

Differences in the Characteristics and Factors Associated with Insulin Resistance in Normal Weight and Overweight Polycystic Ovary Syndrome Women

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ABSTRACT

OBJECTIVE This research aimed to compare clinical and biochemical parameters between normal and overweight-obese polycystic ovary syndrome (PCOS) patients, and to determine the association between insulin resistance (HOMA-IR) and anthropometric, biochemical and hormonal factors in both groups of PCOS.

METHODS This study was of a cross sectional design. PCOS was diagnosed using the Revised Rotterdam Criteria 2003. Participants underwent history taking and physical examination. Blood samples were collected for FPG, insulin, lipid profiles, TSH, FSH, LH, SHBG, total testosterone, and free androgen index (FAI). Insulin resistance index was calculated by homeostatic model assessment (HOMA-IR). Clinical and biochemical parameters were compared between both groups of PCOS. Associations between HOMA-IR and anthropometry, biochemistry and hormonal parameters between groups were determined separately.

RESULTS Sixty-four female with PCOS were enrolled. Comparison of biochemical analyses found that the free androgen index was higher and that LH and SHBG were lower in the overweight-obese group. HOMA-IR was greater in the overweight-obese group. In the overweight-obese group, there were statistically significant correlations between the HOMA-IR with the measured BMI, waist circumference, waist-height ratio, HDL, triglycerides, SHBG and the free androgen index. Interestingly, in the normal weight group, only the triglycerides, SHBG and the free androgen index had a statistically significant correlation with HOMA-IR.

CONCLUSIONS There were clear differences in the parameters related to insulin resistance between overweight-obese and normal weight PCOS; however, triglycerides, SHBG, and the free androgen index played a significant role in insulin resistance in both groups of PCOS.

KEYWORDS homeostasis model assessment (HOMA-IR), insulin resistance, polycystic ovary syndrome

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INTRODUCTION

Over the past few decades, the way of life of the Thai population has undergone rapid changes. Urbanization, less physical activity and consuming a high calorie diet has led to an increasingly overweight and obese population which has also been noted in reproductive age females. Twenty percent of young Thai females have been found to exhibit class I obesity and 12.9% have class II obesity (1). Increased body weight often results in abnormal menstruation in adolescents and adults. PCOS is one of the leading causes of abnormal menstruation in adolescent and adult Thai females and closely correlates with infertility, obesity and diabetes (2). A study of 600 university students in Thailand reported that the prevalence of Polycystic Ovary Syndrome (PCOS) in Thai adolescents was 5.29% (3). In a study of 250 PCOS patients from Siriraj Hospital, Thailand it was found that 49% of the cases had central obesity which was correlated with insulin resistance, abnormal lipid metabolism, and increased male sex hormone production (4). In addition to the Oral Glucose Tolerance Test (OGTT), the insulin resistance in PCOS was also assessed by calculations using the Homeostatic Model Assessment–Insulin Resistance (HOMA–IR) (5, 6). A percentage of these patients progressed to development of diabetes mellitus (7).

Classical PCOS patients have irregular menstruation or amenorrhea together with increased body hair, especially on the lip and upper chest wall as well as increased skin sebaceous glands and acne. Insulin resistance in these patients can lead to hyperinsulinemia. Insulin resistance and hyperinsulinemia cause increased hepatic gluconeogenesis, increased glycogenolysis and can finally progress to diabetes (8). Current concepts of PCOS pathophysiology include functional ovarian hyperandrogenism (FOH) and functional adrenal hyperandrogenism (FAH), polycystic ovarian morphology, LH excess, insulin resistance with hyperinsulinism and obesity. Additionally, FOH plays a principal role in PCOS pathophysiologic abnormality in which increased hyperandrogenemia results in negative feedback on gonadotropin release resulting in excess LH (9). Because PCOS is found in both normal weight and overweight-obese women, many clinical studies have compared biochemical markers between obese and non-obese women with PCOS in order to under-

stand the differences in pathophysiology of PCOS between obese and non-obese PCOS. A study in China found elevated of estradiol (E2) in obese PCOS women compared with non-obese PCOS individuals; however, there were no differences in lipid profile, LH, FSH, testosterone, prolactin, or anti-mullerian hormone (AMH) between the two PCOS groups (10). Additional studies were not able to identify differences in AMH or cardiac risk markers (CRP, ADMA, Hcy, PAI-1, VEGF) between obese and non-obese PCOS (11, 12). Because adiposity plays a role in PCOS, searching for certain adipokines, e.g., endotrophin, adiponectin, ghrelin, as a novel marker in PCOS has been undertaken. One study reported that endotrophin had an association with HOMA-IR, LH/FSH and total testosterone in PCOS (13).

Nevertheless, insulin resistance is the principal pathogenesis in PCOS and affects both obese and non-obese PCOS patients. Previous clinical studies have emphasized the impact of chronic low-grade inflammation on insulin resistance in PCOS (14). Elevation of inflammatory markers such as CRP, WBC concentration, and IL-18, MCP-1 concentration has been observed in PCOS compared with controls (15–18).

The purpose of this research was to identify differences between factors associated with insulin resistance in normal weight PCOS and overweight-obese PCOS women by comparing the associations of insulin resistance (HOMA-IR) with anthropometric, biochemical, and hormonal parameters in both groups of PCOS. This was the first study to compare the correlation of hormonal and biochemical parameters and insulin resistance between lean and overweight-obese adolescent PCOS patients in the Thai population.

METHODS

Study population

Sixty-four Polycystic ovary syndrome (PCOS) patients from the Obstetrics and Gynecology Department of the Medical Center Hospital, Mae Fah Luang University, Chiang Rai, Thailand, from January 2021 to December 2021 were enrolled. PCOS was diagnosed according to The Revised Rotterdam Criteria 2003 (19). Inclusion criteria consisted of amenorrhea/oligomenorrhea and polycystic ovarian morphology confirmed by ultrasonography in all participants done by one of two

gynecologists or by having hirsutism/signs of hyperandrogenemia. Our participants were classified as either PCOS phenotype A or phenotype D. Exclusion criteria were women who had diabetes, took anti-cholesterol medications, corticosteroids, used oral contraception (within 3 months prior to enrollment), had used either injected or implanted contraception, had taken anti-psychotic medications or weight lowering agents or who had previous ovarian surgery. Additionally, patients who had features of hyperthyroidism, hypothyroidism, cushing syndrome, or galactorrhea were also excluded from the study.

The research protocol was approved by the ethical committee of Mae Fah Luang University (COA 183/2020).

Clinical, anthropometric and biochemical measurements

After explaining the study process to the participants and obtaining informed consent, the participants underwent history taking and physical examination. Blood pressure was measured in both arms using a digital sphygmomanometer after a 20-minute rest. The average value of the blood pressure readings was used. Following the American College of Cardiology, pre-hypertension was defined as systolic blood pressure 120-129 mmHg and diastolic blood pressure less than 80 mmHg (20). Height and weight were measured for calculation of BMI. BMI at or above 23 kg/m² was defined as overweight and BMI above 25 kg/m² was defined as obese. Waist circumference was measured by one of the trained nursing staff in centimeters using a measuring tape at the mid-level between the lateral costal margin and iliac crest at full expiration with the patient in the standing position. Waist-height ratio was then calculated.

Acanthosis nigricans present on the back of the neck, axilla, or groin was recorded. The Ferriman-Gallwey scale was used to assess body hair for evidence of hyperandrogenemia. After 12 hours of fasting, blood samples were collected between 8.00 A.M. and 9.00 A.M. to measure plasma glucose, insulin, lipid profiles, TSH, FSH, LH, total testosterone, free testosterone, and SHBG.

Impaired fasting glucose was defined as a fasting plasma glucose from 100 mg/dL to less than 126 mg/dL. Diabetes was defined as fasting plasma

glucose ≥ 126 mg/dL. Metabolic syndrome was defined as agreed to by the International Diabetes Federation 2005, in which participants achieved 3 of 5 criteria including blood pressure $\geq 130/85$ mmHg, waist circumference ≥ 80 cm, triglycerides ≥ 150 mg/dL, HDL-cholesterol < 40 mg/dL, and fasting plasma glucose ≥ 100 mg/dL.

Insulin resistant state (HOMA-IR) was determined by the formula:

$$\text{HOMA-IR} = \frac{\text{Glucose (mg/dL)} \times \text{Insulin (\mu U/mL)}}{405}$$

The cut-off level of HOMA-IR > 2 was defined as insulin resistance (5).

Fasting plasma glucose, TSH, and lipid profiles were measured by photometry (Cobas C 501 analyzer series, Roche Laboratory, Indianapolis, USA). Insulin, FSH, LH, and SHBG were measured by electro-chemiluminescence assay. Free testosterone was calculated based on total testosterone, SHBG, and albumin. Free androgen index (FAI) was calculated using the formula:

$$\text{Total testosterone} = [(\text{nmol/L}) \div \text{SHBG (nmol/L)}] \times 100$$

Statistical analysis

Statistical analysis was performed using SPSS version 25. Data are presented as median and interquartile range. The Mann-Whitney U test was used for comparison of clinical and biochemical parameters, including HOMA-IR and free androgen index, between the normal weight and the overweight-obese group. Spearman correlation coefficient was used for assessment of correlation between HOMA-IR and anthropometric, biochemical, hormonal parameters in both the normal and overweight-obese groups.

RESULTS

There were 64 PCOS patients enrolled in this study (28 in the normal weight group and 36 in the overweight-obese group). In the overweight-obese group 13.9% showed pre-hypertension, 13.9% had impaired fasting glucose but none had diabetes. One of the twenty-eight cases (3.6%) in the normal weight group had metabolic syndrome, whereas eight of thirty-six cases (22.3%) in the overweight-obese group had metabolic syndrome (Table 1). Comparison of clinical features between the normal weight and the

overweight-obese group found that systolic and diastolic blood pressure were higher in the overweight-obese group. The Ferriman-Gallwey scale was also higher in this group compared to the normal weight group (Table 2).

Regarding biochemical parameters, the overweight-obese group had higher fasting insulin levels and triglycerides and lower HDL-cholesterol levels compared to the normal weight group.

Interestingly, the LH level in the normal weight was higher than in the overweight-obese ($p = 0.03$), but the FSH and LH/FSH ratio were not statistically significantly different. In addition, the free androgen index was higher in the overweight-obese group, which is compatible to the higher hirsutism found using the Ferriman-Gallwey scale. SHBG, as expected, was lower in the overweight-obese group ($p < 0.001$). Regarding insulin

Table 1. Prevalence of pre-hypertensive state, impaired fasting glucose, diabetes, metabolic syndrome and insulin resistance in study participants (n = 64)

	BMI < 23 kg/m ² n = 28	BMI ≥ 23 kg/m ² n = 36
Pre-hypertension	0 (0.0%)	5 (13.9%)
Impaired fasting glucose	0 (0.0%)	5 (13.9%)
Diabetes mellitus	0 (0.0%)	0 (0.0%)
Metabolic syndrome	1 (3.6%)	8 (22.3%)
Insulin resistance (HOMA-IR > 2)	2 (7.1%)	23 (64.9%)

BMI, body mass index; pre-hypertension, systolic blood pressure 120-129 mmHg and diastolic blood pressure less than 80 mmHg

Table 2. Comparison of anthropometric and laboratory parameters between BMI < 23 kg/m² with BMI ≥ 23 kg/m² (expressed as median and IQR with Mann-Whitney U test) $p < 0.05$

	BMI < 23 kg/m ² n = 28	BMI ≥ 23 kg/m ² n = 36	p-value
Age	24.00 (20.30-28.00)	22.00 (22.00-27.80)	0.749
Systolic blood pressure (mmHg)	110.00 (102.00-110.00)	120.00 (112.00-125.00)	< 0.001*
Diastolic blood pressure (mmHg)	70.00 (70.00 - 80.00)	80.00 (70.00 - 80.00)	< 0.001*
Height (cm)	158.30 (153.30-163.80)	159.00 (153.50-163.00)	0.082
Weight (kg)	52.10 (48.00-56.40)	74.80 (66.70-89.00)	< 0.001*
BMI (kg/m ²)	20.90 (19.70-21.90)	30.20 (25.80-34.40)	< 0.001*
Waist circumference (cm)	67.50 (63.90-71.50)	88.00 (79.40-97.30)	< 0.001*
Waist-height ratio	0.42 (0.41-0.44)	0.56 (0.49-0.61)	< 0.001*
Ferriman- Gallwey scale	6.00 (3.00- 9.70)	10.50 (5.30-16.80)	0.005*
Fasting plasma glucose (mg/dL)	86.00 (82.00-89.00)	87.00 (82.00-94.00)	0.238
Insulin (μU/mL)	5.20 (3.50-6.30)	13.40 (7.60-20.20)	< 0.001*
HOMA-IR	1.10 (0.70-1.50)	2.90 (1.60-4.30)	< 0.001*
Glucose insulin ratio (mg/dL)/(μU/mL)	16.80 (14.00-24.50)	6.50 (4.50-11.70)	< 0.001*
FAI	3.00 (1.90-5.80)	9.70 (5.50-14.60)	< 0.001*
Free testosterone (ng/dL)	0.57 (0.40-0.89)	1.09 (0.80-1.50)	< 0.001*
SHBG (nmol/L)	48.10 (30.76-70.00)	18.40 (13.73-27.52)	< 0.001*
LH (mIU/mL)	7.69 (5.30-13.64)	6.77 (4.15-8.52)	0.030*
FSH (mIU/mL)	5.34 (3.88-6.73)	4.98 (4.01-5.77)	0.310
LH/FSH ratio	1.60 (1.10-2.20)	1.40 (1.10-1.70)	0.170
Cholesterol (mg/dL)	193.00 (177.00-222.00)	192.00 (180.00-221.00)	0.756
Triglycerides (mg/dL)	79.00 (57.00-103.00)	121.00 (78.00-167.00)	< 0.001*
HDL - C (mg/dL)	65.00 (56.00-74.00)	46.00 (40.00-53.00)	< 0.001*
LDL - C (mg/dL)	110.00 (100.00-143.00)	119.00 (105.00-137.00)	0.593
TSH (μIU/mL)	1.58 (1.35-2.28)	1.90 (1.45-2.91)	0.077

BMI, body mass index; HOMA-IR, homeostasis model assessment-insulin resistance; SHBG, sex hormone binding globulin; LH, luteinizing hormone; FAI, free androgen index; FSH, follicular stimulating hormone; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TSH, thyroid stimulating hormone

resistance, HOMA-IR was significantly higher in the overweight-obese group than in the normal weight group (2.9 compared with 1.1), despite of having the same level of fasting plasma glucose (Table 2).

Correlations among BMI, waist circumference and waist-height ratio in the overweight-obese group showed indications that insulin resistance was related to possessing higher body fat. Analysis of the association between insulin resistance state (HOMA-IR) with anthropometric measurements revealed strong associations between weight, BMI, waist circumference, waist-height ratio and HOMA-IR in the overweight-obese group (Table 3). However, this study found no association between these parameters and HOMA-IR in the normal BMI group (Table 4).

Lipid profiles, both triglycerides and HDL cholesterol, were associated with HOMA-IR in overweight-obese individuals. Importantly, tri-

glycerides, free androgen index and SHBG had a strong association with HOMA-IR in both groups. However, this study found no association between gonadotropin (LH, FSH), LH/FSH ratio and HOMA-IR (Tables 3, 4).

DISCUSSION

This study was designed to compare clinical characteristics, hormonal and metabolic parameters, including insulin resistance, between PCOS women who had normal BMI and PCOS women who presented as being overweight or obese. A study by Wongwananuruk et al. showed 30 percent of PCOS women had hypertension (4). This was higher than in our study which found that 13.9% had pre-hypertension. This could be the result of the lower age of PCOS patients in our study. The 22.3% prevalence of metabolic syndrome in this study was similar to the prevalence in a study by Pantasri et al. (6).

Table 3. Association between insulin resistance (HOMA-IR) with anthropometric and laboratory parameters in the overweight-obese group, BMI ≥ 23 kg/m² (Spearman correlation statistical analysis); $p < 0.05$

	(HOMA-IR)	
	r	p-value
Age	-0.222	0.193
Systolic blood pressure (mmHg)	0.157	0.362
Diastolic blood pressure (mmHg)	0.062	0.721
Height (cm)	0.196	0.251
Weight (kg)	0.675	< 0.001*
BMI (kg/m ²)	0.687	< 0.001*
Waist circumference (cm)	0.669	< 0.001*
Waist - height ratio	0.616	< 0.001*
Ferriman- Gallway Score	0.263	0.121
FAI	0.602	< 0.001*
Free testosterone (ng/dL)	0.459	0.005*
SHBG (nmol/L)	-0.604	< 0.001*
LH (mIU/mL)	-0.150	0.382
FSH (mIU/mL)	-0.061	0.723
LH/FSH ratio	-0.306	0.069
Cholesterol (mg/dL)	0.020	0.906
Triglycerides (mg/dL)	0.366	0.028*
HDL - C (mg/dL)	-0.508	0.002*
LDL - C (mg/dL)	0.157	0.361
TSH (μ IU/mL)	0.057	0.743

BMI, body mass index; HOMA-IR, homeostasis model assessment-insulin resistance; SHBG, sex hormone binding globulin; LH, luteinizing hormone; FAI, free androgen index; FSH, follicular stimulating hormone; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TSH, thyroid stimulating hormone

Table 4. Association between insulin resistance (HOMA-IR) and anthropometric and laboratory parameters in the normal weight group (BMI < 23 kg/m²) (Spearman correlation statistical analysis); $p < 0.05$

	(HOMA-IR)	
	r	p-value
Age	-0.342	0.075
Systolic blood pressure (mmHg)	0.251	0.197
Diastolic blood pressure (mmHg)	0.217	0.268
Height (cm)	0.066	0.738
Weight (kg)	0.244	0.210
BMI (kg/m ²)	0.133	0.501
Waist circumference (cm)	0.299	0.122
Waist - height ratio	0.332	0.084
Ferriman- Gallway Score	-0.073	0.713
FAI	0.594	< 0.001*
Free testosterone (ng/dL)	0.543	0.003*
SHBG (nmol/L)	-0.654	< 0.001*
LH (mIU/mL)	0.257	0.186
FSH (mIU/mL)	0.093	0.637
LH/FSH ratio	0.222	0.257
Cholesterol (mg/dL)	-0.006	0.977
Triglycerides (mg/dL)	0.476	0.010*
HDL - C (mg/dL)	-0.140	0.477
LDL - C (mg/dL)	-0.063	0.752
TSH (μ IU/mL)	0.015	0.940

BMI, body mass index; HOMA-IR, homeostasis model assessment-insulin resistance; SHBG, sex hormone binding globulin; LH, luteinizing hormone; FAI, free androgen index; FSH, follicular stimulating hormone; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TSH, thyroid stimulating hormone

In this study we used an HOMA-IR of 2.0 as the cut off value for insulin resistance based on a study by Siriraj Hospital, Thailand, that compared HOMA-IR with the 75 g oral glucose tolerance test as a standard test for the detection of impaired glucose tolerance in 250 PCOS Thai women. The Siriraj study proposed a cut off value of HOMA-IR of 2.0 which had sensitivity of 84.0%, specificity of 61.0%, positive predictive value of 35.0% and negative predictive value of 93.8% (5). There was a limitation with employing HOMA-IR >2 as an indicator of insulin resistance because the cut-off point of HOMA-IR is different for different races, ages and genders (21).

Comparison of clinical characteristics, biochemical and hormonal features between PCOS women with normal weight those with overweight or obesity, found fasting plasma glucose levels did not differ between groups, while fasting insulin was significantly higher in the overweight-obese group as was HOMA-IR (Table 2). This finding is compatible with a study by Ketel et al. which reported using an euglycemic hyperinsulinemic clamp to assess insulin sensitivity and found that normal weight PCOS women had insulin sensitivity equivalent to normal weight women without PCOS, while overweight PCOS women had remarkably higher insulin resistance than overweight women without PCOS (22). Similarly, in adolescent Thai PCOS women, Sawathipanich et al. reported that obese adolescents with PCOS had significantly higher HOMA-IR scores than those of obese adolescent girls without PCOS (23). However, the use of HOMA-IR in young normal weight patients with PCOS has limitations. Fulghesu et al. reported that the prevalence of hyperinsulinemia in young normal weight PCOS was not detected by HOMA and suggested using a 3-hour OGTT to detect early onset metabolic derangement in young normal weight PCOS individuals (24).

Regarding differences in sex hormone levels between overweight-obese PCOS and normal weight PCOS females, our study found a higher free androgen index and higher levels of free testosterone in the overweight group and higher SHBG and LH in the normal weight group. These findings corresponded with those of previous studies (24, 25). Measurement of gonadotropins found that the LH level in the overweight-obese group was lower than that of the normal weight

group, which corresponds with a study by Katsikis et al. which showed that the LH level has a negative association with BMI (26). However, our study did not find differences in FSH or LH/FSH ratios between the groups. Differences in lipid profiles between overweight PCOS and normal weight PCOS women were similar to non PCOS populations, in that the overweight or obese population had higher triglycerides and lower HDL-cholesterol. Analysis of the association between insulin resistance index (HOMA-IR) and anthropometric factors in overweight-obese and normal weight PCOS found that in the overweight-obese group weight, BMI, waist circumference and waist-height ratio had a positive correlation with HOMA-IR, whereas in the normal BMI group those factors had no association with the insulin resistance index. These findings indicate that insulin resistance plays a major role in the pathophysiology of overweight-obese PCOS patients. These associations are explained by the relationship of both visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) with metabolic disturbance in PCOS women. A study in Austria showed that in overweight-obese PCOS women, lower abdomen and upper back SAT had a significantly positive correlation with insulin resistance but that there was no relationship between SAT and insulin resistance in normal weight PCOS patients. The same study also reported a significantly positive correlation between triglycerides and SAT thickness at the neck, biceps, upper back, front, and lateral side of the chest wall (26). Fruzzetti et al. confirmed that BMI was a good predictor of metabolic abnormalities in PCOS based on the strong association between BMI and HOMA-IR, fasting insulin, blood pressure, and lipid profiles (27).

In our study, waist-height ratio was used as the anthropometric factor instead of waist-hip ratio based on the results of a study in China by Liu et al. which included 1,124 PCOS women that found waist-height ratio was a more accurate anthropometric indicator for predicting insulin resistance (by area under ROC curve and sensitivity of the test to predict insulin resistance state) compared to waist-hip ratio, waist circumference, and BMI. A waist-height ratio of 0.49 was used as the cut off value in that study (28).

Regarding the association between lipid profiles and insulin resistance, our study found that

triglyceride levels had a positive association with HOMA-IR in both the normal weight and overweight-obese groups, while HDL cholesterol had a negative association with HOMA-IR only in the overweight-obese group. This finding indicates that low HDL cholesterol in overweight-obese subjects is of significance in predicting insulin resistance in that group.

The cut-off value of HOMA-IR as an indicator of insulin resistance can differ depending on the population being studied. A study in Thailand used an HOMA-IR of 2.0 (5) while a study from China used a cut off value of 2.77 (29).

In our study, PCOS women with features of metabolic syndrome had higher free testosterone and insulin levels compared with patients without features of metabolic syndrome, but the difference was not statistically significant due to the small sample size. The search for an association between insulin resistance index (HOMA-IR) and hormonal factors found in both the normal weight and overweight-obese groups that the free androgen index had a positive association with HOMA-IR and that SHBG had a negative association with HOMA-IR. However, this study did not find any association between gonadotropin (LH, FSH, LH/FSH ratio) and insulin resistance. Lee et al. studied clinical and biochemical features of PCOS women based on the quartile of HOMA-IR in 458 PCOS. That study showed significant differences in age, body weight, BMI, lipid profiles, glucose metabolic profiles, hs-CRP, apoprotein B, free testosterone, and SHBG between each of the quartiles of insulin resistance (30). Moran et al. studied the relationship between insulin resistance (expressed as glucose/insulin ratio) and gonadotropin dissociation (increased LH level with normal FSH level) in obese and non-obese women with PCOS. They found that insulin resistance did not appear to be related to gonadotropin dissociation (31).

CONCLUSIONS

PCOS is a disease with a high diversity in pathogenesis, clinical manifestations, and complications. Abnormal ovarian steroidogenesis and abnormal folliculogenesis can lead to anovulation, hyperandrogenemia, and infertility. Due to the diversity in the pathogenesis of PCOS, clinical management guidelines should be individualized and may differ between normal weight PCOS

and overweight-obese PCOS patients. This study showed differences in the association of factors with insulin resistance between normal weight PCOS and overweight-obese PCOS. Interestingly, triglycerides, the free androgen index and SHBG played a significant role in identifying the level of insulin resistance in both groups of PCOS. Further studies comparing normal weight and overweight-obese PCOS may lead to better understanding of insulin resistance in PCOS.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to report.

ADDITIONAL INFORMATION

Authors contribution

K.C.: conceptualization, methodology, implementation, data analysis, writing, review and editing; I.W.: methodology, subject recruitment; A.P.: investigations; P.C.: conceptualization, supervision.

Data availability statement

The data used in this study are not publicly available due to participant privacy but are available from the corresponding author upon reasonable request.

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