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HOMA-IR as a secondary diagnostic parameter in the diagnosis and differentiation of the PCOS phenotypic subgroups

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) is a multisystemic endocrine, metabolic and reproductive disorder. The Homeostasis Model Assessment (HOMA-IR), as a clinical marker of insulin resistance (IR), is not included in the Rotterdam diagnostic criteria for the diagnosis of PCOS. The aim of this study was to determine the diagnostic value of HOMA-IR in the diagnosis of PCOS and in the differentiation of its phenotypic forms.

Subjects and Methods: A retrospective case–control study was conducted including 99 women of reproductive age diagnosed with PCOS and 27 women without PCOS, selected according to the ESHRE/ASRM criteria from 2003. All participants were between 21 and 40 years of age. Women with PCOS were classified into four phenotypic groups according to the Rotterdam criteria. The Mann–Whitney U test was used for comparisons between two groups, while the Kruskal–Wallis test was applied for comparisons among more than two groups. Spearman's correlation test was used to analyze correlations between parameters within the groups.

Results: The median HOMA-IR value in women with PCOS was significantly higher compared with the control group without PCOS (2.26 vs. 1.48; $p < 0.001$). All PCOS phenotypic subgroups showed significantly higher HOMA-IR values than the control group: PCOS type A (2.75 vs. 1.48; $p < 0.001$), PCOS type B (2.14 vs. 1.48; $p < 0.001$), PCOS type C (2.22 vs. 1.48; $p < 0.001$), and PCOS type D (2.03 vs. 1.48; $p < 0.001$). A statistically significant difference in median HOMA-IR values was observed among the four PCOS phenotypic subgroups ($\chi^2 = 8.086$; $df = 3$; $n = 99$; $p < 0.04$). A significant positive correlation ($p < 0.05$) was found between HOMA-IR and insulin, glucose, testosterone and body mass index (BMI), while a negative correlation was observed between HOMA-IR and sex hormone–binding globulin (SHBG).

Conclusions: HOMA-IR is a useful additional diagnostic parameter in women with PCOS. When combined with the Rotterdam criteria, it may aid in the differentiation of the PCOS phenotypic forms.

Keywords: *polycystic ovary syndrome; phenotypic forms; homeostasis model assessment (HOMA-IR)*

Introduction

Polycystic ovary syndrome (PCOS), the most common endocrine disorder in women of reproductive age, is characterized by menstrual irregularities, polycystic ovarian morphology and hyperandrogenemia. It is also associated with insulin resistance (IR), chronic inflammation and oxidative stress, which may contribute to the development of autoimmunity (1). Approximately 70% of affected women are subfertile (1), and the prevalence of PCOS ranges from 6% to 20% across different countries (2). From a pathophysiological perspective, PCOS is characterized by chronic oligo-anovulation, elevated circulating androgen levels, polycystic ovarian morphology (PCOM), disordered gonadotropin secretion, insulin resistance/hyperinsulinemia and a frequent association with obesity (3). Women with PCOS are at increased risk of developing type 2 diabetes mellitus and metabolic syndrome (4).

At the Third Amsterdam Consensus Workshop in 2012, the definition of PCOS was redefined. PCOS is now considered an endocrine disorder of reproductive-age women, characterized by menstrual dysfunction, anovulatory infertility, dysfunction of gonadotropin secretion, hyperandrogenism

and insulin resistance, the latter being regarded as a key factor in the development of PCOS (5).

Metabolic insulin resistance arises from serine phosphorylation rather than tyrosine phosphorylation, following insulin binding to the insulin receptor.

Adipocytes, derived from adipose tissue, secrete endocrine and paracrine hormones and metabolites. Free fatty acids and tumor necrosis factor-alpha (TNF-α), produced by adipocytes, contribute to the development of insulin resistance with subsequent compensatory hyperinsulinemia (6,7).

Clinically, insulin resistance can be assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), calculated according to the following formula:

$$\text{HOMA-IR} = \text{fasting insulin } (\mu U/mL) \times \text{fasting glucose (mmol/L)} / 22.5 \text{ (8).}$$

Clinical manifestations of insulin resistance include fasting hyperinsulinemia (>10 μIU/mL), decreased levels of sex hormone–binding globulin (SHBG) and non-alcoholic fatty liver disease (NAFLD) (9).

The aim of this study was to determine the clinical significance of HOMA-IR as a marker of insulin resistance in the diagnosis of PCOS phenotypic forms.

Study design

This retrospective case–control study included 99 women of reproductive age diagnosed with PCOS (study group) and 27 women of reproductive age without a PCOS diagnosis (control group), selected according to the ESHRE/ASRM criteria established in 2003. The study was conducted between May 2022 and December 2024 at the private healthcare institution "PZU Dr. Hajder" in Tuzla.

Study protocol. Inclusion criteria comprised infertile women aged 21–40 years with a diagnosis of PCOS who voluntarily provided written informed consent. Exclusion criteria included women older than 40 years and those with diagnosed endocrinopathies. The PCOS group was further classified into four phenotypic forms (A, B, C, and D) according to the ESHRE/ASRM diagnostic criteria.For all participants, with and without PCOS, the following parameters were analyzed: age, body weight, fasting plasma glucose and insulin levels, HOMA-IR, testosterone, sex hormone–binding globulin (SHBG), free androgen index (FAI) and anti-Muellerian hormone (AMH).

Definition of parameters. Biochemical hyperandrogenemia was defined as a total testosterone level > 2.5 nmol/L. Insulin resistance was defined as HOMA-IR > 2.0 (10). The free androgen index (FAI) was calculated using the formula:

$$\text{FAI} = [\text{testosterone (nmol/L)} / \text{SHBG (nmol/L)}] \times 100 \text{ (11).}$$

An FAI value > 3 indicated hyperandrogenemia. Body mass index (BMI) was calculated as follows:

$$\text{BMI (kg/m}^2\text{)} = \text{weight (kg)} / \text{height}^2 \text{ (m}^2\text{).}$$

Methods

Body weight and height, as well as BMI, were assessed during physical examination. Hormonal parameters, including glucose, insulin, testosterone, SHBG, and AMH, were measured. Age at menarche and menstrual cycle length were also analyzed. Insulin concentrations were measured using the Elecsys Insulin assay (Mannheim, Germany) and AMH levels were determined using the Elecsys AMH assay (Rotkreuz, Switzerland). All laboratory analyses were performed at the Dia Lab laboratory in Bijeljina, Bosnia and Herzegovina.

Statistical analysis. Data analysis was performed using SPSS statistical software (version 22.0; SPSS Inc., Chicago, IL, USA). As the parameters did not follow a normal distribution, results were expressed as nonparametric variables and presented as medians with interquartile ranges. The nonparametric Mann–Whitney U test was used for comparisons between two groups, while the Kruskal–Wallis test was applied for comparisons among more than two groups. In all analyses, a p-value < 0.05 was considered statistically significant. Spearman's rank correlation coefficient was used for bivariate correlation analyses, with $p < 0.05$ also considered statistically significant.

Results

The biggest subgroup was the phenotypic subgroup A and the smallest subgroup the subgroup C.

PCOS type A accounted for 40 (40.40%), PCOS B for 16 (19.19%), PCOS-type C for 14 (14.14%) and PCOS-type D for 29 (29.29%) of the patients (Figure 1).

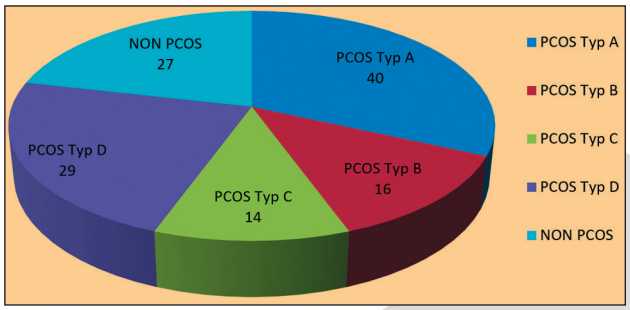

Figure 1. PCOS phenotypes and the non-PCOS group

In PCOS women had a longer menstrual cycle length and earlier menarche. There were no significant differences in age when comparing the PCOS with the control group without PCOS. BMI (26.30 vs. 22.74, $p < 0.001$), insuline (10.30 vs. 7.20, $p < 0.001$), fasting glucose (5.80 vs. 4.82, $p < 0.009$), HOMA-IR (2.26 vs. 1.48, $p < 0.001$), testosterone (2.20 vs. 1.3, $p < 0.001$), FAI (6.21 vs. 1.83, $p < 0.001$) and AMH (5.20 vs. 2.90, $p < 0.001$) were significantly higher in the PCOS when compared with the non-PCOS group, the SHBG (35.30 vs. 62.30, $p < 0.001$) was significantly lower in the PCOS group compared to the non-PCOS group (Table 1).

Patients with PCOS had a significantly higher HOMA-IR median (2.26 vs. 1.48, $p < 0.001$) when compared with the non-PCOS group (table 1). All phenotypic PCOS subgroups had significantly higher HOMA-IR medians ($p < 0.05$) when compared with the non-PCOS group. PCOS subgroup A had a significantly higher HOMA-IR (2.75 vs. 1.48, $p < 0.001$), PCOS subgroup B a significantly higher HOMA-IR (2.14 vs. 1.48, $p < 0.001$), PCOS subgroup C a significantly higher HOMA-IR (2.22 vs. 1.48, $p < 0.001$) and the subgroup D a significantly higher HOMA-IR (2.03 vs. 1.48, $p < 0.001$) when compared with the non-PCOS group (Figure 2). The highest HOMA-IR median was in the PCOS-phenotype A (2.74 vs. 2.03, $p < 0.008$) when compared with the lowest in PCOS-phenotype D. There were no significant differences between the phenotypes B, C and D (Figure 2).

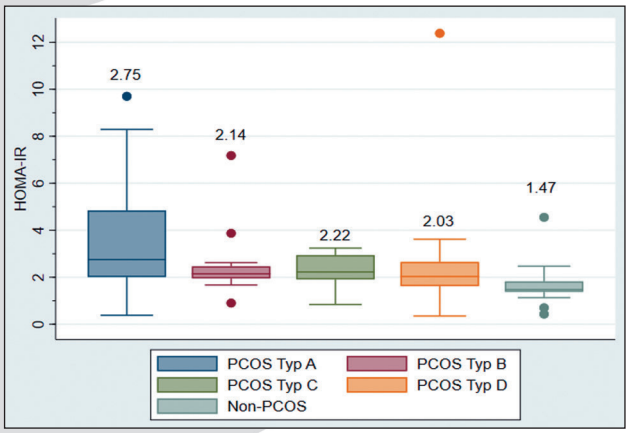

Figure 2. Comparative HOMA-IR analysis in the phenotypic PCOS-subgroups of and the non-PCOS subgroup

Legend: HOMA-IR, Homeostasis Model Assessment For Insulin Resistance Index; p = 0.008 for PCOS Type A vs. PCOS Type D; p = 0.001 for PCOS Typ A vs. Non-PCOS; p = 0.001 for PCOS Type B vs. Non-PCOS; p = 0.001 for PCOS Type C vs. Non-PCOS; p = 0.027 for PCOS Type D vs. Non-PCOS.

Table 1. Basal parameter comparison between the PCOS and the non-PCOS group

Parameters	PCOS (n = 99)	Non-PCOS (n = 27)	p-value
	Median (Q1-Q3)	Median (Q1-Q3)	
Testosterone (nmol/L)	2.20 (1.4-2.9)	1.30 (0.9-1.5)	0.001
SHBG (nmol/L)	35.30 (29.6-51.2)	62.30 (51.6-74.3)	0.001
FAI	6.21 (2.6-9.1)	1.83 (1.3-2.8)	0.001
Insuline (µIU/ml)	10.30 (8.30-13.50)	7.20 (6.30-8.20)	0.001
Glucose (mmol/L)	5.80 (5.2–6.2)	4.82 (4.18–5.32)	0.009
HOMA-IR	2.26 (1.8-3.2)	1.48 (1.38-1.81)	0.001
Menstrual cycle (days)	35.00 (25.1-41.2)	29.00 (28.0-34.0)	0.001
BMI (kg/m^2)	26.30 (23.4-30.0)	22.74 (21.2-25.2)	0.001

Legend: Parameters are expressed as median (range Q1-Q3); FAI, free androgenic index, SHBG, sex hormone binding globuline; BMI, Body Mass Index; HOMA-IR, Homeostasis Model Assessment For Insulin Resistance Index. Two-sample Wilcoxon rank-sum (Mann-Whitney) test, p < 0.05, PCOS vs. non-PCOS; 1 ng/dl = 28.65 nmol/l.

All four PCOS phenotypes showed the presence of insulin resistance (IR), expressed by HOMA-IR values ≥ 2. The highest prevalence of IR shown through the HOMA-IR parameter was 77.50% in PCOS phenotype A and the lowest was 51.72% in PCOS phenotype D (Figure 3).

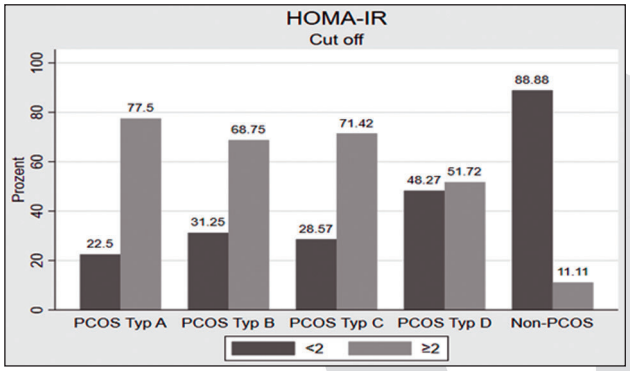

Figure 3. HOMA-IR results in the PCOS phenotypes and the non-PCOS group

Legend: PCOS, Polycystic Ovary syndrome; HOMA-IR, Homeostasis Model Assessment for Insulin Resistance Index.

Table 2. shows the comparison of the HOMA-IR parameters in the 4 phenotypic PCOS subgroups. Kruskal-Wallis test has shown a statistically significant difference in the HOMA results in the 4 phenotypic PCOS-subgroups (n = 40, PCOS subgroup A, n = 16, PCOS subgroup B, n = 14, PCOS subgroup C, n = 29, PCOS subgroup D, X2 = 8.086 (Total n = 99, p < 0.044).

In women with PCOS, a strong positive correlation was observed between HOMA-IR and BMI (ρ = 0.635, n = 99, p < 0.001). A strong positive correlation was also found between HOMA-IR and fasting glucose levels (ρ = 0.598, n = 99, p < 0.001), as well as between HOMA-IR and insulin levels (ρ = 0.980, n = 99, p < 0.001). In addition, HOMA-IR showed a strong positive correlation with testosterone levels (ρ = 0.312, n = 99, p < 0.001) and with the free androgen index (FAI) (ρ = 0.551, n = 99, p < 0.001). Conversely, a strong negative correlation was observed between HOMA-IR and sex hormone–binding globulin (SHBG) levels (ρ = −0.580, n = 99, p < 0.001) (Table 3).

Table 3. Spearman-Rho correlation between the HOMA-IR parameter and other PCOS parameters Parameters

Parameters PCOS	HOMA-IR	
	rho	p-value
BMI (kg/m^2)	0.635**	0.001
Insuline	0.980**	0.001
Menarche (years)	0.19**	0.048
Glucose	0.0.598*	0.001
Testosterone	0.312**	0.001
SHBG	-0.580**	0.001
FAI	0.55**	0.001

*Legend: BMI, body mass index; HOMA-IR, homeostasis model assessment for insulin resistance index; FAI, free androgen index; * weak correlation; ** strong correlation; Spearman rho correlation *p < 0.05.*

Discussion

The key diagnostic parameters of PCOS according to the Rotterdam criteria include clinical and/or biochemical hyperandrogenism (HA), irregular menstrual cycles due to oligo/anovulation (OA) and polycystic ovarian morphology (PCOM). The diagnosis is based on the ESHRE/ASRM criteria and requires the presence of at least two of the three criteria, after exclusion of PCOS-like disorders. The presence of two out of three clinical parameters defines four phenotypic forms of PCOS (A, B, C, and D). At the same ESHRE/ASRM consensus meeting in 2003, four PCOS phenotypes and a PCOM-like phenotype were proposed and remain under consideration today: PCOS phenotype A (HA + OA + PCOM), pheno-

Table 2. Comparison of the HOMA-IR in the 4 PCOS phenotypes

Parametar	Subgroup	n	Mean rank	Chi Square	df	p-value
HOMA-IR	PCOS Type A PCOC Type B PCOC Type C PCOC Type D Kohorta PCOS	40 16 14 29 99	59.43 45.63 48.32 40.22	8.086	3	0.044

Legend: Values are median (range, Q1-Q3), PCOS, Polycystic Ovary Syndrome; n- number of patients; HOMA-IR, homeostasis model assessment for insulin resistance index; Kruskal-Wallis equality-of-populations rank test, p < 0.0001.

type B (HA + OA), phenotype C (HA + PCOM), phenotype D (OA + PCOM) and the PCOM-like phenotype (12).From the three clinical parameters (OA, HA, PCOM), seven different clinical combinations are possible, which complicates the diagnosis of PCOS (13). Although insulin resistance (IR) is a primary disorder with a high prevalence among women with PCOS, it was not included in the Rotterdam diagnostic criteria (12). The prevalence of IR in women with PCOS has been reported to be approximately 70% (14).

There is still no consensus regarding the optimal cut-off value for HOMA-IR. A recently published study reported that a HOMA-IR cut-off value ≥ 2.5 indicates insulin resistance and detects cardiometabolic risk in the general population (15). For women with metabolic syndrome defined by IDF criteria, HOMA-IR cut-off values of 2.11 for those under 30 years of age, 2.05 for those up to 50 years, and 2.37 for those over 70 years have been proposed (16). A HOMA-IR cut-off value ≥ 2.0 has also been recommended for young women with PCOS, and this threshold is used by the European Group for the Study of Insulin Resistance (10).The results of the present study demonstrated that insulin resistance was significantly more prevalent in women with PCOS compared with non-PCOS women (HOMA-IR, $p < 0.01$). All PCOS phenotypic subgroups showed significantly higher HOMA-IR values ($p < 0.05$) than non-PCOS women. The highest HOMA-IR values were observed in phenotype A and were significantly higher ($p < 0.05$) compared with phenotypes B, C, and D. These findings confirm the association between insulin resistance/hyperinsulinemia and hyperandrogenism, which is consistent with the pathogenesis of PCOS. Insulin resistance leads to compensatory hyperinsulinemia, which stimulates pituitary luteinizing hormone (LH) secretion and increases the LH/FSH ratio. Through activation of the enzyme P450c17α, ovarian and adrenal steroidogenesis is enhanced, resulting in hyperandrogenism and irregular menstrual cycles (3). Activation of cytochrome P450c17 (CYP17) due to hyperinsulinemia leads to increased adrenal androgen steroidogenesis (progesterone, 17α-hydroxyprogesterone, and testosterone) compared with normal theca cells (7). Insulin resistance and hyperinsulinemia further increase ovarian androgen production directly and indirectly by reducing the synthesis of sex hormone–binding globulin (SHBG), thereby contributing to hyperandrogenism and estrogen excess in women with PCOS (8).A recently published Indonesian study involving 125 women of reproductive age with PCOS diagnosed according to the Rotterdam criteria (mean age 29.6 years) reported significantly elevated HOMA-IR levels across all PCOS phenotypic subgroups. HOMA-IR values were 4.2 in phenotype A, 3.2 in phenotype B, 2.9 in phenotype C and 2.0 in phenotype D, with a positive correlation between HOMA-IR and AMH levels (17). Panidis et al. (2012), in a Greek study of 1,212 women with PCOS diagnosed according to the Rotterdam criteria, reported that 50% of PCOS women had an increased BMI. Women with phenotypes A and B exhibited significantly greater insulin resistance compared with controls, as reflected by higher insulin and HOMA-IR levels. In contrast, women with phenotype C did not differ from the control group in any insulin resistance markers, while women with phenotype D had borderline higher HOMA-IR values compared with controls ($p < 0.016$) (18). Alebić et al. (2015), in a retrospective analysis of 284 Croatian/French women with PCOS, reported that insulin resistance was significantly present in phenotypic subgroups A and D compared with controls. The highest median HOMA-IR values were observed in phenotype A (3.0) and phenotype D (2.3), while the lowest value was recorded in phenotype C (1.9). Phenotype B was not analyzed due to a small sample size ($n = 12$). No positive correlation between HOMA-IR and AMH was found (19).

Al-Jefout et al. (2017), in an analysis of 159 women with PCOS from Jordan diagnosed according to the Rotterdam criteria, reported that median HOMA-IR values were significantly higher in women with PCOS compared with infertile non-PCOS women. A statistically significant difference ($p < 0.006$) was observed among PCOS phenotypic subgroups. HOMA-IR values were 3.11 in the total PCOS group, 3.38 in phenotype A, 2.86 in phenotype B, 2.95 in phenotype C, and 2.85 in phenotype D. Insulin resistance was most pronounced in phenotype A and lowest in phenotype D (20).Overall, the results of the present study and previously published studies are

largely consistent regarding HOMA-IR values in the overall PCOS population; however, differences exist among individual PCOS phenotypes B, C, and D (17–20).

Conclusion

Insulin resistance is prevalent in women with PCOS and is closely associated with hyperandrogenism, resulting in a complex metabolic–endocrine disorder. HOMA-IR represents a valuable adjunct clinical parameter, alongside Rotterdam criteria and AMH, for the diagnosis of PCOS and its phenotypic forms, particularly for identifying women with PCOS who are at increased cardiometabolic risk.

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