BODY COMPOSITION IN THIN WOMEN WITH PCOS

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Abstract. *Abnormal distribution of fat, especially visceral adipose tissue (VAT), and insulin resistance are key features of PCOS [34]. It remains a question whether lean women with PCOS have a similar pattern of metabolic disturbances and fat distribution as overweight and obese women with PCOS.*

This study aimed to investigate body composition, metabolic characteristics, and insulin resistance in lean women with PCOS.

Keywords: *fat, visceral adipose tissue, insulin resistance, PCOS.*

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy in women of reproductive age, with a prevalence of 8% to 13% depending on the population studied and the diagnostic criteria used [3,5]. PCOS has a complex of reproductive, metabolic and psychological features [16]

Abnormal distribution of fat, especially visceral adipose tissue (VAT), and insulin resistance are key features of PCOS [34]. It remains a question whether lean women with PCOS have a similar pattern of metabolic disturbances and fat distribution as overweight and obese women with PCOS.

This study aimed to investigate body composition, metabolic characteristics, and insulin resistance in lean women with PCOS.

MATERIALS AND METHODS

This was a cross-sectional study in which all included women were divided into two groups according to BMI [16,17] as follows: normal weight PCOS (BMI <25 kg/m2) and a normal weight control group (BMI <25 kg/m2). All women underwent a detailed history taking and physical examination. Irregular menstrual cycles were noted in the form of oligomenorrhea / amenorrhea, hirsutism, infertility, family history (diabetes mellitus / PCOS / hypertension). All women underwent a detailed examination, including anthropometric measurements (height, weight, BMI), blood pressure, assessment of hirsutism (modified Ferriman-Gallway scale; score ≥8 is significant).

All women underwent the following biochemical studies: fasting blood glucose, fasting lipid profile (enzymatic colorimetric assay-oxidase-peroxidase method), fasting insulin (immunochemiluminescent assay), total testosterone (immunochemiluminescent assay), SHBG, and FSH (immunochemiluminescent assay) , LH (chemiluminescent immunoassay) All women also underwent additional tests (prolactin, thyroid stimulating hormone) to rule out secondary causes of PCOS Insulin resistance index [18,19,20,21,22] was used to assess insulin resistance: fasting insulin level (cut-off value; ≥12.2 μU/mL), homeostasis assessment model-insulin resistance (HOMA-IR) (cut-off value; ≥2.7). Whole body dual energy x-ray absorptiometry (DXA) using the Lunar Progidy Primo body composition assessment machine in all women (PCOS and controls).

All parameters including anthropometric, metabolic characteristics, insulin resistance index and body composition parameters were compared between women with PCOS and their age and BMI matched control group.

STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS for Windows, version 21.0. (SPSS Inc., Chicago, USA). A two-sample t-test was used to compare mean differences in continuous outcome variables between groups. Similarly, a chi-square test has been used to link between categorical variables.

RESULTS

In total, 45 women took part in the study, they were divided into 2 groups: women with PCOS - 25 and 21 controls. 73.9% of women with PCOS had oligoanovulation and 91.3% had evidence of polycystic ovaries on ultrasound; hyperandrogenism, biochemical or clinical, was observed in approximately 65.2% of women with PCOS. Demographic and other baseline characteristics of PCOS women and controls are shown in Table 1.

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Table 1.

Initial characteristics of women

	Mean ± Standard Deviation			
	Total (n=45)	PCOS (n=24)	Control (n=21)	p <0,05
Age, years	20,6±2,5	19,9±2,3	20,4±2,6	0,06
Height, sm	158,5±22,8	154,7±30,3	154,7±30,3	0,23
Weight, kg	54,8±7,8	54,8±8,2	55,7±8,1	0,71
BMI, kg/m^2	20,9±2,5	21,2±2,7	20,9±2,6	0,68
FT4, mIU/l	1,7±2,4	2,15±3,3	1,2±0,1	0,47
TSH, mIU/l	2,1±1,1	2,0±1,2	2,3±1,0	0,48
Sex steroid binding globulin, nmol/l	58,2±27,8	43,3±17,4	71,4±29,3	0,001***
DHEA, mIU/l	251,9±111,6	261,5±131,6	194,6±40,7	0,35
Prolactin, ng/ml	15,8±5,8	17,7±5,8	13,8±5,2	0,06
FSH, mIU/l	6,1±2,1	5,9±1,9	6,3±2,3	0,54
LH, mIU/l	12,2±6,4	15,8±6,4	8,2±3,1	<0,001***
Testosteron, nmol/l	1,6±1,5	1,6±1,0	1,6±2,0	0,96

PCOS = polycystic ovary syndrome, BMI = body mass index, SHBG = sex hormone binding globulin, DHEAS = dehydroepiandrosterone sulfate, FSH = follicle stimulating hormone, LH = glutenizing hormone, TSH = thyroid stimulating hormone

The groups did not differ in age and BMI. In the PCOS group, the SHBG level (p= 0.001) and LH level (p<0.001) were statistically significantly lower.

A comparison of metabolic parameters, insulin resistance indices, and body composition parameters among women with PCOS with their age and BMI controls is shown in Table 2.

Table 2.

Comparison of metabolic parameters, insulin resistance index and body composition parameters

	Mean ± Standard Deviation			
	Total (n=45)	PCOS (n=24)	Control (n=21)	p <0,05
Insulin, mIU/l	10,7±4,4	13,2±4,7	8,1±2,3	0,001***
Glucose, mmol/l	4,7±0,4	4,8±0,3	4,6±0,4	0,17
HOMA-IR	1,5±1,3	2,1±1,6	1,1±0,9	0,02*
Cholesterol, mmol/l	4,7±1,04	4,9±0,9	4,4±1,1	0,35
Triglycerides, mmol/l	0,9±0,5	1,2±0,6	0,6±0,2	0,05*
HDL, mmol/l	1,5±0,5	1,5±0,5	1,5±0,6	0,93
LDL, mmol/l	2,4±0,7	2,6±0,5	2,2±0,7	0,27
VLDL, mmol/l,	0,7±0,4	0,8±0,5	0,6±0,4	0,64
Adipose tissue,%	31,2±6,4	33,5±5,4	29,3±6,9	0,03*
Visceral fat, %	3,4±1,1	4,0±0,9	2,8±1,1	0,001***

PCOS = polycystic ovary syndrome, BMI = body mass index, HDL = high density lipoprotein, LDL = low density lipoprotein, VLDL = very low density lipoprotein, HOMA-IR = assessment of homeostatic model - insulin resistance

Both groups had normal levels of insulin and HOMA-IR, but in the group of women with PCOS these values were statistically higher than in the control group, p=0.001 and p=0.02.

Women with PCOS had increased fat mass (as assessed by DXA scan) that was statistically significant (p = 0.03) and significantly higher visceral fat content (p = 0.001) compared to their control group.

DISCUSSION

This study attempted to evaluate metabolic characteristics, insulin resistance indices, and body composition parameters in normal weight women with PCOS compared to their age-matched and BMI-matched controls.

Non-obese women with PCOS had a higher percentage of adipose tissue and visceral fat (as assessed by DXA scan) compared to controls of the same age and BMI.

Overall, the present study suggests that women with PCOS, regardless of BMI, have increased visceral adiposity, which predisposes them to a higher risk of developing metabolic complications in the future. The literature is inconclusive regarding abdominal obesity (as measured either by DXA scan or magnetic resonance imaging [MRI]) in women with PCOS compared to controls. Some studies have demonstrated higher VAT in women with PCOS than controls of the same age and BMI [30,31], however, other studies have not found a statistically significant difference in relation to HPT in women with PCOS [15,23] Available in this regard limited literature.[27] Assessment of visceral fat, either indirectly by waist-to-hip ratio or more

objectively by DXA scan and MRI, is an important determinant and marker of insulin resistance, as it predisposes women with PCOS to a high risk of developing metabolic complications through various mechanisms. [12,26].

In the present study, insulin resistance was observed in 36% of non-obese women with PCOS, based on a fasting insulin threshold of ≥12.2 μU/mL [20, 45]. HOMA-IR ≥2.5, [20,25,55] for insulin resistance. Fasting insulin and HOMA-IR values were statistically significantly higher ($p<0.05$) in both groups of women with PCOS compared to their control group for age and BMI.

Various environmental and genetic factors along with the above mechanisms may further contribute to the increase in abdominal and visceral adiposity even in normal weight women with PCOS.

CONCLUSION

Lean women with PCOS compared with the control group had worse metabolic rates and more pronounced visceral obesity. Non-obese PCOS should be treated in the same way as obese PCOS to prevent future metabolic complications.

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