Correlation of Body Mass Index and Waist Hip Ratio with lipid and hormone profile in women in menopausal transition

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ABSTRACT

Introduction: Menopausal transition is a period characterised by psychic, somatic changes as well as changes in reproductive capabilities of a woman. It occurs as a consequence of ovarian’s function termination, and pertains to the periods of different meanings: perimenopause, menopause and postmenopause. Although there are numerous assessments of behaviour of the lipids and lipoproteins during menopausal transition, their relation to sexual hormones and body mass is still being assessed. The aim of this study is to determine the differences and connections between body mass index (BMI) and waist-hip ratio (WHR) and lipid and hormone profile among the assesses in premenopause, perimenopause and postmenopause.

Methods: The assessment was done on 150 assesses divided in three groups of 50, such as: premenopause, perimenopause and postmenopause. The assessment included the following: interview, determination of BMI, WHR, and taking of blood sample and processing of hormone, lipid and lipoprotein concentration.

Results: Based on the obtained results, it may not be concluded that BMI has a positive correlation with cholesterol and VLDL concentration in postmenopause, positive correlation with apo A in perimenopause and postmenopause, and positive correlation with Lp (a) and apo B in premenopause and perimenopause, while negative correlation with HDL and estradiol concentrations in premenopause. WHR has negative correlation with HDL concentration in premenopause and perimenopause, and a negative correlation with estradiol concentrations in premenopause.

Keywords: menopause, lipids, hormones, body mass index, waist-hip ratio

INTRODUCTION

Menopausal transition is a period in life of a woman, with undefined beginning and duration, but with well-known changes in female organism. Menopausal transition is a period characterized by psychic, somatic and reproductive changes of capabilities at
women (1). Hormonal profile is changing during menopausal transition. Increased level of follicle-stimulating hormone (FSH) stimulates follicles to grow, but those follicles mainly fail to reach the final growth and maturity, which results with frequent anovulation. Progesterone production is 60% lower than in reproduction period. In menopause, metabolic changes occur in different tissues and organs as a result of changed hormonal profile (2). Fat tissue is not only a passive fat depot reflecting the energy balance and thermo-regulation, but is also a significant endocrine organ (3). The main source of estrogen in menopause is estron generated from androstendion in peripheral tissues. Conversion processes of androgen to estrogens in menopause are not performed in fat tissue only, but also in central nervous system (4, 5). One of the most important fat tissue hormones is leptin. During menopause, not only leptin, but also decreased level of growth hormone, E2 and androgens lead to changes of lipogenesis and lipolysis mechanisms, which lead to characteristic distribution of fat tissue in menopause (centripetal obesity). Lipid profile during menopausal transition changes, but all mechanisms of those changes are not clarified. One of the most important factors in that mechanism is the change of fat tissue distribution in postmenopause. Total cholesterol level increases through menopausal transition, with highest values in menopause. LDL increases during menopausal transition, but it decreases after the menopause. The age is significantly related to the changes in the triglyceride, total cholesterol and LDL level, while body mass index (BMI) is significantly related to the changes of level of triglyceride, LDL, HDL (6). With menopause, HDL concentration decreases and HDL structure changes. HDL concentration is inversely proportional to abdominal obesity level (7). Menopause, age and increased distribution of abdominal fat tissue are three independent and important factors violating the lipoprotein profile from the beginning of menopausal transition (8). Age, BMI and menopausal status are significant indicators of the circulating lipoprotein level during menopausal transition (9). BMI is used to calculate the body surface and estimate the body weight. Doctors use BMI to estimate the risk of body weight and cardiac disease. New analysis of 40 studies, published in the Lancet magazine (10), which includes 250 000 patients, shows that the patients with BMI below 20 have higher mortality risk caused by cardiovascular diseases than the obese patients with BMI 30-35. The assesses who smoke have somewhat lower total cholesterol and significantly lower HDL, although there is no difference in BMI distribution between the assesses who smoke and those who do not (11). E2 level is much lower at the obese, premenopausal women than at the premenopausal women of normal body weight. FSH level is also lower at the obese postmenopausal women than at the non-obese postmenopausal women (12). BMI impact to E2 and FSH varies in dependence on menopausal profile (13). Waist-hip ratio, WHR is statistically important predictor of LDL concentration and cholesterol in relation to BMI at women in menopause, which confirms the importance of fat tissue distribution as a risk factor for cardiovascular diseases at this group of women (14). Sultan and associates (15) state that waist-hip ratio may be used as screening for identification of postmenopausal women with higher cardiovascular risk.

The aim of this study was to determine the differences and connection of BMI and WHR with lipid and hormonal profile of women in premenopause, perimenopause and postmenopause.

METHODS

This prospective, comparative study was conducted in period November 2009 – December 2010 and it included a total of 150 assesses aged 40-55, divided into three groups of 50 assesses each (premenopause, perimenopause and postmenopause). The assesses comprised women volunteers who agreed in writing to be included in the study. Each assessee was introduced with the character of the study implemented according to the generally accepted ethical standards for medical research. Qualification criteria for inclusion into the study were: that the candidates do not take hormonal supplemental therapy, that they do not take medicines which could affect the lipid profile, that they do not consume more than twenty cigarettes a day, that their BMI does not exceed 35kg/m². Processing of assesses comprised three phases: interview, taking blood sample, measuring BMI and WHR. For the purpose of analysis, the vein blood sample from cubital vein
was taken, after which the blood was centrifuged, and obtained serum divided into two test tubes. The same day, concentration of lipid, lipoprotein, FSH, LH and E2 was determined. Concentration of the total cholesterol, triglyceride, LDL, HDL, VLDL, apo A, apo B and Lp (a) was also determined. Total cholesterol and triglyceride concentration was determined using enzymatic method at SIEMENS «Dimension RxL» machine. HDL and LDL were determined at the same machine utilizing direct „homogenous“ method. VLDL was calculated with formula: VLDL = total cholesterol - HDL – LDL apo A, apo B and Lp (a) values were determined using immunoturbidimetry method at SIEMENS «Dimension RxL» machine, and reagents of the company „SENTINEL“ were used. FSH, LH and E2 values were determined utilizing the hemiluminescence method. The assesses' body weight and height were measured at the „SECA“ scale with meter. Fattening status was assessed based on the Que- telet index (Devenport-Kaup modification) or BMI where: BMI= body mass in kg/height in m². Based on the waist measuring at the narrowest place and hip measuring at the widest place, WHR was calculated according to the following formula: WH ratio = waist (cm)/hip (cm).

Statistical analysis
To compare the numerical variables among the assesse groups we used the variance analysis (ANOVA) or Kruskal-Wallis non-parametric alternative. To analyse relation of BMI, WHR, menarche and reproductive age factors with lipid and hormonal profile, we used linear regression model. Each factor was tested in bivariate model, adjusted to the group of assesses. Results are presented at regression coefficient with related 95% reliability interval (IP). Statistical significance was confirmed at p<0.05. Statistic programme PASW 18 (SPSS Inc., Chicago, Illinois, USA) was used for data processing.

RESULTS
The assesses included in the study were aged 40 to 55, with average age 48.1 ± 3.9. Statistically, the age between the groups differed significantly: the assesses in perimenopause were 2 years older in average than those in premenopause (95% IP: 0.6-3.5 years), and the assesses in postmenopause were four years older in average than those in perimenopause (95% IP: 2.6-5.4 years) (Table 1). In addition, the assesses from the second group had somewhat lower WHR than the assesses from the third group, although the difference was not statistically significant (average difference = 0.03, 95% IP: -0.003-0.06). There is a significant negative relation between BMI and HDL at assesses in premenopause (Table 2). If the regression coefficient had been standardised to interquartile value of BMI change (IQR=5), the difference of 0.18 mmol/L in medium HDL concentration between low and high BMI would have been suggested. Among the assesses in postmenopause, there is a significant relation between BMI and cholesterol, as well as between BMI and VLDL. For interquartile value of BMI change, standard regression coefficient shows the increase of 0.67 mmol/L in medium cholesterol concentration and increase of 0.22 mmol/L in medium VLDL concentration. There are no proofs of any relation between other lipid and BMI parameters. Multivariate regression analysis showed that the only important lipid fraction affected by BMI is HDL cholesterol. Considering the BMI value in relation to apo A, results indicate that there is a correlation between the groups of assesses in perimenopause and post-

<table>
<thead>
<tr>
<th>Table 1. Main features of assesseses¹</th>
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<tbody>
<tr>
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<tr>
<td>Group I (n=50)</td>
</tr>
<tr>
<td>Age 45.4 ± 3.2 (40-52)</td>
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<tr>
<td>Menarche, age 14.2 ± 1.6 (11-18)</td>
</tr>
<tr>
<td>Age at last menstruation</td>
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<td>Reproductive age</td>
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<tr>
<td>BMI (kg/m²) 26.3 ± 3.7 (20.0-36.0)</td>
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<tr>
<td>WHR 0.81 ± 0.05 (0.70-0.97)</td>
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</table>

¹Values are arithmetic median value ± SD as well as minimum and maximum. Group I = assesseses in premenopause; group II= assesseses in perimenopause; group III= assesseses in postmenopause.
### TABLE 2. Regression coefficient for lipid profile in relation to BMI.

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
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<tr>
<td></td>
<td>Regression coefficient</td>
<td>95% IP</td>
<td>P value</td>
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<td>95% IP</td>
<td>P value</td>
<td>Regression coefficient</td>
<td>95% IP</td>
<td>P value</td>
</tr>
<tr>
<td>Cholesterol, mmol/L</td>
<td>-0.029</td>
<td>-0.104, 0.047</td>
<td>0.445</td>
<td>-0.050</td>
<td>-0.135, 0.034</td>
<td>0.236</td>
<td>0.133</td>
<td>0.032, 0.234</td>
<td>0.011</td>
</tr>
<tr>
<td>Triglyceride, % increase to BMI unit</td>
<td>3.0</td>
<td>-1.1, 7.2</td>
<td>0.146</td>
<td>2.3</td>
<td>-1.6, 6.4</td>
<td>0.232</td>
<td>0.0</td>
<td>-2.9, 2.8</td>
<td>0.935</td>
</tr>
<tr>
<td>HDL, mmol/L</td>
<td>-0.035</td>
<td>-0.057, -0.012</td>
<td>0.004</td>
<td>-0.018</td>
<td>-0.042, 0.006</td>
<td>0.143</td>
<td>0.020</td>
<td>-0.009, 0.050</td>
<td>0.177</td>
</tr>
<tr>
<td>LDL, mmol/L</td>
<td>-0.006</td>
<td>-0.065, 0.052</td>
<td>0.834</td>
<td>-0.048</td>
<td>-0.116, 0.019</td>
<td>0.158</td>
<td>0.070</td>
<td>-0.015, 0.154</td>
<td>0.103</td>
</tr>
<tr>
<td>VLDL, mmol/L</td>
<td>0.012</td>
<td>-0.022, 0.046</td>
<td>0.482</td>
<td>0.016</td>
<td>-0.025, 0.056</td>
<td>0.436</td>
<td>0.043</td>
<td>0.005, 0.080</td>
<td>0.027</td>
</tr>
<tr>
<td>Apo A, g/L</td>
<td>-0.019</td>
<td>-0.048, 0.010</td>
<td>0.191</td>
<td>0.017</td>
<td>-0.014, 0.048</td>
<td>0.283</td>
<td>-0.004</td>
<td>-0.027, 0.020</td>
<td>0.747</td>
</tr>
<tr>
<td>Apo B, g/L</td>
<td>-0.010</td>
<td>-0.034, 0.015</td>
<td>0.426</td>
<td>-0.013</td>
<td>-0.033, 0.006</td>
<td>0.170</td>
<td>-0.011</td>
<td>-0.038, 0.016</td>
<td>0.432</td>
</tr>
<tr>
<td>Lp (a), % decrease to BMI unit</td>
<td>3.5</td>
<td>-4.3, 11.7</td>
<td>0.376</td>
<td>-0.9</td>
<td>-11.2, 8.2</td>
<td>0.833</td>
<td>0.5</td>
<td>-9.8, 11.7</td>
<td>0.948</td>
</tr>
</tbody>
</table>

*Values are arithmetic median value ± SD as well as minimum and maximum. Group I = assesses in premenopause; group II = assesses in perimenopause; group III = assesses in postmenopause.

### TABLE 3. Regression coefficient for hormonal profile in relation to BMI.

<table>
<thead>
<tr>
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<th></th>
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<td>95% IP</td>
<td>P value</td>
</tr>
<tr>
<td>FSH, IU/L</td>
<td>0.602</td>
<td>-0.768, 1.972</td>
<td>0.381</td>
<td>-0.634</td>
<td>-2.383, 1.116</td>
<td>0.470</td>
<td>0.354</td>
<td>-2.344, 3.053</td>
<td>0.793</td>
</tr>
<tr>
<td>LH, IU/L</td>
<td>0.557</td>
<td>-0.383, 1.498</td>
<td>0.239</td>
<td>-0.658</td>
<td>-1.988, 0.673</td>
<td>0.325</td>
<td>0.165</td>
<td>-1.027, 1.358</td>
<td>0.781</td>
</tr>
<tr>
<td>Estradiol, % decrease to BMI unit</td>
<td>8.9</td>
<td>2.8, 15.3</td>
<td>0.005</td>
<td>-3.3</td>
<td>-10.7, 3.6</td>
<td>0.358</td>
<td>3.8</td>
<td>-4.9, 13.2</td>
<td>0.399</td>
</tr>
</tbody>
</table>

Group I = assesses in premenopause; group II = assesses in perimenopause; group III = assesses in postmenopause.
**TABLE 4.** Regression coefficient for lipid profile in relation to WHR, calculated for WHR change of 0.05.

<table>
<thead>
<tr>
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</tr>
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<tbody>
<tr>
<td></td>
<td>Regression</td>
<td>95% IP</td>
<td>Regression</td>
</tr>
<tr>
<td></td>
<td>coefficient</td>
<td>Pvalue</td>
<td>coefficient</td>
</tr>
<tr>
<td>Cholesterol, mmol/L</td>
<td>0.061, -0.204, 0.327</td>
<td>0.645</td>
<td>-0.041, -0.255, 0.173</td>
</tr>
<tr>
<td>Triglyceride, increase coefficient to WHR increase for 0.05</td>
<td>1.13, 0.99, 1.30</td>
<td>0.070, 1.06, 0.96, 1.17</td>
<td>0.232</td>
</tr>
<tr>
<td>HDL, mmol/L</td>
<td>-0.105, -0.186, -0.023</td>
<td>0.013, -0.089, -0.144, -0.034</td>
<td>0.002, 0.17, -0.069, 0.102</td>
</tr>
<tr>
<td>LDL, mmol/L</td>
<td>0.074, -0.130, 0.279</td>
<td>0.468, 0.022, -0.152, 0.195</td>
<td>0.803</td>
</tr>
<tr>
<td>VLDL, mmol/L</td>
<td>0.091, -0.024, 0.207</td>
<td>0.119, 0.026, -0.075, 0.127</td>
<td>0.610</td>
</tr>
<tr>
<td>Apo A, g/L</td>
<td>-0.027, -0.131, 0.076</td>
<td>0.595, -0.040, -0.117, 0.037</td>
<td>0.304, -0.028, -0.094, 0.039</td>
</tr>
<tr>
<td>Apo B, g/L</td>
<td>-0.004, -0.089, 0.082</td>
<td>0.929, 0.006, -0.043, 0.055</td>
<td>0.812, 0.023, -0.053, 0.100</td>
</tr>
<tr>
<td>Lp (a), decrease coefficient to WHR increase for 0.05</td>
<td>1.10, 0.84, 1.44</td>
<td>0.474, 1.07, 0.85, 1.36</td>
<td>0.549, 1.05, 0.78, 1.15</td>
</tr>
</tbody>
</table>

Group I = assesses in premenopause; group II = assesses in perimenopause; group III = assesses in postmenopause.

**TABLE 5.** Regression coefficient for hormonal profile in relation to WHR.

<table>
<thead>
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<td>95% IP</td>
<td>Regression</td>
</tr>
<tr>
<td></td>
<td>coefficient</td>
<td>Pvalue</td>
<td>coefficient</td>
</tr>
<tr>
<td>FSH, IU/L</td>
<td>1.388, -3.436, 6.212</td>
<td>0.566, -3.948, -8.104, 0.408</td>
<td>0.075, 3.889, -3.653, 11.431</td>
</tr>
<tr>
<td>LH, IU/L</td>
<td>0.454, -2.888, 3.796</td>
<td>0.786, -2.018, -5.328, 1.292</td>
<td>0.226, 2.005, -1.315, 5.324</td>
</tr>
<tr>
<td>Estradiol, decrease coefficient to WHR increase for 0.05</td>
<td>1.29, 1.05, 1.59</td>
<td>0.019, 0.93, 0.91, 1.29</td>
<td>0.376, 1.13, 0.88, 1.44</td>
</tr>
</tbody>
</table>

Group I = assesses in premenopause; group II = assesses in perimenopause; group III = assesses in postmenopause.
menopause according to the first significance order (p<0.001), while between the assesses in premeno-
pause and postmenopause there is no significant difference (p=0.516). Correlating BMI value in
relation to apo B, there was statistically significant difference between the assesses in perimenopause
and premenopause (p=0.003), while between the as-

seses in postmenopause and premenopause there
was no statistically significant difference (p=0.367).

Obtained results regarding Lp (a) indicate the sig-
nificant correlation in relation to BMI, only among
the assesses in postmenopause and premenopause
(p<0.001). Of three analysed hormones, significant
relation with BMI is noted only for E2 at women
in premenopause (Table 3). For increase of unit in
BMI, regression coefficient suggests 8.9-percent de-
crease in medium E2 concentration. There is a sig-
nificant negative relation between WHR and HDL
at the assesses in premenopause and perimeno-
pause (Table 4). At WHR change of 0.05, regres-
sion coefficient shows the difference of 0.11 mmol/L
in medium HDL concentration at the assesses in
premenopause, and the difference of 0.09 mmol/L
in medium HDL concentration at the assesses in
perimenopause. The only significant relation be-
tween WHR and hormone parameters is proven for
E2 at the assesses in premenopause (Table 5). Me-
dium estradiol concentration decreased 1.29 times
at WHR increase for 0.05.

DISCUSSION

Numerous studies addressed the issue of lipid chang-
es during menopausal transition or related to meno-
pausal changes of endogenous hormones. Although
the numerous researches have been done on behav-
iour of lipids and lipoprotein during menopausal
transition, their relation with sexual hormones and
body mass is still being assessed. The average age in
time of the last menstruation among the assesses
included in this study is 48. One of the objectives of
this study was to determine the relation of BMI and
WHR with lipid and hormonal profile of assesses
in menopausal transition, and the results show that
at the assesses in postmenopause, there is a posi-
tive correlation between BMI and total cholesterol,
and BMI and VLDL cholesterol, and negative cor-
relation between BMI and HDL in premenopause.

There are no proofs of any relation between other lip-
id and BMI parameters. Other studies also indicate
that women with higher BMI are exposed to the risk
of higher lipid level, although skinner women can
also have higher hormone-related LDL cholesterol
during menopausal transition (16). Duration of
postmenopause, as well as BMI in similar studies do
not show significant correlation with lipid, lipopro-
tein and Lp (a) concentration, while WHR shows
significant positive correlation with cholesterol,
LDL and apo B (14) concentrations. According to
Yamamoto and associates (11), there is no significant
correlation between BMI and serum Lp (a) value,
and medium Lp (a) value shows the possible trend
of increase at women over 40. This study showed
significant correlation between BMI and Lp (a) at
the assesses in postmenopause and premenopause,
which is confirmed by contemporary knowledge on
impact of higher Lp (a) to the increase of cardio-
vascular risk. We determined a significant negative
relation between WHR and HDL cholesterol at
the assesses in premenopause and perimenopause,
while in relation to other lipids, there were no cor-
relations. According to Mešalić (14), among women
in perimenopause, WHR has a significant negative
correlation with HDL and Apo lipoprotein A con-
centration, which, considering the role of these two
lipoproteins in occurrence of cardiovascular diseases,
confirms that even women with regular menstrua-
tions and higher WHR have the risk of cardiovas-
cular diseases. Results of this study indicate nega-
tive correlation between WHR and estradiol at the
asseses in premenopause and negative correlation
between BMI and estradiol in premenopause. How-
ever, with increase of WHR and BMI, the estrogen
level decreases in premenopause, based on which
the women with higher cardiovascular risk may be
identified. Assessment results of the comprehensive
study conducted by the National Health and Nutri-
tion Examination Survey among the assesses aged
35-60 in period 1999-2002 (17) show that there are
no significant differences in total cholesterol, tri-
glyceride, HDL, LDL cholesterol levels adjusted to
the age, among menopausal periods at the group of
women with normal BMI. The difference in HDL
cholesterol values was noticed at the groups with
normal and higher BMI. In the groups of assesses
with normal BMI, LH and FSH hormones activ-
ity was statistically different than at the assesses in premenopause and perimenopause, and between the assesses in premenopause and postmenopause (17). SO, according to Azizi and Ainy (18) BMI, WHR and triglyceride value in blood does not show the significant differences between the assesses in premenopause, perimenopause and postmenopause. Despite numerous studies, physiological role of Lp (a) has not been determined. Its atrogenous influence is attributed to enormous similarity of Lp(a) and LDL in structure, which are very cholesterol rich. In this study, Lp (a) was the highest at the assesses in premenopause and postmenopause. Somewhat different results are presented by Kim and associates (19), who obtained lower Lp (a) values at perimenopausal assesses than at postmenopausal assesses.

**CONCLUSION**

BMI has a positive correlation with cholesterol concentration and VLDL/DLD in postmenopause, positive correlation with apo A in perimenopause and postmenopause, and positive correlation with Lp (a) and apo B in premenopause and perimenopause, while with HDL and estradiol concentration it has a negative correlation in premenopause.

WHR has negative correlation with HDL concentration in premenopause and perimenopause, and negative correlation with estradiol concentrations in premenopause. However, with increase of WHR and BMI, the estrogen level in premenopause decreases, based on which the women with higher cardio-vascular risks may be identified.

**COMPETING INTERESTS**

The author declares no conflict of interest.

**REFERENCES**


