



Original Article

Reproductive Outcomes After *In Vitro* Fertilisation Amongst Different Phenotypes of PCOS

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ABSTRACT

Objectives: To study reproductive outcomes in different polycystic ovary syndrome (PCOS) phenotypes after in vitro fertilisation (IVF) treatments.

Material and Methods: It is a prospective observational study, conducted from October 2024 to April 2025 at the Centre of IVF and Human Reproduction, Sir Ganga Ram Hospital, New Delhi. Women undergoing IVF were screened for the presence of PCOS and diagnosed based on Rotterdam's criteria. These women were then subcategorised into the four PCOS phenotypes based on their presenting features. All women with PCOS who were scheduled for assisted reproductive technologies (ART) procedures during the study period and met the inclusion criteria were enrolled in the study. Metabolic disorders were screened with lipid profiles, mean blood glucose levels, and free androgen index. Primary outcomes studied were the β hCG positive rate, implantation rate per transfer, clinical pregnancy rates after the first transfer, and early pregnancy loss rate. Secondary outcomes studied were total dose of gonadotropins used, days of stimulation, number of oocytes retrieved, number of oocytes fertilised, number of cleaved embryos, fertilisation rate, median utilisable blastocyst number per patient, blastulation rate, and utilisable blastocyst rate.

Results: There were 10 (18.9%) patients with phenotype A, 6 patients in phenotype B (11.3%), 15 in phenotype C (28.3%), and 22 in phenotype D (41.5%). The most prevalent phenotype was D (41.5%). Women with phenotype A though, had higher serum AMH levels (p -value = 0.006), and had a higher median utilisable blastocyst number per patient (p value = 0.024), but the β -hCG positive rate, implantation rates after first embryo transfer, clinical pregnancy rate, and early pregnancy loss rate were similar in the patients with different phenotypes of PCOS. There was no statistical difference in the total dose of gonadotropins used, number of oocytes retrieved, oocytes fertilised, cleaved embryos, fertilisation rate, blastulation rate, and utilisable blastocyst rate. Metabolic disorder screening results indicated higher cholesterol levels in patients with Phenotype B (p value = 0.01), although no difference was noted in levels of HDL, LDL, non-HDL, triglycerides, and mean glucose levels in the patients with different phenotypes of PCOS.

Conclusion: Our study results suggest that reproductive outcomes, including β hCG positive rate, implantation rate per embryo transfer, clinical pregnancy rate after first embryo transfer, and early pregnancy loss rates, were similar amongst patients with different phenotypes of PCOS. Patients with phenotype A had higher serum AMH levels and had a higher median utilisable blastocyst number per patient, but the utilisable blastocyst rate was comparable across all the phenotypes of PCOS. The study is underpowered to detect differences in reproductive outcomes due to the smaller number of patients enrolled in the study.

Keywords: Clinical pregnancy rate, Implantation rate, PCOS phenotypes, Reproductive outcomes, Utilisable blastocyst rate

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a multifactorial condition characterised by several symptoms and is manifested by metabolic diseases along with impairment of reproductive function.^[1] PCOS was diagnosed according to the ESHRE/ASRM Rotterdam criteria (2003), which require the presence of at least two of the following three features: oligo- or anovulation (AO), clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology. The overall prevalence of polycystic ovarian pattern has been reported to be in the range of 6%-21%.^[2] Infertility in patients with PCOS may be attributed to anovulation occurring in these patients.^[3] The Rotterdam criteria identifies four phenotypes in PCOS cases. Phenotype A is characterised by all three features: polycystic ovaries, oligomenorrhea/anovulation, and clinical or biochemical hyperandrogenism (PCO + OA + HA), phenotype B is characterised by hyperandrogenism (clinical or biochemical) and oligomenorrhea/anovulation (HA + OA), phenotype C is characterised by hyperandrogenism (clinical or biochemical) and polycystic ovarian morphology (HA + PCO), and phenotype D is characterised by oligomenorrhea/anovulation and polycystic ovaries (OA + PCO).^[4] Approximately two out of every three individuals diagnosed with PCOS have either the A or B phenotype.^[5] It has been noted in some studies that different phenotypes of PCOS have different responses to gonadotropins in *in vitro* fertilization (IVF) cycles^[6] that might affect the outcomes of the assisted reproductive technologies (ART) cycles. In most of the studies published in the literature, conflicting results were noted, particularly related to outcomes of ART. An individualised approach to management may be needed in patients with different phenotypes of PCOS to ensure better outcomes.

MATERIAL AND METHODS

A prospective observational study was conducted from October 2024 to April 2025 at the Centre for IVF and Human Reproduction, Sir Ganga Ram Hospital, New Delhi, aiming to study reproductive outcomes in different PCOS phenotypes after IVF treatments. The research/study was approved by the Institutional Review Board at the Indian Fertility Society, Independent Ethical Committee, number F.1/IEC/IFS/2024/No.24, dated 27/12/2024. Women undergoing IVF were screened for PCOS and diagnosed according to the Rotterdam criteria, which require the presence of at least two of the following features:

1. Oligomenorrhea or anovulation, defined as menstrual cycles longer than 35 days or fewer than 8 cycles per year, or irregular menstrual cycles (cycle length <21 days);
 2. Signs of hyperandrogenism like acne and/or hirsutism as per the Ferriman-Gallwey scoring system or biochemical hyperandrogenism (free androgen index);
 3. Polycystic ovarian morphology on ultrasound (as per criteria characterised by the presence of ≥ 20 small follicles, measuring 2-9 mm in at least one ovary and/or ovarian volume $>10 \text{ cm}^3$, based on transvaginal ultrasonography with a transducer frequency $\geq 8 \text{ MHz}$).
- All patients with PCOS, diagnosed according to the ESHRE/ASRM Rotterdam criteria and undergoing assisted reproductive treatment, with self-eggs between the ages of 23-40 years, were included after written informed consent. Patients with a history of unilateral oophorectomy, uterine conditions like uterine anomalies, fibroids, and adenomyosis, severe endometriosis, and hydrosalpinx were excluded.
 - All 53 patients included in the study were subcategorised into the four PCOS phenotypes (based on clinical history, examination, and Ferriman-Gallwey scoring), and PCOS panel testing, including lipid profile, mean blood glucose levels, and free androgen index, was done on day 2 of menses.
 - The transvaginal ultrasound (TVS) of the subjects was done by a single investigator (the most senior) once it was decided that the patient would be enrolled. A baseline hormone profile, including serum follicle stimulating hormone (FSH), luteinizing Hormone (LH), and AMH, was done using an automated electrochemiluminescent immunoassay system.
 - Women underwent controlled ovarian stimulation with an antagonist protocol. As per the patient's age, antral follicle count, BMI, and previous ovarian response, the initial dose of gonadotropin was individualised. Gonadotropin doses were adjusted based on the ovarian response. Oocyte aspiration was performed 35.5 hours post-trigger with a TVS-guided single-lumen oocyte aspiration needle under sedation. Following oocyte retrieval, IVF or intra cytoplasmic sperm injection (ICSI) procedures were carried out. A fertilisation check was undertaken at 17 hours, and resultant embryos were cultured up to the cleavage or blastocyst stage and then frozen. Embryo transfer was done using ultrasound guidance. Luteal phase support was given with vaginal progesterone 400 mg twice daily along with oestradiol valerate 2 mg twice daily in the hormone replacement therapy (HRT) cycle and vaginal progesterone 200 mg twice daily in the natural cycle. Serum beta-human chorionic gonadotropin (β hCG) was measured 14 days after the embryo transfer (ET) procedure. Patients were followed up, and pregnancy outcomes were noted till 12 weeks of pregnancy.
 - Baseline characteristics, results of metabolic screening, details of ovarian stimulation cycles, their outcomes, details of embryo transfer cycle, and reproductive outcomes were noted.

- Primary outcomes studied were β hCG positive rate, implantation rate per transfer, clinical pregnancy rates after first transfer, and early pregnancy loss rate. Secondary outcomes analysed were total dose of gonadotropins used, days of stimulation, number of oocytes retrieved, number of oocytes fertilised, number of cleaved embryos, fertilisation rate, mean utilisable blastocyst number per patient, blastulation rate, and utilisable blastocyst rate.

Definition and Criteria Used

- Fertilisation rate = $\frac{\text{oocytes fertilised (2 pn or pro nuclei)} \times 100}{\text{number of eggs}}$
- Median utilisable blastocyst number per patient is the median of the number of blastocysts frozen in each patient.
- Blastulation rate = $\frac{\text{number of blastocyst formed} \times 100}{\text{number of 2 pn (pro nuclei)}}$
- Utilizable blastocyst rate = $\frac{\text{number of blastocyst frozen} \times 100}{\text{number of 2 pn (pro nuclei)}}$
- Implantation rate = $\frac{\text{number of the gestational sacs} \times 100}{\text{number of the transferred embryos}}$
- Clinical pregnancy was defined by the occurrence of an intrauterine gestational sac with the presence of cardiac activity.
- Clinical pregnancy rate = $\frac{\text{number of clinical pregnancies} \times 100}{\text{number of embryo transfer cycle}}$
- Early pregnancy loss includes loss of pregnancy up to 12 weeks of gestation, including biochemical pregnancies, blighted ovum, and missed miscarriage.
- Early pregnancy loss rate = $\frac{\text{number of early pregnancy losses} \times 100}{\text{number of } \beta \text{ hCG positive}}$

Data Management and Statistical Analysis

- Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 28.0.
- Since this study starts with a cohort of PCOS women and not 4 phenotype groups, the sample size estimated by considering the percentage of phenotype C among the PCOS women was 13%, as per the study conducted by Patel *et al.*^[7] With a 95% CI and an error margin of 5%, the sample size was estimated to be 174, but due to time constraints, 53 women were included in the study. This is one of the limitations of the study.
- Continuous variables were reported as mean \pm standard deviation (SD) for normally distributed data, or as median and interquartile range (IQR) for data not following a normal distribution.

- Most variables (e.g., age, weight, Body Mass Index (BMI), high density lipoprotein (HDL), oocyte retrieval (OCR) endometrial thickness, blastulation rate, and fertilisation rate) followed a normal distribution, while several [anti mullerian hormone (AMH), oestradiol, cholesterol, triglycerides, LDL, non-HDL, stimulation dose, oocytes, embryos, Ferriman-Gallwey score, etc.] deviated significantly. This indicates that both parametric and non-parametric tests were appropriately applied depending on the variable distribution.

- Categorical variables were presented as frequencies and percentages.
- Differences in clinical pregnancy rates among various PCOS phenotypes were assessed using the chi-square test or Fisher's exact test, as appropriate.
- For continuous variables, comparisons across phenotypes, such as total gonadotropin dose, number of oocytes retrieved, and fertilisation rate, were carried out using either one-way analysis of variance (ANOVA) (for normally distributed data) or the Kruskal-Wallis test (for non-normally distributed data).

A *p*-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

Baseline Characteristics

In this study, 53 ovarian stimulation cycle data points were collected and analysed. There were 10 (18.9%) patients in phenotype A, 6 (11.3%) in phenotype B, 15 (28.3%) in phenotype C, and 22 (41.5%) in phenotype D. The baseline characteristics were noted in all the phenotypes. It was found that there was no statistical difference in the age distribution, BMI, type of infertility, infertility duration, and history of previous failed embryo transfer cycles, as shown in Table 1; but AMH levels amongst different phenotypes were statistically significantly different. It was noted to be highest in phenotype A with a value of 14 (7.70-18.90) ng/mL compared to 3.99 (3.72-9.00) ng/mL in phenotype B, 4.47 (3.74-6.10) ng/mL in phenotype C, and 5.60 (4.75-7.65) ng/mL in phenotype D (*p* value = 0.006). It was also noted that there was a difference in the causes for which IVF was undertaken in the different phenotype groups, as depicted in Table 1.

Metabolic Screening Results

Metabolic screening was done for all patients. There was a statistically significant difference in the cholesterol levels in the different phenotype groups. The levels of cholesterol were higher in phenotype A: 158.0 (148-170) mg/dL and

Table 1: Baseline characteristics.

Variables	Sub-Division	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 22)	p value	Effect size (95% CI)
Percentage (%)		18.9	11.3	28.3	41.5		
Age (years) (Mean ± SD)		32.30 ± 3.6	34.00 ± 4.6	33.33 ± 3.6	32.5 ± 2.7	0.68	0.030 (0.0-0.115)
BMI (kg/m ²) (Mean ± SD)		27.5 ± 3.3	30.0 ± 7.5	26.2 ± 3.9	26.9 ± 4.5	0.379	0.06 (0.0-0.174)
Sr AMH (ng/mL)		14.00 (7.70-18.90)	3.99 (3.72-9.00)	4.47 (3.74-6.10)	5.60 (4.75-7.65)	0.006	0.19 (0.005-0.472)
Infertility duration		4.0 (3.0-7.0)	2.0 (1.5-2.0)	2.0 (1.0-5.0)	3.0 (2.0-5.0)	0.173	0.04 (-0.01-0.09)
Type of infertility	Primary	5 (50.0%)	5 (83.3%)	9 (60.0%)	15 (68.2%)	0.55	-
	Secondary	5 (50.0%)	1 (16.7%)	6 (40.0%)	7 (31.8%)		
Indication of IVF	Anovulatory PCOS	5 (50.0%)	0 (0.0%)	0 (0.0%)	5 (22.7%)	0.037	-
	Unexplained	3 (30.0%)	2 (33.3%)	9 (60%)	9 (40.9%)		
	Male factor	0 (0.0%)	4 (66.7%)	5 (33.3%)	8 (36.4%)		
	Others	2 (20.0%)	0 (0.0%)	1 (6.7%)	0 (0.0%)		
Previous failed ET	0	10 (100.0%)	5 (83.3%)	13 (86.7%)	19 (86.4%)	0.674	-
	1	0 (0.0%)	1 (16.7%)	1 (6.7%)	1 (4.5%)		
	2	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (9.1%)		
	3	0 (0.0%)	0 (0.0%)	1 (6.7%)	0 (0.0%)		

BMI: Body mass index, AMH: Anti mullerian hormone, IVF: In vitro fertilization, ET: Embryo transfer, CI : Confidence interval, SD: Standard deviation, p value significant for type of infertility: 0.55, p value significant for indications of IVF: 0.037.

Table 2: Metabolic screening results.

Variable	Statistics	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 22)	p-value	Effect size (95% CI)
Cholesterol	Median (IQR)	158.0 (148.0-170.0)	171.0 (168.0-176.0)	150.0 (142.0-160.0)	156.5 (150.0-162.0)	0.01	0.16 (0.06-0.26)
Triglyceride	Median (IQR)	125.0 (106.0-136.0)	121.0 (114.0-132.0)	122.0 (108.0-128.0)	124.0 (122.0-160.0)	0.425	0
HDL	Mean ± SD	54.30 ± 6.86	57.17 ± 7.17	53.13 ± 5.11	51.95 ± 8.98	0.482	0.049 (0.0-0.154)
LDL	Median (IQR)	90.0 (86.0-98.0)	97.0 (93.0-101.0)	88.0 (86.0-92.0)	93.5 (85.0-98.0)	0.255	0.02 (-0.02-0.06)
Non-HDL	Median (IQR)	116.50 (112.00-120.00)	118.00 (106.00-130.00)	124.00 (112.00-130.00)	120.00 (112.00-132.00)	0.884	-0.05
Mean blood glucose	Median (IQR)	90.00 (86.00-100.00)	98.00 (95.00-112.00)	99.00 (88.00-102.00)	100.00 (88.00-103.00)	0.767	-0.04
FAI	Median (IQR)	6.72 (5.53-6.73)	3.15 (3.15-3.15)	3.59 (3.40-3.78)	3.51 (2.40-3.90)	0.225	0.03 (-0.02-0.007)
Ferriman-Gallwey score	Median (IQR)	15.0 (13.25-16.0)	15.0 (13.25-16.0)	16.0 (15.5-20.0)	4.5 (4.0-5.75)	< 0.001	0.46 (0.32-0.59)

HDL: High density lipoprotein, LDL: Low density lipoprotein, FAI: Free androgen index, IQR: Interquartile range. CI: Confidence interval, SD: Standard deviation, Significant p value is <0.05.

phenotype B: 171 (168-176) mg/dL, while they were 150 (142-160) mg/dL in phenotype C and 156.5 (150-162) mg/dL in phenotype D (p value = 0.01). It was also noted that there was no statistically significant difference in HDL levels,

non-HDL levels, low density lipoprotein (LDL) levels, triglyceride levels, mean blood glucose levels, and free androgen index. There was a difference in the Ferriman-Gallwey score amongst different phenotypes, as shown in Table 2.

Table 3: Controlled ovarian stimulation cycle details.

Variable	Sub-Division	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 22)	p-value	Effect size (95% CI)
Protocol	Antagonist	10 (100%)	6 (100%)	15 (100%)	22 (100%)	NA	
Gonadotropin used	FSH	6 (60.0%)	6 (100%)	14 (93.3%)	21 (95.5%)	0.41	-
	FSH + LH	4 (40.0%)	0 (0.0%)	1 (6.7%)	1 (4.5%)		
Total dose (Median (IQR))		1800 (1325-2325)	1763 (1350-2250)	1575 (1350-1775)	1375 (1250-1875)	0.536	-0.02
Subsequent dose change	No	3 (30.0%)	1 (16.7%)	7 (46.7%)	9 (40.9%)	0.529	
	Increase	2 (20.0%)	0 (0.0%)	0 (0.0%)	2 (9.1%)		
	Decrease	5 (50.0%)	5 (83.3%)	8 (53.3%)	11 (50.0%)		
Days of stimulation		8.5 (8.0-13.0)	8.0 (7.0-11.0)	8.0 (7.0-9.0)	8.0 (8.0-9.0)	0.225	0.03 (-0.02-0.008)
Trigger	Decapeptyl	10 (100.0%)	3 (50.0%)	10 (66.7%)	19 (86.4%)	0.087	
	Dual	0 (0.0%)	2 (33.3%)	3 (20.0%)	3 (13.6%)		
	Ovitrelle	0 (0.0%)	1 (16.7%)	2 (13.3%)	0 (0.0%)		
Terminal Oestradiol		2670.5 (1961.0-4381.0)	2303.5 (1725.0-2839.0)	2576.0 (1872.0-4720.0)	2682.0 (1482.0-3826.0)	0.795	-0.04
OCR endometrial thickness		8.0 (7.5-8.8)	9.2 (7.4-11.4)	7.7 (7.5-9.7)	8.5 (7.79.5)	0.331	0.01 (-0.02-0.03)

FSH: Follicle stimulating hormone, LH: Luteinizing hormone, IQR: Interquartile range, OCR: Oocyte retrieval. CI: Confidence interval, NA: Not applicable, Significant p-value is < 0.05.

Table 4: Controlled ovarian stimulation cycle outcomes.

Variable	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 22)	p-value	Effect size (95% CI)
Number of oocytes	23 (27-20)	16 (24-14)	18 (24-10)	19 (23-15)	0.264	0.02 (-0.02-0.06)
Number of oocytes fertilised	14 (219)	9 (14-5)	9 (13-7)	12 (13-9)	0.169	0.04 (-0.01-0.09)
Number of cleaved embryos	13 (21-9)	9 (13-5)	9 (13-7)	13 (15-9)	0.187	0.03 (-0.02-0.08)
Fertilisation rate	70.71 (87.50-44.44)	46.53 (70.8335.71)	58.33 (86.67-41.38)	58.57 (70.37-42.86)	0.499	-0.01
Median utilisable blastocyst number per patient	6 (8-4)	2 (3-1)	2 (4-1)	4 (7-2)	0.024	0.12 (0.03-0.21)
Median blastulation rate	62.4 (75.0-46.3)	34.3 (40.0-20.0)	35.7 (66.7-14.3)	58.2 (75.00-33.3)	0.268	0.02 (-0.02-0.05)
Utilisable blastocyst rate	50.05 (55.60-22.20)	26.65 (40.00-17.60)	28.60 (55.60-14.30)	42.00 (66.70-16.70)	0.531	-0.02

CI: Confidence interval, Significant p-value is <0.05.

Controlled Ovarian Stimulation Cycle Details

Antagonist protocol was used for all the stimulation cycles. It was noted that FSH combined with LH was used more in phenotype A compared to other phenotypes. Though this was mainly due to the clinician's preference for the addition

of LH, it could not be attributed to specific phenotype characteristics. There was no significant difference noted in the total dose of gonadotropins used, subsequent dose change, days of stimulation, trigger, oestradiol levels, and endometrial thickness on the day of trigger, as depicted in Table 3.

Table 5: Embryo transfer cycle details.

Variable	Sub-Division	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 21)	p-value	Effect size (95% CI)
Preparation method	Natural cycle + modified natural cycle	3 (30.0%)	3 (30.0%)	9 (60.0%)	18 (85.7%)	0.015	-
	HRT	7 (70.0%)	3 (50.0%)	6 (40.0%)	3 (14.3%)		
Endometrial thickness		8.0 (7.5-8.8)	9.2 (7.4-11.4)	7.7 (7.5-9.7)	8.5 (7.79-9.5)	0.703	-0.03
Embryo stage	Day 3	0 (0%)	1 (16.7%)	1 (6.7%)	0 (0%)	0.451	
	Day 5	8 (80.0%)	5 (83.3%)	10 (66.7%)	17 (81.0%)		
	Day 6	2 (20.0%)	0 (0%)	4 (26.7%)	4 (26.7%)		
Number transferred		1.00 (1.0-1.0)	1.00 (1.0-1.0)	1.00 (1.0-1.0)	1.00 (1.0-1.0)	0.584	-0.02
Embryo grade	Good	10 (100.0%)	6 (100.0%)	13 (86.7%)	20 (95.2%)	0.487	
	Average	0 (0%)	0 (0%)	2 (13.3%)	0 (0%)		
	Poor	0 (0%)	0 (0%)	0 (0%)	1 (4.8%)		

HRT: Hormone replacement therapy. CI: Confidence interval, Significant p-value is <0.05.

Table 6: Embryo transfer cycle outcomes.

Outcome	Phenotype A (n = 10)	Phenotype B (n = 6)	Phenotype C (n = 15)	Phenotype D (n = 21)	p-value
β-hCG positive	6/10 (60.0%)	6/6 (100.0%)	9/15 (60.0%)	14/21 (66.7%)	0.362
Implantation rate per transfer	6/10 (60.0%)	4/6 (66.7%)	8/15 (53.3%)	12/21 (57.1%)	0.977
Clinical pregnancy rate	6/10 (60.0%)	4/6 (66.7%)	8/15 (53.3%)	11/21 (52.4%)	0.935
Early pregnancy loss rate	1/6 (16.7%)	2/6 (33.3%)	3/9 (33.3%)	4/14 (28.6%)	0.956

hCG: Human chorionic gonadotropin. , Significant p-value is <0.05.

Controlled Ovarian Stimulation Cycle Outcomes

Median utilisable blastocyst number per patient was highest in patients with phenotype A: 6 (8-4) compared to phenotype B: 2 (3-1), phenotype C: 2 (4-1), and phenotype D: 4 (7-2) (p value = 0.024). While the total number of oocytes retrieved, oocytes fertilised, number of cleaved embryos, fertilisation rate, blastulation rate, and utilisable blastocyst rate were similar amongst the patients in different phenotypes as depicted in Table 4.

Embryo Transfer Cycle Details

Out of the 53 ovarian stimulation cycles, 52 patients underwent embryo transfer, as one patient did not form a blastocyst in the initial stimulation cycle. There was a difference in the preparation protocol used for embryo transfer amongst women with different phenotypes of PCOS, as women with phenotype C had ovulatory cycles, so a natural cycle protocol was used for endometrial preparation for embryo transfer in some patients. There was no difference in the endometrial thickness, number of embryos transferred, stage of embryo, and grade of embryo transferred in patients with different phenotypes as depicted in Table 5.

Outcomes of the Embryo Transfer Cycle

The β hCG positive rate (p value = 0.362), implantation rate per embryo transfer (p value = 0.977), and clinical pregnancy rates after the first embryo transfer (p value = 0.935) were similar amongst women with different phenotypes of PCOS. There was no difference in the early pregnancy loss rate amongst women in different phenotypes as well (p value = 0.956) as depicted in Table 6.

DISCUSSION

The study was undertaken to determine if there was any difference in the reproductive outcomes and metabolic disorders amongst women with different phenotypes of PCOS. It was noted that the prevalence of phenotype D was the highest, which was consistent with studies undertaken by Wang *et al.*^[8] and Nayar *et al.*^[9]

In our study, the second most prevalent phenotype was type C, followed by phenotype A and B. When baseline characteristics were analysed, it was found that phenotype A had the highest levels of serum AMH, similar to the results in the study by Patel *et al.*^[7] and Nayar *et al.*^[9]

We observed that there was a difference in the indication for IVF in patients with different phenotypes, but the other baseline characteristics were similar in the patients in different phenotype groups.

Metabolic screening results showed the highest levels of cholesterol in women with phenotype B compared to other phenotypes. While the levels of HDL, LDL, non-HDL, and triglyceride were similar in the patients with different phenotypes, there was no difference in the mean glucose levels, as well as the free androgen index, in the patients with different phenotypes of PCOS. There was a difference in the Ferriman-Gallwey score, as phenotype D was expected to have a lower score, as less or no androgenic features occur in phenotype D. In a study done by Eftekhar *et al.*,^[10] a difference was noted in the fasting blood glucose levels in patients with different phenotypes of PCOS, but currently there is limited literature on the levels of blood sugar in patients with different phenotypes of PCOS.

The analysis of the stimulation cycle used in women with different phenotypes of PCOS suggests that there was no significant difference in the number of days required for the stimulation cycle. In a study undertaken by Patel *et al.*,^[7] similar findings were noted, but the difference was not statistically significant. Also, in the current study, we found no difference in total dose of gonadotropin, subsequent dose change, trigger used, endometrial thickness on the day of trigger, and oestradiol levels on the day of the trigger. Although in a study done by Patel *et al.*,^[7] the authors noted the need for a higher dose of gonadotropin for the stimulation cycle of patients with phenotype A, and in a study by Nayar *et al.*,^[9] the authors noted the need for a higher dose of gonadotropin by women with phenotype B, the study by Wang *et al.*^[8] showed findings similar to our study. They reported no difference in the total dose of gonadotropins used and more number of days of stimulation for women with phenotype A.

In our study, it was noted that although there was a difference in the levels of serum AMH, there was no statistically significant difference in the number of oocytes retrieved, oocytes fertilised, cleaved embryos, and fertilisation rate. Eftekhar *et al.*,^[10] also found no difference in the number of oocytes retrieved despite higher serum AMH levels in women with phenotype A. A higher median number of utilisable blastocysts per patient was noted in women with phenotype A, but the blastulation rate and utilisable blastocyst rate were similar in patients with different phenotypes of PCOS.

When the embryo transfer cycle details were analysed, it was seen that there was a difference in the method used for endometrial preparation for embryo transfer, as few patients with phenotype C were planned with the natural cycle method, as the cycles were ovulatory. Also, the method used was determined by a number of other factors, like the clinician's preference, as well as patient factors like ability

to follow up and response to ovulation induction drugs if used previously. Endometrial thickness, number of embryos transferred, stage of embryo transferred, and grade of embryo transferred were comparable in the patients with different phenotypes.

The reproductive outcomes of the patients in different phenotypes were similar when the β -hCG positive rate, clinical pregnancy rate after first embryo transfer, early pregnancy loss, and implantation rates per embryo transfer were compared. The study by Nayar *et al.*^[9] found a higher statistically significant clinical pregnancy rate in patients with phenotype D, and Patel *et al.*,^[7] reported a higher clinical pregnancy rate in patients with phenotype D, though it was not statistically significant. Eftekhar *et al.*,^[10] reported no difference in the clinical pregnancy rate and implantation rates, similar to our study. Also, studies by Selcuk *et al.*^[11] and Wang *et al.*^[8] also found no difference in the clinical pregnancy and live births amongst women with different phenotypes of PCOS.

Strengths

- Prospective observational study with meticulously collected data.
- Metabolic disorder screening was done with lipid profile, mean blood glucose levels, and free androgen index, which have been studied in a few studies only.

Limitations

This was a single-centre study with a relatively small sample size and a shorter study duration. As a result of the limited timeframe, patients were only followed up after their first embryo transfer.

CONCLUSION

This prospective cohort study suggests that women with phenotype A had higher serum AMH levels and median utilisable blastocyst number per patient, but utilisable blastocyst rate was comparable across all the phenotypes of PCOS. Study results suggest that reproductive outcomes, including β hCG positive rate, implantation rate per embryo transfer, clinical pregnancy rate after first embryo transfer, and early pregnancy loss rates, were similar amongst patients with different phenotypes of PCOS. Future studies with a large sample size are recommended to enhance the accuracy and reliability of the results. Our study is underpowered to detect differences in reproductive outcomes due to the less number of patients enrolled in the study.

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Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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