Original Article

Impact of Polycystic Ovary Syndrome on Fertility and Pregnancy Outcomes: A Prospective Cohort Study

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Abstract

Objective: To assess the impact of PCOS on fertility and pregnancy outcomes by evaluating time to conception, ovulation rates, and pregnancy complications including miscarriage, gestational diabetes mellitus, preeclampsia, preterm birth and neonatal outcomes.

Methodology: This prospective study was conducted in Saidu Teaching Hospital, Swat from January 2021 to December 2021. Participants were enrolled aged 18–40 years who were either diagnosed with PCOS (based on the Rotterdam criteria) or seeking prenatal care/reproductive treatment without PCOS were enrolled. A total of 300 patients were enrolled. Women of comparable age in the control group did not have PCOS and were seeking prenatal care or reproductive treatment. Women with additional endocrinologic conditions (thyroid dysfunction, Cushing's syndrome), type II diabetes mellitus or hypertension, repeated or unexplained pregnancy loss in the past, and women using assisted reproductive technologies (ART) for purposes other than inducing ovulation were excluded. Data were analyzed using SPSS version 25. Descriptive statistics were used to summarize the data, while chi-square tests and t-tests were conducted for group comparisons.

Results: The PCOS group had a significantly longer time to conception (9.2 ± 4.5 vs. 4.6 ± 2.3 months, $p < 0.001$) and lower ovulation rates (61.3% vs. 89.4%, $p < 0.001$) compared to controls. Pregnancy outcomes showed higher rates of miscarriage (22.7% vs. 10.7%, $p = 0.008$), GDM (27.3% vs. 12.0%, $p = 0.014$), preeclampsia (14.7% vs. 6.0%, $p = 0.022$), and preterm birth (18.0% vs. 8.0%, $p = 0.019$) in the PCOS group. Neonatal outcomes revealed lower birth weights (2.89 ± 0.6 vs. 3.14 ± 0.5 kg, $p = 0.031$) and higher NICU admissions (16.0% vs. 7.3%, $p = 0.027$) in the PCOS group.

Conclusion: PCOS significantly impacts a woman's fertility and pregnancy by increasing the chances of prolonged time to conception, miscarriage, gestational diabetes mellitus, preeclampsia, and preterm birth. Optimizing reproductive health in women with PCOS involves early diagnosis with targeted interventions coupled with a personalized approach.

Key words: PCOS, GDM, fertility, pregnancy, miscarriage, diagnosis.

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Introduction

One of the most prevalent endocrine disorders, polycystic ovarian syndrome (PCOS) is estimated to affect 6–12% of women of reproductive age globally.[1] A variety of metabolic disorders and anovulatory infertility are associated with polycystic ovarian syndrome (PCOS), which is defined by lobulated ovarian morphology and hyperandrogenism.[2] Due to its complex causes and risk factors, polycystic ovarian syndrome (PCOS) has a significant impact on the endocrine system and major challenges to be addressed regarding fertility and pregnancy outcomes.[3] Polycystic ovarian syndrome (PCOS) causes prolonged anovulation, making 70–80% of women infertile.[4] As a result, PCOS has a detrimental effect on fertility unequivocally.

Obesity and nearly all types of elevated insulin levels increase the risk of ovulatory and reproductive failure. ([5]). Additionally, PCOS increases the risk of miscarriage, GDM, hypertension, and preterm birth[6] during pregnancy. Several adverse pregnancy outcomes have been linked to unbalanced metabolism and inflammation that actively promote a bad uterine environment before conception and the care of the fetus after conception.[7]

Despite advances in understanding the pathophysiology of PCOS, its association with infertility and adverse

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pregnancy outcomes remains incompletely understood. Recent studies suggest that epigenetic modifications, oxidative stress, and impaired endometrial receptivity contribute to these complications.[8] For instance, oxidative stress markers such as malondialdehyde (MDA) are significantly elevated in women with PCOS, correlating with poorer reproductive outcomes. Additionally, epigenetic changes, such as DNA methylation in genes related to insulin signaling and steroidogenesis, have been implicated in the pathogenesis of PCOS.[9] These factors, combined with the heterogeneity of PCOS phenotypes, complicate the establishment of universal diagnostic criteria and treatment protocols. For example, studies report that up to 70% of women with PCOS experience infertility, and approximately 40-50% of pregnancies in this population are associated with complications such as gestational diabetes mellitus (GDM) and preeclampsia.[10] This variability underscores the need for tailored diagnostic and therapeutic approaches to address the unique needs of women with PCOS.[11]

This study aims to evaluate the impact of Polycystic Ovary Syndrome (PCOS) on fertility and pregnancy outcomes through a prospective cohort design. By examining mechanisms such as hormonal imbalances, oxidative stress, and endometrial receptivity, as well as intersecting risk factors, the study seeks to provide evidence-based insights into the clinical effects of PCOS. The findings will contribute to the development of targeted interventions and personalized treatment strategies to improve reproductive health outcomes for women with PCOS.

Methodology

This prospective cohort study was conducted between January 2021 to December 2021, at Gyne & Obstetrics department, Saidu Teaching Hospital Swat. The sample size was determined using the WHO Sample Size Calculator for cohort studies, working with a power of 80% (β=0.20) and significance level of 5 % (α=0.05). As set forth by earlier literature, the calculations set out an anticipated variation in miscarriage rates of about 20 % between the control groups (PCOS: 30%, Control: 10%). Based on these calculations, the minimum sample size estimated per group was 132 participants. Due to anticipated attrition and missing information, the final sample size was adjusted to 150 per group, equating to 300 participants in total.

Both groups were matched based on age, BMI, and baseline reproductive history to ensure comparability. Women in the control group were selected from those seeking prenatal care or reproductive treatment, with the absence of PCOS confirmed through clinical evaluation, menstrual history, and hormonal assessment, including serum androgen levels and ovulatory status. The hospital's Institutional Review Board (IRB) consented to the protocol prior to this study commencing (reference # 24/ERB/2022, dated 2-10-2022). Participants of the study gave their informed consent prior to enrollment as well. Women aged 18 to 40 years diagnosed with PCOS, based on the Rotterdam criteria, were included in the study.[11] Pregnancies were confirmed through serial β-hCG testing followed by transvaginal ultrasound visualization of intrauterine gestational sac with fetal pole. Women of comparable age in the control group did not have PCOS and were seeking prenatal care or reproductive treatment. Women with other endocrinologic conditions, such as Cushing's syndrome (verified by a 24-hour urine free cortisol or low-dose dexamethasone suppression test) and thyroid dysfunction (diagnosed based on TSH and free T4 values), were not included in the study. The American Diabetes Association's (ADA) criteria for type II diabetes mellitus were HbA1c ≥6.5% or fasting plasma glucose ≥126 mg/dL. Blood pressure ≥140/90 mmHg on two separate occasions or a history of antihypertensive medication use were used to rule out sustained hypertension. Additionally, excluded were women who have experienced recurrent pregnancy loss (≥2 consecutive unexplained miscarriages). Additionally, those who used assisted reproductive technologies (ART) for purposes other than ovulation induction were excluded.

Data on pregnancy outcomes (miscarriage, gestational diabetes mellitus GDM, preeclampsia, preterm birth, and neonatal outcomes) and fertility outcomes (time to conception, ovulation rates) were gathered from participants during their whole pregnancy. Infertility workup included hysterosalpingography/laparoscopy (tubal assessment), partner semen analysis, and baseline pelvic ultrasound to exclude non-PCOS causes. Ovulation induction followed a step-up protocol beginning with letrozole (2.5 mg days 3-7, increasing by 2.5 mg monthly up to 7.5 mg if no response). The following criteria were used to evaluate fertility outcomes: Time to conception (if any) between the start of ovulation induction and a healthy pregnancy. Ovulation rates: Ovulation rates are assessed using serum progesterone levels (>3 ng/mL, indicating ovulation) and serial transvaginal ultrasounds. Ovulation

induction was performed using letrozole (2.5-7.5 mg days 3-7) for PCOS patients and clomiphene citrate (50 mg days 3-7) for controls, with dose escalation every 3 cycles if no response. Transvaginal ultrasound

monitoring began on cycle day 10, with hCG trigger (250 mcg) administered when ≥1 follicle reached 18mm. Luteal support was provided with vaginal progesterone. Ovulation was confirmed by mid-luteal progesterone >10 ng/mL and follicular collapse on ultrasound. Treatment was limited to 6 cycles, with failure defined as either anovulation after 3 maximal-dose cycles or no conception after 6 ovulatory cycles. Pregnancy outcomes were described as follows: A miscarriage occurs when a pregnancy ends before 20 weeks of gestation while the fetus is still developing. According to the International Association of Diabetes and Pregnancy Study Groups' (IADPSG) recommendations, gestational diabetes mellitus (GDM) is diagnosed by a 75-g oral glucose tolerance test (OGTT) performed between weeks 24 and 28 of pregnancy. A diagnosis of GDM was made if fasting plasma glucose was ≥92 mg/dL, 1-hour plasma glucose was ≥180 mg/dL, or 2-hour plasma glucose was ≥153 mg/dL. To evaluate the independent association between PCOS and GDM, relative risk (RR) was calculated, and multivariate logistic regression was performed, adjusting for potential confounders such as age, BMI, and insulin resistance. Pre-eclampsia: It is characterized by proteinuria (≥300 mg/24 hours) and new-onset hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg) after 20 weeks of pregnancy. Giving birth before the 37th week of pregnancy is known as a preterm delivery. Neonatal outcomes include admission to the intensive care unit, birth weight, and Apgar scores.

All statistical analysis was performed using SPSS version 25.0. Continuous variables were described as mean ± SD, whereas categorical variables were described as frequency and percentage. The Student's t-test and Mann-Whitney U test was used for continuous variables, and the chi-square test for categorical variables to compare the baseline characteristics between the PCOS and control groups. Data were pooled and the association between PCOS and fertility and pregnancy outcomes were assessed by calculating relative risk with 95% confidence intervals (CI). Adjusted relative risk estimates were obtained from multivariate Poisson regression with robust error variance, controlling for potential confounders (age, BMI and insulin resistance). Statistical significance was accepted for a p-value < 0.05

Results

Total 300 women were included, with 150 women diagnosed with PCOS and 150 age-matched controls. During follow-up, 18 (6.0%) participants were lost to follow-up (11 from PCOS & 7 from control group). Baseline characteristic of the study participants is presented in (Table I).

Table I: Baseline characteristics.

Characteristic	PCOS Group (n = 139) (mean ± SD)	Control Group (n = 143) (mean ± SD)	p-value
Age	29.1 ± 4.5	28.8 ± 4.2	0.672
BMI (kg/m²)	28.3 ± 4.2	24.7 ± 3.8	<0.001
LH/FSH Ratio	2.4 ± 0.9	1.8 ± 0.6	0.048
Testosterone (ng/mL)	2.1 ± 0.5	1.2 ± 0.3	<0.001
AMH (ng/mL)	6.8 ± 2.4	3.5 ± 1.9	<0.001

Women with PCOS had a significantly longer time to conception (9.2 ± 4.5 months vs. 4.6 ± 2.3 months, p < 0.001) and a lower ovulation rate (61.2% vs. 89.5%, p < 0.001). Pregnancy complications were more frequent in the PCOS group, including higher rates of miscarriage (23.0% vs. 10.5%, RR = 2.19, 95% CI: 1.24–3.85, p = 0.008), GDM (27.3% vs. 11.9%, RR = 2.30, 95% CI: 1.36–3.88, p = 0.002), and preeclampsia (15.1% vs. 6.3%, RR = 2.39, 95% CI: 1.14–5.03, p = 0.022). Preterm birth was also significantly higher in the PCOS group (18.0% vs. 8.4%, RR = 2.14, 95% CI: 1.13–4.05, p = 0.012). Table II

Birth weight was significantly lower in the PCOS group (2.89 ± 0.6 kg vs. 3.14 ± 0.5 kg, p = 0.003). NICU admissions were more frequent in neonates born to mothers with PCOS (15.8% vs. 7.0%, p = 0.015, RR = 2.26, 95% CI: 1.14–4.49). The Apgar score at 5 minutes was comparable between groups (8.9 ± 0.7 vs. 9.0 ± 0.6, p = 0.245). Table II

Multivariate Poisson regression with robust error variance was performed to adjust for age, BMI, and insulin resistance. PCOS remained an independent risk factor for miscarriage (adjusted RR = 2.15, 95% CI: 1.22–3.77, p = 0.009), GDM (adjusted RR = 2.26, 95% CI: 1.33–3.85, p = 0.003), and preeclampsia (adjusted RR = 2.31, 95% CI: 1.09–4.91, p = 0.027). A logistic regression graph illustrating the adjusted risk of adverse pregnancy outcomes in PCOS patients is provided (Figure 1).

Table II: Fertility, Pregnancy, and Neonatal Outcomes.

Outcome	PCOS Group (n = 139)	Control Group (n = 143)	p-value	Relative Risk (95% CI)
Time to Conception (months)	9.2 ± 4.5	4.6 ± 2.3	<0.001	—
Ovulation Rate (%)	85 (61.2%)	128 (89.5%)	<0.001	0.68 (0.57–0.81)
Miscarriage (%)	32 (23.0%)	15 (10.5%)	0.008	2.19 (1.24–3.85)
GDM (%)	38 (27.3%)	17 (11.9%)	0.002	2.30 (1.36–3.88)
Preeclampsia (%)	21 (15.1%)	9 (6.3%)	0.022	2.39 (1.14–5.03)
Preterm Birth (%)	25 (18.0%)	12 (8.4%)	0.012	2.14 (1.13–4.05)
Birth Weight (kg)	2.89 ± 0.6	3.14 ± 0.5	0.003	—
NICU Admission (%)	22 (15.8%)	10 (7.0%)	0.015	2.26 (1.14–4.49)
Apgar Score (5 min)	8.9 ± 0.7	9.0 ± 0.6	0.245	—

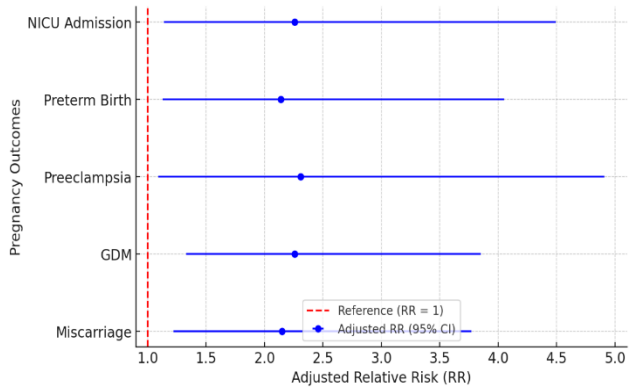

Figure 1. Adjusted relative risk of adverse pregnancy outcomes in PCOS.

Discussion

The higher incidence of miscarriage among women diagnosed with PCOS is compatible with previous reports that early pregnancy loss is associated with dysfunctional endometrium, chronic low-grade inflammation, and hyperinsulinemia.[12] Endometrial receptivity can be affected by insulin resistance, most probably causing implantation failure or miscarriage. Moreover, hyperandrogenism could also influence placentation which further enhances the rate of miscarriage.[12]

The 23.0% miscarriage rate we observed in our PCOS patients is closely in line with the findings of Benito E et al 25.4%[13], reinforcing the argument of hyperandrogenism (and endometrial dysfunction) having a shared mechanism. However, our figure is greater than the reported rate of 15.8% by Lai Q et al[14] because we examined only ultrasound-confirmed clinical miscarriages (biochemical pregnancies were not included), while Benito et al considered both early and late losses. This definition, while increasing the rigor of the study, does detract from more general population-wide applicability.

Our findings show that women with PCOS have a relatively higher prevalence of GDM (27.3%), which is in line with the findings of Chen M et al who observed similar rate of 26.1% in his study.[15] In contrast, Elshewy N et al[16] reported lower rates (18.9%), a discrepancy attributable to their use of older Carpenter-Coustan criteria (fasting glucose ≥95 mg/dL) versus our IADPSG thresholds (≥92 mg/dL), which capture milder dysglycemia. This lending further supports to the previously established association of PCOS with metabolic disturbances in pregnancy. Insulin resistance with hyperinsulinemia of both the mother and the fetus predisposes affected individuals to glucose intolerance and provides a higher risk for GDM. This suggests a need for screening and defined management strategies at an early stage to reduce the risks for maternal and fetal health.

The higher frequency of preeclampsia 15.1% in women with PCOS in our study supported the previous data implicating endothelial dysfunction and chronic inflammatory response as being important in the pathogenesis of hypertensive disorders in pregnancy, this parallels to Liu et al findings 14.7%[17], with both studies implicating endothelial dysfunction in PCOS. PCOS women have higher inflammation markers and also vascular changes, which could, in turn, lead to impaired placental function and predisposition to preeclampsia. With all of these possible downsides, guiding blood strain and blood vessel well being is great in expectant moms with PCOS.

Our study's findings of newborn characteristics further indicate PCOS's detrimental effects on pregnancy. In this study neonates of PCOS mothers had lower birth weights (2.89 kg), which is consistent with the findings of Abdulkhalikova et al[18], who reported lower birth weights of (2.91 kg), this is likely due to shared placental insufficiency mechanisms. In our study the NICU admission rate 15.8% exceeded the reported rate of in a study 12.1% by Cai H et al.[19] This discrepancy likely

reflects our institutional protocol mandating NICU evaluation for all neonates <2.9 kg, in addition to the underlying PCOS-related complications of inflammation, placental insufficiency, and hyperandrogenism that predispose to fetal growth restriction and preterm birth.

This study enhances the knowledge of the risks associated with PCOS due to its prospective design that included both fertility and obstetric outcomes in one cohort during real-world settings. We ensured clinically meaningful phenotyping by applying the Rotterdam criteria rigorously (including hormonal and ultrasound confirmation) and using standardized diagnostic criteria for complications (IADPSG for GDM). The high follow-up rate (94%) and control of important confounding factors, alongside the observed associations strengthen the outcomes. Importantly, our data uniquely quantify the median 9.2-month delay in conception PCOS patients experience in relation to the 15.8% NICU admission rate, elucidating the adverse neonatal outcome, in a developing region that documents evidence of this phenomenon.

However, despite these enlightening results, it is essential to acknowledge the limitations of the study. The results may not be as broadly applicable as they may be due to the single-center methodology and the lack of thorough control for confounding variables including genetic predispositions and lifestyle variations. Future studies should focus on lifestyle modifications and tailored medications to improve obstetric and reproductive outcomes in women with PCOS. In order to manage difficulties and improve pregnancy outcomes, the participation of obstetrical, endocrinological, and maternal fetal medicine specialists is essential. Reproductive health should be improved and negative effects could be reduced with proper risk factor evaluation and management. More work is needed to transform these findings into practical clinical practices, particularly with regard to the biological and environmental relations of these issues and the development of treatment strategies.

Conclusion

Women with PCOS experience significant reproductive and obstetric complications, including increased time to conception, miscarriage, GDM and preeclampsia risks, and adverse neonatal outcomes. While our study was observational and did not test interventions, these findings highlight critical gaps in current PCOS management that warrant further investigation into personalized treatment strategies to improve outcomes.

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Corrigendum

The article titled "Postpartum Depression and Newborn Gender: Investigating Birth Outcomes and Maternal Wellbeing in a Cross-Sectional Study" (website link: https://jsogp.net/index.php/jsogp/article/view/867/913) was published in Volume 14(4), 2024, pages 416–420, DOI: https://doi.org/10.71104/jsogp.v14i4.867.

The spelling of the third author's name was incorrectly written as "Khunsa Iqbal" due to a typographical error. The correct name is "Khansa Iqbal".

This has been rectified in the online version of the article at the above link.