ORIGINAL ARTICLE

Nutritional habits and oxidative stress in postmenopausal age

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KEY WORDS

dietary habits, dyslipidemia, oxidative stress, postmenopausal obesity

ABSTRACT

INTRODUCTION Postmenopausal obesity increases the risk of oxidative stress, but such an association in newly diagnosed dyslipidemia after menopause requires detailed research.

OBJECTIVES The aim of the study was to evaluate the relations between oxidative processes, newly diagnosed dyslipidemia, and nutritional behavior in postmenopausal women who did not receive hypolipidemic treatment.

PATIENTS AND METHODS The nutritional status, dietary habits, and oxidative stress parameters were evaluated in 102 postmenopausal women (51 obese and 51 normal-weight subjects) without lipid disturbances before menopause.

RESULTS In obese subjects, hypercholesterolemia, higher levels of malondialdehyde and advanced oxidation protein products (AOPPs), and a positive correlation between AOPPs and low-density lipoprotein cholesterol (LDL-C) were observed. Plasma superoxide dismutase (SOD) activity positively correlated with high-density lipoprotein cholesterol (HDL-C) and negatively with the ratios of total cholesterol to HDL-C and LDL-C to HDL-C in both groups. In obese women, daily food rations were characterized by a higher intake of copper and of energy from fat and saturated fatty acids (SFA), while the intake of carbohydrates and selenium was lower than that in lean women ($P < 0.05$). The multivariable models showed a significant effect of SFA and selenium intake on the variability of serum SOD activity ($P = 0.003$; $R^2_{adj} = 17\%$) and malondialdehyde concentrations ($P = 0.00001$; $R^2_{adj} = 45\%$) in obese women.

CONCLUSIONS The study showed that oxidative stress processes are present at early stages of hypercholesterolemia in obese postmenopausal women and may be caused by a poorly balanced diet.

INTRODUCTION After menopause, the prevalence of obesity increases, and a tendency to the accumulation of visceral fat tissue is observed.[1,2] Excess of fat in postmenopausal women is usually independently associated with subclinical atherosclerosis, an early stage of atherosclerotic processes within the arterial wall, which confers an increased risk of cardiovascular diseases, and higher mortality compared with the general population. Moreover, obesity, as a component of metabolic syndrome, is frequently associated with a higher level of circulating low-density lipoproteins (LDLs), which are easily oxidized.[3-6]

In lipid disorders, an enhanced synthesis of malondialdehyde (MDA) and advanced oxidation protein products (AOPPs) is observed; however, an intake of 3-hydroxy-3-methyl-glutaryl-CoA reductase inhibitors (statins) affects oxidative stress parameters. AOPPs are the pro-oxidative protein products formed during oxidation that act as mediators of inflammation.[7] Their detrimental effect may be minimized by antioxidant defense systems such as that provided by superoxide dismutase (SOD)—an important sensitive biomarker of antioxidant capacity for age-related changes.[4,6,8] Its activity may be influenced by dietary components, menopause transition, and aging.[8,9] Obesity and dyslipidemia are strongly associated with the formation of reactive oxygen species (ROS). However, there are no data regarding the extent of oxidative processes in newly diagnosed postmenopausal dyslipidemia in patients not receiving hypolipidemic treatment.

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Considering the above rationale, the aim of this study was to characterize the nutritional status and dietary habits and their possible relationship with oxidative stress in obese postmenopausal women with newly diagnosed dyslipidemia. We also sought to investigate whether the levels of MDA and AOPPs as well as the activity of SOD were related to changes in the lipid profile in postmenopausal obese and lean women.

PATIENTS AND METHODS In this study, 451 postmenopausal women aged from 50 to 65 years were recruited from a regional outpatient clinic. The study included only women with natural (not surgical) menopause. From this group, only women without premenopausal dyslipidemia were recruited to the study (documented lipid profile before menopause).

The gynecological interview and hormonal profile, including the measurement of follicle-stimulating hormone (FSH) levels, confirmed the postmenopausal period. The exclusion criteria were used to eliminate the factors that may increase the risk of oxidative stress and metabolic disturbances. We excluded women with diabetes mellitus, endocrine disorders (e.g., thyroid disorders, Cushing syndrome), cardiovascular or infectious diseases, malignancy, hepatic or renal dysfunction, hypolipidemic treatment, cigarette smoking, and alcohol abuse. We selected a homogenous group of 51 postmenopausal obese women (body mass index [BMI] ≥30 kg/m^2) and a control group of 51 aged-matched postmenopausal healthy lean women (BMI <25kg/m^2). None of the selected women used hypolipidemic medications or hormonal replacement therapy. Data on medical and family history were obtained from patients, and blood was sampled for biochemical measurements. The study protocol was approved by the Bioethical Committee of the Poznan University of Medical Sciences (no. 952/11).

The study group comprised obese postmenopausal women who underwent a complete physical examination including the evaluation of anthropometric parameters (measured to the nearest 0.1 kg [weight] and 0.1 cm [height, waist, and hip circumferences]). Weight and height were determined with subjects in underwear using the SECA scale. Waist circumference was measured at the narrowest level between the costal margin and iliac crest, and the hip circumference was measured at the widest level over the buttocks while the subjects were standing normally. Subjects with BMI values of less than 25.0 kg/m^2 were considered to be lean, and those with the values exceeding 30.0 kg/m^2, to be obese.[1] A bioimpedance analyzer with a single frequency of 50 kHz (Bodystat 1500, Bodystat Ltd., United Kingdom) was used to assess the fat content as a proportion of the total body mass.

Nutritional evaluation During the study, all patients were on normal diet (without any dietary modifications). The food intake was assessed using a 24-hour recall during 7 days.[10] The results of the questionnaire were analyzed with both the quantitative and qualitative analyses of the subjects' daily diets[11] using computer databases for Microsoft Access 2000.[12] The food intake recommendations of the National Institute of Food and Nutrition in Warsaw, Poland, were considered to determine whether the Recommended Dietary Allowances (RDAs) specific for age, sex, ideal body mass, height, and physical activity were fulfilled.[9] The reductions of vitamin intake in the cooking process accepted for this study were 25% for vitamin A, 30% for vitamin E, 55% for vitamin C, and 20% for β-carotene. Dietary fiber consumption was compared with the nutritional prophylaxis recommendations at the level between 20 and 40 g, and cholesterol intake at the level of 200 mg (recommended in dyslipidemia).[1,9]

After 12-hour fasting, venous blood samples were obtained from all patients at 7 a.m. Serum samples were taken from clotted (15 min, room temperature) and centrifuged blood (15 min, 3 000 × *g*). The obtained samples were used for the measurements of FSH levels, plasma lipid profile, MDA and AOPP levels, as well as SOD activity.

The lipid profile (total cholesterol [TC], high-density lipoprotein cholesterol [HDL-C], LDL cholesterol [LDL-C], and triglycerides [TGs]) was determined with enzymatic colorimetric assays (Cobas Integra 400 Plus; Roche Diagnostics, Mannheim, Germany). Serum was separated and directly used for the assay. The serum concentration of LDL-C was calculated using the Friedewald formula.[13]

The serum FSH level was measured to confirm the postmenopausal period by specific chemiluminescence assays (Roche Diagnostics). The serum AOPP concentration was assessed using the spectrophotometric method of Witko-Sarsat et al.[14] as modified by Kalousova et al.[15] In this assay, 200 µl of diluted blood serum (1:5) with 0.1 mol/l of phosphate buffer saline (pH 7.4, PBS) was placed in a microtiter well, and 20 µl of acetic acid (14 mol/l) and 10 µl of potassium iodide (1.16 mol/l) were added. Control wells contained PBS. Chloramine-T solution (0–100 mmol/l) was used for calibration. The absorbance was read at 340 nm on a microplate reader (spectrophotometer, Multiscan LS, Labsystems, Finland). AOPP concentrations were expressed as mmol chloramine T equivalent/l.

The total SOD activity was measured using the Oxis Bioxytech®SOD-525™ Assay (Oxis International, Inc., Portland, Oregon, United States). This method is based on a SOD-mediated increase in the rate of autooxidation of 5,6,6a,11b-tetrahydro-3,9,10-trihydroxybenzo[c]fluorene in an aqueous alkaline solution and detects the fluorescence of solution at 525 nm, based on the principle that the autooxidation of tetracyclic cathechol accelerates in the presence of SOD.

Plasma MDA concentrations were assayed colorimetrically by measuring thiobarbituric acid

reactive substances. The pink chromogen produced by the reaction of thiobarbituric acid with MDA was measured at 530 nm.

The intra- and interassay coefficients of variation were less than 5% for all of the assays performed.

Statistical analysis The data analysis was performed using StatSoft, Inc. STATISTICA for Windows, version 10.0. The Shapiro–Wilk test was used to determine whether continuous variables were normally distributed. The mangnitude of association between continuous variables was calculated by the Pearson correlation coefficient. A *P* value of 0.05 or less was considered significant. The differences in AOPP and MDA concentrations and SOD activity between the study and control groups were assessed using the *t* test for independent groups. The significant correlations of saturated fatty acids (SFA) and selenium with both SOD activity and MDA concentrations were estimated, and the parameters were examined using multivariable regression. To accurately describe the examined relations, an additional variable was included in the model, namely, the TG level. The *F*-test based on the determination coefficient of multiple regression was used to compare the models.

RESULTS The study included 102 postmenopausal women (the mean age after the last menstruation was 2.7 y in the obese group and 4.1 years in the lean group). Obese women had high body mass (>90 kg) and increased BMI (35.0–29.9 kg/m^2), reflecting the second degree of obesity (TABLE 1). Fat mass (measured by the bioimpedance method) showed large differences in the amount of fat tissue ($P = 0.00001$), which was distributed viscerally in obese patients and gynoidally in the lean ones.

The levels of FSH and lipid parameters (TC, LDL-C, and TGs) were higher and those of HDL-C fraction were lower in obese subjects compared with the lean ones (TABLE 1). Moreover, a lipid analysis revealed hypercholesterolemia (LDL-C levels, >3.36 mmol/l) only in obese women. In both groups, plasma TC levels exceeded the level of 4.91 mmol/l recommended by the European Society of Cardiology; however, the levels of HDL-C and TGs and the TC-to-HDL ratio were within the normal ranges.

The levels of AOPPs and MDA were higher, while SOD activity was lower in obese women compared with lean subjects ($P = 0.02$, $P = 0.00001$, and $P = 0.03$, respectively). SOD activity positively correlated with HDL-C levels, and negatively with atherogenic parameters such as TC-to-HDL-C and LDL-to-HDL-C ratios in both groups (TABLES 2 and 3). In obese subjects, a negative correlation between SOD activity and TG levels ($P = 0.004$) and a positive correlation between AOPP and LDL-C levels ($P = 0.05$) were observed (TABLE 3).

The multivariable model showed a significant effect of SFA and selenium intake on the variability of serum SOD activity in obese subjects ($P = 0.003$, $R^2_{adj} = 17\%$) (TABLE 4). To describe this phenomenon more precisely, an additional variable was included in the model, namely, serum TG concentration. To compare the models, the *F* test based on the determination of multiple regression was used, and several significant differences between the models were observed. Adding TG levels to the model allowed to better explain the variation in SOD activity: up to 24% ($P = 0.03$).

In the second multivariable model, a major effect of selenium intake and energy from saturated fatty acids on the variability of the serum MDA level in obese women was observed ($P < 0.000001$, $R^2_{adj} = 45\%$; TABLE 4). Adding TG levels to the model allowed to better explain variations in the MDA levels up to 49% ($P = 0.029$).

The daily energy intake in lean subjects corresponded with the RDA specific for age, sex, ideal body mass, height, and physical activity, while obese women showed a higher intake of energy ($P = 0.03$) and fat ($P = 0.02$) (TABLE 1). Nevertheless, irrespective of the energy intake, the diets of both groups were improperly balanced and characterized by excess protein and fat nutrition. Saturated fatty acids exceeded the recommended value by 8% to 10% of energy; however, in dyslipidemia, this value should be even lower than 7% of the energy intake. The consumption of monounsaturated fatty acids (MUFAs) was within the reference range in both groups, but the amount of protective polyunsaturated fatty acids (PUFAs) was low, and the SFA:MUFA:PUFA ratio did not achieve the value of 1:1.5:1 recommended in the Mediterranean diet. Moreover, the consumption of cholesterol twice exceeded the value suggested in nutritional recommendations.

The supply of carbohydrates was higher in the obese group ($P = 0.03$) and complied with the accepted intake level reaching about 52% of the energy intake (including an adequate amount of saccharose). The average dietary fiber intake was close to the lower limit of the reference range of 20 to 40 g/d in both groups.

The intake of zinc was sufficient and comparable in both groups. The consumption of copper was higher ($P = 0.01$) while that of selenium was lower ($P = 0.04$) in obese women compared with the lean ones. Moreover, in both groups, the selenium intake did not achieve the recommended amount of 55 μg/d. The intake of vitamin A was higher than the recommended 700 μg/d, and that of vitamin C was lower than 75 mg/d (only consumption of vitamin E was adequate and exceeded 8 mg/d). The intake of these vitamins did not differ between the lean and obese groups.

In the constructed multivariable model, saturated fatty acids and selenium intake influenced the SOD activity and MDA concentration.

DISCUSSION After menopause, women are at an increased risk of developing visceral obesity

TABLE 1 Clinical, nutritional, and laboratory characteristics of obese and lean postmenopausal women

Parameter	Obese women (n = 51)	Lean women (n = 51)	*P* value
age, y	56.08 ±4.58	57.35 ±4.60	0.1
height, cm	160.91 ±6.45	161.47 ±5.50	0.6
body mass, kg	91.38 ±12.39	62.18 ±6.05	0.0001
BMI, kg/m^2	35.26 ±4.34	23.44 ±1.65	0.0001
FM, %	49.00 ±4.17	31.77 ±4.16	0.0001
WC, cm	101.83 ±9.60	77.46 ±7.20	0.0001
WHR	0.86 ±0.08	0.80 ±0.07	0.0001
FSH, mIU/ml	53.53 ±18.73	79.98 ±29.02	0.0001
TC, mmo/l/l	5.73 ±0.96	5.37 ±0.55	0.04
LDL-C, mmo/l/l	3.56 ±0.86	3.08 ±0.52	0.001
HDL-C, mmo/l/l	1.40 ±0.30	1.78 ±0.35	0.0001
TG, mmo/l/l	1.50 ±0.57	1.12 ±0.62	0.0001
TC-to-HDL-C ratio	2.51 ±0.83	1.82 ±0.56	0.0001
LDL-to-HDL-C ratio	4.01 ±1.02	3.14 ±0.73	0.0001
AOPP, µmol/l	49.28 ±22.24	38.43 ±16.76	0.02
SOD, U/ml	3.92 ±1.46	5.31 ±4.60	0.03
MDA, µmol/l	3.15 ±1.44	1.71 ±0.87	0.0001
energy, kcal	2004.52 ±577.59	1745.96 ±324.13	0.03
protein, g	77.21 ±23.31	68.85 ±14.19	0.2
animal protein, g	51.34 ±18.34	45.15 ±12.22	0.2
plant protein, g	25.63 ±8.42	23.61 ±5.29	0.3
protein, % energy	15.77 ±3.03	16.04 ±2.45	0.6
fat, g	78.10 ±29.79	63.65 ±17.32	0.02
fat, % energy	34.26 ±5.94	31.94 ±4.89	0.04
SFA, % energy	13.98 ±1.85	10.97 ±2.20	0.0001
MUFA, % energy	13.38 ±3.11	12.36 ±2.59	0.08
PUFA, % energy	5.78 ±2.27	5.65 ±1.70	0.9
dietary cholesterol, mg	421.33 ±164.67	386.81 ±138.31	0.2
carbohydrates, g	256.80 ±75.02	227.12 ±46.19	0.03
carbohydrates, % energy	51.70 ±6.56	52.69 ±5.90	0.3
saccharose, % energy	10.47 ±3.65	10.10 ±3.68	0.5
dietary fiber, g	21.70 ±6.33	20.29 ±4.80	0.3
zinc, mg	10.33 ±2.77	9.53 ±2.02	0.3
copper, mg	1.43 ±0.56	1.21 ±0.27	0.01
selenium, µg	50.05 ±23.49	58.99 ±16.30	0.04
vitamin A, µg	913.89 ±578.35	784.77 ±307.64	0.8
vitamin E, mg	8.80 ±4.06	8.19 ±3.25	0.5
vitamin C, mg	63.47 ±38.27	61.68 ±34.31	0.8

Data are presented as mean ± standard deviation.

Abbreviations: AOPP – advanced oxidation protein product, BMI – body mass index, FM – fat mass, FSH – follicle-stimulating hormone, HDL-C – high--density lipoprotein cholesterol, LDL-C – low-density lipoprotein cholesterol, MDA – malondialdehyde, MUFA – monounsaturated fatty acid, PUFA – polyunsaturated fatty acid, SD – standard deviation, SFA – % energy saturated fatty acid, SOD – superoxide dismutase, TC – total cholesterol, TG – triglyceride, WC – waist circumference, WHR – waist-to-hip ratio

owing to the loss of endogenous ovarian hormone production.[16]

In postmenopausal women, estrogens are produced in the adipose tissue via aromatization, which is enhanced in patients with higher BMI.[17,18] Additionally, increased BMI has an inhibitory effect on gonadotropin secretion, and obese women have lower FSH levels compared with normal--weight individuals.[18] Surprisingly, in our study, obese women had higher FSH levels than lean subjects. This can be explained by the change in the levels of both estradiol and FSH, which vary between women and follow several distinct patterns. A large longitudinal study of the menopausal transition distinguished 4 unique estradiol and 3 unique FSH trajectory groups. One of them was characterized by high FSH levels, which has been associated with a slow decline in estradiol levels.[17] Similarly, Hale et al.[18] reported that in 37% of the Caucasian women with menopausal

TABLE 2 Correlations of lipid profile with the parameters of oxidative stress in lean women (n = 51)

Parameter	AOPP, µmol/l				SOD, U/ml				MDA, µmol/l			
	R	R^2	t	*P* value	*R*	R^2	t	*P* value	*R*	R^2	t	*P* value
TC, mmol/l	0.06	0.003	0.41	0.69	–0.1	0.01	–0.73	0.47	–0.18	0.03	–1.34	0.19
HDL-C, mmol/l	–0.004	0.0000	–0.03	0.98	0.33	0.11	2.61	0.01	–0.09	0.01	–0.64	0.53
TC-to-HDL-C	0.08	0.01	0.61	0.55	–0.34	0.12	–2.71	0.01	–0.03	0.001	–0.2	0.84
LDL-C-to--HDL-C	0.07	0.005	0.54	0.59	–0.36	0.13	–2.88	0.01	–0.02	0.001	–0.16	0.87
TG, mmol/l	–0.1	0.01	–0.73	0.47	–0.21	0.05	–1.6	0.11	0.03	0.001	0.19	0.85
LDL-C, mmol/l	0.04	0.002	0.29	0.77	–0.13	0.02	–0.98	0.33	–0.06	0.004	–0.48	0.64

Abbreviations: see **TABLE 1**

TABLE 3 Correlations of lipid profile with the parameters of oxidative stress in obese women

Parameter	AOPP, µmol/l				SOD, U/ml				MDA, µmol/l			
	R	R^2	t	*P* value	*R*	R^2	t	*P* value	*R*	R^2	t	*P* value
TC, mmol/l	0.18	0.03	1.24	0.22	–0.14	0.02	–0.1	0.32	–0.06	0.004	–0.43	0.67
HDL-C, mmol/l	–0.19	0.04	–1.31	0.2	0.3	0.09	2.22	0.03	–0.17	0.03	–1.18	0.24
TC-to--HDL-C ratio	0.27	0.08	1.95	0.06	–0.3	0.09	–2.23	0.03	0.07	0.004	0.45	0.65
LDL-C-to--HDL-C ratio	0.26	0.07	1.85	0.07	–0.35	0.12	–2.6	0.01	0.11	0.01	0.74	0.46
TG, mmol/l	0.07	0.005	0.47	0.64	–0.39	0.15	–2.99	0.004	0.2	0.04	1.44	0.16
LDL-C, mmol/l	0.29	0.08	2.05	0.05	–0.17	0.03	–1.19	0.24	–0.04	0.002	–0.28	0.78

Abbreviations: see **TABLE 1**

TABLE 4 Comparison of the 2 models explaining variations of superoxide dismutase activity and malondialdehyde levels before and after adding the third variable (triglyceryde) to the basic model, which incorporates/includes? selenium amount and energy from saturated fatty acids in obese women

Dependent variable	Statistical parameters	Model including 2 variables: selenium intake and SFA%en	Extended model with a new variable (TG, mmol/l)	Comparison of analyzed models (*F* value)	Comparison of analyzed models (*P* value)
SOD, U/ml	SEe	1.16	1.11	5.38	0.03
	R^2_{adj}	0.17	0.24		
	F	6.26	6.35		
	P value	0.004	0.001		
MDA, µmol/l	SEe	1.08	1.03	5.05	0.03
	R^2_{adj}	0.45	0.49		
	F	21.11	16.93		
	P value	<0.000001	<0.000001		

Abbreviations: SEe — standard error of the estimate (the lower the better fitting model), others – see **TABLE 1**

transition, serum estradiol levels were often elevated and associated with high FSH levels. High estradiol levels with the onset of the menopausal transition could be related to a decrease in the number of ovarian follicles and the resultant impairment of a negative feedback causing elevation in FSH levels.[18,19] Another possibility is that the hypothalamic–pituitary–adrenal axis becomes less sensitive to estrogen during perimenopause.[19]

Not only the cessation of estrogen production but also the aging processes and changes in lifestyle (tendency to physical inactivity) increase the reactive oxygen species formation and lipid disorders.[20] Thus, elevated body mass as well as high BMI and WHR observed in the obese group in our study are risk factors for coronary heart diseases, stroke, and enhanced oxidative stress.[1,21]

The nutritional analysis showed that irrespective of the energy intake, the diets of both groups were poorly balanced and were characterized by

protein and fat overnutrition. High intake of protein was also observed in other studies performed in the European and American populations.[22,23] Based on the previous data, an average amount of protein should not exceed 10% to 15% of the total energy intake because its prolonged intake increases oxidation, correlates positively with insulin resistance, and may cause an increase in amino acid catabolism.[9,10,24] Excess protein is not efficiently used by the body and activates the mitochondrial redox chain, contributing to enhanced oxygen radical production.[22] Moreover, a prolonged high protein intake causes renal damage and calciuria (an increased elimination of calcium with urine), leading to a reduction of bone density, which might be deleterious in postmenopausal age.[23]

A significantly higher intake of total fat and saturated fatty acids in obese women stimulates ROS production that are jointly upregulated in the liver and adipose tissue.[9] If the fat supply is prolonged, the oxidative processes are increased and insulin resistance is initiated. Furthermore, a high intake of cholesterol and low supply of PUFAs may increase the cardiovascular risk independently of plasma cholesterol levels.[3,9,25-27] The Dietary Guidelines of the American Heart Association suggest that cholesterol intake should not exceed 200 mg/d for individuals with elevated cardiovascular risk factors such as high LDL-C levels, diabetes, or ischemic heart disease.[25,26] Food products rich in cholesterol contain high amounts of SFA that enhance the synergistic effect of cholesterol on blood lipid fractions.[1]

In this study, a low supply of dietary fiber was observed in both groups. Dietary fiber increases the sensation of satiety that helps control calorie intake and has an advantageous effect on blood glucose levels. Moreover, in dyslipidemia, soluble fiber intake such as β-glucans and pectins modestly reduce plasma TC and LDL-C levels.[1,2,9]

To diminish the risk of oxidative processes, an adequate amount of antioxidative components should be supplied with the diet. The proper intake of zinc is beneficial because this element has an impact on the oxidative status and lipid metabolism.[28] The higher intake of copper and the inefficient supply of selenium predispose to oxidative stress and metabolic disorders, especially in visceral obesity.[29] Fortunately, the intake of oxidative vitamins assessed in both group was satisfactory, and thus beneficial in reducing oxidative stress.

Newly diagnosed hypercholesterolemia and the elevated LDL-C-to-HDL-C ratio were observed in obese women. The elevated LDL-C level in postmenopausal age is associated with atherosclerosis and high risk of cardiovascular diseases.[10,26,30] Moreover, in this group, the LDL-C-to-HDL-C ratio (the most effective measure of cardiovascular risk in elderly people) exceeded the recommended value of 3.3.[25,31]

Menopause is linked to oxidative stress and a decreased antioxidant defense, which is partially related to aging.[3,5,6] It is associated with a significant change in the antioxidant gene expression, which in turn affects the circulating redox state. Estrogens act as regulators of the key antioxidant gene expression.[16] A number of studies have underlined an imbalance between the oxidant and antioxidant systems in hyperlipidemia.[21] However, there are no data about the postmenopausal early changes of the lipid profile in obese women not receiving hypolipidemic treatment, or the relation of lipid and oxidative stress parameters with the diet. In this study, the AOPP level in obese women was 1.3-fold higher than that in the lean group. High serum AOPP levels are observed in coronary artery diseases, suggesting that an accumulation of AOPPs may be a relevant marker of the atherosclerotic process.[7,32] In clinical studies, increased AOPP levels are highly correlated with the carotid intima–media thickness and may increase the prevalence of cardiovascular events.[32] In this study, the AOPP concentration was positively correlated with LDL (R^2 = 0.08, P = 0.05) in obese women, which suggests that proatherogenic changes of the lipid profile are linked to increased oxidative stress. A similar positive correlation between the AOPP level and atherogenic lipid shifts has been observed in dyslipidemic patients with renal disease.[33]

After the release of ROS, many defense mechanisms are activated and SOD starts to neutralize the effect of superoxide anions.[8] In the current study, the SOD activity in obese women was lower compared with that in the lean group (P = 0.03). Low serum SOD activity in obese patients may be either due to an increased lipid peroxidation in obesity or an elevated production of H_2O_2, which is known to suppress the SOD activity.[34,35] Postmenopausal hypoestrogenism also affects SOD activation, and the activity of this enzyme is decreased in peri- and postmenopausal women with cardiovascular diseases in comparison with the premenopausal stage.[4-6] Also, a diet rich in proatherogenic lipids (e.g., dietary cholesterol or saturated fatty acids) reveals the pro-oxidant effect and change in the SOD activity.[36] In this study, the SOD activity was positively correlated with the HDL fraction, which has a cardioprotective effect; however, it was inversely correlated with proatherogenic parameters such as TC-to-HDL and LDL-to-HDL ratios in both groups. Thus, oxidative stress is related to lipid profile changes as well as to nutritional habits. Excess of saturated fatty acids and insufficient levels of selenium affect the variability of the serum SOD activity in obese subjects in the constructed multivariable model.

A negative correlation of SOD activity with TGs was observed in the obese group. The variation in the SOD activity (an increase from 17% to 24%) could be explained better when TG levels were added to the multivariable model. In conclusion, the SOD activity was linked not only with proatherogenic dietary factors (such as excess

of SFA and insufficient amount of selenium) but also with TG levels.

Our study confirmed that MDA concentrations are increased during oxidative stress in obese postmenopausal women.[37] We also observed the effect of selenium intake and saturated fatty acids on the variability of serum MDA levels. The variation in MDA levels could be explained better when TG levels were added to the multivariable model. Thus, nutritional factors as well as lipid profile changes influence oxidative stress. The modification of dietary habits, such as reduction of fat (mainly saturated fatty acids) and increased intake of dietary antioxidants (such as selenium) may improve the serum lipid profile and diminish oxidative process in postmenopausal women.

Conclusions Our study showed that oxidative stress processes occur at the early stages of dyslipidemia in obese postmenopausal women (increased AOPP and MDA levels and decreased SOD activity). An improperly balanced diet is one of the risk factors of oxidative stress. We observed a significant effect of SFA and selenium intake on the variability of the serum SOD activity and MDA concentration in obese subjects, which also depended on TG levels. Thus, an early diagnosis of dyslipidemia and changes of dietary habits (including an adequate intake of fat and antioxidants) to prevent oxidative stress should be a high priority in postmenopausal age.

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ARTYKUŁ ORYGINALNY

Zwyczaje żywieniowe i stres oksydacyjny w wieku pomenopauzalnym

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dyslipidemia, pomenopauzalna otyłość, zwyczaje żywieniowe, stres oksydacyjny

STRESZCZENIE

WPROWADZENIE Otyłość pomenopauzalna zwiększa ryzyko wystąpienia stresu oksydacyjnego, ale takie powiązanie u osób z nowo rozpoznaną dyslipidemią po menopauzie wymaga dokładnych badań.

CELE Celem badania była ocena zależności pomiędzy procesami oksydacyjnymi, nowo rozpoznaną dyslipidemią oraz sposobem żywienia w grupie pomenopauzalnych kobiet, które nie stosowały leczenia hipolipemizującego.

PACJENCI I METODY Oceniono stan odżywienia, sposób żywienia oraz parametry stresu oksydacyjnego u 102 kobiet po menopauzie (51 otyłych oraz 51 z prawidłową masą ciała), u których nie występowały zaburzenia lipidowe przed menopauzą.

WYNIKI U kobiet otyłych stwierdzono hipercholesterolemię, wieksz stężenia dialdehydu malonowego i zaawansowanych produktów utleniania białek (*advanced oxidation protein products* – AOPP) oraz dodatnią korelację pomiędzy AOPP i frakcją cholesterolu lipoprotein o małej gęstości (*low-density lipoprotein* – LDL). Aktywność dysmutazy ponadtlenkowej w osoczu (*superoxide dismutase* – SOD) dodatnio korelowała z cholesterolem lipoprotein o dużej gęstości (*high-density lipoprotein* – HDL) oraz ujemnie ze wskaźnikami – stosunkiem cholesterolu całkowitego (*total cholesterol* – TC) / HDL i LDL/HDL w obu grupach. U otyłych kobiet całodzienne racje pokarmowe charakteryzowały się większą ilością miedzi oraz energii pochodzącej z tłuszczów i nasyconych kwasów tłuszczowych (*saturated fatty acids* – SFA), natomiast podaż węglowodanów i selenu była mniejsza w porównaniu do kobiet z prawidłową masą ciała ($p < 0,05$). Modele wielowymiarowe wykazały istotny wpływ SFA i selenu na zmienność aktywności SOD w surowicy ($p = 0,003$; $R^2_{adj} = 17\%$) oraz stężenie dialdehydu malonowego ($p = 0,00001$, $R^2_{adj} = 45\%$) u kobiet otyłych (modele te różniły się statystycznie istotnie).

WNIOSKI Badanie wykazało, że procesy stresu oksydacyjnego są obecne na wczesnych etapach rozwoju hipercholesterolemii u otyłych kobiet po menopauzie i mogą wynikać z nieprawidłowo zbilansowanej diety.

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