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PHENOTYPIC DISTRIBUTION OF POLYCYSTIC OVARIAN SYNDROME AND THEIR CLINICAL , METABOLIC AND HORMONAL IMPLICATIONS : A CROSS SECTIONAL ANALYSIS IN MEDICAL COLLEGE KOLKATA, INDIA

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ABSTRACT

Background: Polycystic ovarian disease (PCOD) is one of the most common endocrinopathy of women in reproductive age group and exhibits a heterogenous phenotypic presentation each with distinct clinical, metabolic and hormonal implications.
Aim: This study investigates the distribution of PCOD phenotypes as defined by Rotterdam criteria 2003 and evaluation of their clinical, metabolic and hormonal presentation.
Material and methods: This cross-sectional tertiary hospital based study was conducted with 200 OPD patients after taking consent. They were examined between day 2 to day 7 of menstrual cycle or after prolonged amenorrhoea. Various clinical, metabolic and hormonal parameters were studied as per pre-specified proforma. All patients were divided in 4 phenotypic groups based on Rotterdam consensus 2003 namely hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology and examined the associated profiles.
Results: Study showed most prevalent category was phenotype A (44.5%) followed by phenotype D (26.0%). Least common phenotype was phenotype B (12.5%). Phenotype A and B exhibited more menstrual abnormalities and clinical hyperandrogenism in the form of acne & hirsutism but mean testosterone level is more in type A (69.618 ± 9.445 ng/dL) and least in type D (63.903 ± 10.006 ng/dL). Hormonal imbalance particularly LH/FSH ratio is more elevated in type A (1.4817 ± 0.373) and least in type C (1.265 ± 0.157). Dyslipidaemia was predominantly seen in phenotype A and B.
Conclusion: The phenotypic classification of PCOD reveals significant heterogeneity in clinical, metabolic and hormonal profiles. Hyperandrogenic phenotypes are associated with greater metabolic and clinical severity emphasizing the need for phenotype based individualised management to improve long term reproductive and metabolic outcomes.

Keywords: *Phenotype, Metabolic, Amenorrhoea, Dyslipedemia.*

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age with a global prevalence ranging from 6% to 21% depending on the diagnostic criteria used[1] . In the Indian context studies have reported a prevalence of 9.13% to 22.5% reflecting both genetic and environmental influences [2,3].

The diagnosis of PCOS is based on the Rotterdam criteria 2003 which requires 2 out of 3 following features: oligoovulation/anovulation, clinical or biochemical hyperandrogenism and polycystic ovarian morphology (PCOM) on ultrasound. Based on these criteria, 4 phenotypes of PCOS have been identified:

1. Phenotype A (Hyperandrogenism + ovarian dysfunction + PCOM)
2. Phenotype B (Hyperandrogenism + ovarian dysfunction)
3. Phenotype C (Hyperandrogenism + PCOM)
4. Phenotype D (Ovarian dysfunction + PCOM)

Each phenotype exhibits distinct profiles influencing long term outcomes such as infertility, insulin resistance, metabolic syndrome and cardiovascular risk.[4,5]

Therefore, this cross-sectional study was designed to evaluate the phenotypic distribution of PCOS among women presenting to a tertiary care centre and to assess the clinical, metabolic and hormonal implication of each phenotype.

MATERIALS AND METHODS

This is a hospital based cross-sectional study conducted in the department of Obstetrics and Gynaecology at Kolkata Medical College, Kolkata, West Bengal, over a period of 18 months from 1st January 2023 to 30th June 2024 after taking approval from institute research ethics committee, vide Ref. No. MC/KOL/IEC/NON-SPON/1669/11/2022, Dated 30/11/2022. Study population included women aged 15- 40 years attending the Gynecology outpatient department and diagnosed with PCOD based on the Rotterdam criteria 2003. Patients with other causes of hyperandrogenism such as Cushing's syndrome, androgen secreting tumours, congenital adrenal hyperplasia, thyroid disorders and hyperprolactinemia were excluded. Sample size was calculated using appropriate statistical formula for cross-sectional study [$n=4PQ/d^2$, n=180] and a total of 200 women were enrolled using convenience sampling. After obtaining written informed consent, detailed clinical history and physical examination was conducted. A pre-specified proforma was used for obtaining demographic and clinical examination findings. Detailed clinical assessment was done on hirsutism (assessed by modified Ferriman-Gallway score), acne, acanthosis nigricans. Metabolic data included fasting blood glucose and lipid profile, hormonal profiles on LH, FSH, LH/FSH ratio, total testosterone, TSH and prolactin level. Pelvic ultrasonography was performed to detect polycystic ovarian morphology. All data of 200 women were analysed and they were categorised into four phenotypes A, B, C, and D based on Rotterdam criteria 2003.

STATISTICAL ANALYSIS PLAN: Data was analysed using SPSS software. Descriptive statistics was used for demographic variable. Comparison between phenotypes with respect to clinical, metabolic and hormonal parameters was done using chi square test for categorical variable and ANOVA or Kruskal Wallis test with multiple comparison test for continuous variable. A p value of <0.05 was considered statistically significant.

RESULT

A total 200 women diagnosed with PCOD based on the Rotterdam criteria were included in this study. Based on the presence of Hyperandrogenism (HA), oilgo/anovulation (OA), and polycystic ovarian morphology (PCOM) they were classified into 4 phenotypic categories in which phenotype A was the most prevalent 89 (44.5%) and phenotype B was least prevalent.

Table 1: Distribution of PCOD phenotypes

Phenotype	components	Number of patients	percentage
A	HA+OA+PCOM	89	44.5%
B	HA+OA	25	12.5%
C	HA+PCOM	34	17.0%
D	OA+PCOM	52	26.0%
Total		200	100%

The clinical features varied among different phenotypes. Most women presenting with PCOD were 20-27 years of age and there was no significant age variation among different phenotypes. Hirsutism and menstrual irregularities were more common in phenotype A and B while acanthosis nigricans was noted more in phenotype A and C. Appearance of acne was relatively more in phenotype

TABLE 2: Clinical features among different phenotypes

features	Phenotype A n = 89	Phenotype B n = 25	Phenotype C n = 34	Phenotype D n = 52	P value
Mean age (years)	22.77±2.81	24.04±1.94	24.0±2.08	22.61±2.20	0.0094
BMI (Kg/m^2)	26.25±3.14	26.92±3.00	26.09±3.26	26.64±3.01	0.6707
Hirsutism (mFG>8) %	60 (67.4%)	14(56.0%)	12(35.3%)	24(46.2%)	0.0058
Acne (%)	15(16.9%)	5(20.0%)	6(17.6%)	8(15.4%)	0.9661

Acanthosis nigricans (%)	18(20.2%)	4(16.0%)	7(20.6%)	6(11.5%)	0.5721
Menstrual irregularities (%)	80(89.88%)	21(84.0%)	24(70.58%)	35(67.30%)	0.0045
Waist hip ratio WHR	0.836±0.0321	0.82±0.035	0.81±0.0373	0.82±0.080	0.0037

No significant variations were found in fasting blood sugar level and HDL level among all phenotypes of PCOS. However, triglycerides were higher in phenotype A than type C and D (162.89±7.76 Vs 154.86±9.77). In LDL study, phenotype A had more LDL level than others (phenotype A 101.75±6.93 Vs Phenotype D 95.84±8.08). Results of both harmful lipids are statistically significant ($p<0.0001$ and $p=0.0001$)

Table 3: Metabolic parameters among different phenotypes

parameters	Phenotype A	Phenotype B	Phenotype C	Phenotype D	p value
FBS (mg/dl	76.68±10.95	75.52±8.85	76.529±7.95	77.750±8.31	0.8029
HDL (mg/ dl)	42.280±3.20	42.520±4.17	42.26±3.45	41.61±4.14	0.6764
Triglyceride (mg/dl)	162.89±7.76	158.88±6.32	156.38±9.005	154.86±9.77	<0.0001
LDL (mg/dl)	101.75±6.93	97.040±9.701	97.11±7.78	95.84±8.08	0.0001

More alteration of LH/FSH ratio was seen in phenotype A than other phenotypes. Total Testosterone level in blood of phenotype A was more than type C and D. Results of LH/FSH and mean testosterone level was statistically significant ($p<0.0001$ and $p=0.0019$)

Table 4: hormonal profile among different phenotypes

parameter	Phenotype A n = 89	Phenotype B n = 25	Phenotype C n = 34	Phenotype D n = 52	P value
Mean TSH (μIU/ml)	3.907±0.364	3.996±0.390	3.9412±0.324	3.963±0.414	0.7034
LH/FSH	1.4817±0.373	1.265±0.157	1.285±0.209	1.271±0.171	<0.0001
Mean total testosterone (ng/dl)	69.618±9.445	65.840±8.234	64.617±8.315	63.903±10.006	0.0019

No significant difference in number of ovarian follicles and volume of ovaries were found in all phenotypes of PCOD.

Table 5: ovarian morphology study

parameter	Phenotype A n = 89	Phenotype B n = 25	Phenotype C n = 34	Phenotype D n = 52	P value
No of ovarian follicles	14.191±2.016	15.040±1.593	14.411±1.707	14.576±1.730	0.2135
Ovarian volume (cc)	15.179±2.870	15.56±3.123	15.735±2.608	15.596±2.745	0.7236

DISCUSSION

PHENOTYPIC VARIETY

In the current study, phenotype A emerged as the most common (44.5%) phenotypes of PCOD followed by phenotype D (26.0%) and least common variety was phenotype B (12.5%). Multiple studies across various regions indicate that phenotype A is the most common particularly in hospital-based cohorts. For example, a study by Joshi et al in India reported that phenotype A accounted for 52.6% followed by phenotype B (18.6%), phenotype C (16.3%) and phenotype D (12.5%) [6]. In contrast studies from USA and Europe show a higher frequency of phenotype C and D possibly reflecting differences in genetics, environment, lifestyle and health seeking behaviour. [7,8]

Recognising phenotypic subtype is crucial for both diagnostic clarity and tailored treatment. While phenotypes A and B are more likely to seek medical help due to overt symptoms, phenotype C and D remain undetected unless actively screened. Hence phenotype-based stratification enables more precise risk assessment and management specially in population with diverse clinical presentations.

CLINICAL PROFILE

The present cross-sectional study provides insight into the clinical spectrum of PCOD with particular emphasis on the phenotype distribution and their associated clinical presentations. According to the Rotterdam criteria, PCOD is classified into four phenotypes (A-D) which differ not only in metabolic parameters but also significantly in clinical symptomatology impacting diagnosis and management.

In the present study, menstrual irregularities were noted across all Phenotypic subtypes of PCOD although the frequency and type of abnormalities were varied significantly. We observed 89.88% of phenotype A, 84.0% of phenotype B and 70.5% of phenotype C presented with some form of menstrual irregularities.

In the study of Lizneva D et al, almost all women with phenotype A presented with oligomenorrhoea or amenorrhoea indicating chronic anovulation and menstrual dysfunction which is usually long lasting. [4]

We observed prevalence of acne in PCOD patients were not prominent presentation in any phenotype. Among phenotype B 20.5% and phenotype C 17.6% had complaints of acne. On the contrary Diamanti kandarakis et al 2012 study observed acne was the main or only clinical presentation of phenotype C and they mainly attended in dermatology clinic rather than gynaecology department. [9]

In the current study hirsutism was one of the most common presentations of phenotype A (67.4%) and phenotype B (56%). Lizneva et al 2016[4] and Balen AH et al 1995[10] study concluded hirsutism was the most prominent and often severe, seen in up to 70-80% of PCOD women.

Another clinical presentation, acanthosis nigricans, a clinical marker of insulin resistance was observed in 20.6% women of phenotype C and 20.2% women of phenotype A. Gambineri A et al 2002[11] study noted it to be frequent in phenotype A women and Wijeyaratne CN et al 2002[12] study showed moderate to high prevalence of acanthosis nigricans in phenotype B.

Overall, the clinical presentation of PCOD varies significantly across phenotypes. Phenotype A and B exhibit the most severe hyperandrogenic and metabolic features including hirsutism, acne, acanthosis nigricans along with profound menstrual irregularities, though phenotype C and D women are not excluded totally from menstrual abnormalities.

METABOLIC PROFILE

From metabolic perspective, phenotype A and phenotype B showed significantly higher dyslipidaemia when compared to phenotype C and phenotype D. Although there was no significant fasting blood glucose level variation in all types of PCOD. These findings support the view that hyperandrogenism particularly when combined with anovulation contributes to adverse metabolic profiles as reported by Dunaif et al.[13] These metabolic abnormalities are associated with increased risk of type 2 diabetes mellitus and cardiovascular diseases. [14]

HORMONAL PROFILE

Hormonal profiles study further delineated phenotypic differences. Phenotype A demonstrated highest LH/FSH ratio ($p<0.0001$) and elevated testosterone levels ($p<0.0019$) indicating more profound ovarian dysfunction which has been previously linked to the severity of PCOS shown by Dewailly et al 2011[15] and another study by Sahmay S et al. 2013.[16] These hormonal observations may contribute to the ovulatory dysfunction and subfertility associated with this phenotype. So, despite the increasing awareness, the phenotypic variability of PCOS remains under recognized in clinical practice, especially in low resource settings. Understanding the distribution of PCOS phenotypes and their associated clinical, metabolic and hormonal characteristics is essential for tailoring patient specific management, strategies and anticipating long term complications.

CONCLUSION

The phenotypic distribution of PCOD reflects the complex and heterogeneous nature of the disorder, emphasizing the need for individualized evaluation and management. Among the various phenotypes those fulfilling all three Rotterdam criteria tend to exhibit more pronounced clinical, metabolic and hormonal derangements, including higher prevalence of hyperandrogenism, menstrual irregularities, dyslipidemia and increased cardiometabolic risk. In contrast, ovulatory or normo-androgenic phenotypes often demonstrate milder metabolic burden but remain clinically significant, particularly in reproductive health.

Understanding these phenotype-specific patterns is essential for inproving diagnostic accuracy, tailoring therapeutic strategies and predicting long term outcomes. Early identification of high -risk phenotypes can guide timely interventions targeting weight optimization, insulin sentization, androgen control and reproductive planning. Overall, a phenotype-based approach enhances the precision of PCOD management and contributes to better long term reproductive, metabolic and hormonal health in affected women.

LIMITATIONS: A larger sample size could have increased the power of the study. The effect of socio demographic, environmental and psychological determinants of health were not taken into account in this study. More extensive laboratory tests to rule out androgen excess would have strengthened the study.

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