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A Cross-Sectional Study of the Ultrasonographic and Metabolic Characteristics of Women with Polycystic Ovary Syndrome

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting women of reproductive age and is frequently associated with increased ovarian volume, excess antral follicles, insulin resistance, and lipid abnormalities. Pelvic ultrasonography and fasting metabolic markers provide a practical approach for early risk characterization at diagnosis.

Methods: This cross-sectional study evaluated 120 women aged 18–39 years with PCOS diagnosed according to the Rotterdam criteria. Transabdominal pelvic ultrasound (3.5–7 MHz probe) measured ovarian volume (ellipsoid formula) and antral follicle count (2–9 mm). After 8–12 hours fasting, venous samples analyzed for serum glucose, insulin, triglycerides, total cholesterol, HDL, LDL, and VLDL. HOMA-IR was calculated as (glucose × insulin)/405, with>2.5 defining insulin resistance. Data were expressed as mean±SD or percentages, analyzed using SPSS v25 with Pearson correlation ($p<0.05$).

Results: PCOM was present in 86 women (71.7%); 83 (69.2%) had AFC>12/ovary and 74 (61.7%) showed bilateral involvement. Elevated fasting insulin was found in 63 (52.5%) (mean 18.9±6.2 µIU/mL); 57 (47.5%) had insulin resistance (mean HOMA-IR 3.1±1.2). High TG was seen in 47 (39.2%) (mean 162.7±41.5 mg/dL); low HDL in 37 (30.8%) (mean 41.2±6.6 mg/dL); elevated LDL in 50 (41.7%) (mean 129.6±32.3 mg/dL); composite dyslipidemia in 47 (39.2%). Ovarian volume correlated with HOMA-IR ($r=0.41$, $p<0.001$).

Conclusion: Ovarian enlargement and metabolic dysfunction are frequent at diagnosis. Routine combined ultrasound and fasting metabolic screening may support early risk-based counseling and timely preventive care.

Key-words: Polycystic ovary syndrome, Ovarian volume, Antral follicle count, Insulin resistance, HOMA-IR, Dyslipidemia, Cardiometabolic risk

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a highly prevalent endocrine disorder that impacts women primarily during their reproductive years. Beyond being a leading contributor to ovulatory infertility, the syndrome carries a sustained risk of metabolic dysfunction that may persist long after initial clinical recognition.

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The Rotterdam consensus remains the most widely accepted diagnostic framework for PCOS.[1] Pelvic ultrasonography remains a key clinical tool for evaluating polycystic ovarian morphology (PCOM), which typically manifests as increased ovarian size, stromal hypertrophy, and a high density of small antral follicles, collectively reflecting disordered follicular recruitment and maturation. [2,3] These structural features are not merely diagnostic markers but may also provide indirect insight into underlying ovarian microenvironmental stress. Contemporary evidence suggests that rigid sonographic cut-offs may not apply equally across all age groups, and that individualized interpretation may improve diagnostic accuracy. [4,5]

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A major pathophysiological pillar of PCOS is insulin resistance (IR), a systemic abnormality that influences both ovarian and extra-ovarian physiology. Excess circulating insulin may enhance androgen production, interfere with normal follicular progression, and contribute to metabolic imbalance even before overt biochemical disease is clinically detected [6,7].

Disturbances in lipid metabolism, including elevated triglycerides and LDL, along with reduced HDL, are frequent in PCOS and may accelerate cardiometabolic vulnerability by fostering early endothelial stress and subclinical vascular risk. [8,9] Importantly, metabolic dysfunction in PCOS may not always parallel BMI status; a subset of women with normal body composition may still exhibit impaired insulin sensitivity and lipid irregularities, emphasizing that ovarian dysfunction and metabolic risk can coexist independently of obesity. [7,10] This reinforces the clinical need for early metabolic evaluation rather than delayed testing after symptom progression. This study aimed to characterize baseline ultrasonographic features and metabolic risk profiles in women with PCOS compared with healthy age-matched controls.

MATERIALS AND METHODS

Research Design- This institutional study followed a cross-sectional observational approach to evaluate baseline ovarian ultrasonographic and metabolic parameters in women diagnosed with PCOS. Data collection was performed at the Department of Obstetrics and Gynecology, Adichunchanagiri Institute of Medical Sciences, from January 2023 to July 2024.

Methodology- Fasting venous blood samples were collected after 8–12 hours of overnight fasting and analyzed using calibrated automated analyzers to measure serum glucose, insulin, total cholesterol, triglycerides, HDL, LDL, and VLDL. HOMA-IR was calculated as (fasting glucose × fasting insulin)/405, and values >2.5 were considered indicative of biochemical insulin resistance. [8,9,11,12] Eligible participants also underwent pelvic ultrasonography using a standardized transabdominal scanning protocol with a 3.5–7 MHz curvilinear probe. Ovarian volume was calculated using the ellipsoid formula (length × width × thickness × 0.523), and antral follicles measuring 2–9 mm were manually counted in each ovary. PCOM was defined as ovarian volume >10 mL and/or an antral follicle count >12 per ovary, following accepted sonographic criteria. [13–17] Lipid abnormalities were recorded both as individual markers and as a composite dyslipidemia profile when any lipid parameter exceeded the clinically accepted risk threshold [8,9,18].

Inclusion Criteria

1) Females aged 18–39 years
2) New clinical diagnosis of PCOS based on the Rotterdam 2003 criteria
3) Willing to participate and provide informed consent
4) Recruited consecutively after clinical confirmation to capture baseline status in real time
5) Underwent ultrasonographic and fasting metabolic evaluation** at the time of diagnosis [1,7]

Exclusion Criteria

1) Were pregnant at the time of evaluation
2) Were breastfeeding or in the postpartum period
3) Had diagnosed thyroid disorders (hypo/ hyperthyroidism)
4) Had adrenal or pituitary endocrine disease
5) Had clinical evidence of cortisol excess or related syndrome
6) Had ovarian cysts or tumors unrelated to PCOS
7) Had congenital adrenal hyperplasia or Cushing-like conditions
8) Had taken oral contraceptives, ovulation-inducing hormones, insulin-sensitizing drugs, or lipid-lowering therapy within 12 weeks before enrollment [7,9,11,12]

Statistical analysis- All clinical and biochemical variables were entered into SPSS software (version 25). Quantitative data were expressed as mean±standard deviation, and categorical variables as proportions; Pearson correlation testing was used to examine the direction and strength of association between ovarian measurements and metabolic indices. Statistical significance for all analytical comparisons was set at a two-tailed p-value<0.05.

Ethical Approval- The study protocol was reviewed and approved by the Institutional Ethics Committee of Adichunchanagiri Institute of Medical Sciences. All participants provided written informed consent before enrollment. Privacy, confidentiality, and institutional ethical standards were upheld throughout the study.

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RESULTS

A total of 120 women with clinically confirmed PCOS and 120 age-matched healthy participants were evaluated for ovarian ultrasonographic markers and fasting metabolic parameters.

Baseline demographic analysis showed no significant age difference between the PCOS and control groups (26.7±4.9 vs 26.3±5.1 years, p=0.56), indicating appropriate age alignment. However, BMI was significantly higher in the PCOS cohort (27.1±5.3 kg/m²) than in controls (23.4±3.9 kg/m²; p<0.001). Overweight or obesity was present in 58.3% of women with PCOS versus 22.5% in the control group (p<0.001). Menstrual irregularity, a key clinical manifestation of PCOS, was reported by 100 women (83.3%), while none of the controls exhibited cycle disturbances. Table 1 shows baseline demographic alignment and clinical contrast between PCOS women and healthy participants. Age is comparable, while BMI and menstrual cycle abnormalities are significantly higher in the PCOS group.

Table 1: Baseline Demographic and Clinical Profile of Study Participants

Parameter	PCOS Cases (n=120)	Controls (n=120)	p-value
Age (years)	26.7±4.9	26.3±5.1	0.56
BMI (kg/m²)	27.1±5.3	23.4±3.9	<0.001*
Overweight/Obese (%)	58.3%	22.5%	<0.001*
Menstrual irregularity (%)	83.3%	0%	<0.001*

**p-value<0.05 was significant*

Ultrasonographic assessment revealed that PCOM was present in 86 women (71.7%). The mean ovarian volume was 13.2±3.4 mL in the PCOS group, nearly double that observed in controls (6.8±1.9 mL, p<0.001). An antral follicle count (AFC) exceeding 12 per ovary was detected in 83 women (69.2%), while no control participant crossed this threshold. Bilateral ovarian involvement was present in 74 women (61.7%), reflecting symmetrical ovarian morphological expression in the majority of cases. Table 2 represents ovarian structural changes assessed using transabdominal pelvic ultrasonography. Increased ovarian volume, excess antral follicles, and bilateral involvement are highly prevalent in PCOS, while absent in controls.

Table 2: Ovarian Morphological Features on Pelvic Ultrasonography in PCOS

Ultrasound Marker	PCOS Cases	Controls	p-value
Ovarian volume (mL)	13.2±3.4	6.8±1.9	<0.001*
AFC>12/ovary (%)	69.2%	0%	<0.001*
Bilateral involvement (%)	61.7%	0%	<0.001*
PCOM present (%)	71.7%	0%	<0.001*

**p-value<0.05 was significant*

Metabolic profiling demonstrated that fasting insulin levels were elevated (≥20 µIU/mL) in 63 women with PCOS (52.5%), compared with only 9 women in the control group (7.5%, p<0.001). Fasting glucose impairment (≥100 mg/dL) was observed in 34 women (28.3%) compared with 5 controls (4.2%; p<0.001). The mean HOMA-IR score was 3.1±1.2 in the PCOS group, compared with 1.1±0.4 in controls (p<0.001). 57 women (47.5%) met the biochemical criteria for insulin resistance (HOMA-IR > 2.5), compared with 8 controls (6.7%). Triglyceride elevation (>150 mg/dL) was present in 47 women (39.2%) with a mean TG of 162.7±41.5 mg/dL, while 37 women (30.8%) had reduced HDL (mean 41.2±6.6 mg/dL). LDL levels exceeded 130 mg/dL in 50 women (41.7%) with a mean LDL of 129.6±32.3 mg/dL. Composite dyslipidemia, defined by any lipid abnormality, was present in 47 women (39.2%) compared with 13 controls (10.8%, p<0.001). Table 3 displays fasting metabolic and lipid abnormalities at the

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time of diagnosis. PCOS women exhibit significantly higher rates of insulin elevation, HOMA-IR impairment, triglyceride elevation, reduced HDL, and composite dyslipidemia compared to healthy participants.

Table 3: Cardiometabolic Risk Markers and Lipid Abnormalities in Women with PCOS

Metabolic Marker	PCOS Cases	Controls	p-value
Fasting glucose ≥100 mg/dL (%)	28.3%	4.2%	<0.001*
Fasting insulin ≥20 µIU/mL (%)	52.5%	7.5%	<0.001*
HOMA-IR	3.1±1.2	1.1±0.4	<0.001*
HOMA-IR>2.5 (%)	47.5%	6.7%	<0.001*
TG>150 mg/dL (%)	39.2%	8.3%	<0.001*
HDL <40 mg/dL (%)	30.8%	5.8%	<0.001*
LDL>130 mg/dL (%)	41.7%	9.2%	<0.001*
Composite dyslipidemia (%)	39.2%	10.0%	<0.001*

**p-value<0.05 was significant*

Correlation Analysis, displayed in Table 4, showed that baseline ovarian volume demonstrated a significant positive association with HOMA-IR (r=0.41, p<0.001) and a moderate correlation with fasting triglyceride levels (r=0.32, p=0.002). Additionally, HDL showed a significant inverse correlation with fasting insulin (r = –0.38, p < 0.001). These associations suggest that structural ovarian changes in PCOS may parallel the severity of metabolic dysfunction, particularly insulin resistance and lipid imbalance.

Table 4 summarizes the strength and direction of association between ovarian morphology (ovarian volume, PCOM features) and key metabolic risk indices. A positive r-value indicates a direct relationship, while a negative r-value reflects an inverse association. Ovarian volume showed a strong correlation with insulin resistance estimated using HOMA-IR and a moderate correlation with serum triglyceride levels. HDL demonstrated an inverse relationship with fasting insulin, indicating reduced protective lipid fraction in women with higher insulin levels.

Table 4: Key Correlations Between Ovarian and Metabolic Parameters

Correlation Pair	r-value	p-value
Ovarian volume vs HOMA-IR	0.41	<0.001
Ovarian volume vs Triglycerides (TG)	0.32	0.002
HDL vs Fasting insulin	-0.38	<0.001

DISCUSSION

This study demonstrated that ultrasonographic and metabolic abnormalities are highly prevalent at baseline in women diagnosed with PCOS. The observed prevalence of PCOM in 71.7% of participants aligns closely with findings from large imaging-based cohorts, reinforcing the notion that ovarian enlargement and antral follicle excess are reliable morphological expressions of disordered follicular dynamics in PCOS. [3,4,16] The increase in ovarian volume reflects underlying stromal expansion and follicular arrest, both of which arise from impaired follicular selection and maturation. These changes may also indicate a persistent intra-ovarian imbalance in oxidative and hormonal factors, which disrupts follicular progression beyond the antral stage and contributes to the sonographic signature commonly recognized by clinicians. While ultrasound confirms structural abnormality, its value extends further by offering a non-invasive window into the severity of ovarian dysregulation at the time of diagnosis.

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Insulin resistance was observed in 47.5% of women, supporting the concept that altered insulin sensitivity is not a secondary phenomenon but a central biological component of PCOS. Chronic hyperinsulinemia may directly influence ovarian steroidogenic pathways, promoting excessive androgen synthesis and impairing granulosa cell function, which ultimately interferes with normal follicular development. [6,7,12] The positive correlation between ovarian volume and HOMA-IR suggests that women with more pronounced morphological abnormalities may also harbor more severe metabolic impairment, highlighting the clinical relevance of simultaneous ultrasound-metabolic evaluation. [7,12] This parallel between structure and metabolism indicates that ovarian and systemic dysfunction may evolve in tandem in many patients, even before clinical metabolic syndrome becomes apparent.

Dyslipidemia was identified in 39.2% of PCOS women, predominantly characterized by elevated triglycerides, reduced HDL, and borderline-high LDL. These lipid abnormalities contribute to early endothelial stress, altered lipoprotein transport, and a pro-atherogenic internal milieu, which may accelerate long-term cardiovascular vulnerability. [8,9,18,19] Unlike isolated lipid disorders, dyslipidemia in PCOS emerges in a background of hormonal imbalance and impaired insulin signaling, making the risk more biologically layered and clinically significant. The findings emphasize that metabolic dysfunction in PCOS should not be predicted solely by BMI or physical phenotype, as even women with normal body composition may exhibit meaningful biochemical risk, supporting earlier literature indicating that metabolic impairment may exist independently of obesity status. [7,10]

These observations collectively reinforce that PCOS must be approached as a systemic endocrine-metabolic disorder rather than a diagnosis limited to gynecologic symptomatology. Early metabolic screening at the time of PCOS confirmation can assist clinicians in identifying high-risk subgroups sooner, improving patient counseling, guiding lifestyle modification, and informing therapeutic planning aimed at preventing progression to diabetes and cardiovascular disease [1,7,8,20]. This integrated baseline profiling model supports a shift from reactive management (after metabolic complications appear) to proactive risk-based care (at diagnosis), which is especially relevant in young women, were early clinical decisions influence decades of future health trajectory.

STRENGTHS

This study offers notable strengths that support its clinical applicability and diagnostic relevance. A key advantage is the clear comparative assessment of women with PCOS alongside a well-defined healthy population, enabling reliable differentiation of syndrome-related abnormalities. Ovarian ultrasound parameters and fasting metabolic markers were evaluated within the same clinical timeframe, reducing inter-test variability and supporting a more synchronized interpretation of ovarian and metabolic risk. Correlation analysis further strengthened the findings by highlighting clinically meaningful associations between structural ovarian changes and metabolic indices, reinforcing an integrated endocrine-metabolic perspective of PCOS at diagnosis.

LIMITATIONS

However, certain limitations should be considered. The single-centre recruitment design may limit generalizability to broader populations, and the absence of longitudinal follow-up prevented evaluation of long-term metabolic or cardiovascular outcomes. Future studies incorporating multi-center participation and extended monitoring may help address these gaps.

CONCLUSIONS

This study confirms that a high proportion of women diagnosed with PCOS exhibit classical ultrasonographic ovarian changes along with a considerable burden of subclinical metabolic dysfunction at baseline. The frequent presence of increased ovarian volume, follicle excess, insulin resistance, hyperinsulinemia, and atherogenic lipid trends indicates that metabolic risk in PCOS is common early in the disease course and may evolve independently of visible obesity in many patients. These findings highlight that PCOS evaluation should extend beyond reproductive symptoms and include routine metabolic profiling at diagnosis for early risk recognition. A combined ultrasound-metabolic screening approach may help clinicians identify vulnerable phenotypes earlier, support targeted lifestyle guidance, improve patient counseling, and encourage timely preventive or therapeutic strategies to reduce future

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metabolic and cardiovascular complications.

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