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Relationships between Obesity, Lipids and Fasting Glucose in the Menopause

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SUMMARY

Introduction Menopause leads to the development of central adiposity, a more atherogenic lipid profile and increased incidence of metabolic syndrome independent of age and other factors.

Objective The aim of the study was to investigate the relationships between anthropometric characteristics, sex hormones, lipids and fasting glucose in menopausal women.

Methods The study included 87 menopausal women, who where divided into groups according to two criteria: BMI≥26.7 kg/m^2 and BMI≥25 kg/m^2. Anthropometric characteristics and blood pressure were measured. Blood was taken at 08.00 h for fasting glucose, triglycerides, cholesterol, HDL, LDL, apolipoprotein A, apolipoprotein B, lipoprotein(a) (Lp(a)), C-reactive protein, fibrinogen, follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL), estradiol, progesterone, testosterone and sex hormone binding globulin (SHBG).

Results Significant differences between groups were found for weight, BMI, waist, hips circumference, waist/hip ratio (WHR), systolic and diastolic blood pressure, Lp(a), FSH, LH, PRL (for systolic blood pressure $p<0.05$, for the rest $p<0.01$) and fasting glucose ($p<0.05$). In obese and overweight women with BMI≥26.7kg/m^2 significant negative correlations were found for FSH and glucose, SHBG and LDL, SHBG and total cholesterol, SHBG and glucose, BMI and HDL, WC and HDL. In obese and overweight women with BMI≥25kg/m^2 significant negative correlations were found for BMI and HDL, waist circumference (WC) and HDL, WHR and HDL, FSH and glucose, SHBG and glucose; significant positive correlations were between BMI and glucose, WC and glucose and WHR with triglycerides.

Conclusion Gaining weight and decreased SHBG are related to dyslipidemia and increased fasting glucose confirming increased incidence of metabolic abnormalities in the menopause.

Keywords: obesity; lipids; glucose; menopause

INTRODUCTION

The climacteric period is associated with gradual reduction in the levels of estradiol, but also DHEA and its sulphate (adrenopause), melatonin and growth hormone. These endocrine alternations may result in visceral obesity, insulin resistance, lipid disorders, thrombotic risk and increase in the prevalence of type 2 diabetes [1]. Menopause leads to the development of central adiposity, a more atherogenic lipid profile and an overall increase in the metabolic syndrome (MetS) independent of age and other factors [2]. It is possible that the features of MetS are related to estradiol deficiency. The metabolic syndrome has been defined as a constellation of lipid and non-lipid risk factors that increase subjects' risk for the development of cardiovascular disease (CVD) [3]. The diagnosis of MetS was established by the International Diabetes Federation (IDF) from 2005, and based on the following criteria: central obesity (waist circumference ≥94 cm for European men and ≥80 cm for European women) plus any two of the following four factors: serum triglyceride (TG) levels ≥1.7 mmol/L, serum high density lipoprotein (HDL) cholesterol levels <1.04 mmol/L for man and <1.29 mmol/L for women, systolic blood pressure ≥130 mmHg or diastolic ≥85 mmHg, and fasting glucose ≥5.6 mmol/L or already diagnosed type 2 diabetes mellitus [4].

Sex steroids are involved in the regulation of body fat distribution and adipose tissue metabolism. The increased prevalence of MetS with menopause may be a direct result of ovarian failure, or an indirect result of the metabolic consequences of central fat redistribution with estradiol deficiency [5]. Menopausal women in comparison to premenopausal women have a higher total cholesterol (TC), low density lipoprotein (LDL) cholesterol, triglycerides and lipoprotein(a) [Lp(a)] levels and lower HDL cholesterol levels [6]. ApoB, the primary apolipoprotein of LDL particles, and other Apo B-containing particles are also higher in postmenopausal women compared to premenopausal women. LDL particle composition is also changed in the menopause with the prevalence of small, dense LDL which increases the risk of cardiovascular diseases (CVD). Increasing TG with menopause may be related to the fact that TG levels are highly correlated with increased abdominal fat content and insulin resistance. Several studies have shown a significant rising in Lp(a) levels with menopause, as well as the risk of CVD independently of LDL levels [5].

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A high LDL cholesterol level is well established risk factor for cardiovascular disease. However, most cardiovascular accidents happen to people with normal or borderline high risk LDL cholesterol levels. Jeppesen et al. [7] have shown that MetS, which does not include increased LDL cholesterol level, is at least as powerful as high LDL cholesterol in predicting the risk of cardiovascular disease. The Lipid Research Clinic study demonstrated during follow-up period that a low HDL-cholesterol was the most significant predictor of death from CVD in women, after being adjusted for age [8]. Chamberlain et al. [9] suggested that the decrease in endogenous estradiol as a result of menopause may independently affect lipoprotein concentrations. It is likely that age plays a significant role in developing dyslipidemia. According to the study of Marina et al. [10], women in physiological menopause have much higher levels of total cholesterol, LDL cholesterol, triglycerides and Lp(a) and the risk of CVD than women in early menopause. In the study of Weinberg et al. [11] mean levels of estradiol, testosterone and free testosterone measured through free androgen index (FAI) were higher, and SHBG was lower among women with MetS. Low SHBG, considered as a marker of insulin resistance, was associated with each of the individual components of MetS, even after adjustment for Body Mass Index (BMI).

OBJECTIVE

The aim of this study is two-fold: a) to investigate the relationships between anthropometric characteristics, sex hormones, lipids and fasting glucose in obese, overweight and normal weight postmenopausal women, and b) to disclose how previous relationships influence the incidence of metabolic abnormalities in the menopause.

METHODS

Subject characteristics

This study was carried out at the Institute of Endocrinology, Diabetes and Metabolic Diseases, Clinical Centre of Serbia, from December 2004 till October 2006. The research included 87 women, aged 50-65 years with hypertension, diabetes and a different degree of increased body mass (majority of women were on antihypertensive and antihyperglicemic therapy), and none of them were on hormone replacement therapy (HRT). The participants were divided according to BMI in two groups: a) obese/overweight women and b) controls – normal weight women. Two criteria for division of participants in groups were BMI ≥25 kg/m^2, as suggested by the World Health Organization [12, 13, 14] (63 obese and overweight women and 24 controls) and BMI ≥26.7 kg/m^2 suggested by Ridker et al. [15] (50 obese and overweight women and 37 controls), who found that value was critical for the emergence of MetS in women. We had no data about weight gain for half of the women; five of them have been always obese, and 25 women gained weight during menopause. Women were included in the study if they had stopped menstruating for at least one year.

Anthropometric variables such as body weight, height, waist and hip circumference were measured, as well as blood pressure. Body weight (kg) was measured to the nearest 0.01 kg using a balance scale. Height (m) was measured using a stadiometer to the nearest 0.01 m. Waist and hip circumference were measured to the nearest 0.01 m using a flexible steel metric tape. Waist circumference (WC) was defined as the horizontal distance around the abdomen at the umbilicus level. Hip circumference (HC) was measured as the distance passing horizontally through the two superior iliac bones. One of the indicators of abdominal obesity, waist/hip ratio (WHR), was also determined. The value of more than 0.9 was considered abnormal, but we had not used this indicator for determining abdominal obesity. Abdominal (central) obesity was defined as WC ≥80 cm (for European women). Blood pressure was measured on the right arm in the sitting position, taking the average value of two consecutive measurements in the interval of three minutes. The hypertension was diagnosed by the value of blood pressure ≥130/85 mmHg or if the patient was on antihypertensive therapy. BMI, as the main method for body weight assessment was calculated using the formula weight/height^2 (kg/m^2). In order to collect information about time when the menopause started and possible therapies all women filled in the questionnaire "Obesity and Menopause".

Biochemical analyses

Blood samples were taken via venipuncture at 08.00 h for fasting glucose, triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, apolipoprotein A (ApoA), apolipoprotein B (ApoB), lipoprotein(a) (Lp(a)), C-reactive protein (CRP), fibrinogen, follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL), estrogen (E_2), progesterone (P), testosterone (T) and sex hormone binding globulin (SHBG). Plasma total cholesterol (TC), triglycerides (TG), HDL and LDL were determined by the method of chromatography (Boeringher Mannheim kit). Plasma glucose concentrations were measured using the glucose oxidase method (automated glucose analyzer Beckman Coulter, Inc., Fullerton, CA), while ApoA, ApoB and Lp(a) by nephelometry method (nephelometer BN-100, Behring, Germany). Criterions for elevated levels of triglycerides, fasting glucose and decreased HDL cholesterol were TG ≥1.7 mmol/L, glycemia ≥5.6 mmol/L and HDL <1.29 mmol/L, according to the diagnostic criterions for MetS established by IDF in 2005. The referent value for other parameters depends on the applied method.

Hormonal analyses

All hormones were determined by using RIA method (CIS biointernational set, Filiale de/Subsidiary of Schering S.A.,

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France, for measuring FSH, LH, prolactin, E_2, testosterone and SHBG; set INEP – Vinča, for progesterone). Referent values for hormones are given by the suitable method.

Statistics

Results are expressed as mean value ± standard deviation. Linear correlation was used for estimating the relation between parameters. The Pierson's test of correlation was applied for estimating the statistical significance of coefficients of correlations between groups. Statistical significance of differences between groups was examined by the Student T test. Statistical analysis of data was done using the SPSS 11.5 software.

RESULTS

Anthropometric characteristics

The baseline characteristics are presented in Table 1. In regard to BMI ≥25 kg/m^2, there was no statistically significant difference between obese and overweight and controls for age, time since menopause and height. The differences in body weight, BMI, WC, hip circumference, waist/hip ratio and diastolic blood pressure between obese and overweight and normal weight women were highly significant ($p<0.01$), while in the case of systolic blood pressure they were significant ($p<0.05$).

In regard to BMI ≥26.7 kg/m^2, the results were the same for all variables like that BMI ≥25 kg/m^2, with exception of systolic blood pressure for which there no statistically significant difference.

Additionally, it was found that there was no statistically significant difference between obese and overweight women selected in the groups using the above mentioned BMI criteria, while in regard to controls significant difference ($p<0.05$) was found for body weight and BMI (Table 1).

Biochemical parameters

Among biochemical parameters regarding BMI=25 kg/m^2, no significant differences were found between obese and overweight women and controls. The mean values of TC, HDL and LDL were found to be lower, while for the rest of parameters the average values were higher in the obese and overweight women.

Regarding BMI=26.7 kg/m^2, the mean values of fasting glucose were significantly higher in the obese than in the normal-weight participants (6.22±2.26 vs. 5.49±2.43; $p<0.05$). Concentrations of TC, HDL and LDL cholesterol were higher in controls, while TG levels were higher in a basic group. Nevertheless, the differences of those lipids were not statistically significant. The obese and overweight women had higher Apo A, Apo B and Lp(a) levels, but only the difference for Lp(a) between the groups was significant (0.50±0.36 vs. 0.11±0.03; $p<0.01$). Fibrinogen serum concentrations (one of inflammation markers) were higher in the obese and overweight than in the normal-weight women, but without statistical significance between the groups.

Hormonal analyses

Hormone serum levels and their differences are presented in Table 2. For BMI ≥25 kg/m^2, the average values of all hormones, except for testosterone, were higher in the normal-weight women. The difference for LH serum concentrations between the obese and overweight and controls was statistically significant ($p<0.05$).

Table 1. Anthropometric characteristics and values of systolic and diastolic blood pressure

Variables	Obese and overweight women		Normal weight women		p	
	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2
Age (years)	54.14±3.72	54.40±3.64	54.33±3.93	53.92±3.95	NS	NS
Time since menopause (years)	5.80±5.60	5.90±5.46	6.26±4.06	5.96±4.92	NS	NS
Height (cm)	164.47±5.63	164.10±5.79	162.75±5.50	163.83±5.45	NS	NS
Weight (kg)	83.15±17.28	86.20±17.81	59.00±6.22	62.81±7.90	<0.01	<0.01
BMI (kg/m^2)	30.65±5.77	31.92±5.83	22.28±1.59	23.50±2.13	<0.01	<0.01
Waist circumference (cm)	97.05±15.17	99.96±14.65	77.16±6.26	79.90±8.78	<0.01	<0.01
Hip circumference (cm)	111.41±13.34	114.31±11.00	95.74±7.35	96.93±11.04	<0.01	<0.01
Waist/Hip ratio (cm)	0.87±0.09	0.87±0.09	0.81±0.05	0.83±0.08	<0.01	<0.01
Systolic blood pressure (mmHg)	147.87±27.03	148.23±24.37	134.96±18.85	139.14±26.62	<0.05	NS
Diastolic blood pressure (mmHg)	91.69±13.70	93.08±13.41	85.30±8.28	85.75±10.54	<0.01	<0.01

BMI – Body Mass Index; NS – no significance

Regarding BMI ≥26.7 kg/m^2, the difference for FSH, LH and PRL was highly statistically significant (p<0.01).

Additionally, it was found that there was no a statistically significant difference either between the obese and overweight women selected based on the above mentioned BMI criteria, or in regard to the controls (Table 2).

Correlations

BMI ≥25 kg/m^2

In obese and overweight women, BMI and WC negatively correlated with HDL (r=0.29, r=0.31; p<0.05). On the other hand, BMI and WC positively correlated with glucose (r=0.26, r=0.29; p<0.05). WHR positively correlated with TG (r=0.29; p<0.05) and negatively with HDL (r=0.36; p<0.01). We did not find significant correlations for the mentioned variables among the controls.

Furthermore, in the obese and overweight women, FSH negatively correlated with glucose (r=0.31; p<0.05), as well as SHBG to glucose (r=0.57; p< 0.01). In the normal-weight group estradiol was positively associated with LDL (r=0.49; p< 0.05).

BMI ≥26.7 kg/m2

In the obese and overweight women FSH negatively correlated with glucose (r=-0.30; p<0.05), as well as SHBG with LDL cholesterol, total cholesterol and glucose (r=-0.51; r=-0.50; r=-0.53; p<0.05 respectively). In the controls, significant positive correlation was observed between LH and HDL cholesterol (r=0.34; p<0.05).

Regarding relationships between anthropometric characteristics and lipids, in the obese and overweight women, a significantly negative correlation (p<0.05) was found between BMI and HDL cholesterol (r=-0.31) and WC and HDL cholesterol (r=-0.37).

We did not find significant correlations for the above mentioned variables among the normal-weight group.

DISCUSSION

In the present study we tried to assess the influence of gaining weight and hormonal changes on lipid profile as well as fasting glycemia in the menopause. Systolic and diastolic blood pressures were significantly higher in the obese and overweight women than in the controls. These results are in accordance with previous studies [16, 17, 18]. Increased BMI and increased proportion of visceral adipose tissue were in strong correlation with hypertension as well as the number of metabolic risk factors for CV diseases [17]. Reckelhoff and Fortepiani [18] have found that many postmenopausal women gain weight, which leads to increased blood pressure and greater incidence of type II diabetes. Moreover, overweight and obesity are associated with increased activity of the sympathetic nervous system, especially in kidneys, leading to increased secretion of rennin which attributes to hypertension.

In our study fasting glucose was significantly higher in the obese and overweight subjects. Similarly, fasting glucose was significantly higher in post-menopausal women with greater WC in the study of Ainy et al. [16]. Obesity is related to ectopic accumulation of lipids in the muscles, liver and β-pancreatic cells, leading to insulin resistance (IR) in the muscles and liver and impairment of β-cells function. Free fatty acids (FFA) are in competition with glucose for oxidation, suggesting that increased lipid oxidation worsen IR in obesity [19]. In the present study, regarding BMI ≥26.7 kg/m^2 22% of normal-weight and 42% of obese and overweight women already had been diagnosed diabetes mellitus or glucose intolerance, while in regard to BMI ≥25 kg/m^2 16% of normal-weight and 40% of obese and overweight women had mentioned this diagnosis. Progressive increase in WC, decreased HDL levels, increased TG concentration and blood pressure occurred with changes of glucose homeostasis from normal to impaired glucose tolerance, increased fasting glucose and finally diabetes mellitus [20].

Furthermore, there was a statistically significant difference between the groups for Lp(a), but not for other lipids and fibrinogen. Lipids and lipoproteins are important in

Table 2. Hormonal analyses of obese and overweight and normal weight women

Variables	Obese and overweight women		Normal weight women		p	
	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2	BMI≥25.0 kg/m^2	BMI≥26.7 kg/m^2
FSH (IU/L)	60.00±27.91	54.35±27.16	67.93±33.85	72.32±30.17	NS	<0.01
LH (IU/L)	21.58±11.52	20.33±11.08	30.15±15.11	28.77±14.16	<0.05	<0.01
E_2 (pmol/L)	0.09±0.12	0.09±0.11	0.11±0.16	0.11±0.15	NS	NS
T (nmol/L)	1.61±1.88	1.66±2.11	1.08±0.77	1.24±0.82	NS	NS
P (nmol/L)	4.95±3.98	5.32±4.48	6.18±3.45	5.33±2.91	NS	NS
PRL (nmol/L)	269.24±166.96	251.52±142.60	365.60±252.89	370.27±237.74	NS	<0.01
SHBG (nmol/L)	45.94±23.88	46.94±25.68	63.12±43.11	55.05±36.03	NS	NS

FSH – follicle stimulating hormone; LH – luteinizing hormone; E_2 – estrogen; T – testosterone; P – progesterone; PRL – prolactin; SHBG – sex hormone binding globulin; NS – no significance

the development of atheromatous disease. Lp(a) is atherogenic largely because of its propensity for retention in the arterial wall, but it may also compete with fibrinogen and inhibit fibrinolysis. High levels of Lp(a) are associated with increased risk for CVD, but probably only when LDL levels are also raised. The small, dense LDL particles are particularly atherogenic because they are more likely to become retained in the sub-endothelial space, and to undergo oxidative damage. Studies [21, 22] have shown significant relationship between BMI, abdominal obesity and triglycerides that increase with ageing. Furthermore, Torng et al. [23] showed that menopause was associated with significant increases in TC, LDL cholesterol, TG and Apo B levels. Total cholesterol, LDL cholesterol, TG and Apo B levels increased consistently with BMI in the middle-aged women, regardless of menopausal status.

Regardless of non-significant difference between the groups for fibrinogen, recent studies have demonstrated that chronic subclinical inflammation is important in pathogenesis of obesity and MetS [24, 25]. Estradiol diminished plasma concentrations of fibrinogen, plasminogen, plasminogen activator inhibitor (PAI), homocistein and antithrombin III, and disturbed the function of thrombocytes. Thus, estrogen deficit is related to coagulation disorders, which increases the risk for CV diseases [1, 26].

Hormonal analyses of obese and overweight and normal weight women showed a significant difference for FSH, LH and PRL between the groups when the criterion for BMI ≥26.7 kg/m^2 was used. Only LH was significantly different between the obese and overweight and controls when BMI ≥25 kg/m^2 was used as the criterion for division. Namely, FSH and LH levels were significantly higher in the controls, which could be explained by the fact that due to a lower body fat mass in these women the synthesis of estrone was also decreased, as well as suppression of gonadotropin production in hypophysis. These results are in accordance with those of Malacara et al. [27], who have reported that obese and overweight postmenopausal women have lower FSH concentrations because of higher estradiol. They also disclosed that there was a relationship between FSH and BMI, independent of age, smoking or duration of menopause, but they did not find a direct correlation between FSH and estrogens. Moreover, we found that PRL values were significantly higher in normal-weight women. There is opinion that the number and size of lactotropic cells, as well as PRL secretion are increased by estradiol [28]. It could be expected that obese and overweight women have higher PRL values owing to the increased synthesis of estrone, but in our study that was the case with the normal-weight women. Taking into account the fact that estrone levels were not measured in this study, we could not determine whether it influenced the obtained PRL values.

After these basic analyses, we tested if the correlations existed between sex hormones and lipids, as well as between sex hormones and fasting glucose. We found a significant positive correlation between LH and HDL in the normal-weight women regarding BMI ≥26.7 kg/m^2, and negative one between FSH and fasting glucose in the obese and overweight women (according to both BMI criteria). It is possible that the relationship between FSH and glycemia is influenced by gaining weight which, on the other hand, influences metabolism of glucose and insulin, but we did not find any suitable explanation for these results in the literature available to us.

In the obese and overweight women we found significant negative correlations between SHBG and LDL, TC and blood glucose regarding BMI ≥26.7 kg/m^2. When we selected women using BMI ≥25 kg/m^2 as the criterion, FSH and SHBG negatively correlated with glucose in the obese and overweight women.

One of the best proved metabolic effects of obesity on circulating endogen hormones is a progressive decline of SHBG levels with increasing BMI in both pre- and postmenopausal women. Mechanism responsible for this is related to the increase of insulin concentrations with increasing BMI. Namely, insulin inhibits hepatic synthesis of SHBG [29]. Moreover, Weinberg et al. [11] found a close relationship between SHBG and MetS, as well as between SHBG and individual components of MetS in postmenopausal women. In the study of Mudali et al. [30] SHBG was related to a favorable lipid profile including a lower total LDL cholesterol, TG and a higher HDL cholesterol in postmenopausal women with minimal atherosclerosis. Women with significant atherosclerosis had SHBG levels related only to lower TG and higher HDL cholesterol. According to one explanation, associations between sex steroids and lipids are mediated by obesity and IR. However, the relationship between SHBG and lipid parameters in these postmenopausal women persisted even after adjustment for obesity and IR. SHBG can positively influence the lipid profile by regulating free androgen levels (testosterone). Namely, SHBG binds testosterone with high affinity thus regulating the way of its free levels, so that lower SHBG concentrations mean higher androgenicity [30].

We did not find any correlations of estradiol, T, P and PRL with lipids and fasting glucose. Some researches [30, 31] analyzed relationships between sex hormones, lipids and glucose metabolism. In the study on relatively young postmenopausal women, Kalish et al. [31] found that free and bound estradiol levels were significantly correlated with IR independently of central obesity measured by WHR. After adjustment for BMI only free estradiol remained in relationship with IR. Furthermore, high levels of free testosterone were in relationship with IR independently of BMI and WHR. Another study on overweight postmenopausal women [30] showed that estrone was positively correlated with increased TG and TC in women with significant atherosclerosis. These relationships existed even after adjustment for obesity and IR. It is supposed that estradiol influences enzymes involved in lipid metabolism, like lipoprotein lipase, hepatic lipase and hormone sensitive lipase.

Regarding the relationship between anthropometric characteristics, lipids and fasting glucose, we found significant negative correlations of BMI and WC with HDL in the obese and overweight subjects of both groups. BMI and WC positively correlated with fasting glucose, while

WHR demonstrated positive association with TG but negative one with HDL in the subjects with BMI ≥25 kg/m^2. Feng et al. [32] showed that the post-menopausal women had a significantly lower BMI but higher abdominal fat percentage than the pre-menopausal women. Studies showed that WC, rather than WHR can serve as index of abdominal (visceral, central) obesity and in the assessment of cardiovascular risk [33, 34].

In contrast to our results, Rendell et al. [22] did not find significant correlation between abdominal (visceral) fat mass (measured with computed tomography) and HDL in early postmenopausal women. In a study the premenopausal women [35] BMI and WHR were positively correlated with plasma lipids and lipoproteins (TG and LDL cholesterol), whereas HDL was negatively correlated with BMI. The presence of a significant correlation between abdominal obesity and hyperinsulinemia, IR, increased FFA in plasma, hypertension, tendency to thrombosis, increased TG, small dense LDL particles, as well as decreased HDL cholesterol has been well known for decades [36].

In conclusion, the results of the present research indicate that obese and overweight women have higher anthropometric characteristics, diastolic blood pressure, fasting glucose, Lp(a), but lower FSH, LH and PRL plasma concentrations than normal-weight women. These variables, as well as other lipids and hormones analyzed in this study are most likely influenced by gaining weight, especially in abdominal obesity and menopause. Furthermore, we disclosed significant negative correlations among SHBG, LDL, TC and fasting glucose, as well as among BMI, WC and HDL in obese and overweight subjects. BMI and WC were positively correlated with fasting glucose, while WHR demonstrated a positive association with TG but negative one with HDL in the subjects with BMI ≥25 kg/m^2. This confirmed the finding of many studies that BMI, WC, WHR as well SHBG are related to lipid and glucose metabolism; however, underlying mechanisms are incompletely understood.

CONCLUSION

Our study is limited by a small sample size which may have limited us in a possible disclosure of other differences between the groups. Moreover, a considerable number of women had been on antihypertensive and antihyperglicemic therapy which could have changed the levels of the measured variables. There is no doubt that further studies are necessary to understand cellular and molecular mechanisms involved in relationships among obesity, sex hormones and changes of overall metabolism in the menopause.

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Веза између гојазности, липида и гликемије у менопаузи

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КРАТАК САДРЖАЈ

Увод Менопауза доводи до развоја централне гојазности, атерогеног липидног профила и повећане инциденције метаболичког синдрома независно од старости и других фактора.

Циљ рада Циљ рада је био да се испита повезаност антропометријских обележја, полних хормона, липида и вредности гликемије наташте код жена у менопаузи.

Методе рада Студијом је обухваћено 87 жена у менопаузи које су сврстане у групе према вредностима индекса телесне масе (ИТМ): ИТМ≥26,7 *kg/m*^{2} и ИТМ≥25 *kg/m*^{2}. Испитаницама је измерен крвни притисак и установљена су антропометријска обележја. Венска крв је узимана у осам сати ујутро за следеће параметре: гликемију наташте, триглицериде, укупни холестерол, *HDL*-холестерол, *LDL*-холестерол, аполипопротеин А, аполипопротеин Б, липопротеин(а) (*Lp(a)*), *C*-реактивни протеин, фибриноген, *FSH*, *LH*, пролактин (*PRL*), естрадиол, прогестерон, тестостерон и *SHBG*.

Резултати Утврђене су значајне разлике између група за: телесну тежину, ИТМ, обим струка, обим кукова, однос струк–кук, систолни и дијастолни крвни притисак, *Lp(a)*, *FSH*, *LH*, *PRL* (за систолни крвни притисак $p<0,05$, за остало $p<0,01$) и гликемију наташте ($p<0,05$). Код гојазних жена са ИТМ≥26,7 *kg/m*^{2} значајне негативне корелације су уочене за вредности: *FSH* и гликемије наташте, *SHBG* и *LDL*-холестерола, *SHBG* и укупног холестерола, *SHBG* и гликемије наташте, ИТМ и *HDL*-холестерола, те обима струка и *HDL*-холестерола. Код гојазних жена са ИТМ≥25 *kg/m*^{2} значајне негативне корелације су утврђене за вредности: ИТМ и *HDL*-холестерола, обима струка и *HDL*-холестерола, односа струк–кук и *HDL*-холестерола, као и *FSH* и *SHBG* са глукозом; значајне позитивне корелације су уочене између вредности ИТМ и глукозе, обима струка и глукозе, те односа струк–кук и триглицерида.

Закључак Повећање телесне тежине и смањење *SHBG* су повезани са дислипидемијом и повећаном гликемијом наташте, што потврђује повећање инциденције метаболичких поремећаја у менопаузи.

Кључне речи: гојазност; липиди; гликемија; менопауза

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