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Unraveling the Link: PCOS and Preeclampsia: A Prospective Cohort Study Evaluating Maternal and Fetal Outcomes.

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Abstract

Background: Polycystic ovary syndrome (PCOS) is known to be linked with metabolic and vascular abnormalities that can lead to adverse pregnancy outcomes. The current study aimed to determine the relationship between PCOS and preeclampsia and to examine maternal and fetal outcomes. ***Methods:*** A prospective cohort study of 426 women during pregnancy, including 213 women with PCOS and 213 women without PCOS, was performed. Participants were followed from the time of enrolment in the antenatal clinic until delivery. Statistical analysis was used to compare maternal and fetal outcomes, and multivariable logistic regression was used to detect the independent predictors of preeclampsia. ***Results:*** Women with PCOS were significantly more likely to develop preeclampsia than women without PCOS (p=0.004). Gestational diabetes, cesarean delivery, preterm birth, low birth weight, and neonatal intensive care unit admission were found to be associated with PCOS. The relative risk for preeclampsia was 2.46. PCOS was still independently associated with preeclampsia after adjusting for potential confounders (p=0.012). ***Conclusions:*** PCOS was independently linked with preeclampsia and some maternal and fetal complications. Better antenatal risk assessment and more monitoring between mother and baby could lead to better care for women with PCOS.

Keywords: Polycystic ovary syndrome; PCOS; Preeclampsia; Hypertensive disorders of pregnancy; Maternal outcomes.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex endocrine and reproductive disorder that is characterized by a combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.[1] In addition to the known impacts on fertility, PCOS is now known to be a systemic metabolic disorder linked to insulin resistance, obesity, chronic low-level inflammation, endothelial dysfunction, and an undesirable heart and metabolic profile.[2] The abnormalities can persist or become clinically significant during pregnancy and may increase the risk for obstetric complications in women with PCOS.[3]

One of the largest systematic reviews and meta-analyses published, which included 104 studies and over 106,000 pregnancies, found that women with PCOS had significantly increased risk of gestational diabetes mellitus, gestational hypertension, pre-eclampsia, miscarriage, and cesarean delivery compared to women without PCOS.[4] Importantly, the extra risk of adverse outcomes continued even after the researchers adjusted for age and BMI, indicating that the presence of PCOS may be a factor in adverse pregnancy outcomes.[5] Preeclampsia is regarded as a big hypertensive disorder in pregnancy and is still an important reason for the morbidity and mortality of both the mother and the baby all over the world.[6] The defects in placentation and remodeling of the spiral arteries, endothelial dysfunction, inflammation, oxidative stress, and impaired regulation of angiogenic pathways underlie the pathophysiology.[7] Some of these mechanisms are similar to the metabolic and vascular changes in PCOS.[8] Among other mechanisms, insulin resistance,

hyperandrogenism, obesity, and chronic inflammation could contribute to endothelial dysfunction and abnormal adaptation of the placenta, which could explain the possible link between PCOS and preeclampsia.[9]

The recent meta-analysis suggests that women with PCOS are about two and a half times more likely to develop preeclampsia than women without PCOS, with the association also remaining after adjustment for or matching on important confounders, including BMI and age.[10]. The effects of this relationship could be more far-reaching than maternal hypertension.[11] There are also potential negative fetal and neonatal outcomes that have been associated with pregnancy in women with PCOS, such as low birth weight, fetal growth problems, neonatal morbidity, and premature birth. The 2024 systematic review and meta-analysis, which included 73 studies and 92,881 offspring, identified a higher risk for low birth weight, small-for-gestational age birth, and lower mean birth weight in women with PCOS, with the risk of small-for-gestational age birth and lower mean birth weight being somewhat independent from maternal BMI.[12]

Furthermore, population-based data show that PCOS could exacerbate the negative impact of hypertensive pregnancy complications. In a large cohort of Finnish women, those with PCOS had a higher risk of having a premature child if they had preeclampsia, and women with PCOS and gestational hypertension had a higher risk of having a large-for-gestational-age infant.[13]. Although there is increasing evidence that PCOS is a risk factor for preeclampsia, more questions remain unanswered about how much, and whether women with preeclampsia and PCOS have a unique pattern of maternal and fetal outcomes. Moreover, most of the available evidence is retrospective from databases or international data combined, with prospective data from South Asian populations still being relatively scarce. This association was also confirmed in a recent prospective study conducted in Pakistan, which found increased prevalence of preeclampsia and preterm delivery among pregnant women with PCOS.

In this context, PCOS could be viewed as more than just a reproductive disorder and an early indicator of increased maternal vascular and placental susceptibility during pregnancy. The potential benefit of a prospective assessment from pregnancy to delivery is thus a clinically relevant measurement of the risk for preeclampsia among women with PCOS and whether this is associated with clinical consequences for mother and child. Collecting such evidence in the local population may help reinforce the risk stratification of antenatal care, prompt earlier screening of women with PCOS and help to ensure that women at higher risk receive appropriate interventions. So, the present prospective study was carried out to assess the relationship between PCOS and preeclampsia and to compare the maternal and fetal outcome of pregnant women with and without PCOS.

MATERIALS AND METHODS

A prospective cohort study was conducted in the Department of Obstetrics and Gynecology. This study took place from May 2025 to April 2026 with a 12 month study period which included recruiting the participants, antenatal follow-up, delivery, and recording of maternal and neonatal outcomes. The participants were followed from enrolment to delivery, and neonatal outcomes were recorded prior to discharge.

A sample size was determined by OpenEpi version 3.01, which is a program for comparing two proportions in a cohort study. Ali et al. conducted a prospective study in Pakistan and reported that the incidence of preeclampsia in women with PCOS was 14.7% compared to 6.0% among women without PCOS and this figure was used for the calculation.[14] With a two-sided 95% confidence level, 80% statistical power, and these assumed proportions, the sample size for each cohort was approximately 192 for a total of 384 participants. The final sample size was 426 (including an allowable 10% loss to follow-up, which meant that around 213 women with PCOS and 213 women without PCOS were needed).

A non-probability convenience, or consecutive sampling method, was used for the selection of participants. Eligible women were those who presented for antenatal care before the onset of preeclampsia, had confirmed intrauterine pregnancy, and were aged 18-40 years. Women with PCOS were defined as having a prior diagnosis of PCOS and fulfillment of the Rotterdam criteria, which included at least one of three features: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology (PCOM) on ultrasonography. In comparison cohort women, there was no previous diagnosis or clinical evidence of PCOS. Singleton pregnancies were included and those who gave informed consent and desired to be followed until delivery were enrolled.

Excluded subjects were women with chronic hypertension, with previous type 1 or 2 diabetes mellitus, chronic renal disease, systemic lupus erythematosus or other major autoimmune diseases, antiphospholipid syndrome, significant cardiovascular disease, thyroid disease requiring treatment, and other major endocrine disorders. Women who had previous pregnancies with multifetal, a known major fetal congenital anomaly, pregnancy due to assisted reproductive techniques, and those with preeclampsia at entry to the study were also excluded. Participants who were lost to follow-up and those who withdrew after consent were excluded from the final outcome analysis.

Written informed consent was obtained, and a structured proforma for data collection was used to gather baseline data. Demographic data about the mother such as age, place of residence, parity, gravidity, socioeconomic status and education were recorded. Details regarding the pre-pregnancy weight, height, body mass index (BMI), and medical and obstetric history were recorded. The following information was obtained: age at diagnosis, irregular periods, hyperandrogenic features, previous infertility, and treatment history and ultrasonography results for women with PCOS. All participants were routinely assessed at the institution of their blood pressure, weight, urine protein, and relevant laboratory tests with serial measurements.

The participants were followed up for the duration of pregnancy. Gestational hypertension or preeclampsia was documented based on the following criteria: hypertension developed during pregnancy or at delivery, or preeclampsia symptoms that occurred during pregnancy or at delivery. Preeclampsia was considered to be new-onset hypertension after 20 weeks of gestation, with proteinuria or any other sign of maternal organ dysfunction in a previously normotensive woman. Details of GMH, antepartum haemorrhage, mode of delivery, gestational age at delivery and other maternal complications were noted. Fetal and neonatal outcomes at delivery were recorded, including birth weight, sex, Apgar score, pre-term delivery, small for gestational age or large for gestational age, stillbirth, neonatal intensive care unit admission and early neonatal complications. The primary outcome was preeclampsia and secondary outcomes were maternal complications, mode of delivery, gestational age at delivery, and fetal/neonatal outcome.

IBM SPSS Statistics version 27.0 was used to enter and analyse data. The Shapiro–Wilk test was used for continuous variables to check for normality, and if they were normally distributed, they were expressed as mean ± standard deviation; if non-normally distributed, they were expressed as medians with interquartile range. Categorical variables were reported in frequencies and percentages. Normally distributed continuous variables were compared between the PCOS and non-PCOS groups using the independent-samples t test, and skewed continuous variables were compared using the Mann–Whitney U test. A comparison of categorical variables was made by the chi-square test and the Fisher's exact test.

The frequency of preeclampsia and other maternal and fetal outcomes was compared in women with PCOS and women without PCOS and relative risks (RRs) with 95% confidence intervals (CIs) were computed. To investigate if PCOS was an independent risk factor for preeclampsia after controlling for clinically relevant potential confounders, multivariable binary logistic regression analysis was then conducted. Odds ratios (aORs) with 95% confidence intervals were presented. A p value <0.05 by a two-tailed test was determined as statistically significant.

RESULTS

There were a total of 426 pregnant women, 213 women who had PCOS and 213 women who did not have PCOS. There was no difference in mean age, p=0.397, between the groups. Women with PCOS, however, had significantly higher pre-pregnancy BMI (p<0.001) with a larger percentage of overweight and obese. There were no significant differences between the groups in gravidity, parity, or family history of hypertension or diabetes. (Table 1)

Table 1. Baseline demographic and obstetric characteristics of study participants (n=426)

Variable	PCOS (n=213) n(%)/mean ± SD	Non-PCOS (n=213) n(%)/mean ± SD	p-value
Age (years),	29.1 ± 4.6	28.7 ± 4.8	0.397
Age group			0.682
18–24 years	42 (19.7)	46 (21.6)	
25–29 years	72 (33.8)	69 (32.4)	
30–34 years	70 (32.9)	66 (31.0)	
35–40 years	29 (13.6)	32 (15.0)	
Gravidity, median (IQR)	2 (1–3)	2 (1–3)	0.541
Primigravida	104 (48.8)	99 (46.5)	0.631
Multiparous	109 (51.2)	114 (53.5)	
Pre-pregnancy BMI (kg/m²)	28.4 ± 4.5	25.9 ± 3.8	<0.001
BMI category			<0.001
Normal weight	52 (24.4)	91 (42.7)	
Overweight	83 (39.0)	79 (37.1)	
Obese	78 (36.6)	43 (20.2)	
Family history of hypertension	51 (23.9)	38 (17.8)	0.134
Family history of diabetes	44 (20.7)	32 (15.0)	0.139

Women with PCOS had significantly higher frequencies of previous infertility, menstrual irregularity, clinical hyperandrogenism, and biochemical hyperandrogenism (all p<0.001) compared with women without PCOS. Women with PCOS were also more likely to report having been treated with ovulation-inducing medication. (Table 2)

Table 2. Clinical characteristics of women with PCOS and non-PCOS women

Variable	PCOS (n=213) n(%)	Non-PCOS (n=213) n(%)	p-value
Previous infertility	68 (31.9)	24 (11.3)	<0.001
History of menstrual irregularity	176 (82.6)	18 (8.5)	<0.001
Clinical hyperandrogenism	121 (56.8)	12 (5.6)	<0.001
Biochemical hyperandrogenism	109 (51.2)	9 (4.2)	<0.001
Polycystic ovarian morphology	191 (89.7)	17 (8.0)	<0.001
Previous PCOS treatment	86 (40.4)	—	—
Ovulation-induction treatment,	49 (23.0)	11 (5.2)	<0.001

Women with PCOS had a significantly higher incidence of preeclampsia than women without PCOS (p=0.004). Gestational diabetes mellitus and cesarean delivery were also significantly more prevalent in the PCOS group, but not all differences were statistically significant: gestational hypertension, antepartum hemorrhage, and eclampsia. (Table 3)

Table 3. Maternal pregnancy outcomes according to PCOS status

Maternal outcome	PCOS (n=213)	Non-PCOS (n=213)	p-value
Gestational diabetes mellitus	58 (27.2)	26 (12.2)	<0.001
Gestational hypertension	31 (14.6)	18 (8.5)	0.060
Preeclampsia	32 (15.0)	13 (6.1)	0.004
Severe preeclampsia	12 (5.6)	4 (1.9)	0.049
Early-onset preeclampsia (<34 weeks)	7 (3.3)	2 (0.9)	0.092
Late-onset preeclampsia (≥34 weeks)	25 (11.7)	11 (5.2)	0.016
Cesarean delivery	111 (52.1)	83 (39.0)	0.008
Preterm delivery	39 (18.3)	17 (8.0)	0.002
Antepartum hemorrhage	7 (3.3)	4 (1.9)	0.379
Eclampsia	2 (0.9)	1 (0.5)	0.562

Women with PCOS also had significantly lower mean birth weight of neonates compared to women without PCOS and women with PCOS delivered at a significantly earlier gestation age. There was a significant increase in low birth weight, preterm delivery, and LGA in the children of the women with PCOS, as well as a higher rate of NICU admission. There were also slightly lower mean scores at one and five minutes, and no significant difference was seen in stillbirth or neonatal mortality. (Table 4)

Table 4. Fetal and neonatal outcomes according to PCOS status

Fetal/neonatal outcome	PCOS (n=213) n(%)/mean ± SD	Non-PCOS (n=213) n(%)/mean ± SD	p-value
Birth weight (kg), mean ± SD	2.91 ± 0.54	3.13 ± 0.49	<0.001
Low birth weight (<2.5 kg)	37 (17.4)	20 (9.4)	0.018
Small for gestational age	29 (13.6)	17 (8.0)	0.072
Large for gestational age	25 (11.7)	13 (6.1)	0.038
Preterm birth (<37 weeks)	39 (18.3)	17 (8.0)	0.002
Apgar score at 1 min, mean ± SD	7.5 ± 1.1	7.8 ± 0.8	0.004
Apgar score at 5 min, mean ± SD	8.5 ± 0.8	8.7 ± 0.6	0.006
NICU admission	34 (16.0)	16 (7.5)	0.007
Stillbirth	4 (1.9)	2 (0.9)	0.437
Neonatal mortality	3 (1.4)	1 (0.5)	0.312

Women who were diagnosed with PCOS had slightly higher levels of systolic blood pressure, diastolic blood pressure, fasting blood glucose, and HbA1c at the time of their enrollment. Positive urine protein levels at follow-up were also significantly higher in the PCOS group. There were no differences in mean hemoglobin concentration between groups. (Table 5)

Table 5. Comparison of antenatal clinical and laboratory parameters

Variable	PCOS (n=213) n(%)/mean ± SD	Non-PCOS (n=213) n(%)/mean ± SD	p-value
Systolic BP at enrollment (mmHg)	116.8 ± 9.7	114.9 ± 8.8	0.034
Diastolic BP at enrollment (mmHg)	74.2 ± 7.4	72.8 ± 6.9	0.048
Fasting blood glucose (mg/dL)	91.8 ± 12.7	87.6 ± 9.4	<0.001
HbA1c (%)	5.47 ± 0.49	5.29 ± 0.38	<0.001
Hemoglobin (g/dL)	11.2 ± 1.1	11.3 ± 1.0	0.314
Urine protein ≥1+, n (%)	34 (16.0)	15 (7.0)	0.005
Gestational age at delivery (weeks)	37.1 ± 2.3	38.0 ± 1.7	<0.001

The relative risk analysis showed that women with PCOS faced a risk of about 2.5-fold that of preeclampsia compared to women without PCOS. Excess risks were also noted for gestational diabetes, cesarean delivery, preterm birth, low birth weight, and admission to the NICU. (Table 6)

Table 6. Incidence and relative risk of maternal and fetal outcomes

Outcome	PCOS n/N (%)	Non-PCOS n/N (%)	Relative risk (95% CI)	p-value
Preeclampsia	32/213 (15.0)	13/213 (6.1)	2.46 (1.34–4.52)	0.004
Gestational diabetes	58/213 (27.2)	26/213 (12.2)	2.23 (1.49–3.34)	<0.001
Gestational hypertension	31/213 (14.6)	18/213 (8.5)	1.72 (0.99–2.99)	0.060
Cesarean delivery	111/213 (52.1)	83/213 (39.0)	1.34 (1.10–1.64)	0.008
Preterm birth	39/213 (18.3)	17/213 (8.0)	2.29 (1.35–3.88)	0.002
Low birth weight	37/213 (17.4)	20/213 (9.4)	1.85 (1.11–3.09)	0.018
NICU admission	34/213 (16.0)	16/213 (7.5)	2.13 (1.20–3.79)	0.007

PCOS remained independently associated with preeclampsia in multivariable logistic regression when adjusted for maternal age, BMI, parity, family history of hypertension, gestational diabetes, and SBP. The adjusted odds of preeclampsia were 2.31-fold higher in women with PCOS (p=0.012). Higher systolic blood pressure and family history of hypertension were also independent risk factors for preeclampsia. Additional factors associated with preeclampsia were independently obesity and family history of hypertension and gestational diabetes. (Table 7)

Table 7. Multivariable logistic regression analysis of factors associated with preeclampsia

Predictor	Adjusted OR	95% CI	p-value
PCOS	2.31	1.20–4.45	0.012
Maternal age ≥30 years	1.58	0.86–2.90	0.139
Overweight BMI	1.74	0.91–3.34	0.094
Obesity	2.48	1.28–4.81	0.007
Primigravida	1.46	0.79–2.68	0.226
Family history of hypertension	2.02	1.04–3.92	0.038
Gestational diabetes mellitus	1.89	1.01–3.54	0.047
Systolic BP at enrollment	1.04	1.01–1.07	0.009

In general, PCOS was found to be significantly associated with preeclampsia, gestational diabetes, cesarean delivery, preterm delivery, low birth weight, and NICU admission. However, there were no significant differences in stillbirths or neonatal deaths between the two groups. (Table 8)

Table 8. Summary of primary and secondary outcomes

Outcome	PCOS	Non-PCOS	p-value
Preeclampsia	15.0%	6.1%	0.004
Gestational diabetes	27.2%	12.2%	<0.001
Cesarean delivery	52.1%	39.0%	0.008
Preterm birth	18.3%	8.0%	0.002
Low birth weight	17.4%	9.4%	0.018
NICU admission	16.0%	7.5%	0.007
Stillbirth	1.9%	0.9%	0.437
Neonatal mortality	1.4%	0.5%	0.312

DISCUSSION

In this study, the present prospective cohort study, women with PCOS had a significantly higher risk of adverse pregnancy outcomes compared to women without PCOS. The most significant result was that women with PCOS had a relative risk of 2.46 for preeclampsia (15.0% versus 6.1%). Importantly, PCOS was independently associated with preeclampsia after adjustment for age, BMI, parity, family history of hypertension, gestational diabetes, and systolic blood pressure. The results indicate that PCOS is a significant risk factor for pregnancy and not just a reproductive condition.

Our risk for preeclampsia of 2.46 was very similar to that reported in the 2024 systematic review and meta-analysis by Teede et al. (2024), which reported 104 studies and 106,690 pregnancies, with odds of preeclampsia being 2.30 times higher in women with PCOS. Importantly, the association remained in age- and BMI-matched analyses and in prospective and high-quality studies, which provided further support for the independent association of PCOS beyond obesity. Their pooled estimate is similar to our prospective estimate, which further validates the present results.[15]

The present findings were also similar to those reported by Farland et al. (2022) in a large registry linkage study of 91,825 births in Massachusetts, where women with PCOS had a 25% higher risk of preeclampsia, even after accounting for other maternal and pregnancy factors. The same study found a 51% increased risk of gestational diabetes and a 17% increased risk of preterm birth. While the strength of association was less than that seen in our cohort, it was in the same direction. Variations could be attributed to the diagnosis and ascertainment of PCOS, the population, the rate of obesity, and treatment systems.[16]

In another study in 2022, Joshi et al. assessed the association between PCOS and preeclampsia and confirmed that this association is biologically plausible. The authors emphasized the role of insulin resistance, obesity, hyperandrogenism, endothelial dysfunction, and abnormal placentation in the higher risk of hypertensive pregnancy disorders in PCOS. We found that PCOS was still linked to preeclampsia even after we accounted for some metabolic and demographic factors, which aligns with the idea that there may be several overlapping mechanisms involved in the link between PCOS and preeclampsia.[17]

In particular, the results of Jiang et al. (2024), a retrospective cohort of 616 women with PCOS, were relevant. Of all women with PCOS, 8.28% developed preeclampsia, while 3.22% of women without PCOS did develop it. Hyperandrogenism, pre-pregnancy BMI ≥24 kg/m², a family history of cardiovascular disease, and ART were found to be important predictors. The preeclampsia rate observed (15.0%) was higher, which may be due to population risk, prospective ascertainment, and a tertiary-care setting. However, both studies showed that there was a clear association between PCOS and hypertensive pregnancy outcomes and highlighted the significance of metabolic and androgenic features.[18]

The association between PCOS and preeclampsia was also supported by the national Swedish cohort of Valdimarsdottir et al. (2024), which included 22,947 women with PCOS and 115,272 controls. There was an adjusted OR of 29% for PCOS and preeclampsia, which was higher for early-onset preeclampsia (1.64) than for late-onset preeclampsia (1.26). An increased number of women with PCOS in our study had early onset preeclampsia; however, this relationship was not statistically significant, likely due to a small number of early-onset cases. It is a possibility that the concordance indicates that PCOS is association with specific early placental dysfunction during pregnancy.[19]

The association of increased GDM occurrence in women with PCOS (27.2% vs. 12.2%) was also consistent with the 2024 systematic review by Teede et al., which showed that women with PCOS had 2.41 times greater odds of having GDM. Association remained significant after exclusion of sensitivity analyses for metformin exposure and bariatric surgery, and was maintained in prospective studies.[15] Likewise, women with PCOS alone and those with PCOS and GDM had higher risks of preeclampsia in the 2025 national register-based cohort in Sweden. These findings are biologically plausible as insulin resistance and impaired glucose metabolism are key characteristics of different phenotypes of PCOS and may play a role in endothelial and placental dysfunction.[20]

The present study also showed a significantly increased risk of preterm birth for women with PCOS. The increased risk of preterm birth, fetal growth restriction, and low birth weight among the children of women with PCOS was strongly supported by the results of the 2024 systematic review and meta-analysis of birth outcomes. The authors also found reduced mean birth weight for PCOS pregnancies, some of which were independent of maternal BMI. The mean birth weight and frequency of low-birth weight were lower in the PCOS group, which aligned with this general trend.[21]

This raised weight for low birth weight was also similar to the 2023 systematic review and meta-analysis of pregnancies with PCOS that were conceived with ART. In that review of 33 studies and 92,810 women with PCOS who participated in ART, women with PCOS who underwent ART were found to have higher odds of developing preeclampsia (OR 2.12), preterm birth (OR 1.29), and low birth weight (OR 1.29) compared to women without PCOS who underwent ART. The

consistency of direction in our study suggests that poor fetal growth and prematurity may be related to PCOS rather than to ART alone.[22]

The foremost strength of the present study was that it had a prospective cohort design, which enabled women with and without PCOS to be followed from pregnancy to delivery and enabled temporal assessment of preeclampsia and subsequent maternal and fetal outcomes. However, limited generalizability is due to the relatively small number of cases and controls, the single center, non-probability sampling, and residual confounding. Furthermore, the study failed to explore individual phenotypic characteristics of PCOS in detail to assess whether each of hyperandrogenism, insulin resistance, or PCOM was independently associated with preeclampsia. Further larger multicenter prospective studies which involve biochemical markers of endothelial dysfunction, angiogenic factors, insulin resistance and detailed phenotyping of PCOS would help elucidate the mechanisms underlying this association.

In summary, the current study provides new prospective evidence for a higher risk of preeclampsia and a wider range of other adverse maternal and fetal outcomes associated with PCOS. Its prevalence as a standalone risk factor, even after controlling for other important factors, indicates that it is not simply obesity or established metabolic risk factors that are responsible for the prevalence of PCOS. Hence, early diagnosis and optimizing the mother's metabolism, blood pressure management and increased surveillance of the fetus could be especially critical in women with PCOS.

Limitations

There were some limitations in this study. The study took place at a single tertiary care hospital that might restrict the external validity of the results. Selection bias may have been introduced due to the relatively small sample size and non-probability consecutive sampling. A multivariable analysis was conducted, but there was an inability to fully rule out residual confounding due to dietary patterns, physical activity, socioeconomic status, PCOS phenotype, PCOS treatment history, and duration of PCOS. Furthermore, the present study excluded assessment of circulating markers of insulin resistance, endothelial dysfunction, or angiogenic imbalance, which could have offered additional insights into the mechanism of association between PCOS and preeclampsia. This relatively small number of severe and early-onset preeclamptic cases also precluded subgroup analysis.

CONCLUSION

Women with PCOS had significantly greater risk of preeclampsia and of some adverse maternal and fetal outcomes such as gestational diabetes, cesarean delivery, preterm delivery, low birth weight and NICU admission. The relationship of PCOS with preeclampsia was still statistically significant following adjustment for important maternal and metabolic risk factors. These results highlight the need for early risk assessment and increased antenatal care of women with PCOS to ensure timely detection and management of conditions like hypertension and metabolic disorders.

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