Original Article

Association between Steroid Hormones and Insulin Resistance in Patients with Polycystic Ovary Syndrome

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ABSTRACT

Objectives: Women with Polycystic Ovary Syndrome (PCOS) are more prone to adverse outcomes, including hypertension, obesity, hyperlipidemia, insulin resistance, type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease. This study examined the potential link between abnormal steroid hormone levels and insulin resistance (IR) in reproductive-aged women with PCOS.

Methods: This study involved 61 participants: a case group of 33 patients with confirmed PCOS based on Rotterdam criteria and a control group of 28 healthy individuals without PCOS. Steroid hormone levels, IR indices, metabolic markers, and demographic characteristics of participants were measured.

Results: The results showed significant differences in testosterone (P=0.018), dihydrotestosterone (DHT) (P=0.009), and androstenedione (P=0.002) levels between the two groups. Insulin levels and HOMA-IR were significantly higher in the patients (P=0.034 and 0.025, respectively). Significant correlations were found between androstenedione and insulin (P=0.021), fasting blood sugar, and homeostatic model assessment of insulin resistance (HOMA-IR) levels (P=0.001), as well as between DHT level and IR indices (P=0.03). Additionally, patients with PCOS had higher diastolic blood pressure and lower levels of T4.

Conclusion: The findings of this study showed higher androgen levels in PCOS patients and a significant correlation between DHT and androstenedione levels with IR indices in PCOS patients, which establishes a remarkable connection between hyperandrogenism and insulin resistance in PCOS.

Keywords: Polycystic ovary syndrome, Insulin Resistance, Obesity, Body mass index, Cholesterol, Androstenedione, Testosterone

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Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common metabolic disorders, affecting 5-15% of reproductive-aged women (1,2). According to the Rotterdam criteria, PCOS is diagnosed by the presence of at least two of three diagnostic criteria: oligo-anovulation, clinical or laboratory hyperandrogenism, and polycystic ovaries by ultrasound (3). The cause of PCOS is multi-factorial, involving both genetic and environmental factors (4). Its risk factors include obesity, lack of physical activity, a low fiber diet, and a positive family history (5). It is important to note that other conditions, such as adrenal hyperplasia, hypothyroidism, and hyperprolactinemia, can present similar symptoms and should be ruled out (5,6).

PCOS is a multi-systemic disorder characterized by hormonal imbalances (7,8). It arises from the overproduction of androgen hormones, such as testosterone, in the ovaries. This may occur due to high levels of pulsatile luteinizing hormone (LH) secretion by the anterior pituitary gland or elevated blood insulin levels (hyperinsulinemia) in women with ovaries sensitive to such stimuli (3,9). Obesity-linked insulin resistance is common among women with PCOS (10).

Insulin resistance (IR) refers to a condition in which cells do not respond adequately to insulin, even with normal or elevated insulin levels, leading to elevated blood sugar concentration (10,11). Hyperinsulinemia disrupts the function of the hypothalamus-pituitary-ovary axis, contributing to the development of PCOS (12). It leads to an increase in the pulsatile secretion of hypothalamic gonadotropin-releasing hormone (GnRH), resulting in a higher LH:FSH ratio and subsequent overproduction of androgens in the ovaries (12,13). Hyperinsulinemia also increases α-hydroxylase 17 activity, which is responsible for producing androgen precursors through the PI3K pathway (14).

Obesity is another contributing factor in the development of PCOS. Adipose tissue contains the aromatase enzyme that converts androstenedione to estrone and testosterone to estradiol (15). The increase in adipose tissue in obese women paradoxically increases the levels of androgens and estrogens simultaneously (15,16). Research shows that women with PCOS are more prone to adverse outcomes such as hypertension, obesity, hyperlipidemia, IR, type 2 diabetes mellitus (T2DM), metabolic syndrome (MS), cardiovascular disease, and endometrial hyperplasia (17,18,19). However, there has been a lack of comprehensive research on the relationship between elevated levels of steroid hormones and cardiometabolic factors such as IR, dyslipidemia, and T2DM in PCOS patients. This study aimed to examine the potential link between abnormal steroid hormone levels and IR in reproductive-aged Iranian women with PCOS.

Material and methods

Study subjects

The present retrospective cross-sectional study was conducted on 61 women: a case group with confirmed PCOS based on Rotterdam criteria and a control group without PCOS. At baseline, data were collected following approval from the Ethics Committee of Iran University of Medical Sciences (Ethics code IR.IUMS.FMD.REC.1398.308). The study followed the Declaration of Helsinki.

The participants were thoroughly evaluated and their history was taken. Patients were newly diagnosed and were not receiving any specific medications for PCOS. Inclusion criteria included individuals with PCOS who were diagnosed according to the Androgen Excess and PCOS Society (AE-PCOS) criteria, including: a) evidence of androgen excess; b) ovarian dysfunction recognized by oligo-ovulation and/or polycystic ovaries revealed by ultrasonography; and c) exclusion of related etiologies. Subjects with other accompanying illnesses, including inflammatory diseases, active infections, malignancies, or other endocrine disorders such as Cushing's syndrome, were excluded from the study. Other causes of hyperandrogenism, such as androgen-secreting tumors and non-classic congenital adrenal hyperplasia, were also examined and considered as exclusion criteria. Subjects who proved to be completely healthy were enrolled as the control group. Demographic factors such as age, height, weight, systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded, and body mass index (BMI) was calculated using the formula: $BMI = Weight\ (kg) / Height\ (m)^2$.

Biochemical measurements

In this study, the blood levels of steroid hormones, IR indices, and metabolic markers were measured. Steroid hormones, including estradiol, testosterone, dihydrotestosterone (DHT), androstenedione, dehydroepiandrosterone (DHEA), 17-hydroxyprogesterone (17-OHP), and T4, were measured using chemiluminescence methods. Serum insulin levels were measured by the ELISA method, and an enzymatic method was employed for the measurement of fasting blood sugar (FBS). The homeostatic model assessment of insulin resistance (HOMA-IR) was then calculated using the formula: [glucose (mg/L) * insulin (μU/mL)/405] and was used for the estimation of insulin resistance.

The lipid profile, including triglyceride (TG), cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), as well as liver enzymes such as aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP), were measured by calorimetric

methods. Calcium and phosphorus were measured by the Ion Selective Electrode method using an electrolyte analyzer.

Statistical analysis

Data collection and analysis were carried out using SPSS software version 25.0 (SPSS, Inc., Chicago, IL). The Kolmogorov-Smirnov test was used to assess the normal distribution of the variables. The comparison of variables between case and control subjects was performed using the Student's t-test for variables that were normally distributed and the Mann-Whitney U test for variables with non-normal distribution.

Results

The study population comprised 61 participants, with 33 in the case group and 28 in the control group. The demographic characteristics of the study population are shown in Table 1. The mean age of the case group was lower than that of the control group, with a significant difference between the two groups (P-value = 0.001). The mean height of the patient group was lower than that of the control group; however, the difference was not statistically significant (P-value = 0.837). The mean weight of the patient group was greater than that of the control group, but this difference was not statistically significant (P-value = 0.575). The mean BMI of the case group was higher than that of the control group, but this difference was not statistically significant (P-value = 0.315). The mean SBP and DBP were also compared between the groups, and although both DBP and SBP were higher in the patient group, this difference was only significant for DBP (P-value = 0.475).

The results of the studied biochemical variables are shown in Table 2. Among these parameters, only Ca and T4 levels significantly differed between the case and control groups; such that Ca levels were significantly higher and T4 levels were significantly lower in patients than those in control subjects, although both parameters were within normal ranges in both studied groups.

The hormonal variables studied were estradiol, testosterone, DHT, 17-OHP, androstenedione, DHEA, and insulin. The HOMA-IR was also used as an index of insulin resistance. As shown in Table 3, testosterone, DHT, and androstenedione levels showed a significant difference between the case and control groups, and the mean levels of all these androgens were higher in patients than those in controls. However, estradiol and DHEA did not significantly differ between the two groups. Although non-classical CAH was ruled out in the patients, 17-OHP levels were slightly higher in cases; however, its difference with control subjects did not reach significance.

Table 1: Demographic variables

Variables	Group	Mean	SD	P- value
Age (years)	Case	25.54	7.729	0.001
	Control	32.58	7.421	
Height (cm)	Case	160.79	5.014	0.837
	Control	161.09	6.297	
Weight (kg)	Case	63.75	8.172	0.575
	Control	62.52	8.807	
BMI	Case	24.712	2.655	0.315
	Control	23.988	2.873	
SBP (mmHg)	Case	111.42	8.908	0.475
	Control	109.69	9.757	
DBP (mmHg)	Case	75.53	6.286	0.045
	Control	71.51	8.969	

he data are presented as Mean ± SD. BMI: Body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure.

Table 2: Biochemical variables

Variables	Case	Control	P-value
TG (mg/dl)	87.39 ± 56.1	70.21 ± 24.8	0.137
Cholesterol (mg/dl)	163.11 ± 31.7	154.94 ± 28.1	0.347
HDL-C (mg/dl)	50.59 ± 12.3	51.06 ± 12.7	0.901
LDL-C (mg/dl)	93.77 ± 25.9	89.84 ± 26.5	0.620
FBS (mg/dl)	90.76 ± 8.293	90.42 ± 7.5	0.873
T4 (µg/dl)	8.024 ± 1.2	9.73 ± 2.7	0.005
AST (U/L)	17.30 ± 4.2	16.85 ± 4.6	0.726
ALT (U/L)	18.65 ± 6.7	15.88 ± 7.1	0.169
ALP (U/L)	150.83 ± 39.5	136.95 ± 26.0	0.149
Ca (mg/dl)	9.256 ± 0.55	8.697 ± 0.31	0.001
P (mg/dl)	3.452 ± 0.6	4.021 ± 0.5	0.658

'he data are presented as Mean ± SD. TG: triglycerides; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprote holesterol; FBS: fasting blood sugar; AST: aspartate aminotransferase; ALT: alanine transaminase; ALP: Alkaline Phosphatase.

Table 3: Hormonal variables

	Group	Mean	SD
Variables	**Case**	**Control**	**P-value**
Estradiol (pg/mL)	93.508 ± 105.3	139.77 ± 119.5	0.126
Testosterone (ng/ml)	0.564 ± 0.5	0.267 ± 0.09	0.012
DHT (pg/ml)	412.507 ± 206.8	286.56 ± 122.8	0.005
17-OHP (ng/L)	1.212 ± 1.6	0.561 ± 0.2	0.065
Androstenedione (ng/ml)	3.243 ± 1.86	2.412 ± 1.01	0.036
DHEA (µg/dl)	235.42 ± 87.7	211.31 ± 113.4	0.371
HOMA-IR	4.34 ± 4.2	2.33 ± 1.1	0.025
Insulin (µIU/mL)	11.8 (7.8 - 21.42)	10.33 (7.2 - 12.42)	0.034

he data are presented as Mean ± SD for parametric and median (interquartile range) for non-parametric variables. DHT: Dihydrotestosterone; 7-OHP: 17-Hydroxyprogesterone; DHEA: Dehydroepiandrosterone; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance.

As stated above, FBS was found to be within normal ranges in patients and controls and was not significantly different between the two groups. Nevertheless, the results indicated that insulin levels in the case group were significantly higher compared to the control group (P-value = 0.034). HOMA-IR was also significantly higher in patients, indicating a state of insulin resistance.

The relationship between steroid hormones and IR indices, including FBS, insulin, and HOMA-IR, was investigated and is shown in Table 4. DHT showed remarkable correlations with all the aforementioned indices of insulin resistance. Significant correlations were also observed between androstenedione and insulin, FBS and HOMA-IR levels. There was no significant correlation between IR indices and other steroid hormones. Both HOMA-IR and insulin levels were positively correlated with TG ($r = 0.709$, $P < 0.001$ and $r = 0.690$, $P < 0.001$, respectively), but not with BMI or other metabolic risk factors.

The relationship between steroid hormones and all the demographic and metabolic parameters was also investigated and among them only androstenedione and 17-OHP were found to have negative significant correlations with LDL-C ($r = -0.366$, $P = 0.009$, and $r = -0.286$, $P = 0.046$, respectively), while all other correlation results were non-significant.

Table 4: Correlation between steroid hormones and IR indices

	FBS		Insulin		HOMA-IR	
	R	P	R	P	R	P
Estradiol (pg/mL)	-0.148	0.269	-0.147	0.285	-0.153	0.269
Testosterone (ng/ml)	-0.100	0.453	0.049	0.721	0.176	0.199
DHT (pg/ml)	0.313	0.015	0.330	0.012	0.392	0.003
Androstenedione	0.252	0.048	0.415	0.001	0.444	0.001
DHEA-S	0.188	0.154	-0.020	0.884	0.014	0.920
17-OHP	0.091	0.503	0.081	0.564	0.080	0.571

DHT: Dihydrotestosterone; 17-OHP: 17-Hydroxyprogesterone; DHEA: Dehydroepiandrosterone; FBS: fasting blood sugar; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance.

Discussion

PCOS patients generally encounter a state of insulin resistance compared to healthy individuals, increasing their risk for cardiometabolic complications. Previous reports have attempted to identify predictors of IR and cardiometabolic risks in PCOS patients and found a relationship between steroid hormones and other risk factors with IR and cardiovascular risks. Our study confirms this relationship by finding significant correlations between some steroid hormones and IR indices. The results of our study showed that testosterone, DHT, and androstenedione were significantly higher in patients with PCOS, and androstenedione and DHT were significantly correlated with insulin resistance factors.

In a study by Lazurova et al., the relationship between sex steroid hormones and cardiometabolic risk factors was investigated in PCOS patients. The results showed higher levels of estrone, free testosterone, and free androgen index (FAI) in obese and overweight PCOS patients compared to non-obese PCOS patients. Additionally, it was shown that blood estrone and free testosterone levels can be used as markers to evaluate cardiometabolic risk. A positive correlation was reported between blood estrone, FAI levels, and BMI with HOMA-IR. Similar to the findings of this study, the relationship between estradiol, testosterone, and DHEA with insulin resistance parameters was found to be non-significant. However, inconsistent with the results of this study, no relationship was reported between androstenedione and DHT levels with IR indices (20). The limitation for the measurement of SHBG was present, so FAI could not be calculated and compared.

Yang et al. evaluated the levels of steroid hormones in 141 PCOS patients. Participants were divided into two subgroups based on their testosterone levels, including those with high and normal androgen levels. Additionally, 166 individuals were selected as the control group. Their results showed elevated androstenedione, FAI, and 17-OHP levels in the patient group. These results were consistent with the findings of our study (21).

In a study of the relationship between serum adiponectin levels and IR in PCOS patients, Pekcan et al. showed that lower adiponectin levels were associated with increased IR in these patients. They revealed elevated levels of DHEA, SHBG, FAI, total testosterone, insulin, and HOMA-IR levels were significantly higher in the PCOS group (22).

Xuelin et al. investigated the correlation between glucose metabolism and steroid hormones among 914 PCOS patients. Their study showed a positive and significant correlation between aldosterone, androstenedione, estrone, and FAI levels with abnormal glucose metabolism before adjusting for confounding factors such as age, BMI, and fasting insulin level. However, after adjusting for confounding factors, there was only a significant correlation between aldosterone, androstenedione, and estrone levels with IR. There was also no relationship between abnormal glucose metabolism with progesterone, testosterone, and FAI levels. Among the above factors, the results for androstenedione were similar to our study. However, aldosterone and estrone were not measured in our study (23).

Noor et al. studied 286 women with PCOS and assessed their dyslipidemia. The results showed that 24.13% of the women had dyslipidemia, with a higher prevalence among those of older age and a higher BMI. Our study did not confirm a higher prevalence of dyslipidemia among PCOS patients; however, a significant negative association between LDL-C and androstenedione as well as 17-OHP was observed (24).

Khan et al. explored the relationship between PCOS and metabolic disorders. They analyzed demographic characteristics and metabolic syndrome components and demonstrated a significant association between metabolic syndrome and age, as well as the period of the disease, and a nearly significant association with higher BMI. Fei Guo et al. also studied dyslipidemia and its association with metabolic disorders in PCOS patients and found a high prevalence of dyslipidemia among Chinese PCOS patients, with significantly increased levels of TG and LDL-C in patients with insulin resistance. Besides, they also reported an increasing trend for TG and Apolipoprotein B (Apo-B) with higher

BMI in their studied subjects (26). The findings of our study were different in that no significant difference was observed for metabolic parameters between the case and control groups, which might have been due to the short duration of PCOS and the recent diagnosis of the disease in our patients (25). Therefore, follow-up of patients might better reveal the association between hormonal parameters and metabolic risk factors.

Conclusion

The results of our study showed an elevation of androgen hormones and a state of insulin resistance in patients with PCOS. Among the androgens, the relationship between androstenedione and DHT with insulin resistance was prominent, and these two androgens are suggested to be focused on for the follow-up of patients. No association between androgens and other metabolic parameters existed in patients with newly diagnosed PCOS.

Conflict of Interest

The authors declare that there is no conflict of interest.

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