Original Article

Impact of Polycystic Ovaries on Maternal and Fetal Complications in Pregnant Women: A Case-Control Study

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Abstract

Objective: The objective of this study was to investigate the association between polycystic ovaries (PCOs) and maternal complications during pregnancy.

Methods: This prospective case-control study was conducted at Shahida Islam Medical Complex from November 2024 to June 2025. Data was collected from inpatient pregnant females. Demographic information was recorded, and patients were divided into cases and controls based on the status of PCOs, as diagnosed clinically according to the Rotterdam criteria. Before delivery, 5ml of venous blood was collected for estimation of lab parameters. Patients were followed till delivery, and maternal-fetal outcomes were recorded.

Results: A significant association between PCOS and gestational diabetes was found (χ=11.35, p< 0.0001). Logistic regression analysis, using a backwards stepwise method, identified LH, FSH, Testosterone, Insulin Resistance, and Systolic BP as significant predictors of Gestational Diabetes. Notably, higher levels of LH and FSH were associated with increased risk ($p < 0.0001$ and $p = 0.0000$, respectively). The correlation matrix confirmed strong correlations between Testosterone and LH ($r = 0.83$), and between Insulin Resistance and Glucose levels ($r = 0.83$), highlighting metabolic dysfunctions in PCOS.

Conclusion: These findings underscore the critical role of hormonal imbalances and insulin resistance in increasing the risk of pregnancy complications in PCOS patients, emphasising the need for early screening and intervention.

Keywords: Polycystic Ovary Syndrome, Pregnancy Complications, Gestational Diabetes Mellitus, Insulin Resistance, Pregnancy Outcome, Hypertension, Pregnancy-Induced, Maternal Health, Neonatal Outcome

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Introduction

Polycystic Ovary Syndrome (PCOS) is a significant endocrine disorder due to its vast complications.[1] It has a global prevalence of 20% in females of reproductive age.[2] In Pakistan, a recent study reported that 27.2% of reproductive-age females had PCOS.[3] PCOs are characterised by polycystic ovaries, anovulatory cycles, raised androgens, and/or clinical signs of hyperandrogenism.[4] These features are known to affect fertility and associated mental health. There is also an increased risk of the development of gestational diabetes, hypertensive disorders of pregnancy, preterm delivery, and emergency cesarean section.[5] PCOs are also known to affect pregnancy complications like miscarriages, recurrent miscarriages, and a low probability of having a neonate with optimal birth weight, oversized neonates, and neonates that require admission to the NICU.[6] The inflammatory response and oxidative stress in females with PCOS may lead to hyperinsulinemia, chronic low-grade inflammation, endothelial dysfunction, hyperandrogenemia, and alterations in normal maternofetal outcomes.[3,7] Keeping in view the above-mentioned complications of PCOs, this study aims to investigate the maternal-fetal outcome in females with and without PCOs. This study is done to utilise findings as the departmental protocol to provide better care for pregnant females with PCOS coming to our hospital facility.

Materials And Methods

A case-control study was conducted at Shahida Islam Medical Complex following STROBE guidelines. The duration of the study was 1 year from June 2024- June 2025. Ethical Approval was obtained from the Institutional Review Board (SIMC/ET.C./0040/24), and participants were recruited after informed consent. A sample size of 272 for cases was calculated by the WHO calculator, taking the population proportion as 22.7%,[8] 5% margin of error, and a 95% confidence interval. The sample population was females of reproductive age (15-49 years as set by WHO), divided into cases (pregnant females of PCOs=272) and controls (healthy pregnant females without PCOs, n=272)

Inclusion criteria for cases were inpatient pregnant females diagnosed with PCOS (clinically diagnosed, meeting the Rotterdam criteria for PCOS.[9]). Inpatient pregnant females with no history of PCOS or other known fertility-related issues served as controls. Females with any pre-existing medical conditions like diabetes, hypertension, or autoimmune diseases, or females with a history of pregnancy-related complications in prior pregnancies (to control for confounding factors), or females with elective cesareans were excluded from the study.

After obtaining informed consent, demographic data were collected using a study-designed questionnaire. Blood pressure, BMI, and blood glucose (fasting and postprandial) were recorded on admission day. This was followed by 5ml of intravenous sampling for estimation of serum Luteinizing Hormone (LH), Follicle-Stimulating Hormone (FSH), Insulin Resistance Markers (HOMA-IR), Testosterone (to assess androgen

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excess), and Lipid Profile (Triglycerides, Cholesterol). This sampling was done for those patients who did not have a previous record of the above lab investigations. Upon delivery, fetal weight and fetal heart rate (FHR, normal =110-160bpm) were measured.
The inpatient pregnant females were followed till delivery, and maternal outcomes (History of Gestational Diabetes, Preeclampsia, Cesarean Section or vaginal delivery, weight Gain During Pregnancy, and Postpartum haemorrhage) were noted based on available patient records and history.
Data was analysed with SPSS 26.0. The normality of data was checked with the Shapiro-Wilk test. Categorical variables were expressed as frequency percentages, and quantitative variables were expressed as mean and standard deviation. For continuous variables, group comparison was done by the Mann-Whitney U test, and for categorical variables, the chi-square test was used. To analyse the odds of developing maternal and fetal complications based on PCOS status, regression analysis was done with the backwards method after adjusting for confounding variables like age and BMI.

Results

The study included 544 age and BMI-matched cases and controls. Table 1:

Table 1: Baseline characteristics of the whole study population, n=544

Variable	Mean	STD	25%	50%	75%
Age (Years)	28.33036	2.888471	140.75	280.5	420.25
BMI (kg/mÂ²)	29.98143	2.938343	26	28	30
Previous Pregnancies	2.032143	0.826032	28	30	31.9
Testosterone (ng/dL)	53.07982	11.92967	0	0	1
LH (mIU/mL)	15.96607	6.869644	44.175	51.7	61.725
FSH (mIU/mL)	7.387679	2.172501	9.8	13.85	22.025
Insulin Resistance (HOMA-IR)	3.073571	0.970263	5.7	6.9	8.925
Systolic BP (mmHg)	125.0875	10.28004	2.3	2.9	3.8
Diastolic BP (mmHg)	79.63571	7.967699	118	125	132
Glucose Level (mg/dL)	119.6875	27.58121	74	79	85
Postpartum Hemorrhage	0.146429	0.353851	98.75	117	141
Estradiol (pg/mL)	90.25268	30.18425	0	0	0
Progesterone (ng/mL)	12.27696	5.053636	71.9	89.1	110.025
Prolactin (ng/mL)	16.12661	5.737491	8.6	12	15.9
TSH (mIU/L)	2.781607	0.940978	12.175	16.1	20.025
Fetal Growth Restriction (FGR)	0.153571	0.36086	0	1	2
Gestational Age at Delivery	39.46607	1.708317	0	0	0
Fetal Heart Rate (FHR)	140.1125	12.02129	38	40	41

Correlation analysis was done to find hormonal, emotional, and physical factors on pregnancy complications. We found a strong positive correlation between Testosterone and LH (r = 0.83), and Testosterone and FSH (r = 0.61), reflecting typical PCOS characteristics. Insulin resistance correlates strongly with Testosterone (r = 0.73) and glucose levels (r = 0.83), showing a metabolic disturbance linked to PCOS. Systolic and Diastolic BP have a strong correlation (r = 0.88), and Triglycerides are negatively correlated with HDL (r = -0.76), indicating cardiovascular risk. Insulin resistance and glucose levels correlate with gestational diabetes, while high BP and glucose are linked to preterm birth and FGR. Figure 1

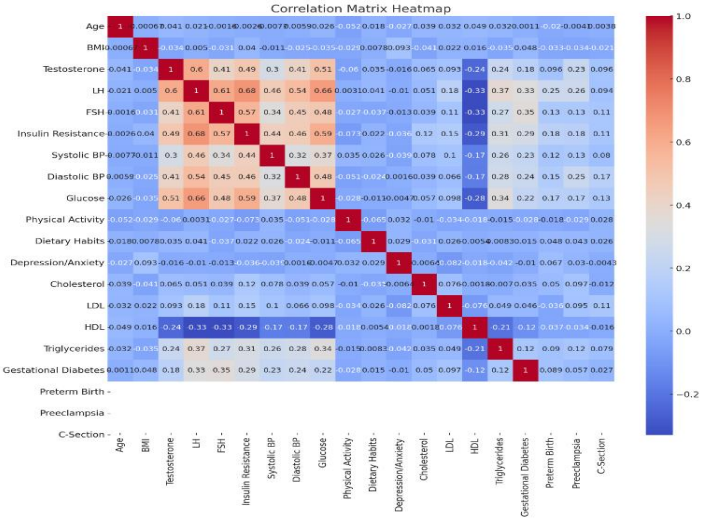

Figure 1: Heatmap for Correlation Matrix. Values close to 1 indicate a strong correlation, while values close to zero indicate a weak to no correlation. Negative sign indicates negative correlation, and positive sign indicates positive correlation

The baseline characteristics revealed a significant incidence of preeclampsia, c-section, presence of gestational diabetes, and preterm birth in females with PCOs. Females with PCOs had significantly greater insulin resistance and elevated testosterone levels (p=<0.0001, p=0.0002, respectively). Table 2: To find the association of maternal and fetal outcomes with the status of PCOs, chi chi-square test of association was applied, and a p-value of <0.05 was considered statistically significant. Table 2

Table 2: Group comparison of baseline characteristics based on presence (Cases) or absence (Controls) of PCOs

Variable	Cases (n=272, mean ± SD)	Control (n=272, mean ± SD)	p-value
Age (Years)	27.46 ± 2.96	28.20 ± 2.82	0.2859
BMI (kg/m²)	29.95 ± 2.86	30.01 ± 3.02	0.8205
Previous Pregnancies	1.97 ± 0.82	2.09 ± 0.83	<0.0001
Testosterone (ng/dL)	61.05 ± 10.07	45.11 ± 7.51	<0.0001
LH (mIU/mL)	22.10 ± 3.91	9.84 ± 1.96	<0.0001
FSH (mIU/mL)	8.90 ± 1.94	5.88 ± 1.04	<0.0001
Insulin Resistance (HOMA-IR)	3.83 ± 0.69	2.32 ± 0.51	0.0225
Systolic BP (mmHg)	130.41 ± 9.50	119.76 ± 8.03	0.0175
Diastolic BP (mmHg)	84.56 ± 7.27	74.71 ± 5.07	0.0163
Glucose Level (mg/dL)	139.99 ± 20.77	99.39 ± 16.30	0.0285
Total Cholesterol (mg/dL)	192.96 ± 25.82	188.85 ± 17.69	<0.0001
LDL (mg/dL)	103.19 ± 30.60	93.47 ± 20.19	<0.0001
HDL (mg/dL)	49.90 ± 11.09	58.87 ± 11.55	<0.0001
Triglycerides (mg/dL)	176.47 ± 46.60	138.14 ± 38.25	0.0003
Fetal Heart Rate (FHR)	140.09 ± 11.78	140.14 ± 12.28	0.958
Gestational Age at Delivery	39.41 ± 1.7	39.52 ± 1.72	0.4148
	n		**χ/p**
Gestational Diabetes			
No	165	252	25.44/ <0.0001
Yes	115	28	
Preeclampsia			
No	194	255	11.01/ <0.0001
Yes	86	25	
Preterm Birth			
No	209	254	12.12/<0.0001
Yes	71	26	
C-Section			
No	179	217	13.56/ 0.0015
Yes	101	63	
Postpartum Hemorrhage			
No	218	260	24.0167/)0.765
Yes	62	20	
Physical Activity (0-3)			
0 (Sedentary, no exercise)	72	66	
1 (1-2 days/week)	69	70	5.22/0.1565
2 (2-3 days/ week)	80	64	
3 (Daily Exercise 7days/week)	59	80	
Dietary Habits (0-2)			
0 (Balanced)	93	85	2.60/0.2727
1 (More vegetables)	85	103	
2 (More Meat)	102	92	
Depression/Anxiety Score (0-3)			
0 (No)	71	71	
1 (Mild)	79	81	1.93/0.5865
2(Moderate)	68	56	
3 (Severe)	62	72	
Fetal Growth Restriction (FGR)			
0 (No)	221	253	13.20/ 0.0003
1 (Yes)	59	27	

**Mann-Whitney U test applied for quantitative variables and chi-square test of association applied for qualitative variables. A p-value of <0.05 was considered statistically significant.*

Logistic regression was performed with a backwards method. Pregnancy-related complications were taken as outcomes, and hormonal, physical, and emotional factors were considered as predictors. Age and BMI were controlled, so confounders did not need to be excluded. The backwards method excluded weak to moderate correlation variables, and the strong ones were included in estimating the odds ratio. We found that higher levels of LH and FSH are associated with an increased risk of Gestational Diabetes. Table 3

Table 3: Logistic regression analysis. The outcome was gestational diabetes, and the predictors were LH and FSH

	Coef.	Std. Err.	Z	p	0.025	0.975
Intercept (const)	-4.30885	0.429	-10.03	0.0000	-5.150	-3.467
LH (mIU/mL)	0.070246	0.018	3.7887	0.0002	0.0339	0.1065
FSH (mIU/mL)	0.259504	0.057	4.5103	0.0000	0.1467	0.3722

Discussion

PCOS is inherently characterised by hormonal imbalances, particularly elevated levels of Testosterone, LH, and FSH.[10] The current study reported a significant association of LH, FSH with Gestational Diabetes ($p = 0.0002$ for LH, $p = 0.0000$ for FSH), and other pregnancy-related complications in this study. Table 2-4, Figure 1. This supports the hypothesis that hormonal disturbances are central to the pathophysiology of PCOS and crucial for understanding its impact on pregnancy outcomes. A previous study found that PCOS patients have a significantly higher incidence of Gestational Diabetes, consistent with the findings of this study ($p < 0.0001$).[11] While contrary to current study findings, some did not find any significant role of FSH in pregnancy-related complications.[12] These elevated hormone levels are known to contribute to insulin resistance, which exacerbates metabolic disturbances during pregnancy. In the present study, insulin resistance was significantly higher in the PCOS group ($p = 0.0225$) and correlated strongly with blood glucose levels ($r = 0.83$), validating the role of insulin resistance in adverse pregnancy outcomes.[7] The findings align with existing literature that shows insulin resistance as a primary factor in PCOS-related complications.[13] A meta-analysis demonstrated the strong association between insulin resistance and pregnancy complications in PCOS, similar to the HOMA-IR findings in this study ($p = 0.0225$).[10] While this study found insulin resistance to be a significant predictor of C-sections ($p = 0.0425$), other studies did not establish a direct link between insulin resistance and C-section.[14] Early intervention strategies like metformin, dietary modifications, and weight management should be considered to manage these risks. PCOS patients have an increased risk of Gestational Diabetes, Preeclampsia, Preterm Birth, and C-sections, as shown by significant findings in this study ($p < 0.0001$ for Gestational Diabetes, $p = 0.0006$ for Preeclampsia). Lipid abnormalities, including elevated triglycerides ($p = 0.0003$) and lower HDL levels ($p < 0.0001$), were found in the PCOS group, contributing to cardiovascular risks. The correlation between insulin resistance and lipid disturbances ($r = 0.73$) in PCOS suggests that metabolic dysfunction impacts both maternal and fetal health. This underscores the importance of monitoring lipid profiles and managing cardiovascular risks during pregnancy in PCOS patients. Studies also showed that women with PCOS have higher chances of developing gestational diabetes mellitus (GDM), hypertensive disorders, premature births, and cesarean delivery as compared to non-PCOS women.[15] Newborn complications, such as low birthweight, macrosomia, and a higher NICU admission rate, were also often reported in women with PCOS.[16] Prospective interventions in this high-risk group are highlighted by these outcomes. This could be attributed to insulin resistance; PCOS is the reason behind the high rate of GDM in women of reproductive age. Hyperinsulinemia that accompanies insulin resistance not only causes hyperglycemia during pregnancy but may worsen other pregnancy complications such as hypertensive disorders.[1] Higher preeclampsia and gestational hypertension identified in this study are consistent with endothelial dysfunctions and chronic inflammation seen in PCOS.[17] In addition, the PCOS group is more likely to have higher cesarean delivery rates due to complications like macrosomia and failed labour induction. The neonatal complications in pregnancy affected by PCOS are the result of the poor-quality intrauterine environment due to maternal hyperglycemia, obesity, and other metabolic derangements. While low birth weight of a baby can be due to placental dysfunction, macrosomia can be due to maternal hyperglycemia.[5,15] Thus, the rising rates of NICU admission point to the clinical relevance of these complications and underscore the importance of continued observation of the neonates. There are various ways through which PCOS leads to these outcomes of the following diseases, and these include various factors. Hyperandrogenism and insulin resistance are two key components, if not the root of the matter, in PCOS and are believed to cause complications through maternal metabolism, placental function, and fetal development.

Conclusions

This study reports a significant impact of polycystic ovarian syndrome on maternal and fetal outcomes. This is greatly associated with increased risk of Gestational Diabetes. There is a strong chance of fetal growth restriction in females with PCOS. Early intervention on control of FSH and LH levels, along with insulin sensitivity, is essential for improving maternal and fetal outcomes in women with PCOS.

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