UDC 618.11-006-031.13-616-056.52: 616.1

Age and Body Mass Related Changes of Cardiovascular Risk Factors in Women With Polycystic Ovary Syndrome

Đuro Macut*†, Dragan Micić*†, Biljana Parapid†, Goran Cvijović*, Mirjana Šumarac*†, Aleksandra Kendereški*†, Nataša Milić‡, Lidija Tulić†, Azra Muharemagić†, Svetlana Zorić*, Danica Pejković*

Clinical Center of Serbia, *Institute of Endocrinology, Diabetes and Diseases of Metabolism, **Belgrade, University of Belgrade Medical School†,** Institute of Medical Statistics‡, **Belgrade**

Polycystic ovary syndrome (PCOS) is considered a metabolic disorder closely related to obesity, insulin resistance (IR), hyperinsulinemia and unfavorable lipid profile, all increasing the risk for the occurrence of cardiovascular diseases. The aim of this study was to assess age and body mass index (BMI) related changes of cardiovascular risk factors in 90 women with PCOS. The cut-off age point was 30 years and for BMI 27.8 kg/m^2. In all patients systolic and diastolic blood pressure (BP), metabolic parameters comprising values of glucose and insulin during oral glucose tolerance test (OGTT), and basal lipid values were determined. Significant increase in blood pressure (BP) indices, basal insulin values and insulin resistance (IR) assessed by HOMA model were observed with aging and the increase of BMI, while the parameters of glucose metabolism, total cholesterol and triglycerides were significantly elevated only with aging. However, the correlation between the indices of arterial blood pressure, and lipid and glucose metabolism parameters occurred only in patients over 30 years of age, pointing to the causative relation and the consequent deterioration of IR and lipid profile with aging, influencing cardiovascular function in women with PCOS.

K e y w o r d s : **polycystic ovary syndrome; cardiovascular diseases; risk factors; age factors; body mass index; insulin resistance; lipids; blood pressure; glucose tolerance test.**

Introduction

Polycystic ovary syndrome (PCOS) in women of reproductive age is defined as hyperandrogenism with chronic anovulation in the absence of adrenal or pituitary disorder. For years, following the first description of Stein and Leventhal in 1935, PCOS had been diagnosed on the basis of clinical manifestation featuring obesity, hirsutism, amenorrhea and enlarged ovaries (1). Nowadays, in diagnosing PCOS we mostly rely on far more sensitive diagnostic methods: increased levels of luteinizing hormone (LH), ratio LH:FSH (follicle stimulating hormone), free and bound testosterone, androstenedion or dehydroepiandrosterone - sulfate (DHEA-S) and sex hormone binding globulin (SHBG).

Today, PCOS is considered a metabolic disorder closely related to obesity, insulin resistance and hyperinsulinemia. The severe insulin resistance is associated with the alterations in β-cell function. Moreover, the prevalence of the impaired glucose tolerance and diabetes is increased in PCOS (2). It is postulated that the disorder begins at menarche, and its characteristics do not change with aging (1). Females with PCOS below 18 years of age have comparable clinical, neuroendocrine and ultrasonographic features as the adult women with PCOS (3). It was demonstrated that the adolescents with PCOS were severely insulin resistant (4) and that the adolescent PCOS group could represent a cohort with a potential of developing cardiovascular diseases in the adulthood due to the aggravation of dyslipidemia and fibrinolysis with aging (5, 6).

Studies of the lipid profiles in adult women with PCOS had shown the increase in total cholesterol, triglycerides and LDL-cholesterol decrease in Apo-A1 and HDL-cholesterol levels (7, 8). Seemingly, the same pattern of changes within the lipid family had occurred in the adolescent patients with PCOS (6). These findings increased the overall risk for cardiovascular diseases and were positively correlated with the increased occurence of coronary artery disease (7, 9). Angiographic studies of coronary arteries in women discovered more significant atherosclerotic plaques in the subgroup of hyperandrogenic women, while women with PCOS were found to have a seven-fold increased risk for the acute myocardial infarction (AMI) (10, 11). Furthermore, the relationship between elevated plasma insulin levels and hypertension was shown (9).

The aim of our study was to assess glucose and lipid metabolism according to age and body mass index (BMI), and to determine possible associations between the investigated cardiovascular risk factors in women with PCOS.

Methods

This study was conducted at the Institute of Endocrinology, Diabetes and Diseases of Metabolism of the Clinical Center of Serbia, affiliated to the Belgrade University School of Medicine. In all the patients PCOS was diagnosed according to the standard criteria: hyperandrogenism, oligoamenorrhea and ultrasonographic findings of polycystic ovaries (1). Three months prior to the investigation patients had normal fasting glucose and did not take any medication. They were investigated in the follicular phase of the menstrual cycle, or randomly, if amenorrhoeic.

Within the anthropometric research patients' body mass index (BMI) and indices of arterial blood pressure (BP) - systolic and diastolic (mmHg) were assessed. Respective weights and heights [BMI: weight(kg)/height(m)^2] were used for the former. According to age, patients were divided into ones below 30 – group A (n=52) and ones aged 30 or older – group B (n=38). Regarding BMI, they were assessed at 27.8 kg/m^2 obesity cutt-off, being divided into ones with BMI below 27.8 kg/m^2 – group C (n=44) and ones with BMI of 27.8 kg/m^2 or higher – group D (n=46).

Total cholesterol (mmol/l; normal value: 3.1–6.5) and triglycerides (mmol/l; normal value: <1.95) were measured by standard enzymatic methods (cholesterol: cholesterol oxidase, Randox, UK; triglycerides: glycerol-3-phosphate oxidase, Randox, UK); HDL-cholesterol (mmol/l; normal value for women: 0.78–1.95) was measured by the direct method (Randox, UK); LDL-cholesterol (mmol/l; normal value for women: 1.55–4.14) was determined by Friedwald method.

A 75g oral glucose tolerance test (OGTT) was performed on each patient after an overnight fast. Plasma glucose (mmol/l; normal value: 3.3–6.1) was determined by the glucose-oxidase method (Randox, UK) using auto-analyser (Beckman, Austria). Plasma insulin (mIU/l; normal value: <20) was measured by radioimmunoassay (RIA, INEP, Zemun). Coefficient of variation (CV) for intra-assay was 20.4, 7.6 and 6.1% for the concentration of 9.4, 23.7 and 181.6 mIU/l; for inter-assay CV was 22.1, 10.6 and 16.8% for the concentration of 8.6, 33.8 and 153.9 mIU/l, respectively. For the measurement of glucose and insulin venous blood was drawn at 30 minute intervals over 2 hrs following glucose ingestion. The integrated area under the curve (AUC) analysis for glucose and insulin was determined according to the trapezoidal rule. Insulin sensitivity index (S_I) was calculated from fasting glucose-to-insulin ratio (FGIR), homeostasis model assessment (HOMA) and quantitative insulin sensitivity check index (QUICKI) (12–14).

Data are presented as the mean value ± standard deviation (mean ± SD). Student's t-test was used for the assessment of respective parameters between groups. Correlation between the observed parameters was determined using Pearson's coefficient of correlation. The statistical level of significance was set at $p<0.05$.

Results

The results of respective anthropometric, glucose, insulin and lipid parameters according to the age and body mass index of the investigated patients are presented in Table 1 and Table 2, respectively. Regarding the age of the examined women with PCOS, older patients showed significantly higher values of systolic and diastolic BP, fasting glucose, glucose at the end of 2 hours-lasting OGTT, integrated glucose response (AUC for glucose) during OGTT, and total cholesterol and trygliceride levels (Table 1). Analyses of PCOS patients regarding respective BMI showed that only systolic and diastolic BP were significantly elevated in the obese group of patients with PCOS (Table 2).

The obtained significant correlations in respect to the age and BMI for the whole group of patients with PCOS are presented in Table 3.

The correlations presented in Table 4 for the subgroup of patients below 30 and in Table 5 for the subgroup of those aged 30 and over were obtained after the assessment of patients according to their respective age groups.

Discussion

In this study anthropometric and metabolic characteristics of the group of women with polycystic ovary syndrome admitted for further evaluation were examined at the Clinical Center of Serbia's Institute of Endocrinology, Diabetes and Diseases of Metabolism. The entire group of patients was further analyzed regarding their age and BMI.

Regarding the age at which patients were assessed and BMI-related, it was observed that there was a significant increase in systolic and diastolic blood pressure with aging, as well as the increase of body weight. Interestingly, women became hypertensive with aging in spite of the fact that

Table 1.

Age related differences in anthropometric and metabolic parameters in PCOS women

	Age (years)				
	Group A (<30) n=52		Group B (≥30) n=38		p
Anthropometric parameters					
Age (years)	22.02	± 4.01	36.50	± 5.88	0.001
BMI (kg/m^2)	27.44	± 7.33	30.27	± 8.93	> 0.05
Systolic BP (mmHg)	122.76	±10.36	140.44	±25.51	0.001
Diastolic BP (mmHg)	82.65	± 9.95	90.59	±15.51	0.006
Glucose and insulin parameters					
Fasting glucose (mmol/l)	4.37	± 0.65	5.00	± 1.01	0.021
120 min glucose (OGTT) (mmol/l)	5.74	± 1.11	8.09	± 2.61	0.001
AUC for glucose (mmol/l/120 min)	748.04	±144.54	982.98	±234.22	0.001
Fasting insulin (mIU/l)	26.73	±21.79	26.54	±23.07	>0.05
AUC for insulin (mIU/l/120 min)	8762.05	±6147.14	8769.14	±5656.0	>0.05
FGIR (mg/dl x 10^{-2})	5.75	± 4.82	5.29	± 3.25	>0.05
HOMA	4.87	± 3.75	6.25	± 5.17	>0.05
QUICKI	0.54	± 0.11	0.52	± 0.09	>0.05
Lipid parameters					
Total cholesterol (mmol/l)	5.03	± 1.22	6.11	± 0.86	0.03
HDL-cholesterol (mmol/l)	1.17	± 0.24	1.22	± 0.31	>0.05
LDL-cholesterol (mmol/l)	2.92	± 1.01	3.68	± 1.10	>0.05
Triglycerides (mmol/l)	1.59	± 0.48	2.95	± 0.66	0.012

Table 2.

BMI related differences in anthropometric and metabolic parameters in PCOS women

	BMI (kg/m^2)				
	Group C (<27.8) n=44		Group D (≥27.8) n=46		p
Anthropometric parameters					
Age (years)	27.62	± 8.59	28.65	± 8.54	>0.05
BMI (kg/m^2)	22.22	± 3.33	34.97	± 6.54	0.001
Systolic BP (mmHg)	123.90	±14.85	136.67	±14.85	0.003
Diastolic BP (mmHg)	81.95	± 9.54	90.12	± 15.0	0.004
Glucose and insulin parameters					
Fasting glucose (mmol/l)	4.53	± 0.64	4.67	± 0.97	>0.05
120 min glucose (OGTT) (mmol/l)	6.06	± 2.30	7.01	± 2.23	>0.05
AUC for glucose (mmol/l/120 min)	768.26	±227.77	873.81	±206.82	>0.05
Fasting insulin (mIU/l)	21.21	±17.48	29.0	±23.06	>0.05
AUC for insulin (mIU/l/120 min)	7326.04	±4517.47	8880.47	±6588.96	>0.05
FGIR (mg/dl x 10^{-2})	4.96	± 2.14	5.74	± 4.78	>0.05
HOMA	4.80	± 3.97	5.74	± 4.73	>0.05
QUICKI	0.53	± 0.02	0.53	± 0.11	>0.05
Lipid parameters					
Total cholesterol (mmol/l)	5.18	± 1.30	5.75	± 1.74	>0.05
HDL-cholesterol (mmol/l)	1.31	± 0.18	1.12	± 0.33	>0.05
LDL-cholesterol (mmol/l)	3.05	± 1.22	3.48	± 0.97	>0.05
Triglycerides (mmol/l)	1.43	± 1.50	2.72	± 1.78	>0.05

Table 3.

Respective correlations for the whole group of patients with PCOS

Correlated Parameters	r	p
Age in relation to basal glucose	0.418	0.007
Age in relation to glucose at 120 min	0.619	0.01
Age in relation to AUC for glucose	0.583	0.01
Age in relation to total cholesterol	0.425	0.01
Age in relation to LDL-cholesterol	0.426	0.038
BMI in relation to glucose at 120 min	0.403	0.011
BMI in relation to AUC for glucose	0.423	0.007
BMI in relation to total cholesterol	0.302	0.09

Table 4.

Respective correlations within the age-related subgroups of patients with PCOS (age < 30 years)

Correlated parameters	r	p
BMI in relation to systolic BP	0.302	0.037
BMI in relation to diastolic BP	0.422	0.003
BMI in relation to glucose at 120 min	0.527	0.01
BMI in relation to AUC for glucose	0.518	0.011
BMI in relation to HDL-cholesterol	-0.722	0.012
Total cholesterol in relation to glucose at 120 min	0.612	0.003
Total cholesterol in relation to AUC for glucose	0.487	0.025
Triglycerides in relation to glucose at 120 min	0.628	0.002
Triglycerides in relation to AUC for glucose	0.520	0.013
FGIR in relation to HOMA	-0.701	0.004
FGIR in relation to QUICKI	0.921	0.001

Table 5.

Respective correlations within the age-related subgroups of patients with PCOS (age ≥ 30 years)

Correlated parameters	r	p
BMI in relation to systolic BP	0.496	0.003
BMI in relation to diastolic BP	0.471	0.006
BMI in relation to triglycerides	0.540	0.003
BMI in relation to basal insulin	0.579	0.049
Systolic BP in relation to total cholesterol	0.626	0.001
Systolic BP in relation to triglycerides	0.682	0.001
Systolic BP in relation to FGIR	-0.750	0.02
Systolic BP in relation to QUICKI	-0.748	0.02
Diastolic BP in relation to total cholesterol	0.524	0.004
Diastolic BP in relation to triglycerides	0.577	0.001
Diastolic BP in relation to basal glucose	0.652	0.012
Diastolic BP in relation toAUC for glucose	0.579	0.038
Diastolic BP in relation to basal insulin	0.630	0.038
Diastolic BP in relation to HOMA	0.735	0.024
FGIR in relation to triglycerides	0.76	0.018
FGIR in relation to QUICKI	0.797	0.006

there was no difference in their body mass, which was the case in the BMI-related analysis. Therefore, it was speculated that the deterioration of arterial blood pressure could be a consequence of PCOS independent of the body mass. It was previously shown that the obese women with PCOS had higher arterial blood pressure (7). Our results were in agreement with the data from previous studies, indicating that systolic and diastolic blood pressure elevated significantly in the groups over 35 years of age (5).

Significant increase in the fasting glucose, as well as global deterioration of glucose tolerance, was observed in the group of the assessed metabolic parameters. Namely, women with PCOS over than 30 years of age had impaired glucose tolerance after the glucose challenge as well as pronounced integral glucose response after OGTT expressed by the area under the glucose curve. However, basal insulin levels and integral insulin response after the glucose challenge were constantly high and did not differ between the respective age groups. This was in accordance with the view that insulin levels were higher in all PCOS age categories than in the respective controls (5). At the same time positive correlation between the age and basal glucose was observed for the whole group of PCOS patients, as well as positive correlation between both age and BMI and blood glucose after the glucose challenge test, total cholesterol and LDL-cholesterol levels, respectively.

Nestler indicated that approximately 75% of women with PCOS were insulin resistant and hyperinsulinemic, and demonstrated the increasing incidence of diabetes mellitus, hypertension, dyslipidemia and atherosclerosis (15). Impaired glucose tolerance and non-insulin dependent diabetes mellitus (NIDDM) was reported to be in range of 18–35% and 7.5–10%, respectively, in women with PCOS (12, 16, 17). Dunaif suggested that obese women with PCOS were at high risk of developing NIDDM in their 20s and 30s, compared to normal population, and proposed that non-obese women with PCOS were probably more likely to develop NIDDM if followed up to their 40s (16). Our results were in agreement with the above mentioned data, as we supposed that our group of women with PCOS developed hyperinsulinemia and insulin resistance already in the third decade of their life,

even earlier (6, 16). Insulin resistance found in patients with PCOS was due to the defect in postreceptor signal transduction between receptor kinase and glucose transporters (18). At the same time, there was a speculation on the existence of a PCOS-specific adipocyte insulin resistance resulting in neutralisation of the stimulatory influence of hyperinsulinemia on insulin secretion (19).

According to the latter, our patients – even under 30 years of age – expressed the decreased insulin sensitivity assessed by the basal insulin value and HOMA model. Other indices of insulin action, as FGIR and QUICKI, seemed not specific and accurate in the assessment of insulin sensitivity in our group of women with PCOS.

Contrary to the results obtained in relation to their age, our group of women with PCOS did not show deterioration in glucose tolerance and integral glucose response regarding their BMI. In the non-obese group of women with PCOS, basal insulin level was over the upper limit of the normal range and was elevating with aging. Other parameters for the assessment of insulin sensitivity showed the same pattern of change as those obtained for age-related differences.

It was shown that traditional cardiovascular risk factors were more prevalent in hyperinsulinemic women with PCOS than in their normoinsulinemic counterparts. It was also shown that normoinsulinemic women with PCOS were under higher cardiovascular risk than unselected normal healthy controls (20). In spite of the non-significant difference in insulin levels between the examined subgroups of PCOS women, high levels of insulin with the advanced increase with aging and aggravation of BMI were obtained, which was in accordance with the previous results (6, 20). Furthermore, when the peripheral insulin effect was assessed with the indexes of insulin sensitivity, only HOMA model clearly showed the deterioration of insulin sensitivity with aging and the aggravation of BMI in our patients with PCOS.

The group of patients with PCOS showed an increase in all observed lipid parameters between the third and the fourth decade of life, which was significant for total cholesterol and triglycerides. The increase was observed for total cholesterol, LDL-cholesterol and triglycerides, after the assesment of the patients towards their BMI. Total cholesterol in the whole group of PCOS patients was in correlation with age and BMI. When the patients were analyzed in relation to their age, in patients younger than 30 years of age, correlation was observed between total cholesterol and triglycerides with BMI and glucose response after glucose challenge, while only triglycerides correlated significantly with BMI in patients older than 30 years of age.

It was shown that hyperinsulinemia in PCOS correlated with low HDL and high triglycerides' level (7), which was not found in our group of patients, in spite of the elevated insulin levels and insulin resistance present in all the analyzed groups. The dyslipidemia of the insulin resistance syndrome (i.e., increased triglyceride levels, decreased HDL, and compositional changes in LDL) appeared to be caused by characteristics of the insulin-resistant state itself, rather than by elevated insulin concentrations or obesity. Proved persistent defect of β-cells of the endocrine pancreas and their secretion was found in spite of significant improvement in insulin sensitivity, suggesting that it could represent a primary abnormality of the polycystic ovary syndrome (21).

The atherogenic potential of the decreased HDL levels is a well known fact. Unexpectedly, elevated LDL was not a characteristic of the dyslipidemia of insulin resistance. However, in the insulin-resistant state, the composition of LDL was altered. It was demonstrated that in women with PCOS there existed an elevation of LDL-cholesterol subfraction III (22). Recently, it was observed that in PCOS patients HDL and LDL-cholesterol concentrations remained within the range of the healthy population, and that there was a significant derangement within LDL/Apo-B related complex with aging. Also, a significant increase in triglycerides was verified in the same study, which, together with the derangement in LDL/Apo-B complex, pointed to the altered metabolism of very low-density lipoproteins (VLDL) in relation to aging (6) in PCOS women.

Conclusion

The results of this study demonstrated a deterioration of glucose and lipid metabolism presumably related to the age of the examined patients with PCOS. Consequently, a substantial degree of insulin resistance was shown, best assessed through the indices of insulin action as its basal insulin value and HOMA model.

A significant increase in systolic and diastolic arterial blood pressure with aging and the increase in body weight was noted in the examined group of PCOS patients. However, only in patients over 30 years of age correlation between the indices of arterial blood pressure and lipid and glucose metabolism parameters was found, pointing out their causative relation and consequent deterioration of insulin resistance and lipid profile with aging, influencing the cardiovascular function in women with PCOS.

R E F E R E N C E S

1. *Franks S.* Polycystic ovary syndrome. N Engl J Med 1995; 333(13): 853–61.
2. *Legro RS, Kunselman AR, Dodson WC, Dunaif A.* Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome. A prospective, controlled study in 254 affected women. J Clin Endocrinol Metab 1999; 84(1): : 165–9.
3. *Gülekli B, Turhan NÖ, Senöz S, Kükner S, Oral H, Gökmen O.* Endocrinological, ultrasonographic and

clinical findings in adolescent and adult polycystic ovary patients. A comparative study. Gynecol Endocrinol 1993; 7(4): 273–7.

4. *Arslanian SA, Lewy VD, Danadian K.* Glucose intolerance in obese adolescents with polycystic ovary syndrome: roles of insulin resistance and beta-cell dysfunction and risk of cardiovascular disease. J Clin Endocrinol Metab 2001; 86(1): 66–71.
5. *Talbott E, Clerici A, Berga SL, Kuller L, Guzick D, Detre K,* et al. Adverse lipid and coronary heart disease risk profiles in young women with polycystic ovary syndrome: results of a case-control study. J Clin Epidemiol 1998; 51(5): 415–22.
6. *Macut D, Micić D, Cvijović G, Šumarac M, Kendereški A, Zorić S,* et al. Cardiovascular risk in adolescent and young adult obese females with polycystic ovary syndrome (PCOS). J Pediatr Endocrinol Metab 2001; 14 Suppl 5: 1353–9.
7. *Conway GS, Agrawal R, Betteridge DJ, Jacobs HS.* Risk factors for coronary artery disease in lean and obese women with the polycystic ovary syndrome. Clin Endocrinol 1992; 37(2): 119–25.
8. *Lobo RA, Carmina E.* The importance of diagnosing the polycystic ovary syndrome. Ann Intern Med 2000; 132(12): 989–93.
9. *Talbott E, Guzick D, Clerici A, Berga S, Detre K, Weimer K,* et al. Coronary heart disease risk factors in women with polycystic ovary syndrome. Arterioscler Thromb Vasc Biol 1995; 15(7): 821–6.
10. *Wild RA, van Nort JJ, Grubb B, Bachman W, Hartz A, Bartholomew M.* Clinical signs of androgen excess as risk factors for coronary artery disease. Fertil Steril 1990; 54(3): 255–9.
11. *Dahlgren E, Janson PO, Johansson S, Lapidus L, Oden A.* Polycystic ovary syndrome and risk for myocardial infarction. Evaluated from a risk factor model based on a prospective population study of women. Acta Obstet Gynecol Scand 1992; 71(8): 599–604.
12. *Legro RS, Finegood D, Dunaif A.* A fasting glucose to insulin ratio is a useful measure of insulin sensitivity in women with polycystic ovary syndrome. J Clin Enocrinol Metab 1998; 83(8): 2694–8.
13. *Kousta E, Cela E, Lawrence N, Penny A, Millauer B, White D,* et al. The prevalence of plycystic ovaries in women with a history of gestational diabetes. Clin Endicrinol 2000; 53(4): 501–7.
14. *Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G.* Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000; 85(7): 2402–10.
15. *Nestler JE.* Insulin regulation of human ovarian androgens. Hum Reprod 1997; 12 Suppl 1: 53–62.
16. *Dunaif A.* Insulin resistance in polycystic ovarian syndrome. Ann N Y Acad Sci 1993; 687: 60–4.
17. *Rajkhowa M, Bicknell J, Jones M, Clayton RN.* Insulin sensitivity in women with polycystic ovary syndrome: relationship to hyperandrogenemia. Fertil Steril 1994; 61(4): 605–12.
18. *Homburg R.* Polycystic ovary syndrome. Med Forum Int – Gyn Forum 1999; (Spec Issue): 3–11.
19. *Brzechffa PR, Jakimiuk AJ, Agarwal SK, Weitsman SR, Buyalos RP, Magoffin DA.* Serum immunoreactive leptin concentrations in women with polycystic ovary syndrome. J Clin Endocrinol Metab 1996; 81(11): 4166–9.
20. *Mather KJ, Kwan F, Corenblum B.* Hyperinsulinemia in polycystic ovary syndrome correlates with increased cardiovascular risk independent of obesity. Fertil Steril 2000; 73(1): 15–6.
21. *Holte J, Bergh T, Berne C, Wide L, Lithell H.* Restored insulin sensitivity but persistently increased early insulin secretion after weight loss in obese women with polycystic ovary syndrome. J Clin Endocrinol Metab 1995; 80(9): 2586–93.
22. *Pirwany IR, Fleming R, Greer IA, Packard CJ, Sattar N.* Lipids and lipoprotein subfractions in women with PCOS: relationship to metabolic and endocrine parameters. Clin Endocrinol 2001; 54(4): 447–53.

Rad je primljen 4. I 2002. god.

A p s t r a k t

Macut Đ, Micić D, Parapid B, Cvijović G, Šumarac M, Kendereški A, et al.
Vojnosanit Pregl 2002(6): 593–599.

KARDIOVASKULARNI FAKTORI RIZIKA KOD ŽENA SA SINDROMOM POLICISTIČNIH JAJNIKA U ODNOSU NA PROMENE VEZANE ZA ŽIVOTNO DOBA I TELESNU MASU

Sindrom policističnih ovarijuma (PCOS) predstavlja metabolički poremećaj blisko povezan sa gojaznošću, insulinskom rezistencijom (IR), hiperinsulinemijom i prognostički nepovoljnim lipidogramom, što u celini povećava rizik za nastanak kardiovaskularnih oboljenja. Cilj je bio da se proceni uloga godina života i indeksa telesne mase (BMI) u promeni kardiovaskularnih faktora rizika u 90 žena sa PCOS koristeći kao granične vrednosti starost od 30 godina i BMI od 27,8 kg/m^2. Kod svih bolesnica određivani su sistolni i dijastolni arterijski krvni pritisak, glikemija i nivo insulina u krvi tokom oralnog testa tolerancije na glukozu (OGTT) i bazalni lipidni status. Zapažen je značajan porast parametara arterijskog krvnog pritiska, bazne vrednosti insulina i insulinske rezistencije procenjene pomoću HOMA modela sa starenjem i porastom BMI, dok su se parametri metabolizma glukoze, ukupnog holesterola i triglicerida značajno povećavali sa starenjem. Međutim, samo kod bolesnica sa PCOS starijih od 30 godina uočena je korelacija između pokazatelja arterijskog krvnog pritiska i metabolizmna lipida i glukoze, što ukazuje na njihov uzročni odnos i posledično pogoršanje IR i lipidograma sa starenjem i uticaj na kardiovaskularnu funkciju.

K lj u č n e r e č i : **jajnik, policistični, sindrom; kardiovaskularne bolesti; faktori rizika; životno doba, faktori; telesna masa, indeks; insulin, rezistencija; lipidi; krvni pritisak; glukoza, test tolerancije.**