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Original Research Article

A Retrospective Comparative Study of Metabolic Parameters among Different Phenotypes of Polycystic Ovary Syndrome

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Abstract:

Background: Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder with variable metabolic risk across its phenotypes. Hyperandrogenic phenotypes are believed to carry greater cardiometabolic burden.

Aim: To evaluate and compare metabolic and hormonal profiles among different PCOS phenotypes based on the Rotterdam criteria.

Methodology: This retrospective comparative study was conducted using hospital medical records of 90 women aged 18–40 years diagnosed with PCOS. Patients were classified into four phenotypes (A, B, C, and D) according to the Rotterdam criteria. Data on anthropometric measurements, blood pressure, hormonal parameters, glucose metabolism indices, lipid profile, and insulin resistance assessed by HOMA-IR were extracted and analyzed using appropriate statistical methods.

Results: Phenotype A was most prevalent (35.6%). BMI, waist circumference, and blood pressure were significantly higher in Phenotypes A and B ($p<0.01$). Phenotype A showed the highest LH/FSH ratio, testosterone, fasting glucose, insulin, HOMA-IR, and adverse lipid parameters ($p<0.01$). Insulin resistance was most common in Phenotype A (62.5%), followed by B (50%), C (33.3%), and D (25%). Metabolic syndrome prevalence was highest in Phenotype A (37.5%) and lowest in Phenotype D (10%).

Conclusion: Hyperandrogenic phenotypes, particularly Phenotype A, exhibit greater metabolic dysfunction. Phenotype-based risk stratification is essential for early identification and prevention of long-term cardiometabolic complications.

Keywords: Polycystic Ovary Syndrome, PCOS Phenotypes, Insulin Resistance, Metabolic Syndrome, Hyperandrogenism, Lipid Profile.

Introduction

Polycystic ovary syndrome (PCOS) belongs to the number of the most common endocrine conditions in women of the reproductive age [1]. It is typified by menstrual irregularities, hyperandrogenism and polycystic ovarian morphology. PCS diagnosis heavily relies on the Rotterdam criterion suggested by the European Society of Human Reproduction and Embryology (ESHRE) and the American Society of Reproductive Medicine (ASRM) that include any two out of the following three characteristics: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovaries on ultrasound, following the elimination of other related conditions [2]. According to these criteria, PCOS is divided into four phenotypes (Phenotype A: hyperandrogenism + ovulatory dysfunction + polycystic ovaries), Phenotype B (hyperandrogenism + ovulatory dysfunction), Phenotype C (hyperandrogenism + polycystic ovaries) and Phenotype D (ovulatory dysfunction + polycystic ovaries). This classification shows the heterogeneity of PCOS and indicates that clinical and metabolic phenotypes may vary across phenotypes.

As much as PCOS was originally viewed as a reproductive disorder, the disorder nowadays is considered a serious metabolic disorder [3]. Insulin resistance is considered to be a primary pathogenesis event in PCOS. This elevated insulin concentration stimulates ovarian androgen synthesis and

suppresses sex hormone-binding globulin synthesis causing high circulating androgens. This is a hormonal disorder that leads to anovulation and clinical effects including hirsutism and acne. Nevertheless, insulin resistance and metabolic disturbance differ in the phenotypes of type 2 diabetes, which is why phenotype-based comparison is significant.

It is proven by several researches that both classical phenotypes, especially Phenotype A and Phenotype B are connected to more severe metabolic abnormalities [4]. These groups of women usually have elevated body mass index (BMI), excessive waist circumference, insulin resistance, and undesirable lipid profiles. High levels of fasting glucose, triglycerides and low levels of high-density lipoprotein cholesterol are also characteristic of hyperandrogenic phenotypes [5]. Such malformations predispose people to such diseases as metabolic syndrome, type 2 diabetes mellitus and cardiovascular diseases. Phenotype D (without hyperandrogenism) on the other hand typically exhibits relatively milder metabolic alterations, though there can be low-profile insulin resistance. Phenotype C can exhibit intermediate metabolism risk because of the existence of hyperandrogenism even though ovulation was preserved [6].

Obesity also aggravates the metabolic profile of all PCOS phenotypes; however, intrinsic insulin resistance can also develop in lean women with PCOS suggesting that body weight is not the sole determinant [7]. Thus, metabolic parameters including the level of fasting blood glucose, insulin, lipid profile, and blood pressure should be evaluated in all women with PCOS, irrespective of their phenotype and BMI. Another factor that affects the metabolic presentation of PCOS is ethnic and regional differences. As an illustration, South Asian women are more likely to develop insulin resistance and central obesity at lower BMI, and this could pose a metabolic risk. This underlines the necessity of individualized assessment and care depending on phenotype and population appearance.

PCOS is a heterogenous disorder with four different phenotypes which are not only different in their reproductive features, but also metabolic risk. There is a general insulin resistance and poor metabolic profile of hyperandrogenic phenotypes than non-hyperandrogenic phenotypes. The knowledge of these differences will be essential to detect the high-risk individuals early and design the right lifestyle and therapeutic interventions in order to minimize the metabolic complications in the long-term.

Methodology

Study Design: This hospital-based retrospective comparative study was conducted to evaluate and compare metabolic profiles among different phenotypes of polycystic ovary syndrome (PCOS) as defined by the Rotterdam criteria. The study aimed to identify variations in metabolic abnormalities across the four recognized phenotypes of PCOS.

Study Area: The study was carried out in the Department of Obstetrics and Gynaecology, Nalanda Medical College and Hospital, Patna, Bihar, India.

Study Duration: The study was conducted over a period of one year. December 2023 to November 2024.

Study Participants: A total of 90 women diagnosed with PCOS were enrolled and categorized into different phenotypes based on the Rotterdam diagnostic criteria.

Inclusion Criteria

- Women aged 18–40 years.
- Women diagnosed with PCOS according to the Rotterdam criteria (presence of any two of the following three features):
    1. Chronic ovulatory dysfunction (oligo-/anovulation).
    2. Clinical and/or biochemical hyperandrogenism.
    3. Polycystic ovarian morphology (PCOM) on ultrasonography (≥12 follicles measuring 2–9 mm in either ovary and/or ovarian volume ≥10 mL).
- Willingness to participate and provide written informed consent.

Exclusion Criteria

- Women with hypothalamic amenorrhea.
- Hyperprolactinemia.
- Thyroid dysfunction (hypo- or hyperthyroidism).
- Congenital adrenal hyperplasia.
- Premature ovarian insufficiency or premature ovarian failure.
- Cushing's syndrome or androgen-secreting tumors.
- Known type 1 or type 2 diabetes mellitus diagnosed prior to study enrollment.
- Use of hormonal medications (oral contraceptive pills, anti-androgens, glucocorticoids) in the last 6 months.
- Use of drugs affecting metabolic parameters (metformin, insulin, statins) within the past 6 months.
- Pregnant or lactating women.

Sample Size: The total sample size was 90 participants. The sample size was determined based on feasibility within the study duration and expected prevalence of metabolic abnormalities among women with PCOS attending the outpatient department.

Procedure: Eligible women attending the gynecology outpatient department were screened for PCOS using detailed history, clinical examination,

biochemical evaluation, and pelvic ultrasonography. After obtaining written informed consent, demographic details including age, menstrual history, and family history were recorded. Anthropometric measurements such as height, weight, waist circumference, and hip circumference were measured using standardized techniques, and body mass index (BMI) was calculated.

Clinical hyperandrogenism was assessed using the modified Ferriman–Gallwey (mFG) score, with a score >3 considered significant. Blood pressure was recorded using a calibrated sphygmomanometer.

Biochemical investigations were performed in the early follicular phase (day 2–4 of spontaneous or progesterone-induced menstruation). Fasting venous blood samples were collected after 8–12 hours of overnight fasting to measure fasting blood glucose, fasting insulin, lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C), serum testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), and anti-Müllerian hormone (AMH). An oral glucose tolerance test (OGTT) was conducted by administering 75 g of oral glucose, and blood glucose levels were measured after 2 hours.

Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) calculated as:

HOMA-IR = Fasting glucose (mmol/L) × Fasting insulin (μIU/mL) / 22.5.

A HOMA-IR value >3.0 was considered indicative of insulin resistance.

Participants were categorized into four PCOS phenotypes:

- Phenotype A (OD + HA + PCOM)
- Phenotype B (OD + HA)
- Phenotype C (HA + PCOM)
- Phenotype D (OD + PCOM)

Metabolic abnormalities including impaired glucose tolerance, insulin resistance, dyslipidemia, and metabolic syndrome were assessed and compared among phenotypes.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 27.0. Continuous variables were tested for normality using the Kolmogorov–Smirnov test. Normally distributed data were expressed as mean ± standard deviation and compared using Student's t-test or one-way ANOVA as appropriate. Non-normally distributed data were analyzed using the Mann–Whitney U test or Kruskal–Wallis test. Categorical variables were expressed as frequency and percentage and compared using the chi-square test or Fisher's exact test. Multivariate logistic regression analysis was performed to determine the association between PCOS phenotypes and metabolic abnormalities after adjusting for confounding variables such as age and BMI. A p-value <0.05 was considered statistically significant.

Result

Table 1 shows the distribution of different PCOS phenotypes among the 90 study participants. The most common phenotype was Phenotype A (OD + HA + PCOM), observed in 32 patients (35.6%), indicating that over one-third of the participants presented with the complete classical triad of ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Phenotypes B (OD + HA) and D (OD + PCOM) were equally distributed, each accounting for 20 cases (22.2%), suggesting a comparable prevalence of these two partial phenotypic expressions. Phenotype C (HA + PCOM) was the least common, identified in 18 participants (20.0%). Overall, the findings demonstrate that the full-blown phenotype (A) predominates in the study population, while the other three phenotypes show relatively similar and moderate distribution patterns.

Table 1: Distribution of PCOS Phenotypes (n = 90)

PCOS Phenotype	Criteria	Frequency (n)	Percentage (%)
Phenotype A	OD + HA + PCOM	32	35.60%
Phenotype B	OD + HA	20	22.20%
Phenotype C	HA + PCOM	18	20.00%
Phenotype D	OD + PCOM	20	22.20%
Total		**90**	**100%**

Table 2 demonstrates that while the mean age was comparable across all PCOS phenotypes ($p = 0.48$), indicating no significant age difference, marked variations were observed in anthropometric and blood pressure parameters. Phenotype A exhibited the highest mean BMI (28.6 ± 3.5 kg/m²), followed by Phenotype B (27.8 ± 3.1 kg/m²), whereas Phenotypes C and D had comparatively lower BMI values (25.9 ± 2.8 kg/m² and 24.8 ± 2.6 kg/m², respectively), with the difference being statistically significant ($p < 0.01$). A similar trend was noted for waist circumference, where Phenotype A showed the greatest central adiposity (90.4 ± 6.2 cm), and Phenotype D the lowest (82.6 ± 5.1 cm) ($p < 0.01$). Additionally, both systolic and diastolic blood pressure were significantly higher in Phenotypes A and B compared to C and D ($p < 0.01$). Overall, the findings suggest that Phenotypes A and B are associated with a more adverse metabolic and cardiovascular risk profile compared to Phenotypes C and D.

Table 2: Baseline Demographic and Anthropometric Characteristics According to PCOS Phenotypes					
Parameter	**Phenotype A (n=32)**	**Phenotype B (n=20)**	**Phenotype C (n=18)**	**Phenotype D (n=20)**	**p-value**
Age (years)	25.8 ± 4.2	26.5 ± 3.8	24.9 ± 3.9	25.2 ± 4.1	0.48
BMI (kg/m²)	28.6 ± 3.5	27.8 ± 3.1	25.9 ± 2.8	24.8 ± 2.6	<0.01
Waist Circumference (cm)	90.4 ± 6.2	88.2 ± 5.8	83.5 ± 4.9	82.6 ± 5.1	<0.01
Systolic BP (mmHg)	128.4 ± 10.5	125.3 ± 9.2	118.6 ± 8.4	116.8 ± 7.9	<0.01
Diastolic BP (mmHg)	82.6 ± 6.8	80.4 ± 5.9	75.8 ± 5.1	74.9 ± 4.7	<0.01

Table 3 demonstrates significant variations in hormonal profiles among different PCOS phenotypes. Luteinizing hormone (LH) levels were highest in Phenotype A (11.8 ± 3.4 IU/L) and progressively decreased across Phenotypes B, C, and D, with the lowest levels observed in Phenotype D (8.8 ± 2.3 IU/L), showing a statistically significant difference ($p < 0.01$). In contrast, follicle-stimulating hormone (FSH) levels were relatively similar across all phenotypes, with no statistically significant difference ($p = 0.41$). Consequently, the LH/FSH ratio was significantly elevated in Phenotype A (1.93 ± 0.6) and gradually declined through Phenotypes B, C, and D ($p < 0.01$). Total testosterone levels were also significantly higher in Phenotypes A, B, and C compared to Phenotype D, with the lowest mean value recorded in Phenotype D (1.3 ± 0.3 nmol/L) ($p < 0.01$). Additionally, anti-Müllerian hormone (AMH) levels were highest in Phenotype A and C and lowest in Phenotype D, showing a significant difference among groups ($p = 0.02$). Overall, Phenotype A exhibited the most pronounced hormonal imbalance.

Table 3: Hormonal Profile Comparison Among PCOS Phenotypes					
Parameter	**Phenotype A**	**Phenotype B**	**Phenotype C**	**Phenotype D**	**p-value**
LH (IU/L)	11.8 ± 3.4	10.6 ± 2.9	9.2 ± 2.6	8.8 ± 2.3	<0.01
FSH (IU/L)	6.1 ± 1.2	6.3 ± 1.4	6.5 ± 1.3	6.7 ± 1.1	0.41
LH/FSH Ratio	1.93 ± 0.6	1.68 ± 0.5	1.41 ± 0.4	1.31 ± 0.3	<0.01
Total Testosterone (nmol/L)	2.4 ± 0.6	2.2 ± 0.5	2.1 ± 0.4	1.3 ± 0.3	<0.01
AMH (ng/mL)	6.8 ± 1.5	5.9 ± 1.3	6.5 ± 1.4	5.4 ± 1.1	0.02

Table 4 demonstrates significant differences in glucose metabolism and insulin resistance across the four PCOS phenotypes. Phenotype A exhibited the highest mean fasting glucose levels (102.4 ± 12.5 mg/dL), followed by Phenotype B (98.6 ± 11.8 mg/dL), while Phenotypes C and D showed comparatively lower values (92.5 ± 9.6 and 90.4 ± 8.7 mg/dL, respectively), with the difference being statistically significant ($p < 0.01$). A similar trend was observed for 2-hour OGTT values, fasting insulin levels, and HOMA-IR scores, all of which were highest in Phenotype A and progressively decreased from Phenotype B to D ($p < 0.01$). Notably, the prevalence of insulin resistance was greatest in Phenotype A (62.5%), followed by Phenotype B (50%), Phenotype C (33.3%), and lowest in Phenotype D (25%), with a significant association ($p = 0.01$). Overall, the findings indicate that Phenotype A demonstrates the most pronounced metabolic dysfunction and insulin resistance among the PCOS phenotypes.

Table 4: Glucose Metabolism and Insulin Resistance Among PCOS Phenotypes					
Parameter	**Phenotype A**	**Phenotype B**	**Phenotype C**	**Phenotype D**	**p-value**
Fasting Glucose (mg/dL)	102.4 ± 12.5	98.6 ± 11.8	92.5 ± 9.6	90.4 ± 8.7	<0.01
2-hour OGTT (mg/dL)	148.2 ± 20.4	138.5 ± 18.6	122.6 ± 15.2	118.4 ± 14.8	<0.01
Fasting Insulin (μIU/mL)	16.8 ± 4.5	14.2 ± 3.8	11.6 ± 3.2	10.8 ± 2.9	<0.01
HOMA-IR	4.2 ± 1.3	3.5 ± 1.1	2.6 ± 0.9	2.4 ± 0.8	<0.01
Insulin Resistance (%)	20 (62.5%)	10 (50%)	6 (33.3%)	5 (25%)	0.01

Table 5 demonstrates a significant variation in lipid profile parameters and metabolic abnormalities among the different PCOS phenotypes. Phenotype A exhibited the most adverse metabolic profile, with the highest mean total cholesterol (202.5 ± 28.4 mg/dL), triglycerides (168.4 ± 32.5 mg/dL), and LDL-C levels (132.8 ± 21.6 mg/dL), along with the lowest HDL-C level (39.6 ± 6.4 mg/dL). In contrast, Phenotype D showed the most favorable lipid profile, characterized by the lowest total cholesterol (172.6 ± 19.4 mg/dL), triglycerides (126.5 ± 20.8 mg/dL), LDL-C (105.4 ± 16.2 mg/dL), and the highest HDL-C levels (47.4 ± 6.5 mg/dL). Phenotypes B and C demonstrated intermediate values. The prevalence of metabolic syndrome was also highest in Phenotype A (37.5%) and lowest in Phenotype D (10%), with statistically significant differences across groups ($p = 0.02$). Overall, the findings

indicate that Phenotype A is associated with greater metabolic derangement compared to other phenotypes.

Table 5: Lipid Profile and Metabolic Abnormalities Among PCOS Phenotypes

Parameter	Phenotype A	Phenotype B	Phenotype C	Phenotype D	p-value
Total Cholesterol (mg/dL)	202.5 ± 28.4	194.6 ± 24.8	178.3 ± 21.5	172.6 ± 19.4	<0.01
Triglycerides (mg/dL)	168.4 ± 32.5	154.8 ± 29.6	132.6 ± 22.4	126.5 ± 20.8	<0.01
LDL-C (mg/dL)	132.8 ± 21.6	124.2 ± 19.8	110.6 ± 17.5	105.4 ± 16.2	<0.01
HDL-C (mg/dL)	39.6 ± 6.4	41.2 ± 5.8	45.8 ± 6.2	47.4 ± 6.5	<0.01
Metabolic Syndrome (%)	12 (37.5%)	6 (30%)	3 (16.7%)	2 (10%)	0.02

Discussion

The research demonstrates that PCOS exhibits multiple phenotypes which display distinct clinical and hormonal and metabolic characteristics. The study found that Phenotype A (OD + HA + PCOM) was the most common phenotype in our group with 35.6% of participants showing this phenotype followed by Phenotypes B and D and Phenotype C which occurred less frequently. The distribution of this study matches the results from Bozdag et al. (2016) [8] meta-analysis which found that classical phenotype constitutes around 30 to 40 percent of worldwide PCOS cases. The study by Lizneva et al. (2016) [9] showed that different ethnic groups and regions display distinct patterns of phenotype distribution which supports our finding that PCOS presents multiple clinical expressions instead of one standard clinical presentation.

Our results showed that hyperandrogenic phenotypes (A and B) had higher BMI and waist circumference measurements than the non-hyperandrogenic individuals who exhibited Phenotype D. This is consistent with the findings of Barber et al. (2007) [10], who reported that normoandrogenemic women with PCOS had metabolic profiles that matched those of controls and showed less central adiposity than hyperandrogenic women. Our research showed that both systolic and diastolic blood pressure levels were elevated in both Phenotypes A and B. The research from Moran et al. (2010) [11] showed a 2 to 3 times higher rate of impaired glucose tolerance and metabolic syndrome among women with PCOS compared to control groups with higher rates in obese hyperandrogenic women, which matched our findings.

Insulin resistance (IR) showed its highest level in Phenotype A which had 62.5% of cases while Phenotype D showed the least level of IR with 25% of cases. The results of this study match the findings of Goverde et al. (2009) [12] which showed that hyperandrogenic phenotypes had HOMA-IR scores that were significantly higher than those of non-hyperandrogenic groups. The research conducted by Zeng et al. (2020) [13] established a strong connection between serum testosterone levels and obesity and insulin resistance among women suffering from PCOS. Our data showed that Phenotypes A and B exhibited increased fasting insulin and HOMA-IR results which demonstrated that hyperandrogenism functions as the main cause of metabolic dysfunction.

The study results showed the highest metabolic syndrome prevalence in Phenotype A which matched the findings of Wu et al. (2015) [14] who found that 31.9% of Chinese women with PCOS experienced metabolic syndrome and they showed higher rates of the condition in hyperandrogenic phenotypes. Moran et al. (2010) estimated that metabolic syndrome appeared in PCOS populations with a range of 33% to 47% which matched the rates we observed in our hyperandrogenic groups. The normoandrogenic phenotypes showed lower metabolic abnormality rates which confirmed the results of Rimmer et al. (2020) [15] who found that hyperandrogenic PCOS patients showed more severe metabolic inflexibility.

The study observed lipid abnormalities which developed through a gradient that showed Phenotype A had increased total cholesterol and triglycerides and LDL-C levels while maintaining reduced HDL-C levels. The study results match the findings from Li et al. (2019) [16] which discovered that hyperandrogenic PCOS patients showed increased triglyceride and LDL-C levels compared to non-hyperandrogenic PCOS patients. The research conducted by Kempegowda et al. (2020) [17] established that androgen excess causes changes in fat metabolism through its impact on visceral adiposity and dyslipidemia which leads to increased long-term cardiovascular metabolic issues.

Our research found that Phenotype A exhibited higher LH levels and LH/FSH ratios which matched previous endocrine research findings that established classical phenotypes show greater gonadotropin dysregulation. The total testosterone levels found in all three Phenotypes A B and C showed higher levels than those found in Phenotype D which demonstrated that hyperandrogenism serves as the main factor driving metabolic severity. The relationship between excessive androgen production and insulin resistance strengthens the pathophysiological model which Barber et al. (2016) [18] developed to

show how hyperandrogenism and metabolic dysfunction interact with each other.

Overall, our study supports previous evidence that PCOS phenotypes differ significantly in metabolic burden, with Phenotype A showing the most severe metabolic and endocrine disturbances. These findings highlight the importance of phenotype-based risk stratification and individualized metabolic screening in women with PCOS rather than adopting a uniform management approach.

Conclusion

The present study confirms that PCOS is a heterogeneous disorder with significant variation in metabolic and hormonal profiles across different phenotypes. Phenotype A was the most prevalent and demonstrated the most severe metabolic disturbances, including higher BMI, central obesity, elevated blood pressure, insulin resistance, adverse lipid profile, and greater prevalence of metabolic syndrome. Phenotype B also showed considerable metabolic risk, though less pronounced than Phenotype A. In contrast, Phenotype D exhibited the most favorable metabolic profile, while Phenotype C showed intermediate characteristics. These findings emphasize that hyperandrogenic phenotypes are associated with greater cardiometabolic risk. Therefore, phenotype-based evaluation and individualized management strategies are essential for early identification of high-risk women and for preventing long-term complications such as diabetes and cardiovascular disease.

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