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Association Between Polycystic Ovary Syndrome Phenotypes and Treatment Outcomes in Infertile Women.

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Name of Author:
Dr. AIMEN KALEEM[1*], Dr. SADIA HABIB MEMON[2], Dr. ZULEKHA NASIM[3],
Dr. NADIA JABEEN[4], Dr. ROSHNI MUMTAZ[5], Dr. HINA AYUB[6].

Affiliation: *[1]MBBS, FCPS (Obstetrics & Gynaecology) SPR gynae c unit Women and children hospital MTI Dera Ismail Khan.*
[2]MBBS, FCPS Obstetrics & gynaecology Liaqat University Hospital Hyderabad/Jamshoro.
[3]MBBS, FCPS, gynaecology, Islam Medical College Sialkot.
[4]Associate professor, gynae and obstetrics, Rawal institite of health sciences Affiliqted with Rawal Medical And Dental College Islamabad.
[5]2nd year obgyn resident, Department obstetretics and gynaecolgy. Kth /Khyber medical college.
[6]Associate Professor, Obstetrics and Gynecology, Gomal Medical College.

Corresponding Author:
Dr. AIMEN KALEEM.

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Abstract: ***Background:*** Polycystic Ovary Syndrome (PCOS) is a heterogeneous endocrine disorder and is a major cause of infertility in reproductively-aged women. Clinical variability and response to treatment across phenotypes of PCOS present a challenge in the optimization of fertility outcomes. ***Objective:*** To assess the relationship between PCOS phenotypes and treatment responses in infertile women. ***Methodology:*** This comparative cross-sectional study was carried out over a period of one year Women and Children Hospital, MTI, Dera Ismail Khan, and involved 165 infertile women with PCOS as per the Rotterdam criteria. The participants were divided into phenotypes A, B, C, and D. Demographic, clinical, and hormonal profiles were obtained. The patients all received standard infertility therapies, such as ovulation stimulation using letrozole or clomiphene citrate with or without metformin. The analysis was done through SPSS version 26, and $p \leq 0.05$ was regarded as significant. ***Results:*** The most common was the classical phenotype (A). Hormonal profiles showed significant differences among phenotypes, with increased levels of androgen and insulin in severe phenotypes. Compared to the classical phenotypes, milder phenotypes produced higher ovulation and pregnancy rates and poorer outcomes. High BMI and hyperandrogenism were both significantly related to the decreased success of the treatment. ***Conclusion:*** PCOS phenotypes play a great role in determining hormone features and infertility treatment response. An individualized management approach based on phenotype could enhance reproductive success in infertile women with PCOS.

Keywords: Polycystic Ovary Syndrome, PCOS Phenotypes, infertility, Ovulation induction, hyperandrogenism.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a widespread endocrine disorder that afflicts women of reproductive age, which is defined by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.[1] It is an unequal disorder with reproductive, metabolic, and psychological consequences that differ across individuals. PCOS has a prevalence of about 6% to 21% among women worldwide, depending on diagnostic criteria, with recent estimates indicating a prevalence of about 11% to 13% globally.[2] The syndrome is believed to be the cause of the most frequent anovulatory infertility and has a significant impact on the global burden of fertility and infertility problems.

Infertility is a major community health problem, especially in developing nations, where its levels are said to be as high as 21.9% in Pakistan.[3] PCOS affects a significant percentage of cases in affected women, with research showing that as many as 72% of women infected with PCOS are infertile, as opposed to 16% of women not infected with the condition.[4] The incidence of infertility caused by PCOS has been on the rise, with the cases worldwide rising to more than 12 million in 2019 in comparison to the levels of the 1990s, at about 6 million cases.[5] This upward trend highlights the growing clinical and socioeconomic effects of the disorder.

One of the main characteristics of PCOS is its phenotypic heterogeneity. According to the Rotterdam criteria, PCOS has four phenotypes (A, B, C, and D) based on the presence or absence of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.[6, 7] Such phenotypes vary greatly in hormonal profiles, metabolic risks, and clinical manifestations. New evidence indicates that these differences also affect reproductive outcomes and the response to infertility treatments. Some of these phenotypes are linked to an increased risk of poor pregnancy outcomes, whereas some have shown a more positive reproductive outcome or a response to assisted reproductive methods.[8]

Treatment options for infertility in PCOS range from lifestyle changes, ovulation induction using medications like clomiphene citrate or letrozole, insulin sensitizers like metformin, and advanced assisted reproductive procedures.[9] The success rates of treatment may be promising, up to 70 to 80% conception rates with some regimens, but not all patients respond in the same way to treatment. The variability can be in part attributed to underlying phenotypic differences, which are often not considered in normal clinical practice.[10]

Although the heterogeneity of PCOS is becoming increasingly acknowledged, the majority of clinical strategies currently utilize a generalized treatment approach, as opposed to phenotype-based treatment. The existing knowledge gap in the literature about the effects of various PCOS phenotypes on treatment effects in infertile women is still very huge, especially in resource-constrained environments. The identification of phenotype-specific responses would help to increase individualized treatment, improve success rates, and decrease the number of unnecessary interventions. Because of the heterogeneity of PCOS and the variability in treatment effects, there is a strong rationale to examine the relationship between various PCOS phenotypes and infertility treatment outcomes. The knowledge of these relationships can aid in more individualized and successful management techniques that will eventually lead to better reproductive outcomes. Thus, the goal of the research was to determine the correlation between polycystic ovary syndrome phenotypes and treatment outcomes among infertile women.

METHODOLOGY

The current research was done as a cross-sectional study at the Department of Obstetrics and Gynecology, Women and Children Hospital, MTI, Dera Ismail Khan, for a time period of 1 year from 1st January 2025 to 31st December 2025. The infertile women reporting to seek evaluation and treatment were recruited. Women with polycystic ovary syndrome (PCOS), according to the Rotterdam criteria, were included as the study population, and further divided into various phenotypes (A, B, C, and D) to compare the results of the treatment.

The OpenEpi version 3 was used to determine the sample size. Based on the confidence level of 95%, power of 80%, and the expected proportion of positive treatment of around 60% of PCOS patients, as a study on a similar topic had already established.[11] The required minimum sample size was estimated to be 165 patients.

The sampling technique was a non-probability consecutive sampling method where all eligible infertile women with PCOS who presented themselves within the study period were included until the sample size was reached. The study involved women aged 18-40 years with PCOS diagnosed using the Rotterdam criteria and with either primary or secondary infertility. Also, it was only the patients who volunteered and gave informed consent that were enrolled. Women with other causes of infertility, like tubal blockage, male factor infertility, hyperprolactinemia, thyroid disorders, endometriosis, or chronic systemic diseases like diabetes mellitus, hypertension, or renal disease were excluded. Patients with a history of assisted reproductive methods or who were receiving long-term hormonal treatment were also not included to prevent any confounding effect on the treatment outcome.

A pre-tested and structured proforma was used to collect data. Following ethical approval by the institutional review board and informed consent of the participants,

extensive demographic data were gathered, such as age, body mass index (BMI), infertility duration, and type, and menstrual history. Clinical examination involved examination of hyperandrogenism signs like hirsutism and acne. Laboratory studies were conducted to determine hormonal profiles such as serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), testosterone levels, and fasting insulin levels as necessary. The ovarian morphology was measured by transvaginal or transabdominal ultrasound. On the basis of these results, the participants were classified into various PCOS phenotypes.

Polycystic ovary syndrome phenotypes were categorized based on the Rotterdam criteria into four different groups depending on the presence or absence of three main characteristics, namely hyperandrogenism (clinical or biochemical), ovulatory dysfunction, and polycystic ovarian morphology.[12] Phenotype A (classic PCOS) entailed those patients with all three phenotypes, such as hyperandrogenism, oligo/anovulation, and polycystic ovaries, and is said to be the most serious form with both a metabolic and reproductive abnormality. Phenotype B (non-polycystic ovaries) comprised patients with hyperandrogenism and ovulatory dysfunction but without polycystic ovarian morphology on ultrasound. Phenotype C (ovulatory PCOS) consisted of patients who were hyperandrogenic and had polycystic ovaries, but had normal ovulatory cycles. Phenotype D (non-hyperandrogenic PCOS) included patients who had ovulatory dysfunction and polycystic ovarian morphology without hyperandrogenism and is generally regarded as the milder phenotype that has relatively lower metabolic complications.

Every patient received the regular infertility treatment regimens, which included lifestyle change, ovulation induction with either letrozole or clomiphene citrate, and adjunctive therapy with metformin in cases where necessary. A comparison of treatment outcome was made in the ovulation rate, clinical pregnancy rate, and time to conception during a specific follow-up period. Serial ultrasound scans and biochemical confirmation were done, as necessary, to monitor patients.

The statistical package, Statistical Package for Social Sciences (SPSS) version 26, was used to enter and analyze the collected data. The quantitative variables (age, BMI, hormone levels) were represented as mean ± standard deviation (SD), whereas the qualitative variables (PCOS phenotypes and treatment outcomes) were represented as frequencies and percentages. The Shapiro-Wilk test was used to measure the normality of data distribution. The Chi-square test of categorical variables and one-way ANOVA of continuous variables were used to compare the different PCOS phenotypes and treatment results based on the data distributions. The p-value of ≤ 0.05 was considered significant.

RESULTS

The study population was mainly young women in their late twenties, with a majority of the participants being in the overweight to obese range of BMI. Primary infertility was more common than secondary infertility, and menstrual irregularities, especially oligomenorrhea, were very common as a manifestation of underlying ovulatory dysfunction that is typical of PCOS. The endocrine nature of the disorder was supported by the fact that a significant percentage of the participants had clinical features of hyperandrogenism, such as hirsutism and acne (Table 1).

In terms of phenotypic distribution, phenotype A (Classical) was the most prevalent phenotype, with almost equal distributions of phenotypes B and D, and phenotype C being rather rare. This distribution indicates that more severe phenotypes with combined hyperandrogenism, ovulatory dysfunction, and/or polycystic ovarian morphology are predominant and indicate a greater burden of more complicated clinical manifestations in the population under study (Table 2).

Hormonal and metabolic parameter analysis revealed that there were clear differences between PCOS phenotypes. The more extreme phenotypes had a relatively higher level of luteinizing hormone, testosterone, and fasting insulin, which is more indicative of endocrine and metabolic derangements. Conversely, the less severe phenotype (Phenotype D) had lower androgen and insulin concentrations, which means a less severe metabolic imbalance. Most of the parameters were statistically significant (Table 4).

The phenotypes also showed a great variation in treatment outcomes, and ovulation and pregnancy rates varied among the groups. The less severe types, especially those not characterized by severe hyperandrogenism, had relatively more successful reproductive outcomes, such as increased ovulation and conception rates and reduced time to conception. On the other hand, the classical phenotype had relatively reduced success rates and slower conception, which indicates a potential adverse effect of combined abnormalities of endocrine conditions on treatment responsiveness. These results highlight the significance of phenotypic variation in determining the success of the therapeutic outcome (Table 4).

Regarding treatment methods, the most common forms of treatment were ovulation induction agents, the most common of which was letrozole, which was either used alone or in combination with metformin. In general, the percentage of patients

who successfully ovulated was positive, and almost half of the group became clinically pregnant during the follow-up. The mean time to conception was not out of range, thus pointing out to the overall efficacy of the standard treatment regimens within this group, yet the results were not consistent (Table 5).

Additional examination showed that rising BMI was linked to a steady drop in both ovulation and pregnancy rates, which showed the negative impact of excessive weight on the results of reproductive functions. Moreover, hyperandrogenism was also associated with a relatively poorer response to treatment, and lower conception rates were found with affected individuals. These relationships were statistically significant, which indicates the impact of metabolic and endocrine factors on fertility results in PCOS patients (Table 6).

Table 1: Baseline Demographic and Clinical Characteristics (n = 165)

Variable	Mean ± SD / n (%)
Age (years)	27.8 ± 4.6
BMI (kg/m²)	29.4 ± 5.2
Duration of infertility (years)	3.6 ± 2.1
Type of Infertility	
Primary infertility	108 (65.5%)
Secondary infertility	57 (34.5%)
Menstrual History	
Regular cycles	48 (29.1%)
Oligomenorrhea	89 (53.9%)
Amenorrhea	28 (17.0%)
Clinical Hyperandrogenism	
Hirsutism	96 (58.2%)
Acne	74 (44.8%)

Table 2: Distribution of PCOS Phenotypes

Phenotype	Description	Frequency (n)	Percentage (%)
Phenotype A	HA + OD + PCO	68	41.2%
Phenotype B	HA + OD	34	20.6%
Phenotype C	HA + PCO	29	17.6%
Phenotype D	OD + PCO	34	20.6%

Table 3: Hormonal and Metabolic Profile

Parameter	Mean ± SD
LH (IU/L)	10.8 ± 3.5
FSH (IU/L)	6.2 ± 1.8
LH/FSH ratio	1.74 ± 0.62
Serum Testosterone (ng/dL)	72.5 ± 18.4
Fasting Insulin (µIU/mL)	14.9 ± 6.3

Table 4: Comparison of Hormonal Profile and Treatment Outcomes Among PCOS Phenotypes

Variable	Phenotype A (n=68)	Phenotype B (n=34)	Phenotype C (n=29)	Phenotype D (n=34)	p-value
Hormonal Profile					
LH (IU/L)	11.9 ± 3.4	11.2 ± 3.1	10.1 ± 2.8	8.9 ± 2.6	0.002
FSH (IU/L)	5.9 ± 1.6	6.1 ± 1.7	6.4 ± 1.9	6.8 ± 2.0	0.081
Testosterone (ng/dL)	82.3 ± 15.2	78.6 ± 14.9	75.4 ± 13.6	52.1 ± 10.5	<0.001
Fasting Insulin (µIU/mL)	17.8 ± 6.5	16.3 ± 5.8	13.5 ± 4.9	11.2 ± 4.1	<0.001
Treatment Outcomes					
Ovulation achieved n (%)	45 (66.2%)	23 (67.6%)	25 (86.2%)	29 (85.3%)	0.018
Clinical pregnancy n (%)	27 (39.7%)	13 (38.2%)	19 (65.5%)	19 (55.9%)	0.021
Time to conception (months)	4.8 ± 2.0	4.5 ± 1.8	3.6 ± 1.5	3.9 ± 1.6	0.034

Table 5: Treatment Modalities and Overall Treatment Outcomes (n = 165)

Variable	Frequency (n)	Percentage (%) / Mean ± SD
Treatment Type		
Letrozole	72	43.6%
Clomiphene Citrate	54	32.7%
Letrozole + Metformin	25	15.2%
Clomiphene + Metformin	14	8.5%
Treatment Outcomes		
Ovulation achieved	122	73.9%
No ovulation	43	26.1%
Clinical pregnancy	78	47.3%
Time to conception (months)	—	4.2 ± 1.9

Table 6: Association of BMI and Hyperandrogenism with Treatment Outcomes.

Variable	Ovulation n (%)	Pregnancy n (%)	p-value
BMI Category			
Normal (<25)	32 (88.9%)	24 (66.7%)	
Overweight (25–29.9)	46 (75.4%)	30 (49.2%)	
Obese (≥30)	44 (66.7%)	24 (36.4%)	0.027
Hyperandrogenism			
Present	68 (70.8%)	40 (41.7%)	
Absent	54 (78.3%)	38 (55.1%)	0.049

DISCUSSION

The current study has shown that most of the infertile women with PCOS conformed to the classical phenotype, and then other phenotypic variants, which indicates a high proportion of more severe clinical manifestations. This observation aligns with the cross-sectional study of Abdalla Moustafa Elsayed et al. (2023) that found that phenotype A was the one with the strongest endocrine and reproductive disturbances in comparison with other phenotypes. Their analysis also highlighted that phenotype A generally correlates with an increase in androgen levels, menstrual abnormalities, and infertility, which correlates with its increased prevalence and clinical severity.[13] Nevertheless, there have been some regional studies that have associated a comparatively higher proportion of milder phenotypes, implying the possibility of ethnic and environmental influences on the distribution of phenotypes.

Hormonally, our research showed that luteinizing hormone, testosterone, and insulin were significantly higher in the more severe of the phenotypes as opposed to the milder phenotypes. The results are consistent with the research conducted by Zaixin Guo et al. (2023), which noted that hormonal and metabolic imbalances, especially hyperinsulinemia and hyperandrogenism, are the major predictors of poor response in ovulation among PCOS patients undergoing treatment.[14] On the same note, reproductive outcomes were also reported to be strongly linked with high levels of androgens and insulin resistance, which, in our case, supports our findings that the severity of phenotypes is related to biochemical derangement.

In response to treatment, our results showed that ovulation and pregnancy rates were much better in less severe phenotypes, especially those not with marked hyperandrogenism. This is heavily substantiated by the recent prospective cohort study by Yavuz Emre Şukele et al. (2026), revealing that treatment effectiveness differs greatly among PCOS phenotypes, with higher ovulation rates in patients with less severe hyperandrogenism.[15] They also noted better results with letrozole over clomiphene citrate, especially in hyperandrogenic patients, which is consistent with our report of overall good ovulation rates with letrozole-based programs.

Our study recorded similar ovulation and pregnancy rates as Priyanka Sharma et al. (2023), who reported a better fertility rate of ovulation induction therapies, especially with combination regimens.[16] Their conclusions corroborate our results that conventional treatment regimes can deliver adequate results, but there is still variability based on patient characteristics. Additionally, the results of research assessing the efficacy of assisted reproductive methods have also demonstrated that letrozole enhances follicular growth and pregnancy success, especially in overweight PCOS patients, which supports the validity of the treatment procedures adopted in our cohort study.[17]

A key result of our research was the adverse effect of the rising BMI on the outcomes of treatment. There was a decreased ovulation and pregnancy rates in obese patients as compared to normal-weight patients. This is in line with the findings of several recent studies which show that obesity worsens insulin resistance and hormonal imbalance leading to the ultimate impairment

of fertility in PCOS patients. To illustrate, it has been studied that weight-related metabolic disturbances are the main factors that diminish the responsiveness to ovulation induction and delay conception, and which indicate the need to manage weight in enhancing reproductive success.

Also, our research determined that hyperandrogenism was linked with worse treatment outcomes. This finding agrees with the current evidence that high levels of androgen adversely affect follicular growth and ovulatory response. This association is also supported by the study of Yavuz Emre Şuktur et al. where the authors showed that the success of the treatment was lower in patients with higher levels of androgen concentration in their bodies.[15] The results support the importance of hyperandrogenism as an important determinant of treatment response in PCOS.

The heterogeneity of PCOS and clinical implications is another crucial point that our results reveal. As with our results, recent literature highlights that PCOS is not a single homogenous condition but a continuum of phenotypes that have different metabolic and reproductive phenotypes. It has been demonstrated repeatedly that phenotype-specific strategies can enhance the results of treatment and minimize unnecessary interventions, which is why our study is rational.

Further, a study has found that ethnicity, baseline hormonal status, and metabolic profile play an important role in determining the treatment outcome in PCOS. Indicatively, a study comparing ovulation induction in diverse populations displayed inconsistency in the rates of response indicating that personalized treatment plans are crucial to the best outcomes.[18] This also validates our results that patient-specific variables, such as phenotype, BMI, and hormone profile are instrumental in influencing treatment success.

Translational and experimental studies have also been useful in learning the pathophysiology of PCOS and its repercussions on fertility. As an example, animal models have recently demonstrated that metabolic interventions can revert hormonal and reproductive abnormalities in PCOS, again highlighting that metabolic regulation can be used to enhance fertility outcomes.[19] Even though these results are preclinical, they offer biological plausibility of the associations that we found in our study.

Generally, the results of the current paper correspond to the increasing body of research that emphasizes the significance of PCOS phenotypes in clinical expression, hormonal patterns, and response to treatment. Although our findings are consistent with the majority of the recent literature, there can be slight differences in the distribution of phenotypes and treatment results which could be explained by the differences in the populations of the study, sample size and clinical protocol. Together, these results highlight the importance of a phenotype-driven, personalized approach when addressing infertility in PCOS women.

There are limitations to this study that must be taken into consideration. As a single-center study with a small sample size, the extrapolation of findings to the broader population is limited. Selection bias can also be introduced by non-probability consecutive sampling. More studies are needed in the direction of insulin resistance and long follow-up is needed to see long term metabolic and treatment effects.

CONCLUSION

Finally, the current paper demonstrates that PCOS remains a heterogeneous disease, and phenotypic differences play a large role in determining the outcome of hormonal profile and infertility treatment. Milder phenotypes were found to exhibit relatively higher ovulation and pregnancy rates compared to classical phenotypes with marked hyperandrogenism and metabolic disturbance that exhibited lower responsiveness to treatment. Additional contributors of poor reproductive outcomes were higher BMI and hyperandrogenism. These results highlight the need to adopt an individualized approach to infertility management in women with PCOS that relies on phenotype to enhance the likelihood of treatment success and clinical outcomes.

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