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A Cross-Sectional Study on the Prevalence of Polycystic Ovary Syndrome and Its Association with Metabolic Syndrome in Reproductive-Age Women

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder among reproductive-age women and is frequently associated with metabolic derangements. The coexistence of Metabolic Syndrome (MetS) in PCOS increases the risk of cardiovascular disease and diabetes, making early detection clinically essential. Aim: To determine the prevalence of PCOS and its association with Metabolic Syndrome in reproductive-age women.
Methods: A cross-sectional study was conducted among 180 women aged 18-40 years attending the outpatient department of a tertiary care hospital. PCOS was diagnosed using Rotterdam criteria (2003), while MetS was defined according to NCEP ATP III guidelines. Anthropometric measurements, blood pressure, fasting glucose, and lipid profile were recorded. Data were analyzed using SPSS v25.0, with χ^2 and t-tests applied as appropriate; $p < 0.05$ was considered statistically significant.
Results: The mean age of participants was 27.9 ± 4.2 years, with a mean BMI of 26.8 ± 3.9 kg/m². The overall prevalence of PCOS was 34.4% (95% CI: 27.9-41.6%), while MetS prevalence was 26.7% (95% CI: 20.9-33.3%). Among women with PCOS, 41.9% had concurrent MetS compared to 18.6% among non-PCOS women (RR = 2.25; 95% CI: 1.40-3.63; p = 0.0008). Central obesity (56.5% vs 35.6%), elevated triglycerides (51.6% vs 33.1%), and low HDL-C (61.3% vs 42.4%) were significantly more common in the PCOS group. The prevalence of PCOS rose progressively with increasing BMI (p = 0.007).
Conclusion: A strong association exists between PCOS and MetS in reproductive-age women, driven mainly by central obesity and dyslipidemia. Screening for metabolic risk factors should form an integral part of PCOS management to prevent future cardiometabolic complications.
Keywords: Polycystic Ovary Syndrome, Metabolic Syndrome, Reproductive-age Women

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, characterized by a constellation of symptoms including menstrual irregularities, hyperandrogenism, and polycystic ovarian morphology on ultrasonography. It

represents a multifactorial condition resulting from the complex interplay of genetic, hormonal, metabolic, and environmental factors. The global prevalence of PCOS ranges between 6% and 20%, depending on the diagnostic criteria used [1]. In India, the prevalence appears to be increasing rapidly, with studies suggesting that nearly one in five reproductive-aged women may be affected [2].

PCOS is not merely a reproductive disorder but a systemic condition with profound metabolic implications. Insulin resistance, obesity, dyslipidemia, and glucose intolerance are frequently encountered in women with PCOS and serve as important links between PCOS and metabolic syndrome (MetS). MetS, as defined by the International Diabetes Federation (IDF) and the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), comprises a cluster of cardiovascular risk factors, including central obesity, dyslipidemia, hypertension, and impaired glucose tolerance [3]. Several studies have demonstrated that women with PCOS are at a two- to four-fold increased risk of developing MetS compared to age- and BMI-matched controls, highlighting the overlapping pathophysiology of these two syndromes [4].

Insulin resistance appears to be the cornerstone of both PCOS and MetS. Hyperinsulinemia promotes ovarian androgen production and decreases hepatic synthesis of sex hormone-binding globulin (SHBG), resulting in elevated circulating androgens. Simultaneously, chronic low-grade inflammation and adipokine dysregulation contribute to endothelial dysfunction and atherogenesis. The coexistence of PCOS and MetS therefore markedly increases the risk of type 2 diabetes mellitus, hypertension, and premature cardiovascular disease. Moreover, lifestyle factors such as sedentary behavior, unhealthy dietary patterns, and increasing obesity rates in urban India have further exacerbated the prevalence of these interlinked conditions [5].

To determine the prevalence of Polycystic Ovary Syndrome and its association with Metabolic Syndrome in reproductive-age women.

Objectives:

1. To estimate the prevalence of Polycystic Ovary Syndrome (PCOS) among reproductive-age women.
2. To determine the prevalence of Metabolic Syndrome (MetS) in women diagnosed with PCOS.
3. To assess the association between individual components of Metabolic Syndrome and the presence of PCOS.

MATERIAL AND METHODOLOGY

Source of Data: The data were obtained from women attending the Outpatient Department (OPD) of Obstetrics and Gynecology and the Department of Endocrinology at a tertiary care teaching hospital. All eligible participants were evaluated after obtaining written informed consent.

Study Design: The study was designed as a hospital-based, cross-sectional observational study.

Study Location: This study was conducted at the Department of Obstetrics and Gynecology in collaboration with the Department of Biochemistry, Bidar Institute of Medical Sciences, Bidar a tertiary care hospital situated in North Karnataka, India.

Study Duration: The study was conducted over a period of 18 months.

Sample Size: A total of 180 reproductive-age women (18-40 years) were enrolled in the study using a convenient sampling method.

Inclusion Criteria:

- Women aged 18-40 years attending OPD with menstrual irregularities or symptoms suggestive of PCOS.
- Willingness to provide informed consent.
- Women not on hormonal medication for at least three months prior to enrollment.

Exclusion Criteria:

- Women with known thyroid disorders, hyperprolactinemia, or adrenal disorders.
- Pregnant or lactating women.
- Women with chronic systemic illness or on long-term corticosteroid therapy.

Procedure and Methodology: All participants underwent detailed history taking, including menstrual, obstetric, and family history, followed by physical examination. Anthropometric parameters such as height, weight, waist circumference, and body mass index (BMI) were measured. Blood pressure was recorded in the sitting position. Laboratory investigations included fasting plasma glucose, fasting lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C), and fasting insulin. PCOS diagnosis was made based on the Rotterdam criteria (2003)-requiring at least two of the following: (1) oligo/anovulation, (2) clinical or biochemical hyperandrogenism, (3) polycystic ovaries on ultrasound. Metabolic Syndrome was diagnosed using NCEP ATP III criteria, requiring three or more of the following: waist circumference ≥88 cm, fasting glucose ≥110 mg/dL, triglycerides ≥150 mg/dL, HDL-C <50 mg/dL, and blood pressure ≥130/85 mmHg.

Sample Processing: Fasting venous blood samples (5 mL) were collected after overnight fasting of 8-10 hours. Serum glucose and lipid parameters were analyzed using an automated biochemistry analyzer. Ultrasound pelvis was performed by a trained radiologist to assess ovarian morphology.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as proportions. The Chi-square test and Fisher's exact test were used to analyze associations between PCOS and metabolic parameters. Independent t-tests were applied to compare means between groups. A p-value <0.05 was considered statistically significant.

Data Collection: Information was collected using a pretested structured proforma that included demographic details, clinical features, biochemical results, and ultrasonographic findings. All collected data were coded and maintained confidentially for analysis and reporting.

OBSERVATION AND RESULTS

Table 1. Overall cohort profile and key outcomes (N = 180)

Measure	Value n (%) or mean ± SD	95% CI
Age (years)	27.9 ± 4.2	27.3 to 28.5
BMI (kg/m²)	26.8 ± 3.9	26.2 to 27.4
Waist circumference (cm)	87.3 ± 8.1	86.1 to 88.5
SBP (mmHg)	118.7 ± 12.9	116.8 to 120.6
DBP (mmHg)	77.6 ± 8.7	76.3 to 78.9
Fasting glucose (mg/dL)	97.4 ± 12.6	95.5 to 99.3
Triglycerides (mg/dL)	154.2 ± 41.7	148.1 to 160.3
HDL-C (mg/dL)	44.1 ± 9.3	42.7 to 45.5
PCOS prevalence	62 (34.4%)	27.9% to 41.6%
MetS prevalence	48 (26.7%)	20.9% to 33.3%
MetS among PCOS	26/62 (41.9%)	30.5% to 54.3%
MetS among non-PCOS	22/118 (18.6%)	12.6% to 26.6%
Association: PCOS ↔ MetS	RR = 2.25	1.40 to 3.63

Table 1 presents the baseline demographic and biochemical characteristics of the 180 reproductive-age women enrolled in the study. The mean age of participants was 27.9 ± 4.2 years (95% CI: 27.3-28.5), indicating that the cohort largely represented young adults in the reproductive age range. The mean BMI was 26.8 ± 3.9 kg/m² (95% CI: 26.2-27.4), with an average waist circumference of 87.3 ± 8.1 cm, suggesting a

predominance of overweight and mildly obese women. The mean systolic and diastolic blood pressures were 118.7 ± 12.9 mmHg and 77.6 ± 8.7 mmHg, respectively, reflecting an overall normotensive cohort. Fasting glucose averaged 97.4 ± 12.6 mg/dL, while triglyceride and HDL-C levels were 154.2 ± 41.7 mg/dL and 44.1 ± 9.3 mg/dL, respectively, showing mild dyslipidemia. The prevalence of PCOS was 34.4% (95% CI: 27.9-41.6%), while Metabolic Syndrome (MetS) was present in 26.7% (95% CI: 20.9-33.3%) of the total cohort. Among women with PCOS, 41.9% had concomitant MetS, compared to 18.6% among non-PCOS participants. The relative risk (RR) of MetS in PCOS women was 2.25 (95% CI: 1.40-3.63), with a highly significant association (χ^2 = 11.28, p = 0.0008). This demonstrates that women with PCOS were more than twice as likely to have MetS as those without PCOS, emphasizing the strong metabolic burden associated with PCOS.

Table 2. Prevalence of PCOS and distribution across BMI categories (N = 180)

Category	n in category	PCOS, n (%)	95% CI (PCOS within category)
Normal BMI	52	10 (19.2%)	10.7% to 31.7%
Overweight	39	12 (30.8%)	18.4% to 46.9%
Obese	89	40 (44.9%)	34.5% to 55.8%
Overall PCOS prevalence	180	62 (34.4%)	27.9% to 41.6%

Tests of significance: Across BMI categories (PCOS vs non-PCOS, 3×2): χ^2 = 9.91, df=2; p = 0.007. One-sample check vs a reference prevalence of 25% (literature-based): z = 2.93; p = 0.0034.

Table 2 illustrates the distribution of PCOS prevalence across BMI categories. Among the 180 women, PCOS was diagnosed in 19.2% of those with normal BMI, 30.8% of overweight women, and 44.9% of obese women. The overall PCOS prevalence was 34.4%, consistent with regional Indian data. A statistically significant trend was observed between BMI and PCOS occurrence (χ^2 = 9.91, df = 2, p = 0.007), confirming that the likelihood of PCOS increased with rising BMI. Furthermore, when compared with a reference prevalence of 25% reported in prior literature, the observed prevalence was significantly higher (z = 2.93, p = 0.0034). This indicates that obesity is a major contributing factor to PCOS pathogenesis in this cohort, supporting the established link between excess adiposity, insulin resistance, and hyperandrogenism.

Table 3. Prevalence of Metabolic Syndrome (MetS) in women with PCOS (N=180; PCOS n=62)

Group	Met S present, n (%)	95% CI	Comparator	Effect (95% CI)	Test (df)	p-value
PCOS (n=62)	26 (41.9%)	30.5% to 54.3%	vs non-PCOS	RR = 2.25 (1.40-3.63)	χ^2=11.28 (1)	0.0008
Non-PCOS (n=118)	22 (18.6%)	12.6% to 26.6%	-	-	-	-

Note: **RR = risk ratio of MetS comparing PCOS to non-PCOS; CI via log method.**

Table 3 highlights the comparative prevalence of Metabolic Syndrome among PCOS and non-PCOS women. Among 62 women diagnosed with PCOS, 26 (41.9%) had MetS (95% CI: 30.5-54.3%), whereas only 22 (18.6%) of the 118 non-PCOS participants were affected (95% CI: 12.6-26.6%). The calculated relative risk was 2.25 (95% CI: 1.40-3.63), with a statistically significant association (χ^2 = 11.28, p = 0.0008). These findings suggest that the coexistence of PCOS and MetS is common and that women with PCOS are more than twice as likely to develop MetS as their non-PCOS counterparts. This relationship underscores the shared metabolic pathways-particularly insulin resistance and central

obesity-that link these two disorders and contribute to long-term cardiovascular risk.

Table 4. Association between individual MetS components and presence of PCOS (PCOS n=62; non-PCOS n=118)

MetS component (ATP III cut-offs)	PCOS n/N (%)	Non-PCOS n/N (%)	Effect size	95% CI	χ^2 (df=1)	p-value
Central obesity (waist ≥88 cm)	35/62 (56.5%)	42/118 (35.6%)	RR 1.59	1.14-2.20	7.22	0.0072
Triglycerides ≥150 mg/dL	32/62 (51.6%)	39/118 (33.1%)	RR 1.56	1.10-2.22	5.86	0.0155
HDL-C <50 mg/dL	38/62 (61.3%)	50/118 (42.4%)	RR 1.45	1.08-1.93	5.82	0.0160
BP ≥130/85 mmHg	21/62 (33.9%)	27/118 (22.9%)	RR 1.48	0.92-2.39	2.51	0.113
Fasting glucose ≥110 mg/dL	19/62 (30.6%)	22/118 (18.6%)	RR 1.64	0.97-2.80	3.33	0.068

Table 4 analyzes the relationship between each component of MetS and PCOS. Central obesity (waist ≥ 88 cm) was significantly higher in the PCOS group (56.5%) compared with non-PCOS women (35.6%) with a RR = 1.59 (95% CI: 1.14-2.20; p = 0.0072). Similarly, elevated triglycerides (≥150 mg/dL) and low HDL-C (<50 mg/dL) were significantly more prevalent in PCOS women, with RRs of 1.56 (95% CI: 1.10-2.22; p = 0.0155) and 1.45 (95% CI: 1.08-1.93; p = 0.0160), respectively. Elevated blood pressure and fasting glucose abnormalities were more frequent in the PCOS group (33.9% and 30.6%, respectively) but did not reach statistical significance (p > 0.05).

DISCUSSION

In our hospital-based cohort of 180 reproductive-age women, the mean age (27.9 ± 4.2 years) and adiposity profile (BMI 26.8 ± 3.9 kg/m²; mean waist 87.3 ± 8.1 cm) indicate a predominantly overweight population using Indian cardiometabolic risk cut-offs, a pattern that mirrors urban Indian clinic samples and aligns with the higher body-fat-at-given-BMI paradigm described for South Asians Sharma A et al.(2025)[6] The overall prevalence of PCOS was 34.4% (95% CI: 27.9-41.6). This is considerably higher than community estimates from Western cohorts (=8-13%) and many Indian population studies, but is consistent with symptomatic, hospital-based sampling and the use of Rotterdam criteria, which are known to broaden case ascertainment Joham AE et al. (2025) [7]. Indeed, the "phenotypic expansion" under Rotterdam-by allowing any two of oligo-ovulation, hyperandrogenism, and polycystic ovaries-has repeatedly been shown to inflate prevalence compared with NIH criteria Fatima SH et al. (2022) [8]

Metabolic risk in the cohort was substantial: MetS prevalence was 26.7% overall, but 41.9% among women with PCOS versus 18.6% among non-PCOS, yielding a relative risk of 2.25 (95% CI: 1.40-3.63; p = 0.0008). This magnitude is concordant with meta-analytic data showing =2-3-fold higher odds of MetS in PCOS after accounting for age and adiposity Zeng W et al. (2025) [9]. Classic clinic-based series also reported high MetS burdens in PCOS (=33-43%), particularly where central obesity and atherogenic dyslipidemia co-cluster Naeem I et al. (2025) [10]. Our lipid pattern (mean triglycerides 154.2 ± 41.7 mg/dL; HDL-C 44.1 ± 9.3 mg/dL) and fasting glucose (97.4 ± 12.6 mg/dL) reflect precisely that dyslipidemic signature; it is the TG-↑/HDL-↓ axis-rather than frank hyperglycemia-that most clearly separates PCOS from non-PCOS at these ages Muhaidat N et al. (2023) [11].

The BMI-stratified analysis demonstrated a clear dose-response: PCOS prevalence rose from 19.2% in normal-BMI women to 30.8% in overweight and 44.9% in obesity (χ^2 = 9.91, p = 0.007). This gradient supports the mechanistic link between adiposity, insulin

resistance, and hyperandrogenism emphasized in pathophysiologic reviews Giri A et al. (2022) [1]. It also echoes Indian meta-analytic evidence that higher regional estimates of PCOS are partly driven by rising obesity and central adiposity Joham AE et al. (2025) [7]. Notably, our one-sample comparison showed the overall PCOS prevalence exceeded a reference 25% ($z = 2.93$, $p = 0.0034$), underscoring the burden in clinic-attending women.

When individual MetS components were contrasted by PCOS status, central obesity (RR 1.59; $p = 0.0072$), triglycerides ≥ 150 mg/dL (RR 1.56; $p = 0.0155$), and HDL-C < 50 mg/dL (RR 1.45; $p = 0.0160$) were significantly enriched in PCOS, whereas elevated blood pressure and impaired fasting glucose trended higher but were not statistically significant. This prioritization of adiposity and atherogenic dyslipidemia over overt glycemia at a mean age =28 years matches the trajectory described in longitudinal and cross-sectional reports-lipid abnormalities and central obesity manifest early, with glycemic thresholds crossed later as β-cell compensation wanes Tanveer U et al. (2025) [12]. The pattern also dovetails with ATP-III/IDF frameworks where waist circumference and the TG/HDL pair dominate risk clustering in young women Sharma A et al. (2025) [6].

CONCLUSION

The present cross-sectional study demonstrated a high prevalence of Polycystic Ovary Syndrome (PCOS) (34.4%) among reproductive-age women, with a significant association between PCOS and Metabolic Syndrome (MetS). Nearly 42% of women with PCOS fulfilled the criteria for MetS compared to 18.6% among non-PCOS women, indicating that PCOS confers over two-fold increased metabolic risk (RR = 2.25, $p < 0.001$). Central obesity, hypertriglyceridemia, and low HDL-C were the most prominent metabolic abnormalities contributing to this association. The prevalence of PCOS was also found to increase with higher BMI, underscoring obesity as an important modifiable risk factor. These findings highlight the need for routine metabolic screening and lifestyle interventions in all women with PCOS to prevent long-term complications such as type 2 diabetes mellitus, hypertension, and cardiovascular disease. Early detection and integrated management addressing both reproductive and metabolic aspects can substantially reduce morbidity and improve quality of life in this population.

Limitations

1. The study was hospital-based and may not reflect the true prevalence of PCOS and MetS in the general community.
2. Being a cross-sectional design, the study establishes association but not causation between PCOS and metabolic abnormalities.
3. The sample size (N = 180), although adequate for prevalence estimation, may have limited power to detect subtle subgroup differences.
4. Insulin resistance markers such as HOMA-IR and fasting insulin were not analyzed, which could have provided deeper insight into the metabolic mechanisms.
5. Lifestyle factors, dietary intake, and physical activity patterns were not comprehensively evaluated and may have influenced metabolic outcomes.
6. Hormonal assays and ultrasound findings were based on single-time assessments, which could vary with menstrual phase or inter-observer differences.

Declaration by Authors

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Conflict of Interest: Nil

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