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Estimation of Subfatin, Fetuin-A and other Parameters in Obese Menopause Women

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ABSTRACT

Background: Menopause is an aging in women, it begins in most of them at 45 years old, the main reason of menopause is the decline of the Estrogen result from decline of ovaries function, it's include many of symptoms such as nocturnal sweats, hot flashes, depression, elevated of anxiety, insomnia, sleep disturbance, genitalia include inflammation, dryness, urinary tract infections, painful urination, gaining weight, usually in the abdomen, hips and joint pain. Menopause leads to many disease include insulin resistance, atherosclerosis, chronic hearts disease, obesity, osteoporosis. The adipose tissue it's secretes active proteins such as subfatin which is adipocytokines regulates food intake. Fetuin-A is adipokines and hepatokiens that generated mainly by liver it serves to act as carrier of free fatty acid in circulation.

Materials& Methods: Ninety women ages (50-65) participated in the study. (45) non-obese menopausal women with BMI (22.08±3.39) Kg/m^2 as a control group. (45) obese women in the menopause with a BMI (35.38±2.52) Kg/m^2 as the second group. **Results:** The results showed that there was a significant decrease at (P≤ 0.01) in the level of Subfatin and increase at (P≤ 0.01) in Fetuin-A in the second group compared to the control group.

Conclusion: This study found significant negative correlation between Subfatin and lipid profile and significant positive correlation between Fetuin-A and lipid profile.

Keywords: Menopause; adipocytokines; hepatokiens; Fetuin-A and Subfatin.

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INTRODUCTION

Menopause, a natural occurrence that occurs between the ages of 45 and 55 as a natural mean of biological ageing, is the permanent cessation of menstruation due to loss of ovarian follicular function. It is identified by 12 months of amenorrhea [1]. The transition to menopause accrue gradually, beginning with changes in the menstrual cycle known as 'Perimenopause' which is the period can last several years and can affect physical, emotional, mental and social well-being [2].Ovulatory dysfunction may theoretically raise the risk of obesity since it prevents women from experiencing the customary "reduced appetite" time. The female sex steroid hormones, especially progesterone and estradiol, play a major role in mediating this relationship between body weight regulation and reproductive function. Estradiol generally controls women's homeostatic nutrition by reducing food intake and raising energy expenditure [3] in other word (with the loss of estradiol activity) lead to an increase in food consumption[4] .

Adipokines comprise a variety of bioactive molecules, such as growth factors, stress hormones, acute phase proteins, complement system and proteins implicated in glucose homeostasis. Interactions between adipokine secretion and adipose tissue govern several physiological processes, including inflammation, metabolism, and general homeostasis. Adipokine levels are positively correlated with the quantity of adipose tissue in the human body [5]. Angiotensinogen, plasminogen activator inhibitor, adiponectin, retinol binding protein, blood pressure regulation, chemokines like monocyte chemoattractant protein-1, pro-inflammatory cytokines such as TNF-α and IL-6, proteins linked to vascular homeostasis, glucose homeostasis, lipid metabolism, and involvement in angiogenesis are some of the characteristics that define adipokines [6]. Adipokines that have been described in this study subfatin which is first identified as a novel secreted protein that in conjunction with meteorin, constitutes a new family of evolutionary conserved proteins [7]. Subfatin was discovered as a new adipokine by Li [8]. According to bioinformatics study the 311 amino acid subfatin protein encoded in human genomes has an NH2-terminal signal patch of 45 amino acids and no transmembrane region, indicating that it is a mature protein that, when secreted, has 266 amino acids, Research have revealed that a long-term high-fat diet, inflammation, physical activity, exposure to the cold and other variables all stimulated its production[9]. Elevated levels of subfatin in the bloodstream encourage energy expenditure and enhance mice's glucose tolerance [10] subfatin not only stimulates the expression of

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genes linked to thermogenesis in beige/brown adipose tissue, but it can also control insulin resistance, lipid-mediated inflammation, and adipocyte differentiation[11 ,12].

Fetuin-A is a liver heterodimeric plasma glycoprotein It is expressed most frequently in adult hepatocytes and embryonic cells, and less frequently in adipocytes and monocytes [13] . In animal experiments, the link between circulation levels of fetuin-A and fat mass was examined under a variety of situations, including diet-induced obesity, exercise-induced weight loss, and anorexia [14]. The relationship between body weight, fat mass, and secreted fetuin-A is indicated by the expression and secretion pattern of fetuin-A by adipose tissue from visceral and subcutaneous stores under different physiological and nutritional situations. Visceral adipose tissue from anorectic animals secretes less fetuin-A. It has also been demonstrated that lowering body weight lowers human circulation fetuin-A levels[15] Furthermore, evidence suggests that fetuin-A may affect adipose tissue and trigger the generation of inflammatory cytokines by adipocytes and macrophages, ultimately leading to the development of overall insulin resistance.

MATERIAL AND METHODS

This study involved (90) women aged between (50-65) were selected from private laboratories in the city of Mosul from the period 15/8/2023 to 18/11/2023.The diagnosis of women based on the calculation of BMI and last 12 months with amenorrhea. The women in this study were given a preliminary questionnaire that had multiple items : age, body mass index, time of fasting, time of last menstruation, and signs of menopause such as fatigue**,** hot flashes and increase in food intake (obesity). 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 divided two groups:

Group$_1$: (Control group), contain 45 non-obese menopause women with BMI (22.08±3.39) Kg/m^2.

Group$_2$: contain 45 obese menopause women with BMI (35.38±2.52) Kg/m^2.

Collecting sample

Following a 12-hour fast, five milliliters of the women's venous blood were extracted. After the serum was separated by centrifugation, it was placed in a sterile plastic Eppendorf tube and frozen at -20 °C until it was time to conduct the test.

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Hormonal and biochemical test

The following experiments were carried out in the lab: BMI, which is expressed in kg/m2. To assess serum levels, the Sunlong Biotech Company (China) ELISA kit has been utilized. To estimate Sulfating and Kits for determination of Fetuin-A manufactured by My BioSource Company (USA), lipid profiles TG, TC, LDL-c, VLDL-c, HDL-c determined by Fujifilm device Japan in origin.

Statical analysis

Data was gathered, edited, documented, and imported into the statistical program IBM SPSS version (16). Once it was shown that the quantitative data distribution followed a parametric distribution, the mean, standard deviations, and ranges were displayed. The data was analyzed using a system of straightforward experiments and a completely random design. Additionally, there is no results that represent the correlation coefficient (r) between the variables was obtained [16].

RESULTS

Depending on the table (1), the results showed that there was a significant decrease at ($P \leq 0.001$) in the levels of subfatin (0.51±0.67) ng/ml, in the second group, compared to the control group (3.28±0.69) ng/ml. The results showed that there was a significant increase at ($P \leq 0.001$) in the level of fetuin-A (70.0±3.34) ng/ml in second group compared to the control group (44.6±4.99) ng/ml, the result show (in table 2) a significant increase at ($P \leq 0.001$). In the TG, TC, LDL-c and VLDL-c (2.48±52.9) mg/dl, (2.55±47.63) mg/dl, (1.79±50.08) mg/dl, (49.76±10.58) mg/dl respectively and there are significant decrease in HDL-c (25.36±3.61) mg/dl in the second group compered to control group TG, TC, LDL-c and VLDL-c (1.69±33.54) mg/dl, (1.91±29.69) mg/dl, (1.17±33.19) mg/dl, (33.86±6.70) mg/dl respectively and HDL-c (40.12±5.02) mg/dl.

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Table (1): The levels of hormones in the two groups.

Groups / Parameters	Group[1] (means ± SD)	Group[2] (means ± SD)	p-value
Sufatin (ng/ml)	3.28±0.69 a	0.51±0.67 b	0.000 **
Fetuin-A (ng/ml)	44.6±4.99	70.0±3.34	0.000 **

The values are means ± standard deviation SD.

Table 2: Lipid profile in the two groups.

Groups / Parameters	Group[1] (means ± SD)	Group[2] (means ± SD)	p-value
TG (mg/dl)	1.69±33.54	2.48±52.9	0.000 **
TC (mg/dl)	1.91±29.69 b	2.55±47.63 a	0.000 **
LDL-c (mg/dl)	1.17±33.19 b	1.79±50.08 a	0.000 **
VLDL-c (mg/dl)	33.86±6.70 b	49.76±10.58 a	0.000 **
HDL-c (mg/dl)	40.12±5.02 a	25.36±3.61 b	0.000 **

The values are means ± standard deviation SD.

DISCUSSION

From a physiological standpoint, each of the four varieties of adipose cells possesses endocrine properties, white adipocytes release a variety of adipokines that affect eating patterns and metabolism, brown or beige adipocytes also release growth factors and hormones, pink adipocytes release leptin in addition to milk-related substances. [17]. Our results showed that decrease in subfatin concentration had been linked with obesity due to increase in a petites, from these basis it is believed to regulated of energy homeostasis [18] low of subfatin in menopause due to the fact that the menopausal state consider chronic

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inflammation and inflammation accrue in all tissue particular adipose tissue which is the main source of subfatin, inflammation suppress subfatin secretion. The study of Rupérez [19] lined with our result by ageing could lead to a significant reduction in circulating subfatin levels so, treatment with subfatin reduced lipid-induced inflammation [20], The lack of adipose tissue subfatin worsened the development of high fat diet-induced hypertriglyceridemia [21]. Subfatin also increased the expression of genes related to lipid metabolism and enhanced the activity of lipase in adipose tissue [22]. The reason for the connection between reduced levels of circulating subfatin and unfavorable lipid profile is as follows: low levels of subfatin can inhibit lipoprotein lipase, resulting in decreased fatty acid oxidation and increased production of triglycerides in adipose tissue and liver. This, in turn, contributes to hypertriglyceridemia and disrupts the metabolism of cholesteryl esters (HDL-c and LDL-c) [23]. The protective impact of subfatin on adipose tissue and skeletal muscle is achieved by increasing subfatin levels. This rise in subfatin stimulates whole-body energy expenditure by activating a wide range of genes related to beige/brown fat thermogenesis. Additionally, subfatin in adipocytes also plays a role in triglyceride metabolism [10].

Menopause lead to inflammation of tissue which eventually contributes to the development of obesity. The level of fetuin-A is positively correlated with a reduction in Estradiol (E2). A decrease in E2 levels in menopausal women may lead to an increase in total body fat, particularly in the central region. This is followed by a loss of subcutaneous fat and an increase in visceral fat, which in turn leads to an increase in fetuin-A concentration in the bloodstream [24]. Prior studies have shown that a lack of E2 not only causes a redistribution of fat, with a decrease in subcutaneous fat and an increase in harmful visceral fat [25], but also increases appetite by influencing the activity of molecules involved in regulating food intake [26]. This leads to obesity, which aligns with the findings of our study. Additionally, E2 reduces adiposity by promoting the utilization of lipids as a source of energy through the activation of processes that enhance fat oxidation in muscle, inhibit lipogenesis in adipose tissue, liver, and muscle, and improve the rate of adipocyte lipolysis. Consequently, lower levels of fatty acids in circulation result in decreased levels of fetuin-A in the presence of E2 .[27]

Fetuin-A is a protein produced by adipocytes, which are fat cells. The level of expression of Fetuin-A is directly correlated with the quantity of fat present in these cells. Additionally, Fetuin-A is classified as both a hepatokine and an adipokine. Hyperlipidemic

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conditions lead to an increase in the expression of fetuin-A, which is regulated by the transcription factor NFκB [28]. This upregulation occurs through the stimulation of fetuin-A's promoter activity by free fatty acids (FFA). As a result, fetuin-A promotes the uptake and storage of FFA in adipocytes, while also negatively affecting adipocyte function by inhibiting the phosphorylation of the peroxisome proliferation-activating receptor γ (PPARγ) [29] . PPARγ is a nuclear transcription factor that regulates adipogenesis, lipid and glucose metabolism, and insulin sensitivity. It plays a crucial role in controlling the expression of various target genes, including adiponectin, fatty acid-binding proteins 4 (FABP4) [30], fetuin-A, and adiponectin levels. It has been observed that fetuin-A suppresses adiponectin mRNA in cultured human adipocytes, and treatment of wild-type mice with fetuin-A leads to lower serum adiponectin levels[31] . Decreased adiponectin concentrations have been associated with higher LDL-c and TG concentrations, possibly due to the direct impact of adiponectin on lipoprotein lipase [32].

CONCLUSION

In conclusion menopause revealed a significant increasing in subfatin and TG, TC, LDL and VLDL lipid levels and significant decreasing in fetuin-A and HDL in obese menopause women

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Conflict of interests.

There are no conflicts to declare.

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الخلاصة

المقدمة: يعد سن الياس (شيخوخة النساء) وغالبا ما يبدئ بعمر 45 ويعود السبب الأساسي الى نقص هرمون الانوثة الاستروجين نتيجة ضمور وظيفة المبايض ويشمل سن الياس العديد من الاعراض ومنها التعرق الليلي, الهبات الساخنة ,الاكتاب, القلق, قلة النوم, التهاب الاعضاء التناسلية, الجفاف, التهابات المسالك البولية المتكررة, سلس البول, زيادة الوزن خاصة في البطن والوركين والم المفاصل وكما يؤدي سن الياس الى العديد من الامراض ومنها اضطراب ايض الدهون ,تصلب الشرايين, مقاومة الانسولين, امراض القلب المزمنة, السمنة وهشاشة العظام. تلعب الأنسجة الدهنية، التي تُعرف بأنها أحد أكبر الغدد الصماء داخل الجسم، تقوم بافراز بروتينات تنظم الشهية وتناول الطعام ومنها السبفاتن ويعتبر اديبونيكتين ولكن الفتوين أ يعتبر اديبوكاين وهيباتوكاين يفرز بشكل أساسي من الكبد ويعمل على نقل الاحماض الدهنية الحرة في الجهاز الدوران طرق العمل: كان الأفراد المشاركون في هذه الدراسة 90 امرأة تتراوح أعمارهم بين (50–65) سنة. وللمقارنة، شملت الدراسة 45 امرأة غير بدينة في سن الياس حيث كان مؤشر كتلة الجسم **(22.08±3.39) Kg/m^2** كمجموعة ضابطة. و 45 امرأة بدينة في سن الياس حيث كان مؤشر كتلة الجسم **(35.38±2.52) Kg/m^2** ضمن المجموعة الثانية. النتائج: أظهرت النتائج وجود انخفاض معنوي في مستوى هرمون السبفاتن وارتفاع معنوي في هرمون الفتوين ا في المجموعة الثانية مقارنة بمجموعة السيطرة.

الاستنتاج: أظهرت هذه الدراسة أن هرمون السبفاتن يظهر علاقة عكسية مع مستويات الدهون في الجسم بينما يظهر هرمون الفتوين ا علاقة طردية مع مستويات الدهون في الجسم

الكلمات المفتاحية: سن الياس , السيتوكينات, الاديبونيكينات , سبفاتن, فيتوين أ.