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Polycystic Ovary Syndrome and Cardiometabolic Risk in Reproductive Age Women: A Phenotype-Stratified Analysis

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Abstract — Polycystic ovary syndrome (PCOS) is the most common endocrine disorder affecting reproductive-age women, with established associations with metabolic syndrome, type 2 diabetes, and cardiovascular disease. The Rotterdam criteria define four PCOS phenotypes (A-D) based on combinations of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, with the hyperandrogenic phenotypes (A and B) generally associated with greater cardiometabolic risk. We undertook a cross-sectional analysis of 312 reproductive-age women with PCOS at a tertiary endocrinology service, stratified by Rotterdam phenotype and characterised across multiple cardiometabolic dimensions. The classical hyperandrogenic phenotype A accounted for the largest fraction (39.7%), with phenotypes B, C, and D accounting for 17.9%, 25.0%, and 17.3% respectively. Metabolic syndrome prevalence reached 62.4% in obese phenotype A women, contrasting with 9.4% in lean phenotype D women. HOMA-IR correlated strongly with BMI (r = 0.62) but showed substantial residual variance attributable to PCOS phenotype. Strongest independent predictors of metabolic syndrome included BMI ≥30, HOMA-IR ≥2.5, phenotype A, waist-to-hip ratio, and free androgen index. The findings support phenotype-aware cardiometabolic surveillance and targeted intervention in PCOS clinics.

Keywords — *Polycystic Ovary Syndrome; PCOS; Metabolic Syndrome; Hyperandrogenism; Insulin Resistance; Cardiometabolic; Reproductive Endocrinology.*

1. Introduction

Polycystic ovary syndrome affects approximately 8-13% of reproductive-age women globally and substantially higher fractions in some South Asian populations where the syndrome appears particularly prevalent. The clinical presentation combines reproductive features menstrual irregularity, anovulatory infertility, hyperandrogenic skin and hair manifestations with metabolic features that include insulin resistance, dyslipidaemia, and an elevated risk of type 2 diabetes and cardiovascular disease. The condition emerged from gynaecological clinics decades ago but has since been recognised as a systemic metabolic disorder that extends well beyond the reproductive system. Diagnostic frameworks have evolved over time. The 2003 Rotterdam criteria requiring two of three features (clinical or biochemical hyperandrogenism, ovulatory dysfunction, polycystic ovarian morphology) yields four diagnostic phenotypes (A, B, C, D) that differ both in clinical presentation and in associated cardiometabolic risk. The hyperandrogenic phenotypes (A and B), which include biochemical or clinical androgen excess, generally show stronger metabolic associations than the non-hyperandrogenic phenotype D. Phenotype-based stratification is therefore increasingly used to guide cardiometabolic surveillance (Jha, Kumar,, & Neha, 2026; Kumar, Gautam,, & Maitiy, 2026; Yatish, Khatoon,, & Kumar, 2026). We undertook a cross-sectional analysis at our tertiary endocrinology service to characterise the phenotype distribution of PCOS, examine cardiometabolic features across phenotypes and BMI strata, identify independent predictors of metabolic syndrome, and inform stratified surveillance and intervention strategies for women with PCOS at our institution and similar settings.

2. Methods

We conducted a cross-sectional analysis of women aged 18-40 years with PCOS diagnosed at the endocrinology clinic of a tertiary medical centre between June 2023 and May 2024. Diagnosis required Rotterdam criteria (at least two of three: clinical or biochemical hyperandrogenism, ovulatory dysfunction, polycystic ovarian morphology on transvaginal ultrasound) after exclusion of alternative causes including hyperprolactinaemia, congenital adrenal hyperplasia, androgen-secreting tumour, Cushing syndrome, and thyroid dysfunction. The final cohort comprised 312 women. Each patient was classified into one of the four Rotterdam phenotypes. Hyperandrogenism (HA) was defined as clinical hirsutism (modified Ferriman-Gallwey score ≥8) or biochemical hyperandrogenism (free androgen index >5 or free testosterone above the assay-specific 95th percentile). Ovulatory dysfunction (OD) was defined as oligomenorrhoea (<8 cycles per year) or amenorrhoea. Polycystic ovarian morphology (PCOM) required ≥12 follicles per ovary of 2-9 mm diameter or ovarian volume >10 mL on transvaginal ultrasound. All patients underwent comprehensive cardiometabolic assessment

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including anthropometry (height, weight, BMI, waist and hip circumference, waist-to-hip ratio), blood pressure, fasting glucose, fasting insulin (with HOMA-IR calculation), HbA1c, 75-g oral glucose tolerance test, lipid profile, high-sensitivity CRP, and reproductive hormones including LH, FSH, total and free testosterone, sex-hormone binding globulin, DHEAS, anti-Müllerian hormone, and 17-OH-progesterone. Metabolic syndrome was defined per modified NCEP-ATP III criteria with South Asian waist thresholds. Continuous variables are summarised as median (IQR) or mean (SD) and compared across phenotypes using Kruskal-Wallis or one-way ANOVA. Categorical variables used chi-squared testing. Multivariable logistic regression identified independent predictors of metabolic syndrome incorporating phenotype alongside other clinical and biochemical covariates. The interaction between phenotype and BMI on metabolic syndrome prevalence was examined.

3. Results

3.1 Cohort Characteristics and Phenotype Distribution

Phenotype distribution across the cohort is shown in Figure 1. Phenotype A (hyperandrogenism + ovulatory dysfunction + PCOM), the classical fully expressed PCOS, was the most common at 39.7% (124 patients). Phenotype C (hyperandrogenism + PCOM without ovulatory dysfunction) was the second most common at 25.0%. Phenotypes B (without PCOM) and D (without hyperandrogenism) accounted for 17.9% and 17.3% respectively.

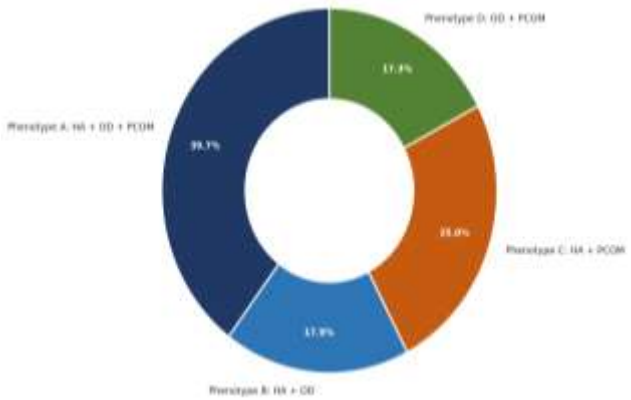

Fig.1: PCOS phenotype distribution by Rotterdam criteria

Table 1. Characteristics by PCOS Rotterdam phenotype

Characteristic	Pheno A (n=124)	Pheno B (n=56)	Pheno C (n=78)	Pheno D (n=54)
Age, mean (SD), years	27.4 (5.6)	26.8 (5.8)	28.2 (5.4)	26.4 (5.2)
Age at menarche, mean (SD), years	12.8 (1.6)	13.2 (1.4)	12.6 (1.6)	13.0 (1.4)
BMI, mean (SD), kg/m²	29.4 (5.6)	28.2 (5.2)	26.2 (4.8)	25.6 (4.4)
BMI ≥30, n (%)	48 (38.7)	18 (32.1)	16 (20.5)	8 (14.8)
Waist circumference, mean, cm	92.4 (12.4)	88.6 (11.2)	84.2 (10.8)	82.4 (10.4)
Waist-to-hip ratio, mean	0.88 (0.06)	0.86 (0.06)	0.84 (0.06)	0.82 (0.06)
Hirsutism (mFG ≥8), n (%)	118 (95.2)	52 (92.9)	72 (92.3)	0 (0.0)
Modified Ferriman-Gallwey score, mean	12.4	11.8	11.2	4.2
Acne, n (%)	82 (66.1)	36 (64.3)	48 (61.5)	16 (29.6)
Acanthosis nigricans, n (%)	48 (38.7)	18 (32.1)	16 (20.5)	6 (11.1)
Cycle length, median, days	>90	>90	32	>90
Oligomenorrhoea/amenorrhoea, n (%)	124 (100.0)	56 (100)	0 (0.0)	54 (100.0)
Documented infertility, n (%)	48 (38.7)	22 (39.3)	12 (15.4)	18 (33.3)
Family history of T2DM, n (%)	68 (54.8)	28 (50.0)	32 (41.0)	18 (33.3)
Family history of PCOS, n (%)	42 (33.9)	18 (32.1)	26 (33.3)	16 (29.6)

3.2 Metabolic Syndrome by Phenotype × BMI

Metabolic syndrome prevalence varied substantially across PCOS phenotype and BMI strata (Figure 2). Within each phenotype, metabolic syndrome prevalence rose steeply with increasing BMI. Within each BMI stratum, the hyperandrogenic phenotypes A and B showed higher prevalence than the non-hyperandrogenic phenotype D. The highest prevalence 62.4% was observed in obese phenotype A women, while the lowest 9.4% was in lean phenotype D women. The roughly six-fold gradient across this matrix demonstrates the joint contribution of androgen burden and adiposity to cardiometabolic risk in PCOS

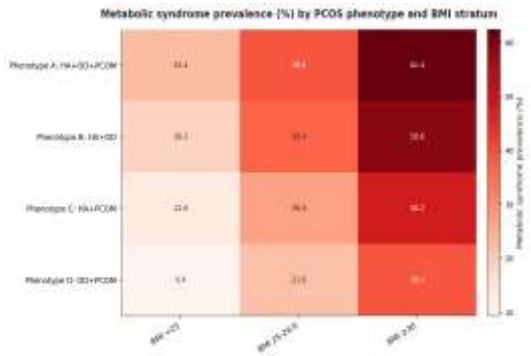

Fig. 2: Metabolic syndrome prevalence by PCOS phenotype and BMI stratum

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3.3 Biochemical and Metabolic Profile

Table 2. Biochemical and metabolic parameters by PCOS phenotype

Parameter	Pheno A (n=124)	Pheno B (n=56)	Pheno C (n=78)	Pheno D (n=54)
Free androgen index, mean (SD)	8.4 (4.2)	7.8 (3.8)	6.4 (3.4)	2.6 (1.4)
Total testosterone, mean (SD), ng/dL	68 (22)	62 (20)	58 (18)	32 (12)
SHBG, mean (SD), nmol/L	28 (12)	32 (14)	36 (16)	52 (22)
DHEAS, mean (SD), µg/dL	248 (98)	224 (92)	218 (88)	158 (72)
AMH, mean (SD), ng/mL	8.4 (4.2)	6.2 (3.4)	8.6 (4.4)	8.2 (4.0)
Fasting glucose, mean (SD), mg/dL	102 (24)	98 (22)	94 (18)	92 (16)
HbA1c, mean (SD), %	5.8 (0.8)	5.6 (0.8)	5.4 (0.6)	5.4 (0.6)
Fasting insulin, mean (SD), µIU/mL	18.4 (10.6)	15.2 (8.4)	12.6 (7.2)	10.4 (6.4)
HOMA-IR, mean (SD)	4.8 (2.8)	3.8 (2.2)	2.9 (1.8)	2.4 (1.4)
HOMA-IR ≥2.5, n (%)	78 (62.9)	32 (57.1)	36 (46.2)	18 (33.3)
Total cholesterol, mean (SD), mg/dL	198 (38)	192 (36)	186 (34)	178 (32)
Triglycerides, median (IQR), mg/dL	148	138	118	104
HDL-cholesterol, mean (SD), mg/dL	42 (10)	44 (10)	48 (12)	52 (12)
hsCRP, median, mg/L	3.2	2.8	1.8	1.4
Vitamin D deficiency (<20 ng/mL), n (%)	78 (62.9)	32 (57.1)	42 (53.8)	26 (48.1)

3.4 HOMA-IR and BMI Relationship

Insulin resistance assessed by HOMA-IR correlated strongly with BMI across the cohort (Pearson r = 0.62; Figure 3). The relationship was broadly linear, with each unit increase in BMI associated with approximately 0.18 increase in HOMA-IR. Substantial residual variance the scatter around the regression line was attributable to PCOS phenotype, with hyperandrogenic phenotypes showing higher HOMA-IR at any given BMI than non-hyperandrogenic phenotypes. The implication is that adiposity-independent insulin resistance in hyperandrogenic PCOS warrants attention beyond weight-based interventions alone.

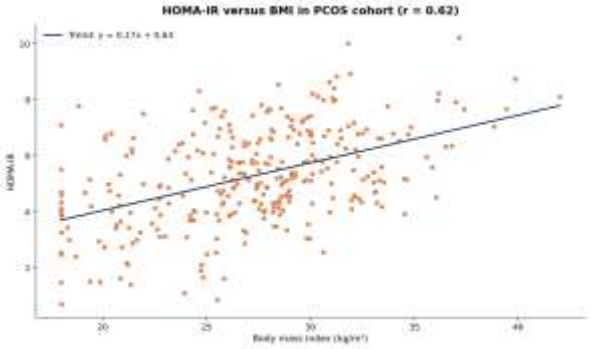

Fig.3: HOMA-IR versus BMI in the PCOS cohort

3.5 Predictors of Metabolic Syndrome

Multivariable logistic regression identified ten independent predictors of metabolic syndrome in this PCOS cohort (Figure 4). Obesity (BMI ≥30) carried the strongest single association (adjusted OR 4.21), followed by HOMA-IR ≥2.5 (OR 3.84) and phenotype A (OR 3.46 versus phenotype D). Waist-to-hip ratio, free androgen index, acanthosis nigricans, family history of type 2 diabetes, and advanced age all retained independent predictive value. Notably, the predictors include both inherent biological features (phenotype, hormone profile) and modifiable lifestyle and weight-related features.

Table 3. Cardiometabolic comorbidity and reproductive outcomes by phenotype

Outcome	Pheno A (n=124)	Pheno B (n=56)	Pheno C (n=78)	Pheno D (n=54)
Metabolic syndrome, n (%)	48 (38.7)	18 (32.1)	16 (20.5)	6 (11.1)
Pre-diabetes (HbA1c 5.7-6.4 or IGT), n (%)	42 (33.9)	16 (28.6)	18 (23.1)	8 (14.8)
Type 2 diabetes, n (%)	18 (14.5)	6 (10.7)	4 (5.1)	2 (3.7)
Hypertension on treatment, n (%)	32 (25.8)	12 (21.4)	12 (15.4)	6 (11.1)
Documented NAFLD on imaging, n (%)	48 (38.7)	18 (32.1)	16 (20.5)	8 (14.8)
Carotid IMT abnormal, n (%)	38 (30.6)	14 (25.0)	12 (15.4)	6 (11.1)
Reported infertility, n (%)	48 (38.7)	22 (39.3)	12 (15.4)	18 (33.3)
Documented infertility treatment, n (%)	32 (25.8)	16 (28.6)	6 (7.7)	12 (22.2)
Pregnancy during PCOS care, n (%)	18 (14.5)	6 (10.7)	12 (15.4)	12 (22.2)

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Gestational diabetes (of pregnancies), n	6/18	2/6	2/12	2/12
Documented endometrial sampling, n	12	4	2	6

3.6 Implications for Stratified Care

Table 4. Surveillance and intervention recommendations by phenotype-BMI category

Category	**Number in cohort**	**Suggested intervention priority**
Phenotype A, BMI ≥30	48 (15.4 of cohort)	Most intensive: structured weight management, metformin, statin if dyslipidaemic
Phenotype A or B, BMI 25-30	42 (13.5)	Lifestyle intervention, consider metformin if HOMA-IR ≥2.5
Phenotype A or B, BMI <25	32 (10.3)	Lifestyle counselling, monitor HOMA-IR and lipid profile
Phenotype C or D, BMI ≥30	24 (7.7)	Weight management; lower metabolic urgency than hyperandrogenic
Phenotype C, BMI 25-30	32 (10.3)	Lifestyle, annual metabolic screen
Phenotype D, BMI <25	28 (9.0)	Lowest metabolic risk; focus on reproductive concerns
Documented cardiometabolic surveillance frequency	-	Annually for phenotypes A, B; 2-yearly for C, D if metabolically normal
Endometrial protection in oligomenorrhoea	-	Cyclic progesterone or OCP in all OD phenotypes

4. Discussion

Across 312 women with PCOS evaluated cross-sectionally, three observations have direct implications for clinical care delivery. First, the cardiometabolic risk gradient across phenotype-BMI combinations is substantial six-fold between the lowest-risk (lean phenotype D) and highest-risk (obese phenotype A) groups. This gradient is operationally actionable: women in higher-risk cells warrant more intensive surveillance, earlier pharmacotherapy consideration (metformin, statin), and more aggressive lifestyle intervention. Women in lower-risk cells may appropriately receive lighter-touch monitoring with focus on reproductive concerns. Second, the strong correlation between HOMA-IR and BMI (r = 0.62) confirms the dominant role of adiposity in PCOS-associated insulin resistance, but the substantial residual variance attributable to phenotype reminds us that PCOS involves intrinsic insulin resistance beyond what BMI alone explains. Hyperandrogenic phenotypes show higher HOMA-IR at any given BMI, supporting the case for considering metformin or other insulin-sensitising therapy in women with hyperandrogenic PCOS even when BMI is in the non-obese range (Jha, Kumar,, & Neha, 2026; Kumar, Gautam,, & Maitiy, 2026; Yatish, Khatoon,, & Kumar, 2026). Third, the multivariable predictors identified offer a concise clinical risk-stratification framework. BMI ≥30, HOMA-IR ≥2.5, phenotype A, waist-to-hip ratio, and family history of T2DM together capture most of the cardiometabolic risk variance in this population. A simple structured assessment incorporating these features would provide adequate risk stratification for most clinical decisions without requiring sophisticated biomarker panels (Bhatnagar, Kumar,, & Shivam, 2026; Jha, Kumar,, & Neha, 2026). Implementation considerations include integration of structured cardiometabolic assessment into PCOS clinic templates (rather than the historical reproductive-only focus), patient education about the systemic nature of PCOS, longitudinal weight-management support which remains the cornerstone intervention, and access to evidence-based pharmacotherapy including metformin and (in selected cases) GLP-1 receptor agonists.

Digital tools for menstrual cycle tracking, dietary monitoring, and structured behavioural intervention show promise particularly for younger women (Deepa et al., 2026; Catherine, Gupta, Gopi,, & Swadhi, 2025; Swadhi, Gayathri, Suresh, Catherine,, & Velmurugan, 2025; Vettriselvan, Ramya, et al., 2026; Vinodh, Subramani,, & Vettriselvan, 2026; Subramani, Chillagattu, et al., 2026; Selvi et al., 2026). Reproductive concerns including infertility, pregnancy outcomes, and endometrial protection remain integral but should be treated as one component of comprehensive PCOS care alongside cardiometabolic management. Limitations include the cross-sectional design which constrains causal inference about phenotype-to-cardiometabolic-outcome trajectories; the single-centre tertiary setting which may attract a sample with more severe PCOS than community-level samples; the absence of long-term cardiovascular event data; and the imperfect phenotype boundary classifications, particularly between phenotypes B (no PCOM) and C (ovulatory hyperandrogenism). Anti-Müllerian hormone thresholds for PCOM definition vary across populations and assays, introducing some variability into phenotype assignment.

5. Conclusion

PCOS-associated cardiometabolic risk varies substantially by Rotterdam phenotype and BMI, with a six-fold gradient from lean non-hyperandrogenic women to obese fully-expressed hyperandrogenic women. The risk is driven jointly by adiposity, insulin resistance, and androgen burden, with each contributing independent predictive value. Phenotype-aware structured cardiometabolic

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surveillance, combined with targeted lifestyle and pharmacological intervention, offers a practical framework for risk reduction in this common condition whose long-term consequences extend well beyond the reproductive system.

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