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Original Research Article

Phenotypic spectrum of polycystic ovary syndrome in a tertiary care rural hospital in Eastern India

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ABSTRACT

Background: PCOS is a common, complicated endocrine disorder affecting women of reproductive age causing wide-spectrum of clinical, metabolic and hormonal abnormalities whose characteristics are poorly understood. This research will investigate the phenotypic prevalence of PCOS as well as clinical, hormonal and metabolic aspects to identify risks and therapeutic requirements.
Methods: The Rotterdam criteria were used to classify 204 women with PCOS into four phenotypes: A (hyperandrogenism + oligo/anovulation + polycystic ovaries), B (hyperandrogenism + oligo/anovulation), C (hyperandrogenism + polycystic ovaries) and D (oligo/anovulation + polycystic ovaries). BMI, waist circumference, hip circumference, waist-to-hip ratio and clinical features (menstrual abnormalities, acanthosis nigricans) were examined. To compare the phenotypes, luteinizing hormone, follicle-stimulating hormone, testosterone and metabolic indicators (lipid profiles, glucose metabolism parameters) were examined. The phenotypes were also examined for the prevalence of metabolic syndrome.
Results: A (58.33%) was the most prevalent phenotype, followed by D (32.84%), C (4.90%) and B (3.43%). Phenotype A had the highest BMI and most menstrual abnormalities and also the highest LH/FSH ratio and the highest testosterone, indicating a severe endocrine disorder. Phenotype A also had the worst lipid profile and the highest metabolic syndrome (24.37%).
Conclusion: Phenotype A is the most severe form of PCOS, characterized by clinical, metabolic and hormonal abnormalities and increases the risk of cardiovascular and metabolic problems. These data suggest that PCOS therapy should be phenotype-specific to address the risks and health problems of each phenotype and treatment individualised.

Keywords: PCOS phenotypes, Rotterdam criteria, Metabolic syndrome, Hormonal profile, Reproductive health, Cardiovascular risk

INTRODUCTION

Polycystic ovary syndrome (PCOS), which affects 5–15% of women of reproductive age worldwide, is the most common endocrine disorder: menstrual irregularities, polycystic ovaries and hyperandrogenism are typical causes of infertility in women of reproductive age.[1,2] In addition to reproductive problems, the condition is also associated with obesity, dyslipidemia, insulin resistance, type 2 diabetes and metabolic syndrome.[3] However, polycystic ovaries (PCOM), oligo-/anovulation (OA) and hyperandrogenism/hyperandrogenaemia (HA) are the most common clinical signs of this condition.[4,5] The prevalence of PCOS can be influenced by various diagnostic criteria, such as those of the National Institutes of Health (NIH), the Rotterdam 2003 and the Androgen

Excess Society (AES).[5] In women with PCOS, there are four phenotypes based on the Rotterdam criteria. PCOS prevalence estimates may also be influenced by the lack of standard diagnostic techniques within each criteria set, such as the detection of androgen excess and oligo/novulation and a technology issue in PCOM assessment. However, systemic treatment with hormonal contraceptives and the expression of PCOS are also an issue.[6]

Many reproductive, obstetric, metabolic and psychosocial features are associated with PCOS.[7,8] Reproductive and obstetric symptoms include menstrual abnormalities, infertility, hyperandrogenism and pregnancy problems. Boomsma et al also mention early pregnancy loss, gestational diabetes, pregnancy-induced hypertension and neonatal problems.[9]

Metabolic consequences include metabolic syndrome, increased cardiovascular risk factors, impaired glucose tolerance (IGT), type 2 diabetes (DM2) and a probable increased risk of cardiovascular disease.[10,11] Himelein et al and Thatcher et al found that women with PCOS are more likely to be sad, have self-doubt and poorer quality of life.[12] Recent economic evaluations of PCOS advocate screening, diagnosis and treatment to avoid catastrophic consequences. The International Diabetes Federation recommends early treatment, prevention, or deferral of DM2.[13] Before advocating for this, women with PCOS need to understand appropriate screening, long-term risks, and effective treatments for metabolic morbidity.[14]

Hyperandrogenism, insulin resistance and anovulation are the most common acute problems. Periods are irregular with anovulation. Acne and hirsutism are the result of hyperandrogenism. Most PCOS women secrete more LH, which exacerbates hyperandrogenism. Central obesity and hyperinsulinemia due to PCOS exacerbate insulin resistance and hyperandrogenism. Different endocrine and metabolic disorders in PCOS women can produce a different biochemical and clinical profile, making diagnosis difficult. Patients with PCOS usually have obesity, dyslipidemia, insulin resistance and hyperinsulinemia.[15]

Insulin resistance (IR) causes the metabolic and reproductive problems of PCOS. Insulin increases total and free androgens, decreases SHBG, and increases ovarian androgen production, leading to PCOS.[16] Insulin resistance is associated with obesity, type II diabetes, and poor lipids. Weight, genetics, ethnicity, geographic diversity and lifestyle affect disease symptoms and severity. People with PCOS may have greater overweight and abdominal obesity, which exacerbates insulin resistance. Obesity-specific IR exacerbates intrinsic or PCOS-specific IR in lean women with PCOS.[17] The optimal PCOS diagnostic criteria are still under debate. The 1992 NIH criteria, which included hyperandrogenism and anovulation, now include the non-NIH diagnostic criteria.[6] Two of the three ESHRE/ASRM diagnostic criteria-hyperandrogenism, polycystic ovaries on ultrasound (PCO), and irregular periods with anovulation-were created in 2003. In 2004, hyperandrogenic ovulatory and non-hyperandrogenic an ovulatory PCO phenotypes were developed. In 2006, the AES recommended changing these settings to remove PCO and irregular cycles without hyperandrogenism. Most studies of phenotypic metabolic effects not conducted by the NIH have focused on PCOS. The prevalence of DM2 and cardiovascular disease in reproductive PCOS phenotypes is unknown.[18] Clarification of this issue will help to determine whether non-NIH phenotypes are part of the complex PCOS syndrome and whether reproductive PCOS phenotypes have greater metabolic risks. In this study we aim to enhance our understanding of the heterogeneous nature of PCOS and potentially improve diagnosis, treatment, and management strategies tailored to individual phenotypic presentations.

METHODS

This hospital-based observational and cross-sectional study involved a total of 204 women of childbearing age (18 to 49 years) who were diagnosed with PCOS and attended the outpatient clinic of the Department of Gynecology and Obstetrics at Diamond Harbor Government Medical College and Hospital, South 24 Parganas, from 1 January 2023 to 31 December 2023. Women who had taken birth control pills in the last three months, women with known thyroid, hypothalamic, diabetic, pituitary, adrenal or neoplastic disorders, with a history of ovarian surgery, cytotoxic drugs or radiotherapy, pregnant women, patients taking insulin-sensitizing or lipid-lowering agents, and women with endocrine disorders were excluded. Clinical parameters (BMI, acanthosis nigricans, hirsutism, acne and waist-to-hip ratio), endocrine parameters (FSH, LH, testosterone, LH/FSH ratio, TSH and prolactin) and metabolic parameters (lipid profile, fasting plasma glucose, waist circumference, blood pressure, HbA1c and 75 g glucose challenge test) were documented. The study was conducted with the approval of the institutional research ethics committee under the auspices of the Department of Obstetrics and Gynecology at Diamond Harbor Government Medical College and Hospital, South 24 Parganas. After proper counseling, each patient who was included in the study according to the inclusion and exclusion criteria gave her signed informed consent.

A total of 204 patients were systematically recruited for the study. According to the Rotterdam criteria, these individuals were categorized into four phenotypes. Two thousand and three. I. Phenotype A of traditional PCOS (H+O+P); II. Traditional PCOS with healthy ovaries (H+O); Phenotype B III. Phenotype C of ovulatory polycystic ovary syndrome (H+P) IV. Normal androgenic phenotype D of polycystic ovary syndrome (O+P). The different clinical, metabolic and endocrine characteristics of these groups were then investigated. An existing questionnaire was used to collect clinical and demographic

information. As part of the clinical assessment, a detailed menstrual, personal, historical and family history was obtained from each patient, followed by a thorough physical examination. The Ferriman-Galway (FG) score of ≥8 or a total testosterone (TT) level of ≥60 ng/dl in the blood was used to assess hirsutism. Pelvic ultrasound was used to assess the volume, size, echogenicity and stromal thickness of the ovaries as well as the number and distribution of cysts characteristic of PCOS. During the physical examination, anthropometric measurements such as height, weight, waist circumference, BMI and hip circumference were determined. Acne (grade 1 to 4) and acanthosis nigricans (grade 1 to 4) were assessed for their presence and distribution. Laboratory data were used to compare the levels of FSH, LH, LH/FSH ratio, testosterone, TSH and prolactin in the different groups. The IDF criteria for metabolic syndrome were used to assess metabolic syndrome, including lipid profiles, blood pressure measurements and fasting plasma glucose levels.

RESULTS

A total of 204 patients were consecutively recruited for the study. These patients were categorised into four phenotypes based on the 2003 Rotterdam criteria. I. Classic PCOS (H+O+P); phenotype A II. Classic PCOS but normal ovaries (H+O); phenotype B III. Ovulatory PCOS (H+P); phenotype C IV. Normal androgenic PCOS (O+P); phenotype D. The study analyzed the prevalence of PCOS in women. The average age was 24.18 years, with similar ages for phenotypes A, B, C, and D.

Table 1: Association of baseline characteristics with various PCOS phenotypes.

	Phenotype A (n=119)		Phenotype B (n=7)		Phenotype C (n=10)		Phenotype D (n=67)		F	P value
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD		
Age (in years)	24.18	6.10	19.57	2.51	25.20	6.49	23.64	4.36	1.77	0.154
Weight (kg)	60.58	10.27	58.86	4.06	56.80	3.82	55.21	8.81	4.73	0.003
Height (cm)	154.50	6.05	156.14	4.63	156.30	9.45	155.34	5.80	0.56	0.643
BMI (kg/m2)	25.40	4.24	24.15	1.56	23.40	2.45	22.87	3.38	6.45	0.000
Waist circumference (cm)	86.71	12.66	82.14	10.42	81.80	14.78	87.43	14.26	0.79	0.500
Hip circumference (cm)	98.66	17.23	87.57	11.09	97.20	15.41	100.07	16.03	1.23	0.301
WC/HC	0.90	0.19	0.96	0.21	0.84	0.04	0.88	0.15	0.70	0.552
SBP (mmHg)	115.10	11.48	122.00	14.47	117.80	16.85	118.30	13.93	1.40	0.245
DBP (mmHg)	77.77	8.59	78.86	9.58	79.60	10.80	78.36	9.26	0.18	0.909

Table 2: Association of mean hormonal and metabolic profile with various PCOS phenotypes.

	Phenotype A (n=119)		Phenotype B (n=7)		Phenotype C (n=10)		Phenotype D (n=67)		F	P value
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD		
LH	10.13	2.78	7.66	3.48	9.45	1.56	9.04	2.37	3.91	0.010
FSH	4.86	1.49	5.69	1.67	5.45	2.02	4.43	1.27	3.06	0.029
LH/FSH	2.21	0.66	1.43	0.68	1.93	0.64	2.17	0.71	3.28	0.022
Total testosterone	42.19	24.56	31.06	10.90	37.00	12.24	28.23	12.34	6.85	0.000
TSH	2.60	1.44	2.16	0.17	2.80	1.04	1.99	1.06	3.63	0.014
S. Prolactin	11.22	10.19	8.53	0.64	9.03	3.81	7.62	2.57	2.97	0.033
T. Cholesterol	170.11	29.91	144.29	18.91	149.20	21.99	154.66	28.13	6.07	0.001
LDL	112.54	23.98	105.57	24.51	88.70	18.17	103.25	27.73	4.10	0.008
TG	112.71	57.45	95.29	30.93	92.20	27.92	98.51	35.06	1.62	0.186
HDL	40.49	14.32	49.86	4.26	50.70	31.79	44.87	7.47	3.39	0.019
FBS	86.74	16.66	86.14	8.34	88.80	12.59	90.36	16.68	0.74	0.528
75 mg OGCT	114.69	31.21	99.14	13.93	128.30	46.50	112.90	33.46	1.18	0.320
HbA1C	5.16	1.65	6.26	0.99	5.19	0.69	5.14	1.69	1.06	0.369

The mean weight, height, and BMI varied among phenotypes, with weight and BMI being greater in phenotype A. The mean waist circumference, hip circumference, and waist-hip circumference were similar across phenotypes, but not significantly different. The mean SBP and DBP values were similar across phenotypes, but not significantly different. The mean waist

circumference, hip circumference, and WC/HC were not significantly different among phenotypes. The mean SBP and DBP values were not significantly different among phenotypes A, B, C, and D (Table 1).

Table 3: Association of frequencies of various clinical and metabolic syndrome with various PCOS phenotypes.

		Phenotype A (n=119)		Phenotype B (n=7)		Phenotype C (n=10)		Phenotype D (n=67)		P value
		N	%	N	%	N	%	N	%	
Metabolic syndrome	Present	29	24.37	0	0.00	2	20.00	16	23.88	0.516
	Absent	90	75.63	7	100.00	8	80.00	51	76.12	
Hypo-menorrhoea	Present	31	26.05	5	71.43	5	50.00	48	71.64	<0.001
	Absent	88	73.95	2	28.57	5	50.00	19	28.36	
Oligo-menorrhoea	Present	56	47.06	3	42.86	0	0.00	27	40.30	0.036
	Absent	63	52.94	4	57.14	10	100.00	40	59.70	
Amenorrhoea	Present	4	3.36	0	0.00	0	0.00	4	5.97	0.675
	Absent	115	96.64	7	100.00	10	100.00	63	94.03	
Marital status	Married	73	61.34	1	14.29	3	30.00	38	56.72	0.002
	Unmarried	46	38.66	6	85.71	7	70.00	29	43.28	
Menstrual cycle abnormality	Yes	119	100	7	100	10	100	67	100	-
	No	0	0.00	0	0.00	0	0.00	0	0.00	
Acanthosis nigricans	Yes	41	34.45	2	28.57	4	40.00	20	29.85	0.874
	No	78	65.55	5	71.43	6	60.00	47	70.15	
Hirsutism	Yes	119	100.00	7	100.00	10	100.00	0	0.00	<0.001
	No	0	0.00	0	0.00	0	0.00	67	100.00	

Table 2 reveals the association of mean hormonal and metabolic profiles with various PCOS phenotypes. The mean LH, FSH, LH/FSH, Total testosterone, TSH, and S. Prolactin levels were significantly different between PCOS phenotypes A, B, C, and D. These levels were more in phenotype A. The mean T. Cholesterol, LDL, TG, and HDL levels were also significantly different between PCOS phenotypes A, B, C, and D.

The mean FBS, 75 mg OGCT, and HbA1C levels were also significantly different between PCOS phenotypes. The mean T FBS, 75 mg OGCT, and HbA1C levels were not significantly different between PCOS phenotypes A, B, C, and D. The study highlights the importance of understanding the relationship between hormonal and metabolic profiles and PCOS phenotypes to improve treatment strategies and overall health.

The study shows a correlation between the frequency of clinical and metabolic syndrome and different PCOS phenotypes (Table 3). Metabolic syndrome was found in 24.37% of phenotype A, while hypomenorrhoea was found in 26.05% of phenotype A, 71.443% of phenotype B, 50.00% of phenotype C, and 71.64% of phenotype D. Oligomenorrhoea was found in 47.06% of phenotype A, 42.86% of phenotype B, 0.00% of phenotype C, and 40.30% of phenotype D.

Amenorrhoea was found in 3.36% of phenotype A, 0.00% of phenotype B, 0.00% of phenotype C, and 5.97% of phenotype D. Married and unmarried PCOS phenotypes were significantly different based on marital status. Menstrual cycle abnormalities were found in all PCOS phenotypes. Acanthosis Nigricans was found in 34.45% of phenotype A, 28.57% of phenotype B, 40.00% of phenotype C, and 29.85% of phenotype D. However, phenotypes A, B, C, and D were comparable based on the presence of amenorrhoea.

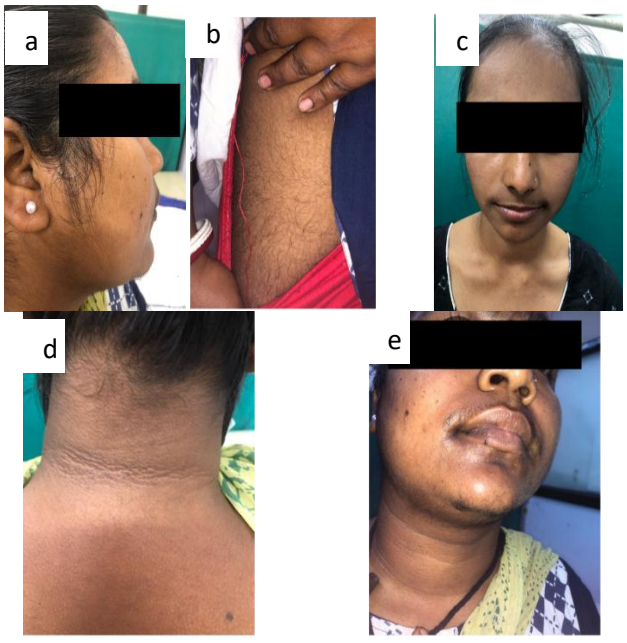

Figure 1 (a-e): Signs of hirsutism Sideburn, Abdomen, Upper lip brow, Acanthosis, Chin.

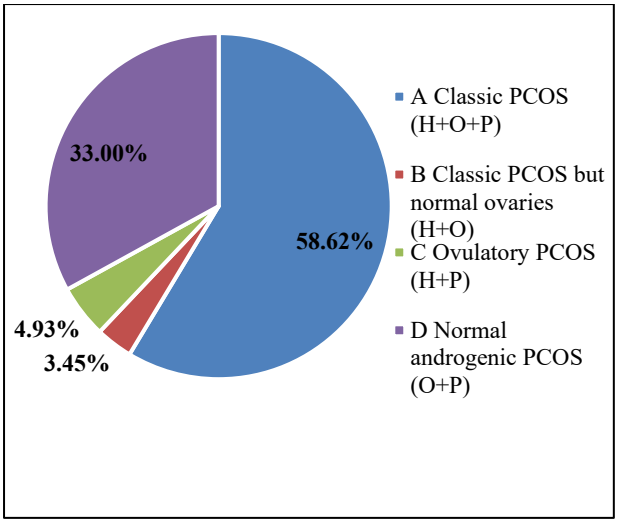

Figure 2: The distribution of various PCOS phenotypes.

DISCUSSION

The study revealed that phenotype A (H+O+P) of PCOS was the most prevalent (58.33%), followed by D (32.84%), C (4.90%) and B (3.43%). These findings are consistent with various studies, who found genetic, racial and regional differences in phenotypic prevalence.[19-21] Recruitment of the research population from infertility, gynecology, dermatology or medical clinics influences these differences. Figure 2 show distribution of various PCOS phenotypes. The percentage of A, B, C, and D phenotypes of PCOS were 58.33%, 3.43%, 4.90% and 32.84%, respectively. And Phenotype A has the highest prevalence. The mean age for PCOS phenotypes A, B, C and D in our study was 24.18±6.10, 19.57±2.51, 25.20±6.49 and 23.64±4.36 years, respectively Sachdeva et al and Pikee et al found a similar age distribution, with individuals with phenotype A being slightly older.[19,22]

In our study the BMI differed significantly between the phenotypes. Previous studies reported that the PCOS phenotypes had higher BMI and central obesity than controls.[19,23] Phenotype A had the highest percentage. Phenotype B had the most obesity, followed by A, C and D. Another study found increased obesity rates in phenotype B.[24] A cross-phenotype comparison of waist circumference (WC), hip circumference (HC) and waist-to-hip ratio (WHR) showed no significant differences. Phenotypes A and D had slightly larger WC.[23] A study found that PCOS phenotypes had a larger waist circumference than controls, although the differences were not always statistically significant.[22] According to previous studies, PCOS phenotypes had similar systolic and diastolic blood pressure values.[19,20] The study discovered significant variations in LH, FSH, testosterone and other hormones between phenotypes. A study found that PCOS women had higher LH and testosterone levels than controls.[22] Zhang et al and Li et al found a higher LH/FSH ratio in phenotype A.[25,26] Phenotype A could have a more severe endocrine disorder due to these hormonal differences. In the study, the phenotypes A, C and D had higher total cholesterol, LDL and triglycerides and lower HDL. A study observed that PCOS women, especially phenotype A, had worse lipid profiles than control subjects.[20] Another study also showed that a greater cardiovascular risk is associated with phenotype A.[22] In the study the glucose metabolism was not significant changes in FBS, OGCT or HbA1C were found between phenotypes. However, previous studies showed that poor glucose tolerance and diabetes were more common in phenotypes A and D.[20,27] PCOS phenotypes A and B, which had a lower glucose-to-insulin ratio, had more glucose abnormalities.[22]

The study found that metabolic syndrome was most common in phenotype A (24.37%), followed by B and C. A study found increased insulin resistance and metabolic abnormalities in phenotype A.[28] These metabolic differences suggest that phenotype A women may be more susceptible to metabolic syndrome and cardiovascular outcomes. In this study the hypomenorrhea occurred most frequently in phenotypes B and D, with significant differences. This is consistent with the findings of Legro et al who found that phenotypes with more severe PCOS symptoms, such as A, had more menstrual irregularities, including oligomenorrhea and amenorrhea. Another study also found that phenotype A was more frequently associated with oligomenorrhea and amenorrhea.[29,30]

In this study we found that the Oligomenorrhea was more common in A and B, but amenorrhea was more common in D. Previous study found that women with all three PCOS diagnostic criteria were more likely to have oligomenorrhea and amenorrhea.[29] In the study, phenotype A had the most married women (61.34%) and phenotype B had the fewest. These differences in marital status were statistically significant and suggest that lifestyle and socioeconomic variables may influence PCOS in different groups. The phenotype C (40.00%) had the most acanthosis nigricans, with significant similarities. These data support Das et al who reported that acanthosis nigricans, a marker of insulin resistance, is more common in PCOS women, especially those with metabolic abnormalities.[31] The severity of acanthosis nigricans is associated with hyperinsulinemia and insulin resistance in phenotypes A and B.[32]

The data confirm previous studies on PCOS traits. Phenotype A increased BMI, metabolic and hormonal imbalances, and cardiovascular risk. C and D had milder symptoms than phenotype B, in which obesity and menstrual irregularities were more pronounced. Due to these clinical, metabolic and hormonal differences, women with PCOS require phenotype-specific therapy. To improve the outcomes of PCOS treatment, future research should investigate the causes of these phenotypic differences. Limitations of the study include the small sample size, the single-centre study, and the

recommendation to conduct a larger sample size and a multicentre study.

CONCLUSION

We concluded that PCOS phenotype A is more common in our Tertiary care rural hospital in eastern India. Obesity, Oligomenorrhoea, Amenorrhoea, disordered lipid profile, and metabolic syndrome are more common in phenotypic group A, putting them at risk for metabolic and cardiovascular issues. Phenotypic division allows for improved knowledge of PCOS pathophysiology and prediction of poor metabolic and cardiovascular effects. Identifying phenotypes will help identify, treat and prognose PCOS-related infertility patients.

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