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Vitamin D Status and Dyslipidaemia Among Obese Individuals in Ibadan, Nigeria: A Pilot Study

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Abstract

Objective. Obesity is a major global health concern characterized by an accumulation of excessive body fat, dyslipidaemia and low vitamin D levels. This study aimed to assess the relationship between dyslipidaemia and vitamin D status in obese individuals and the risk of atherogenesis.

Methodology. In this cross-sectional study, 140 participants aged 18 to 65 years, were categorized into 4 equal groups of 35 each, based on their body mass index BMI. Baseline and demographic data were obtained using a semi-structured questionnaire. The serum levels of vitamin D, total cholesterol (TC), triglyceride (TG) and HDL-cholesterol (HDL-c) were measured using standard methods. Low-density lipoprotein cholesterol (LDL-c) and atherogenic index in plasma (AIP) were calculated.

Results. The vitamin D status was sufficient in all groups but its concentrations decline significantly as BMI increases. Serum TC, TG, LDL-c concentration significantly increases as BMI increases, but HDL-c concentration decreases. The AIP increases as BMI increases.

Conclusion. The study provided possible evidence to support the association between dyslipidaemia and inadequate vitamin D status in this cohort of adults with obesity in Ibadan, Nigeria.

Key words: obesity, dyslipidaemia, vitamin D status, atherogenic index

INTRODUCTION

Obesity is a multifaceted, long-term metabolic disorder characterized by an excessive buildup of body fat due to an imbalance between the amount of energy consumed and expended.[1] It is linked to the occurrence of cardiovascular diseases (CVD), hypertension, type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), polycystic ovary syndrome (PCOS), insulin resistance (IR), dyslipidaemia (which are features of metabolic syndrome) and different types of cancers. These factors are associated with increased rates of morbidity and mortality, worse quality of life, and societal stigmatization.[2] Dyslipidaemia is characterized by elevated concentrations of serum total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-c), as well as low concentrations of high-density lipoprotein cholesterol (HDL-c), thus, these lipid markers can be used to assess the presence of obesity.

Visceral obesity causes insulin resistance through the action of adipokines and free fatty acids (FFA). Adipokines, such as resistin and retinol-binding proteins, reduce insulin sensitivity; whereas leptin and adiponectin have the opposite impact. Obesity not only increases the likelihood of developing insulin resistance and diabetes mellitus, but it also plays a role in causing atherogenic dyslipidaemia. It directly contributes to inflammation and enhances the development of atherosclerosis, regardless of its impact on insulin resistance or lipoproteins.[2]

Vitamin D (25 [OH] D) is produced by the action of ultraviolet (UVB) rays on the skin where it converts 7-dehydrocholesterol to cholecalciferol (vitamin D3) or when ergocalciferol (vitamin D2) and vitamin D3 are consumed orally. The best method for determining a person's vitamin D status is to measure the amount of circulating 25(OH) vitamin D because of its longer half life.[3] There is growing consensus that the ideal circulating

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25(OH) vitamin D level should be approximately 30.0–32.0 ng/ml or more, notwithstanding disagreements on the definition of low vitamin D status.[4] Inadequate levels of vitamin D are also linked to metabolic derangements that negatively impact one's health.

Deficient/insufficient vitamin D status has recently been linked to several disorders, including metabolic syndrome, malignancies, autoimmune diseases, psychiatric disorders and neurodegenerative diseases. However, the exact mechanism of vitamin D deficiency in causing these disorders is still uncertain.[5] Vitamin D deficiency in individuals may result in an increased susceptibility to obesity-related metabolic diseases due to the absence of potential anti-inflammatory effects on chronic low-grade inflammation.

Currently, the most likely explanation for the negative correlation between vitamin D serum levels and body mass index (BMI) is the process of volumetric dilution of vitamin D. Despite obese and lean individuals having comparable levels of vitamin D, overweight individuals have lower blood concentrations due to the higher distribution volume of vitamin D in their bodies. Specifically, 25 (OH) D is mostly distributed in the serum, muscle, fat, and liver compartments, which are found to be enhanced in individuals with obesity.[6] Nevertheless, if the primary factor leading to low serum 25(OH) D levels in obese individuals is volumetric dilution, it implies that losing weight would subsequently elevate the levels of vitamin D in the bloodstream. However, researches on weight loss demonstrated contradictory findings.[7] A group conducted a study to determine the levels of vitamin D in the plasma, omental, and subcutaneous tissue of individuals with obesity and a control group (normal weight).[8] They discovered that the correlation between plasma vitamin D and concentrations of vitamin D in the subcutaneous and omental fat compartments was comparable in both groups. Additionally, they observed a similar distribution pattern of vitamin D between these two fat tissues. These findings suggest that adipose tissue does indeed function as a storage site for vitamin D.

Alternatively, the hypothesis of vitamin D sequestration in adipose tissue has been proposed.[7] It has been reported that while the production of vitamin D in the skin does not vary between obese and normal weight individuals, individuals with obesity experience smaller increases in plasma levels of 25(OH) D after exposure to sunlight and oral intake of vitamin D compared to individuals with normal weight. It was proposed that vitamin D, being a fat-soluble vitamin, is stored and preserved in adipose tissue, resulting in reduced levels of vitamin D in the bloodstream of individuals with a significant amount of adipose tissue. The sequestration concept served as the foundation for the previously mentioned volumetric dilution hypothesis.[7] However, when compared to volumetric dilution, the sequestration of the prohormones ergocalciferol (vitamin D2) and cholecalciferol (vitamin D3) is characterized by their hydrophobic nature and ability to dissolve in adipose tissue. Additionally, sequestration refers to their inability to return to the circulation as a substrate for liver 25-hydroxylase, which converts these prohormones to 25(OH) D after they are stored.[9]

The lower levels of 25(OH) D observed in individuals with obesity can also be attributed to reduced sunlight exposure. This is likely due to their limited mobility and participation in outdoor activities, and different clothing habits compared to individuals with normal weight.[10]

However, the specific mechanisms behind the lower 25(OH) D concentrations in obesity are not fully understood, and there is uncertainty regarding the potential health consequences of these lower concentrations. This present study aims to elucidate the possible protective role of vitamin D against atherogenic lipids in individuals with obesity by assessing the relationship between vitamin D status and dyslipidaemia in this population.

METHODOLOGY

Study design

A cross-sectional study was carried out among 140 individuals aged 18 to 65 years, for a period of 4 months (March to July 2022). A convenient (non-random) sampling technique was used to select the study participants. The participants were recruited from the popular Bodija market, Ibadan, Nigeria and the University College Hospital, Ibadan, Nigeria. The individuals were grouped into 4 based on their BMI as normal weight i.e., controls (18.0 to 24.9 kg/m^2), overweight (25.0 and 29.9 kg/m^2), moderately obese (35.0 and 39.9 kg/m^2) and severely obese (>40 kg/m^2).

Inclusion/Exclusion criteria

Individuals with consent to participate were recruited into the study, while those with chronic illness (such as hypertension, diabetes mellitus, thyroid disorders) or on medication for chronic illness were excluded from the study because they have metabolic disorders that can affect the outcome of the study. Thyroid-stimulating hormone screening was done in all participants to rule out thyroid disorders while other chronic illnesses were excluded through the questionnaire.

Sample size

The minimum sample size for the study was calculated with the formula proposed by Charan and Biswas[11] which is as follows: - $Z_{1\ a/2}{}^2$ p (1-p)/d^2 where; $Z_{1\ a/2}$ = is the standard normal variate [at 5% type 1 error (p<0.005) it is 1.96]; p = expected proportion in population based on previous studies or pilot studies (estimated as 8.1% from study by Chukwuonye et al., 2013);[12] d = absolute error or precision (this is chosen to be 5%). The minimum sample size calculated was 114.

Data and specimen collection

Following ethical approval from the University of Ibadan/ University College Hospital (UI/UCH) Joint Ethics Committee (UIUCH/EC/21/0726) and after obtaining informed consent from participants, a total of 10 ml of venous blood was withdrawn from each participant. The blood was withdrawn after an overnight fast through venipuncture using pyrogen-free disposable needles and syringes. Five milliliters of whole blood was dispensed into fluoride oxalate bottle and the obtained plasma after centrifugation was used for fasting plasma glucose measurement. The remaining 5 ml was dispensed into serum separator tubes (SST) and 30 minutes was allowed for clot retraction; and the obtained serum after centrifugation was used for vitamin D, TSH, fasting lipid profile assay. Each specimen bottle was assigned an identity number that will match the ID number on each data collection form used for participant recruitment. This separation process was achieved by spinning the specimen bottles in a centrifuge made by UNISCOPE at a speed of 4,000 revolutions per minute for duration of 10 minutes. The resultant plasma and serum were dispensed into their respective well-labeled plain bottle and stored at a temperature of -20^{0}C until analysis was performed within 3 months of collection.

Assay methods

Fasting plasma glucose was determined using glucose oxidase method as described by Trinder[13] on an automated chemistry analyzer, LandWind C-100 plus. The serum vitamin D concentration was quantitatively measured using an enzyme-linked immunosorbent assay (ELISA) as described by Holick MF[15] on StatFax 400 ELISA reader. Vitamin D status is classified based on the ELISA kit manufacturers' manual (Calbiotech, Inc) as follows: levels <20 ng/ml, 21 ng/ml-29 ng/ml, 30 ng/ml-150 ng/ml, >150 ng/ml were considered as deficiency, insufficiency, sufficiency and intoxication respectively. Thyroid-stimulating hormone was assayed by ELISA method as described by Frank et al.[16] The lipid profile (TC, TG and HDL-c) was done using the enzymatic method as described by Fredrickson et al.,[17] Hainline et al.,[18] and Albers et al.,[19] respectively on LandWind C-100 plus chemistry auto-analyzer. Low-density lipoprotein cholesterol was calculated using the using the formula described by Friedwald et al.[20] The equation is expressed as:

LDL-c (mg/dl) = TC−VLDL-c− HDL-c; where VLDL-c= TG/5

Atherogenic index in plasma (AIP) was determined by a method proposed by Dabiasova and Frohlic in 2001;[21] AIP= Log (TG/HDLc), value is expressed in mmol/L.

Statistical analysis

The data analysis was conducted using the statistical package software for social scientists (SPSS) version 25.0. Kolmogrov-Smirnov test of normality was used to test the distribution of obtained data and non-Gaussian parameters were transformed. The data were presented as the mean value plus or minus the standard deviation (mean ± SD). The method of analysis of variance (ANOVA) was employed to assess and compare the differences in means among all groups, while the Duncan multiple range test was used to determine the differences within the BMI groups. The Pearson correlation coefficient was employed to ascertain the link between all parameters within each group. The level of significance was taken to be p <0.05.

RESULTS

At the conclusion of the study, there was a total of 134 participants remaining, with 104 females and 30 males. Six (6) participants were excluded due to findings of thyroid disorders obtained from TSH screening and the remaining numbers for each group were described as follows: control (34), over-weight (33), moderately obese (32), and severely obese (34).

Figure 1 represents the mean vitamin D levels in all the participants and shows that the vitamin D concentration

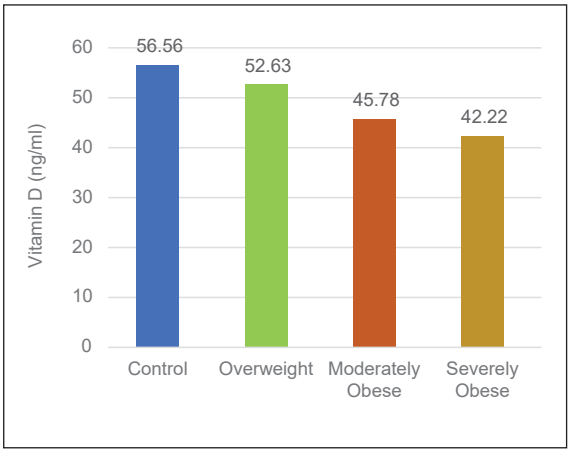

Figure 1. Mean serum vitamin D levels in the control, overweight, moderately obese and severely obese groups.

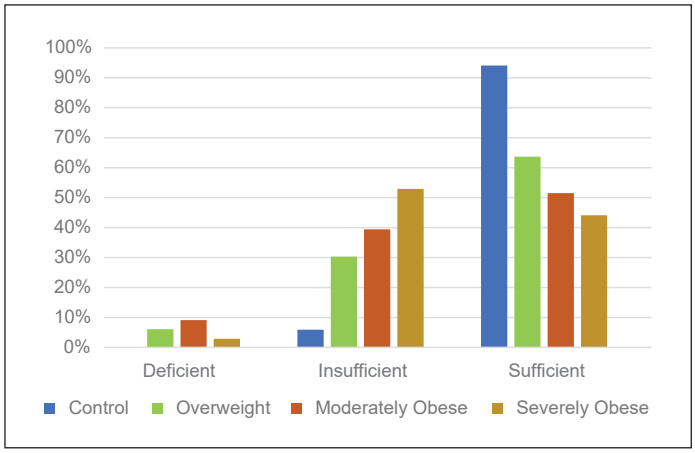

Figure 2. Classification of vitamin D status in the control, overweight, moderately obese and severely obese groups.

Table 1. Serum lipid profile levels in control, overweight, moderately obese and severely obese groups

Serum lipids	Control (n = 34)	Overweight (n = 33)	Moderately obese (n = 32)	Severely obese (n = 34)	*p*-value
T. Chol (mg/dl)	165.50 ± 24.15[a]	191.80 ± 15.93[b]	221.41 ± 15.17[c]	241.53 ± 41.96[d]	<0.001*
TG (mg/dl)	106.26 ± 27.21[a]	129.06 ± 33.00[b]	158.66 ± 17.35[c]	189.32 ± 45.78[d]	<0.001*
HDL (mg/dl)	67.35 ± 7.10[a]	59.94 ± 11.14[b]	39.09 ± 8.73[c]	29.62 ± 13.37[d]	<0.001*
LDL (mg/dl)	46.60 ± 22.65[a]	67.33 ± 22.83[b]	102.98 ± 18.45[c]	118.72 ± 31.49[d]	<0.001*

*p-value significance at $p \leq 0.05$

The Duncan multiple range test was used to determine the differences within the groups, with corresponding the compact letter display, a, b, c and d.

Values of the same superscript within the same column are not statistically different at $p > 0.05$ while values with different superscript are significantly different at $p < 0.05$. For all the lipid profile, a ≠ b ≠ c ≠ d.

despite being sufficient in all the groups (≥30.0 ng/ml), declines significantly as BMI increases (p = 0.000). The serum vitamin D concentration is 56.56 ± 14.25 ng/ml, 52.63 ± 22.02 ng/ml, 45.78 ± 30.59 ng/ml and 42.22 ± 31.59 ng/ml in the controls, overweight, moderately obese and severely obese group respectively.

Figure 2 represents the classification of all the participants according to their vitamin D status (deficient/insufficient/sufficient), and it shows that 0%, 5.9 and 94.1% of the normal weight participants (controls) had vitamin D deficiency, insufficiency and sufficiency respectively; while 6.1%, 30.3% and 63.7% of the overweight participants had vitamin D deficiency, insufficiency and sufficiency respectively. 9.1%, 39.4% and 51.5% of the moderately obese group had vitamin D deficiency, insufficiency and sufficiency respectively; while 2.9%, 52.9% and 44.1% of the severely obese group had vitamin D deficiency, insufficiency and sufficiency respectively. Thus, the percentage of participants with vitamin D sufficiency decreases as BMI increases.

Table 1 shows the serum fasting lipid profile in the control, overweight, moderately obese and severely obese groups. It was observed that there were significant differences between the mean ± SD in the four BMI groups; where the serum concentrations of TC, triglyceride and LDL-c of normal weight (i.e., controls) is significantly lower than that of overweight (p <0.001), that of overweight is significantly lower than that of moderately obese group (p <0.001), that of moderately obese is significantly lower than that of severely obese group (p <0.001). Meanwhile the serum concentration of HDL-c of the control is significantly higher than that of the overweight group (p <0.001), that of the overweight is significantly higher than that of moderately obese group (p <0.001) and that of moderately obese is significantly higher than that of severely obese group (p <0.001). Therefore, the serum TC, triglycerides and LDL-c significantly increase across the four BMI groups with the severely obese group having the highest concentrations; while the opposite is observed for the serum HDL-c with the severely obese group having the lowest concentration.

The correlation between BMI and lipid profile in the severely obese group is shown in Table 2. There is a positive significant correlation (p = 0.001) between BMI and TC (r = 0.685), BMI and TG (r = 0.656), BMI and LDL-c (r = 0.645), and a significant negative correlation (p = 0.001) between BMI and HDL-c (r = -0.654) in the severely obese group.

Table 2. Correlation between BMI and Lipid Profile in severely obese group, n=34

Parameters	r	*P* Value
BMI vs T. Chol	0.685**	0.001**
BMI vs TG	0.656**	0.001**
BMI vs HDL-c	-0.654**	0.001**
BMI vs LDL-c	0.645**	0.001**

** Correlation is significant at the p-value ≤0.01 level (2-tailed).

Table 3. Correlation between vitamin D and lipid profile in control, overweight, moderately obese and severely obese groups

Parameters	Control (n = 34)	Overweight (n = 33)	Moderately obese (n = 32)	Severely obese (n = 34)
Vit. D vs T Chol	-0.113	-0.040	0.060	-0.189
Vit. D vs TG	-0.261	-0.176	-0.044	-0.145
Vit. D vs HDL-c	-0.092	-0.051	-0.215	0.160
Vit. D vs LDL-c	0.107	0.125	0.171	-0.179

** Correlation is significant at the p-value ≤0.01 level (2-tailed).

Table 4. Atherogenic index in plasma (AIP) level in all participants

BMI	N	Mean	SD
Normal	34	-0.19	0.22
Overweight	33	-0.05	0.25
Moderately obese	32	0.25	0.11
Severely obese	34	0.37	0.20

Correlation between serum vitamin D and lipid profile in the control, overweight, moderately obese and severely obese groups is shown in Table 3. There is no significant correlation between vitamin D and lipid profile parameters (TC, TG, LDL-c and HDL-c) in all the 4 groups studied.

Table 4 shows the level of atherogenic index in plasma (AIP) in all the participants, and there is increasing value of AIP from the normal weight participants (-0.19 ± 0.22), to the overweight group (-0.05 ± 0.25), to the moderately obese group (0.25 ± 0.11) and severely obese participants (0.36 ± 0.20). Thus, the AIP increases as BMI increases.

DISCUSSION

The aetiology of obesity is multifactorial. It is a subject of controversy whether vitamin D deficiency is a consequence of obesity or a factor predisposing to obesity.[22] In recent

years, the correlation between vitamin D and obesity has attracted considerable attention in the world, in which individuals with obesity might present with inadequate vitamin D status. Dyslipidaemia is a major feature of obesity and a major risk factor for atherogenesis and insulin resistance; thus, this present study explored the relationship between serum vitamin D levels and dyslipidaemia in obesity states, with the intent of determining the possible role of vitamin D in the management of lipid disorders in individuals with obesity.

In our study, though the mean serum vitamin D concentration in all the participants is sufficient (≥30 ng/ml), approximately half of the moderately obese and severely obese individuals have inadequate vitamin D status; this observation could be due to regular exposure to sunlight in the participants from this region, South-Western Nigeria. Most of these participants are sedentary traders who are exposed to sunlight for almost eight (8) hours a day. Sunlight exposure on the skin triggers the synthesis of vitamin D, which is crucial for maintaining optimal levels in the body and ameliorates vitamin D deficiency.

The inadequate vitamin D level found in half of the moderately and severely obese individuals might be as a result of the volumetric dilution principle in obesity.[7] The more obese an individual is, the more likely vitamin D becomes insufficient or deficient because vitamin D is trapped in the adipose tissue. It was reported by Gonzalez et al., that the lower levels of 25(OH) vitamin D were associated with higher BMI,[23] suggesting that vitamin D might play an important regulatory role in the development of obesity. This present study found that vitamin D levels decrease significantly as BMI increases; this is consistent with the finding of Orces in 2019, which indicated that low vitamin D status is likely to contribute to the development of overweight/obesity.[24]

Decreased vitamin D levels might not only lead to obesity. It has been proposed that as the weight of an individual increases, the likelihood of dyslipidaemia becomes higher. As seen in this present study, the TC, TG and LDL-c are significantly elevated in individuals with obesity compared with normal weight individuals; while the "good cholesterol" HDL-c is significantly lower in the obese group compared to normal weight participants. This trend occurs because obesity is considered to be potentially linked with abnormal lipid levels which can contribute to dyslipidaemia and an increase in cardiovascular risk outcomes. Thus, the observation from our study validates the findings of Xiongjing et al., and the prospective cohort studies of Wang et al., where they reported association between low vitamin D levels and dyslipidaemia.[25,26]

A positive correlation was observed in this study between BMI and the lipid profile especially in the severely obese individuals; this observation concurs with the findings of Ofori and Angmorterh, where a positive correlation between BMI and lipid profile was also reported.[2] Thus, as BMI increases, there is a tendency for lipid parameters to exhibit less favorable values. The mechanism underlying the positive correlation between BMI and lipid profile is considered multifactorial and complex. Such factors include increased adipose tissue mass, insulin resistance and altered lipoprotein metabolism.

In this present study, there was no significant correlation found between the levels of vitamin D and lipid profile in the different categories of all participants studied. This can be explained by different factors such as dietary habits, sunlight exposure, physical activity levels, and individual metabolic variations. These factors interact in intricate ways, providing the body with a substantial amount of vitamin D which mitigates the elevation of lipids.

It was found in this study that as an individual's BMI increases, AIP, a marker of CVD risk also increases, with higher values in the moderately and severely obese groups. The high prevalence of insulin resistance, adipose tissue inflammation, changes in lipoprotein composition and dyslipidaemia in obesity could explain this finding. AIP is calculated based on the ratio of triglyceride to HDL cholesterol,[19] thus an increase in triglyceride and decrease in HDL cholesterol could contribute to an elevated AIP. Nevertheless, findings by Fernandez et al., and Niroumand et al., revealed why a better biomarker, such as AIP, is needed to predict cardiovascular disease (CVD) compared with conventional biomarkers.[26,27] For instance, abnormally increased LDL-c levels indicate higher risk of coronary artery disease (CAD) events. However, many CVD patients do not exhibit elevated LDL-c levels. A similar finding was observed in this present study that as the mean LDL-c concentrations in the moderately and severely obese participants were at low risk rather than high risk category level. Therefore, it is not sufficient to use merely conventional lipid profiles to predict CVD. Rather, a calculated AIP based on the ratio of triglyceride to HDL is better used to predict cardiovascular disease.

In general, this present study highlights that, participants who maintained a healthy diet, engaged in outdoor activities, who are often exposed to sunlight and engage in physical exercise, who are not overweight or obese, would have sufficient 25(OH) vitamin D level with lipid profile within optimal limits and will be at low risk for cardiovascular diseases; this is shown by the AIP result of this study. However, some of those who are overweight and obese, who are also engaged in outdoor activities, who occasionally are exposed to sunlight and maintained a healthy diet from time to time, would have inadequate 25 (OH) vitamin D levels as well as some degree of derangement in lipid profile. These groups can be referred to as healthy obese as the exposure to sunlight ameliorates dyslipidaemia and increases vitamin D level. Also, individuals with sedentary lifestyle, indoor occupational activities, who do not engage in physical activities, who wore covered clothes when they went outside and who have genetic predisposition, would have deficient vitamin

D concentration, abnormal lipid levels and will be at higher risk for cardiovascular diseases. This is also in agreement with the work of Lagunova et al., and Nejabat et al., expressing the possible association between low vitamin D status, BMI and cardiovascular diseases.[28,29]

CONCLUSION

The study provided possible evidence to support the association between dyslipidaemia and inadequate vitamin D status in this cohort of adults with obesity in Ibadan, Nigeria. It also supports the hypothesis that individuals with obesity who often expose themselves to sunlight are rarely vitamin D deficient but rather have inadequate vitamin D status. As individuals with obesity expose themselves to sunlight, the exposure prevents them from being at risk of vitamin D deficiency regardless of their vitamin D status. Exposure to sunlight mitigates the effect of sedentary lifestyle and dyslipidaemia by raising the serum vitamin D (25[OH] D) levels.

Recommendations

More attention needs to be focused on the approaches to prevention of dyslipidaemia in obesity, through proper dietary modifications, exercise and deliberate exposure to sunlight. Vitamin D nutritional fortifiers should be supplemented properly when indicated to prevent dyslipidaemia and other cardiovascular abnormalities caused by vitamin D deficiency.

Anthropometric measurement is important in the evaluation of patients with obesity. It is a cheap, non-invasive objective monitoring index for patients with this condition; thus it should be maintained as a standard practice.

Study limitation

This study did not investigate the advantages of vitamin D supplementation in raising the vitamin D level in the body and its effect on reducing the body weight as well as metabolic abnormalities. It was observed that the study population consists mostly of females, which may limit generalization; underscoring the need to recruit more males into the study to be conclusive. A larger sample size and a longer study duration to examine an interventional arm of participants with insufficient vitamin D levels, and the effect of weight management strategies in improving metabolic health outcomes in individuals with obesity would be optimal.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement
OOS: Conceptualization, Methodology, Validation, Investigation, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization; **ZOS:** Conceptualization, Methodology, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Project administration, Funding acquisition; **AAS:** Software, Validation, Formal analysis, Investigation, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization; **KSA:** Conceptualization, Methodology, Validation, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Supervision, Project administration, Funding acquisition; **CFA:** Methodology, Investigation, Resources, Project administration.

Data Availability Statement
Datasets generated and analyzed are included in the published article.

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References
1. Lin X, Li H. Obesity: Epidemiology, pathophysiology, and therapeutics. Front Endocrinol (Lausanne). 2021;12:706978. PMID: 34552557 PMCID: PMC8450866 DOI: 10.3389/fendo.2021.706978
2. Jin X, Qiu T, Li L, et al. Pathophysiology of obesity and its associated diseases. Acta Pharm Sin B. 2023;13(6):2403-24. PMID: 37425065 PMCID: PMC10326265 DOI: 10.1016/j.apsb.2023.01.012
3. Holick MF. Vitamin D status: Measurement, interpretation, and clinical application. Ann Epidemiol. 2009;19(2);73-8. PMID: 18329892 PMCID: PMC2665033 DOI: 10.1016/j.annepidem.2007.12.001
4. Binkley N, Ramamurthy R, Krueger D. Low vitamin D status: Definition, prevalence, consequences, and correction. Endocrinol Metab Clin North Am. 2010;39(2):287-301. PMID: 20511052 PMCID: PMC4315502 DOI: 10.1016/j.ecl.2010.02.008
5. Kaur J, Khare S, Sizar O, Givler A. Vitamin D deficiency. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024. https://www.ncbi.nlm.nih.gov/books/NBK532266/
6. Walsh JS, Bowles S, Evans AL. Vitamin D in obesity. Curr Opin Endocrinol Diabetes Obes. 2017;24(6):389-94. PMID: 28915134 DOI: 10.1097/MED.0000000000000371
7. Vranić L, Mikolašević I, Milić S. Vitamin D deficiency: Consequence or cause of obesity? Medicina (Kaunas). 2019;55(9):541. PMID: 31466220 PMCID: PMC6780345 DOI: 10.3390/medicina55090541
8. Carrelli A, Bucovsky M, Horst R, et al. Vitamin D storage in adipose tissue of obese and normal weight women. J Bone Miner Res. 2017; 32(2):237-42. PMID: 27542960 PMCID: PMC5577589 DOI: 10.1002/jbmr.2979
9. Gangloff A, Bergeron J, Lemieux I, Després JP. Changes in circulating vitamin D levels with loss of adipose tissue. Curr Opin Clin Nutr Metab Care. 2016;19(6):464-70. PMID: 27537278 DOI: 10.1097/MCO.0000000000000315
10. Lukaszuk JM, Luebbers PE. 25(OH)D status: Effect of D3 supplement. Obes Sci Pract. 2017;3(1):99-105. PMID: 28392936 PMCID: PMC5358080 DOI: 10.1002/osp4.85
11. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? Indian J Psychol Med. 2013;35(2): 121-6. PMID: 24049221 PMCID: PMC3775042 DOI: 10.4103/0253-7176.116232
12. Chukwuonye II, Chuku A, John C, et al. Prevalence of overweight and obesity in adult Nigerians: A systematic review. Diabetes Metab Syndr Obes. 2013;6:43-7. PMID: 24049221 PMCID: PMC3775042 DOI: 10.4103/0253-7176.116232
13. Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Ann Clin Biochem. 1969;6(1):24. DOI: 10.1177/000456326900600108
14. Frank JE, Faix JE, Hermos RJ, et al. Thyroid function in very low birth weight infants: Effects on neonatal hypothyroidism screening. J Pediatr. 1996;128(4):548-54. PMID: 8618191 DOI: 10.1016/s0022-3476(96)70368-2
15. Fredrickson DS, Lees RS. In: Stanbury JB, Wyngaarden JB, Fredrickson DS, eds. The metabolic basis of inherited disease. 2nd ed. New York, NY: McGraw-Hill; 1967.
16. Hainline A Jr, Cooper GR, Olansky AS, et al. CDC survey of high-density lipoprotein cholesterol measurements: A report. Atlanta, GA: Centers for Disease Control; 1980.
17. Albers JJ, Tollefson JH, Chen CH, Steinmetz A. Isolation and characterization of human plasma lipid transfer proteins. Arteriosclerosis. 1978;4(1):49-58. PMID: 6691846 DOI: 10.1161/01.atv.4.1.49

18. Friedewald WT, Levy RT, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6): 499-502. PMID: 4337382
19. Dobiášová M, Frohlich J. The plasma parameter log(TG/HDL-C) as an atherogenic index: Correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FERHDL). Clin Biochem. 2001;34(7):583-8. PMID: 11738396 DOI: 10.1016/s0009-9120(01)00263-6
20. Yao Y, Zhu L, He L, et al. A meta-analysis of the relationship between vitamin D deficiency and obesity. Int J Clin Exp Med. 2015;8(9): 14977-84. PMID: 26628980 PMCID: PMC4658869
21. González L, Ramos-Trautmann G, Díaz-Luquis GM, Pérez CM, Palacios C. Vitamin D status is inversely associated with obesity in a clinic-based sample in Puerto Rico. Nutr Res. 2015;35(4):287-93. PMID: 25708459 PMCID: PMC4376559 DOI: 10.1016/j.nutres.2015.02.001
22. Orces C. The association between body mass index and vitamin D supplement use among adults in the United States. Cureus. 2019;11(9):e5721. PMID: 31720189 PMCID: PMC6823089 DOI: 10.7759/cureus.5721
23. Jiang X, Peng M, Chen S, Wu S, Zhang W. Vitamin D deficiency is associated with dyslipidemia: A cross-sectional study in 3788 subjects. Curr Med Res Opin. 2019;35(6):1059-63. PMID: 30479168 DOI: 10.1080/03007995.2018.1552849
24. Wang Y, Si S, Liu J, et al. The associations of serum lipids with vitamin D status. PLoS One. 2016;11(10):e0165157. PMID: 27768777 PMCID: PMC5074586 DOI: 10.1371/journal.pone.0165157
25. Ofori EK, Angmorterh SK. Relationship between physical activity, body mass index (BMI) and lipid profile of students in Ghana. Pan Afr Med J. 2019;33:30. PMID: 31384345 PMCID: PMC6658156 DOI: 10.11604/pamj.2019.33.30.17889
26. Fernandez-Macías JC, Ochoa-Martínez AC, Varela-Silva JA, Perez-Maldonado IN. Atherogenic index of plasma: Novel predictive biomarker of cardiovascular illness. Arch Med Res. 2019;50(5): 285-94. PMID: 31593853 DOI: 10.1016/j.arcmed.2019.08.009
27. Niroumand S, Khajedaluee M, Khadem-Rezaiyan M, et al. Atherogenic index of plasma (AIP): A marker of cardiovascular disease. Med J Islam Repub Iran. 2015;29:240. PMID: 26793631 PMCID: PMC4715400
28. Lagunova Z, Porojnicu A, Lindberg F, Hexeberg S, Moan J. The dependency of vitamin D status on body mass index, gender, age and season. Anticancer Res. 2009;29(9):3713-20. PMID: 19667169
29. Nejabat A, Emamat H, Afrashteh S, et al. Association of serum 25-hydroxy vitamin D status with cardiometabolic risk factors and total and regional obesity in southern Iran: Evidence from the PoCOsteo study. Sci Rep. 2024;14(1):17983. PMID: 39097599 PMCID: PMC11297962 DOI: 10.1038/s41598-024-68773-1