

Research Article

Single vs Repeat Courses of Antenatal Corticosteroids in Women at Risk of Preterm Birth

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ABSTRACT

Background: Antenatal corticosteroids are an established intervention for women at risk of preterm birth. The role of repeat courses remains clinically important when substantial risk persists after an initial course.

Objective: To compare maternal and neonatal outcomes associated with single versus repeat courses of antenatal corticosteroids.

Materials and methods: This observational cohort study included 200 women at risk of preterm birth. Participants receiving a single course (n=100) were compared with those receiving repeat courses (n=100). Maternal, obstetric, treatment, and neonatal variables were analyzed. Crude associations were followed by multivariable logistic regression, penalized regression for sparse outcomes, propensity-score matching, and sensitivity analyses as reported in the study analysis.

Results: Repeat-course recipients had older maternal age, higher BMI, earlier gestational age at first course, longer interval to delivery, and substantially higher rates of preeclampsia, PPROM, and hypertension. Crude neonatal morbidity was higher in the repeat group. RDS occurred in 68% versus 8% (OR 24.3, 95% CI 8.5-69.4; $p < 0.001$), while NICU stay was 34 versus 9 days. After adjustment, the RDS association attenuated to OR 2.1 (95% CI 0.8-5.4; $p = 0.14$). Propensity matching likewise showed no statistically significant association.

Conclusion: Repeat corticosteroid courses were associated with substantially worse crude outcomes, but these associations markedly attenuated after adjustment for baseline risk, supporting substantial confounding by indication rather than an independently demonstrated adverse treatment effect.

Keywords: Antenatal Corticosteroids, Preterm Birth, Repeat Course, Betamethasone, Dexamethasone, Respiratory Distress Syndrome, Neonatal Outcomes, Propensity Score.

INTRODUCTION

Preterm birth is a major contributor to neonatal morbidity and mortality because immaturity of the lungs, brain, gastrointestinal tract, and immune system increases the risk of respiratory disease, neurological injury, infection, and prolonged neonatal hospitalization. The burden is particularly important in settings where premature infants require substantial neonatal intensive care resources. Epidemiological studies have established preterm birth as a major global public-health problem, with substantial variation in rates and causes across populations (1,2).

Antenatal corticosteroids are an established intervention for women at substantial risk of preterm birth. Randomized evidence and

systematic reviews show that an appropriately timed course accelerates fetal lung maturation and reduces neonatal respiratory morbidity and other serious complications (3). Clinical guidance from professional bodies and international recommendations supports antenatal corticosteroid administration when the likelihood of preterm birth within the relevant treatment window is sufficiently high (4,5). Betamethasone and dexamethasone are the principal corticosteroids used for fetal maturation, and evidence also supports their use in selected women at risk of late preterm birth (6).

The clinical question becomes more complex when delivery does not occur after the initial course and substantial risk persists. Repeat or

rescue courses have been studied because the biological effect of an initial course may diminish over time. Randomized trials have evaluated repeat-course strategies and have generally demonstrated short-term respiratory or neonatal benefits without establishing a simple justification for repeated exposure in every pregnancy (7,8). Longer-term follow-up has also examined childhood growth and neurodevelopment after repeat antenatal corticosteroid exposure, making the balance between immediate neonatal benefit and potential longer-term effects clinically important (9,10).

The interpretation of repeat-course treatment is further complicated in observational studies by confounding by indication. Women who receive repeat courses may differ systematically from women who receive a single course in gestational age, duration of pregnancy after the first course, PPRM, hypertensive disease, preeclampsia, and other factors that independently affect neonatal outcomes. Previous collaborative trials and follow-up studies provide evidence relevant to safety and growth, but observational comparisons require careful adjustment for the underlying risk of preterm birth (11,12). Respiratory morbidity itself is also strongly influenced by gestational maturity and contemporary neonatal respiratory-support practices (13,14).

The present study compares maternal and neonatal outcomes between women receiving a single course and those receiving repeat courses of antenatal corticosteroids. In addition to crude comparisons, the reported analysis incorporates multivariable adjustment, penalized regression for sparse outcomes, propensity-score matching, inverse-probability weighting, and subgroup analyses. These approaches are relevant because the epidemiology and causes of preterm birth are heterogeneous (15,16), and differences in gestational age and clinical indication can strongly influence observed neonatal outcomes. The study therefore focuses not only on crude outcome differences but also on whether those differences persist after accounting for measured baseline risk (17,18).

MATERIALS AND METHODS

This observational cohort study was done at Department of Obstetrics and Gynaecology, Ayub Teaching Hospital Abbottabad, from June 2025 to December 2025 which included 200 women at risk of preterm birth who

received antenatal corticosteroids. Participants were divided equally into a single-course group (n=100) and a repeat-course group (n=100). Maternal variables included age, BMI, gravidity, parity, maternal hypertension, preeclampsia, and PPRM. Treatment-related variables included gestational age at first corticosteroid course, corticosteroid type, number of antenatal steroid doses, and interval from the first course to delivery. Delivery mode and neonatal outcomes were also recorded.

Neonatal variables included birth weight, sex, 5-minute Apgar score, respiratory distress syndrome (RDS), bronchopulmonary dysplasia (BPD), necrotizing enterocolitis (NEC), intraventricular hemorrhage (IVH), neonatal sepsis, neonatal death, and NICU length of stay. Maternal infection was evaluated as a maternal outcome.

Continuous variables were summarized as mean $\pm$ standard deviation or median [interquartile range], as appropriate, and categorical variables as frequencies and percentages. Between-group comparisons used independent-samples t tests or Mann-Whitney U tests for continuous variables and χ^2 or Fisher's exact tests for categorical variables. Standardized mean differences (SMDs) were used to assess baseline balance, with an absolute SMD <0.1 considered indicative of adequate balance.

Crude odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for binary outcomes. Multivariable logistic regression adjusted for gestational age at first course, interval to delivery, preeclampsia, PPRM, maternal hypertension, BMI, and maternal age. Firth's penalized likelihood approach was used for outcomes with zero cells. NICU length of stay was examined using multivariable linear regression. Propensity scores were estimated using logistic regression with baseline covariates, followed by 1:1 nearest-neighbor matching using a caliper of 0.2 times the standard deviation of the logit of the propensity score. The reported matched cohort comprised 72 pairs. Sensitivity analyses included Firth regression, inverse probability of treatment weighting (IPTW), restriction by gestational age and delivery interval, and comparison of complete-case and multiple-imputation analyses.

The supplied study information does not provide the institutional study site, recruitment dates, ethical approval number, consent procedures, or detailed eligibility criteria; these

items are therefore not independently specified here. Because treatment allocation was observational and the groups were substantially imbalanced at baseline, the results are interpreted as associations rather than proof of a causal effect of repeat corticosteroid exposure.

RESULTS

A total of 200 women at risk of preterm birth were included, with 100 (50.0%) receiving a single course and 100 (50.0%) receiving repeat courses. Mean maternal age was 27.9 ± 4.2 years in the single-course group and 31.4 ± 4.8 years in the repeat-course group ($t=-5.48$, $p<0.001$). Mean BMI was also higher in the repeat group (28.6 ± 2.7 kg/m²) than in the single group (24.3 ± 2.1 kg/m²; $t=-12.61$, $p<0.001$).

Gestational age at first corticosteroid administration was significantly earlier in the repeat group (26.2 ± 0.8 weeks) than in the single group (29.6 ± 1.2 weeks; $t=22.87$, $p<0.001$). The median interval to delivery was longer after the first course in the repeat group (18 days [IQR 15–22]) than in the single group (5 days [IQR 3–7]; $U=128$, $p<0.001$). Preeclampsia, PPRM, and maternal hypertension were substantially more frequent among repeat-course recipients. Mean birth weight was lower in the repeat group (1080 ± 130 g) than in the single group (1690 ± 95 g; $t=38.14$, $p<0.001$). All reported baseline SMDs exceeded 0.1, demonstrating substantial imbalance and supporting concern for confounding by indication.

Table 1. Baseline Characteristics of the Study Population

Variable	Single (n=100)	Repeat (n=100)	Test	p	SMD
Maternal age, years	27.9 ± 4.2	31.4 ± 4.8	$t=-5.48$	<0.001	0.77
BMI, kg/m ²	24.3 ± 2.1	28.6 ± 2.7	$t=-12.61$	<0.001	1.75
Gravidity, median [IQR]	2 [1–3]	3 [2–4]	$U=3210$	<0.001	0.62
Parity, median [IQR]	1 [0–2]	2 [1–3]	$U=3450$	<0.001	0.58
GA at first course, weeks	29.6 ± 1.2	26.2 ± 0.8	$t=22.87$	<0.001	3.31
Interval to delivery, days	5 [3–7]	18 [15–22]	$U=128$	<0.001	3.89
Preeclampsia, n (%)	2 (2.0)	72 (72.0)	$\chi^2=103.7$	<0.001	2.87
PPROM, n (%)	0 (0.0)	98 (98.0)	$\chi^2=192.1$	<0.001	6.24
Maternal hypertension, n (%)	6 (6.0)	56 (56.0)	$\chi^2=60.2$	<0.001	1.45
Betamethasone, n (%)	72 (72.0)	68 (68.0)	$\chi^2=0.38$	0.54	0.09
Antenatal steroid doses, median	2	4	$U=1520$	<0.001	2.45
Birth weight, g	1690 ± 95	1080 ± 130	$t=38.14$	<0.001	5.24
Male neonate, n (%)	52 (52.0)	48 (48.0)	$\chi^2=0.32$	0.57	0.08

Continuous variables are mean $\pm$ SD or median [IQR]; categorical variables are n (%). SMD=standardized mean difference; GA=gestational age; PPRM=preterm premature rupture of membranes. The repeat-course group had higher crude rates of the major neonatal outcomes. RDS occurred in 68% of repeat-course infants versus 8% of single-course infants (crude OR 24.3, 95% CI 8.5–69.4; $\chi^2=76.3$, $p<0.001$). BPD occurred in 12% versus 0% (Fisher's

exact $p<0.001$), IVH in 14% versus 0% ($p<0.001$), neonatal sepsis in 28% versus 0% ($p<0.001$), and neonatal death in 6% versus 0% ($p=0.03$). NEC occurred in 4% versus 0%; the reported comparison was not statistically significant ($p=0.12$). Median NICU stay was 34 days [IQR 28–52] in the repeat group versus 9 days [IQR 7–12] in the single group ($U=145$, $p<0.001$). Mean 5-minute Apgar was 5.6 ± 1.1 versus 8.4 ± 0.7 , respectively ($t=21.4$, $p<0.001$).

Table 2. Crude Neonatal Outcomes by Treatment Group

Outcome	Single (n=100)	Repeat (n=100)	Crude OR (95% CI)	p
RDS, n (%)	8 (8.0)	68 (68.0)	24.3 (8.5–69.4)	<0.001
BPD, n (%)	0 (0.0)	12 (12.0)	Not estimable	<0.001
NEC, n (%)	0 (0.0)	4 (4.0)	Not estimable	0.12

IVH, n (%)	0 (0.0)	14 (14.0)	Not estimable	<0.001
Neonatal sepsis, n (%)	0 (0.0)	28 (28.0)	Not estimable	<0.001
Neonatal death, n (%)	0 (0.0)	6 (6.0)	Not estimable	0.03
Apgar at 5 min, mean $\pm$ SD	8.4 $\pm$ 0.7	5.6 $\pm$ 1.1	—	<0.001
NICU LOS, days, median [IQR]	9 [7–12]	34 [28–52]	—	<0.001

OR=odds ratio; CI=confidence interval; RDS=respiratory distress syndrome; BPD=bronchopulmonary dysplasia; NEC=necrotizing enterocolitis; IVH=intraventricular hemorrhage; NICU LOS=neonatal intensive care unit length of stay.

Maternal infection occurred in 54% of the repeat group and in none of the single-course group (Fisher's exact $p < 0.001$). Cesarean delivery occurred in 52% of repeat-course recipients compared with 34% of single-course recipients (crude OR 2.10, 95% CI 1.18–3.74; $\chi^2 = 6.56$, $p = 0.01$).

Table 3. Crude Maternal Outcomes

Outcome	Single (n=100)	Repeat (n=100)	Crude OR (95% CI)	p
Maternal infection, n (%)	0 (0.0)	54 (54.0)	Not estimable	<0.001
Cesarean delivery, n (%)	34 (34.0)	52 (52.0)	2.10 (1.18–3.74)	0.01

Adjusted, Propensity-Matched, and Sensitivity Analyses

Because baseline imbalance was substantial, multivariable logistic regression adjusted for gestational age at first course, interval to delivery, preeclampsia, PPRM, maternal hypertension, BMI, and maternal age. Firth's penalized likelihood was used for outcomes with zero cells. After adjustment, the associations between repeat corticosteroid courses and adverse outcomes attenuated and were no longer statistically significant. RDS had an adjusted OR of 2.1 (95% CI 0.8–5.4; $p = 0.14$), BPD 1.4 (0.4–4.9; $p = 0.60$), IVH 1.2 (0.3–4.6; $p = 0.79$), neonatal sepsis 1.8 (0.6–5.2; $p = 0.28$), neonatal death 1.1 (0.2–5.8; $p = 0.91$), and maternal infection 1.5 (0.6–3.8; $p = 0.39$). For NICU length of stay, the adjusted mean difference was +2.3 days (95% CI –1.1 to +5.7; $p = 0.18$).

The reported propensity-score analysis used 1:1 nearest-neighbor matching and produced 72 matched pairs, with all SMDs below 0.1 after matching. In the matched cohort, repeat corticosteroids were not significantly

associated with RDS (OR 1.9, 95% CI 0.7–5.1; $p = 0.21$), BPD (1.3, 0.3–5.0; $p = 0.68$), IVH (1.1, 0.3–4.2; $p = 0.89$), neonatal sepsis (1.7, 0.5–5.6; $p = 0.38$), or NICU length of stay (+1.8 days, 95% CI –2.0 to +5.6; $p = 0.35$).

Sensitivity analyses were consistent with the primary adjusted analysis. Firth regression yielded the same RDS estimate (OR 2.1, 95% CI 0.8–5.4; $p = 0.14$), while IPTW produced an OR of 2.0 (95% CI 0.7–5.6; $p = 0.19$). The reported E-value for RDS was 3.4 (lower confidence-limit E-value 1.3). Restriction to gestational age <28 weeks yielded an adjusted OR of 1.6 (0.6–4.3; $p = 0.35$), while the ≥ 28 -week subgroup had an OR of 2.4 (0.7–8.2; $p = 0.17$); the interaction p value was 0.52. For interval to delivery, adjusted ORs were 1.8 (0.5–6.1; $p = 0.36$) for <7 days and 2.3 (0.7–7.4; $p = 0.16$) for ≥ 7 days, with interaction $p = 0.61$. Complete-case and multiple-imputation analyses were reported as materially unchanged.

Table 4. Multivariable, Propensity-Matched, and Sensitivity Analyses

Analysis/outcome	Effect estimate (95% CI)	p-value
Adjusted RDS OR	2.1 (0.8–5.4)	0.14
Adjusted BPD OR	1.4 (0.4–4.9)	0.60
Adjusted IVH OR	1.2 (0.3–4.6)	0.79
Adjusted neonatal sepsis OR	1.8 (0.6–5.2)	0.28
Adjusted neonatal death OR	1.1 (0.2–5.8)	0.91
Adjusted maternal infection OR	1.5 (0.6–3.8)	0.39
Adjusted NICU LOS mean difference, days	+2.3 (–1.1 to +5.7)	0.18

Propensity-matched RDS OR	1.9 (0.7–5.1)	0.21
Propensity-matched BPD OR	1.3 (0.3–5.0)	0.68
Propensity-matched IVH OR	1.1 (0.3–4.2)	0.89
Propensity-matched sepsis OR	1.7 (0.5–5.6)	0.38
Propensity-matched NICU LOS mean difference, days	+1.8 (–2.0 to +5.6)	0.35
IPTW RDS OR	2.0 (0.7–5.6)	0.19
GA <28 weeks RDS OR	1.6 (0.6–4.3)	0.35
GA ≥28 weeks RDS OR	2.4 (0.7–8.2)	0.17
Interval <7 days RDS OR	1.8 (0.5–6.1)	0.36
Interval ≥7 days RDS OR	2.3 (0.7–7.4)	0.16

DISCUSSION

This study found a pronounced crude difference in neonatal morbidity between women receiving single and repeat antenatal corticosteroid courses. Repeat-course recipients had markedly higher crude rates of RDS, BPD, IVH, neonatal sepsis, neonatal death, and longer NICU hospitalization. However, the crude estimates should not be interpreted as causal effects of repeat corticosteroid exposure because the treatment groups were substantially different at baseline. The reported adjusted and propensity-matched estimates were much closer to the null. This pattern is consistent with the established observation that neonatal outcomes are strongly determined by gestational maturity and the severity of the pregnancy complications leading to preterm delivery (19).

The baseline imbalance provides an important explanation for this attenuation. Women receiving repeat courses were older, had higher BMI, received their first course at substantially earlier gestational ages, had longer intervals to delivery, and had markedly higher frequencies of preeclampsia, PPROM, and maternal hypertension. Gestational age is particularly important because it is strongly related to respiratory morbidity, neurological injury, infection, mortality, and duration of neonatal intensive care. The relationship between prematurity and neonatal outcome is well established, and contemporary estimates of neonatal risk must therefore account for gestational age and the level of neonatal care available (20,21).

The findings are consistent with the broader evidence that repeat antenatal corticosteroid therapy should be considered selectively rather than interpreted as an exposure that can be evaluated independently of the underlying risk of preterm birth. Randomized evidence has generally evaluated specific repeat-course regimens in women who remain at risk after an initial course, rather than

indiscriminate repeated administration (22,23). The present observational data similarly suggest that women selected for repeat treatment had a substantially different clinical risk profile before neonatal outcomes occurred.

The respiratory findings should also be interpreted in the context of neonatal management. Respiratory distress syndrome remains a major complication of prematurity, and contemporary respiratory-support strategies influence both its clinical expression and subsequent morbidity. Current consensus guidance emphasizes gestational age, antenatal corticosteroid exposure, respiratory support, and individualized neonatal management when evaluating respiratory outcomes (24). Therefore, the crude difference in RDS between groups should not by itself be taken as evidence that repeat corticosteroids caused respiratory disease.

The propensity-score analysis strengthens the interpretation because adequate post-matching balance was reported, with all SMDs below 0.1. The matched estimates were close to the null and statistically non-significant, while IPTW, Firth regression, gestational-age subgroup analyses, interval-to-delivery subgroup analyses, and the reported E-value analysis were directionally consistent. These complementary analyses reduce, but do not eliminate, concern that the principal crude associations were driven by measured baseline differences. The analysis also illustrates why causal interpretation of treatment comparisons in observational obstetric cohorts requires explicit attention to selection and baseline risk. The maternal infection and neonatal death findings require particular caution. Both outcomes were more common in the repeat-course group in crude analysis, but the supplied data do not provide enough information about the timing, diagnostic criteria, organism profile, treatment, or clinical severity to determine whether these outcomes were caused by corticosteroid exposure.

Similarly, NICU length of stay is affected by gestational age, birth weight, respiratory support, infection, neurological complications, and local discharge practices. Contemporary neonatal-care recommendations emphasize that respiratory and intensive-care outcomes should be interpreted within the broader clinical context rather than as isolated treatment effects (25).

The principal limitation is residual confounding. Even extensive adjustment cannot fully account for clinical severity when important variables are unmeasured or measured imperfectly. The supplied dataset does not document the precise indication for each repeat course, exact steroid administration schedule, detailed neonatal respiratory-support exposure, antenatal infection status before treatment, or all clinical factors influencing the decision to repeat corticosteroids. In addition, the advanced regression, propensity-score, IPTW, and E-value results used in this manuscript were supplied as study-analysis results and were not independently rerun from the raw 200-row dataset during preparation of this revision. They should therefore be checked against the original statistical output before submission.

Despite these limitations, the study has several strengths, including individual-level maternal and neonatal data, assessment of multiple clinically relevant outcomes, explicit quantification of baseline imbalance using SMDs, and use of complementary adjusted and propensity-based approaches. The overall pattern is clinically coherent: a very large crude difference was substantially attenuated after adjustment for measured obstetric risk. The results therefore support cautious interpretation of crude associations and reinforce the importance of distinguishing treatment exposure from the underlying clinical circumstances that prompted repeat treatment.

CONCLUSION

In this observational cohort, repeat antenatal corticosteroid courses were associated with substantially worse maternal and neonatal outcomes in crude analyses. However, repeat-course recipients also had substantially greater baseline obstetric risk and earlier gestational age at first corticosteroid administration. After multivariable adjustment and propensity-score matching, the associations were markedly attenuated and no longer statistically significant. The findings therefore indicate

substantial confounding by indication and do not demonstrate an independent adverse effect of repeat corticosteroid courses on the evaluated outcomes.

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