



Neonatal outcomes in infants born to mothers with polycystic ovary syndrome (PCOS): a hospital-based longitudinal case-control study.

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ABSTRACT

Background:

One of the most common endocrine-metabolic conditions affecting women of reproductive age worldwide, polycystic ovarian syndrome (PCOS), has been shown to have negative effects on maternal health. However, the specific effects of this maternal illness on the newborn are still not well understood, especially in hospital settings in low-to-middle-income countries.

Methods:

A six-month longitudinal study was carried out in a hospital. Age- and parity-matched controls without PCOS (n = 85) and consecutive pregnant women with confirmed PCOS (n = 85) were included. At delivery and 24, 48, and 72 hours after delivery, neonatal parameters were noted. SPSS v26.0 was used for the statistical analysis; p < 0.05 was deemed significant.

Results:

Neonates of PCOS mothers demonstrated significantly higher rates of preterm birth (28.2% vs. 11.8%; p = 0.012), low birth weight (23.5% vs. 9.4%; p = 0.018), NICU admissions (21.2% vs. 8.2%; p = 0.024), neonatal hypoglycaemia (17.6% vs. 5.9%; p = 0.021), and lower mean APGAR scores at 1 minute (6.8 ± 1.2 vs. 7.6 ± 0.9 ; p = 0.003). Large-for-gestational-age (LGA) infants were also more frequent in the PCOS group (16.5% vs. 7.1%; p = 0.049).

Conclusion:

Maternal PCOS is independently associated with adverse neonatal outcomes encompassing both growth restriction and macrosomia, respiratory distress, and metabolic complications. Early antenatal identification and structured surveillance protocols for pregnancies complicated by PCOS are warranted to mitigate neonatal risk.

Recommendation:

Pregnancies complicated by PCOS should receive closer antenatal surveillance and neonatal monitoring to facilitate early identification and management of adverse neonatal outcomes.

Keywords: Polycystic ovary syndrome; neonatal outcomes; preterm birth; low birth weight; neonatal hypoglycaemia.

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INTRODUCTION

Depending on the diagnostic criteria used, the prevalence of PCOS is estimated to be between 8% and 13% worldwide, making it the most frequent reproductive endocrinopathy in women of childbearing capacity.[1] The pathogenesis of PCOS is complex, involving insulin resistance, chronic oligo-anovulation, and androgen excess, all of which affect the regulation of the hypothalamic-pituitary-ovarian axis.[2] Although PCOS has historically been thought of as an illness

of reproductive failure, new research increasingly suggests that it is a systemic metabolic disorder with far-reaching effects beyond fertility.

When pregnancies do occur in women with PCOS — whether spontaneously or following assisted reproduction — they carry a distinctly elevated risk profile relative to the general obstetric population.[3] Gestational diabetes mellitus (GDM) is reported in up to 40% of PCOS-affected pregnancies, driven by pre-existing insulin resistance that is further compounded



by placental hormonal shifts.[4] Hypertensive disorders of pregnancy, including gestational hypertension and preeclampsia, occur at two- to threefold higher rates in PCOS pregnancies compared to unaffected counterparts.[5] Miscarriage rates are also considerably elevated, particularly in women with concurrent hyperandrogenism and metabolic dysfunction.

Despite growing recognition of these maternal complications, the direct neonatal consequences attributable to maternal PCOS have received comparatively less systematic investigation.[6] It remains debated whether adverse neonatal outcomes arise primarily from comorbid gestational conditions — such as GDM or preeclampsia — or whether PCOS exerts an independent teratogenic or growth-modulatory effect on the developing foetus.[7] Several biological mechanisms have been proposed, including transplacental transfer of maternal androgens that alters foetal growth trajectories, epigenetic programming secondary to hyperinsulinaemia, and dysfunctional placental vascularity that impairs nutrient delivery.[8]

Published data on neonatal outcomes in PCOS pregnancies have yielded heterogeneous findings. Some investigations report a predominance of large-for-gestational-age (LGA) neonates — consistent with the macrosomic tendency observed in GDM-complicated pregnancies[9] — while others document significantly elevated rates of SGA and IUGR, suggesting a biphasic growth phenotype contingent on the degree of androgen excess and metabolic derangement.[10] Preterm birth, NICU admission, neonatal RDS, and neonatal hypoglycaemia have each been reported at elevated frequencies in this population, though systematic hospital-based longitudinal data from South Asian settings remain scarce.

India carries a substantial burden of PCOS, with regional prevalence studies reporting rates of 9.13% to 22.5% among adolescent and reproductive-age women.[11] Given the concurrent high burden of gestational diabetes and pregnancy-induced hypertension in this region, PCOS-complicated pregnancies represent a compounded risk scenario. Neonates born within this epidemiological milieu may therefore face greater vulnerability than those studied in high-income country cohorts.[12]

The present longitudinal study was undertaken at a tertiary care hospital to systematically characterise neonatal outcomes in infants delivered to PCOS-diagnosed mothers over a six-month observation period. By employing a matched control design and conducting serial neonatal assessments within the first 72 hours of life, the study aims to contribute robust, contextually relevant data to this evolving literature. The findings are intended to inform clinical surveillance protocols

and support evidence-based antenatal counselling for pregnant women with PCOS.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based longitudinal case-control study was conducted at a Tertiary Care Teaching Hospital, providing comprehensive obstetric, neonatal, and emergency healthcare services, over six months. The study aimed to compare neonatal outcomes between infants born to mothers diagnosed with polycystic ovary syndrome (PCOS) and those born to mothers without PCOS.

Ethical Considerations

The study protocol was reviewed and approved by the **Institutional Ethics Committee**. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment after explaining the objectives, procedures, potential benefits, and possible risks of the study. Participation was voluntary, and confidentiality of participants' information was maintained throughout the study.

Study Participants

Pregnant women attending the antenatal clinic or admitted to the labour room during the study period were screened for eligibility. Eligible participants were recruited consecutively until the required sample size was achieved.

The study included two groups:

Case Group (PCOS): Pregnant women with a confirmed diagnosis of PCOS established before pregnancy or during the first trimester according to the Rotterdam 2003 criteria, requiring the presence of at least two of the following: (1) oligo- or anovulation, (2) clinical or biochemical hyperandrogenism, and (3) polycystic ovaries on ultrasonography.

Control Group: Age- and parity-matched pregnant women without a previous diagnosis of PCOS, having regular menstrual cycles before conception and no evidence of clinical or biochemical hyperandrogenism.

Inclusion criteria

Included singleton pregnancies, maternal age between 18 and 40 years, gestational age of 28–42 weeks at delivery, and willingness to participate by providing written informed consent.



Exclusion criteria

Included multiple pregnancies, pre-existing type 1 or type 2 diabetes mellitus, thyroid disorders, major fetal congenital anomalies detected on antenatal ultrasonography, severe maternal systemic illnesses unrelated to PCOS, and participants who were lost to follow-up before delivery.

Sample Size

The sample size was calculated using a two-proportion formula based on previously reported NICU admission rates among neonates born to mothers with and without PCOS. Assuming 80% statistical power, a 5% level of significance, and expected NICU admission rates of 20% and 8%, respectively, the minimum required sample size was 78 participants in each group. To compensate for possible attrition, 85 participants were enrolled in each group, resulting in a total sample of 170 participants.

Data Collection

Baseline maternal demographic and clinical characteristics, including age, parity, pre-pregnancy body mass index (BMI), PCOS diagnostic parameters, glycaemic status, blood pressure profile, mode of delivery, and gestational age at delivery, were recorded using a structured data collection form.

Neonatal outcomes were assessed immediately after birth and at 24, 48, and 72 hours of life. Data collected included gestational age at birth, birth weight, APGAR scores at 1 and 5 minutes, small-for-gestational-age (SGA) and large-for-gestational-age (LGA) status, neonatal hypoglycaemia, respiratory distress syndrome, NICU admission, duration of NICU stay, and other early neonatal complications.

Measures to Minimize Bias

Several measures were implemented to minimize potential sources of bias. Consecutive recruitment of eligible participants reduced selection bias, while age- and parity-matching of controls minimized confounding. Uniform diagnostic criteria (Rotterdam criteria) were used for diagnosing PCOS, and standardized protocols were followed for neonatal assessment. Data collection was performed using predefined case record forms, and multivariable logistic

regression analysis was conducted to adjust for important confounding variables, including gestational diabetes mellitus, gestational hypertension, preterm birth, and mode of delivery.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean $\pm$ standard deviation (SD) and compared using the independent-samples t-test. Categorical variables were presented as frequencies and percentages. Binary logistic regression analysis was performed to determine whether maternal PCOS independently predicted adverse neonatal outcomes after adjustment for potential confounding variables, including gestational diabetes mellitus, gestational hypertension, preterm birth, and mode of delivery. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported, and a two-tailed p-value <0.05 was considered statistically significant.

RESULTS

During the study period, **214** pregnant women were screened for eligibility. Of these, **44** were excluded because they did not meet the inclusion criteria ($n = 21$), declined participation ($n = 15$), or had incomplete clinical records ($n = 8$). The remaining **170** participants fulfilled the eligibility criteria and were enrolled in the study, comprising **85 women with PCOS** and **85 age- and parity-matched controls**. All enrolled participants completed the study and were included in the final analysis.

Baseline Maternal Characteristics

The final analysis comprised 170 mother-neonate dyads ($n = 85$ in each group). The mean mother age (27.4 ± 3.8 years in cases versus 26.9 ± 4.1 years in controls; $p = 0.42$) and parity distribution of the two groups were similar. Pregnancy-induced hypertension (21.2% vs. 8.2%; $p = 0.018$), GDM (37.6% vs. 11.8%; $p < 0.001$), and mean pre-pregnancy BMI (26.8 ± 4.2 vs. 23.1 ± 3.5 kg/m²; $p < 0.001$) were all considerably higher in PCOS moms. The PCOS group had a considerably higher rate of Caesarean sections (49.4% vs. 29.4%; $p = 0.009$).



Table 1: Baseline Maternal Characteristics

Variable	PCOS Group (n = 85)	Control Group (n = 85)	p-value
Maternal Age (years, mean $\pm$ SD)	27.4 $\pm$ 3.8	26.9 $\pm$ 4.1	0.42
Pre-pregnancy BMI (kg/m ²)	26.8 $\pm$ 4.2	23.1 $\pm$ 3.5	<0.001
Gestational Diabetes Mellitus (%)	32 (37.6%)	10 (11.8%)	<0.001
Pregnancy-Induced Hypertension (%)	18 (21.2%)	7 (8.2%)	0.018
Caesarean Section (%)	42 (49.4%)	25 (29.4%)	0.009

Gestational Age and Delivery Outcomes

PCOS cases had a considerably higher rate of preterm birth than controls (28.2% vs. 11.8%; $p = 0.012$). The case group had a lower mean gestational age at delivery (37.6 $\pm$ 2.1 vs. 38.9 $\pm$ 1.4 weeks; $p = 0.001$). The majority of preterm births in both groups were late preterm deliveries (34–36+6 weeks).

Table 2: Gestational Age and Delivery Outcomes

Variable	PCOS Group	Control Group	p-value
Preterm Birth (<37 weeks)	24 (28.2%)	10 (11.8%)	0.012
Mean Gestational Age (weeks)	37.6 $\pm$ 2.1	38.9 $\pm$ 1.4	0.001

Birth Weight and Growth Classification

Neonates of PCOS women had a substantially lower mean birth weight (2,748 $\pm$ 512 g vs. 2,974 $\pm$ 448 g; $p = 0.002$). Compared to 9.4% of controls, 23.5% of PCOS neonates had

low birth weight (<2,500 g) ($p = 0.018$). In contrast, the PCOS group had a higher prevalence of LGA newborns (birth weight >90th centile) (16.5% vs. 7.1%; $p = 0.049$), which is consistent with concomitant GDM. SGA neonates made up 14.1% of cases compared to 5.9% of controls ($p = 0.067$).

Table 3: Birth Weight and Growth Classification

Variable	PCOS Group	Control Group	p-value
Mean Birth Weight (grams)	2748 $\pm$ 512	2974 $\pm$ 448	0.002
Low Birth Weight (<2500 g)	20 (23.5%)	8 (9.4%)	0.018
Small for Gestational Age (SGA)	12 (14.1%)	5 (5.9%)	0.067
Large for Gestational Age (LGA)	14 (16.5%)	6 (7.1%)	0.049

APGAR Scores and Neonatal Condition at Birth

PCOS-group neonates demonstrated significantly lower mean APGAR scores at 1 minute (6.8 $\pm$ 1.2 vs. 7.6 $\pm$ 0.9; $p = 0.003$).

At 5 minutes, mean APGAR scores remained lower in the case group (8.1 $\pm$ 1.0 vs. 8.7 $\pm$ 0.7; $p = 0.001$). Neonates with 5-minute APGAR scores <7 were significantly more common among PCOS deliveries (10.6% vs. 2.4%; $p = 0.029$).

Table 4: APGAR Scores and Neonatal Condition at Birth

Variable	PCOS Group	Control Group	p-value
APGAR at 1 min (mean $\pm$ SD)	6.8 $\pm$ 1.2	7.6 $\pm$ 0.9	0.003
APGAR at 5 min (mean $\pm$ SD)	8.1 $\pm$ 1.0	8.7 $\pm$ 0.7	0.001
APGAR <7 at 5 min	9 (10.6%)	2 (2.4%)	0.029



NICU Admission and Neonatal Morbidity

NICU admission rates were significantly higher in the PCOS group (21.2% vs. 8.2%; $p = 0.024$). Primary indications for NICU admission included neonatal respiratory distress (45%), hypoglycaemia (30%), jaundice requiring phototherapy

(15%), and feeding difficulties (10%). Neonatal hypoglycaemia was documented in 17.6% of PCOS neonates vs. 5.9% of controls ($p = 0.021$). Respiratory distress syndrome was diagnosed in 12.9% of PCOS infants vs. 4.7% of controls ($p = 0.047$). Duration of NICU stay was longer in PCOS neonates (4.2 ± 2.1 vs. 2.8 ± 1.4 days; $p = 0.038$).

Table 5: NICU Admission and Neonatal Morbidity

Variable	PCOS Group	Control Group	p-value
NICU Admission	18 (21.2%)	7 (8.2%)	0.024
Neonatal Hypoglycaemia	15 (17.6%)	5 (5.9%)	0.021
Respiratory Distress Syndrome	11 (12.9%)	4 (4.7%)	0.047
Duration of NICU Stay (days)	4.2 ± 2.1	2.8 ± 1.4	0.038

Multivariate Analysis

On binary logistic regression adjusting for GDM, gestational hypertension, preterm birth, and mode of delivery, maternal PCOS remained an independent predictor of NICU admission (adjusted OR 2.18; 95% CI 1.12–4.24; $p = 0.022$) and neonatal hypoglycaemia (adjusted OR 2.47; 95% CI 1.08–5.63; $p = 0.031$), suggesting a PCOS-specific neonatal risk beyond that attributable to associated comorbidities alone.

DISCUSSION

The findings of this hospital-based longitudinal study affirm that maternal PCOS is associated with a significantly elevated burden of adverse neonatal outcomes, extending beyond the contribution of recognised comorbidities such as gestational diabetes and hypertensive disorders of pregnancy. These results align with and extend prior literature suggesting that PCOS imposes an independent neonatal risk not fully explained by its associated metabolic complications.[13]

The elevated rate of preterm birth (28.2%) observed in PCOS pregnancies in this cohort is consistent with findings from large Nordic registry studies.[14] Kollmann et al. reported a significantly increased risk of preterm delivery in PCOS pregnancies compared to spontaneously conceived controls, a finding replicated in a subsequent meta-analysis by Qin et al.[15] The biological basis for this association is multifactorial: uterine hyperandrogenism may alter myometrial contractility; concurrent preeclampsia frequently necessitates iatrogenic preterm delivery; and endometrial dysfunction inherent to PCOS may impair placental anchoring, predisposing to cervical incompetence.

The dual phenotype of growth outcomes — simultaneous elevation of both LGA and LBW rates — is a clinically important finding that mirrors observations reported by Boomsma et al. in a Dutch prospective cohort.[16] This

bidirectional growth perturbation is mechanistically explicable: LGA/macrosomic infants predominantly arise from PCOS pregnancies complicated by GDM, wherein foetal hyperinsulinaemia drives excess adipogenesis[17]; while LBW and SGA outcomes likely reflect placental dysfunction driven by androgen-mediated vascular impairment and intrauterine growth restriction.

The significantly lower APGAR scores in PCOS neonates, particularly at 1 minute, are clinically meaningful and have not been uniformly reported across prior studies.[18] Possible explanations include higher rates of prematurity and emergency caesarean section in the PCOS cohort, both of which are associated with suboptimal immediate neonatal adaptation. Perinatal asphyxia risk is further compounded by potential placental insufficiency in the hyperandrogenic milieu.

Neonatal hypoglycaemia, recorded in 17.6% of PCOS-exposed neonates, reflects the downstream metabolic consequences of maternal hyperinsulinaemia and GDM. Transplacental glucose transfer stimulates foetal pancreatic beta-cell hyperplasia; following cord clamping, persistent foetal hyperinsulinaemia in the absence of maternal glucose supply precipitates hypoglycaemia.[19] This risk is not confined to clinically diagnosed GDM cases — subclinical insulin resistance in PCOS, even without formal GDM criteria, may be sufficient to disrupt foetal glucose homeostasis.[20]

The elevated NICU admission rate (21.2%) and its persistence as an independent risk factor on multivariate analysis (adjusted OR 2.18) highlight that maternal PCOS imparts neonatal risk beyond what is accounted for by concurrent gestational complications. This finding has immediate clinical relevance: it supports the case for designating all deliveries to PCOS-diagnosed mothers as high-risk irrespective of



comorbidity status, with neonatology team presence at delivery and structured glucose monitoring protocols applied universally.[21]

The androgen-mediated pathway merits specific discussion. Women with PCOS exhibit substantially elevated circulating androgens, which cross the placental barrier and may induce epigenetic alterations in the developing foetus.[22] Animal models of prenatal androgen exposure have documented offspring effects including altered glucose metabolism, disrupted hypothalamic-pituitary function, and cardiovascular remodelling.[23] Whether similar programming effects operate in human PCOS pregnancies is an area of active investigation, with implications extending well beyond the neonatal period into long-term offspring health.

Respiratory distress syndrome, observed in 12.9% of PCOS neonates vs. 4.7% of controls, is largely attributable to preterm delivery in this cohort. Prematurity-associated surfactant deficiency remains the dominant pathological mechanism. However, there is emerging evidence that foetal lung maturation may be delayed in GDM-complicated pregnancies independent of gestational age, as hyperinsulinaemia antagonises glucocorticoid-mediated surfactant synthesis.[24] This mechanism may be operative even in near-term PCOS neonates, contributing to the observed excess of RDS.

A notable strength of this study is its longitudinal design with serial neonatal assessments extending to 72 hours, allowing capture of delayed-onset complications such as hypoglycaemia and jaundice that might be missed by cross-sectional delivery-room assessment alone. The matched control design minimises confounding by maternal age and parity. However, several limitations merit acknowledgement.

Generalizability

Although this study was conducted at a single tertiary care teaching hospital, the findings provide valuable insights into the neonatal outcomes associated with maternal polycystic ovary syndrome (PCOS) in a hospital-based setting. The use of age- and parity-matched controls and standardized neonatal assessments enhances the internal validity of the findings. However, variations in patient demographics, healthcare infrastructure, and clinical practices across different institutions and regions may limit the direct applicability of the results to all populations. Therefore, multicentre studies involving larger and more diverse populations are warranted to improve the external validity and generalizability of these findings.

CONCLUSION

This hospital-based longitudinal study provides systematic evidence that infants born to mothers with polycystic ovary syndrome face a substantially elevated risk of adverse neonatal outcomes, including preterm birth, low birth weight, compromised APGAR scores, neonatal hypoglycaemia, and NICU admission. Critically, maternal PCOS retained its independent association with NICU admission and neonatal hypoglycaemia even after adjusting for concurrent gestational diabetes and hypertensive disorders, suggesting biological mechanisms intrinsic to PCOS that operate beyond its recognised comorbidities.

These findings underscore the imperative of recognising maternal PCOS as a distinct, independent neonatal risk determinant within antenatal risk stratification frameworks. Clinically, this necessitates multidisciplinary antenatal surveillance incorporating early glycaemic screening, growth monitoring with serial foetal biometry, planned delivery at a centre with tertiary neonatal facilities, and standardised postpartum neonatal glucose monitoring for all PCOS-affected pregnancies. Future longitudinal research tracking PCOS-exposed offspring into childhood and adolescence is warranted to characterise long-term metabolic and neurodevelopmental trajectories in this high-risk cohort.

Limitations

This study has several limitations. First, it was conducted at a single tertiary care centre, which may limit the generalizability of the findings. Second, the relatively small sample size may have reduced the statistical power to detect differences in less frequent neonatal outcomes. Third, neonatal follow-up was limited to the first 72 hours after birth, preventing assessment of long-term developmental and metabolic outcomes. Additionally, residual confounding due to unmeasured maternal or environmental factors cannot be completely excluded despite statistical adjustment for major confounders. Future multicentre studies with larger sample sizes

Recommendations

Pregnancies complicated by PCOS should be recognized as high-risk and managed through multidisciplinary antenatal care involving obstetricians, endocrinologists, and neonatologists. Early screening for gestational diabetes mellitus and hypertensive disorders, regular fetal growth surveillance, and appropriate intrapartum planning should be incorporated into routine clinical practice. Neonates born to mothers with PCOS should undergo careful monitoring for hypoglycaemia, respiratory complications, and the need for neonatal intensive care. Future multicentre prospective studies



with long-term follow-up are required to evaluate the metabolic and neurodevelopmental outcomes of offspring born to mothers with PCOS.

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List of Abbreviations

- PCOS – Polycystic Ovary Syndrome
- BMI – Body Mass Index
- GDM – Gestational Diabetes Mellitus
- PIH – Pregnancy-Induced Hypertension
- NICU – Neonatal Intensive Care Unit
- LBW – Low Birth Weight
- SGA – Small for Gestational Age
- LGA – Large for Gestational Age
- RDS – Respiratory Distress Syndrome
- OR – Odds Ratio
- aOR – Adjusted Odds Ratio
- CI – Confidence Interval
- SD – Standard Deviation

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Conflict of Interest

The authors declare that they have no competing interests.

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Data Availability

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

Author Contributions

Author Contribution

Ranvijay Kumar Conceptualization, study design, data collection, statistical analysis, manuscript drafting
Ilika Bharti Methodology, supervision, manuscript review and editing

Omkar Kumar Literature review, data collection, data interpretation, manuscript editing

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