Original Article

Comparison of Effect of Antenatal Betamethasone Versus Dexamethasone on Antepartum Cardiotocography

Ifra Shafique[1], Amara Arooj[2], Humaira Bilqis[3], Lubna Ejaz Kahloon[4], Tallat Farkhanda[5]

[1]Gynecology and Obstetrics trainee, [2]Assistant Professor, Gynecology and Obstetrics
[3]Associate Professor, Gynecology and Obstetrics, [4]Ex Dean and Head of Department Gynecology and Obstetrics,
[5]Dean and Head of department,
(Gynecology and Obstetrics, Holy Family Hospital, Rawalpindi)

Correspondence: *Dr Ifra Shafique*
Gynecology and Obstetrics trainee
Holy Family Hospital, Rawalpindi
ifra.shafique1944@gmail.com

Abstract

Objective: To compare the outcome of antenatal dexamethasone versus betamethasone on antepartum cardiotocography in terms of mean number of accelerations and long-term variability at day 0, 2 and 4 following drug administration

Methodology: This single blind Randomized Controlled Trial was carried out at Department of Obstetrics & Gynecology, Holy Family Hospital, Rawalpindi from 13th July 2025 to 15th October 2025. The trial was registered with clinicaltrials.gov (NCT07078786). The study enrolled 110 patients with 55 in each group. In Group A, patients received a 24 mg intramuscular injection of dexamethasone, administered as two doses of 12 mg 24 hours apart. In diabetic patients, the total dose was administered in four equal divided doses 12 hours apart. In Group B, patients received a 24 mg intramuscular injection of betamethasone, administered as two doses of 12 mg 24 hours apart.

Results: Baseline characteristics were comparable between groups (all $p>0.05$). Acceleration counts were similar at baseline ($p=0.881$) but were significantly higher in the dexamethasone group at day 4 ($p<0.001$), with significant within-group decline from baseline in both groups ($p<0.001$). Fetal heart rate variability was significantly better with dexamethasone at day 2 ($p=0.006$) and day 4 ($p=0.013$).

Conclusion: Administration of both Dexamethasone and Betamethasone to mother causes transient decrease in both fetal heart rate accelerations and variability with greater effect seen in case of Betamethasone.

Keywords: Pre term neonate, Antenatal corticosteroids.

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Introduction

The incidence of pre-term birth is 10% and about 40-45% of these cases occur secondary to pre-term labor with intact membranes.[1] This leads to delivery of fetus at an age where it may not cope with the stress of external environment. The pre-term neonate may suffer from a common yet life threating condition that is known as respiratory distress syndrome. The incidence varies inversely with gestational age. The incidence is 98% among neonates born at 24 weeks while it is 5% at 34 weeks and 1% at 37 weeks.[2] This occurs due to lack of surfactant within the lungs. The lung maturity and therefore surfactant production is accelerated by administration of steroids to the mother who are at risk of pre-term labor and birth. Antenatal corticosteroids not only reduces the incidence of respiratory distress syndrome but they also reduces neonatal mortality.[3] Two of the commonly administered corticosteroids to accelerate fetal lung development include Dexamethasone and Betamethasone. Furthermore, the steroids administered may affect organ systems other than the lungs. One of the systems that antenatal corticosteroids can affect is fetal cardiovascular system.[4] The cardiovascular effect can affect the indices of fetal well being seen on cardiotocography.[4] If these indices are negatively affected this could in turn increase the iatrogenic risk of pre-term birth particularly that with C-section.

Recent study suggested that antenatal corticosteroids result in suppression of fetal sympathetic activity leading to significant heart rate reduction, increased complexity

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of heart rate patterns while short term variability remained unchanged.[4] Antenatal Betamethasone administration resulted in significant changes on CTG.[5] In more than half of these patients the change was decrease in baseline fetal heart rate and fetal heart rate variability.[5] However long term follow up of patients exposed to Betamethasone in the intrauterine life revealed no increase in cardiovascular risk factors.[6,7] Therefore, the effects may be short lived. Similarly antenatal administration of Dexamethasone was associated with transient decrease in parameters of fetal heart rate including baseline heart rate variability and accelerations more pronounced on second day after administration which can mimic fetal compromise triggering iatrogenic delivery.[8] However in another study, fetal heart activity remained unchanged up to 96 hours following maternal administration of Dexamethasone but there was significant decrease in fetal movements at 24 hours following administration.[9]

Although dexamethasone and betamethasone are commonly used for similar clinical indications, existing literature provides limited direct comparative evidence, especially within the national context. Most available studies are either international, heterogeneous in design, or underpowered to detect clinically meaningful differences. Given potential regional differences in patient profiles, comorbidities, and practice patterns, a locally conducted comparative study is warranted to inform optimal drug selection and improve patient outcomes. In a direct comparative study, there was significant decrease in the number of accelerations (15.9 ± 7.4 vs 8.1 ± 5.3, P=0.01) and long term variation (msec) (46.2±6.7 vs 41.5±10.6) on day 2 after administration of dexamethasone, as compared to day 0(10). Similar there was more profound decrease in the number of accelerations (20.1 ± 7.4 vs 5.3 ± 4.9, P=0.001) and long term variation (55.6±8.5 vs 34.3±6.7) on day 2 after administration of betamethasone, as compared to day 0.[10] All the fetal heart parameters returned to baseline value on day 4 in Dexamethasone group but betamethasone exposed fetuses had a significantly lower return to baseline values as compared to dexamethasone group.[10] However contradictory findings were found in another study revealing no significant difference between CTG parameters while comparing Dexamethasone and Betamethasone.[11] Despite widespread clinical use of dexamethasone and betamethasone, the evidence base remains limited and heterogeneous, precluding firm conclusions regarding their comparative effectiveness. This evidence gap contributes to variability in clinical practice and uncertainty in guideline formulation. A rigorously designed head-to-head study is therefore warranted to clarify their relative benefits and inform standardized practice.

Methodology

This single blind Randomized Controlled Trial was carried out at Department of Obstetrics & Gynecology, Holy Family Hospital, Rawalpindi from 13th July 2025 to 15th October 2025. The study was started after taking ethical approval from Institutional ethical review board (1080/IREF/RMU/2024). The trial was registered with clinicaltrials.gov (NCT07078786). The study enrolled 110 patients with 55 in each group. The sample size was calculated according to WHO calculator with 5% level of significance, 80% power of study and taking mean number of accelerations after 4 days in antenatal dexamethasone group as 6.9 ± 5.3 and in betamethasone group as 5.3 ± 4.4(10). Patients fulfilling the inclusion criteria that was antenatal patients between gestational age of 30 to 37 weeks with singleton pregnancy requiring steroid prophylaxis who are at risk of threatened preterm deliveries such as previous history of preterm labor, Preterm prolabor rupture of membranes, mild third trimester bleeding due to placenta previa were included in the study. Patients were randomly divided into two groups that was group A and B using computer generated numbers. While patients with Uteroplacental insufficiency/IUGR/Oligohydramnios, drug therapy interfering with fetal heart rate such as labetalol, hydralazine, magnesium sulphate and nifedipine), contraindication to intramuscular injections and non-reassuring CTG were excluded from the study. Age, gestational age, parity (primiparous/multiparous), height, weight and BMI were noted for all patients. In the Group A, patients were given 24mg dexamethasone intramuscular injection in two doses of 12mg, 24 hours apart and in equal four divided doses 12 hours apart in case of diabetics. While in Group B patients, 24mg Intramuscular injection of betamethasone in two doses of 12 mg, will be administered 24 hours apart.

Cardiotocography examination was done before (day 0), day 2 and 4 after the first dose of steroid administration. CTG was performed at described interval from the first dose (day 0, day 2 and day 4) except if the patient complained a significant decrease in fetal movements, then CTG was performed earlier. In both groups number of accelerations and variability before, after 2 and 4 days

of first dose of antenatal steroid was measured and compared. Variability was classified as absent—amplitude range undetectable, minimal—amplitude range detectable but ≤ 5 bpm, moderate—amplitude range 6– 25 bpm and marked—amplitude range > 25 bpm. In case CTG was indicative of fetal compromise other parameters of fetal well being were assessed according to departmental protocol and decision was made accordingly.

Results

Baseline demographic profile was similar across two groups with no statistically significant difference as per p value obtained from independent t test. This is shown in table I.

For comparison of mean number of accelerations across two groups, data was checked for normality using Shapiro Wilk test which showed data distribution to be non-parametric. At all point of times Shapiro wilk test p value was 0.000 indicating that data was significantly deviated from normal distribution.

At day 2 no acceleration was observed across two groups. Comparison of mean number of accelerations across two groups at different time frames is shown in table II.

Number of accelerations following administration of drugs at day 4 were significantly higher in Dexamethasone group as compared to Betamethasone group. (Table III)

While making comparison of number of accelerations at day 4 from day 0 within the groups, Wilcoxon test was used. The p value for both groups was found to be 0.000 indicating significant difference in number of accelerations at day 4 from baseline. (Table IV)

Discussion

This provided important information on effects of two commonly used steroids Betamethasone and Dexamethasone on fetal cardiotocography tracing. The mean maternal age of presentation was comparable across two groups. The mean age among patients receiving Dexamethasone was 32.94 ± 3.65 while it was 32.42 ± 3.76 years among patients who received Betamethasone. Younger age group that was mean age of 26.10±4.53 in years was studied by Ishrat et al([12]). Comparable age groups were studied by a number of different authors.[13,11] The studied included patients with gestational age between 30-37 weeks with mean gestational age in group A being 33.36 ± 1.79 weeks while it was 33.25 ± 2.00 weeks in group B. This is comparable with a number of studies reporting almost similar gestational age.[(14),(15),(16)]. The main outcome variable was number of accelerations on day 2 and 4 across two groups. The comparison showed significant difference between both groups with number of accelerations being significantly higher in group A than B on day 4.

Table I: Baseline demographic profile across two groups.

Parameter	Group A (Dexamethasone) Mean ± SD	Group B (Betamethasone) Mean ± SD	P value
Age	32.94 ± 3.65	32.42 ± 3.76	0.457
Height	160.71 ± 3.78	162.03 ± 5.48	0.142
Weight	77.42 ± 7.87	77.25 ± 6.93	0.908
Gestational age	33.36 ± 1.79	33.25 ± 2.00	0.764

Table II: Comparison of Number of accelerations across two groups.

Parameter	Group A (Dexamethasone) Median (IQR)	Group B (Betamethasone) Median (IQR)	P value
Accelerations at day 0	2 (1)	2 (1)	0.881
Accelerations at day 4	1 (1)	0 (0)	0.000

Table III: Comparison of fetal heart rate variability at Day 2.

Variability	Group A (Dexamethasone) n (%)	Group B (Betamethasone) n (%)	P value
Minimal	36	49	0.006
Moderate	19	6	
Total	55	55	

Table IV: Comparison of fetal heart rate variability at Day 4.

Variability	Group A (Dexamethasone) n (%)	Group B (Betamethasone) n (%)	P value
Minimal	0	7	0.013
Moderate	55	48	
Total	55	55	

There was a significant reduction in the number of accelerations on day 4 compared with day 0 within both groups. However, Dexamethasone group had higher number of accelerations on day 4 when compared to Betamethasone group with p value being 0.000. Limited literature is available in which a direct comparison has been made between Betamethasone and Dexamethasone fetal CTG tracing. In a study done, it was found that maternal exposure to both Betamethasone and Dexamethasone reduces number of accelerations seen on CTG with greater reduction in Betamethasone group particularly at 48 hours.[10] This study shows that 48 hours both groups lack acceleration when compared to baseline. Similar to our findings, a study observing the effect of Dexamethasone on fetal heart accelerations on CTG showed that there was significant reduction in fetal heart rate accelerations from 3.84 to 3.17 at 48 hours following drug administration.[12] This study also showed that at day 4 Dexamethasone group had significantly higher number of accelerations than Betamethasone group but the number of accelerations were still lower than that seen at baseline. Similar findings were seen by Rotmensch S et al with mean number of acceleration at day 4 in Betamethasone group were 15.8 ± 7.6 that were significantly lower than baseline (p value <0.01) and mean number of accelerations in Dexamethasone group were 20.2 ± 14.9.[10] In other studies it was found that Betamethasone is associated with transient worsening of CTG parameters such as decrease baseline fetal heart rate variability and presences of decelerations which are not related to hypoxemia.[14,15] On the other hand ,Dexamethasone administration was associated with increased fetal heart rate variability with no other adverse effect on CTG.[16]

In this study, fetal heart rate variability was better in Dexamethasone group as compared to Betamethasone group both at day 2 and day 4 following drug administration. At day 2 following drug in dexamethasone group 36 had minimal while 19 had moderate variability while in Betamethasone group 49 had minimal and 6 had moderate variability. The difference was statistically significant with p value of 0.006. At day 4 following drug administration all fetus in Dexamethasone had moderate fetal heart rate variability while in Betamethasone group, 7 had minimal and 48 had moderate fetal heart rate variability. The difference was significant with p value was 0.013. The findings are similar to number of studies in the already available literature. Previously a study had revealed that there was no significant difference in both short and long term variability in fetal heart activity from baseline at day 2 and 4 following Dexamethasone administration while both short- and long-term variability decreased from baseline in fetus exposed to Betamethasone at day 2 with p value being less than 0.05.[10] Dexamethasone group had better variability than Betamethasone group on day 2.[10] Similarly in another study there was a transient decrease in fetal heart rate variability at 24 hours following drug administration which was significantly greater in Betamethasone group than Dexamethasone group.[13] With passage of time there was greater improvement in fetal heart rate variability in Dexamethasone group than Betamethasone group.[13]

These findings reported by Chaurasia et al is similar to our study which also revealed better fetal heart rate variability in Dexamethasone group than Betamethasone group at different points of time from 24h to 72h following drug administration.[13] However contrary findings of fetal heart rate variability were reported by Henry A et al.[11] They found no significant difference in fetal heart rate variability from baseline or between groups at different points time after drug administration.[11] On the other hand, non-comparative studies on individual drugs have shown a transient decrease in fetal heart rate variability following drug administration. This is seen at 48 hours following Dexamethasone administration which returns to baseline at day 4.[12] Similarly in majority of the patients fetal heart rate variability was found to be significantly reduced after administration of Betamethasone.[14]

Although prior studies have described transient changes in fetal heart rate parameters following antenatal corticosteroid administration, high-quality head-to-head data comparing dexamethasone and betamethasone in our setting remain limited. In this study, while the overall trends were consistent with existing literature, we further delineated the temporal pattern and relative magnitude of these effects between the two agents. A transient reduction in fetal heart accelerations was observed up to day 4, and fetal heart rate variability decreased most prominently on day 2, with more pronounced changes in the betamethasone group. These findings provide locally relevant comparative evidence and underscore the need for cautious CTG interpretation after maternal steroid administration to avoid unnecessary iatrogenic deliveries. The study reports transient decrease in fetal heart accelerations up to day 4 as well as in fetal heart rate variability that is more pronounced on day 2. The transient decrease in these parameters is greater in

Betamethasone group than Dexamethasone group. Therefore, fetal CTG should be carefully evaluated after administration of maternal steroids so that un-necessary iatrogenic deliveries can be avoided.

Conclusion

Administration of both dexamethasone and betamethasone was associated with a transient reduction in fetal heart rate accelerations and variability, with a more pronounced effect observed following betamethasone. These findings suggest that dexamethasone may have a comparatively lesser impact on fetal surveillance parameters. While both corticosteroids remain clinically effective for women at risk of preterm delivery, the observed differences—along with considerations of accessibility and cost—may support the preferential use of dexamethasone in appropriate settings. Importantly, clinicians should recognize the transient pharmacological effects of antenatal corticosteroids on fetal heart rate parameters to avoid misinterpretation and unnecessary iatrogenic preterm delivery.

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