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ORIGINAL ARTICLE

Metabolic Syndrome in Lean Versus Obese Polycystic Ovarian Syndrome (PCOS) Phenotype

Navjyoti Goyal,[1] Sarita Sharma,[1, 2] Ruby Bhatia,[3] Sumeet Gupta,[1] Raghavendra Pai[4]

Abstract

Background/Aim: Polycystic ovarian syndrome (PCOS) is the most common metabolic, hormonal and endocrinological condition. Compared to lean, obese PCOS women are more likely to experience infertility, irregular menstruation, acanthosis and hirsutism. Aim of this study was to investigate the prevalence of metabolic syndrome in lean and obese PCOS patients as well as its correlation with age, body mass index (BMI) and obesity.

Methods: The 450 research participants in the cross-sectional study visited the tertiary care hospital. The individuals were chosen using the updated Rotterdam criteria and then divided into lean and obese groups according to their BMI. Clinical, biochemical, anthropometric, metabolic and demographic information about the patients were assessed and compared.

Results: The average age of obese patients was 29.8 ± 8.47 years, whereas the average age of lean patients was 24.8 ± 7.07 years. Obese PCOS individuals had significantly higher levels of all examined parameters, including age, height, weight, waist-to-hip ratio (WHR), hip and waist circumferences and BMI. Obese people were significantly more likely than thin people to have clinical parameters such infertility, alopecia, hirsutism, acanthosis and irregular menstruation. When comparing biochemical parameters such total testosterone (TT), luteinising hormone (LH): follicle stimulating hormone (FSH) ratio, thyroid stimulating hormone (TSH), dehydroepiandrosterone sulphate (DHEAS), the fasting oral glucose tolerance test (OGTT) and blood pressure, there were significant differences between PCOS patients who were thin and obese. Compared to lean individuals, obese patients showed higher levels of the lipid profile ($p < 0.001$). Metabolic syndrome was far more common in obese PCOS patients than in lean ones.

Conclusion: Study showed that both lean and obese persons might have metabolic syndrome. Consequently, all PCOS patients require monitoring for cardio-metabolic risk, irrespective of age, weight, or BMI.

Key words: Age; Body mass index; Polycystic ovary syndrome; Metabolic syndrome; Obesity.

1. M.M. College of Pharmacy, Maharishi Markandeshwar (Deemed to be university), Mullana-133207, Ambala, Haryana, India.
2. Amity Institute of Pharmacy, Amity University Madhya Pradesh, Gwalior, India.
3. Maharishi Markandeshwar Institute of Medical Science and Research, Mullana, Ambala Haryana, India.
4. Pharmacovigilance, India-South East Asia, Takeda Singapore, Singapore.

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Corresponding author:
SARITA SHARMA
E: sharmasarita.4416@gmail.com
T: 7083566900

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Introduction

Women in their reproductive years are susceptible to the metabolic, hormonal and endocrine disorder known as polycystic ovarian syndrome (PCOS).[1] Depending on the diagnosis, sample strategy, race, ethnicity and diagnostic criteria, PCOS affects more than 15–25 % of women

worldwide.[2] According to a survey by the World Health Organization (WHO), between 6 and 13 % of women who are of reproductive age have PCOS and up to 70 % of these women do not receive a diagnosis.[3] This condition is characterised by a large number of ovarian cysts, high testosterone levels and persistent anovulation.[4] A variety of features, including as insulin resistance, hirsutism, acne, obesity, dysmenorrhoea and *acanthosis nigricans* are clinical manifestations of PCOS.[5] It plays a significant role in infertility and is associated with an increased risk of endometrial cancer, depression, cardiovascular disease, metabolic syndrome, diabetes and obstructive sleep apnoea (OSA).[6] To be diagnosed with this condition, the patient must have signs of hyperandrogenism, oligo or anovulation, or polycystic ovarian morphology.[7] The pathophysiology of PCOS includes aberrant gonadotropin-releasing hormone (GnRH) pulsation, insulin resistance, hyperandrogenism, ovulatory dysfunction and an imbalance in the hypothalamus-pituitary-ovary (HPO) axis.[8]

Studies have indicated a correlation between PCOS and a higher body mass index (BMI). With a frequency of 38–88 %, being overweight or obese is very prevalent among PCOS patients.[9] Hyperandrogenism, insulin resistance and an irregular distribution of androgenic body fat might be the cause of this.[10] However, PCOS can also occurs in lean patient with prevalence of 20-50 % as per several studies. But still, there is very limited study conducted on lean PCOS patients. The phenotypic, metabolic, haematologic and neurologic features of lean PCOS women differ from those of obese PCOS women. Comprehending the distinctions between two subgroups is essential for devising a suitable management strategy.[11]

Metabolic syndrome is a cluster of interrelated metabolic abnormalities that increase the risk of cardiovascular disease and type 2 diabetes. It is characterised by central obesity, dyslipidaemia (elevated triglycerides and/or reduced HDL cholesterol), elevated blood pressure and impaired glucose regulation. The presence of three or more of these components, as defined by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) or other international guidelines, constitutes a diagnosis of metabolic syndrome. According to a number of studies, up to 38–47 % of women with PCOS have metabolic syndrome, making it a rather common disorder in this population. Hypertension, insulin resistance, abdominal obesity, dyslipidaemia and hyperandrogenism are the components of the metabolic syndrome linked to PCOS. As a result, metabolic syndrome affects 43 % of adult women and around one-third of PCOS teens.[12] In PCOS patients, an excess of testosterone initiates a vicious cycle of metabolic problems. By producing hyperinsulinemia, which in turn raises the production of androgen from the ovaries and adrenal glands, hyperandrogenism is thought to have a role in the formation of visceral fat.[13] Those who have metabolic syndrome are five times more likely to develop type II diabetes. Understanding the connections between metabolic dysfunctions and PCOS will lead to a better comprehension of customised therapy for effective PCOS management.[14]

However, due to little information on relationship of metabolic syndrome with PCOS with respect to age, BMI and obesity, a cross-sectional study was carried out at a hospital with tertiary care. The purpose of this study was to investigate the metabolic syndrome prevalence in PCOS patients within lean and obese bodies, as well as the relationship between metabolic syndrome and age, BMI and obesity.

Methods

An outpatient obstetrics and gynaecology department in a tertiary care hospital served as the site of this hospital-based cross-sectional research. All reproductive women between the ages of 15 to 45 years, who had been diagnosed with PCOS using the 2003 modified Rotterdam criteria, made up the research population. Two of the three conditions have to be met in order for the Rotterdam definition to apply: i) Male baldness, acne, or hirsutism are indicators of clinical hyperandrogenism; and higher levels of androstenedione, dehydroepiandrosterone sulphate, or total or free testosterone are biochemical hyperandrogenism; ii) A menstrual interval of more than 35 days or less than eight cycles in a 12-month period is known as amenorrhea or oligomenorrhea, iii) either transvaginal or abdominal ultrasounds that reveal polycystic ovaries, often indicate at least one ovary with a minimum ovarian capacity of 10 mL and/or 12 or more peripheral follicles that range in diameter 2-9 mm. In addition to other causes of hyperandrogenism,

women with a history of Cushing's syndrome, diabetes mellitus androgen-secreting tumours, or congenital adrenal hyperplasia were not included. Participation was prohibited for patients who were unable to give informed permission. Metabolic syndrome was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria, applying the South Asian–specific cut-off for central obesity. The diagnosis required the presence of three or more of the following: waist circumference ≥ 80 cm, fasting plasma glucose ≥ 100 mg/dL or treatment for hyperglycaemia, serum triglycerides ≥ 150 mg/dL or treatment for elevated triglycerides, high-density lipoprotein (HDL) cholesterol < 50 mg/dL or treatment for low HDL and blood pressure ≥ 130/85 mm Hg or use of antihypertensive medication.

The anticipated sample size was 450 women with PCOS, with a power of 80 % and an alpha value of 0.05. The study was started after the approval from the institutional ethics committee. Prior to the research participants being recruited, written informed consent was acquired. The participants were fully informed about the study's methodology. The research participants' information was gathered using a semi-structured questionnaire that had been pre-tested. To determine if they fulfilled the study's eligibility conditions, their whole medical history was obtained, clinical exams were performed and ultrasounds were performed. The demographic data included age, marital status, social level, education and ethnicity of the patient. The obstetric and gynaecological history was collected, along with details on recent weight gain, irregular menstruation and infertility.

A systematic framework was also used to gather clinical characteristics of hyperandrogenism, such as hirsutism, alopecia, acne and dysmenorrhoea. The waist-hip ratio was calculated, blood pressure and the modified Ferriman-Gallwey scores. Standard instruments were used to record anthropometric variables such as measurements of height (in cm), weight (in kg), BMI, hip circumference (in cm) and waist circumference (in cm). To calculate BMI, weight in kilograms was divided by height in meters squared. The waist circumference was measured in the horizontal plane, midway between the lowest ribs and the iliac crest. A measurement of hip circumference was made while the subjects were standing upright using non-stretchable plastic tape at the level of the hips' greatest lateral extension. To measure biochemical indicators, including the fasting oral glucose tolerance test (OGTT) and the lipid profile result, an automated biochemical analyser was used. The biochemical tests were performed on PCOS patients as part of standard examinations. The following serum levels were also measured: follicle stimulating hormone (FSH), luteinising hormone (LH), thyroid stimulating hormone (TSH), total testosterone (TT), dehydroepiandrosterone sulphate (DHEAS) and others.

Each member of the research population was divided into two groups according to their BMI (normal, overweight, obese and underweight). Those with normal BMI and underweight (18.5-24.9) were classified as having lean PCOS. Obese PCOS was defined as PCOS in patients who were overweight or obese (BMI ≥ 25). After being imported into Microsoft Excel 2013, all of the data was examined using IBM SPSS for Windows version 26.0. The parameters for each subject were recorded as mean ± SD. The two groups were compared using an independent t-test to see if the data were regularly distributed. Depending on the circumstance, proportions were compared using the Chi-square test. All statistical tests were two-sided and performed at a significance threshold of $p < 0.05$.

Results

Participants in the study were divided into groups based on their body mass index, in accordance with World Health Organisation (WHO) criteria and different age category (Table 1). A total of 232 patients (51.5 %) were categorised as obese PCOS patients because they were overweight or obese, whereas 218 patients (48.5 %) were categorised as lean PCOS patients because they were underweight or of normal weight. It also displays the age distribution of the study population. 101 (22.5 %) of the patients were between the ages of 15 and 20, whereas the majority of research volunteers 113 (25.2 %) were between the ages of 21 and 25.

The socio-demographic profile of the research participants is shown in Table 2. Rural areas accounted for the majority of obese patients (144, or 55.17 %) and lean patients (117, or 44.82 %).

Table 1: Distribution of patients with polycystic ovarian syndrome (PCOS) in relation to body mass index (BMI) and age

Parameter	N	%
BMI (kg/m^2)		
< 18.5 (Underweight)	39	8.75
18.5-24.9 (Normal)	179	39.75
25.0-29.9 (Overweight)	154	34.25
≥ 30 (Obese)	78	17.25
Age (years)		
15-20	101	22.50
21-25	113	25.20
26-30	99	22.00
31-35	68	15.20
36-40	31	6.90
41-45	38	8.20

Out of all the obese patients, 153 (60.23 %) were married, but only 101 (39.76 %) of the total lean individuals were. There were 184 lean patients (47.17 %) and 206 obese individuals (52.82 %) in the initial cases. The bulk of participants were either undergrad 160 (35.6 %) or graduates 138 (30.7 %) with a degree or above. According to the modified Kuppuswamy scale, the most prevalent socioeconomic category among patients who were obese (108 or 51.1 %) and lean (103 or 48 %), making up 211 (46.9 %) of the population overall, was lower middle class (III).

The clinical profile and BMI (lean versus obese) association for women with PCOS is shown in Table 3. Infertility was more common in PCOS-afflicted women with higher BMI (70 (66 %)) than in those with lean PCOS (36 (33.96 %); p = 0.00). Obese PCOS patients had statistically insignificantly higher rates of depression, menstrual irregularity, painful periods, clots, acne, amenorrhoea, alopecia and exhaustion than lean PCOS patients. Lean individuals had higher rates of oligomenorrhoea 145 (50.6 %) than obese ones 141 (49.3 %) (p > 0.05). Obese individuals had a statistically significant higher prevalence of acanthosis nigricans 109 (83.2 %) compared to thin individuals 22 (16.7 %) (p = 0.00). Mood swings, recent weight gain and irregular sleep were higher in high BMI participants, whereas pain in abdomen was higher in low BMI participants. Based on the Modified Ferriman Gallwey score, hirsutism is classified as either non-existent, normal, mild, moderate, or severe hair growth. It is a clinical indicator of hyperandrogenism. Severe hirsutism was uncommon, with 302 (67 %) of the individuals having either no hirsutism or very moderate symptoms.

The hormonal, anthropometrical and biochemical characteristics of lean PCOS patients and obese PCOS patients are contrasted in Table 4. In comparison to the lean group, the obese group was

Table 2: Socio-demographic characteristics of polycystic ovarian syndrome (PCOS) phenotypes

Parameter	Obese (N = 240)	Lean (N = 210)	Total (N = 450)
Ethnicity			
Rural	144 (55.2)	117 (44.8)	261 (58.0)
Urban	93 (49.2)	96 (50.7)	189 (42.0)
Marital status			
Married	153 (60.2)	101 (39.8)	254 (56.5)
Unmarried	84 (42.9)	112 (57.1)	196 (43.6)
Cases			
Initial	206 (52.8)	184 (47.2)	390 (86.7)
Follow-Up	31 (51.7)	29 (48.4)	60 (13.4)
Education			
Primary education	20 (62.5)	12 (37.5)	32 (7.2)
Secondary education	70 (58.4)	50 (41.7)	120 (26.7)
Under-graduation	85 (53.1)	75 (46.8)	160 (35.6)
Graduation or more	78 (56.5)	60 (43.4)	138 (30.7)
Socioeconomic status			
Lower (V)	10 (58.8)	7 (41.2)	17 (3.8)
Upper lower (IV)	64 (59.2)	44 (40.7)	108 (24.0)
Lower middle (III)	108 (51.1)	103 (48.0)	211 (46.9)
Upper middle (II)	54 (52.9)	48 (47.0)	102 (22.7)
Upper (I)	6 (50.0)	6 (50.0)	12 (2.7)

Data are presented as n (%) for categorical variables.

Table 3: Polycystic ovarian syndrome (PCOS) comorbidities and clinical symptoms

Parameter	Obese (N = 240)	Lean (N = 210)	Total (N = 450)	p-value
Infertility	70 (66.0)	36 (34.0)	106 (23.6)	< 0.006
Depression	84 (56.4)	65 (43.6)	149 (33.2)	0.101
Menstrual irregularity	216 (53.1)	191 (46.9)	407 (90.5)	0.262
Painful periods	138 (52.2)	126 (47.7)	264 (58.7)	0.150
Clots	126 (52.5)	114 (47.5)	240 (53.4)	0.415
Acne	89 (53.2)	78 (46.7)	167 (37.2)	0.539
Oligomenorrhoea	141 (49.3)	145 (50.6)	286 (63.6)	0.127
Amenorrhoea	80 (55.9)	63 (44.1)	143 (31.8)	0.369
Alopecia	138 (54.7)	114 (45.2)	252 (56.0)	0.207
Acanthosis nigricans	109 (83.2)	22 (16.7)	131 (29.2)	< 0.001
Recent weight gain	185 (84.1)	35 (15.9)	220 (48.9)	< 0.001
Fatigue	184 (51.9)	170 (48.1)	354 (78.7)	0.256
Mood swings	124 (56.1)	97 (43.8)	221 (49.2)	0.030
Irregular sleep	142 (55.2)	115 (44.7)	257 (57.2)	0.040
Pain in abdomen	53 (43.4)	69 (56.5)	122 (27.2)	0.020
Hirsutism				
Absence	79 (51.6)	74 (48.3)	153 (34.0)	
Mild	90 (60.4)	59 (39.5)	149 (33.0)	
Moderate	15 (60.0)	10 (40.0)	25 (5.6)	0.231
Normal	58 (51.7)	54 (48.2)	112 (24.9)	
Severe	10 (90.9)	1 (9.1)	11 (2.5)	

Data are presented as n (%) for categorical variables. Comparisons between groups were performed using chi-square test for categorical variables.

larger in terms of age, height, weight, waist circumference (WC), hip circumference (HC), waist to-hip ratio (WHR) and BMI. The statistical significance of these discrepancies was established. Lean and obese PCOS patients differed significantly ($p < 0.05$) in several parameters, including haemoglobin, erythrocyte sedimentation rate (ESR), pulse rate (PR), DHEAS, total testosterone, FSH, thyroid stimulating hormone (TSH), LH:FSH and the fasting oral glucose tolerance test (OGTT). However, the study found no significant change in the levels of prolactin, LH, Triiodothyronine (T3) and thyroxine (T4). Every lipid indication Low density lipoprotein (LDL), high density lipoprotein (HDL), triglycerides, cholesterol and very low-density lipoprotein (VLDL) were all greatly elevated in the obese group compared to the thin group ($p = 0.00$).

Table 4: Comparison of hormonal, anthropometrical and biochemical parameters in obese and lean polycystic ovarian syndrome (PCOS) phenotypes

Parameter (N = 450)	Obese (N = 240)	Lean (N = 210)	p-value
Age (years)	29.80 ± 8.47	24.80 ± 7.07	0.006
Height (cm)	158.02 ± 8.31	152.01 ± 7.70	0.005
Weight (kg)	69.97 ± 8.77	54.05 ± 8.11	< 0.001
BMI (kg/m^2)	30.02 ± 4.28	24.37 ± 3.50	< 0.001
WC (cm)	98.42 ± 6.63	82.36 ± 5.31	< 0.001
HC (cm)	107.55 ± 7.91	88.44 ± 7.35	< 0.001
WHR	0.94 ± 1.02	0.88 ± 1.12	< 0.001
SBP (mm Hg)	126.78 ± 12.27	106.19 ± 11.37	< 0.001
DBP (mm Hg)	75.04 ± 7.48	78.25 ± 8.20	0.020
PR (bpm)	85.25 ± 6.15	82.22 ± 5.33	< 0.001
Total testosterone (ng/mL)	16.05 ± 4.90	14.58 ± 6.22	< 0.001

LH	10.40 ± 5.65	8.46 ± 4.99	0.050
FSH	4.57 ± 3.00	3.71 ± 2.81	< 0.001
LH : FSH ratio	1.82 ± 0.41	2.02 ± 0.49	< 0.001
T3 (ng/mL)	1.93 ± 1.76	2.69 ± 2.23	0.340
T4 (ug/mL)	9.15 ± 1.79	8.57 ± 1.80	0.120
TSH (uIU/mL)	4.61 ± 2.05	3.48 ± 1.10	< 0.001
DHEAS (ug/dL)	175.12 ± 87.95	145.29 ± 89.96	0.006
Prolactin (ng/mL)	19.15 ± 11.63	18.48 ± 15.22	0.622
OGTT (mg/dL)	101.21 ± 34.77	85.17 ± 31.24	< 0.001
Haemoglobin (g %)	12.68 ± 1.40	10.62 ± 1.52	0.015
ESR	20.29 ± 10.27	15.73 ± 6.96	< 0.001
VLDL (mg/dL)	114.26 ± 37.59	96.73 ± 29.09	< 0.001
HDL (mg/dL)	60.64 ± 13.75	55.47 ± 10.87	< 0.001
LDL (mg/dL)	153.29 ± 41.60	136.38 ± 39.84	< 0.001
Cholesterol (mg/dL)	210.42 ± 45.73	178.53 ± 26.09	< 0.001
Triglycerides (mg/dL)	151.85 ± 41.65	105.67 ± 42.37	< 0.001

Data are presented as mean ± SD for continuous variables. Comparisons between groups were performed using Student's t-test for continuous variables; BMI: body mass index; WC: waist circumference; HC: hip circumference; WHR: waist to-hip ratio; SBP: systolic blood pressure; DBP: diastolic blood pressure; PR: pulse rate; LH: luteinising hormone; FSH: follicle stimulating hormone; T3: triiodothyronine; T4: thyroxine; TSH: thyroid stimulating hormone; DHEAS: dehydroepiandrosterone; OGTT: fasting oral glucose tolerance test; ESR: erythrocyte sedimentation rate; LDL: low density lipoprotein; HDL: high density lipoprotein; VLDL: very low density lipoprotein;

Table 5: Metabolic syndrome prevalence in polycystic ovarian syndrome (PCOS) individuals (obese and lean)

Metabolic syndrome	Obese PCOS	Lean PCOS	Total	p-value
Yes	80 (57.97)	58 (42.02)	138 (30.70)	< 0.001
No	150 (48.07)	132 (42.30)	312 (69.40)	

Data are presented as n (%) for categorical variables. Comparisons between groups were performed using chi-square test for categorical variables.

Metabolic syndrome affected about 58 (42.02) of lean people and 80 (57.97 %) of obese patients (Table 5). Metabolic syndrome was seen in 138 (30.7 %) of PCOS patients overall.

Discussion

PCOS is a prevalent condition affecting women of reproductive age, increasingly manifesting during adolescence. It is characterised by obesity, hyperandrogenism and insulin resistance, with obesity likely being the primary cause. A study indicates that obese PCOS patients show a significantly higher prevalence than their lean counterparts, although normal or underweight individuals can also have PCOS. Irregular periods, hyperandrogenism and elevated testosterone levels are common in all PCOS patients, irrespective of BMI.

According to Ali et al,[15] Hamed et al[16] and Abdelazim et al,[17] PCOS patients exhibit higher BMI, predominantly falling within the overweight and obese categories. Factors such as sedentary lifestyles, environmental influences, genetics and access to obesogenic foods are linked to this obesity prevalence. In this research, obese PCOS patients showed significantly elevated lipid profiles, OGTT, TSH, blood pressure and total serum testosterone levels compared to lean patients. Infertility rates, due to ovarian dysfunction, are higher in obese women. Increased insulin resistance in these patients leads to lower HDL levels and higher triglycerides, contributing to greater risks of hypertension and cardiovascular disease. Moreover, hyperinsulinemia results in heightened blood levels of free testosterone due to decreased sex hormone binding globulin (SHBG) among the obese.[18]

Presented research indicates that the prevalence of PCOS is highest among individuals aged 21 to 25, aligning with earlier data from Delhi-NCR, which reported a 17.4 % prevalence in this age group. This suggests that the early reproductive years are crucial for PCOS onset, particularly in college-age women in metropolitan areas, em-

phasising the need for early screening and preventative measures.[19] It is also significant to highlight that some of the observed group differences may have been influenced by the fact that the obese individuals were older than the lean PCOS women.

Many PCOS patients face a heightened risk of developing metabolic syndrome, particularly those who are obese.[20] This study indicates that metabolic syndrome prevalence is more significant in obese PCOS patients compared to lean ones, aligning with findings from other researchers like Karee et al[21] and Kaur et al.[22] According to Kayali et al,[23] infertile PCOS women also exhibit higher rates of metabolic syndrome than their non-PCOS counterparts. Factors such as visceral obesity, insulin resistance and chronic low-grade inflammation contribute to this risk. Interestingly, our study found a higher prevalence of metabolic syndrome in lean PCOS women, diverging from earlier reports, potentially due to variations in diagnostic criteria, ethnic susceptibility and methodological differences. Additionally, factors like age and lack of data on visceral fat or inflammatory markers may have influenced these results.

Current research indicates that participants with a higher BMI exhibit more clinical symptoms of conditions such as depression, infertility, acne, hirsutism, alopecia and irregular menstruation. Weight fluctuations can disrupt hormonal balance and trigger ovulation, yet no significant statistical differences were found between PCOS groups. Obesity exacerbates reproductive issues related to PCOS and contributes to metabolic problems, including diabetes, hypertension, endometriosis, cardiovascular disease and dyslipidaemia.

Numerous studies indicate that metabolic abnormalities significantly affect women with PCOS, with prevalence rates varying by phenotype and obesity levels. A study of Indian women revealed that 17.8 % of PCOS patients had metabolic syndrome compared to 3.3 % of controls, with higher rates in obese phenotypes and notable instances in lean subgroups (8.3 % in phenotype A and 2 % in phenotype D).[24] According to another clinic-based study in Southern India, 53.3 % of PCOS women met the modified NCEP ATP III metabolic syndrome criteria.[25] Recent research also demonstrated that lean PCOS is associated with metabolic disturbances, affirming the issue across both lean and obese cases, though more pronounced in the latter.[26]

A multimodal approach is essential for managing PCOS, incorporating therapies tailored to the patient's age, BMI and comorbidities. Interventions aim to address insulin resistance, metabolic abnormalities and hormonal irregularities. Weight loss through lifestyle changes has shown benefits for obese PCOS patients, improving insulin sensitivity. Women with a normal BMI should focus on moderate lean proteins, healthy fats and complex carbohydrates while engaging in regular exercise and limiting sugar and processed food intake.[27] The investigation highlights the importance of early detection of metabolic risk in both obese and lean PCOS phenotypes, suggesting that targeted treatments can be guided accordingly. Obese patients should focus on weight loss, while lean patients are advised to maintain their weight. Personalised care pathways should consider age, BMI and metabolic indicators to help gynaecologists identify and treat metabolic syndrome in PCOS patient early, potentially reducing morbidity from preventable diseases.

This study has several limitations that should be considered when interpreting the findings:

Study design – The cross-sectional nature of the study limits the ability to establish causal relationships. Future case–control or cohort studies with larger sample sizes are needed to strengthen the evidence.

Study setting – As the research was conducted in a tertiary care hospital, the findings may not be fully representative of the general community.

Measurement scope – Visceral fat measurements and inflammatory markers (eg C-reactive protein, adiponectin) were not assessed. Inclusion of these parameters could provide further insights into phenotype-specific risks.

Age difference between groups – The obese participants were older than the lean PCOS women, which could have influenced the observed differences between groups.

Conclusion

This study highlights the importance of age, obesity, BMI and metabolic problems in women

with PCOS. According to presented analysis, obese women with PCOS are far more likely to acquire metabolic syndrome. However, metabolic syndrome in lean patients demonstrates that metabolic dysfunction is not limited to obesity, pointing to underlying pathophysiological processes that extend beyond adiposity. Findings emphasise the necessity of phenotype-specific treatment regimens, customised risk assessment and early screening in PCOS to reduce long-term cardio-metabolic effects. Further long-term studies are required to investigate the underlying causes and provide customised treatments for PCOS patients.

Ethics

Data was collected after obtaining approval from the Institutional Ethics Council of Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR) in Mullana, Ambala, Haryana (Project No: IEC-2801), dated 20 March 2024 for a recommended period of 2 years. Research participants were recruited from 1st April 2024 till July 2025, after acquiring written informed consent, predominantly from Haryana state only. The authors gave importance to the participant data information and assured the confidentiality of the individual information.

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Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

Author ORCID numbers

Navjyoti Goyal (NG):
0000-0002-8746-7556
Sarita Sharma (SS):
0000-0003-4010-1562
Ruby Bhatia (RB):
0009-0008-1971-5950
Sumeet Gupta (SG):
0000-0003-2980-7791
Raghavendra Pai (RP):
0009-0006-4150-213X

Author contributions

Conceptualisation: NG
Methodology: NG
Writing - original draft: NG
Supervision: SS, RB
Project administration: SG, RP.

References

1. Shukla A, Rasquin LI, Anastasopoulou C. Polycystic ovarian syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [Cited: 22-Jul-2025]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459251/.
2. Moulana M. Androgen induced cardiovascular risk in polycystic ovary syndrome: the role of T lymphocytes. Life. 2023 Apr 14;13(4):1010. doi: 10.3390/life13041010.
3. World Health Organization (WHO). Polycystic ovary syndrome [Internet]. 7 Feb 2025. [Cited: 22-Jul-2025]. Available from: https://www.who.int/news-room/fact-sheets/detail/polycystic-ovary-syndrome.
4. Singh S, Pal N, Shubham S, Sarma DK, Verma V, Marotta F, et al. Polycystic ovary syndrome: etiology, current management, and future therapeutics. J Clin Med. 2023 Feb 11;12(4):1454. doi: 10.3390/jcm12041454.
5. Bai H, Ding H, Wang M. Polycystic ovary syndrome (PCOS): symptoms, causes, and treatment. Clin Exp Obstet Gynecol. 2024 May 21;51(5). doi: 10.31083/j.ceog5105126.

6. Li J, Huang Y, Xu S, Wang Y. Sleep disturbances and female infertility: a systematic review. BMC Womens Health. 2024 Dec 20;24(1). doi: 10.1186/s12905-024-03508-y.
7. Kiran Z. Diagnostic criteria for polycystic ovary syndrome. In: Rehman R, Sheik A, Eds. Polycystic Ovary Syndrome. Amsterdam: Elsevier 2024. p.61-74. doi: 10.1016/B978-0-323-87932-3.00018-9.
8. Di Lorenzo M, Cacciapuoti N, Lonardo MS, Nasti G, Gautiero C, Belfiore A, et al. Pathophysiology and nutritional approaches in polycystic ovary syndrome (PCOS): a comprehensive review. Curr Nutr Rep. 2023 May 22;12(3):527-44. doi: 10.1007/s13668-023-00479-8.
9. Barber TM, Hanson P, Weickert MO, Franks S. Obesity and polycystic ovary syndrome: implications for pathogenesis and novel management strategies. Clin Med Insights Reprod Health. 2019 Jan;13:1179558119874042. doi: 10.1177/1179558119874042.
10. Unluhizarci K, Karaca Z, Kelestimur F. Role of insulin and insulin resistance in androgen excess disorders. World J Diabetes. 2021 May 15;12(5):616-629. doi: 10.4239/wjd.v12.i5.616.
11. Barrea L, Frias Toral E, Verde L, Ceriani F, Cucalón G, Garcia Velasquez E, et al. PCOS and nutritional approaches: differences between lean and obese phenotype. Metabolism Open. 2021 Dec;12:100123. doi: 10.1016/j.metop.2021.100123.
12. Sanchez Garrido MA, Tena Sempere M. Metabolic dysfunction in polycystic ovary syndrome: pathogenic role of androgen excess and potential therapeutic strategies. Mol Metab. 2020 May;35:100937. doi: 10.1016/j.molmet.2020.01.001.
13. Hirschberg AL. Hyperandrogenism and cardiometabolic risk in pre and postmenopausal women—what is the evidence? J Clin Endocrinol Metab. 2023 Oct 27;109(5):1202-13. doi: 10.1210/clinem/dgad590.
14. Chen W, Pang Y. Metabolic syndrome and PCOS: pathogenesis and the role of metabolites. Metabolites. 2021 Dec 14;11(12):869. doi: 10.3390/metabo11120869.
15. Amer Ali E, AL Jedda WA, AL Khateeb SMJ, AL Samarriae AY. The association between serum nesfatin 1 level and body mass index in Iraqi patients with polycystic ovary syndrome (PCOS). Indian J Forensic Med Toxicol. 2021 Mar 24;15(2):1490-5. doi: 10.37506/ijfmt.v15i2.14549.
16. Hamed EA, Sayyed HG, Abbas AM, Abdel Gaber MM, Abd El Aleem HM. Nesfatin 1, dopamine, and NADPH levels in infertile women with polycystic ovary syndrome: is there a relationship between their levels and metabolic and hormonal variables. J Reprod Infertil. 2022 Jul 11;23(3). doi: 10.18502/jri.v23i3.10006.
17. Abdelazim I, Alanwar A, AbuFaza M, Amer O, Bekmukhambetov Y, Zhurabekova G, et al. Elevated and diagnostic androgens of polycystic ovary syndrome. Menopausal Rev. 2020;19(1):1-5. doi: 10.5114/pm.2020.95293.
18. Amisi CA. Markers of insulin resistance in polycystic ovary syndrome women: an update. World J Diabetes. 2022 Mar 15;13(3):129-49. doi: 10.4239/wjd.v13.i3.129.
19. Sharma A, Sarwal Y, Devi NK, Saraswathy KN. Polycystic ovary syndrome prevalence and associated sociodemographic risk factors: a study among young adults in Delhi NCR, India. Reprod Health. 2025 Apr 28;22(1). doi: 10.1186/s12978-025-02019-9.
20. Zaeemzadeh N, Jahanian Sadatmahalleh S, Ziaei S, Kazemnejad A, Mottaghi A, Mohamadzadeh N, et al. Prevalence of metabolic syndrome in four phenotypes of PCOS and its relationship with androgenic components among Iranian women: a cross sectional study. Int J Reprod BioMed. 2020 May 5;18(4):253-64. doi: 10.18502/ijrm.v13i4.6888.
21. Karee M, Gundabattula SR, Sashi L, Boorugu H, Chowdhury A. Prevalence of metabolic syndrome in women with polycystic ovary syndrome and the factors associated: a cross sectional study at a tertiary care center in Hyderabad, south eastern India. Diabetes Metab Syndr. 2020 Jul;14(4):583-7. doi: 10.1016/j.dsx.2020.05.006.
22. Kaur R, Kaur M. Evaluation of potential association of metabolic syndrome in obese and non obese PCOS women. Middle East Fertil Soc J. 2024 Jan 26;29(1). doi: 10.1186/s43043-024-00170-0.
23. Kayali S, Chitra T, Kamalanathan S, Nandeesha H. Assessment of metabolic syndrome in infertile women with polycystic ovary syndrome in a rural population of South India: a cross sectional study. Int J Reprod BioMed. 2022 Apr 21;20(3):161-8. doi: 10.18502/ijrm.v20i3.10707.
24. Dadachanji R, Patil A, Joshi B, Mukherjee S. Elucidating the impact of obesity on hormonal and metabolic perturbations in polycystic ovary syndrome phenotypes in Indian women. PLoS One. 2021 Feb 26;16(2):e0246862. doi: 10.1371/journal.pone.0246862.
25. Varghese J, Kantharaju S, Thunga S, Joseph N, Singh P. Prevalence and predictors of metabolic syndrome in women with polycystic ovarian syndrome— a study from southern India. Int J Reprod Contracept Obstet Gynecol. 2015;1:113-8. doi: 10.5455/2320-1770.ijrcog20150222.
26. Arunachalam R, Priya K, Brindha R, Parthiban K. Understanding metabolic patterns in polycystic ovary syndrome: comparing lean and obese women at a family medicine clinic. J Family Med Prim Care. 2024 May;13(5):1837-42. doi: 10.4103/jfmpc.jfmpc_1425_23.
27. Kim JJ. Obesity and polycystic ovary syndrome. J Obes Metab Syndr. 2024 Dec 20;33(4):289-301. doi: 10.7570/jomes24035.