ORIGINAL RESEARCH ARTICLE

Obesity-Driven Variations in Endocrine Profiles among Polycystic Ovarian Syndrome Phenotypes in A Cross-Sectional Epidemiologic Study: Obesity and Metabolic Variations in PCOS

Soujanya J Pujar[1*], Neelamma Patil[2]

[1]Department of Obstetrics and Gynaecology Nursing, BLDEA's Shri B M Patil Institute of Nursing Sciences, Vijayapura, Karnataka, India
[2]Department of Obstetrics and Gynaecology, BLDE Deemed to be University, Shri B M Medical College, Hospital and Research Centre, Vijayapura, Karnataka, India

DOI: 10.55489/njcm.161220256014

ABSTRACT

Background: The management approaches for polycystic ovary syndrome (PCOS) vary between lean and overweight patients which led to this research investigating clinical and endocrine differences among lean and obese/ overweight PCOS individuals. Objective was to evaluate the clinical and hormonal differences between obese/ overweight and lean phenotypes of women with PCOS**.**

Methods and Materials: The study executed between January 2021 and March 2022 at B M Patil Medical College and Hospital in Vijayapur, included 77 women aged 21-35 who met the Rotterdam criteria for PCOS diagnosis. Participants were dichotomized as lean (≤23 kg/m^2) or obese/ overweight (>23 kg/m^2), with the sociodemographic information collected as well as clinical assessments and hormone measurements.

Results: The 77 participants comprised 33 lean and 44 obese/ overweight individuals. Lean women experienced more irregular menstrual cycles, while obese/overweight women had higher rates of dysmenorrhea. Waist-hip ratios differed significantly, with 60.6% of lean women having a ratio ≤0.8 and 86.4% of obese/ overweight women exceeding 0.8. Hirsutism and acne were more prevalent in obese/ overweight individuals, with statistical significance. Lean women had higher proportion of elevated free testosterone levels (>6.3pg/mL) at 39% compared to 22.7% of obese/overweight women had a higher proportion. Obese/ overweight women had a higher proportion of LH: FSH ratios ≥1(59.1%) compared to lean participants (45.5%).

Conclusion: The findings highlight the clinical and hormonal disparities between lean and obese/overweight PCOS patients, underscoring the importance of BMI in PCOS management. Limitations include the small size and lack of multivariate analysis, which may affect generalizability and control for confounding variables.

Keywords: PCOS, Obesity, Lean, Clinical Parameters, BMI, Testosterone

ARTICLE INFO

Financial Support: This work obtained support from BLDE Deemed to be University through Grant No. 439485 which was issued on 31/03/2023.

Conflict of Interest: The authors have declared that no conflict of interests exists.

Received: 14-09-2025, **Accepted**: 18-10-2025, **Published**: 01-12-2025

***Correspondence:** Soujanya J Pujar (Email: jpsoujanya141@gmail.com)

How to cite this article: Pujar SJ, Patil N. Obesity-Driven Variations in Endocrine Profiles among Polycystic Ovarian Syndrome Phenotypes in A Cross-Sectional Epidemiologic Study: Obesity and Metabolic Variations in PCOS. Natl J Community Med 2025;16(12):1181-1187. DOI: 10.55489/njcm.161220256014

www.njcmindia.com | pISSN: 0976-3325 | eISSN: 2229-6816 | Published by Medsci Publications

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is the one of the most prevalent endocrinopathies, affects 6.5-8% of women.[1] The association between obesity, hirsutism, amenorrhea and polycystic ovaries was first described by Stein and Leventhal in 1935.[2]

According to the 2003 consensus meeting of ESHRE and ASRM in Rotterdam, a diagnosis of PCOS requires at least two of the following three criteria- (a) oligo/ anovulation (b) polycystic ovaries on USG, and (c) hyperandrogenism (HA). A polycystic ovary is currently defined as having 12 or more follicles that are 2-9mm in size or an ovarian volume greater than 10cm.[3] These criteria have since been widely adopted and validated in clinical and research settings, forming the cornerstone of PCOS diagnosis globally.

The PCOS is an oligogenic condition with diverse clinical and biochemical phenotypes, determined by complex interplay of genetic and environmental factors.[4] Primary abnormalities in insulin resistance; ovarian function and the hypothalamic pituitary axis are all part of the pathogenesis of PCOS.[5] Insulin resistances contribute to metabolic dysfunction, while hyperandrogenism promotes adipose tissue accumulation and dysfunction, leading to oxidative stress.[6] This syndrome exhibits the full clinical spectrum, combining metabolic disturbances with reproductive and psychological dysfunctions concurrently.[7]

PCOS is associated with ovulatory dysfunction, menstrual irregularities while also causing infertility and insulin resistance. The presentation of symptoms encompasses acne development alongside hirsutism and weight gain.[8] Progression of PCOS can is associated with long term health complications such as obesity, Type 2 diabetes, endometrial cancer, elevated cholesterol levels and heart diseases.[9]

Between 25% and 70% of women with PCOS are obese/ overweight, yet many affected individuals have normal BMI values (≤25 kg/m^2). The elements involved create intricate obstacles for developing effective diagnostic and treatment approaches.[10]

Although obesity is a well-established contributor to adverse outcomes in PCOS, emerging evidence suggest that South Asian women may exhibit significant metabolic risks even at lower BMI thresholds. Women often exhibit disproportionately high levels of visceral adipose tissue even in the absence of obesity, a condition frequently accompanied by elevated insulin resistance. This distinct metabolic profile characterized by excess visceral fat and impaired insulin sensitivity despite a normal BMI significantly increases their vulnerability to cardio metabolic disorders such as type 2 diabetes, hypertension, and dyslipidemia.[11] This raises the possibility that lean women with PCOS may also be prone to significant metabolic and hormonal risks, which could be overlooked if clinical attention focuses predominantly on obesity.

This cross-sectional study was designed to compare the clinical and endocrinological profiles of lean and obese/ overweight women with PCOS in Indian population. We hypothesize that BMI influences the clinical and metabolic presentation of PCOS with distinct differences between obese and lean phenotypes. Specifically, we expect that lean PCOS women may show higher androgen levels due to reduced adipose tissue buffering, while overweight/ obese women may exhibit more pronounced clinical signs of hyperandrogenism. Clarifying these BMI related variations is important for developing phenotype-specific diagnostic and management strategies in PCOS particularly among the South Asian Population.

METHODOLOGY

Study design and setting: The hospital-based observational case control study was conducted at the Gynecology OPD at B M Patil Medical College, Hospital and research Centre in Vijayapur, Karnataka, India from January 2021 and March 2022. The study was the hospital based observational case control study conducted over a one-year period. The study was approved by the Institutional Ethical Committee (IEC no. BMPRC/IEC/2020/45, dated 15/12/2020), and informed written consent was obtained from all participants.

Sample size calculation: The sample size was estimated using the formula of comparing proportions between two independent groups:

$$n = ((Z_{1-\alpha/2} + Z_{1-\beta})^2 \cdot [P_1(1-P_1 1) + P_2(1-P_2)]/(P_1 - P_2)^2$$

Where, $Z_{1-\alpha/2=}$ 1.96 standard normal deviate corresponds to a 95% confidence level, an alpha of 0.05, Z_β: for 80% power, p_1 and p_2 are based on prior study by Carmina et al (2022)[12]; p_1-$p_{2:}$ expected difference of 20% in prevalence of PCOS.

Based on this assumption, the minimum required sample size was 36 participants per group. Accounting for an anticipated 10% dropouts, a total 77 women were included in the study.

Study participants: A total of 77 women aged 21-35 years, who attended the Gynecology OPD were the study participants. Women who met Rotterdam criteria for PCOS were eligible; those outside the age range, unwilling to participate, or with known causes of hyperandrogenism (HA), primary hypothalamic amenorrhea, primary ovarian failure, thyroid disease or prolactin disorders were excluded.

Data collection and tools: The socio-demographic survey included age, education, occupation, and residence and subsequently included a clinical examination for height, weight, and BMI. Participants were categorized into two groups based on BMI as per WHO Asia- Pacific Guidelines: Group I (Obese /overweight) with BMI >23 kg/m^2 and Group II (normal weight and lean) with BMI of ≤23kg/m^2.[13] Overweight and obese participants were analyzed as

a combined group. Menstrual history was documented. Oligo- or an ovulation was defined as a cycle length exceeding of 35 days or less than 8 cycles per year or absence of menstruation for more than 3 months. Dysmenorrhoea was recorded as self- reported moderate to severe pelvic pain during menstruation. Menstrual flow was classified based on volume and pad using pictorial blood loss assessment chart as follows, heavy flow was defined as >80mL or cycle or saturation of a pad for every 1-2 hours; moderate flow as 40-80mL or cycle or saturation of a pad for every 3-4 hours; and scanty flow as <40mL or cycle or saturation of a pad for 6 hours or longer. Waist circumference (WC) and hip circumference (HC) were both measured by the researcher using an inch tape. Waist circumference was measured in the horizontal plane between the lower margin of the last rib and the iliac crest yielding a value to the nearest 0.1 cm. The measurement of hip circumference (HC) was taken at the widest part of the participant's buttocks. This standard approach was used to minimize measurement bias. The waist to hip ratio (WHR) was calculated by dividing WC by HC and an abnormal ratio was accepted above 0.8. Presence of acne, hirsutism, and acanthosis nigricans were observed as clinical signs of hyperandrogenism. The diagnosis of acne was established with the presence of comedones, papules, nodules, cysts, and scarring. Modified Ferriman-Gallwey score was used to grade terminal hair growth across 9 androgen sensitive areas of the body.[14] The participants with score of ≥ 8 were considered as clinically hirsute.

The hormonal assessment included measurement of serum levels of LH, FSH, prolactin and free testosterone. Blood samples were collected in the morning after an overnight fast, preferably on cycle's days 2 or 3 or on any day for ammenorrheic participants. Hormone levels were analyzed using chemiluminescent immunoassay (CLIA) on the Abbott Architect i2000SR platform. The intra-assay coefficient of variation (CV) was maintained below 5%, and inter-assay CV below 10%, ensuring analytical precision. Reference ranges used for interpretation were: LH (2-12 IU/L), TSH (0.4-4.0 IU/ mL), prolactin (5-25 IU/L), and free testosterone (0.5-2.5pg/mL), consistent with clinical standards.

Statistical analysis: The collected data was analysed through SPSS version 20 and the results appeared as frequencies as well as percentages and mean ± standard deviation. They analyzed data using Student's t-test for continuous variables together with chi-squared test for categorical data with p values less than 0.05 deemed significant.

Approval of Institutional Ethical Review Board: This study was done in accordance with Declaration of Helsinki and approved by Institutional Ethical Committee at (Ref no: BLDE (DU)/IEC/584/2021-22) at BLDE (DU) Shri B M Patil Medical College, Hospital, and Research Centre, Vijayapur.

RESULTS

Across all participants (n=77), the mean age was 25.27 ± 2.92 years, mean BMI was 24.8 ± 4.2 kg/m^2, mean waist- hip ratio was 0.84 ± 0.09, and mean prolactin level was 312.4 ± 98.7 ng / mL 0.15, mean TSH was 2.93 ±1.08 mIU/L, 0.69, mean free testosterone was 3.12 ± 1.64 pg/mL, 0.04, and mean LH: FSH ratio was 1.42 ± 0.061, 0.03.

Table 1: The demographic profile of the study subjects

Parameters	Lean (N=33) (%)	Obese/overweight (N=44) (%)	P value
Age in years (Mean± SD)	25.33 ± 2.93	25.92 ± 2.92	0.88
Residence			
Rural	15(45.5)	22 (50.0)	0.69
Urban	18 (54.5)	22 (50.0)	
Religion			
Hindu	27 (81.8)	38 (86.4)	0.86
Muslim	4 (12.1)	4 (9.1)	
Christian	2 (6.1)	2 (4.5)	
Education			
High school	5 (15.2)	6 (13.6)	0.67
Degree	25 (75.8)	31 (70.5)	
PG	3 (9.1)	7 (15.9)	
Marital status			
Married	22 (66.7)	26 (59.1)	0.49
Unmarried	11 (33.3)	18 (40.9)	
Type of marriage			
Consanguineous	9 (27.3)	15 (34.1)	0.40
Non consanguineous	13 (39.4)	11 (25.0)	
Employment			
House maker	19 (57.5)	28 (63.6)	0.66
Student	7 (21.2)	9 (20.5)	
Working	7 (21.2)	8 (18.2)	
Diet			
Mixed	9 (27.3)	13 (29.5)	0.82
Vegetarian	24 (72.7)	31 (70.5)	

Note: * P value less than 0.05

Table 2: Correlation of clinical profile and BMI (lean versus obese) in women with PCOS

Parameters	Lean (N=33) (%)	Obese/overweight (N=44) (%)	P value
Menstrual Regularity			
Irregular	27 (81.8)	32 (72.7)	0.02*
Regular	6 (18.2)	12 (27.3)	
Dysmenorrhea			
No	18 (54.5)	15 (34.1)	0.07
Yes	15 (45.5)	29 (65.9)	
Menstrual flow pattern			
Heavy	7 (21.2)	8 (18.2)	0.71
Moderate	18 (54.5)	28 (63.6)	
scanty	8 (24.2)	8 (18.2)	
Waist Hip Ratio			
=or <0.8	20(60.6)	6(13.6)	0.001*
>0.8	13 (39.4)	38 (86.4)	
Hirsutism			
Grade-I	21 (63.6)	28 (63.6)	0.98
Grade-II	3 (9.1)	4 (9.1)	
Grade-III	5 (15.2)	8 (18.2)	
Grade-IV	4 (12.1)	4(9.09)	
Acanthosis Nigerians			
No	19 (57.6)	30 (68.2)	0.34
Yes	14 (42.4)	14 (31.8)	
Acne			
No	17 (51.5)	12 (27.3)	0.03*
Yes	16 (48.5)	32 (72.7)	

Note: * P <0.05, Statistically significant

Table 3: Correlation of endocrinal profile and BMI (lean versus Obese/ Overweight) in women with PCOS

Hormones	Lean (33) (mean ± SD)	Obese/ Overweight (44)	p- value
Prolactin (ng/mL)	287.6 ± 112.3	312.4 ± 98.7	0.15
TSH (mIU/L)	2.81 ± 1.12	2.93± 1.08	0.69
Free Testosterone (pg/mL)	4.21 ± 1.76	3.12 ± 1.64	0.04*
LH: FSH ratio	1.18 ± 0.52	1.42 ± 0.61	0.03*

Note: * Significant at p < 0.05; TSH- Thyroid Stimulating Hormone; LH- Luteinizing Hormone; FSH- Follicle Stimulating Hormone

Table 4: Multivariate logistic regression analysis of predictors of Obese/ Overweight status in women with PCOS

Predictor	Odds Ratio (OR)	95% of CI	P value
Age	0.97	0.85- 1.11	0.65
Irregular menstrual cycle	1.42	0.55- 3.65	0.47
Dysmenorrhoea	2.18	0.89- 5.34	0.09
Waist- Hip ratio	6.75	2.45- 18.6	< 0.001*
Acne	2.65	1.05- 6.68	0.04*
Prolactin (ng/mL)	1.03	1.01- 1.06	0.01*
LH: FSH ratio	1.88	0.74- 4.76	0.19
Free Testosterone (pg/mL)	0.72	0.28 1.85	0.49

Note: *Significant at p < 0.05; CI- Confidence Interval

Participant characteristics: A total of 77 women with PCOS records, 33 were categorized as lean PCOS (BMI<23 kg/m^2) and 44 as obese/ overweight (BMI>23 kg/m^2). As shown in Table 1. The majority of the participants in both groups were Hindu, college- educated, married, and homemakers, with a balanced rural – urban distribution. None of these demographic variables differed significantly

Correlation between clinical characteristics and BMI (lean versus obese/ overweight) in women with PCOS: In the current research, lean subjects presented with a greater frequency of irregular menstrual cycles (81.8%) than obese/ overweight subjects (72.7%), with a statistically significant difference (p = 0.02). Dysmenorrhea was more frequent in obese/ overweight subjects (65.9%) than in lean subjects (45.5%), although not statistically significant (p = 0.07).

Waist-hip ratios were quite different between groups: lean subjects mostly had ratios ≤0.8 (60.6%), whereas obese/ overweight subjects had ratios >0.8 (86.4%) (p <0.01). Hirsutism was also quite different, with more severe grades more prevalent in those with obesity (p = 0.98). Acne prevalence was greater in obese/ overweight subjects (72.7%) compared to lean subjects (48.5%) (p = 0.03). The other factors, including menstrual flow pattern and acanthosis nigricans, were not significantly different.

Correlation between endocrine profile and BMI (lean versus obese/ overweight) in women with PCOS: Table no 3 illustrates that the lean and obese/ overweight participant's exhibit differences in hormone levels. Mean prolactin levels were significantly higher in obese/ overweight (312.4 ± 98.7 ng/mL) compared to lean participants (287.6 ± 112.3 ng/mL) (p= 0.15). Similarly, mean TSH levels were elevated in obese/ overweight women (2.93 ± 1.08 m IU/L) versus lean (2.81± 1.12 m IU/L) (p= 0.69). Free testosterone levels were significantly higher in lean participants (4.21 ± 1.76 pg/mL) compared to obese/ overweight (3.12 ± 1.64 pg/mL) (p = 0.04). The LH ratio was also significantly higher in the obese/ overweight group (1.42 ± 0.61) than in lean participants (1.18 ± 0.52) (p = 0.03), indicating a greater degree of hormonal imbalance.

Independent predictor of obese/ overweight status: Table 4 presents the results of a multivariate logistic regression analysis conducted to identify independent predictors of obese/ overweight status among women with PCOS. After adjusting for age, three variables emerged as statistically significant: waist-hip ratio >0.8(OR= 6.75, 95% CI: 2.45- 18.6, p <0.001), acne presence of acne (OR= 2.65, 95% CI: 1.05-6.68, p= 0.04), and prolactin level (OR= 1.03, 95% CI: 1.01-1.06, p= 0.01) these findings suggests that central obesity, acne, and elevated prolactin are independently associated with the obese/ overweight PCOS phenotype. Other variables such as dysmenorrhoea, LH: FSH ratio, free testosterone, menstrual irregularity and age did not show significant associations in the adjusted model.

DISCUSSION

Polycystic ovary syndrome is a common endocrine disorder affecting 5% to 15% of among women of childbearing age. Although gynaecological expertise is essential for the management of PCOS, polycystic ovaries seldom correlate with typical symptoms. While its presentation varies, this case control study exhibited hallmark symptoms such as irregular menses, hirsutism, acne, and menstrual disturbances, particularly among obese/overweight participants. These findings are consistent with prior reports describing the clinical spectrum of PCOS manifestations.[15] Consistent with our findings, previous studies have shown obesity and a tendency toward weight gain are prevalent even among women with normal BMI.[16]

Intermittent GnRH release from the hypothalamus influences PCOS by modulating FSH and LH release, which causes irregular follicular function. Androgen-secreting cysts induce virilisation in females with symptoms such as weight gain, hirsutism, and acne.[17] Lean PCOS subjects have characteristic metabolic and neurologic features that are different from their obese counterparts.[18]

In our study, 77 women with PCOS were enrolled over one year, with 44 (57.1%) classified as obese/ overweight (BMI >23 kg/m^2) and 33 (42.8%) as lean (BMI <23 kg/m)^2.This distribution aligns with previous reports by SS Lim et al and Alvarez, who observed increased PCOS prevalence among obese/ overweight women.[19-20] Similarly Makhija N et al[21] (2023) reported a predominance of overweight / obese women among PCOS cases, with 60% of their cohort falling into higher BMI category. Their findings also highlighted that obese participants exhibited clinical features such as hirsutism, acne and menstrual irregularities.

However, unlike our study, Makhija N et al[21] observed a higher prevalence of menstrual irregularities among lean PCOS women, whereas we found it more common among obese/ overweight PCOS women. This discrepancy may be attributed to differences in population characteristics, as their study was conducted in a tertiary care centre in the centre India, while this study was based in a regional hospital in south India. Additionally, variations in sample size, diagnostic criteria application and hormonal assay methods may have influenced the clinical patterns. These contrasts underscore the importance of considering regional, methodological and demographical factors when interpreting PCOS phenotypes.[21]

Both lean and obese women participated, mostly aged 21-25. Chi-square tests failed to show any statistically significant relationships between body type and categorical variables among lean and obese participants, mirroring the findings of Mohapatra (2024).[22]

Irregular menstrual cycles were significantly more frequent among lean participants (81.8%) compared to the obese/ overweight (72.7%). McCartney and Campbell (2020) suggest that this may reflect a distinct neuroendocrine dysfunction in lean PCOS women, where hypothalamic-pituitary axis distribution plays a central role.[23] In the absence of obesity–related hyperinsulinemia, lean PCOS patients often exhibit altered Gonadotrophin- Releasing Hormone (GnRH) pulsatility, leading to increased luteinizing hormone (LH) secretion and impaired follicular development.[24,25] This dysregulation is compounded by elevated Anti-Mullerian Hormone (AMH) level, which have been shown to directly influence hypothalamic neurons and further disrupt GnRH pulse generation.[26] Together, these factors contribute to anovulation and menstrual irregularity, particularly in lean PCOS women where insulin mediated feedback is less dominant. Similar findings were reported by Liao B et al.[27] The likelihood of acne occurrence was found to be significantly related to BMI, with obese/ overweight participants being more disposed toward these conditions (51.5% vs. 27.3%, p=.04), as observed in Makhija N et al (2023).[21] Further supporting this association, Benyaminpour and Sachmechi(2004) conducted a systematic review and found that women with PCOS had a 1.6 fold increased risk of acne compared to healthy controls, with insulin resistance and hyperandrogenism iden-

tified as key contributing factors.[28] Hirsutism and acanthosis were noticed among lean as well as obese/ overweight PCOS subjects, with both 56 (70%) of obese/ overweight and 3 (15%) of lean PCOS women exhibiting a WHR >0.85 (central obesity) according to Akshaya S (2016) et al.[29]

The recent study suggested that obese/ overweight PCOS women showed greater free testosterone and LH ratio compared to lean women, validating the findings of Kiddy DS et al[30] (1990), Kumar N[31] (2022), and Remsberg KE et al[32] (2002). Interestingly some studies have reported higher free testosterone levels in lean PCOS women, which may be attributed to lower sex hormone- binding globulin (SHBG) levels due to reduced peripheral conversion of estrogens in adipose tissue, resulting in increased bio available androgens. Additionally, obesity has been shown to suppress LH pulsatility through leptin- mediated hypothalamic feedback and chronic low- grade inflammation, potentially altering the LH: FSH ratio.[33] Both the groups in this study showed comparable levels of TSH and prolactin, aligning with Nayak PK et al[34] (2020). No such differences were observed in this study.

These findings underscore the need for phenotype-specific management strategies. Lean PCOS women may require closer monitoring for hormonal imbalances despite normal BMI, while obese /overweight women may benefit from insulin-sensitizing therapies and lifestyle interventions targeting adiposity. In South Asian populations, where metabolic risk is elevated even at lower BMI, screening lean women for insulin resistance, lipid abnormalities, and cardiovascular risk is crucial.

Limitations

This study was limited its cross-sectional design, which restricts casual inference. The sample size was modest and drawn from a single centre, potentially introducing selection bias. Confounding factors such as diet, physical activity, and socioeconomic status were not adjusted for. BMI, while widely used, dies not capture body composition nuances such as visceral fat or lean mass.

Conclusion

Tailored management strategies based on BMI phenotypes are essential in addressing the diverse clinical and hormonal presentations of PCOS. Lean women with PCOS may require more vigilant hormonal monitoring, particularly for elevated free testosterone and menstrual irregularities, which may be overlooked due to their normal BMI. Conversely, obese /overweight women often present with more pronounced clinical signs such as hirsutism, acne, and dysmenorrhoea, necessitating interventions targeting insulin resistance and adiposity. In South Indian populations, where metabolic risks manifest at lower BMI thresholds, clinicians should adopt ethnicity- specific screening protocols to identify high risk individuals regardless of weight status. These findings support the need for phenotype- specific diagnostic and therapeutic approaches in PCOS, emphasizing the importance of individualized care beyond BMI categorization.

Future directions

Future research should include longitudinal studies to tract hormonal and metabolic changes overtime, incorporate genetic and epigenetic analysis, and use advanced body composition metrics like DEXA, bioimpedance etc. Expanding the sample to include diverse geographic and ethnic backgrounds will enhance generalizability.

Acknowledgement: We extend our sincere gratitude to the BLDE (DU) Shri B M Patil Medical College, Hospital, and Research Centre for their support and resources throughout this study. Special thanks to the participants for their invaluable contribution and cooperation.

Individual Authors' Contributions: The corresponding author SP collaborated with NP on the concept and designs of this project. NP provided revisions and oversight throughout the project. Both authors were involved in data collection from participants. NP handled the review process and managed resources. The SP performed the data analysis and drafted the manuscript. Both authors reviewed and approved the final version of the article and accept responsibility for all aspects of it.

Availability of Data: Data generated during this study available from corresponding author upon reasonable request.

Declaration of Non-use of Generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

References

1. Veltman-Verhulst SM, Boivin J, Eijkemans M, Fauser B. Emotional distress is a common risk in women with polycystic ovary syndrome: a systematic review and meta-analysis of 28 studies. Hum Reprod Update. 2012;18(6):638-651. DOI: https://doi.org/10.1093/humupd/dms029 PMid:22824735
2. Kiałka M, Milewicz T, Sztefko K, Rogatko I, Majewska R. History of discovery of polycystic ovary syndrome. Adv Clin Exp Med. 2017; 26(3):555-558. DOI: https://doi.org/10.17219/acem/61987 PMid:28791833
3. Bonnar J, Dunlop W. Recent advances in obstetrics and gynaecology. 23rd ed. London: Royal Society of Medicine; 2005.
4. Xita N, Georgiou I, Tsatsoulis A. The genetic basis of polycystic ovary syndrome. Eur J Endocrinol. 2002;147(6):717-725. DOI: https://doi.org/10.1530/eje.0.1470717 PMid:12457445
5. Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, et al. Endocrine-disrupting chemicals: an Endocrine Society scientific

statement. Endocr Rev. 2009;30(4):293-342. DOI: https://doi.org/10.1210/er.2009-0002 PMid:19502515

6. Dumesic DA, Abbott DH, Sanchita Sharma, Chazenbalk GD. Endocrine-Metabolic Dysfunction in Polycystic Ovary Syndrome: an Evolutionary Perspective. Curr Opin Endocr Metab Res. 2020;12:41-48. DOI: https://doi.org/10.1016/j.coemr.2020.02.013

7. Teede HJ, Joham AE, Paul E, et al. Longitudinal weight gain in women identified with polycystic ovary syndrome: results of an observational study in young women. Obesity (Silver Spring). 2013;21(8):1526-1532. DOI: https://doi.org/10.1002/oby.20213 PMid:23818329

8. Jahan S, Munir F, Razak S, et al. Ameliorative effects of rutin against metabolic, biochemical and hormonal disturbances in polycystic ovary syndrome in rats. J Ovarian Res. 2016; 9(1): 86. DOI: https://doi.org/10.1186/s13048-016-0295-y

9. Henney AE, Gillespie CS, Lai JY, et al. Risk of Type 2 Diabetes, MASLD and Cardiovascular Disease in People Living With Polycystic Ovary Syndrome. J Clin Endocrinol Metab. 2025; 110 (5):1235-1246. DOI:https://doi.org/10.1210/clinem/dgae481

10. Idicula-Thomas S, Gawde U, Bhaye S, Pokar K, Bader GD. Meta-analysis of gene expression profiles of lean and obese PCOS to identify differentially regulated pathways and risk of comorbidities. Comput Struct Biotechnol J. 2020;18:1735-1745. DOI: https://doi.org/10.1016/j.csbj.2020.06.023 PMid:32695266 PMCid:PMC7352056

11. Mohan D, Pradeepa R, Venkatesan U et al. High prevalence of metabolic obesity in India: The ICMR-INDIAB national study (ICMR-INDIAB-23). Indian J Med Res. 2025 May;161(5):461-472. DOI: https://doi.org/10.25259/IJMR_328_2025. PMID: 40844097 PMCID: PMC12550443

12. Carmina E, Lobo RA. Comparing Lean and Obese PCOS in Different PCOS Phenotypes: Evidence That the Body Weight Is More Important Than the Rotterdam Phenotype in Influencing the Metabolic Status. Diagnostics. 2022; 12(10):2313. DOI: https://doi.org/10.3390/diagnostics12102313

13. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363(9403):157-163. DOI: https://doi.org/10.1016/S0140-6736(03)15268-3

14. Kahraman FC, Erdoğan SS. Grading of hirsutism: a practical approach to the modified Ferriman-Gallwey scoring system. Postepy Dermatol Alergol. 2022; 39(4):744-748. DOI: https://doi.org/10.5114/ada.2021.108455 PMid:36090715

15. Rosenfield RL, Ehrmann DA. The pathogenesis of polycystic ovary syndrome (PCOS): The hypothesis of PCOS as functional ovarian hyperandrogenism revisited. Endocr Rev. 2016; 37(5):467-520. DOI: https://doi.org/10.1210/er.2015-1104

16. Choudhary A, Jain S, Chaudhari P. Prevalence and symptomatology of polycystic ovarian syndrome in Indian women: is there a rising incidence? Int J Repro'd Contracept Boster Gynecol. 2017; 6(11):4971- 4975. DOI: https://doi.org/10.18203/2320-1770.ijrcog20175010

17. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy. J Steroid Biochem Mol Biol. 2018;182:27-36. DOI: https://doi.org/10.1016/j.jsbmb.2018.04.008 PMid:29678491

18. Goyal M, Dawood AS. Debates Regarding Lean Patients with Polycystic Ovary Syndrome: A Narrative Review. J Hum Reprod Sci. 2017 Jul-Sep;10(3):154-161. DOI: https://doi.org/10.4103/jhrs.JHRS_77_17 PMid:29142442

19. Lim SS, Davies MJ, Norman RJ, Moran LJ. Overweight, obesity and central obesity in women with polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod Update. 2012;18(6):618-637. DOI: https://doi.org/10.1093/humupd/dms030 PMid:22767467

20. Alvarez-Blasco F, Botella-Carretero JI, San Millán JL, Escobar-Morreale HF. Prevalence and characteristics of the polycystic ovary syndrome in overweight and obese women. Arch Intern Med. 2006;166(19):2081-2086. DOI: https://doi.org/10.1001/archinte.166.19.2081

21. Makhija N, Tayade S, Toshniwal S, Tilva H. Clinico-metabolic profile in lean versus obese polycystic ovarian syndrome women. Cureus. 2023;15(4):e37809. DOI: https://doi.org/10.7759/cureus.37809

22. Mohapatra I, Samantaray SR. BMI and Polycystic Ovary Syndrome: Demographic Trends in Weight and Health. Cureus. 2024;16(3):e55439. https://doi.org/10.7759/cureus.55439

23. McCartney CR, Campbell RE. Abnormal GnRH Pulsatility in Polycystic Ovary Syndrome: Recent Insights. Curr Opin Endocr Metab Res. 2020;12:78-84. DOI: https://doi.org/10.1016/j.coemr.2020.04.005 PMid:32676541 PMCid:PMC7365617

24. Okigbo CC, Gill S, Hall JE. The Hypothalamic-Pituitary Axis in PCOS. In: Polycystic Ovary Syndrome. Springer; 2022;73-93. DOI: https://doi.org/10.1007/978-3-030-92589-5_5

25. Barbotin AL, Mimouni NEH, Kuchcinski G, et al. Hypothalamic neuroglial plasticity is regulated by anti-Müllerian hormone and disrupted in polycystic ovary syndrome. E-BioMedicine. 2023; 90:104535. DOI: https://doi.org/10.1016/j.ebiom.2023.104535 PMid:37001236 PMCid:PMC10070524

26. Abbott DH, Hutcherson BA, Dumesic DA. Anti-Müllerian Hormone: A Molecular Key to Unlocking Polycystic Ovary Syndrome? Semin Reprod Med. 2024 Mar;42(1):41-48. DOI: https://doi.org/10.1055/s-0044-1787525 PMid:38908381

27. Liao B, Qiao J, Pang Y. Central Regulation of PCOS: Abnormal Neuronal-Reproductive-Metabolic Circuits in PCOS Pathophysiology. Front Endocrinol (Lausanne). 2021;12:667422. DOI: https://doi.org/10.3389/fendo.2021.667422

28. Benyaminpour S, Sachmechi I. PCOS and Acne: A Systematic Review. Int J Clin Med Case Rep. 2024;37: 000906. DOI: https://doi.org/10.46998/IJCMCR.2024.37.000906

29. Akshaya S, Bhattacharya R.Comparative study of clinical profile of lean and obese polycystic ovary syndrome women. Int J Reprod Contracept Obstet Gynecol. 2017;11;5(8):2530-2533. DOI: https://doi.org/10.18203/2320-1770.ijrcog20162173

30. Kiddy DS, Sharp PS, White DM, Scanlon MF, Mason HD, Bray CS. Differences in clinical and endocrine features between obese and non-obese subjects with polycystic ovary syndrome: An analysis of 263 consecutive cases. Clinical Endocrinology. 1990;32(2):213-220. DOI: https://doi.org/10.1111/j.1365-2265.1990.tb00857.x

31. Kumar N, Agarwal H. Early Clinical, Biochemical and Radiological Features in Obese and Non-Obese Young Women with Polycystic Ovarian Syndrome: A Comparative Study. Horm Metab Res. 2022; 54(9):620-624. DOI: https://doi.org/10.1055/a-1880-1264 PMid:35858631

32. Remsberg KE, Talbott EO, Zborowski JV, Evans RW, McHugh-Pemu K. Evidence for competing effects of body mass, hyperinsulinemia, insulin resistance, and androgens on leptin levels among lean, overweight, and obese women with polycystic ovary syndrome. Fertil Steril. 2002;78(3):479-486. DOI: https://doi.org/10.1016/S0015-0282(02)03303-4

33. Quennell JH, Mulligan AC, Tups A, et al. Leptin indirectly regulates gonadotropin-releasing hormone neuronal function. Endocrinology. 2009;150(2):772-779. DOI: https://doi.org/10.1210/en.2008-1693 PMid:19179437

34. Nayak PK, Mitra S, Sahoo J, Mahapatra E, Agrawal S, Lone Z. Relationship of subclinical hypothyroidism and obesity in polycystic ovarian syndrome patients. J Family Med Prim Care. 2020; 9(1):147-150. DOI: https://doi.org/10.4103/jfmpc.jfmpc_654_19 PMid:32110581