The Medical Journal of Tikrit University (2025) 31 (2): 145-154
DOI: http://dx.doi.org/10.25130/mjotu.31.2.16

IRAQI Academic Scientific Journals

ISSN: 1813-1638

The Medical Journal of Tikrit University

Available online at:www.mjotu.com

MJTU

The Medical Journal of Tikrit University

The Effect of Vitamin D deficiency on Central Obesity and Fatty Liver in Female within Reproductive Age

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Keywords: Central obesity, vitamin D, waist hip ratio, Body mass index, Nonalcoholic fatty liver disease.

ARTICLE INFO

Article history:

Received	01 Jul 2025
Accepted	01 Sep 2025
Available online	31 Dec 2025

Citation:

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ABSTRACT

Background: The complications of central obesity and the rapidly increasing burden have become important health issues. There is inverse relationship between the status of vitamin D (VD) and obesity .VD deficiency is a risk factor for the development of Nonalcoholic fatty liver disease (NAFLD), also VD deficiency was associated with atherosclerotic cardiovascular disease. The aim of the study is to assess the level of VD in young female with central obesity, assess the parameters of central obesity and its association with VD deficiency.

Methods: This study was based on a case control with sample of (80) young females' aged from14-45 years. The females in study were single or married but not pregnant. Females divided in to 2 groups' forty having central obesity and forty did not have central obesity, the parameters measured are VD level by i-chroma-II, BMI (kg/m^2) and waist-hip ratio. Fatty liver diagnosed by liver ultrasound .The differences in parameter measured between two groups were analyzed.

Results: Eighty females divided into two groups: 40 with central obesity and 40 without central obesity, VD (10.6) was significantly low among female with central obesity. The BMI (30.0) and WHR (0.95) among the central obesity group were significantly high. The incidence of fatty liver within central obesity group was significant. The correlation of VD with BMI, WHR and fatty liver were weak and indirect.

Conclusion: There is a positive correlation between VD deficiency and the pathogenesis of central obesity in young female, the study found the low VD levels associated significantly with developing NAFLD. So it is important to monitor VD levels, decrease the risk of obesity and screen for NAFLD.

INTRODUCTION

The complications associated with obesity and rapidly increasing burden have become worldwide health issues [1]. In 2016, More than 650 million adults age 18 years and older were obese. Most of the world's population who live in countries killed by overweight and obesity more than underweight [2]. Vitamin D (VD) is essential micronutrient that perform many biological processes [3].Vitamin D, lipid soluble hormone, produced on exposure to ultraviolet light, because this lipophilic nature there is a relationship between the level of VD and adipocyte thus causes dilution in the adipose tissue and reduce the availability of biological active form VD. There is inverse relationship between the status of micronutrient and obesity. In fact obese individuals appear with micronutrient deficiency, one of these is VD deficiency. [4].

In general, it is known n that lean and obese adults contain equivalent amounts of VD, but the most likely explanation for the inverse association between VD serum levels and body mass index (BMI) is volumetric dilution of VD. The wide distribution of VD into the serum, muscle, fat, and liver compartments are increased in obesity. In overweight individuals, VD distributes throughout a larger volume, resulting in lower blood concentrations [5].

Wortsman et al. discovered that, in comparison to normal weight people, obese subjects had lesser increases in plasma levels of vitamin D after exposure to sunshine, .Despite the cutaneous synthesis of VD does not differ between obese and normal subjects after oral supplementation. The rationale is that subjects with high levels of adipose tissue have lower plasma levels of VD due to the fact that VD (fat-soluble vitamin) accumulates and is kept in adipose tissue [6].

Although neither of these theories can fully explain the inverse relationship between low plasma VD levels and abdominal obesity, it is possible that these mechanisms will have an impact on how VD, obesity and diseases associated with disturbances interact between them [7].

Obesity associated with increased risk of Non-alcoholic fatty liver NAFLD which is a benign adipose tissue accumulation in the liver in which abundance of fat deposits in liver cells. It is very common in obesity. The prevalence of NAFLD is 24% all over the world. In a powerful European study it was observed that NAFLD is present in 94% of obese patients with BMI> 30Kg/ m^2 [8]. The association between VD and NAFLD actually still undetermined, although several studies suggest the association between VD deficiency with presence of fatty liver [9,10], while other studies considered VD deficiency a risk factor for the development of NAFLD [11].

Low VD is linked to health issues, this deficiency has been linked to a variety of conditions, including cancer, neurological illnesses, and metabolic syndrome, however it is still unknown how VD deficiency cause many of these conditions [12]. One study suggests the changes in the expression of genes regulated by the VD receptor contribute to the progression of these diseases [7].

Also, VD deficiency causes insufficient insulin levels by disturbing synthesis of insulin and secretion thus accelerated the development of type 2 diabetes, obesity and metabolic syndrome [13].Because VD

deficiency is linked to visceral adiposity, it is also used as a biomarker of a dysmetabolic state (cardiovascular diseases, dyslipidemia, type 2 diabetes, and arterial hypertension).

There is a role for VD deficiency in atherosclerotic cardiovascular disease by complicating dyslipidemia profile [14,15]. So it's important to investigate the level of VD in women with central obesity, to decrease the occurrence of complication that associated with central obesity and its consequence of VD deficiency.

The aim of this study is to assess the level of VD in young female with central obesity, assess the parameters of central obesity and its association with VD deficiency

MATERIALS AND METHODS

Ethical approval

The consent was taken from the Participant's after explaining to them the aim of our study that may help them and the community for better health care, we promise the participants to protect their information and use only the necessary information for educational purposes. The protocol of the study was approved by regional research Committees at the College of Pharmacy Nineveh University 18/1/2023

Study design and Setting

This study was based on a case control with a representative sample of (80) young females aged from 14-45 years. The study commenced in 2023 (from January to April) and conducted in a clinic and laboratory of private medical center in Mosul city.

Participants

The female participate in this study are single or married. The pregnant women, alcohol drinking, those having liver disease and use hepatotoxic medication were excluded.

Collection of samples and parameters measured

After informing the female and taking their agreement for inclusion in the study, a sample volume 2.5ml of venous blood were collected to prepare a serum for measuring VD concentrations level (ng/ml) by using fluorescence immunoassay (FIA) was performed with semiautomatic i-Chroma -II readers. The VD level in the subjects is diagnosed to be deficient when the level less than 30 ng/ml, insufficient when the level ranges from 30-39 ng/mL and sufficient when the level between 40-100 ng/mL.

The females involved in this study divided in to 2 groups:

1- Forty females having central obesity, 39 of them have abnormal level of VD, either deficiency or insufficient VD level.

2- 40 females did not have central obesity (control) but have either normal or abnormal level of vitamin D3.

Diagnosis of Central obesity

The diagnosis of central obesity was based on measuring BMI and WHR BMI (kg/m^2) calculated by dividing weight (in kilograms) by height (in meters) squared. and classified as normal weight ($\geq$18.5 and <25 kg/m^2), Overweight ($\geq$25 and <30 kg/m^2) and obesity ($\geq$30 kg/m^2) [16].

The waist-hip ratio (WHR), is the waist circumference in centimeters divided by the hip circumference in centimeters. For woman more than 0.9 was used to define abdominal obesity. Based on waist

measured at baseline with a metric tape placed midway between the final rib and the top border of the iliac crest, central obesity was investigated. At the conclusion of the respiratory cycle's expiration phase, two measurements were made. A third was taken if there was a measuring discrepancy of more than 3 cm. The two closest measurements were averaged. For measurement of hip circumference, stand up straight and wrap a tape measure around the widest part of the hips. Take the measurement at the ends of the tape measure overlap [17].

After eliminating alcohol consumption and other liver illnesses, NAFLD was diagnosed by abdominal ultrasound examination the liver, gallbladder, kidneys, and spleen were evaluated during the examination. The liver size, hepatic steatosis and presence of localized lesions were assessed. The results were recorded on the presence or absence of fatty liver [18].

Statistical Analyses:

According to whether central obesity is present or not in each person, the descriptive statistics were employed to determine their characteristics. The data management for the study was documented by using Microsoft Excel 2007 sheets. Mean quartiles (25^{th} and 75^{th}) were used to express the numerical data, whereas frequencies and percentages were used to indicate categorical data.

Mann-Whitney U test has been used to find the association between the numerical data while, Chi square test was used for comparison between categorical variable Statistics were deemed significant when a p-value was less than 0.05.

RESULTS

Table (1) demonstrated a comparison of the age and anthropometric measurements between patients and controls female without central obesity**,** the results showed that the median age among the female with central obesity group was higher than that among controls group but not statistically significant (p=0.595). The medians of BMI (30.0) and WHR (0.95) among the central obesity group were significantly higher than that among the controls group (26.0) and (0.76) in that order at (p=0.001) and (p=0.000) respectively.

Comparison of the Blood tests between cases of central obesity and controls showed that the median of VD among the female with central obesity group (10.6) was lower in a statistically significant way (p=0.000) from that among the controls (21.0).

Table (2) Comparison of the Study parameters between cases with central obesity and controls was shown that positive family history among female with central obesity group were significantly differed from these among controls group, also a positive and significant difference ultrasound findings for fatty liver within central obesity group, while marital status showed no significant differences.

Table (3) show the correlation of VD with BMI, WHR and fatty liver. The correlation were weak and indirect and significant at (p=0.012), (p=0.015) and (p=0.012) respectively.

DISCUSSION

It's becoming increasingly evident for the presence of link between VD deficiency, central obesity and NAFLD [19]. Abdominal obesity is associated with the risk of incidence of low V D concentrations [20].

Many studies found VD deficiency is common in subjects with central obesity, the explanation is that VD has a role in fats function and metabolism and at low level of VD fat cells may be store fat and reduce release it for energy [19,20].

Obesity is a risk factor for many chronic diseases, especially cardiovascular diseases also V D deficiency contributes to a higher risk for subject of these diseases given its association with atherosclerosis, cardiac disease that appear in early age [20].

VD deficiency was associated with central obesity in young female in our research. In consistent with many prior studies and a systematic review study, that show obesity and comorbidities was associated with significantly lower VD levels, although controversial results were found from these studies because the variation in parameters and the sample include both sex ,while in our study we analyzed only young women [21,22,23,24].

One hypotheses have been put forward to explain relation of obesity with VD deficiency is that leptin have a role in the cause and development of obesity [25]. VD regulates adipogenesis, energy metabolism, and regulation of leptin pathways and inhibition of adipogenesis. The leptin level is negatively controlled by VD, some invitro studies suggest that addition of VD to adipocyte culture inhibits leptin secretion [26]. So by this mechanism VD deficiency can cause obesity.

In our study we take anthropometric measurements (age, BMI and WHR) between cases and controls since these measurements provide important evidence on prevalence of central obesity. The results showed that the median age among the central obesity group was lower than that among control group but statistically, not significant. Our results disagreed with a recent study conducted in Baghdad on women during 2021 that found significant association between serum VD level and participants age at P- value of (0.013) among (30-39) years old [27]. While other study in Basrah [28], found VD level increased with increasing age.

In Our study found the difference in medians of (BMI and WHR) among the central obesity group were significantly higher than that among the controls group, thus compatible with another studies which assumed that VD level is inversely correlated with many parameters of obesity, such as BMI, waist circumference and fat mass [29]. Our results regarding BMI disagree with Baghdad study that shown no significant correlation between serum VD and BMI was found, while this study in agreement with our results about WHR, as shown by the significant correlation between serum VD level and WHR, as the WHR increase, VD level decrease [27].The explanation is that VD have a role in fat cell function and metabolism, in low VD level fat cells become more likely to store fat and less likely to release it for energy [30].

Remarkable difference for VD level was observed between cases and controls, regarding positive ultrasound findings of fatty liver. This study confirmed with several studies and meta- analysis that found a significant correlation between serum VD concentration and NAFLD [31,32, 33]. The explanation for correlations between VD and NAFLD remains entirely unclear, however according to the pathophysiology of NAFLD in obese subjects there is increase in fatty acid flux to the liver, this

imbalance between the rate of import/ synthesis and the rate of export/catabolism of fatty acid in the liver causes the development of steatosis [8].

There's also a theory that fat people's livers produce less VD, which is why persons who are V D deficient have greater levels of proinflammatory cytokines, which can cause NAFLD to develop. [34].

By attaching to the VD receptor and activating hepatocyte nuclear factor 4α (HNF4α), VD controls the transport of lipids [35]. Because of this, the person with VD deficit has an increase in the blood's flow of free fatty acids (FFAs), which hastens the deposition of fat into hepatocytes and advances NAFLD [36]. Additional investigation is required to elucidate these possibilities.

Limitation of the study:

Our study had some limitation include the small sample size because it based on the special criteria of the participant that include young females have central obesity. liver biopsy was not taken in this study and us is made because its a suitable diagnostic test for NAFLD ,safe, sensitive and specific for identifying fatty infiltration. Liver enzyme also not made because it used in diagnosis of steatosis. The measurement of leptin is not feasible.

CONCLUSION

1-There is a positive correlation between VD deficiency and the pathogenesis of central obesity in young female.

2-The study demonstrated a relation between VD levels and NAFLD. Low VD levels associated significantly with developing NAFLD.

3-VD deficiency is a common problem in our city (Mosul) due to poor exposure to sun light and the diet not contain adequate amount of VD. So it is important to monitor VD levels, lower the risk of obesity and screen for NAFLD, VD supplementation is recommended and exposure to sun light

CONFLICT OF INTEREST

No conflict of interest during our research.

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TABLES

Table (1): Comparison of the age and anthropometric measurements and VD between cases and controls.

Age and anthropometric measurements	Central obesity cases Median (25^{th}, 75^{th})Q Range(Min, Max)	Controls group Median (25^{th}, 75^{th})Q Range(Min, Max)	p-value *
Age	23.5(17.0, 30.8) 28.0(14.0,42.0)	25.5(17.8, 31.0) 30.0(14.0, 44.0)	0.595

BMI	30.0(26.0,32.4) 17.0(23.0,40.0)	26.0(24.0,29.5) 8.0(23.0,31.0)	**0.001**
WHR	0.95(0.9, 1.2) 101.3(0.7,1.02.0)	0.76(0.7, 0.8) 0.4(0.6, 1.0)	**0.000**
VD	10.6(8.0,15.0) 27.0(6.0,33.0)	21.0(11.3,37.5) 45.7(7.3,53.0)	**0.000**

Table (2): Comparison of the Study parameters between cases and controls.

Study parameters		**With Central obesity cases (n=40) No. (%)**	**Controls without central obesity group (n=40) No. (%)**	**p-value***
Marital status	Married	22(55.0)	26(65.0)	0.361
	Single	18(45.0)	14(35.0)	
Ultrasound of fatty liver	Positive	33(82.5)	1(2.5)	**0.000**
	Negative	7(17.5)	39(97.5)	
Family history	Positive	12(30.0)	2(5.0)	**0.003**
	Negative	28(70.0)	38(95.0)	

Table (3): The correlation of VD with BMI, WHR and Fatty liver.

Spearman Correlation of Vitamin D	**Value**	**Asymp. Std. Error[a]**	**Approx. T[b]**	**p-value**
BMI	-0.278	0.100	-2.561	0.012[c]
WHR	-0.270	0.104	-2.477	0.015[c]
Fatty liver	-0.256	0.102	-2.367	0.012[c]