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FULL PAPER

1, 25-Dihydroxyvitamin D3 level and lipids profile in some obese adults in Samarra city, Iraq

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Obesity is associated with community and medicinal risks that individually make it a problem. The current study aimed to evaluate vitamin D and lipids profile in some obese adults in Samarra City, Iraq. The study design included two groups; the first group was obese people and the second one was non-obese people as a control group. Each group consisted of fifty persons, and the ages of the two groups ranged between (20-40) years. Blood samples were collected for the period from 2/1/2021 to 3/2/2021. The results displayed a significant increase in BMI in obese group compared with non-obese group, a significant increase in the levels of total cholesterol, triglycerides, low density lipoproteins-cholesterol and very low density lipoproteins-cholesterol in obese group compared with non-obese group, while there was a non-significant difference in the levels of vitamin D_3 and high-density lipoprotein-cholesterol between two the groups.

KEYWORDS

Vitamin D_3; lipids profile; obesity; body mass index.

Introduction

Overweightness is described as state in which there is an unusual or extra fat collection in the body that can influence the health of a human, and is one of the greatest health problems worldwide [1,2]. Obesity is a health problem that increases the possible risk of developing other diseases and health complications, for example hypertension, heart disease, cardiovascular, diabetes, insulin resistance, lipids metabolism variations, metabolic syndrome and cancers [3-5]. There are many factors that lead to obesity such as genetic factors, metabolic syndrome, lifestyle choices and age [6-8]. Diet regime, physical activity, and life style changes can help weight loss [9].

Body mass index (BMI) is an easy weight to height relation that is commonly used to classify overweight and obesity in adults. It is explained as a body's weight in kilograms divided *via* the square of his height in meters (kg/m^2) [4].

Vitamin D is a fat-soluble vitamin. It is a secosteroid with an endocrine mechanism of action, which is sequentially synthesized in humans in the skin, liver and kidneys [10]. Vitamin D has a vital role in glucose homeostasis regulation, mechanisms of insulin secretion, and inflammation related with obesity [11]. In addition, vitamin D has an important function in conserving fine health, and its values or significance hail from its function in absorbing of phosphorus and calcium ions from the gastrointestinal track, which is why it can be utilized for treating and inhibiting bone and muscle pains, chronic tiredness, teeth and dental problem, and osteoporosis, as well as other cell functions [12,13]. In obesity, vitamin D influences secretion of insulin, tissue sensitivity to

insulin, and systemic infection. The direct and paracrine properties of vitamin D cause activation of vitamin D receptor in pancreatic beta cells [14,15].

Vitamin D deficiency may lead to diabetes mellitus, hypertension, osteoporosis, chronic fatigue syndrome, obesity, Alzheimer's disease, depression, fibromyalgia, heart disease, stroke, cancer, periodontitis, and autoimmune diseases. Considering that vitamin D acts a function in managing the immune system, there is a probability of lessening one's danger of autoimmune disease and cancers [16,17]. Abnormalities in lipids metabolism are very commonly observed in obese patients, so obesity is related with raised blood lipids and lipoproteins [4].

This study aimed to assess the relationship between $DHVD_3$ and lipids profile levels with obesity in some adults in Samarra city.

Materials and methods

Sampling

This study was conducted on eighty people including fifty obese people and thirty healthy people as control groups (not suffering from any disease). Serum was collected from obese and healthy people attended to outpatient clinics in Samarra for the period from firs of January2021 to tenth of March 2021. The ages of them ranged between (twenty to forty) years.

Blood collection

After ten hours of fasting, the blood was taken and left at 25 °C for coagulation for a period of 15 min. The serum was expelled at 5000 x g and the separated sera were frozen at -20 °C for future biochemical analysis [18].

Analysis of biochemical parameters

Vitamin D was determined by ichroma technique; a fluorescence immunoassay (FIA) for the quantitative determination, lipids profile (Total Cholesterol (TC), Triglycerides (TG), high density lipoprotein- cholesterol (HDL-C)) were assessed by used kits supplied from Biolabo Co, France.

Very Low density lipoprotein- cholesterol (VLDL-C) was determined by using the following formula: VLDL-C=TG/5. Low density lipoprotein- cholesterol (LDL-C) was determined by the following equation:

LDL-C= TC-(HDL-C+VLDL-C).

Statistical analysis

The results were represented as mean ± SD for all values. The data were analyzed for critical contrast utilizing t-test ($P \leq 0.05$) by Minitab program to compare the chemical variables between two groups [19].

Results and discussion

The results of BMI and age in obese and non-obese are shown in Table 1; mean ± SD for the BMI of obese group was (36.419 ± 2.98) compared with the non-obese group (٢٣.474±١.01). The results showed that BMI level a momentous rise, $P \leq 0.05$ in the obese group compared with non-obese group, as shown in Figure 1. Results showed no significant difference in age between two groups.

TABLE 1 BMI, age, lipid profile and D3 levels in the obese and non-obese groups

Parameters	Mean ± SD		P-Value
	Non-obese group	Obese group	
BMI	٢٣.474±١.01	36.419±2.98	0.001*
Age	32.20±8.12	32.90±7.16	0.531
Vitamin D_3	19.374±8.15	15.738±4.49	0.١١٤
Cholesterol	158.35±26.36	192.02±40.44	0.001*
Triglycerides	95.04±43.65	158.43±74.71	0.001*
HDL-C	39.351±21.85	34.806±17.56	0.466
LDL-C	99.994±30.00	125.523±42.46	0.001*
VLDL-C	19.007±8.73	31.686±14.94	0.001*

*This sign means different significant at P ≤0.05.

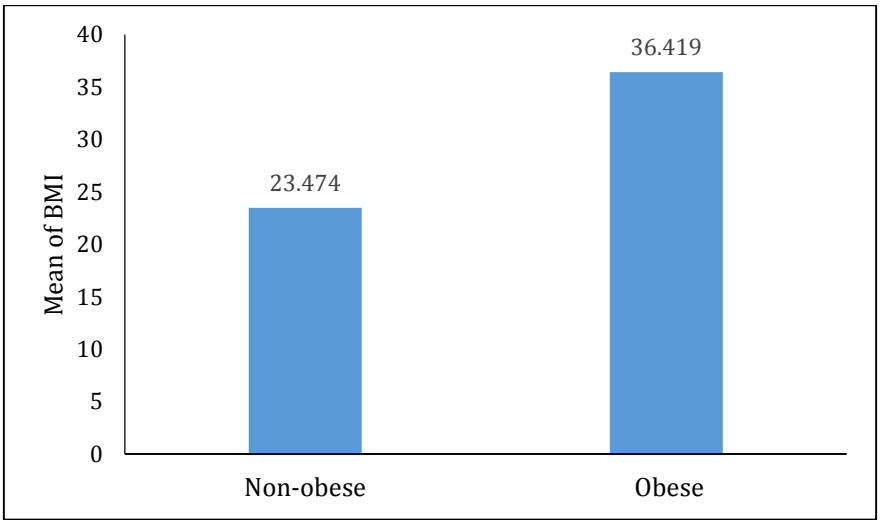

FIGURE 1 Mean of BMI between two groups

The results in Table 1 shows non-significant decrease in the concentration of vitamin D_3 for obese group; the mean ± SD for obese group was (15.738±4.49) compared with non-obese group (19.374±8.15), as shown in Figure 2.

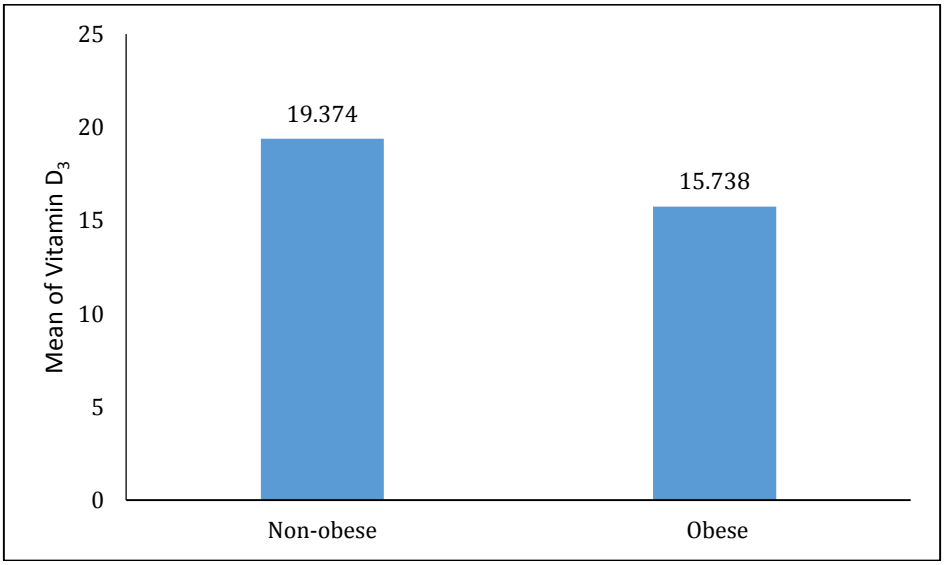

FIGURE 2 Mean of vitamin D_3 between two groups

The results showed a significant increase (P ≤0.05) in the concentration of total cholesterol (TC), triglycerides (TGs), low density lipoproteins-cholesterol (LDL-C) and very low density lipoproteins-cholesterol (VLDL-C) in obese group compared with non-obese group, while there was no significant difference in the concentration of high density lipoproteins-cholesterol (HDL-C) between two groups, as shown in Table 1.

The mean ± SD of TC, TGs, LDL-C and VLDL-C in obese group were (192.02±40.44), (158.43±74.71), (125.52±42.46) and (31.68±14.94), respectively, compared with the non-obese group (158.35±26.36), (95.04±43.65), (99.99±30.00) and (19.01±8.73) respectively, as shown in Figures 3, 4, 5, and 6. The mean ± SD of HDL-C was (34.81±17.56) in obese group, while it was (39.35±21.85) in non-obese group, as shown in Figures 7. These results are in agreement with those of Shahid and Sarwar, reporting the increase of the concentration of TC, TGs and LDL-C [20]. Similar results were found in a study carried out by Silitonga *et al.* [21].

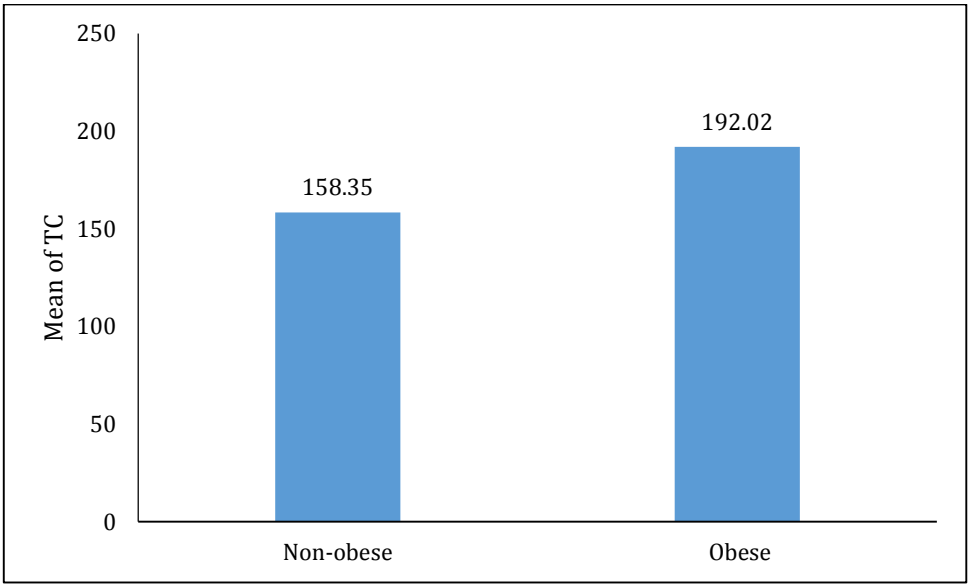

FIGURE 3 Mean of TC between two groups

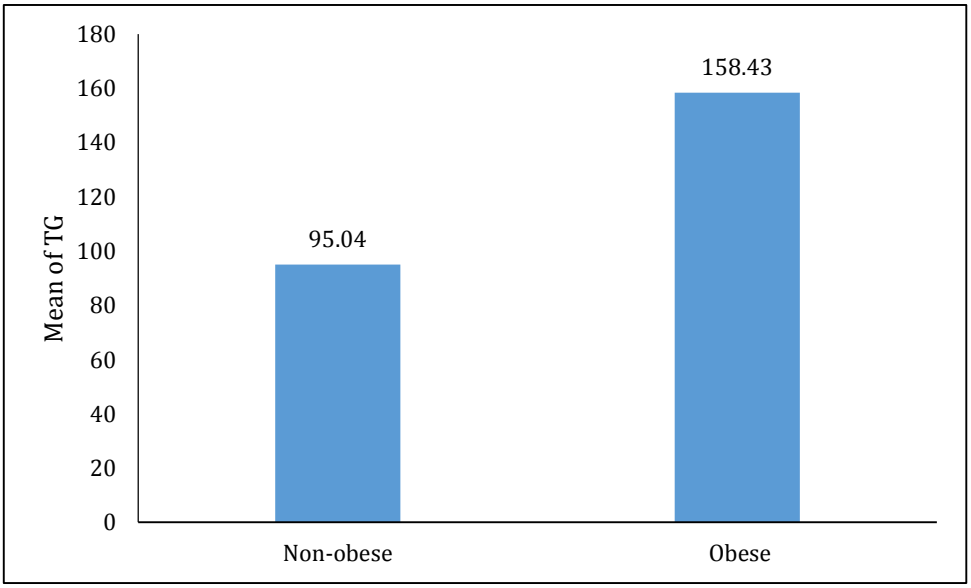

FIGURE 4 Mean of TGs between two groups

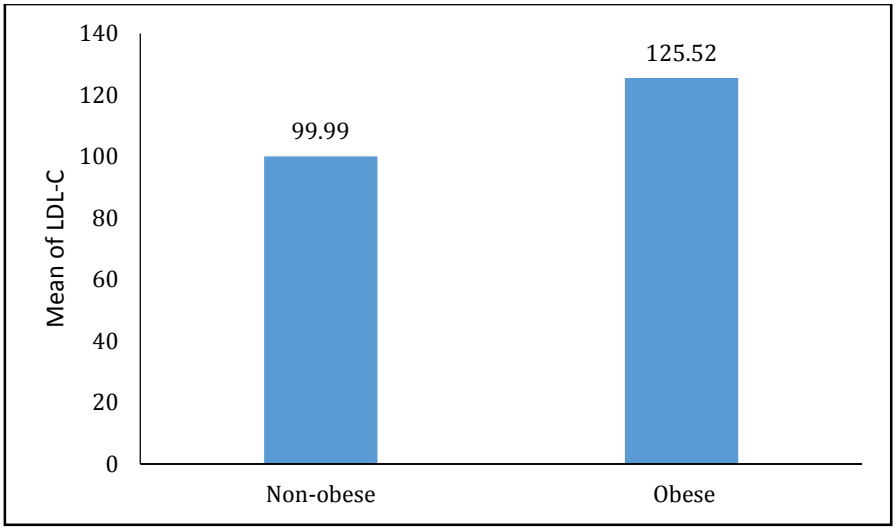

FIGURE 5 Mean of LDL-C between two groups

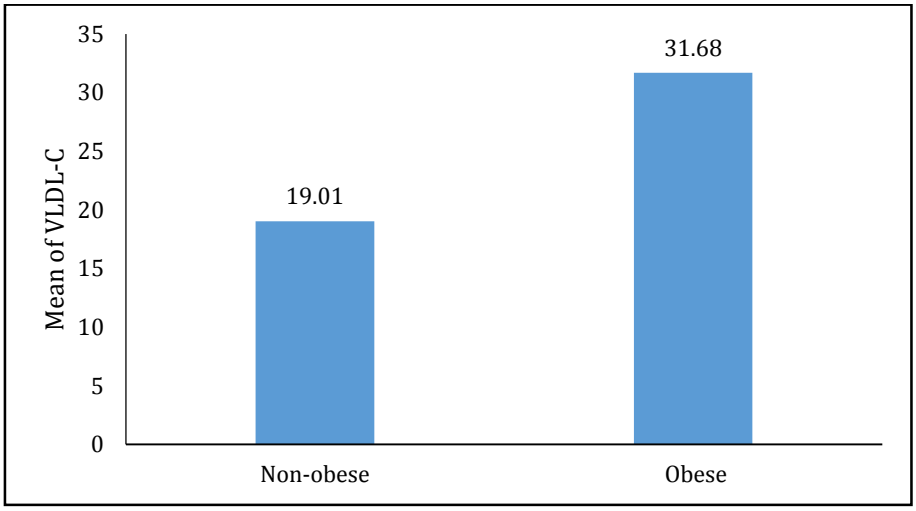

FIGURE 6 Mean of VLDL-C between two groups

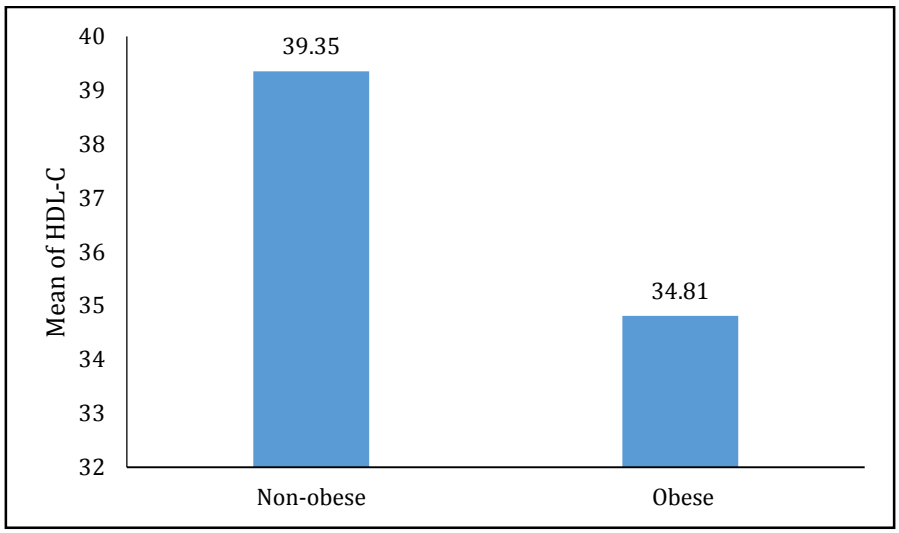

FIGURE 7 Mean of HDL-C between two groups

Several factors lead to obesity, but despite the genetic role, it has been detected to happen chiefly due to unhealthy lifestyle behaviors [1].

Obesity-associated vitamin D_3 insufficiency is likely due to the decreased bioavailability of vitamin D_3 from cutaneous and dietary sources because of its deposition in body fat compartments. These results are consistent with those of Joseph and Karani (2013), finding that obesity can lead to vitamin D_3 deficiency. They were the first to link a high body mass index with low levels of vitamin D_3, as the study indicated the increasing rates of obesity and vitamin D_3 deficiency in the world, which may lead to many serious health problems [22]. Obesity contributes to vitamin D_3 deficiency by reducing the body's ability to convert this vitamin into its active hormonal form, as fat cells isolate vitamin D_3 in a way that they stop its secretion in the body, so those who suffer from obesity must take higher doses of vitamin D_3 supplements to maintain its levels in addition to increasing the intake of sources of this vitamin from the diet along with increased exposure to sunlight [23]. The cause for low vitamin D_3 is staying indoors for a long time, especially in the summer months, and thus low exposure to sunlight, and low consumption of foods rich in vitamin D3 such as cereals contribute to this problem [24].

The reason for high cholesterol is due to the nutritional pattern, which is one of the factors that causes a high concentration of lipids in the blood plasma and thus leads to a high level of cholesterol, in addition to the body's ability to quickly produce cholesterol and quickly get rid of it. The results of this study are in agreement with those of Rexrode, reporting that fat cells located in the abdominal area play the largest role in causing the imbalance in the values of the lipid profile in obese people compared with peripheral fats [25], and central fats play a greater role in causing insulin resistance and more amenable to recycling fatty acids during lipolysis [26]. Other studies have also confirmed that high cholesterol levels are affected by the nutrient type and genetic predisposition, such as genetic cholesterol and obesity [27]. Taskinen *et al.* concluded that the increase the fat mass in the waist circumference plays an important role in the high level of total cholesterol as a result of the defect in metabolism [28]. The disturbances in the physiological activities of lipid metabolism in obese individuals were reflected in the high lipid levels, including total cholesterol [29].

In this study, the increase of TGs may be due to eating foods rich in fat, which increases the production of chylomicron in the intestine, and when it is decomposed, fatty acids are released, leading to an increase in their quantities in the liver, which causes an increase in the release of TGs, especially if it is associated with the absence of insulin. Bays *et al.*[30] concluded that the impairment of fat cells in the body as a result of obesity leads to changes in the percentage of fat and loses the ability to store fat, causing the body to increase the breakdown of TGs and a rise in the level of fatty acids in the blood, which negatively affects the liver tissue.

The lack or absence of insulin leads to the decomposition of lipids stored in adipose tissue, which results in an increase fatty acids levels, and leads to the conversion to VLDL-C, phosphorylated LDL-C and cholesterol. They move with the TGs formed in the liver to enter the blood and thus results in an increase the lipids levels in the blood [31].

Abbasi *et al.* showed that TGs concentration has a strong relationship with insulin resistance, as the increase in TG concentration tends to decrease HDL-C levels. Moreover, the insulin resistance is independently associated with high TG and low HDL-C [32].Chatrath et al. displayed that eating a high-fat or high-calorie diet causes the body to increase the breakdown of TGs and the high level of fatty acids in the blood,

which negatively affects the liver tissue, causing a state of non-alcoholic fatty liver [33]. The relationship between fatty liver and a high lipids levels in the blood with insulin resistance has been found [34]. The high level of lipids in the blood affects the process of lipid metabolism and storage and leads to diseases related to the heart and arterial bleeding [35].

The decrease in HDL-C in the obese group is because of the rise in VLDL-C and chylomicrons, which leads to an increase in the level of triglyceride-rich lipoprotein, and this causes a disturbance in the activity of cholesterol ester transfer protein. This acts to transfer the TGs from the triglyceride-rich lipoprotein to HDL-C and LDL-C, and this disorder results in decrease in HDL-C level. Also, it has been found that HDL-C is inversely proportional with the distribution of visceral lipids in the body, known by measuring the waist circumference, as the increase in WHR decreases the levels of HDL-C in contrast to the increase in the levels of cholesterol, TGs, LDL-C and VLDL-C [36]. It has been reported that a decrease level of HDL-C negatively affects the values of the lipids profile and the central role that HDL-C plays in converting cholesterol in excess of the cells' need and transporting it through the plasma to the liver [37]. Therefore, low levels of HDL-C mean a decrease in the effectiveness of cholesterol utilization [38]. Also, a study has confirmed the relationship between obesity and lipid profile [39]. The low value of HDL-C leads to health problems in obese individuals such as heart disease and atherosclerosis due to the accumulation of fat on the inner walls of the bloodstream [40].

The reason for the increase in the levels of LDL-C and VLDL-C is as follows: First, the lack of insulin leads to the decomposition of fats stored in the fatty tissues, which results in high levels of these compounds, resulting from the activation of lipoprotein lipase in the fatty tissue, and this leads to the release of fats into the blood circulation. Second, the increase in oxidative stress is linked to the pathophysiological manifestations of metabolic syndrome, and LDL-C cholesterol, which is linked to its high sensitivity to oxidative stress, as LDL-C is the main component of the metabolic syndrome, resulting in an imbalance in the level of body lipid. This is what is found in cases of obesity. It was found by Rubins *et al.* that obese people notice an increase in the value of LDL-C/VLDL-C [41].

Lteif *et al.* showed that obese people have an increase in the value of both LDL-C/VLDL-C as a result of the high value of TGs resulting from the defect in the lipoprotein lipase responsible for breaking down TGs found in the chylomicrons and VLDL-C [42]. Another study reported the increase in the value of LDL-C/VLDL-C levels in obese people as a result of the increase in waist circumference caused by fatty density [43). Karpe et. al. observed that obesity leads to insulin resistance in the body as a result of a defect in the function of adipose tissue, and consequently, an increase in the values of LDL-C/VLDL-C levels occurs [44].

Conclusion

The results showed the increase in the levels of TC, TG and LDL-C. Also, no difference in the HDL-C and vitamin D_3 levels was observed in obese people group compared with non-obese group.

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