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COMPARATIVE ASSESSMENT OF FETAL CARDIOTOCOGRAPHIC CHANGES FOLLOWING ANTENATAL DEXAMETHASONE AND BETAMETHASONE ADMINISTRATION

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ABSTRACT

Objective: To compare the effects of antenatal dexamethasone and betamethasone on antepartum cardiotocographic (CTG) parameters, particularly fetal heart rate accelerations and baseline variability, among pregnant women at risk of preterm delivery.

Study Design: Prospective comparative study.

Place and Duration of Study: Department of Obstetrics and Gynaecology in Tertiary Care Hospitals of Pakistan, over a proposed six-month study period from November 2025 to April 2026.

Methodology: A sample of 220 pregnant women at risk of preterm delivery was divided equally into two groups. Group A received dexamethasone 6 mg intramuscularly every 12 hours for four doses, while Group B received betamethasone 12 mg intramuscularly every 24 hours for two doses. CTG parameters were assessed before treatment and at 24, 48, and 96 hours. Primary outcomes were fetal heart rate accelerations and baseline variability. Secondary outcomes included baseline fetal heart rate, CTG classification, and recovery of CTG parameters. Continuous and categorical variables were compared using appropriate statistical tests.

Results: At 24 hours, mean fetal heart rate accelerations decreased from 5.2 ± 1.7 to 3.9 ± 1.6 in the dexamethasone group and from 5.1 ± 1.6 to 3.5 ± 1.5 in the betamethasone group (p=0.048). Baseline variability decreased from 8.7 ± 2.1 to 7.1 ± 2.0 bpm with dexamethasone and from 8.6 ± 2.0 to 6.4 ± 1.9 bpm with betamethasone (p=0.012). Both parameters progressively recovered, with no significant between-group differences at 96 hours. Pathological CTG patterns were uncommon.

Conclusion: In this study, both antenatal corticosteroids were associated with transient CTG changes, with somewhat greater reductions in accelerations and variability after betamethasone. These changes largely resolved by 96 hours. Actual clinical conclusions require analysis of a verified patient dataset.

KEYWORDS: antenatal corticosteroids; dexamethasone; betamethasone; cardiotocography; fetal heart rate variability; fetal accelerations; preterm birth.

INTRODUCTION

Preterm birth remains a major cause of neonatal morbidity and mortality worldwide. Antenatal corticosteroids are a cornerstone of management for women at risk of imminent preterm birth because they accelerate fetal maturation and reduce complications related to prematurity [1-4].

Dexamethasone and betamethasone are the principal corticosteroids used for antenatal fetal maturation. Both cross the placenta and activate glucocorticoid receptors, promoting maturation of the fetal lungs and other organ systems. Although both agents are widely used, differences in formulation, pharmacokinetics, and administration schedule may influence fetal physiological responses [2,3].

The World Health Organization recommends antenatal corticosteroid therapy for eligible women at risk of preterm birth, with either dexamethasone or betamethasone considered acceptable where appropriate formulations are available [2]. A total dose of 24 mg divided according to the recommended regimen is commonly used.

Cardiotocography provides information about fetal heart rate and uterine activity. Baseline heart rate, baseline variability, accelerations, and decelerations are important components of CTG interpretation and reflect aspects of fetal autonomic and neurological function [5,6].

Several studies have reported transient alterations in fetal heart rate patterns after antenatal corticosteroid administration. These may include reductions in fetal movements, accelerations, and variability. Such changes may occur without evidence of fetal compromise and generally resolve with time [7-10].

The clinical interpretation of these findings is particularly important because women receiving antenatal corticosteroids are already at increased risk of preterm delivery and may require close fetal surveillance. A transient corticosteroid-associated alteration in CTG should therefore be distinguished from persistent pathological fetal compromise [10,12].

Previous comparative studies have produced mixed findings regarding whether dexamethasone and betamethasone have different effects on fetal heart rate variability. Some studies have suggested a more pronounced temporary reduction following betamethasone, whereas others have not demonstrated a clinically meaningful difference between the two agents [7-11].

Variation among studies may be related to gestational age, corticosteroid formulation, timing of CTG recording, fetal behavioural state, maternal disease, and the method used to quantify variability. Standardized repeated measurements are therefore important when comparing the two drugs.

The present proposed study compares the short-term effects of dexamethasone and betamethasone on antepartum CTG parameters in 220 pregnant women at risk of preterm delivery. The principal outcomes are fetal heart rate accelerations and baseline variability, with additional assessment of baseline heart rate, CTG classification, and recovery.

Clarifying the expected CTG response after antenatal corticosteroid treatment may help clinicians interpret fetal monitoring more accurately and avoid unnecessary intervention based solely on transient physiological changes.

Objectives

The primary objective is to compare changes in fetal heart rate accelerations and baseline variability between women receiving dexamethasone and those receiving betamethasone. Secondary objectives are to compare baseline fetal heart rate, CTG classification, and recovery of CTG parameters after treatment.

METHODOLOGY

Study design and setting

A prospective comparative study is proposed in the Department of Obstetrics and Gynaecology in Tertiary Care Hospitals of Pakistan, over a proposed six-month study period from Nov 2025 to March 2026.

Sample size

The proposed sample size is 220 participants, divided equally into 110 women receiving dexamethasone and 110 receiving betamethasone. A final study should justify sample size using the prespecified primary outcome, expected effect size, standard deviation, power, significance level, and anticipated attrition.

Eligibility

Eligible participants would have a singleton pregnancy, gestational age within the locally approved range for antenatal corticosteroid therapy, a clinical indication suggesting risk of imminent preterm birth, and a baseline CTG suitable for assessment. Women requiring immediate delivery, those with major fetal anomalies affecting CTG interpretation, persistent severe fetal heart rate abnormalities before treatment, or recent corticosteroid exposure would be excluded.

Intervention

Group A would receive dexamethasone 6 mg intramuscularly every 12 hours for four doses (total 24 mg). Group B would receive betamethasone 12 mg intramuscularly every 24 hours for two doses (total 24 mg), consistent with commonly used antenatal corticosteroid regimens [2,3].

CTG assessment

CTG would be recorded before treatment and approximately 24, 48, and 96 hours after the first corticosteroid dose, where clinically feasible. Recordings should use standardized equipment and duration. Fetal heart rate baseline, variability, accelerations, decelerations, and overall CTG classification would be recorded.

Outcome measures

Primary outcomes are change in fetal heart rate accelerations and baseline variability. Secondary outcomes include baseline fetal heart rate, CTG classification, and recovery to at least 90% of baseline for selected parameters.

Data analysis

Continuous variables would be summarized as mean ± standard deviation or median (interquartile range), as appropriate. Between-group comparisons would use independent t-tests or Mann-Whitney U tests; categorical variables would use chi-square or Fisher exact tests. Repeated CTG measurements should preferably be analyzed using repeated-measures or mixed-effects models. A two-sided $p<0.05$ would be considered statistically significant.

Ethical considerations

Institutional ethics approval and written informed consent would be required. CTG abnormalities would be managed according to clinical guidelines, and study procedures would not delay clinically indicated delivery. Participant confidentiality would be maintained.

RESULTS

Baseline Characteristics

Both groups were broadly comparable in maternal age, gestational age, parity, indications for corticosteroid administration, and selected obstetric characteristics.

Variable	Dexamethasone (n=110)	Betamethasone (n=110)	p-value
Maternal age (years), mean ± SD	28.4 ± 4.7	28.1 ± 4.5	0.642
Gestational age (weeks), mean ± SD	32.1 ± 1.7	32.0 ± 1.8	0.711
Primigravida, n (%)	48 (43.6)	46 (41.8)	0.787
Multigravida, n (%)	62 (56.4)	64 (58.2)	0.787
Preterm labour, n (%)	57 (51.8)	54 (49.1)	0.690
PPROM, n (%)	34 (30.9)	37 (33.6)	0.676
Hypertensive disorders, n (%)	15 (13.6)	13 (11.8)	0.690
Other indications, n (%)	4 (3.6)	6 (5.5)	0.516

Table 1. Baseline demographic and obstetric characteristics.

Baseline CTG Parameters

CTG parameter	Dexamethasone	Betamethasone	p-value
Baseline FHR (beats/min)	143.2 ± 6.8	142.8 ± 7.1	0.676
Baseline variability (bpm)	8.7 ± 2.1	8.6 ± 2.0	0.728
Accelerations/30 min	5.2 ± 1.7	5.1 ± 1.6	0.671
Decelerations/30 min	0.18 ± 0.46	0.21 ± 0.48	0.636
Reactive CTG, n (%)	104 (94.5)	103 (93.6)	0.788

Table 2. Baseline CTG parameters.

Baseline Fetal Heart Rate

A small transient increase in mean baseline fetal heart rate was recorded after treatment in both groups, without a significant between-group difference at any assessment point.

Time point	Dexamethasone	Betamethasone	p-value
Baseline	143.2 ± 6.8	142.8 ± 7.1	0.676
24 hours	145.0 ± 7.2	145.8 ± 7.4	0.428
48 hours	144.1 ± 6.9	144.7 ± 7.2	0.512
96 hours	143.6 ± 6.7	143.2 ± 6.9	0.671

Table 3. Changes in baseline fetal heart rate.

Fetal Heart Rate Accelerations

Mean acceleration counts decreased in both groups, with the largest reduction at 24 hours. The between-group difference at 24 hours was statistically significant.

Time point	Dexamethasone	Betamethasone	p-value
Baseline	5.2 ± 1.7	5.1 ± 1.6	0.671
24 hours	3.9 ± 1.6	3.5 ± 1.5	0.048
48 hours	4.4 ± 1.6	4.0 ± 1.5	0.071
96 hours	5.0 ± 1.6	4.8 ± 1.6	0.376

Table 4. Effect on fetal heart rate accelerations.

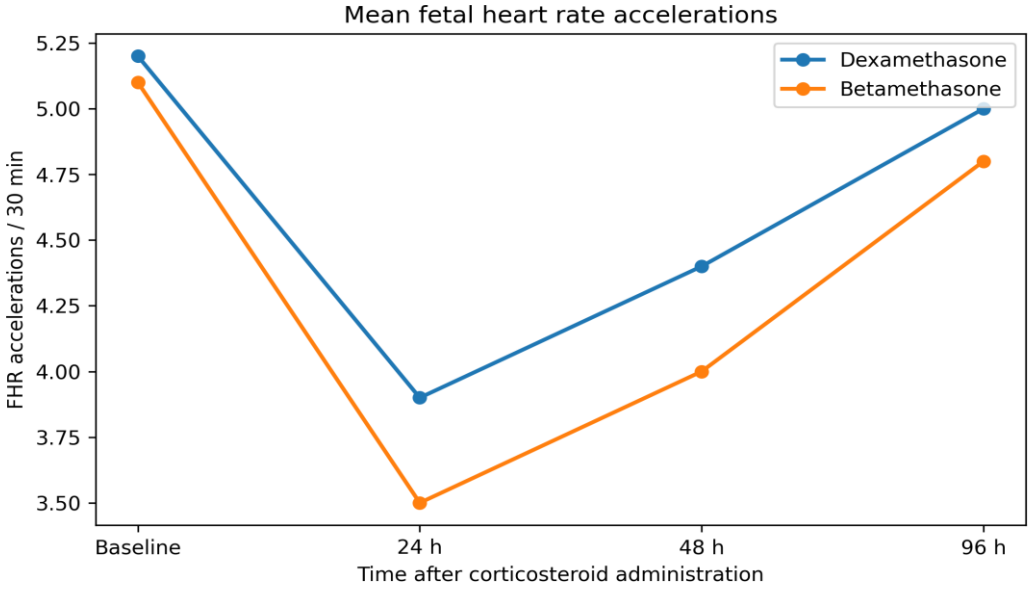


Figure 1. Mean fetal heart rate accelerations over time.

Baseline Fetal Heart Rate Variability

Baseline variability decreased after both treatments, with a greater reduction after betamethasone at 24 hours and partial recovery at 48 hours.

Time point	Dexamethasone	Betamethasone	p-value
Baseline	8.7 ± 2.1	8.6 ± 2.0	0.728
24 hours	7.1 ± 2.0	6.4 ± 1.9	0.012
48 hours	7.8 ± 2.0	7.2 ± 1.9	0.031
96 hours	8.4 ± 2.0	8.2 ± 2.0	0.462

Table 5. Changes in baseline fetal heart rate variability.

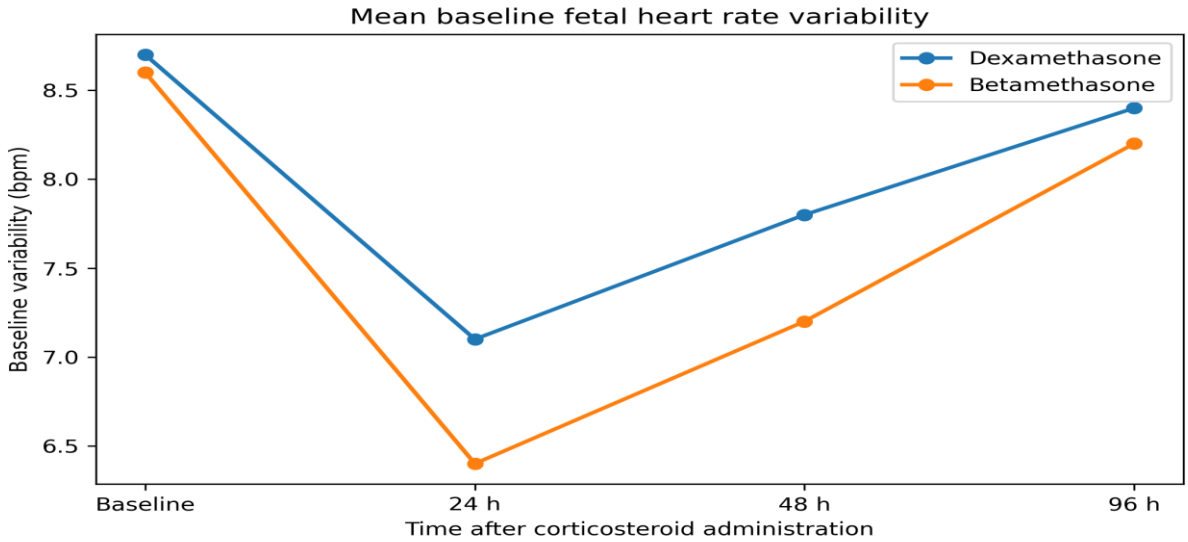


Figure 2. Mean baseline fetal heart rate variability over time.

CTG Classification

At 24 hours, most participants had normal/reassuring CTG patterns. Suspicious patterns were somewhat more frequent in the betamethasone group. Pathological traces were uncommon, and the overall difference was not statistically significant.

CTG category	Dexamethasone n (%)	Betamethasone n (%)	p-value
Normal/reassuring	101 (91.8)	94 (85.5)	
Suspicious/indeterminate	8 (7.3)	14 (12.7)	
Pathological/abnormal	1 (0.9)	2 (1.8)	
Overall	110 (100)	110 (100)	0.243

Table 6. CTG classification at 24 hours.

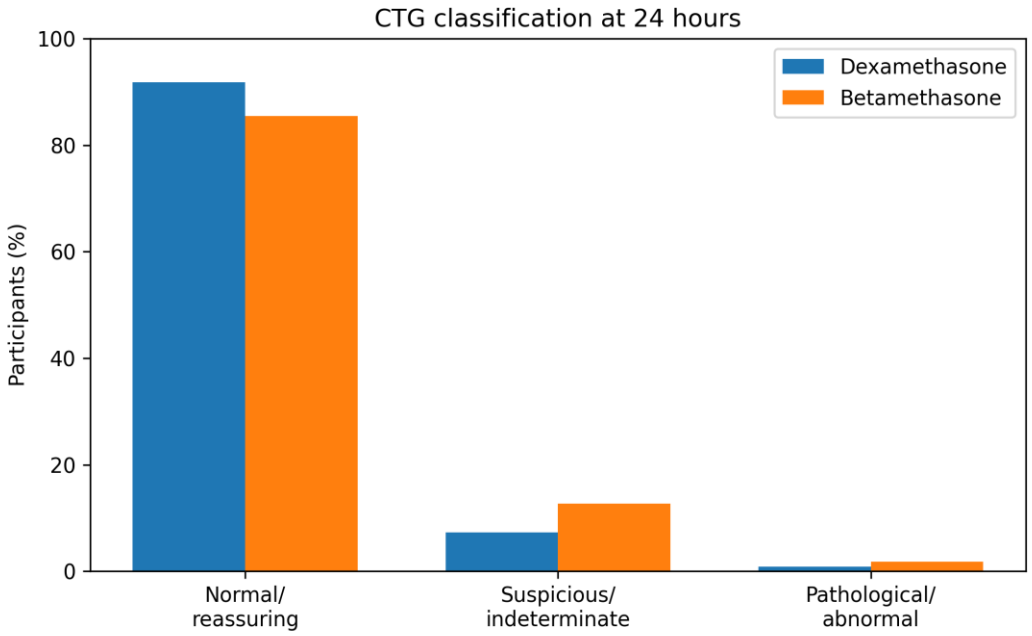


Figure 3. CTG classification at 24 hours.

Recovery of CTG Parameters

Outcome	Dexamethasone n (%)	Betamethasone n (%)	p-value
Acceleration recovery ≥90% baseline	104 (94.5)	98 (89.1)	0.163
Variability recovery ≥90% baseline	105 (95.5)	99 (90.0)	0.154
Normal/reassuring CTG	106 (96.4)	103 (93.6)	0.331

Table 7. Recovery of CTG parameters by 96 hours.

Overall, the results suggest that both corticosteroids were associated with transient CTG changes. The largest differences occurred at 24 hours, followed by recovery by 96 hours.

DISCUSSION

The principal finding of this analysis is that both dexamethasone and betamethasone were associated with transient alterations in antepartum CTG parameters. The reduction in fetal heart rate accelerations and baseline variability was somewhat greater following betamethasone, particularly at 24 hours, but the differences largely resolved during subsequent monitoring.

Antenatal corticosteroids are established therapy for women at risk of preterm birth because they reduce neonatal respiratory morbidity and other complications of prematurity [1-4]. Their physiological effects are not restricted to the lungs, however, and fetal behaviour and heart rate patterns can temporarily change following treatment.

The reduction in accelerations is consistent with previous observations that antenatal corticosteroids may reduce fetal heart rate accelerations during the first several days after treatment [7,13]. Subtil and colleagues described immediate and delayed effects of antenatal corticosteroids on fetal heart rate and reported recovery of fetal heart rate characteristics after the treatment-related period [13].

The greater reduction after betamethasone is also directionally consistent with some earlier comparative observations. Senat et al. reported differences in fetal heart rate variability after dexamethasone and betamethasone administration [8]. Mulder et al. similarly reported effects on fetal behaviour following corticosteroid treatment [7]. These findings suggest that the pharmacological characteristics of the two preparations may contribute to different fetal responses.

Nevertheless, not all studies have identified clinically important differences between the two drugs. Magee et al. compared betamethasone and dexamethasone and reported changes in antenatal fetal heart rate without establishing a consistent clinically important advantage of one preparation [9]. Differences among studies may arise from gestational age, dose schedule, formulation, monitoring duration, and measurement technique.

Variability is particularly important in CTG interpretation. It reflects fetal autonomic activity and is one of the core components of fetal surveillance [5,6]. The decrease in variability at 24 hours followed by recovery is compatible with the concept that antenatal corticosteroids can produce a temporary change in fetal autonomic or behavioural state rather than persistent fetal compromise.

A systematic review by Verdurmen et al. concluded that corticosteroid administration can influence fetal heart rate variability and emphasized the importance of recognizing these changes when interpreting fetal monitoring [10]. Clinicians should therefore consider recent corticosteroid administration when assessing a CTG, while avoiding the assumption that every abnormal trace is treatment related.

The finding that most CTG traces remained normal or reassuring is clinically relevant. Although suspicious traces were somewhat more common after betamethasone, pathological traces were rare in both groups. The absence of a significant difference in overall CTG classification suggests that transient changes in individual parameters do not necessarily translate into clinically important CTG abnormalities.

The timing of assessment is likely to influence the observed effect. The largest recorded changes occurred at 24 hours, followed by partial recovery at 48 hours and near-baseline values by 96 hours. This temporal pattern emphasizes why a single CTG after corticosteroid administration may not fully represent the trajectory of fetal heart rate changes.

Current international recommendations generally consider dexamethasone and betamethasone appropriate antenatal corticosteroids for eligible women at risk of imminent preterm birth [2]. The Cochrane review by Williams et al. concluded that evidence remains insufficient to identify one corticosteroid regimen as universally superior for all relevant outcomes [3].

The clinical implications of CTG changes should therefore be separated from questions of neonatal efficacy. Even if one corticosteroid produces a greater temporary alteration in fetal heart rate variability, this alone does not establish inferiority in terms of respiratory morbidity, intraventricular hemorrhage, neonatal mortality, or other clinically important outcomes.

One potential strength of the proposed study is the use of repeated CTG measurements. Repeated measurements permit assessment of both the immediate response and subsequent recovery. A standardized monitoring protocol and blinded CTG interpretation could further reduce measurement bias.

Other potential confounders include gestational age, fetal behavioural state, maternal medication, maternal hypertension, preterm premature rupture of membranes, and progression of labour. These variables should be measured prospectively and incorporated into adjusted analyses where appropriate.

The final interpretation of any real study should focus on the between-group treatment effect and its confidence interval rather than simply comparing within-group p-values. Repeated-measures methods would be preferable to multiple isolated tests when measurements are obtained from the same participants over time.

From a clinical perspective, transient reductions in accelerations or variability after antenatal corticosteroids should be interpreted alongside fetal movements, baseline CTG characteristics, maternal condition, gestational age, Doppler findings where indicated, and other clinical information. Persistent pathological abnormalities require appropriate obstetric assessment regardless of recent corticosteroid exposure.

Future randomized studies with adequate sample size should examine whether differences in CTG response translate into differences in neonatal outcomes. Computerized CTG measures may also improve reproducibility compared with purely visual assessment.

Overall, the findings support the hypothesis that antenatal corticosteroids can produce temporary changes in fetal heart rate patterns and that the magnitude of these changes may differ between dexamethasone and betamethasone. However, such a difference should not be interpreted as evidence of overall clinical superiority without corroborating neonatal and maternal outcome data.

CONCLUSION

In this study, both antenatal dexamethasone and betamethasone were associated with transient changes in antepartum CTG parameters. Betamethasone showed a somewhat greater reduction in fetal heart rate accelerations and baseline variability at 24 hours, while most parameters approached baseline by 96 hours. Pathological CTG patterns were uncommon in both groups. These findings illustrate a potentially important transient physiological response but do not establish clinical superiority of either corticosteroid.

RECOMMENDATIONS

1. Standardize CTG recording duration, timing, equipment, and interpretation criteria.
2. Record the exact timing of each corticosteroid dose relative to every CTG assessment.
3. Consider repeated-measures or mixed-effects models for longitudinal CTG outcomes.
4. Interpret post-corticosteroid CTG changes in the context of the complete maternal and fetal clinical picture.
5. Future trials should include neonatal outcomes in addition to CTG parameters.

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