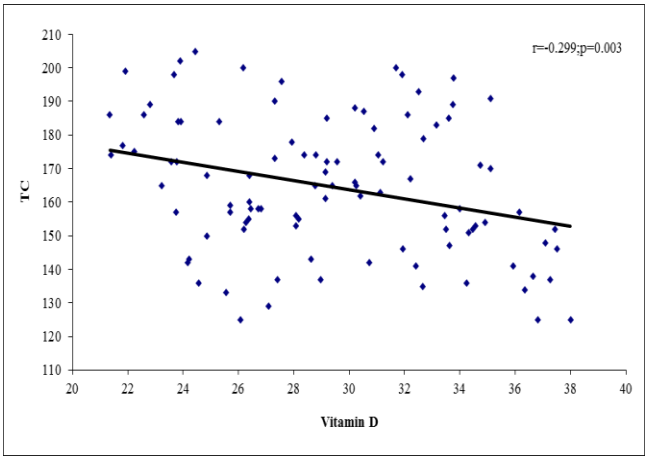

Figure 5: Scatter diagram showing negative correlation (r= 0.299; p<0.01) between vitamin D and TC.

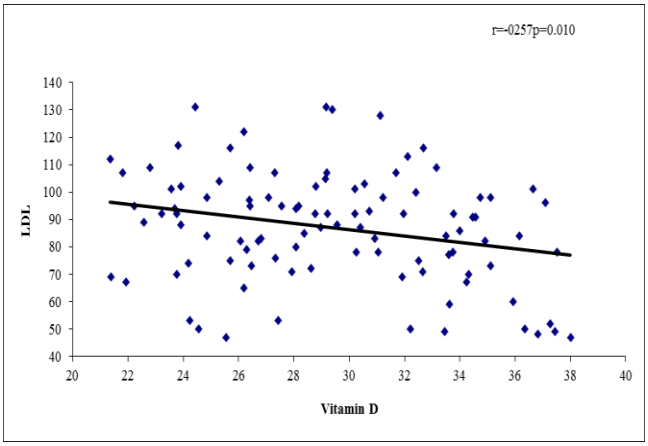

Figure 7: Scatter diagram showing negative correlation (r= -0.257; p<0.05) between vitamin D and LDL-C.

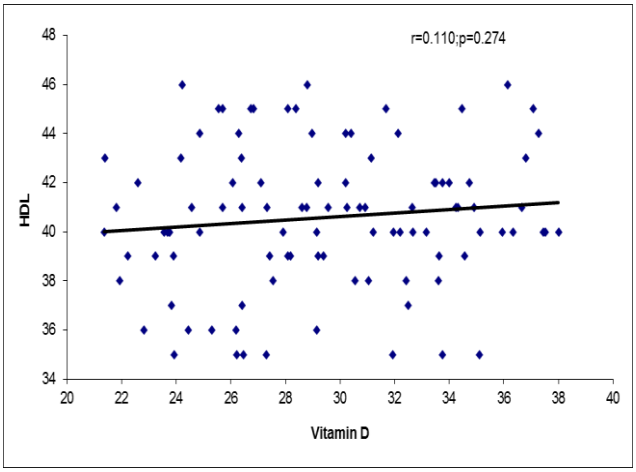

Figure 8: Scatter diagram showing correlation (r= 0.110; p= 0.274) between vitamin D and HDL-C.

DISCUSSION

A total of 100 young women aged 19-29 years were included in this cross sectional study based on predefined enrollment criteria. Among them, 50 were non-obese and 50 were obese women. This study was conducted in the Department of Biochemistry, Sir Salimullah Medical College & Mitford Hospital, Dhaka during July`20 - June`21 to assess the relationship between obesity and serum vitamin D levels.

Vitamin D showed inverse relationship with TC, TAG and LDL-C and it had no relationship with HDL-C in the present study. The serum 25(OH)D levels were inversely associated with TAG and LDL‑C and positively associated with TC in Chinese adults as observed by Wang et al. (2016).[17] Paul et al. (2020) reported that 25(OH)D levels did not correlate with either total cholesterol, HDL-C, or triacylglycerol levels; only LDL-C showed a significant positive correlation with vitamin D level.[15] It was observed through the study that

vitamin D had an inverse relationship with BMI, WC and WHR. These findings were consistent with the studies of Paul et al. (2020); Taheri et al. (2012) and Wortsman et al. (2000).[18,19]

Frequency of vitamin D insuffiency were 32(64%) and 27(54%) in obese and non-obese young women respectively. Jungert, Roth and Neuhauser-Berthold, (2012) and Karatas et al. (2013) reported that the prevalence of 25(OH)D deficiency as 40% to 80% among obese people.[20] Hamza and Hasan (2020) also found vitamin D deficiency was more prevalent in overweight and obese patients.[21] In a meta-analysis Yao et al. (2015) indicated that vitamin D deficiency is more common among obese subjects and the obesity-vitamin D deficiency association is stronger in the Asian population than in the European–American population.[22]

It remains unclear whether low vitamin D status is responsible for the development of obesity or whether obesity results in vitamin D insufficiency or deficiency. Paul et al. (2020) mentioned that low serum 25(OH)D is a consequence of obesity not the cause of obesity.[15] The underlying mechanisms of hypovitaminosis D in obesity is not clear. There could be lower vitamin D input because of lower dietary intake, lower sunlight exposure, or impaired skin synthesis of vitamin D.[15] Some studies suggested that vitamin D deficiency can favor higher adiposity by promoting elevated parathyroid hormone levels and more calcium inflow into adipocytes which will increase lipogenesis through which acetyl-CoA is converted to triacylglycerol for storage in fat and packaged within lipid droplets.[23,24] Obesity has been shown to be associated with decreased serum 25(OH)D level.[25,26] The possible reason for this association backs to the characteristics of Vitamin D itself is a fat soluble vitamin. Higher body fat decreases the availability of circulating 25(OH)D.[27] On the other hand obese persons have higher fat content, which could block 25(OH)D to be halted into the body tissue and consequently lowers the circulating serum 25(OH)D.[21] A large genetic study found that high BMI and genes that predispose to obesity decrease serum 25(OH)D. Alterations in protein binding or faster metabolic clearance in obesity could lead to lower serum 25(OH)D. The lower serum 25(OH)D could be because of the distribution of 25(OH)D into a larger whole body tissue volume, particularly if 25(OH)D were actively sequestered in other tissues.[19,28] Lenchik et al. (2003) reported that the expression of enzymes affecting vitamin D metabolism has been demonstrated in adipocytes, and the relationship between obesity and vitamin D is increasing.[29] Obesity is known to decrease vitamin D synthesis by increasing the expression of enzymes that adversely affect vitamin D metabolism.[30,31] Therefore, further research is needed to clarify the mechanism involved in humans.

CONCLUSIONS

In conclusion, we can say that young obese women had significantly lower serum vitamin D levels than non-obese young women. There is a significant correlation between levels of serum 25(OH)D and lipid profile components in young women.

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