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CORRELATION OF LIPID ABNORMALITIES, ANDROGEN LEVELS, AND BODY MASS INDEX IN WOMEN WITH POLYCYSTIC OVARIAN SYNDROME: A CROSS-SECTIONAL EVALUATION

Dr. Madhab Chandra Mali[1], Dr. Debdutta Gautom[2], Dr Bhaskar Jyoti Dutta[3]

[1] Assistant Professor, Department of Obstetrics and Gynaecology, Nalbari Medical College & Hospital.
[2] Assistant Professor, Department of Medicine, Gauhati Medical College & Hospital.
[3] Associate Professor, Department of Pharmacology, Gauhati Medical College & Hospita.

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Corresponding Author:

Dr. Madhab Chandra Mali

Assistant Professor, Department of Obstetrics and Gynaecology, Nalbari Medical College & Hospital.

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) represents the most prevalent endocrinopathy affecting women of reproductive age, characterized by a constellation of metabolic, hormonal, and reproductive abnormalities. The syndrome exhibits significant heterogeneity in clinical presentation, with variable manifestations of hyperandrogenism, ovulatory dysfunction, and metabolic derangements across different body mass index (BMI) categories.

Objectives: This study aimed to evaluate the relationship between BMI and serum lipid profile parameters, assess the association between BMI and serum testosterone levels, and establish correlations among BMI, lipid profile components, and androgen status in women diagnosed with PCOS according to Rotterdam criteria. timeofcare

Methods: A hospital-based cross-sectional analytical study was conducted among 100 consecutive women diagnosed with PCOS in the reproductive age group (15-44 years) at a tertiary care center. Participants were stratified into lean (BMI <23 kg/m²) and obese/overweight (BMI ≥23 kg/m²) categories based on Asian-specific BMI cut-offs. Fasting venous blood samples were analyzed for lipid profile parameters (total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol) using digital photocolorimetry, and serum testosterone was quantified using enzyme-linked fluorescent assay (ELFA) technique on MINIVIDAS platform.

Results: The study cohort comprised 41 lean PCOS women (mean BMI 21.13±1.21 kg/m²) and 59 obese/overweight PCOS women (mean BMI 26.65±2.08 kg/m²), with highly significant BMI differences between groups ($p<0.01$). Obese PCOS women exhibited significantly lower HDL-cholesterol levels (52.29±10.49 mg/dL) compared to lean counterparts (57.78±10.48 mg/dL; $p<0.05$) and markedly elevated triglycerides (143.59±23.89 mg/dL versus 127.98±19.47 mg/dL; $p<0.01$). No statistically significant differences were observed in LDL-cholesterol (69.97±30.22 versus 65.78±29.35 mg/dL; $p>0.05$) or total cholesterol (151.42±26.28 versus 149.15±25.32 mg/dL; $p>0.05$) between groups. Serum testosterone levels were elevated above normal reference range (>0.9 ng/mL) in both lean (1.44±0.543 ng/mL) and obese (1.62±0.47 ng/mL) PCOS women, though the difference was not statistically significant ($p>0.05$). Correlation analysis revealed moderate positive correlation between BMI and triglycerides ($r=0.51$), negative correlation with HDL-cholesterol ($r=-0.41$), weak positive correlations with LDL-cholesterol ($r=0.24$) and total cholesterol ($r=0.19$), and negligible correlation with testosterone ($r=0.09$).

Conclusions: PCOS promotes development of an atherogenic lipid profile characterized by decreased HDL-cholesterol and elevated triglycerides, with metabolic abnormalities significantly influenced by obesity status.

Hyperandrogenism manifests independently of BMI, supporting the concept that androgen excess represents a fundamental pathophysiological feature of PCOS rather than a consequence of adiposity. These findings underscore the importance of comprehensive cardiometabolic risk assessment in PCOS women regardless of body weight, with particular attention to lipid profile monitoring in obese patients.

Keywords*: Polycystic ovary syndrome, PCOS, hyperandrogenism, dyslipidemia, body mass index, testosterone, cardiovascular risk, metabolic syndrome, lipid profile.*

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) constitutes the most common endocrine disorder affecting women during their reproductive years, with global prevalence estimates ranging from 5% to 20% depending on diagnostic criteria and population studied. Originally described by Stein and Leventhal in 1935 as an association between bilateral polycystic ovaries and clinical features including amenorrhea, oligomenorrhea, hirsutism, and obesity, PCOS is now recognized as a complex heterogeneous disorder with multifaceted metabolic, reproductive, and psychological manifestations.

The syndrome exhibits familial clustering patterns suggesting inheritance as a complex genetic trait, though specific causative genes remain incompletely characterized. Contemporary understanding recognizes PCOS not merely as a gynecologic condition but as a systemic metabolic disorder with significant long-term health implications extending beyond reproductive years.

1.1 Diagnostic Criteria Evolution

The Rotterdam ESHRE/ASRM consensus established revised diagnostic criteria in 2003, requiring presence of two of three features: (1) oligo-ovulation or anovulation, (2) clinical and/or biochemical hyperandrogenism, and (3) polycystic ovarian morphology on ultrasonography. Sonographic criteria mandate identification of 12 or more follicles measuring 2-9 mm in diameter or ovarian volume exceeding 10 mL in at least one ovary. The Androgen Excess Society (AES) proposed alternative diagnostic criteria in 2006 emphasizing hyperandrogenism as an essential feature, requiring both hyperandrogenism (clinical or biochemical) and ovarian dysfunction (oligo-anovulation and/or polycystic ovaries), with exclusion of other androgen excess disorders.

These varying diagnostic approaches have generated four distinct PCOS phenotypes with potentially different metabolic profiles and cardiovascular risk trajectories. The Rotterdam criteria's broader inclusivity has facilitated identification of milder phenotypes but also raised concerns regarding diagnostic heterogeneity and treatment standardization.

1.2 Hyperandrogenism: The Central Feature

Hyperandrogenism represents the hallmark pathophysiological feature of PCOS, resulting primarily from excessive androgen production by ovarian theca cells, with lesser contributions from adrenal sources. Testosterone, the principal ovarian androgen, serves as the usual basis for biochemical diagnosis of hyperandrogenemia, with most PCOS women demonstrating elevated circulating testosterone concentrations. Free testosterone measurement provides superior diagnostic sensitivity compared to total testosterone, as it reflects biologically active unbound hormone concentrations.

Clinical manifestations of hyperandrogenism include hirsutism, acne, and androgenic alopecia, all mediated through androgen effects on pilosebaceous units. However, the correlation between clinical features and biochemical hyperandrogenemia remains relatively poor due to significant inter-individual variation in pilosebaceous unit sensitivity to androgens. The relationship between androgens and insulin resistance appears bidirectional, with hyperinsulinemia promoting ovarian androgen production while androgens impair insulin signaling, creating a self-perpetuating cycle.

1.3 Obesity and Body Mass Index in PCOS

Obesity affects more than 50% of PCOS patients, with characteristic central distribution pattern showing greater accumulation of visceral compared to subcutaneous adipose tissue. This android fat distribution pattern contributes significantly to metabolic dysfunction and insulin resistance characteristic of the syndrome. Geographic location and ethnicity substantially influence adiposity patterns in PCOS populations, with considerable variability observed across different regions.

The relationship between BMI and biochemical hyperandrogenism demonstrates complexity, with studies showing strong correlations between BMI and free androgen index (FAI) mediated through sex hormone-binding globulin (SHBG) suppression, though direct causal links remain incompletely established. Hyperinsulinemia associated with obesity suppresses hepatic SHBG production, increasing free testosterone availability despite potentially unchanged total testosterone concentrations.

1.4 Dyslipidemia and Cardiovascular Risk

Dyslipidemia represents perhaps the most prevalent metabolic abnormality observed in PCOS patients, with approximately 70% demonstrating at least one borderline or elevated lipid parameter according to National Cholesterol Education Program (NCEP) guidelines, though many women with PCOS maintain entirely normal lipid profiles. The characteristic dyslipidemic pattern includes elevated triglycerides, decreased HDL-cholesterol, and variably elevated LDL-cholesterol and total cholesterol, creating an atherogenic lipid profile that substantially increases cardiovascular disease risk.

Recent meta-analyses confirm that PCOS women demonstrate significantly elevated risks for cardiovascular events including myocardial infarction (hazard ratio 1.79), stroke (hazard ratio 1.66), coronary artery disease (hazard ratio 2.92), and arrhythmias (hazard ratio 1.37) compared to age-matched controls. These cardiovascular risks manifest even in young women aged 30-40 years, with 19% higher risk of developing cardiovascular disease attributable to clustering of obesity, hypertension, and type 2 diabetes.

Insulin resistance and compensatory hyperinsulinemia contribute significantly to dyslipidemia through multiple mechanisms, including decreased HDL-cholesterol and elevated triglyceride concentrations. Studies demonstrate that insulin resistance associates with dyslipidemia in PCOS women independent of obesity status, though obesity significantly amplifies metabolic derangements.

1.5 Rationale and Objectives

Despite extensive research characterizing metabolic abnormalities in PCOS, the relationships among BMI, lipid profile components, and androgen status require further elucidation, particularly in diverse geographic and ethnic populations. The present study was designed to comprehensively evaluate these relationships in a Northeast Indian population attending a tertiary care hospital, with the following specific

1. To assess the relationship between body mass index and serum lipid profile parameters in women with PCOS
2. To evaluate the association between body mass index and serum testosterone levels in PCOS women
3. To establish correlations among body mass index, serum lipid profile components, and serum testosterone in women diagnosed with PCOS

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This investigation employed a hospital-based cross-sectional analytical study design conducted at a tertiary care medical center in Northeast India. The study protocol received approval from the Institutional Ethical Committee, and written informed consent was obtained from all participants prior to enrollment.

2.2 Study Population and Sample Size

The study population comprised 100 consecutive women diagnosed with polycystic ovarian syndrome in the reproductive age group (15-44 years) attending the Obstetrics and Gynaecology outpatient department. Sample size calculation employed the formula:

$$n = \frac{\left\{Z_{(1-\alpha/2)} \times Z_{(1-\beta)} \times \sigma^2\right\}}{\Delta^2}$$

where $Z_{(1-\alpha/2)}$represents the confidence interval at 95%, $Z_{(1-\beta)}$denotes the power of test at 80%, σ^2indicates variance (standard deviation squared), and Δ^2represents the square of mean difference.

2.3 Diagnostic Criteria

PCOS diagnosis was established according to the Rotterdam ESHRE/ASRM revised 2003 criteria, requiring presence of at least two of three features: (1) oligo-ovulation or anovulation, (2) clinical and/or biochemical hyperandrogenism, and (3) polycystic ovarian morphology on ultrasonography.

2.4 Inclusion Criteria

- Women aged 15-44 years diagnosed with PCOS fulfilling Rotterdam criteria
- No history of medications affecting hormonal or lipid metabolism
- Willingness to participate with informed consent provided

2.5 Exclusion Criteria

- Diabetes mellitus, hypertension, chronic kidney disease, or hepatic failure
- Other endocrine disorders altering lipid profile or testosterone levels (Cushing's syndrome, testosterone-secreting tumors, thyroid dysfunction)
- Other androgen excess disorders (congenital adrenal hyperplasia, androgen-secreting tumors)
- Current use of hormonal or non-hormonal treatments for PCOS or other conditions

2.6 Anthropometric Measurements

Height Measurement: Height was measured using a portable vertical stadiometer marked with metric measurement scale, with participants standing barefoot in erect posture.

Weight Measurement: Body weight was measured using calibrated Libra flat model 770 weighing machine (machine number 53084), with participants wearing light clothing and no footwear.

Body Mass Index Calculation: BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Based on World Health Organization recommendations for Asian populations, participants were categorized into two groups: (A) Lean PCOS with BMI <23 kg/m^2, and (B) Obese/Overweight PCOS with BMI ≥23 kg/m^2.

2.7 Sample Collection and Processing

Under aseptic precautions, 3 mL of venous blood was collected from each participant following 12 hours of overnight fasting. Blood samples were transferred to sterile vials and allowed to clot at room temperature for 30-45 minutes. Following clot formation, samples were centrifuged at 3000 rpm for 5 minutes, and supernatant serum was collected in sterile vials using sterile pipettes. When immediate analysis was not feasible, serum was stored at 2-8°C for maximum duration of five days.

2.8 Biochemical Analyses

Lipid Profile Estimation: Biochemical estimation of lipid profile parameters (total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol) was performed using digital photocolorimeter with standard enzymatic colorimetric methods.

Serum Testosterone Measurement: Testosterone quantification was performed using MINIVIDAS automated immunoassay analyzer employing Enzyme-Linked Fluorescent Assay (ELFA) technique, which combines enzyme immunoassay competition methodology with fluorescent detection.

2.9 Reference Ranges and Cut-off Values

Dyslipidemia Criteria: Lipid abnormalities were defined according to NCEP-ATP III guidelines as:

- Total cholesterol ≥200 mg/dL
- Triglycerides ≥150 mg/dL
- HDL-cholesterol <50 mg/dL
- LDL-cholesterol ≥130 mg/dL

Testosterone Reference Range: Normal range for reproductive-aged women: 0.1-0.9 ng/mL.

2.10 Statistical Analysis

Data were entered into Microsoft Excel and analyzed using GraphPad InStat software. Continuous variables are presented as mean ± standard deviation. Unpaired Student's t-test was employed to compare parameters between lean and obese PCOS groups, with statistical significance established at $p<0.05$ and $p<0.01$. Pearson's correlation coefficient (r) was calculated to assess relationships between BMI and all biochemical parameters across the entire study population, with correlation coefficient range of -1 to +1. Scatter diagrams were constructed to visualize correlations between variables.

3. RESULTS

3.1 Study Population Characteristics

The study cohort comprised 100 women diagnosed with PCOS, with 41 participants (41%) classified as lean PCOS (BMI <23 kg/m^2) and 59 participants (59%) categorized as obese/overweight PCOS (BMI ≥23 kg/m^2).

3.2 Body Mass Index Comparison

Mean BMI in the lean PCOS group was 21.13±1.21 kg/m^2, while the obese/overweight PCOS group demonstrated mean BMI of 26.65±2.08 kg/m^2. Unpaired Student's t-test revealed highly significant difference in BMI between groups ($t=15.296$, $df=98$, $p<0.01$), confirming appropriate group stratification.

3.3 Lipid Profile Parameters

HDL-Cholesterol: Lean PCOS women exhibited mean HDL-cholesterol of 57.78±10.48 mg/dL, while obese PCOS women demonstrated significantly lower levels at 52.29±10.49 mg/dL ($t=2.576$, $df=98$, $p<0.05$). The obese PCOS category demonstrated statistically significant reduction in this cardioprotective lipoprotein fraction compared to lean counterparts.
Triglycerides: Highly significant difference in triglyceride levels was observed between groups, with lean PCOS women showing mean triglycerides of 127.98±19.47 mg/dL compared to 143.59±23.89 mg/dL in obese PCOS women ($t=3.461$,

df=98, p<0.01). This represents an approximately 12% elevation in triglyceride concentrations associated with obesity in PCOS.

LDL-Cholesterol: Mean LDL-cholesterol was 65.78±29.35 mg/dL in lean PCOS women and 69.97±30.22 mg/dL in obese PCOS women, with no statistically significant difference between groups (t=0.689, df=98, p>0.05).

Total Cholesterol: Total cholesterol levels showed minimal difference between lean (149.15±25.32 mg/dL) and obese (151.42±26.28 mg/dL) PCOS groups, without achieving statistical significance (t=0.433, df=98, p>0.05).

3.4 Serum Testosterone Levels

Both lean and obese PCOS groups demonstrated elevated serum testosterone concentrations exceeding the normal reference range upper limit of 0.9 ng/mL. Lean PCOS women exhibited mean testosterone of 1.44±0.543 ng/mL, while obese PCOS women showed mean testosterone of 1.62±0.47 ng/mL. Although obese PCOS women demonstrated approximately 12.5% higher mean testosterone compared to lean counterparts, this difference did not achieve statistical significance (t=1.79, df=98, p>0.05).

3.5 Correlation Analyses

BMI and HDL-Cholesterol: Pearson's correlation coefficient revealed negative correlation (r=-0.41) between BMI and HDL-cholesterol, indicating that increasing BMI associates with decreasing HDL-cholesterol concentrations across the study population.

BMI and Triglycerides: Moderate positive correlation (r=0.51) was observed between BMI and triglycerides, demonstrating linear relationship wherein higher BMI associates with elevated triglyceride levels.

BMI and LDL-Cholesterol: Weak positive correlation (r=0.24) was identified between BMI and LDL-cholesterol, suggesting modest tendency for LDL elevation with increasing BMI.

BMI and Total Cholesterol: Low positive correlation (r=0.19) was observed between BMI and total cholesterol, indicating minimal relationship between these parameters.

BMI and Testosterone: Negligible correlation (r=0.09) was found between BMI and serum testosterone, suggesting that androgen levels in PCOS women are largely independent of body weight.

4. DISCUSSION

4.1 Principal Findings

This cross-sectional analysis of 100 women with PCOS diagnosed according to Rotterdam criteria demonstrates that obesity significantly influences specific lipid profile parameters while hyperandrogenism manifests independent of BMI status. The study reveals a biphasic pattern of metabolic dysfunction wherein certain lipid abnormalities (HDL-cholesterol reduction and triglyceride elevation) show clear BMI dependency, while others (LDL-cholesterol, total cholesterol, and testosterone elevation) occur irrespective of obesity status.

4.2 Dyslipidemia and Body Mass Index

The significant reduction in HDL-cholesterol and elevation in triglycerides observed in obese compared to lean PCOS women aligns with established understanding of obesity-related metabolic dysfunction. HDL-cholesterol serves critical cardioprotective functions through reverse cholesterol transport, anti-inflammatory effects, and endothelial protection, making its reduction particularly concerning for long-term cardiovascular health.

These findings corroborate previous research by Saxena et al., who reported lipid profile derangements in 14.2% of lean versus 31% of obese PCOS women, with significantly decreased HDL-cholesterol and increased triglycerides in the obese group. Similarly, studies by Moini et al. demonstrated high prevalence of metabolic syndrome components in PCOS women, particularly decreased HDL-cholesterol levels, associated with insulin resistance and hyperinsulinemia.

The absence of significant differences in LDL-cholesterol and total cholesterol between lean and obese PCOS groups contrasts with some previous studies but aligns with research by Fulghesu et al., who found no differences in lipid profile between adolescent PCOS patients and controls. This variability may reflect population-specific characteristics, age distribution differences, or varying PCOS phenotypes within study cohorts.

4.3 Insulin Resistance as Unifying Mechanism

Insulin resistance represents the crucial pathophysiological defect accounting for most endocrine and metabolic abnormalities observed in PCOS. Within this framework, insulin resistance produces abnormal follicular responses to FSH, leading to anovulation and excessive androgen secretion. Enhanced androgen production results in non-cyclic peripheral

aromatization to estrogens, which combined with hyperinsulinemia causes aberrant gonadotropin release patterns favoring continuous LH secretion.

In adipose tissue, insulin resistance promotes lipolysis of stored triglycerides, elevating circulating free fatty acid concentrations. These free fatty acids impair hepatic insulin signaling, suppress SHBG production, and contribute to hepatic VLDL overproduction, creating the characteristic dyslipidemic pattern. Studies by Kalra et al. confirmed that insulin resistance associates with dyslipidemia in PCOS women independent of obesity, though obesity substantially amplifies metabolic disturbances.

4.4 Hyperandrogenism Independent of BMI

The observation that serum testosterone levels were elevated above normal reference range in both lean and obese PCOS groups, without statistically significant difference between groups, supports the hypothesis that hyperandrogenism represents a fundamental pathophysiological feature of PCOS rather than a consequence of obesity. This finding carries important clinical implications, indicating that androgen excess requires attention regardless of patient body weight.

The negligible correlation ($r=0.09$) between BMI and testosterone across the entire study population further substantiates this independence. However, previous research by Barth et al. demonstrated strong correlation between BMI and free androgen index (FAI) mediated through SHBG suppression, though not with total testosterone. This apparent discrepancy likely reflects the differential effects of obesity on total versus free testosterone concentrations, with hyperinsulinemia suppressing SHBG production and thereby increasing bioavailable free testosterone despite stable total testosterone levels.

Recent studies confirm that free testosterone and SHBG play independent roles in insulin resistance, with low SHBG and high free testosterone independently associating with insulin resistance, and coexistence of these features enhancing metabolic abnormalities. Total testosterone significantly correlates with insulin resistance in PCOS patients and can serve as a predictor of insulin resistance, acceptably discriminating HOMA-IR values.

4.5 Cardiovascular Risk Implications

The atherogenic lipid profile observed in this study population, particularly in obese PCOS women, carries significant implications for long-term cardiovascular health. Recent meta-analyses confirm that PCOS women demonstrate substantially elevated cardiovascular disease risks, with hazard ratios of 2.33 when diagnosed by Rotterdam criteria and 2.47 when diagnosed by NIH criteria.

Importantly, cardiovascular risks diverge markedly starting at age 35, with women aged 30-40 years showing 19% higher risk of cardiovascular disease development. These risks manifest even in absence of obesity or dyslipidemia, with young PCOS women exhibiting premature endothelial dysfunction and carotid intima-media thickening characteristic of early atherosclerosis.

The constellation of dyslipidemia, hyperandrogenism, insulin resistance, and obesity observed in PCOS women comprises the metabolic syndrome or "Syndrome X" originally described by Reaven, which substantially amplifies cardiovascular disease risk through multiple synergistic mechanisms. Obesity has been identified as a key predictor of metabolic syndrome development in PCOS participants, warranting aggressive lifestyle intervention and pharmacological management when appropriate.

4.6 Clinical Implications

These findings underscore the necessity for comprehensive cardiometabolic risk assessment in all women diagnosed with PCOS, regardless of body weight. Particular emphasis should be placed on lipid profile monitoring in obese PCOS patients, given their significantly increased triglyceride levels and decreased HDL-cholesterol concentrations.

The presence of hyperandrogenism across BMI categories indicates that androgen excess evaluation and management should constitute standard care for all PCOS women. Weight reduction through lifestyle modification remains a cornerstone therapeutic intervention for obese PCOS patients, with documented benefits for menstrual regularity, ovulatory function, insulin sensitivity, and lipid profile improvement.

Given the systemic nature of metabolic disturbances in PCOS, management approaches must extend beyond gynecologic concerns to encompass comprehensive cardiometabolic risk reduction strategies. This requires multidisciplinary collaboration among gynecologists, endocrinologists, cardiologists, nutritionists, and mental health professionals to address the full spectrum of PCOS-related health impacts.

4.7 Limitations

Several limitations warrant acknowledgment. The cross-sectional study design precludes establishment of causal relationships between variables. The sample size of 100 participants, while adequate for initial evaluation, limits

generalizability of findings to broader populations. The study did not include measurement of insulin levels or insulin resistance indices (HOMA-IR, QUICKI), which would have provided valuable mechanistic insights into relationships among hyperandrogenism, obesity, and dyslipidemia.

Additional unmeasured variables including physical activity levels, dietary patterns, parity, and family history of metabolic disorders may influence observed relationships. The study population derived from a single tertiary care center in Northeast India, potentially limiting applicability to other geographic regions and ethnic groups given known population-specific variations in PCOS manifestations.

Future research should incorporate longitudinal designs with larger representative samples, comprehensive assessment of insulin resistance parameters, evaluation of free testosterone and SHBG concentrations, characterization of PCOS phenotypes, and investigation of genetic, lifestyle, and environmental factors influencing metabolic and hormonal profiles in PCOS.

CONCLUSION

This cross-sectional evaluation demonstrates that polycystic ovarian syndrome promotes development of an atherogenic lipid profile characterized by reduced HDL-cholesterol and elevated triglycerides, with obesity significantly amplifying these metabolic derangements. The findings confirm that hyperandrogenism manifests as a fundamental feature of PCOS independent of body mass index, supporting the concept that androgen excess requires clinical attention regardless of patient adiposity status.

The observed metabolic abnormalities place PCOS women at substantially elevated risk for cardiovascular disease and metabolic syndrome, necessitating comprehensive cardiometabolic risk assessment and proactive management strategies. Particular emphasis should be directed toward aggressive lifestyle intervention and pharmacological therapy when indicated for obese PCOS patients, given their markedly adverse lipid profiles.

Recognition of PCOS as a systemic metabolic disorder rather than solely a gynecologic condition mandates integrated multidisciplinary care approaches addressing reproductive, metabolic, cardiovascular, and psychological health dimensions. Early identification and intervention may mitigate long-term cardiovascular morbidity and mortality in this high-risk population.

Further research with larger sample sizes, longitudinal designs, comprehensive insulin resistance assessment, and phenotype-specific analyses is warranted to fully elucidate the complex relationships among obesity, hyperandrogenism, insulin resistance, and dyslipidemia in PCOS, thereby optimizing individualized therapeutic strategies.

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