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Estimation of Vaspin Hormone in Obese Women With Vitamin D3 Deficiency

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ABSTRACT

Background: Obesity currently stands as one of the prevailing and widespread endocrine disorders globally, it has evolved into a worldwide epidemic over the last five decades. Adipose tissue, identified as an endocrine organ within the body, plays a role in certain obesity-related conditions including insulin resistance, diabetes mellitus, and atherosclerosis. Adipose cytokines are among the factors derived from adipose tissue and act as hormones. Vaspin, among these adipocytokines, is generated by both visceral and subcutaneous adipose tissues, it serves to regulate the metabolism of adipose tissue and stimulates processes such as the maturation and differentiation of adipocytes.

Materials& Methods: The individuals who participated in this study were 90 women between (18-50) years old. The research encompassed 45 healthy women with regular menstrual cycles, serving as an age-matched control group. In contrast, the second group comprised 45 obese women who had previously been diagnosed with a vitamin D deficiency.

Results: The results showed that there was a significant increase at ($P \leq 0.01$) in the level of Vaspin, Parathyroid hormone (PTH) and Osteopontin (OPN) in the group$_2$ compared to the control group, while there was a significant decrease at ($P \leq 0.01$) in Vitamin D_3 (VD_3), Vitamim K_2 (VK_2), Phosphorus(P) and Calcium(Ca^{+2}) in group$_2$ compared to the control group.

Conclusion: This study found that vaspin, osteopontin, and PTH show an inverse association with vitamin D_3. While vitamin K_2, calcium, and phosphorus show a positive association with vitamin D_3.

Keywords: Obesity, adipocytokines, vaspin, vitamin D and osteopontin.

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INTRODUCTION

Obesity is related to VD deficiency, which in turn, is recognized as a global issue. Medications, inadequate sun exposure, nutritional deficiencies, disorders impeding absorption, liver and kidney illnesses, and metabolic deficiencies are a few of the factors leading to deficiencies. According to certain observational studies low serum levels of 25(OH)D have been associated with conditions such as obesity, diabetes mellitus, and metabolic syndrome. There is compelling proof that VD deficiency decreases lipolysis and increases lipogenesis, inhibits uncoupling protein-2 (UCP-2), and modifies intracellular ionized calcium signaling in adipocytes. It's believed that excess body fat keeps VD metabolites intact and that body fat partially absorbs cholecalciferol that's either produced by the skin or consumed through food before it's transferred to the liver for the first hydroxylation. The bulk of evidence linking vitamin D (VD) and obesity is observational. In contrast, experimental findings suggest that vitamin D deficiency may contribute to increased adipocytes by stimulating elevated levels of parathyroid hormone and promoting an augmented influx of calcium into adipocytes. This indicates a potential link between VD status, parathyroid hormone regulation, and adipose tissue function [1].

Adipose tissues (AT) are loose connective tissue that is primarily composed of adipocytes as well as pre-adipocytes, macrophages, fibroblasts, endothelial cells, and leucocytes [2]. As a lipid reservoir, it is widely dispersed throughout the body and is essential to maintaining energy homeostasis. Additionally, adipocytes, especially those found in visceral fat, are endocrinologically active [3]. AT content and lipid composition are highly influenced by a variety of factors, such as climate, diet, and biological species [4]. There are two main ways that AT can grow: either by the enlargement of already existing adipocytes (hypertrophy) or through hyperplasia, which is the differentiation of resident tissue precursors into new adipocytes [5]. AT can be classified into several types (white, brown, beige/ brite, and pink adipose tissue) based on its characteristics and functions [6].

Adipokines are a diverse group of bioactive substances including stress hormones, proteins involved in glucose homeostasis, complement system proteins, neuromodulators, growth factors, and acute phase proteins. The amount of adipose tissue in the human body is correlated with the circulating levels of particular adipokines, this interplay between adipose tissue and the secretion of adipokines contributes to the regulation of various physiological processes, including metabolism, inflammation, and overall homeostasis [7]. Adipokines could hold clinical significance as indicators of adipose tissue function and heightened metabolic risk, given their pivotal role in various metabolic processes, including the regulation of appetite and energy expenditure [8], insulin sensitivity, inflammation, and cardiovascular activity. Adipokines exhibit several characteristics, including pro-inflammatory classical cytokines like TNF-α and IL-6, chemokines such as monocyte chemoattractant protein-1, proteins associated with vascular homeostasis (plasminogen activator inhibitor), blood pressure regulation (angiotensinogen), glucose homeostasis (adiponectin), lipid metabolism (retinol binding protein), and involvement in angiogenesis through vascular endothelial growth factor (VEGF) [9], also in puberty (Leptin) [10]. Vaspin is an additional example of adipokines that have been studied for its roles in various physiological processes, including inflammation and metabolic regulation [11].

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Vaspin is a protein hormone belonging to the serine protease inhibitor family [12], with a molecular mass of 47 kDa, and it is secreted from AT. In rats, mice, and humans, the vaspin hormone is composed of 412, 414, and 415 amino acids, respectively [13]. The visceral secretion of vaspin is notably correlated with BMI, body fat percentage, and blood glucose tolerance. Certain studies suggest that the upregulation of vaspin mRNA in human adipose tissue may represent a mechanism linked to both obesity and insulin sensitivity [14]. Research findings indicate that serum vaspin levels rise in cases of type 2 diabetes, obesity, and impaired insulin sensitivity, while they decrease with the reversal of diabetes, weight loss, and improved insulin sensitivity. Following metformin treatment, a significant reduction in serum vaspin levels has been observed [12]. In general, Serine proteases are a category of proteolytic enzymes, constituting over one-third of all known enzymes with proteolytic activity. Serine proteases are widely distributed in nature, and found in diverse living organisms, including viral genomes. The active site of serine proteases is defined by three essential amino acids: serine, histidine, and aspartate, collectively referred to as the "catalytic triad." These proteases play various roles in human health, encompassing general digestion as well as intricately regulated processes like embryonic development, immune response, blood coagulation, and sperm penetration into the egg [15].

MATERIALS AND METHODS

A total of 90 women between the ages of 18 and 50 were chosen of females only from private laboratories in Mosul city from the period 13/10/2022 to 26/12/2022.

The cases were diagnosed based on the calculation of BMI and determined vitamin D_3 concentration. An initial questionnaire was prepared for the women included in this study, including several items: age, body mass index, blood pressure, hyperthyroidism, and signs of vitamin D3 deficiency such as fatigue, hair loss, and osteoporosis. Work was done in private laboratories, and data for the women who were samples were taken according to a special form provided in the appendix. All women were separated into two groups:

Group 1: Control, includes 45 non-obese women with normal levels of serum parameters and no vitamin D_3 deficiency with BMI (22.40±1.41) Kg/m^2.

Group 2: This group included 45 obese women with vitamin D deficiency, with a BMI (38.81±3.98)Kg/m^2.

COLLECTING SAMPLES

Five milliliters of the women's venous blood were drawn after they had fasted for 12 hours. The serum was centrifuged to separate it, and the separated serum was put into an Eppendorf tube made of sterile plastic and frozen at (- 20) °C until it was tested.

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HORMONAL AND BIOCHEMICAL TESTS

The following laboratory investigations were conducted: BMI, (calculated as Kg/m2). The ELISA kit manufactured by Sunlong Biotech Company (China) has been used to estimate serum Vaspin and K2, OPN and PTH estimated by the ELISA kit manufactured by My Biosource Company (USA), Phosphorus and Calcium determined manually by Linear Chemical Company (Spain).

STATISTICAL ANALYSIS

Data was collected, revised, recorded, and entered into the IBM SPSS statistical software application. When the distribution of the quantitative data was determined to be parametric, the mean, standard deviations, and ranges were presented. Duncan's multiple range test showed that different letters of the alphabet significantly distinguished the different coefficients at the probability level of ($P \leq 0.001$), so a system of simple experiments and an entirely random design were used to analyze the data. The correlation between the variables was also examined [16].

RESULTS

Depending on the table below, the results also showed that there was a significant increase in the probability level ($P \leq 0.001$) in the levels of vaspin (3.15±0.55) ng/ml, osteopontin (33.37±5.27) ng/ml, and PTH (43.01±11.88) pg/ml in the second group, compared to the control group (0.82±0.22) ng/ml, (9.67±0.49) ng/ml, and (31.55±8.14) pg/ml, respectively. The results showed that there was a significant decrease at the probability level ($P \leq 0.001$) in the level of vitamin D_3 (5.78±2.66) ng/ml, VK_2 (292.44±30.12) pg/ml, calcium (7.59±0.47) mg/100 ml, and phosphorus (0.33±2.95) mg/100 ml in the second group compared to the control group (39.22 ±8.46) ng/ml, (23.94±2.28) pg/ml, (638.58±63.48) pg/ml, (9.41±0.83) mg/100 ml, (3.25±0.39) mg/100 ml, respectively.

Table 1: The value of study hormones in the different groups.

Groups / Parameters	Group 1 (means ± SD)	Group 2 (means ± SD)	p-value
Vaspin (ng/ml)	0.82 ± 0.22 b	3.15± 0.55 a	0.000 **
Osteopontin (ng/ml)	9.67± 0.49 b	33.37± 5.27 a	0.000 **
PTH (pg/ml)	31.55± 8.14 b	43.01± 11.88 a	0.000 **

The no. followed by different letters means there is a significant difference.
The values are means ± standard deviation SD

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Table 2: The value of study parameters in the different groups.

Groups / Parameters	Group 1 (means ± SD)	Group 2 (means ± SD)	p-value
Vitamin D (ng/ml)	39.22± 8.46 a	5.78± 2.66 b	0.000 **
Vitamin K_2 (pg/ml)	638.58± 63.48 a	292.44± 30.12 b	0.000 **
Calcium (mg/dl)	9.41± 0.83 a	7.59± 0.47 b	0.000 **
Phosphorus (mg/dl)	3.25± 0.39 a	2.95± 0.33 b	0.000 **
Body mass index	22.40± 1.41 b	38.81± 3.98 a	0.000 **

The no. followed by different letters means there is a significant difference.
The values are means ± standard deviation SD

Table 3: Correlations between Vitamin D and other parameters

	Vitamin D (ng/ml)
Vaspin (ng/ml)	.031
Osteopontin (ng/ml)	-.891**
PTH (pg/ml)	-.480**
K_2 (pg/ml)	.921**
Calcium (mg/dl)	.790**
Phosphorus (mg/dl)	.362**

**. Correlation is a significant at the 0.01 level

DISCUSSION

From a physiological perspective, endocrine characteristics are present in all four types of adipose cells. Numerous adipokines secreted by white adipocytes influence metabolism and eating habits. Hormones and growth factors are also secreted by brown or beige adipocytes. In addition to releasing components of milk, pink adipocytes secrete leptin [17]. The results of the current study showed that insufficient vitamin D concentration had been linked with obesity, and one effect affects the other, obesity can lead to vitamin D deficiency. Conversely, women with vitamin D deficiency suffer from obesity [18].

Recent research and experimental evidence support the idea that VD may play a role in the pathogenesis of obesity, instead of merely an effect. According to some experimental data, a VD deficiency-related rise in parathyroid hormone may encourage lipogenesis via increasing calcium flow in adipocytes. Another more

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likely theory is that 1,25(OH)D, the active form of VD, inhibits adipogenesis by modulating VD receptors. By inhibiting the transcription factor C/EBP that promotes adipocytes, VD receptors prevented differentiation in 3T3-L1 preadipocytes in the presence of 1,25(OH)D. Additionally, by maintaining the WNT/-catenin pathway, which decreases activity during adipogenesis, 1,25(OH)D can inhibit adipogenesis. Therefore, pre-adipocyte differentiation into adipocytes may be enhanced by lower VD levels. Furthermore, two detached longitudinal studies detected that subjects with lower baseline VD concentrations are more likely to become obese and experience greater weight gain than those with higher baseline VD concentrations but less weight gain. The precise function of VD deficiency in the predisposition and development of obesity is still unclear because there are few clinical studies. VD deficiency is most likely just a side effect of volumetric dilution and other causes, but since experimental data suggested positive outcomes, pathogenesis could not be completely ruled out. Therefore, more prospective randomized controlled trials are required to develop VD supplementation as a treatment option in the prevention of obesity. VD deficiency could work as a biomarker for a dysmetabolic state associated with visceral obesity, such as arterial hypertension, type 2 diabetes, dyslipidemia, and cardiovascular diseases because it is related to visceral adiposity. However, as a result of changes in gene expression controlled by VD receptors Its independent contribution to the development and course of these illnesses is still unknown. Additionally, the absence of potential anti-inflammatory effects on chronic low-grade inflammation in VD-deficient subjects may increase the risk of obesity-related metabolic disorders [19].

The subcutaneous AT of the obese group showed a 71% reduction in the expression of the cytochrome P450 2J2 gene, which codes for the enzyme 25-hydroxylase, and a 49% reduction in the expression of cytochrome P450 27B1, which codes for the enzyme 1-hydroxylase, in comparison to lean subjects [20]. The noticing of a decrease in the gene coding for these enzymes would suggest a deficiency of the bioactive form of VD and a diminished impact in the body because these enzymes are involved in specific steps of hydroxylation or the bioactive form of prohormone conversion. Additionally, there was no difference between the obese and normal weight groups in the expression of cytochrome P450 24A1, which codes for the enzyme that inactivates 1,25(OH)2D (a bioactive form). However, after losing weight, this gene's expression increased by 79% [19].

Our results were consistent with previous research which showed that obese people had higher vaspin concentrations than subjects of normal weight. Additionally, this study demonstrated that serum vaspin concentration was independently determined by BMI value [21]. Similar to our study, other study demonstrated elevated serum vaspin levels in obese patients in comparison to non-obese controls. Moreover, the research revealed numerous positive correlations between serum vaspin concentration and cardiometabolic risk-related parameters in both obese patients and the overall studied population [22].

The level of vaspin is influenced by a range of genetic and environmental factors, with body weight and the quantity of adipose tissue being among the most significant determinants. Obesity and decreased insulin sensitivity are linked to vaspin mRNA and serum levels. Adipose tissue's level of vaspin mRNA is correlated with an increase in body mass [23]. The level of vaspin is influenced by a range of genetic and environmental factors, with body weight and the quantity of

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adipose tissue being among the most significant determinants. Obesity and decreased insulin sensitivity are linked to vaspin mRNA and serum levels. Adipose tissue's level of vaspin mRNA is correlated with an increase in body mass [24], these factors increase with vitamin D deficiency which causes increased food intake and obesity [25]. The increase in serum vaspin concentration is linked to insulin resistance, obesity, and decreased insulin sensitivity and fitness. The hypothesis that vaspin is linked to body fat mass is supported by the fact that serum vaspin also has a significant correlation with leptin. In one study, there was a notable correlation between serum vaspin levels and the ratio of visceral to subcutaneous vaspin expression, as well as with the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) and fasting insulin levels [26]. Fat cell hypertrophy and an increase in the number of fat cells are common in obese people, and these factors can affect how endogenous hormones are expressed in fat cells. Insulin resistance could result from this. Serum vaspin levels were higher in obese and abnormally insulin-sensitive patients. Moreover, obese patients in the same group exhibited higher serum vaspin levels compared to their non-obese counterparts [27].

The results about the correlation between vaspin and VD were consistent with previous studies, where results showed a negative correlation between vaspin and VD in their study on obese diabetic female patients. Regarding the suggestion that preadipocytes have VD receptors, which explains the significant negative correlation between VD and vaspin. The active form of VD acts through these receptors and modifies adipokines to produce a variety of effects through various mechanisms [28]. According to recent research, adipocytes in both humans and rodents express the genes for 25-hydroxyvitamin D 1α-hydroxylase and VD receptors, suggesting that VD actions may target adipose tissue, this shows that VD may have an impact on the adipose tissue's release of adipokines such as vaspin [29].

The primary adipokine involved in attracting and accumulating ATMs in the tissue is osteopontin (OPN) [30]. In both rat and human obesity models, adipocytes, dendritic, stromal, and vascular cells, as well as ATMs, overexpress OPN, which is the primary cytokine overexpressed in adipose organs. This is supported by the observation that the inflammation and infiltration of macrophages into adipose tissue caused by a high-fat diet are significantly reduced in obese OPN-knockout rodents. This is why it is stressed that the primary source of OPN during obesity is VAT [31].

The hypothesis of OPN's function as a crucial regulator of the inflammatory mechanisms causing adipose tissue inflammation brought on by obesity is also advanced by the metabolic changes orchestrated by OPN in VAT [32] and develops into a potential treatment target for conditions linked to adipose tissue, like cardiac and renal diseases [31].

In a previous study, Osteopontin (OPN) levels were observed to be higher in overweight adolescents as opposed to obese adolescents. This finding is likely attributed to overweight being an early stage of inflammation and the onset of metabolic disorders, while obesity represents a condition where chronic pathological processes have already been established. The molecular expression profiles of natural killer cells have also disclosed immunological distinctions between overweight and obese individuals. indicating distinct immunological responses in the former group with a decline in response in the latter [33]. These

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results about the correlation between osteopontin and Vitamin D were consistent with a previous study, according to their findings, vitamin D3 is effective at lowering the RNA levels of the proinflammatory mediators IL-6 and OPN in the liver tissue of diabetic mice. Moreover, as repletion of serum 25OHD3 down-regulated OPN and IL-6 expression, They concluded that a lack of vitamin D_3 might encourage the activity of these inflammatory factors [34].

OPN-mediated gene expression of interleukin-6, OPN action in the liver is probably related to its binding to integrin receptor αvβ3 located on hepatic macrophages and other leukocytes, followed by stimulation of proinflammatory signaling transduction in these cells and subsequent expression of proinflammatory cytokines, including IL-6. vitamin D_3 exerts anti-inflammatory effects. Our results have shown the efficiency of vitamin D_3 in lowering RNA levels of IL-6 and OPN, two pro-inflammatory mediators, in liver tissue. Moreover, as the repletion of serum 25OHD3 down-regulated OPN and IL-6 expression, we can guess that a vitamin D_3 deficiency may make these proinflammatory factors more active. It can be suggested that vitamin D_3 action on IL-6 mRNA expression is mediated through its regulatory effect on the OPN gene. Several studies have reported the involvement of 1,25(OH)2D3 in the transcriptional regulation of various osteokines, including OPN [34].

Results of PTH in our study were consistent with previous studies conducted on obese individuals, they found that BMI and serum PTH level had a positive correlation. They concluded that fat mass significantly affects serum PTH independently of the relationship between vitamin D and parathyroid hormone [35]. The parathyroid hormone (PTH) controls the concentration of 25-hydroxyvitamin D, also known as vitamin D, which is essential for maintaining the balance of calcium and phosphorus in the body. Additionally, numerous authors have discussed the idea of functional vitamin D deficiency, which is characterized by secondary hyperparathyroidism and refers to high PTH levels combined with low vitamin D levels [36]. The most active form of vitamin D, 1,25-dihydroxyvitamin D, is synthesized and is tightly controlled by PTH, calcium, and phosphorus. Therefore, without taking into account the potential influence that other metabolites may have on the association, low levels of 1,25-dihydroxyvitamin D cannot necessarily be associated with a 25-hydroxyvitamin D deficiency [37].

The production of 1α,25(OH)2D in the kidney is controlled by parathyroid hormone (PTH), establishing a unique feedback loop between the kidney and the parathyroid gland. PTH exerts its effects through the G-protein-coupled receptor known as parathyroid receptor 1 (PTHR1), and all of its actions contribute to elevated blood calcium levels. PTH signaling stimulates the synthesis of 1,25(OH)2D in the proximal renal tubules, indirectly facilitating increased absorption of Ca^{+2} from the intestines. This process further enhances osteoclast activity, leading to the release of Ca^{+2} from the bone. Additionally, PTH signaling promotes calcium retention by increasing Ca^{+2} reabsorption in both the thick ascending limb and the distal convoluted tubule of the kidney. As a result, PTH secretion is reduced by elevated circulating 1,25(OH)2D and the normalization of blood calcium levels. Thus, a closely regulated feedback loop is established between 1,25(OH)2D and PTH, where PTH initiates 1,25(OH)2D synthesis, while 1,25(OH)2D suppresses PTH release [38].

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Phylloquinone (vitamin K_1), the most widely recognized form of vitamin K, plays a crucial role in activating various coagulation factors. Another significant vitamin K species is menaquinone-7 (MK-7), also known as vitamin K_2. MK-7, with higher concentrations outside the liver, primarily exerts effects beyond hepatic functions. While some pathways overlap, vitamin K_2 is considered the primary activator of matrix-Gla proteins (MGP), non-hepatic proteins crucial for inhibiting arterial calcification. Nevertheless, vitamin D_3 once again plays a role in stimulating the synthesis of these vital proteins, essential for their activation. The synthesis and activation of relevant proteins are essential for maintaining the balance of cellular calcium uptake and the mineralization process in both bone and blood vessels [39].

The findings regarding vitamin K_2 and vitamin D_3 in our study align with previous research on older adults, which highlighted a favorable relationship between vitamin K and D. This study observed that low levels of vitamin D and vitamin K were associated with negative cardiac remodeling and an elevated risk of all-cause mortality in both men and women [40]. Additionally, individuals with combined low vitamin status were found to have an increased risk of mortality compared to those with adequate levels of vitamins K and D [41]. These results suggest a collaborative effect of the two vitamins in promoting bone and cardiovascular health [39].

Results show a significant decrease at ($P \leq 0.001$) in levels of Calcium in obese women with VD deficiency. One of the main skeletal system's mineral components, calcium is also crucial for blood clotting, nerve conduction, muscle contraction, hormone and enzyme secretion, and muscle contraction. For healthy bone mineralization and growth of the skeleton and teeth, adequate calcium intake is necessary [42]. Recently, calcium and vitamin D have been linked to obesity. Emerging research indicates that low calcium intake and low vitamin D status may help control weight gain, especially when incorporated into a diet low in calories, even though maintaining energy balance is the most important aspect of body composition maintenance. In epidemiological studies, Reduced vitamin D levels and low calcium intake have been linked to an increased risk of obesity. A growing body of evidence suggests that having less calcium may lead to having more fat mass [43].

A low calcium intake aggravates the consequences of vitamin D deficiency, this suggests an interaction between vitamin D and calcium intake. By binding to the vitamin D receptor (VDR) in intestinal cells, the active calcium transport from the intestine to the circulation is stimulated by 1,25-dihydroxyvitamin D (1,25(OH)2D), a vitamin D metabolite. Active calcium absorption decreases when the serum 25-hydroxyvitamin D (25(OH)D) concentration is < 20 nmol/L [44].

Results of Calcium and obesity in our study were consistent with previous studies, where there is evidence of a positive correlation between P level and waist circumference and an inverse correlation between plasma zinc, calcium, and magnesium levels and BMI. Also, the results conducted in their study on adolescent girls show that in the relationship test between Ca^{+2} and obesity, were show the direction of the relationship is inverse. This means that the lower the calcium intake, the higher the BMI/U percentile [45]. Low calcium intake can stimulate calciotropic hormones (calcitriol and parathyroid hormones) thereby increasing the concentration of calcium in adipose tissue. In addition, it can reduce lipolysis and thermogenesis and increase lipogenesis [46].

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Results show a significant decrease at ($P \leq 0.001$) in levels of phosphorus in obese women with vitamin D deficiency. In 46,798 healthy Korean adults who participated in a health-check survey, plasma phosphate is negatively correlated with BMI, uric acid, fasting glucose, insulin, HOMA-IR, high-sensitive CRP (HS-CRP), triglyceride levels, systolic and diastolic blood pressure, and waist circumference. First, a clinical study has shown that low serum phosphate concentrations in obese people are inversely correlated with BMI. Later, it was believed that an excessive energy intake (nutrient-poor diet), specifically an intake of carbohydrates that was low in protein, which is a major source of phosphate, was the cause of low phosphate levels in obese subjects [47]. Therefore, it is possible to hypothesize that phosphorus plays a role in controlling body weight. Numerous studies back up the idea that phosphorus or hepatic ATP plays a role in controlling body weight and food intake [48].

CONCLUSION

The results showed that there was a significant decrease at the probability level ($P \leq 0.001$) in the level of vitamin D_3 (5.78±2.66) ng/ml and Omentin (15.81±0.93) pg/ml, in addition to vitamin K_2 (292.44±30.12) pg/ml, calcium (7.59±0.47) mg/100 ml, and phosphorus (0.33±2.95) mg/100 ml in the second group compared to the control group (39.22 ±8.46) ng/ml, (23.94±2.28) pg/ml, (638.58±63.48) pg/ml, (9.41±0.83) mg/100 ml, (3.25±0.39) mg/100 ml, respectively.

The results also showed that there was a significant increase at the probability level ($P \leq 0.001$) in the levels of vaspin (3.15±0.55) ng/ml, osteopontin (33.37±5.27) ng/ml, and PTH (43.01±11.88) pg/ml in the second group, compared to the control group (0.82±0.22) ng/ml, (9.67±0.49) ng/ml, and (31.55±8.14) pg/ml, respectively.

According to Pearson's correlation, vaspin, osteopontin, and PTH show an inverse association with vitamin D_3. While vitamin K_2, calcium, and phosphorus show a positive association with vitamin D_3.

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Conflict of interests.

There is no conflict interest

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الخلاصة

المقدمة: تعتبر السمنة حاليًا واحدة من اضطرابات الغدد الصماء السائدة والمنتشرة على مستوى العالم، وقد تطورت لتصبح وباءً عالميًا على مدار العقود الخمسة الماضية. تلعب الأنسجة الدهنية، التي تُعرف بأنها أحد أعضاء الغدد الصماء داخل الجسم، دورًا في بعض الحالات المرتبطة بالسمنة بما في ذلك مقاومة الأنسولين، ومرض السكري، وتصلب الشرايين. تعتبر الستوكينات الدهنية من بين العوامل المشتقة من الأنسجة الدهنية، وتعمل كهرمونات. يتم إنتاج الفاسبين، من بين هذه السيتوكينات الدهنية، بواسطة الأنسجة الدهنية الحشوية وتحت الجلد، وهو يعمل على تنظيم عملية التمثيل الغذائي للأنسجة الدهنية ويحفز عمليات مثل نضوج الخلايا الشحمية وتمايزها.

طرق العمل: كان الأفراد المشاركون في هذه الدراسة 90 امرأة تتراوح أعمارهم بين (18–50) سنة. وللمقارنة، شملت الدراسة 45 امرأة سليمة ذات دورة شهرية منتظمة، والمناسبة لعمرها كمجموعة ضابطة. تم بالفعل تشخيص إصابة 45 امرأة بدينة بنقص فيتامين D_3 ضمن المجموعة الثانية.

النتائج: أظهرت النتائج وجود ارتفاع معنوي في مستوى الفاسبين وهرمون الغدة الجار درقية والأوستيوبونتين في المجموعة الثانية مقارنة بمجموعة السيطرة، في حين كان هناك انخفاض معنوي في مستوى فيتامين K_2 والفوسفور والكالسيوم في المجموعة الثانية مقارنة مجموعة السيطرة.

الاستنتاج: أظهرت هذه الدراسة أن الفاسبين والأوستيوبونتين وهورمون الغدة جار الدرقية يظهرون علاقة عكسية مع فيتامين D_3, بينما يظهر فيتامين K_2 والكالسيوم والفوسفور ارتباطًا إيجابيًا بفيتامين D_3.

الكلمات المفتاحية: السمنة, السيتوكينات الدهنية, فاسبين, فيتامين D_3, أوستيوبونتين.