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ORIGINAL RESEARCH

Impact of Polycystic Ovary Syndrome Hyperandrogenic Phenotypes A and Non-Hyperandrogenic D on Pregnancy Outcomes After in vitro Fertilization (IVF)/Intracytoplasmic Sperm Injection (ICSI)

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Objective: This study aims to evaluate the association between polycystic ovary syndrome (PCOS) phenotypes A and D with primary and secondary pregnancy outcomes among PCOS vs non-PCOS patients after in-vitro fertilization (IVF)/intracytoplasmic sperm injection (ICSI).

Methods: A retrospective cohort design using data of eligible women aged 20–40 years who had their first IVF treatment. The data were obtained from medical records. The women were either diagnosed with PCOS according to Rotterdam Criteria, namely phenotypes A and D or non-PCOS. Pregnancy outcome including (1) the rate of biochemical pregnancy and (2) ongoing clinical pregnancy and (3) the rate of miscarriage were measured. Furthermore, secondary outcome data were collected on (1) the number of oocytes retrieved, (2) the number and percentage of mature oocytes, (3) fertilization rate, (4) the percentage of grade 1 embryos on day 3 and (5) the percentage of grade 1 embryos on day 5.

Results: A sample of 149 women was analyzed. For secondary pregnancy outcomes, PCOS women in groups A and D have lower oocyte maturity rate and lower fertilization oocyte ratio, as well as fewer grade 1 oocytes on day 3 in comparison to the control. Primary pregnancy outcomes have significantly lower values among PCOS women in group A with respect to biochemical pregnancy and ongoing clinical pregnancy and higher miscarriage rates in comparison to group D and the control group. Finally, group D and control did not differ significantly in the primary pregnancy outcomes.

Conclusion: Patients with phenotype D have better IVF outcomes in comparison to phenotype A. Phenotype A, affects the IVF outcomes negatively possibly due to hyperandrogenism.

Keywords: polycystic ovary syndrome, IVF, ICSI, pregnancy outcomes, oocytes, hyperandrogenism

Introduction

A common endocrine and metabolic condition affecting women of reproductive age is polycystic ovarian syndrome (PCOS), as it manifests as infertility, hyperandrogenism (HA), and menstrual disruption (oligomenorrhoea).[1] For women with PCOS who are not trying to conceive, cyclic progestogens or the combined oral contraceptive pill can help them get back on track with their monthly cycles. Anti-androgens can be used to treat HA. PCOS can significantly impact fertility and the outcomes of assisted reproductive technologies such as in vitro Fertilization (IVF).[2] Ovulation induction and

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lifestyle modifications are the main forms of treatment for infertile people. Nonetheless, there are instances in which infertility brought on by a male factor or an inability to trigger ovulation calls for IVF.

Since 1990, there have been modifications to the PCOS diagnosis National Institutes of Health criteria. Before the advent of ultrasonography technology, the diagnosis of PCOS was based on the presence of two criteria: HA and irregular menstruation. After late-onset congenital adrenal hyperplasia, Cushing's syndrome, androgen-secreting tumors, and hyperprolactinemia have been ruled out, the most recent and widely used assessment tool for PCOS is the Rotterdam criteria; however, it requires the presence of at least two of the three manifestations: polycystic ovarian appearance on ultrasound, clinical and/or biochemical HA, and oligomenorrhoea.[3] Thus, since the introduction of Rotterdam consensus on diagnosis of PCOS four different PCOS phenotypes emerged[4]:

Phenotype A: HA, oligomenorrhoea, and polycystic ovarian morphology (PCOM).

Phenotype B: HA, oligomenorrhoea but with normal ovaries.

Phenotype C: HA and PCOM but regular ovulatory cycle.

Phenotype D: Oligomenorrhoea, PCOM but no clinical or biochemical HA.

It is critical to mention the 2003 Rotterdam criteria are considered in need of an update; furthermore, the name PCOS has come into question in recent years, as evidenced by the new classification proposed by EGOI-PCOS scientific society. Particularly, it is aimed to differentiate the hyperandrogenic patients (A, B, C) into type 1, type 2, and type 3 endocrine- metabolic syndrome, and non-hyperandrogenic patients (formerly phenotype D) as true PCOS.[5] This new classification steers the incorporation of insulin resistance as a diagnostic parameter. While it has often been excluded in various classifications over the years, insulin resistance plays a predominant role in the etiopathogenesis of hyperandrogenic phenotypes.[6,7]

The prevalence of these phenotypes tends to vary between countries given that regional variables and ethnic/racial variances might influence how the condition manifests clinically. Using the Rotterdam 2003 criteria, the prevalence of PCOS varies from 6% to 21% worldwide.[1,8] According to available data, approximately two-thirds of PCOS cases identified in clinical settings are the typical form of PCOS, phenotypes A and B. However, according to the few studies that studied PCOS phenotypes, distribution across unselected, medically impartial population indicates that phenotypes B and C could account for about two-thirds of PCOS patients seen, while phenotypes A and D are nearly equally common.[8] In Jordan, very few studies on PCOS have been published. According to a study of 159 PCOS-afflicted infertile women in Jordan, the results showed that type A (50.3%) was the most common phenotype, followed by type C (29.6%), type B (14.5%), and type D (5.7%).[9] Thus, the distribution of PCOS phenotypes among Jordanian women revealed that Phenotype A was the most prevalent, which aligns with findings in other populations.

Obesity, defined as BMI >30 kg/m^2, is commonly present in PCOS 33–70% depending on the population studied. One study showed that insulin resistance measured by the homeostasis model assessment-estimated insulin resistance (HOMA-R), a feature of PCOS increased from 37.7% to 75.8% as BMI increased.[10] Response to ovarian stimulation seems to be affected by the phenotype of PCOS, as the number of oocytes and risk of ovarian hyperstimulation syndrome (OHSS) is higher in phenotype A and B[11], which is mainly due to the high ovarian reserve by having the highest levels of anti-Mullerian hormone (AMH).[12,13]

A recent study from Turkey on PCOS phenotypes and IVF outcome reported significantly higher numbers of oocytes retrieved in phenotype A and D compared to patients in the control group.[14] Regarding the ratio of Metaphase II oocytes to implantation rates, there was no statistically significant variation observed across the groups. Patients in phenotype A had a much reduced number of high-quality embryos on transfer day; nonetheless, all groups' rates of pregnancy were determined to be identical in terms of biochemical, clinical, and ongoing pregnancy outcomes. They came to the conclusion that HA had no detrimental effects on the success of IVF.[14] Hyperandrogenic PCOS phenotypes had a reduced cumulative pregnancy rate, according to another study by De Vos et al,[15] while a study by Hu et al revealed HA may have a negative impact on conception rates.[16]

Studies on the effects of PCOS on pregnancy outcomes are quite limited. Therefore, we carried up this retrospective analysis to examine (1) the distribution of various PCOS phenotypes among Jordanian women, and (2) the impact of the unfavorable pregnancy outcomes in women with PCOS by comparison with the control group, in order to facilitate a more

accurate assessment of the risks of pregnancy difficulties in these women. Our study was to determine the impact of PCOS, namely phenotypes A and D, which are prevalent in Jordan, on the success rate of IVF and its outcomes among these women.

Methods

Study Setting and Participants

The study used a retrospective cohort design using data from the Arab Medical Centre for IVF, a gynecology and fertility clinic in Amman, Jordan, of eligible women aged 20–40 years who had their first IVF treatment between January 2018 till December 2019. The data were obtained from medical records. We excluded couples with male factor infertility, women aged <20 years old as it is difficult to make diagnosis of PCOS in this group due to the adolescent rise in antral follicles[17] and women aged >40 years old.

The study population were divided into the PCOS group and the non-PCOS group. The women were either diagnosed with PCOS according to Rotterdam Criteria,[18] namely phenotypes A (group A) and D (Group D), or non-PCOS (Control). It is important to note that the new proposed classification to differentiate the hyperandrogenic patients (A, B, C) as type 1, type 2, and type 3 endocrine – metabolic syndrome, and non-hyperandrogenic patients (formerly phenotype D).[5,6]

PCOS Diagnosis According to Rotterdam Criteria

Data on women who underwent their first IVF cycle were extracted from the notes in the Arab Medical Centre IVF. The diagnosis of PCOS was made according to Rotterdam criteria if patients meet two or three of the following criteria:

1. The presence of oligomenorrhoea with cycle more than 35 days long or serum progesterone level <3 ng/mL on day 18–21 of cycle or of amenorrhoea as in absence of menses in the last 6 or more months.
2. Biochemical HA confirmed by an elevated serum testosterone >2.2 nmol/L) and/or serum Androstenedione >3.8 ng/mL and/or clinical HA a Ferriman Gallwey score >8.[19]
3. Transvaginal ultrasound scan showing the presence of 12 or more peripheral antral follicles 2–8 mm in diameter in either ovary and/or increased ovarian volume of each ovary >10 mL.[20]

However, according to the newly proposed classifications, the inclusion of insulin resistance as a diagnostic parameter is critical as it plays a major role in the etiopathogenesis of the hyperandrogenic phenotypes (formerly known as A, B, C) into type 1, type 2, and type 3 endocrine–metabolic syndrome in comparison to the non-hyperandrogenic phenotypes (formerly phenotype D).

IVF and Embryo Transfer Protocols

A complete record of the IVF protocol was obtained as well as age, BMI, oocyte collection and pregnancy outcomes. All patients included underwent IVF cycles with a standard luteal phase down-regulation protocol using a GnRH antagonist. The starting dose of gonadotropin and the GnRH antagonist were chosen at the physician's discretion. Ovarian response was monitored via serum E2 levels and transvaginal ultrasound. According to the follicular response and serum E2, the physician would decide on the final oocyte maturation trigger shot by either with an injection of human chorionic gonadotropin (hCG) or GnRH analogue when at least two follicles reached 17–18 mm. Oocyte retrieval was performed 36 hours after the hCG injection using transvaginal ultrasound guidance. All cases underwent intracytoplasmic sperm injection (ICSI). Embryo quality was assessed by two experienced embryologists based on the size and number of blastomeres and the degree of fragmentation and were graded based on Graduated Embryo Score (GES) criteria.[21] Ultrasound-guided fresh embryo transfer took place on either day 3, 4 or 5 post-fertilization.

Measurement of Outcomes

Pregnancy outcome including (1) the rate of biochemical pregnancy and (2) ongoing clinical pregnancy and (3) the rate of miscarriage were collected and measured and labeled as primary outcome measurements. The presence of a gestational sac at 7–8 weeks of gestation using ultrasound was defined as ongoing clinical pregnancy. There were two types of miscarriages included: early miscarriage, which occurred before 12 weeks of gestation, and late miscarriage, which occurred between 12 and 28 weeks of gestation.

Furthermore, secondary outcome measurements' data were collected on (1) the number of oocytes retrieved, (2) the number and percentage of mature oocytes, (3) fertilization rate which was calculated as percentage of oocytes that were fertilized 24 hours after ICSI, (4) the percentage of grade 1 embryos on day 3 and (5) the percentage of grade 1 embryos on day 5.

Ethical Considerations

The study protocol was approved on July 15 2024 by the Institutional Review Board (IRB) of Al Balqa Applied University, Al-Salt, Jordan, with approval reference number: 2024/2023/7/66. Due to the retrospective nature of the study, the requirement of informed consent was waived. The privacy of all patients was protected and data was collected anonymously keeping patients' data confidential, while no identifying information was used in the analysis or distribution of the results, and all protocols complied with the Declaration of Helsinki's ethical standards and regulations.

Statistical Analysis

Statistical analysis will be performed using SPSS version 20 (SPSS Inc., Chicago, IL, USA). Continuous variables are presented as the mean ± SD, whereas categorical variables are shown as the number of cases and percentages. Mean differences between the two groups were compared using Student's *t* test or the Mann–Whitney test, whichever was applicable. *P*-values <0.05 were considered statistically significant.

Results

Patient Clinical Characteristics

A total of 149 women aged 20–40 years undergoing their first IVF treatments between January 2018 and December 2019 met the criteria and were enrolled in this cohort study, with 52 women diagnosed with PCOS phenotype A, 47 women diagnosed with PCOS phenotype D and 49 controls. The women were diagnosed with PCOS mainly patients with phenotype A has higher BMI and hyperandrogenic, while patients with phenotype D had lower BMI and non-androgenic type. Results are summarized in Tables 1 and 2. The range of BMI in PCOS women in this study was 17–40 kg/m^2. For phenotype A, there were 73.6% (39/53) PCOS women with BMI >25 kg/m^2 and 26.4% (14/53) PCOS women with BMI ≤25 kg/m^2, while for phenotype D, there were 46.8% (22/47) PCOS women with BMI >25 kg/m^2 and 53.2% (25/47) PCOS women with BMI ≤25 kg/m^2, similar to the range of control group. Compared to the control group, phenotype A had higher BMI (29.38 vs 26.59; *P*=0.01).

Table 1 Clinical Characteristics, Primary and Secondary Pregnancy Outcomes of Women with PCOS Phenotype A and D vs Controls During Their First IVF Treatment Cycle

Variables	Group A (n = 53)	Group D (n = 47)	Control (n = 49)
Maternal Age (y)	28.13 ± 4.76 (20–38)	27.36 ± 4.38 (21–40)	29.67 ± 4.22 (23–40)
Maternal BMI (kg/m^2)	29.38 ± 5.03 (17–40)	26.59 ± 4.40 (19–37)	27.09 ± 3.67 (22–38)
Primary outcome			
Biochemical pregnancy	48.7% (20/41)	51.3% (20/39)	54.2% (26/47)
Ongoing Clinical pregnancy	26.8% (11/41)	41.0% (16/39)	43.8% (21/47)
Miscarriages rates	45.0% (9/20)	20.1% (4/20)	19.2% (5/26)
Secondary outcome			
Oocyte number	22.23 ± 9.48 (10–44)	21.83 ± 8.73 (10–44)	11.74 ± 4.57 (2–23)
Oocyte maturity rate	74.7% ± 0.10	71.8% ± 0.07	84.4% ± 0.12
Fertilized oocyte ratio	73.5% ± 0.10	74.8% ± 0.09	84.7% ± 0.11
Grade 1 oocyte, day 3	57.9% ± 0.12	60.1% ± 0.11	67.8% ± 0.16
Grade 1 blastocyst	24.6% ± 0.11	29.6% ± 0.12	32.8% ± 0.18

Notes: Values are numbers or percentages of cases, mean ± standard deviation. BMI, body mass index. Group A is PCOS phenotype A patients; group D is PCOS phenotype D patients and control is non-PCOS patients. Bold represents the groups and the list of variables analyzed.

Table 2 One-Way ANOVA Analysis of Clinical Characteristics, Primary and Secondary Pregnancy Outcomes of Women with PCOS Diagnosis in Groups A and D Versus Controls

Variables	Groups		
	A vs C	D vs C	A vs D
Maternal Age (y)	0.087	0.010*	0.404
Maternal BMI (kg/m^2)	0.010*	0.549	0.004*
Primary outcome			
Biochemical pregnancy	0.026*	0.205	0.048*
Ongoing Clinical pregnancy	0.022*	0.221	0.028*
Miscarriages rates	0.032*	0.251	0.048*
Secondary outcome			
Oocyte number	<0.001*	<0.001*	0.829
Oocyte maturity rate	<0.001*	<0.001*	0.101
Fertilized oocyte ratio	<0.001*	<0.001*	0.505
Grade 1 oocyte, day 3	<0.001*	0.006*	0.330
Grade 1 blastocyst	0.006*	0.308	0.035*

Notes: Statistical significances were considered as $p<0.05$, and noted with *. BMI, body mass index. Group A is PCOS phenotype A patients; group D is PCOS phenotype D patients and control is non-PCOS patients. Bold represents the groups and the list of variables analyzed.

To prevent ovarian hyperstimulation syndrome in PCOS women groups A and D, freeze all strategy was done where all embryos were frozen and no fresh embryo transfer was performed in 12 and 8 cases, respectively, while only in one case in the control group. Furthermore, one case in the control group had no embryo transfer due to absence of grade 1 oocytes on day 3.

Secondary Pregnancy Outcomes

In the present study, Tables 1 and 2 show PCOS women, in both phenotypes A and D, had a trend toward more oocytes retrieved (~22 vs 12; $p<0.001$). However, PCOS women in groups A and D has lower oocyte maturity rate (~73% vs 84%; $p<0.001$) and lower fertilization oocyte ratio (~74% vs 84%; $p<0.001$), as well as fewer grade 1 oocytes on day 3; (~59% vs 68%, $p<0.001$) in comparison to the control. Interestingly, the percentage of grade 1 blastocyst did not differ significantly between group D and control ($p=0.308$), while group A has significantly lower percentage in comparison (vs C; $p=0.006$ and vs D; $p=0.035$).

Primary Pregnancy Outcomes

The pregnancy rate was calculated from cases who had fresh embryo transfer, and not from subsequent frozen embryo transfer cycles. PCOS women in group A had significantly lower biochemical pregnancy (48.7% vs 51.3%,), lower ongoing clinical pregnancy (26.8% vs 41.0%) and higher miscarriage rates (45.0% vs 20.1%), as summarized in Table 1 and Table 2, in comparison to group D ($p=0.048$, $p=0.028$, $p=0.048$, respectively). The same significant pattern was detected when comparing PCOS women in group A and the control group (48.7% vs 54.2%, 26.8% vs 43.8%, 45.0% vs 19.2%, respectively; $p=0.026$, $p=0.022$, $p=0.032$, respectively). Finally, group D and control did not differ significantly in the primary pregnancy outcomes.

Discussion

In the present study, we found that the full-blown PCOS type A had higher obesity and worst primary pregnancy outcomes in comparison to phenotype D, the least severe type, and the control group. Being overweight is seen as a common metabolic phenotype and is associated with ovulatory, metabolic, and HA problems.[22] Thus, it is essential for

overweight or obese women with PCOS to adopt a proper lifestyle focused on a balanced diet and regular physical activity, as these measures can significantly improve both metabolic and reproductive outcomes.[23,24] Particularly, the consumption of a very-low-calorie ketogenic diet as nutritional therapy in the management of overweight/obese PCOS patient candidates for IVF showed promising results.[25] Furthermore, a high BMI associated with PCOS appears to be linked to the higher risk of unfavourable pregnancy outcomes seen in women with PCOS undergoing IVF.[26,27] Consistent with these findings, the overweight phenotype A exhibited worst primary pregnancy outcome. There were no significant differences between PCOS phenotype D and non-PCOS group in terms of biochemical, ongoing clinical pregnancies and miscarriage rates.

We propose the culprit for having higher miscarriage rate and lower biochemical and ongoing clinical pregnancies in phenotype A compared to phenotype D is the negative effect of HA, as numerous previous publications indicated. A meta-analysis of IVF in patients with PCOS showed a linked of HA on the pregnancy outcomes, with increased miscarriage rates in phenotype A, especially amongst Asian ethnicity.[28] In addition, compared to patients without HA, hyperandrogenic patients with phenotype A had a noticeably inferior outcome from IVF compared to phenotype D.[14,29]

It is important to highlight the different etiopathogenesis of Phenotype A and Phenotype D in PCOS, and how these differences could justify their varying outcomes in ART. The full-blown PCOS phenotype A, which is typified by polycystic ovarian morphology, hyperandrogenism, and ovulatory dysfunction, is the classic presentation of PCOS. Insulin resistance, hyperinsulinemia, and dysregulation of the hypothalamic-pituitary-ovarian (HPO) axis are among the metabolic and endocrine changes that comprise the underlying etiopathogenesis.[30] Chronic anovulation and high androgen production are caused by these hormonal and metabolic abnormalities. These patients are frequently less receptive to ART due to insulin resistance and metabolic dysfunction, which also increases their risk of ovarian hyperstimulation syndrome (OHSS), lowers implantation rates, and results in worse oocyte quality.[31,32]

On the other hand, phenotype D, also referred to as "non-hyperandrogenic PCOS" is characterized by ovulatory dysfunction and polycystic ovarian morphology but lacks hyperandrogenism and significant metabolic alterations such as insulin resistance. Because Phenotype D does not include the same level of endocrine-metabolic disruption, its etiopathogenesis is unique. Rather than systemic insulin resistance or hyperinsulinemia, the disorder may result from intrinsic ovarian malfunction or minor anomalies in folliculogenesis. IGF-1 (insulin-like growth factor 1) levels are much higher than normal, leading to the arrest of follicle maturation. A recent study published by Dai et al using mouse models showed treatment with increasing doses of IGF-1 on healthy mice (without endocrine-metabolic alterations) exhibited follicular maturation arrest when stimulated with the highest dose.[33] These individuals typically have better ART outcomes, such as higher oocyte quality, enhanced endometrial receptivity, and better implantation rates, because they do not have the complicating complication of metabolic dysfunction. Thus, the stark differences in the etiopathogenesis of these two phenotypes largely explain their divergent ART outcomes and thus, requiring different therapy approaches. For instance, managing insulin resistance and hyperandrogenism is crucial to improving ART success for phenotype A, while the absence of systemic metabolic disturbances allows for a more favorable response to ART, often requiring less aggressive treatment protocols for non-hyperandrogenic phenotype D. Tailoring ART protocols based on the specific phenotype and its underlying etiopathogenesis can optimize outcomes and minimize risks for women with PCOS.

Numerous studies have been conducted on the metabolic perspective of PCOS, and published research has found connections between hyperinsulinemia, insulin resistance (IR), and metabolic syndrome (MS).[34] This is especially true for phenotypes A, where a possible correlation between HA and abnormalities in metabolism has been observed and ultimately linked to infertility and unfavorable IVF outcomes.[35] Tripathi et al's study of the prevalence of MS in the PCOS phenotypes showed phenotype D having the lowest levels—provides evidence in favor of this theory.[36]

This is not uniformly reported across all studies, where some showed both phenotypes A and D having significant upsurge in the rates of ectopic pregnancy, miscarriage, and preterm pregnancy compared to the control group.[15,37] This indifference among phenotype A and D is seen in our study regarding the secondary pregnancy outcomes, as there were no significant differences between the two phenotypes of PCOS groups, in terms of the number of oocytes retrieved, maturity rate, fertilization rate and the number of day 3 grade 1 oocyte. A meta-analysis of 29 studies by She et al showed women with PCOS having similar pregnancy-related outcomes and complication rates compared with women without PCOS.[38] A limitation of this systematic analysis is they were unable to conduct additional analysis to assess the impact of

PCOS phenotypic variants. One of the few studies that have assessed the results of IVF among the various PCOS phenotypes of women,[39] with variations in the embryo cryopreservation technique and the embryo transfer stage revealed a fourfold increase risk for obstetric or neonatal complication due to ovarian dysfunction and biochemical hyperandrogenism among PCOS women. Therefore, to determine whether pregnancy outcomes and complications differ between PCOS and other causes of infertility after IVF treatment, more carefully planned, prospective studies with more detailed data, including the techniques used for assisted treatment as well as detailed treatment protocols, are required.

Fertility issues are unfortunately common among PCOS patients, thus selecting the appropriate therapy is critical to meeting the patient's demands. Patients with PCOS undergoing ovarian stimulation often result in varied clinical outcomes because of the diverse hormone profiles associated with different PCOS phenotypes and inconsistent progesterone resistance.[40] As a result, the impact of PCOS phenotypes on fertility treatment should be considered when advising PCOS patients who are attempting to conceive. The etiopathogenesis of PCOS differ among phenotypes. To be concise, phenotypes A, B, and C, there is an endocrine-metabolic alteration, with insulin resistance and hyperandrogenism present in 75% of patients (3 out of 4 women).[41,42] In contrast, for Phenotype D, where these two conditions are absent, the cause could be an alteration in the IGF-1 signalling pathway that blocks follicle maturation, resulting in menstrual cycle disturbances.[41,42] It is exactly in consideration of these fresh perspectives that we need to assign the suitable therapeutic rationale which cannot overlook an appropriate lifestyle. If we are managing a hyperandrogenic patient with insulin resistance, a treatment utilizing an insulin-sensitizing agent (myo-inositol) will be effective: this will diminish insulin resistance, thereby reducing male hormones, resulting in a recovery of ovarian function and the reinstatement of the menstrual cycle as suggested in the study by Unfer et al.[6] Regarding phenotype D, the therapeutic rationale shifts completely, as these potentially have disruption in the IGF-1 signalling pathway, which could an alternative therapy.[41]

Our current study lacked the report on live birth rate which due to the difficulty of tracking patients after their clinical pregnancy was confirmed. Furthermore, the pregnancy rate was calculated from cases who had fresh embryo transfer, but not from subsequent frozen embryo transfer cycles, thus this study did not measure cumulative pregnancy rate, and this is considered a limitation. On the other hand, our results were based on a carefully selected population of patients with PCOS who had no known risk factors for a complicated pregnancy.

In conclusion, the current study validates that PCOS is a composite risk factor for the success of IVF and pregnancy outcomes. Depending on the various PCOS phenotypes and characteristics that each woman has, there are wide variations in the increased risk for adverse pregnancy results and IVF results. To validate the current findings, a larger, prospectively planned studies are necessary for future research in order to confirm and broaden the existing conclusions about the influence of PCOS on IVF results, especially in relation to the rarer PCOS phenotypes B and C. Deeper insights into individualized treatment plans and better patient care would result from such studies, which would offer a more thorough understanding of the condition's impact on reproductive success. Finally, for women with PCOS phenotypes A and D, individualized treatment during assisted reproduction and close follow-up after clinical pregnancy are necessary.

Data Sharing Statement

The data presented in this study are available on request from the corresponding author due to ethical reasons to ensure privacy and confidentiality of the patients and their information.

Informed Consent Statement

Patient consent was waived due to the retrospective nature of the study.

Institutional Review Board Statement

The study was conducted and approved by the Institutional Review Board of Al Balqa Applied University, Al-Salt, Jordan (protocol code 2024/2023/7/66 approved on July 15, 2024).

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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