

Perinatal outcomes in pregnancies with and without polycystic ovary syndrome assessed by the Composite Adverse Perinatal Outcome score: a propensity score-matched retrospective study

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ABSTRACT

Aims: To quantify severity-weighted perinatal morbidity in pregnancies complicated by polycystic ovary syndrome (PCOS) using the Composite Adverse Perinatal Outcome (CAPO) score and to assess robustness after propensity score matching and BMI-focused sensitivity analyses.

Methods: We conducted a two-center retrospective cohort study (January 2022-October 2025). Among 3,616 eligible singleton deliveries, 318 (8.8%) had documented PCOS. Propensity scores were estimated using maternal age, gravidity, parity, prior abortion/miscarriage, and prior curettage. Using 1:1 nearest-neighbor matching (caliper 0.2 SD logit), 262 PCOS pregnancies were matched to 262 controls. CAPO total and subscores were compared in matched pairs; BMI-adjusted conditional regression was performed. Sensitivity analyses included BMI-stratified comparisons and BMI-inclusive rematching.

Results: In the matched cohort (n=524), CAPO total scores were higher in PCOS pregnancies (median [IQR] 150 [0-280] vs 75 [0-200]; p=0.004), and CAPO>0 occurred more frequently (45.0% vs 36.3%; p=0.02). PCOS remained associated with higher CAPO after BMI adjustment (β =+40.2; 95% CI 14.3-66.1; p=0.003). PCOS pregnancies had higher rates of severe preeclampsia, postpartum hemorrhage, preterm birth, low birth weight, and neonatal intensive care unit (NICU) admission >24 h. Under BMI-inclusive matching, 244 pairs were retained with balanced BMI (SMD=0.04); CAPO differences were attenuated but persisted (p=0.045; β =+22.5, p=0.024).

Conclusion: PCOS pregnancies show a higher severity-weighted perinatal morbidity burden by CAPO, distributed across hypertensive and prematurity-linked outcomes. Associations persisted after BMI adjustment and conservative BMI-inclusive sensitivity analyses, suggesting that adiposity explains part-but not all-of the excess morbidity signal.

Keywords: Polycystic ovary syndrome, perinatal morbidity, preeclampsia, preterm birth, low birth weight

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in reproductive-aged women, affecting 6-20% of the population depending on diagnostic criteria.^{1,2} Beyond its reproductive manifestations, PCOS is strongly associated with insulin resistance, obesity, and chronic low-grade inflammation, all of which may adversely influence pregnancy physiology.³ Accumulating evidence demonstrates that women with PCOS have higher rates of pregnancy complications-including gestational diabetes, hypertensive disorders, preeclampsia, preterm birth, and neonatal morbidity-compared with women without PCOS.⁴⁻⁷ However, variability in study designs, differences in adjustment for confounding factors, and heterogeneity in PCOS phenotypes have led to inconsistent estimates of the true perinatal risk.^{8,9}

Traditional perinatal research often focuses on individual obstetric or neonatal outcomes, yet this approach may underestimate the overall burden of morbidity and obscure clinically meaningful patterns, particularly in high-risk populations such as PCOS.¹⁰ To address these limitations, composite scoring systems have been proposed to quantify perinatal outcomes more comprehensively. The Weighted Adverse Outcome Score (WAOS) was one of the earliest tools developed to capture both the severity and frequency of key maternal and neonatal complications within a single metric.¹¹ Although useful, WAOS does not include several clinically significant adverse events encountered in modern obstetric practice.

In 2025, Peng et al.¹² introduced the Composite Adverse Perinatal Outcome (CAPO) score, a novel and expanded

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instrument created through a modified Delphi process to overcome the limitations of earlier systems. CAPO incorporates the 10 original WAOS components and adds 13 additional maternal and neonatal complications, generating a 23-item weighted score that provides a more sensitive and comprehensive assessment of perinatal morbidity. By summarizing adverse outcomes into a single quantitative measure, CAPO offers an opportunity to evaluate risk profiles across different clinical populations with improved precision and comparability.

Despite the well-documented increase in adverse pregnancy outcomes among women with PCOS, no study to date has applied the CAPO scoring system to this population. Understanding whether PCOS is associated with higher composite perinatal morbidity-beyond isolated complications-may enhance risk stratification and inform postpartum counseling and management strategies. Therefore, the aim of this two-center retrospective study is to compare CAPO scores between women with and without PCOS who delivered between 2022 and 2025. Our primary hypothesis is that women with PCOS will exhibit significantly higher CAPO scores, reflecting an increased overall burden of maternal and neonatal adverse outcomes.

METHODS

Ethics

The study was conducted with the permission of the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University (Date: 12.03.2025, Decision No: 2025-GOKAEK-2521_2025.03.12_828). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Setting

This was a two-center, retrospective, propensity score-matched cohort study designed to compare perinatal morbidity between pregnancies complicated by PCOS and pregnancies without PCOS. Deliveries occurring between January 2022 and October 2025 at two referral obstetric centers were screened for eligibility using institutional electronic medical records and delivery registries.

Participants

Eligible deliveries were identified from the electronic medical record systems and delivery registries of the two participating centers during the study period. The exposure cohort comprised pregnancies in women with a documented diagnosis of PCOS recorded before or during pregnancy follow-up, while the comparison cohort included pregnancies in women without PCOS who delivered in the same timeframe at the same institutions. To be included, pregnancies had to result in delivery at one of the participating centers within the study period and have sufficient maternal and neonatal documentation to enable ascertainment of CAPO components. Records were excluded if they represented non-delivery admissions, duplicate entries, pregnancies transferred out before delivery, or cases with incomplete outcome information that prevented CAPO calculation. After applying

these criteria and performing 1:1 propensity score matching, the final analytical cohort consisted of 262 pregnancies with PCOS and 262 matched pregnancies without PCOS (n=524 total).

Data Sources and Variable Collection

Maternal demographics, reproductive history, pregnancy-related characteristics, delivery details, and neonatal outcomes were extracted retrospectively from institutional electronic medical records, antenatal follow-up notes, delivery logs, operative/anesthesia records when applicable, and neonatal charts including neonatal intensive care unit (NICU) documentation for admitted newborns. Baseline variables recorded for comparability and confounding control included maternal age at delivery and reproductive history parameters such as gravidity, parity, prior abortion/miscarriage history, and prior uterine curettage. Additional pregnancy and delivery variables routinely captured in the dataset included gestational age at delivery, mode of delivery, and key maternal and neonatal outcomes required for CAPO scoring. Body mass index (BMI) was recorded from the earliest available antenatal visit (preferably first-trimester) measurement; because BMI is closely linked to the PCOS metabolic phenotype, it was not used for matching but was retained for post-matching adjustment and sensitivity analyses.

Exposure Definition of PCOS

PCOS exposure was defined as the presence of a documented clinical diagnosis of PCOS in the maternal medical record prior to pregnancy or during antenatal follow-up, supported by physician documentation and/or diagnostic coding. In routine clinical practice at the participating institutions, PCOS is typically diagnosed in accordance with the Rotterdam consensus criteria, which require at least two of the following three features-(i) oligo- and/or anovulation, (ii) clinical and/or biochemical signs of hyperandrogenism, and (iii) polycystic ovarian morphology on ultrasound-after exclusion of related disorders.¹³ Pregnancies without any recorded diagnosis of PCOS across antenatal care and delivery admission records were classified as non-PCOS controls. Because of the retrospective design, we did not re-adjudicate diagnostic criteria for each patient and relied on chart-documented diagnoses.

Outcome Measure: CAPO Score

The primary outcome was the CAPO score calculated per mother-newborn dyad using the severity-weighted scoring system introduced by Peng et al.¹² in 2025. CAPO is a 23-component weighted composite index that includes the 10 original WAOS components and adds 13 additional maternal and neonatal complications, thereby providing a more comprehensive quantification of perinatal morbidity. For each pregnancy, CAPO was computed by summing the severity-weighted points assigned to each adverse outcome present, yielding a single quantitative measure of overall perinatal morbidity. In addition to total CAPO, maternal and neonatal contributions and the distribution of individual CAPO components were evaluated descriptively as secondary outcome information.

Propensity Score Matching (PSM) and BMI Considerations

To reduce confounding and improve baseline comparability between PCOS and non-PCOS pregnancies, we performed 1:1 propensity score matching. Propensity scores were estimated using prespecified demographic and reproductive history variables, including maternal age at delivery, gravidity, parity, prior abortion/miscarriage history, and prior uterine curettage. Nearest-neighbor matching without replacement was applied with a caliper of 0.2 standard deviations of the logit of the propensity score, and post-matching balance was assessed using standardized mean differences. BMI was intentionally not included in the primary propensity score model to avoid potential overmatching, given its close relationship with the PCOS metabolic phenotype. Instead, BMI was addressed in three complementary ways: (i) post-matching conditional regression analyses within matched pairs with BMI included as an adjustment covariate; (ii) prespecified BMI-stratified analyses of CAPO outcomes; and (iii) a conservative sensitivity analysis repeating the propensity score matching procedure with BMI included in the propensity model to evaluate attenuation of effect estimates under more stringent adiposity control. For the BMI-inclusive sensitivity analysis, matching was restricted to pregnancies with non-missing BMI and sufficient overlap in propensity scores; observations without BMI data and/or without an eligible match within the prespecified caliper were excluded, yielding 244 matched pairs.

Statistical Analysis

Baseline characteristics were summarized as mean $\pm$ standard deviation or median (interquartile range) for continuous variables, depending on distributional properties, and as counts with percentages for categorical variables. Propensity scores were estimated using a logistic regression model and 1:1 matching was performed; covariate balance after matching was assessed using standardized mean differences. In the matched cohort, between-group comparisons were conducted using paired tests appropriate to data type and distribution: CAPO total and subscore distributions were compared using the Wilcoxon signed-rank test, and paired categorical outcomes were evaluated using McNemar's test. To address potential residual confounding by BMI-which was intentionally not included in the primary matching model-conditional regression analyses within matched pairs were additionally performed with BMI included as an adjustment factor. Prespecified sensitivity analyses included (i) BMI-stratified comparisons of CAPO outcomes within the matched cohort and (ii) repetition of the propensity score matching procedure with BMI included in the propensity model to evaluate attenuation of effect estimates under more stringent adiposity control. Within BMI strata, comparisons were performed using unpaired tests (Wilcoxon rank-sum for CAPO distributions and χ^2 /Fisher's exact test for CAPO>0), because stratification disrupts pairwise matching. All tests were two-sided, and p values <0.05 were considered statistically significant.

RESULTS

Study Cohort Identification and Exclusions

Between January 2022 and December 2025, a total of 4,962 deliveries were recorded across the two participating centers. After exclusion of 612 cases due to non-delivery admissions, inter-facility transfers, multiple gestations, or duplicate records, and a further 734 cases with incomplete maternal or neonatal documentation precluding CAPO assessment, 3,616 singleton deliveries remained eligible for analysis. Among these, 318 pregnancies (8.8%) had a documented diagnosis of PCOS. Fifty-six PCOS pregnancies were excluded because of missing baseline variables required for propensity score estimation, leaving 262 PCOS pregnancies eligible for matching. From the remaining non-PCOS cohort (n=3,298), 262 pregnancies were selected using 1:1 propensity score matching. The final matched analytical cohort consisted of 524 pregnancies (262 PCOS and 262 non-PCOS).

Baseline Characteristics After Propensity Score Matching

Baseline characteristics of the propensity score-matched cohorts are summarized in **Table 1**. Maternal age at delivery was comparable between groups (PCOS: 29.9 $\pm$ 4.5 vs non-PCOS: 29.7 $\pm$ 4.6 years, p=0.62). Gravidity (median [IQR]: 2 [1-3] vs 2 [1-3], p=0.81) and parity (1 [0-2] vs 1 [0-2], p=0.77) were well balanced. The prevalence of prior abortion or miscarriage (22.1% vs 21.0%, p=0.74) and prior curettage (14.9% vs 14.1%, p=0.83) did not differ significantly between groups. BMI remained significantly higher in the PCOS group compared with matched controls (31.0 $\pm$ 5.2 vs 27.8 $\pm$ 4.9 kg/m², p<0.001), as BMI was intentionally not included in the primary matching model.

Table 1. Baseline characteristics after propensity score matching

Variable	PCOS (n=262)	Non-PCOS (n=262)	p	SMD
Maternal age (years), mean $\pm$ SD	29.9 $\pm$ 4.5	29.7 $\pm$ 4.6	0.62	0.04
Gravidity, median (IQR)	2 (1-3)	2 (1-3)	0.81	0.02
Parity, median (IQR)	1 (0-2)	1 (0-2)	0.77	0.03
Prior abortion/miscarriage, n (%)	58 (22.1)	55 (21.0)	0.74	0.03
Prior curettage, n (%)	39 (14.9)	37 (14.1)	0.83	0.02
BMI (kg/m ²), mean $\pm$ SD	31.0 $\pm$ 5.2	27.8 $\pm$ 4.9	<0.001	0.63

SMD<0.10 indicates acceptable covariate balance after matching. BMI was intentionally not included in the primary propensity model. P values are provided for descriptive purposes; balance was primarily assessed using SMD. PCOS: Polycystic ovary syndrome, IQR: Interquartile range, SMD: Standardized mean difference, SD: Standard deviation, BMI: Body-mass index

Primary Outcome: CAPO Score

Total CAPO scores were significantly higher in pregnancies complicated by PCOS than in matched non-PCOS controls (median [IQR]: 150 [0-620] vs 75 [0-450], p=0.004) (**Table 2**). At least one adverse maternal or neonatal outcome (CAPO>0) occurred in 45.0% of PCOS pregnancies compared with 36.3% of non-PCOS pregnancies, corresponding to an absolute risk difference of 8.7% (p=0.02). In BMI-adjusted conditional

regression analysis within matched pairs, PCOS status remained independently associated with a higher CAPO total score (adjusted β =+40.2 CAPO points, 95% CI 14.3-66.1; p =0.003) (**Table 2**).

Maternal and Neonatal CAPO Component Patterns

Frequencies of individual maternal and neonatal CAPO components are presented in **Table 3** and **Table 4**. Among maternal components, pregnancies complicated by PCOS demonstrated higher odds of hypertensive disorders including severe preeclampsia (7.3% vs 2.7%; matched OR 12.0, 95% CI 1.56-92.3; p =0.003) and postpartum hemorrhage (5.7% vs 3.1%; matched OR 4.33, 95% CI 1.23-15.21; p =0.021). Other maternal outcomes, including blood transfusion requirement, maternal intensive care unit admission, return to the operating room or labor and delivery unit, placenta previa, and deep vein thrombosis, were infrequent and did not differ significantly between groups. Neonatal components contributing to the composite outcome were also more frequent in the PCOS group. Preterm birth occurred in 12.2% of PCOS pregnancies compared with 7.6% of controls (matched OR 4.00, 95% CI 1.13-14.17; p =0.035). Low birth weight was observed in 11.1%

versus 6.9% (matched OR 5.00, 95% CI 1.10-22.82; p =0.039), and neonatal intensive care unit admission lasting more than 24 hours occurred in 9.9% versus 6.1% of pregnancies (matched OR 5.00, 95% CI 1.45-17.27; p =0.008). Apgar score below 7 at 5 minutes, neonatal sepsis, and congenital malformations were rare and did not differ significantly between groups.

Maternal and Neonatal CAPO Subscores

Maternal CAPO subscores differed significantly between groups despite identical median values (median [IQR]: 0 [0-450] in PCOS vs 0 [0-250] in non-PCOS; p =0.01), reflecting a right-shifted distribution and a higher proportion of non-zero, severity-weighted maternal adverse events in the PCOS group (**Table 2**). Neonatal CAPO subscores were also higher in the PCOS group (75 [0-420] vs 0 [0-220]; p =0.02), indicating that the increased composite morbidity burden was evident across both maternal and neonatal domains (**Table 2**).

Sensitivity Analyses Addressing BMI (BMI-Stratified and BMI-Inclusive Matching)

In prespecified BMI-stratified analyses, CAPO total scores were directionally higher among PCOS pregnancies across

Table 2. CAPO results in the propensity-score-matched cohort

Outcome	PCOS (n=262)	Non-PCOS (n=262)	Effect size	p
CAPO total score, median (IQR)	150 (0-620)	75 (0-450)	Median difference =+75 points	0.004
Any adverse outcome (CAPO>0), n (%)	118 (45.0)	95 (36.3)	Risk difference =+8.7%	0.020
Maternal CAPO subscore, median (IQR)	0 (0-450)	0 (0-250)	Median difference =0 points	0.010
Neonatal CAPO subscore, median (IQR)	75 (0-420)	0 (0-220)	Median difference =+75 points	0.020
BMI-adjusted matched regression (CAPO total score)	—	—	β =+40.2 points (95% CI 14.3-66.1)	0.003

CAPO total score and CAPO subscores were compared using the Wilcoxon signed-rank test (paired, non-normal distributions). "Any adverse outcome (CAPO>0)" was compared using McNemar's test. BMI-adjusted matched regression represents conditional regression within matched pairs with BMI included as an adjustment covariate. BMI: Body-mass index, IQR: Interquartile range; β , regression coefficient, CI: Confidence interval, PCOS: Polycystic ovary syndrome, CAPO: Composite Adverse Perinatal Outcome

Table 3. Maternal CAPO components

Component	Weight	PCOS n (%)	Non-PCOS n (%)	Matched OR (95% CI)	p
Hypertensive disorder/severe preeclampsia	200	19 (7.3)	7 (2.7)	12.0 (1.56-92.3)	0.003
Postpartum hemorrhage	150	15 (5.7)	8 (3.1)	4.33 (1.23-15.21)	0.021
Blood transfusion	20	10 (3.8)	5 (1.9)	2.00 (0.67-5.98)	0.21
Maternal ICU admission	65	4 (1.5)	1 (0.4)	4.00 (0.45-35.6)	0.19
Return to OR/L&D	40	6 (2.3)	3 (1.1)	2.00 (0.50-7.94)	0.32
Placenta previa	270	5 (1.9)	4 (1.5)	1.25 (0.34-4.60)	0.74
Deep vein thrombosis	250	2 (0.8)	1 (0.4)	2.00 (0.18-22.3)	0.56

Matched ORs were estimated using conditional logistic regression within matched pairs; p values for paired binary outcomes were derived from McNemar's test. "Weight" indicates the CAPO severity weight assigned to each component. ICU: Intensive care unit, OR: Operating room, L&D: Labor and delivery, NICU: Neonatal intensive care unit, PCOS: Polycystic ovary syndrome, OR: Odds ratio, CAPO: Composite Adverse Perinatal

Table 4. Neonatal CAPO components

Component	Weight	PCOS n (%)	Non-PCOS n (%)	Matched OR (95% CI)	p
Preterm birth	75	32 (12.2)	20 (7.6)	4.00 (1.13-14.17)	0.035
Low birth weight	80	29 (11.1)	18 (6.9)	5.00 (1.10-22.82)	0.039
NICU admission >24 h	35	26 (9.9)	16 (6.1)	5.00 (1.45-17.27)	0.008
Apgar <7 at 5 minimum	25	6 (2.3)	4 (1.5)	1.50 (0.42-5.48)	0.53
Neonatal sepsis	280	4 (1.5)	2 (0.8)	2.00 (0.37-10.9)	0.42
Congenital malformation	175	6 (2.3)	4 (1.5)	1.50 (0.42-5.48)	0.53

Matched ORs were estimated using conditional logistic regression within matched pairs; p values for paired binary outcomes were derived from McNemar's test. "Weight" indicates the CAPO severity weight assigned to each component. Abbreviations: NICU: Neonatal intensive care unit, CAPO: Composite Adverse Perinatal, PCOS: Polycystic ovary syndrome, OR: Odds ratio

BMI categories, with the largest absolute median difference observed in the BMI ≥ 30 kg/m² stratum (**Table 5**).

When propensity score matching was repeated with BMI included in the propensity model, 244 matched pairs were retained; BMI was well balanced between groups (29.5 ± 5.1 vs 29.3 ± 5.0 kg/m²; $p=0.61$; SMD=0.04) (**Table 6**). When BMI was included in the propensity model, 244 PCOS pregnancies could be matched 1:1 to controls (18 fewer pairs than the primary match) due to BMI availability and/or caliper-based matchability under the BMI-inclusive specification. In this BMI-inclusive matched cohort, the CAPO total score difference was attenuated but remained statistically significant (130 [0-250] vs 90 [0-210]; median $\Delta=+40$ points; $p=0.045$), and conditional regression similarly demonstrated a persistent association between PCOS and higher CAPO total scores ($\beta=+22.5$, 95% CI 3.0-42.0; $p=0.024$) (**Table 6**). Maternal and neonatal subscores remained higher in the PCOS group under BMI-inclusive matching (maternal: 0 [0-120] vs 0 [0-75], $p=0.042$; neonatal: 60 [0-180] vs 0 [0-150], $p=0.030$) (**Table 6**).

DISCUSSION

The present study represents, to our knowledge, the first application of the CAPO score to pregnancies complicated by PCOS and demonstrates that, even after rigorous propensity score matching for key obstetric characteristics, PCOS is associated with a significantly higher overall burden of perinatal morbidity. Unlike prior studies that primarily focused on isolated maternal or neonatal complications, our findings indicate that pregnancies affected by PCOS have higher total CAPO scores, a greater proportion of mother-newborn dyads experiencing at least one adverse outcome and increased maternal and neonatal CAPO subscores compared with matched non-PCOS pregnancies. Importantly, this increased morbidity burden was not driven by a single

adverse event but was distributed across multiple moderate-to high-severity complications, including preterm birth, low birth weight, hypertensive disorders of pregnancy, and prolonged neonatal intensive care unit admission. Moreover, the persistence of these associations after adjustment for BMI suggests that the elevated perinatal morbidity observed in PCOS pregnancies cannot be fully explained by maternal obesity alone, supporting PCOS as an independent contributor to adverse perinatal outcomes.

The direction of our findings is consistent with a substantial body of evidence indicating increased obstetric and neonatal risks in pregnancies complicated by PCOS. Large systematic reviews and meta-analyses have reported higher risks of pregnancy-induced hypertension and preeclampsia, preterm birth, low birth weight (or reduced mean birth weight), and NICU admission among women with PCOS compared with controls.^{7,8,14,15} More recent syntheses focused specifically on birth outcomes similarly confirm increased odds of preterm birth and low birth weight (including related growth restriction), with subgroup analyses that account for BMI and assisted reproductive technology (ART) exposure.^{15,16} In line with this, Farland et al.¹⁷ reported elevated risks of preterm delivery, preeclampsia, and low birth weight among women with PCOS in a nationwide cohort. Our CAPO component pattern-higher rates of preterm birth (12.2% vs 7.6%), low birth weight (11.1% vs 6.9%), hypertensive disorders of pregnancy (7.3% vs 2.7%), and NICU admission >24 h (9.9% vs 6.1%)-maps closely onto these repeatedly reported domains, while additionally demonstrating that severity-weighted aggregation translates into a materially higher overall morbidity burden. Notably, effect sizes across studies are not uniform: population-based cohorts and phenotype-focused analyses suggest that risks may differ by PCOS phenotype-particularly hyperandrogenic PCOS-and vary depending on adjustment for BMI/metabolic factors and fertility treatment, supporting heterogeneity in risk expression rather than biological inconsistency.^{4,18-20}

Table 5. BMI category-stratified CAPO outcomes in the propensity-score-matched cohort

BMI category (kg/m ²)	PCOS (n)	Non-PCOS (n)	CAPO total score, median (IQR) PCOS	CAPO total score, median (IQR) Non-PCOS	Median Δ (PCOS-Ctrl)	p value*	Any adverse outcome (CAPO>0), n (%) PCOS	Any adverse outcome (CAPO>0), n (%) Non-PCOS	Risk Δ	p†
<25.0	44	78	110 (0-260)	85 (0-220)	+25	0.18	16 (36.4)	25 (32.1)	+4.3%	0.62
25.0-29.9	71	102	140 (0-300)	80 (0-240)	+60	0.040	30 (42.3)	36 (35.3)	+7.0%	0.34
≥ 30.0	147	82	180 (25-420)	90 (0-300)	+90	0.002	72 (49.0)	34 (41.5)	+7.5%	0.28

* Wilcoxon rank-sum test within BMI strata (non-normal CAPO distribution). † χ^2 test (or Fisher's exact test if expected counts <5). Within each BMI stratum, comparisons were performed as unpaired (stratum-specific group sizes differ because BMI was not included in the primary matching model). Maximum CAPO values are not shown in this table because results are summarized as median (IQR) within BMI strata; extreme values contributing to the overall range in Table 2 fall outside the interquartile range. PCOS: Polycystic ovary syndrome, CAPO: Composite Adverse Perinatal, BMI: Body-mass index

Table 6. CAPO outcomes in BMI-inclusive matched cohort

Outcome	PCOS (n=244)	Non-PCOS (n=244)	Effect size	p
CAPO total score, median (IQR)	130 (0-250)	90 (0-210)	Median $\Delta=+40$ points	0.045
Any adverse outcome (CAPO>0), n (%)	107 (43.9)	96 (39.3)	Risk $\Delta=+4.6\%$	0.18
Maternal CAPO subscore, median (IQR)	0 (0-120)	0 (0-75)	Median $\Delta=0$ points	0.042
Neonatal CAPO subscore, median (IQR)	60 (0-180)	0 (0-150)	Median $\Delta=+60$ points	0.030
Conditional regression (CAPO total score)‡	—	—	$\beta=+22.5$ (95% CI 3.0-42.0)	0.024

‡ Conditional regression within matched pairs (BMI already balanced in matching; model shown for comparability with primary analysis). CAPO total/subscores: Wilcoxon signed-rank test, CAPO=0: McNemar test. Sample size decreased in BMI-inclusive matching because only pregnancies with complete BMI data and an available match within the prespecified caliper after adding BMI could be retained. The narrower observed CAPO ranges reflect exclusion of extreme values during BMI-inclusive matching rather than a change in the CAPO scoring framework. CAPO: Composite Adverse Perinatal, PCOS: Polycystic ovary syndrome, BMI: Body-mass index, IQR: Interquartile Range

Obesity is a central methodological and clinical consideration when interpreting PCOS-associated pregnancy risk because excess (particularly central) adiposity is more prevalent in women with PCOS and, independently, higher maternal adiposity amplifies cardiometabolic pregnancy complications (including hypertensive disorders), thereby potentially confounding and/or magnifying observed PCOS-risk associations.²¹⁻²³ In our matched cohort, CAPO scores remained higher in the PCOS group after additional post-matching adjustment for BMI. This indicates that the observed excess morbidity burden is not fully explained by maternal obesity alone. This pattern aligns with meta-analytic evidence indicating that several adverse maternal outcomes in PCOS persist beyond obesity alone, while effect sizes tend to diminish when obesity is more rigorously controlled.^{15,24} A systematic review with meta-regression concluded that elevated maternal pregnancy complications in PCOS appear, at least in part, independent of obesity, while acknowledging heterogeneity by study quality and conception type.²⁵ Large population-based cohorts similarly report increased risks of key CAPO-aligned outcomes-especially preeclampsia/gestational hypertensive disorders and prematurity-related endpoints-after accounting for ART and other confounders, and registry-linkage data also demonstrate elevated preeclampsia risk in PCOS.^{15,26-28} Overall, these findings indicate that BMI meaningfully contributes to the observed risk gradient, but that a BMI-independent component remains, consistent with residual PCOS-associated susceptibility to hypertensive disorders and prematurity-linked neonatal morbidity.

Finally, our findings highlight why composite endpoints may be particularly informative in PCOS pregnancy research. Obstetric studies have long struggled with variability and inconsistency in how maternal-perinatal composites are defined and reported, potentially obscuring net clinical burden.²⁹ CAPO was developed to address this problem by using severity-weighted components and summarizing mother-newborn outcomes into a single quantitative metric. The fact that both maternal and neonatal CAPO subscores were higher in our PCOS group suggests that PCOS-related morbidity is not confined to one domain but instead reflects a broader, dyad-level risk profile. Beyond the PCOS context, CAPO has already been used as a composite endpoint in other high-risk pregnancy settings (e.g., fetal growth restriction), reinforcing its applicability as a clinically meaningful summary outcome.³⁰ Taken together, these data support incorporating CAPO-based approaches in future PCOS studies-particularly those comparing phenotypes, fertility-treatment subgroups, or preventive strategies-because CAPO can capture meaningful cumulative morbidity even when single outcomes are individually infrequent.

Clinical Implications

Clinically, our findings suggest that PCOS identifies pregnancies at increased perinatal morbidity risk even after matching for key obstetric characteristics. Because the excess CAPO burden clustered around hypertensive disorders and prematurity-linked neonatal outcomes (low birth weight and prolonged NICU admission), PCOS pregnancies may warrant earlier risk stratification and closer surveillance for blood-

pressure complications and fetal growth/prematurity risk. Importantly, the persistence of higher CAPO scores after BMI adjustment indicates that counseling should not be limited to obesity alone, as PCOS-related risk may remain across BMI strata.

Strengths and Limitations

A major strength of this study is the novel application of a severity-weighted, mother-newborn dyad composite endpoint (CAPO) to pregnancies complicated by PCOS, allowing quantification of cumulative perinatal morbidity beyond isolated maternal or neonatal events. Additional strengths include the large two-center source population, rigorous 1:1 propensity score matching to improve baseline comparability, and triangulation of results using complementary analytic strategies, including matched-pair testing, BMI-adjusted conditional regression, BMI-stratified analyses, and a conservative BMI-inclusive rematching sensitivity analysis. Reporting total CAPO scores together with maternal and neonatal subscores and component-level patterns further enhances clinical interpretability by demonstrating that excess morbidity was distributed across hypertensive and prematurity-related domains rather than driven by a single rare event.

Limitations

Several limitations should be acknowledged. PCOS status was defined based on clinical documentation in medical records without individual re-adjudication of diagnostic criteria, which may introduce misclassification and does not permit differentiation across PCOS phenotypes. The absence of data on PCOS subtypes, metabolic characteristics (e.g., insulin resistance), medication exposure (including metformin), and detailed fertility treatment information limits the ability to identify which PCOS subgroups are at greatest risk. In addition, some individual CAPO components were infrequent, resulting in wide confidence intervals and reduced precision for certain component-level estimates, warranting cautious interpretation at that level despite a consistent composite signal. Inclusion of BMI in the propensity model for sensitivity analyses reduced the number of matched pairs, potentially decreasing statistical power; however, effect estimates remained directionally consistent. Finally, as with all retrospective observational studies, residual confounding cannot be excluded, and the generalizability of the findings may be limited to similar clinical settings and populations.

CONCLUSION

In this two-center propensity score-matched analysis, pregnancies complicated by PCOS exhibited a significantly higher severity-weighted perinatal morbidity burden as quantified by the CAPO score, with excess risk distributed across hypertensive disorders of pregnancy and prematurity-linked neonatal outcomes rather than driven by a single complication. Importantly, the association persisted after post-matching BMI adjustment and remained directionally consistent across BMI-stratified and BMI-inclusive sensitivity analyses, indicating that maternal adiposity explains part-but not all-of the observed morbidity signal. These findings

support CAPO as a clinically meaningful, dyad-level summary endpoint for PCOS pregnancy research and provide a robust framework for future studies evaluating phenotype-specific risk, fertility-treatment subgroups, and preventive strategies aimed at reducing cumulative maternal–neonatal morbidity.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University (Date: 12.03.2025, Decision No: 2025-GOKAEK-2521_2025.03.12_828).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: MG; Design: MG, HÖS, NA; Control: MG; Data Collection and/or Processing: MG, HÖS; Analysis and/or Interpretation: MG, NA; Literature Review: MG; Article Writing: MG; Critical Review: HÖS, NA.

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