International Journal of Women's Health

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Metabolic Risk Factors Evaluation of Obstructive Sleep Apnea Among Patients with Polycystic Ovary Syndrome

Ling-Xia Ha[1], Jin-Juan Wang[1], Ying-Ying Yuan[2], Hu Liu[3], Jing Yang[3]

[1]Reproductive Center, Peking University First Hospital Ningxia Women and Children's Hospital, Ningxia Hui Autonomous Region Maternal and Child Health Hospital, Yinchuan, 750004, People's Republic of China; [2]Reproductive Medicine Center, General Hospital of Ningxia Medical University, Key Laboratory of Fertility Preservation and Maintenance of Ministry of Education, Ningxia Medical University, Yinchuan, 750004, People's Republic of China; [3]Ningxia Medical University, General Hospital of Ningxia Medical University, Yinchuan, 750004, People's Republic of China

Correspondence: Ling-Xia Ha, Reproductive Center, Peking University First Hospital Ningxia Women and Children's Hospital, Ningxia Hui Autonomous Region Maternal and Child Health Hospital, Yinchuan, 750004, People's Republic of China, Tel +86 13629500990, Email halingxia@126.com

Objective: Women diagnosed with PCOS exhibit a high prevalence of obstructive sleep apnea (OSA). This study aims to assess risk factors of OSA among patients with PCOS.

Methods: This retrospective study included 126 patients with PCOS who were categorized into an OSA group (n = 30) and a non-OSA group (n = 96) according to the apnea-hypopnea index (AHI). A control group comprised 72 patients without PCOS who presented during the same period for infertility due to fallopian tube, pelvic, or male factors. Patients with PCOS A multivariate logistic regression model was used to analyze independent risk factors for OSA in the PCOS group.

Results: Patients with PCOS had significantly higher AHI values and elevated values for various physical indicators, including body mass index (BMI) and neck, waist, and hip circumferences; prolactin (PRL); fasting plasma glucose (FPG); insulin (FINS); triglycerides (TG); homeostasis model assessment of insulin resistance (HOMA-IR); 2-hour postprandial glucose (2-hPG) and insulin (2-hINS); AHI; and oxygen desaturation index (ODI). Conversely, levels of high-density lipoprotein cholesterol (HDL-C) and lowest oxygen saturation ($LSaO_2$) were significantly lower ($p < 0.05$). AHI was positively correlated with BMI, neck circumference, waist circumference, hip circumference, 2-hPG, 2-hINS, and apolipoprotein B/ apolipoprotein A1 (apoB/apoA1) ratio ($p < 0.05$). BMI and neck circumference as independent risk factors for OSA in patients with PCOS ($p < 0.05$). The ROC curve analysis of BMI, neck circumference and BMI + neck circumference to predict PCOS patients with OSA showed that AUC=0.838,0.842 and 0.859, respectively, all exhibiting high sensitivity and specificity.

Conclusion: OSA in PCOS patients is linked to metabolic indicators. High neck circumference and BMI levels were independent risk factors, highlighting the need for OSA in routine PCOS screening, particularly in the context of metabolic dysregulation.

Keywords: body mass index, neck circumference, obstructive sleep apnea, polycystic ovary syndrome

Introduction

Polycystic ovary syndrome (PCOS) is among the most prevalent chronic endocrine–metabolic disorders affecting females of reproductive age, with a global prevalence estimated at 10–13%.[1,2] The condition is characterized by clinical and/or biochemical manifestations of hyperandrogenism, persistent anovulation, and polycystic ovarian morphology, often presenting with heterogeneous clinical features.[3] Advancements in medical knowledge and increased awareness of health-related conditions have led to broader recognition of PCOS as a complex, multisystem disorder. In addition to its impact on reproductive function, PCOS has been associated with a range of metabolic abnormalities, including hyperinsulinemia, insulin resistance (IR), obesity, and dyslipidemia.[4,5] Furthermore, evidence has suggested a significant association between PCOS and sleep-related disorders.[6] Individuals with PCOS are at increased risk of developing type 2 diabetes mellitus (T2DM), cardiovascular disease, and various

Received: 27 May 2025
Accepted: 31 October 2025
Published: 13 November 2025

mental health conditions compared to those without PCOS. As a systemic disorder, PCOS has been shown to substantially impair overall quality of life.

Obstructive sleep apnea (OSA), the most common form of sleep-disordered breathing, is a condition characterized by recurrent episodes of decreased oxygen saturation, sleep fragmentation, upper airway occlusion, and sleep analysis findings that reveal significant disruptions in breathing, specifically the apnea hypopnea index (AHI). Furthermore, evidence indicates that OSA contributes to a range of adverse health outcomes, including cardiovascular disease, metabolic syndrome, diabetes, chronic pulmonary disease, cognitive impairment, and reproductive function.[7–9] Yet, it is often overlooked and unassessed. A recent meta-analysis highlighted the high prevalence of OSA among women with PCOS.[10] Observational evidence in a limited number of studies suggests that metabolic outcomes are worsened among PCOS women who have OSA.[11,12] So, Early detection and management of OSA are therefore considered essential in clinical practice.

In this context, the present study aimed to identify metabolic risk factors associated with OSA among individuals diagnosed with PCOS. By analyzing key metabolic indicators, the study aimed to provide a basis for early screening and diagnosis of OSA in this population.

Materials and Methods

Study Population

This retrospective study included 126 individuals diagnosed with PCOS who received care at the Reproductive Medicine Center of the General Hospital of Ningxia Medical University between June 2023 and January 2025. Additionally, 72 patients without PCOS, presenting with infertility attributed to isolated tubal factor or male factor, were included as the control group during the same period. This study was approved by the Ethics Committee of the General Hospital of Ningxia Medical University (Approval number: KYLL-2024-0205). Written informed consent was obtained from all participants.

Inclusion Criteria

Eligible participants were aged 40 years or younger. Inclusion in the PCOS group was based on the diagnostic criteria established by the 2003 Rotterdam consensus.[4] Individuals in the control group were diagnosed with infertility, exhibited regular menstrual cycles, and demonstrated normal sex hormone profiles.

Diagnostic Criteria for PCOS

The diagnosis of PCOS was established based on the 2003 Rotterdam criteria, which require the presence of at least two of the following three conditions, following the exclusion of other causes of hyperandrogenemia and oligo-amenorrhea:[4]

1) Oligo-ovulation or anovulation;
2) Clinical and/or biochemical evidence of hyperandrogenemia;
3) Polycystic ovarian morphology on ultrasonography, defined as the presence of ≥ 12 small follicles with a diameter of 2–9 mm in at least one ovary and/or an ovarian volume > 10 cm^3.

Diagnostic Criteria for OSA

A diagnosis of OSA was established based on the occurrence of ≥ 30 episodes of apnea and hypopnea during a 7-hour nocturnal sleep period or an apnea-hypopnea index (AHI) of ≥ 5 events per hour, in accordance with established diagnostic guidelines.[11]

Exclusion Criteria

(1) Androgen-secreting tumors, Cushing syndrome, prolactinoma, congenital adrenal hyperplasia, and thyroid diseases.
(2) Presence of other types of sleep-disordered breathing, including central sleep apnea or mixed sleep apnea syndrome; comorbid neurological or psychiatric disorders; or other endocrine-metabolic conditions.
(3) Use of glucocorticoids or anti-androgen medications within the preceding 3 months, or ongoing use of insulin sensitizers for more than 6 months.
(4) History of chronic smoking, alcohol use disorder, or disruption of circadian rhythm.

(5) History of pharyngeal surgery or anatomical abnormalities such as severe nasal septal deviation or adenoid hypertrophy.

Research Methods

Baseline Information

Baseline demographic and anthropometric data included age, height, weight, neck circumference, waist circumference, and hip circumference.

Neck circumference was measured with the participant in a standing position and the head held upright. A flexible measuring tape was positioned horizontally at the level of the thyroid cartilage.

Waist circumference was measured with the participant standing upright, feet together, and breathing normally. The abdominal muscles were relaxed during the procedure. A soft measuring tape was positioned at the midpoint between the lowest point of the rib cage and the upper edge of the iliac crest. The measurement was taken at the end of a normal exhalation and before the next inhalation.

Hip circumference was measured as the horizontal circumference at the level of the most prominent point of the buttocks, using a non-elastic measuring tape while the participant remained in a standing position.

Laboratory and Clinical Parameters

Reproductive Endocrine Hormones

Fasting venous blood samples were collected from all participants between days 2 and 4 of the menstrual cycle. Follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL), total testosterone (T), estradiol (E2) and anti-Müllerian hormone (AMH) were measured using chemiluminescent analyzer (Beckman Coulter Inc, Fullerton, CA, USA).

Glucose and Lipid Metabolism Parameters

Serum glucose levels, insulin levels, total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) were measured on an automated biochemistry analyzer (Olympus 600, Olympus Diagnostica Gmbh, Ireland).Participants also underwent a 2-hour 75-g oral glucose tolerance test including 2-hour glucose and 2-hour insulin values.The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated according to the following equation: fasting serum glucose (FPG, mmol/l) × fasting insulin (FINS, mIU/l)/22.5.

Polysomnography (PSG) Monitoring Parameters

All patients were evaluated by a sleep medicine provider followed by formal testing for OSA with a polysomnography (PSG) (SF-A9,Hunan Wanmai Medical Technology Co, Ltd,Chin). OSA severity was defined based on apnea-hypopnea index (AHI) as mild (5–15/h), moderate (15–30/h), or severe (>30/h). From the PSG, information on AHI, lowest oxygen saturation (SpO2), time spent below SpO2 <89% and 4% oxygen desaturation index (ODI) was obtained.

Statistical Analysis

Statistical analyses were performed using SPSS version 26.0. The Shapiro–Wilk test was used to determine the normality of continuous data. Variables following a normal distribution were expressed as mean ± standard deviation ($\bar{x} \pm s$), and intergroup comparisons were conducted using independent sample *t*-tests. Non-normally distributed variables were expressed as median with interquartile range [M (P_{25}, P_{75})] and compared using the Mann–Whitney *U*-test.

Pearson's correlation analysis was used to evaluate linear associations between variables. Multivariate logistic regression analysis was employed to determine independent factors associated with the presence of OSA in patients with PCOS. Categorical data were analyzed using the chi-square test.

Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the predictive efficacy of body mass index (BMI) and neck circumference for the diagnosis of OSA. The area under the curve (AUC) and 95% confidence interval (CI) were calculated to evaluate diagnostic performance. A p-value of < 0.05 was considered statistically significant. GraphPad Prism 8.0 software was used to generate graphical representations.

Results

Comparison of Baseline Characteristics Between PCOS and Non-PCOS Groups

Participants diagnosed with PCOS exhibited significantly higher values of BMI, AMH, neck circumference, waist circumference, hip circumference, T, and LH compared to the non-PCOS group ($p < 0.05$). No statistically significant differences were observed between the two groups in terms of age, FSH, E2, or PRL levels ($p > 0.05$) (Table 1).

Comparison of Metabolic Indicators Between PCOS and Non-PCOS Groups

The PCOS group demonstrated significantly elevated levels of FINS, homeostatic model assessment for IR (HOMA-IR), TG, and apoB, along with reduced levels of HDL-C, relative to the non-PCOS group ($p < 0.05$). No statistically significant differences were detected in FPG, TC, LDL-C, apoA1, or apoB/apoA1 ratio between the two groups ($p > 0.05$) (Table 2).

Table 1 Comparison of Baseline Characteristics Between PCOS and Non-PCOS Groups

Group	PCOS Group *n* = 126	Non-PCOS Group *n* = 72	*t*/*Z*	*P*
Age (y)	28.53±4.05	27.50±4.53	1.804	0.073
BMI (25kg/m^2)	26.77±4.84	24.60±5.80	2.025	0.045*
AMH (ng/mL)	6.87±1.63	2.67±1.21	17.032	0.000*
Neck circumference (cm)	34.74±3.99	32.96±3.44	2.832	0.005*
Waist circumference (cm)	86.12±12.37	80.05±9.24	3.125	0.002*
Hip circumference (cm)	98.38±8.64	94.85±7.77	3.583	0.000*
FSH (mIU/mL)	6.38±1.61	6.22±1.40	0.558	0.578
LH (mIU/mL)	7.12 (4.43, 9.97)	4.45 (2.59, 8.36)	3.053	0.002*
E_2 (pg/mL)	46.25 (39.25, 55.48)	42.80 (32.53, 56.07)	0.732	0.464
PRL (ng/mL)	9.49 (7.52, 13.21)	9.29 (7.26, 13.06)	0.590	0.556
T (ng/dl)	33.37±11.80	27.46±9.49	2.828	0.005*

Note: *Significant; *t*-test.
Abbreviations: BMI, Body Mass Index; AMH, Anti-Müllerian Hormone; FSH, Follicle Stimulating Hormone; LH, Luteinizing Hormone; E2, Estradiol; PRL, prolactin; T, Testosterone.

Table 2 Comparison of Metabolic Indicators Between PCOS and Non-PCOS Groups

Group	PCOS Group *n* = 126	Non-PCOS Group *n* = 72	t	*P*
FPG (mmol/L)	5.15±0.61	5.01±0.45	1.552	0.122
FINS (mU/L)	16.27±7.34	11.68±4.02	4.297	0.000*
HOMA-IR	3.49±2.16	1.97±1.45	5.297	0.000*
2- hPG (mmol/L)	6.89±2.30	–		
2-hINS (mU/L)	89.61±51.97	–		
TG (mmol/L)	1.66±0.68	1.16±0.35	5.116	0.000*
TC (mmol/L)	4.68±0.81	4.60±0.75	0.591	0.555
HDL-C (mmol/L)	1.21±0.25	1.33±0.24	−2.964	0.003*
LDL-C (mmol/L)	2.98±0.79	2.76±0.51	1.868	0.064
apoA1 (g/L)	1.36±0.20	1.32±0.16	0.810	0.420
apoB (g/L)	0.89±0.22	0.80±0.12	2.137	0.035*
apoB/apoA1	0.67±0.19	0.61±0.13	1.428	0.156

Note: *Significant; *t*-test.
Abbreviations: FPG, Fasting Plasma Glucose; FINS, fasting insulin; 2-hBG, 2-hours Plasma Glucose; 2-hINS, 2-hours Insulin; TG, Triglyceride; TC, Total Cholesterol; HDL, High-density Lipoprotein Cholesterol; LDL, Low-density Lipoprotein Cholesterol.

Comparison of OSA-Related Indices Between PCOS and Non-PCOS Groups

Compared to the non-PCOS group, the PCOS group demonstrated significantly elevated AHI and ODI values, along with decreased $LSaO_2$. ($p < 0.05$) (Table 3).

Comparison of Baseline Characteristics Between OSA and Non-OSA Groups

Among the PCOS cohort, participants with OSA had significantly higher BMI, neck circumference, waist circumference, hip circumference, and PRL levels compared to those without OSA ($p < 0.05$). No statistically significant differences were noted in age, AMH, FSH, LH, E2, and T levels between the two groups ($p > 0.05$) (Table 4).

Comparison of Metabolic Indicators Between OSA and Non-OSA Groups

Participants with OSA exhibited significantly higher levels of FPG, FINS, HOMA-IR, 2-hPG, 2-hINS, and TG, along with lower levels of HDL-C, compared to those without OSA ($p < 0.05$). No statistically significant differences were observed in TC, LDL-C, apoA1, apoB, or apoB/apoA1 ratio ($p > 0.05$) (Table 5).

Comparison of Sleep Characteristics Between OSA and Non-OSA Groups in Patients with PCOS

Within the PCOS group, those diagnosed with OSA demonstrated significantly higher values for AHI and ODI, along with significantly lower minimum $LSaO_2$, compared to the non-OSA group ($p < 0.05$) (Table 6).

Table 3 Comparison of OSA Occurrence Between PCOS and Non-PCOS Groups

Group	PCOS Group *n* = 126	Non-PCOS Group *n* = 72	t	P
AHI (times/hour)	3.17±5.04	1.31±2.01	3.826	0.000*
ODI (times)	5.53±7.37	2.61±4.58	4.517	0.000*
LSaO2 (%)	88.48±4.39	94.27±5.36	−4.024	0.000*

Note: *Significant; *t*-test.
Abbreviations: AHI, Apnea Hypopnea Index; ODI, Oxygen depletion index.

Table 4 Comparison of Baseline Characteristics Between OSA and Non-OSA Groups

Group	OSA Group *n* = 30	Non-OSA Group *n* = 96	t/Z	P
Age (y)[t]	27.90±4.28	28.72±3.97	−0.978	0.330
BMI (25kg/m^2)[t]	31.61±3.70	25.32±4.17	7.174	0.000*
AMH (ng/mL)[t]	7.07±1.67	6.91±1.63	0.756	0.431
Neck circumference (cm)[t]	38.32±2.98	33.64±3.61	6.342	0.000*
Waist circumference (cm)[t]	96.89±11.95	82.84±10.54	6.088	0.000*
Hip circumference (cm)[t]	105.51±10.28	96.20±6.77	5.684	0.000*
FSH (mIU/mL)[t]	5.98±1.06	6.49±1.73	−1.202	0.121
LH (mIU/mL)[m]	7.27 (4.98, 8.15)	7.12 (4.40, 14.24)	0.500	0.617
E_2 (pg/mL)[m]	43.94 (37.01, 48.70)	47.09 (39.25, 56.80)	−1.096	0.273
PRL (ng/mL)[m]	8.11 (6.48, 9.60)	10.14 (7.71, 14.24)	−2.074	0.038*
T (ng/dl)[t]	36.04±11.72	32.47±11.53	1.130	0.262

Note: *Significant; m: Mann–Whitney U; t: *t*-test.
Abbreviations: BMI, Body Mass Index; AMH, Anti-Műllerian Hormone; FSH, Follicle Stimulating Hormone; LH, Luteinizing Hormone; E2, Estradiol; PRL, prolactin; T, Testosterone.

https://doi.org/10.2147/IJWH.S543184

Table 5 Comparison of Metabolic Parameters Between OSA and Non-OSA Groups

Group	OSA Group *n* = 30	Non-OSA Group *n* = 96	*t*	*P*
FPG (mmol/L)	5.35±0.87	5.09±0.48	2.025	0.045*
FINS (mU/L)	21.45±7.21	14.70±6.65	4.531	0.000*
HOMA-IR	4.70±2.75	3.11±1.80	3.680	0.000*
2-hPG (mmol/L)	7.73±3.38	6.64±1.84	2.227	0.028*
2-hINS (mU/L)	120.39±57.05	80.14±46.69	3.779	0.000*
TG (mmol/L)	1.98±0.73	1.56±0.64	2.721	0.008*
TC (mmol/L)	4.78±0.84	4.65±0.80	0.724	0.471
HDL-C (mmol/L)	1.11±0.20	1.23±0.26	−2.173	0.032*
LDL-C (mmol/L)	3.17±0.69	1.23±0.26	1.409	0.162
apoA1 (g/L)	1.32±0.17	1.36±0.21	−0.650	0.518
apoB (g/L)	0.93±0.17	0.88±0.24	0.860	0.393
apoB/apoA1	0.71±0.13	0.65±0.20	0.917	0.362

Note: *Significant; *t*-test.
Abbreviations: FPG, Fasting Plasma Glucose; FINS, fasting insulin; 2-hBG, 2-hours Plasma Glucose; 2-hINS, 2-hours Insulin; TG, Triglyceride; TC, Total Cholesterol; HDL, High-density Lipoprotein Cholesterol; LDL, Low-density Lipoprotein Cholesterol.

Table 6 Comparison of Sleep Characteristics Between OSA and Non-OSA Groups in Patients with PCOS

Group	OSA Group *n* = 30	Non-OSA Group *n* = 96	*t*	*P*
AHI (times/hour)	8.95±7.18	1.15±0.95	9.900	0.000*
ODI (times)	13.13±10.64	2.99±3.16	7.242	0.000*
LSaO2 (%)	83.41±4.68	90.18±2.67	−8.735	0.000*

Note: *Significant; *t*-test.
Abbreviations: AHI, Apnea Hypopnea Index; ODI, Oxygen depletion index.

Comparison of the Prevalence of Metabolic Syndrome Between OSA and Non-OSA Groups in Patients with PCOS

The prevalence of metabolic syndrome in OSA groups is higher than non-OSA groups in patients with PCOS ($p < 0.05$) (Table 7).

Correlation Analysis of Factors Associated with Elevated AHI Levels in Patients with PCOS

In patients with PCOS, AHI showed a statistically significant positive correlation with BMI, neck circumference, waist circumference, hip circumference, 2-hPG, 2-hINS, and the apoB/apoA1 ratio ($p < 0.05$). Although positive correlations

Table 7 Comparison of The Prevalence of Metabolic Syndrome Between OSA and Non-OSA Groups in Patients with PCOS

	OSA Group *n* = 30	Non-OSA Group *n* = 96	χ^2	*P*
With MetS	11(36.67%)	17(17.71%)	4.753	0.029*
Without MetS	19(63.33%)	79(82.29%)		

Note: *Significant; chi-square test.

were also observed between AHI and FPG, FINS, HOMA-IR, TG, TC, LDL-C, apoA1, and apoB, a negative correlation was observed with HDL-C. These associations were not statistically significant ($p > 0.05$) (Table 8).

Multivariate Logistic Regression Analysis of Independent Risk Factors for OSA in Patients with PCOS

A multivariate logistic regression analysis was performed with OSA status as the dependent variable and BMI, neck circumference, waist circumference, hip circumference, 2-hPG, 2-hINS, and the apoB/apoA1 ratio as independent variables. The analysis identified BMI (odds ratio (OR) = 1.446, 95% CI 1.236–1.692) and neck circumference (OR = 1.814, 95% CI 1.384–2.377) as independent risk factors for OSA in patients with PCOS ($p < 0.05$) (Table 9).

Table 8 Correlation Analysis Between AHI Levels and Clinical Parameters in Patients with PCOS

Indicator	r	P
BMI (kg/m^2)	0.414	0.000*
Neck circumference (cm)	0.568	0.000*
Waist circumference (cm)	0.353	0.000*
Hip circumference (cm)	0.383	0.000*
FPG (mmol/L)	0.022	0.800
FINS (mU/L)	0.065	0.473
2-hPG (mmol/L)	0.202	0.030*
2-hINS (mU/L)	0.175	0.061*
HOMA-IR	0.090	0.276
TG (mmol/L)	0.037	0.676
TC (mmol/L)	0.113	0.197
HDL-C (mmol/L)	−0.009	0.917
LDL-C (mmol/L)	0.072	0.412
apoA1 (g/L)	0.091	0.386
apoB (g/L)	0.109	0.297
apoB/apoA1	0.280	0.004*

Note: *Significant; Pearson's correlation analysis.
Abbreviations: BMI, Body Mass Index; FPG, Fasting Plasma Glucose; FINS, fasting insulin; 2-hBG, 2-hours Plasma Glucose; 2-hINS, 2-hours Insulin; TG, Triglyceride; TC, Total Cholesterol; HDL, High-density Lipoprotein Cholesterol; LDL, Low-density Lipoprotein Cholesterol.

Table 9 Multivariate Logistic Regression Analysis of Independent Factors for OSA in Patients with PCOS

Factor	B	SE	Wald χ^2	P	OR	95% CI
BMI (kg/m^2)	0.369	0.080	21.165	0.000*	1.446	1.236–1.692
Neck circumference (cm)	0.595	0.138	18.599	0.000*	1.814	1.384–2.377
Waist circumference (cm)	2.584	1.415	3.332	0.068	0.370	0.861–1.057
Hip circumference (cm)	0.060	0.085	0.501	0.479	1.062	0.899–1.253
2-hPG (mmol/L)	0.095	0.118	0.163	0.686	0.909	0.721–1.147
2-hINS (mU/L)	0.006	0.011	0.364	0.546	1.006	0.986–1.027
apoB/apoA1	0.043	0.041	1.079	0.299	1.044	0.963–1.132

Note: *Significant.
Abbreviations: BMI, Body Mass Index; 2-hBG, 2-hours Plasma Glucose; 2-hINS, 2-hours Insulin.

Table 10 Diagnostic Value of BMI and Neck Circumference for Predicting OSA in Patients with PCOS

Indicator	Cut-Off Value	AUC	Sensitivity	Specificity	*P*	95% CI
BMI (kg/m^2)	28.22	0.838	0.85	0.76	<0.001*	0.726–0.950
Neck Circumference (cm)	34.75	0.842	0.95	0.66	<0.001*	0.757–0.927
BMI+Neck Circumference	–	0.859	0.95	0.66	<0.001*	0.781–0.937

Note: *Significant; Receiver operating characteristic (ROC) curve analysis.
Abbreviations: BMI, Body Mass Index; AUC, area under the curve.

Predictive Value of BMI and Neck Circumference for OSA in Patients with PCOS

ROC curve analysis demonstrated that BMI had predictive value for OSA in patients with PCOS, with an AUC of 0.838 (95% CI: 0.726–0.950). At a BMI threshold of 28.22 kg/m^2, the sensitivity for diagnosing OSA was 85% and the specificity was 76%. ROC curve analysis for neck circumference predicting OSA in PCOS patients showed AUC of 0.842 (95% CI: 0.757–0.927); a cut-off value of 34.75 cm yielded a sensitivity of 95% and specificity of 66%. The combined predictive model incorporating BMI and neck circumference yielded an AUC of 0.859 (95% CI: 0.781–0.937), with a sensitivity of 95% and specificity of 66% for diagnosing OSA. (Table 10 and Figure 1).

Discussion

PCOS is a prevalent endocrine-metabolic disorder, affecting approximately 10 to 13% of females of reproductive age.[13,14] Typical clinical manifestations include menstrual irregularities, infertility, hyperandrogenism (HA), and polycystic ovarian morphology. These features are frequently accompanied by metabolic disturbances, including obesity, IR, and dyslipidemia. These comorbidities are recognized risk factors for diseases such as T2DM, cardiovascular diseases, cerebrovascular events, and endometrial carcinoma.[15]

Previous studies have indicated that approximately 50% to 70% of patients with PCOS exhibit varying degrees of IR.[16] The presence of IR leads to compensatory hyperinsulinemia, which promotes adipose tissue accumulation and increases the risk for metabolic conditions such as T2DM, obesity, and metabolic syndrome.[17] Dysregulation of lipid metabolism contributes to arterial fat deposition and atherosclerotic plaque formation, which in turn predisposes individuals to hypertension and coronary heart disease.[18–20] Furthermore, central obesity, particularly visceral fat

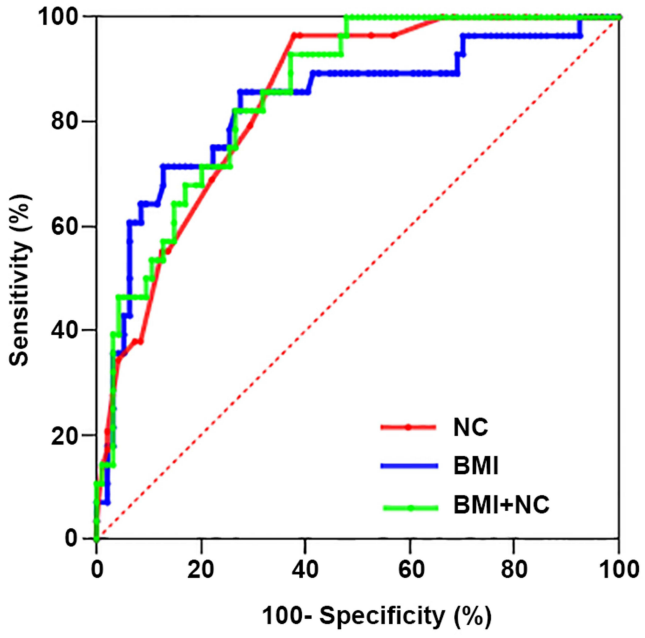

Figure 1 ROC Curve Analysis of BMI and Neck Circumference for Predicting OSA in Patients with PCOS.

accumulation, plays a key role in the development of IR. The presence of IR is often accompanied by lipid metabolism disorders, further increasing the risk of cardiovascular morbidity.[21]

Obesity has been recognized as a key risk factor for the development of OSA in patients with PCOS. Previous studies have reported that approximately half of all individuals with PCOS are either overweight or obese.[22] Among those with central (abdominal) obesity, the prevalence of OSA has been observed to reach 40%–60%, which is significantly higher than in the non-obese subgroup.

In the present study, patients with PCOS and coexisting obese demonstrated significantly higher AHI and ODI values, along with $LSaO_2$, compared to their non-obese counterparts. These findings suggest that obesity may exacerbate the severity of OSA among patients with PCOS, with a positive correlation observed between BMI and AHI, indicating that increasing BMI levels are associated with an elevated risk of OSA. These results align with findings from both domestic and international research, which have similarly identified obesity as a contributing factor to OSA in the PCOS population.[21,22]

Multiple pathophysiological mechanisms have been proposed to explain the interaction between obesity and the development of OSA. First, excessive adipose tissue deposition in the upper respiratory tract area may lead to mechanical pressure on surrounding structures, increased airflow resistance, eventual obstruction during sleep, thereby contributing to apneic events.[23] Second, increased intrathoracic pressure associated with obesity may restrict pulmonary expansion, impair respiratory function, and elevate the resistance of the upper respiratory tract, thereby promoting airway collapsibility. Third, obesity is associated with a chronic low-grade inflammatory state. Adipocytes particularly those located in visceral fat depots, secrete pro-inflammatory cytokines, which can lead to systemic inflammation and edema of the upper airway soft tissues, contributing to further airway narrowing and obstruction.[24] Fourth, obesity has been associated with alterations in sleep architecture, including an increased proportion of rapid eye movement (REM) sleep. REM sleep is characterized by pronounced muscle hypotonia, increasing susceptibility to airway collapse and the severity of OSA events.[25]

In summary, obesity contributes to OSA pathogenesis through anatomical mechanical, inflammatory, and neurophysiological pathways. Weight loss is commonly regarded as a key therapeutic strategy to alleviate OSA symptoms and mitigate associated risks in patients with PCOS.

IR has been shown to play a critical role in the pathogenesis and progression of PCOS. Its presence contributes not only to disturbances in the menstrual cycle, anovulation, and reduced fertility, but also to an increased risk of excessive weight gain, diabetes, and cardiovascular disease. Previous studies have reported that the prevalence of OSA among patients with T2DM ranges from 18% to 36%, while the probability of T2DM among patients with OSA is estimated to approach 40%, suggesting that IR may represent a pivotal pathophysiological link between T2DM and OSA.[26]

In the present study, patients with PCOS and concomitant IR exhibited higher AHI values compared to those without IR, indicating a potential association between IR and the development or exacerbation of OSA. These findings are consistent with those of Kahal et al who reported that the coexistence of PCOS and OSA was associated with more pronounced IR and impaired glucose regulation.[27] Similarly, a prospective study conducted by Chen demonstrated a positive correlation between HOMA-IR and OSA severity in patients with PCOS.[28]

Several mechanisms have been proposed to explain the association between IR and OSA. First, IR and obesity are frequently accompanied by low-grade systemic inflammation, which may result in increased relaxation and collapsibility of upper airway musculature and soft tissues, promoting obstruction during sleep. Second, IR is often accompanied by autonomic nervous system dysfunction. Excessive excitation of the sympathetic nervous system may lead to increased tension in airway muscles, thereby compromising the stability of the airway.[29] Third, intermittent hypoxia in patients with OSA has been shown to impair glucose homeostasis by reducing pancreatic β-cell responsiveness to glucose, thereby diminishing insulin sensitivity and perpetuating a cycle of metabolic dysfunction.[30] Early detection and management of IR in individuals with PCOS may improve metabolic and respiratory outcomes, thereby enhancing overall health status and quality of life in this population.

In patients with PCOS, the interplay between obesity and dysregulation of glucose and lipid metabolism has been observed to mutually reinforce each other, thereby contributing to the pathogenesis of OSA. Correlation analysis indicated that the AHI in patients with PCOS demonstrated significant positive associations with BMI, neck circumference, waist circumference, hip circumference, 2-hPG, 2-hINS, and the apoB/apoA1 ratio.

Subsequent multivariate logistic regression analysis identified BMI and neck circumference as independent predictors of elevated AHI levels. ROC curve analysis showed that the combination of BMI and neck circumference yielded a sensitivity of 95% and specificity of 66% for predicting the presence of OSA in patients with PCOS. These findings suggest that anthropometric measures may serve as practical, non-invasive screening tools for identifying individuals at elevated risk of OSA. Their application in clinical practice could facilitate timely diagnostic evaluation and support the implementation of targeted interventions.

In our study, it was found that compared with the non-OSA group, the prevalence of metabolic syndrome was higher in polycystic ovary syndrome (PCOS) patients with OSA. Among PCOS patients, obesity, insulin resistance, and hyperlipidemia are all closely associated with the development of metabolic syndrome.There is also some evidence to suggest that OSA may contribute to insulin resistance and glucose intolerance among women PCOS, and thus increase their metabolic risk.

However, the study had several potential limitations. First, the sample size is relatively small. polysomnography is the gold standard for diagnosing OSA.This requires patients to take the device home and return it to the hospital the next day. However, due to inconvenience, patients are uncooperative with the examination, making it difficult to collect a large number of cases in the study. Second, the proportion of obese participants is relatively high, this limited the generalizability of the results to other populations. So, large-sample, randomized controlled studies are still needed.

Conclusion

In conclusion, patients with PCOS represent a population at elevated risk for developing OSA. In this study, BMI and neck circumference demonstrated independent associations with AHI levels and demonstrated utility as predictive markers for OSA risk stratification. These findings highlight the importance of incorporating routine anthropometric assessments into the clinical evaluation of individuals with PCOS. For high-risk groups, weight loss may be one of the important measures to improve the symptoms of OSA and reduce the incidence of metabolic syndrome.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki (as was revised in 2013). The study was approved by Ethics Committee of the General Hospital of Ningxia Medical University (Approval number: KYLL-2024-0205). Written informed consent was obtained from all participants.

Acknowledgments

We are particularly grateful to all the people who have given us help on our article.

Funding

The Key Research and Development Program of Ningxia Hui Autonomous Region (Grant No.2024BEG02019).

Disclosure

The authors declare that they have no competing interests in this work.

References

1. Sadeghi HM, Adeli I, Calina D, et al. Polycystic ovary syndrome: a comprehensive review of pathogenesis, management, and drug repurposing. *Int J Mol Sci*. 2022;23(2):583. doi:10.3390/ijms23020583
2. Teede HJ, Tay CT, Laven J, et al. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. *Fertil Steril*. 2023;120(4):767–793. doi:10.1016/j.fertnstert.2023.07.025
3. Meier RK. Polycystic Ovary Syndrome. *Nurs Clin North Am*. 2018;53(3):407–420. doi:10.1016/j.cnur.2018.04.008

4. Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod*. 2004;19(1):41–47. doi:10.1093/humrep/deh098
5. Ganie MA, Chowdhury S, Malhotra N, et al. Prevalence, phenotypes, and comorbidities of polycystic ovary syndrome among indian women. *JAMA Netwo Open*. 2024;7(10):e2440583. doi:10.1001/jamanetworkopen.2024.40583
6. Kujanpää L, Arffman RK, Pesonen P, et al. Polycystic ovary syndrome presents as a multimorbid condition by age 50: birth cohort linkage to national register data. *Eur J Endocrinol*. 2024;190(6):409–420. doi:10.1093/ejendo/lvae057
7. Chinese Medical Association. Chinese Medical Journals Publishing House, Chinese Society of General Practice, et al. Diagnosis and treatment guidelines for obstructive sleep apnea in adults in primary care settings. *Chin J Gen Pract*. 2019;18(1):21–29.
8. Sunwoo BY, Owens RL. Sleep deficiency, sleep apnea, and chronic lung disease. *Sleep Med Clin*. 2024;19(4):671–686. doi:10.1016/j.jsmc.2024.07.012
9. Jurado-Robles I, Jurado-Gámez B, Feu Collado N, Molina-Luque R, Molina-Recio G. Influence of comorbidity and obesity on the occurrence of vascular events in obstructive apnoea treated with CPAP. *Nutrients*. 2024;16(18):3071. doi:10.3390/nu16183071
10. Kahal H, Kyrou I, Uthman OA, et al. The prevalence of obstructive sleep apnoea in women with polycystic ovary syndrome: a systematic review and meta-analysis. *Sleep Breath*. 2020;24(1):339–350. doi:10.1007/s11325-019-01835-1
11. Christ JP, Shinkai K, Corley J, Pasch L, Cedars MI, Huddleston HG. Metabolic and endocrine status associate with obstructive sleep apnea risk among patients with polycystic ovary syndrome. *J Clin Sleep Med*. 2024;20(6):871–877. doi:10.5664/jcsm.11012
12. Sam S, Tasali E. Role of obstructive sleep apnea in metabolic risk in PCOS. *Curr Opin Endocr Metab Res*. 2021;17:46–51. doi:10.1016/j.coemr.2021.01.002
13. Huddleston HG, Dokras A. Diagnosis and Treatment of Polycystic Ovary Syndrome. *JAMA*. 2022;327(3):274–275. doi:10.1001/jama.2021.23769
14. Dilliyappan S, Kumar AS, Venkatesalu S, et al. Polycystic ovary syndrome: recent research and therapeutic advancements. *Life Sci*. 2024;359:123221. doi:10.1016/j.lfs.2024.123221
15. Stener-Victorin E, Teede H, Norman RJ, et al. Polycystic ovary syndrome. *Nat Rev Dis Primers*. 2024;10(1):27. doi:10.1038/s41572-024-00511-3
16. Kohlhoff G, Kirwan R, Mushtaq S. The effect of vitamin D supplementation on markers of insulin resistance in women with polycystic ovarian syndrome: a systematic review. *Eur J Nutr*. 2024;63(8):2859–2869. doi:10.1007/s00394-024-03489-6
17. Alissa EM, Algarni SA, Khaffji AJ, Al Mansouri NM. Relationship between metabolic syndrome and polycystic ovary syndrome with special reference to c-reactive Protein. *J Obstet Gynaecol Can*. 2024;46(3):102255. doi:10.1016/j.jogc.2023.102255
18. Glintborg D, Rubin KH, Nybo M, Abrahamsen B, Andersen M. Cardiovascular disease in a nationwide population of Danish women with polycystic ovary syndrome. *Cardiovasc Diabetol*. 2018;17(1):37. doi:10.1186/s12933-018-0680-5
19. Jabczyk M, Nowak J, Jagielski P, Hudzik B, Borszcz J, Zubelewicz-Szkodzińska B. Interplay between lipid profile and anthropometric measures as indicators of cardiometabolic risk in women with polycystic ovary syndrome. *Front Endocrinol*. 2024;15:1398017. doi:10.3389/fendo.2024.1398017
20. Yang X, Jiang H. Research progress on cardiovascular diseases in patients with polycystic ovary syndrome. *China Med*. 2021;16(12):1911–1913.
21. Hellberg A, Salamon D, Ujvari D, Rydén M, Hirschberg AL. Weight changes are linked to adipose tissue genes in overweight women with polycystic ovary syndrome. *Int J Mol Sci*. 2024;25(21):11566. doi:10.3390/ijms252111566
22. Seravalle G, Grassi G. Sleep Apnea and Hypertension. *High Blood Press Cardiovasc Prev*. 2022;29(1):23–31. doi:10.1007/s40292-021-00484-4
23. Mokhlesi B, Scoccia B, Mazzone T, Sam S. Risk of obstructive sleep apnea in obese and nonobese women with polycystic ovary syndrome and healthy reproductively normal women. *Fertil Steril*. 2012;97(3):786–791. doi:10.1016/j.fertnstert.2011.12.024
24. Pinto JA, Ribeiro DK, Cavallini AF, Duarte C, Freitas GS. Comorbidities associated with obstructive sleep apnea: a retrospective study. *Int Arch Otorhinolaryngol*. 2016;20(2):145–150. doi:10.1055/s-0036-1579546
25. Hachul H, Polesel DN, Tock L, et al. Sleep disorders in polycystic ovary syndrome: influence of obesity and hyperandrogenism. *Rev Assoc Med Bras*. 2019;65(3):375–383. doi:10.1590/1806-9282.65.3.375
26. Hill EA, Williams LJ, Cooper SA, Riha RL. Objective and subjective prevalence of obstructive sleep apnoea/hypopnoea syndrome in UK adults with down syndrome: a strong marker for diurnal behavioural disturbances. *Brain Sci*. 2021;11(9):1160. doi:10.3390/brainsci11091160
27. Kahal H, Kyrou I, Uthman O, et al. The association between obstructive sleep apnea and metabolic abnormalities in women with polycystic ovary syndrome: a systematic review and meta-analysis. *Sleep*. 2018;41(7):10.1093/sleep/zsy085. doi:10.1093/sleep/zsy085
28. Chen L. Correlation between polycystic ovary syndrome and obstructive sleep apnea syndrome. *Pract J Gynecol Endocrinol*. 2023;10(30):1–3,8.
29. Protasiewicz-Timofticiuc DC, Bădescu D, Moța M, et al. Back to roots: dysbiosis, obesity, metabolic syndrome, type 2 diabetes mellitus, and obstructive sleep apnea-is there an objective connection? a narrative review. *Nutrients*. 2024;16(23):4057. doi:10.3390/nu16234057
30. Qian Y, Xu H, Wang Y, Yi H, Guan J, Yin S. Obstructive sleep apnea predicts risk of metabolic syndrome independently of obesity: a meta-analysis. *Arch Med Sci*. 2016;12(5):1077–1087. doi:10.5114/aoms.2016.61914