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Original Research Article

The transient effect of dexamethasone on foetal cardiotocography in antenatal period

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ABSTRACT

Background: Preterm birth is a major contributor to neonatal morbidity and mortality worldwide. This study aimed to evaluate the impact of antenatal dexamethasone on CTG patterns, specifically FHR variability, accelerations, and foetal movements, in women at high risk of preterm labour.
Methods: A cross-sectional, comparative observational study was conducted in a Kamla Nehru hospital Shimla from June 23 to June 24. The study included 102 women aged 18-35 years, with a gestational age of 28-34 weeks, at risk for preterm delivery. Participants received a full course of dexamethasone (24 mg in divided doses 12 hours apart). CTG was recorded before administration and at 48- and 96-hours post-dexamethasone. Statistical analysis was performed using Chi-square tests with significance set at $p<0.05$.
Results: Before dexamethasone administration, all participants reported normal foetal movement, but 48 hours later, 53.9% reported decreased foetal movement ($p<0.001$). By 96 hours, the perception of foetal movement returned to normal in 98% of participants ($p=0.498$). FHR variability significantly decreased with moderate variability observed in 74.5% of cases at 48 hours ($p<0.001$). FHR accelerations decreased significantly from 94.1% before administration to 79.4% at 48 hours ($p=0.002$), but returned to baseline by 96 hours ($p=0.774$).
Conclusions: Dexamethasone administration resulted in transient changes in FHR patterns, including decreased accelerations, decreased moderate variability, and reduced foetal movement within the first 48 hours. However, these effects were short-lived, with normalization occurring by 96 hours.

Keywords: Antenatal corticosteroids, Dexamethasone, Preterm labour, Cardiotocography, Foetal heart rate variability, Foetal monitoring

INTRODUCTION

Preterm birth is a significant global public health issue, being a leading cause of perinatal mortality and morbidity.[1] Preterm labour, which occurs in about 12% of pregnancies, is responsible for nearly half of all preterm births. Neonatal morbidity is strongly associated with preterm birth, with 70% of such complications linked to premature delivery, while mortality in very premature infants born before 32 weeks is approximately 2%.[2] The world health organization estimates that preterm birth occurs in 5-18% of pregnancies across 184 countries worldwide. In approximately half of the cases of spontaneous preterm labour, the cause remains unknown, highlighting the limited understanding of the underlying mechanisms of preterm labour.[3]

To prevent premature birth and its complications, common strategies include bed rest, tocolysis, antibiotics, and antenatal corticosteroid administration.[4-6]

Research has shown that administering corticosteroids to women at risk of preterm delivery can significantly reduce the likelihood of complications such as fetal and neonatal death, respiratory distress syndrome (RDS), intraventricular hemorrhage, necrotizing enterocolitis,

systemic infections, and developmental delays.[7-9] Antenatal corticosteroids are particularly effective for fetal lung maturation when given 24 hours before preterm delivery, with the most significant benefits observed between 24 and 34 weeks of gestation. The greatest reduction in RDS risk-up to 50%-occurs when delivery happens within 7 days after completing the full steroid regimen.[1,10]

Betamethasone and dexamethasone are commonly used synthetic glucocorticoids for maternal treatment in cases of anticipated preterm birth. Both medications cross the placenta in their active form and promote foetal organ maturation by modifying gene expression.[11-14] The glucocorticoid effects of these drugs lead to rapid physiological changes, such as increased lung protein production, phospholipid synthesis, and surfactant formation, which are beneficial for foetal lung maturation. However, corticosteroids can also cause side effects, including elevated foetal blood pressure and reduced cerebral blood flow, which may reduce the risk of intraventricular haemorrhage in the short term.[15,16]

It is therefore important to understand short-term effects of corticosteroids on the foetus in utero, and whether there are correlations between these effects and neonatal outcomes and/or later outcomes. Awareness of the pharmacologic effect of corticosteroids on parameters for the assessment of foetal well-being might prevent unnecessary iatrogenic delivery of preterm foetus. This study aimed to study the CTG before and after antenatal corticosteroids administration in patients at high risk of preterm labour to reduce preterm risks.

The aim of this study was to evaluate the changes in the cardiotocograph (CTG) before and after the administration of antenatal corticosteroids in patients at high risk of preterm labour. Additionally, the study also seeks to investigate the effects of maternal dexamethasone on the foetal CTG in patients who present with decreased foetal movement but have a previously normal CTG.

METHODS

A cross-sectional comparative observational study was conducted in department of obstetrics and gynaecology, in a tertiary care hospital of Shimla, for a period of one year from 24/08/2023. The study included only women who received dexamethasone according to the hospital protocol and met the specified inclusion criteria. Eligible participants were women aged 18-35 years, with a gestational age between 28 and 34 weeks (calculated from the first day of their last menstrual period or the first ultrasound), carrying a singleton pregnancy and at risk of preterm delivery. The inclusion criteria specifically included women with a history of preterm labour, premature rupture of membranes (within 12 hours), placenta previa, pregnancy-induced hypertension (PIH), preeclampsia, preterm uterine contractions, or polyhydramnios.

Exclusion criteria encompassed women who were in active labour, had foetal growth restriction (FGR), had received corticosteroids during the current pregnancy, or were carrying a foetus with suspected structural abnormalities. Additionally, women with multiple pregnancies, those with contraindications to corticosteroid administration, or those with a history of abnormal Doppler studies were excluded from the study. Cases with history of foetal distress, medical disorders and chorioamnionitis were also excluded from the study.

Sample size was calculated by taking confidence level as 95%, expected percentage of non-reactive cardiotocography as 84%.[17] Absolute error as 5% and finite population as 200 patients as per inclusion criteria in 9 months. Final sample size came out to be 102 patients. Sample size was calculated using Open Epi software version 3.

The ethical approval was obtained from Institutional ethical committee and informed written consent was obtained from all study participants after explaining the study in their vernacular language. A detailed history of the patient was recorded in a pre-validated proforma designed for the study. Detailed obstetrical examination including per abdomen examination and per speculum examination (when required) was done.

Women received dexamethasone intramuscularly with a total steroid dose of 24 mg in equal four divided doses 12 hours apart according to the hospital protocol. All patients were asked about foetal movements and activity every day from day 1- day 4 from the first dose of dexamethasone. CTG examination was done to all patients for foetal heart monitoring as a non-stress. Foetal heart rate was monitored with Heteromach Meditronix ISO 13485 foetal monitor cardiotocogram. The neonatal outcome was monitored in form of severe neonatal respiratory distress within 24 hours, and severe neonatal respiratory distress within first week of birth, neonatal death within first week of birth, neonatal death until 28 completed days of life, still birth or neonatal death (any baby beath).

After the completion of data collection, Data was entered in MS excel spreadsheet, cleaned for errors and were analysed using EpiInfo software version 7.2.2.5. Qualitative variables were presented as frequencies, percentages. Inferential analyses were done for qualitative data using Chi square test for independent groups. The level of significance was taken at $p<0.005$ as significant, otherwise non-significant.

RESULTS

A total of 102 ANC mothers fulfilling the inclusion criteria were enrolled in the study. The socio-demographic characteristics of study participants are shown in Table 1. Nearly half of the study participants were between 18-24 years of age (49%), belonged to lower-middle class (50%), normal BMI range (53.9%) with majority being from rural

area (65.7%). Most of the participants in the study were multigravida (75 subjects, 73.5%) Among the multigravida mothers 39 subjects (38.2%) had previous full-term delivery, 25 participants (24.5%) had pre-term delivery and 11 participants (10.8%) had ended in abortion.

PPROM (25.5%) and previous history of preterm (24.5%) were the most common indications for administration of dexamethasone followed by preterm uterine contractions (18 cases, 17.7%), placenta previa and PIH (15 cases each, 14.7%). Three mothers (2.9%) were administered dexamethasone due to polyhydramnios (Figure 1).

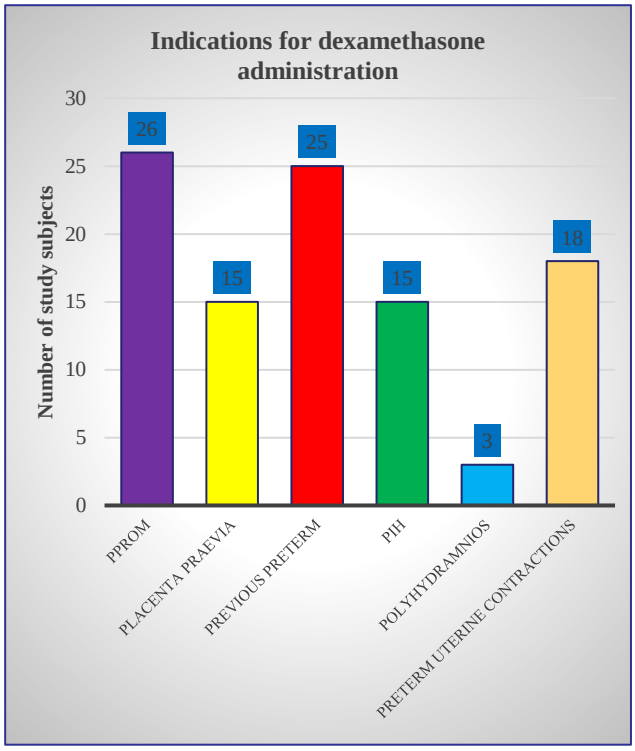

Figure 1: Indications for dexamethasone administration among study participants.

Before the administration of dexamethasone, all participants (102 women) reported normal foetal movement. However, 48 hours after administration, there was a significant shift, with 47 women (46.1%) perceiving normal foetal movement and 55 women (53.9%) reporting decreased movement (p<0.001). By 96 hours after administration, the perception of foetal movement returned to near normal, with 100 women (98%) reporting normal foetal movement and only 2 women (2.0%) reporting decreased movement (p=0.498) (Figure 2).

On comparison of FHR before and 48 hours after administration of first dose of dexamethasone (Table 2), it was noted that FHR remained within the normal range (110-160 BPM) for all 102 participants both before and 48 hours after administration, with no cases of out-of-range FHR (<110 or >160 BPM). A significant increase in minimal variability (<5 BPM) was observed 48 hours after administration, with 26 participants (25.5%) showing minimal variability compared to just 8 (7.8%) before dexamethasone. Meanwhile, the number of participants with moderate variability (6-25 BPM) decreased from 94 (92.2%) to 76 (74.5%) with the difference being statistically significant (p<0.001). The presence of foetal heart rate acceleration decreased significantly, from 96 participants (94.1%) before administration to 81 (79.4%) 48 hours after administration (p=0.002).

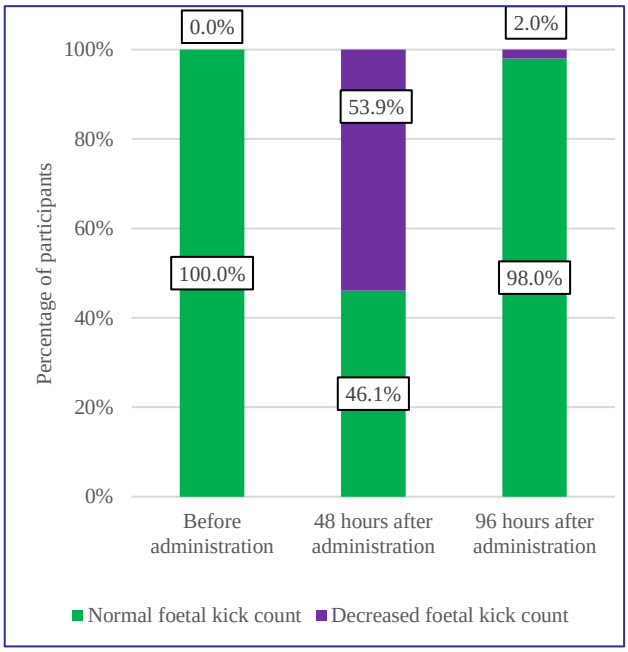

Figure 2: Comparison of perception of foetal kick count before and after administration of dexamethasone.

Table 1: Socio-demographic parameters of study participants.

Parameters	N	Percentage (%)
Age groups (in years)		
18-24	50	49.0
25-30	38	37.3
31-35	14	13.7
Residence		
Rural	67	65.7
Urban	35	34.3
Socio-economic status		
Upper middle	42	41.2
Lower middle	51	50.0
Lower	9	8.8
BMI (kg/m^2)		
<18.5	24	23.5
18.5-24.9	55	53.9
25.0-29.9	23	22.6
Gravida		
Primigravida	27	26.5
Multigravida	75	73.5
Total	102	100.0

Table 2: Comparison of baseline FHR variables to FHR variables 48 hours (day 2) after first dose of dexamethasone administration.

Foetal heart rate	Time duration with respect to dexamethasone administration, N (%)		P value
	Before administration	48 hours after administration	
Count-beats per minute (BPM)			
Within normal limit (110-160 BPM)	102 (100)	102 (100)	-
Out of range (<110 or >160 BPM)	0	0	
Variability			
Minimal (<5 BPM)	8 (7.8)	26 (25.5)	<0.001
Moderate (6-25 BPM)	94 (92.2)	76 (74.5)	
Acceleration			
Present	96 (94.1)	81 (79.4)	0.002
Absent	6 (5.9)	21 (20.6)	
Total	102 (100)	102 (100)	

Table 3: Comparison of baseline FHR variables to FHR variables 96 hours (day 4) after first dose of dexamethasone administration.

Foetal heart rate	Time duration with respect to dexamethasone administration, N (%)		P value
	Before administration	96 hours after administration	
Count-beats per minute (BPM)			
Within normal limit (110-160 BPM)	102 (100)	102 (100)	-
Out of range (<110 or >160 BPM)	0	0	
Variability			
Minimal (<5 BPM)	8 (7.8)	9 (8.8)	0.800
Moderate (6-25 BPM)	94 (92.2)	93 (91.2)	
Acceleration			
Present	96 (94.1)	95 (93.1)	0.774
Absent	6 (5.9)	7 (6.9)	
Total	102 (100)	102 (100)	

On comparison of FHR before and 96 hours after the first dose of dexamethasone administration, it was observed that FHR remained within the normal range (110-160 BPM) for all 102 participants and variability (<5 BPM) remained relatively stable, with 8 participants (7.8%) showing minimal variability before administration and 9 participants (8.8%) showing it 96 hours later (p=0.800). Foetal heart rate acceleration was also comparable between pre-administration and 96 hours after administration of dexamethasone (Table 3).

On further follow-up all the study subjects (100.0%) had live-birth with nearly three-forth (73.5%) having no neonatal complications. Nineteen neonates (18.6%) developed RDS within 24 hours of birth, while another eight neonates (7.9%) had RDS within first week of birth. There was no incidence of either early or late neonatal deaths.

DISCUSSION

Preterm birth is a major cause of perinatal death and disability, representing a significant global public health concern.[18] Synthetic corticosteroids, such as betamethasone and dexamethasone, are commonly given to pregnant women at risk of preterm delivery to help prevent or reduce complications associated with prematurity, including the need for respiratory support, intensive care admissions, RDS, intraventricular haemorrhage, and necrotizing enterocolitis. The administration of antenatal steroids can influence foetal heart rate patterns, which may be detected in the CTG.[16,19]

In the present study, we included 102 participants who received the standard dosage regimen recommended by the RCOG, which involves a single course of dexamethasone 6 mg intramuscularly, administered 12 hours apart for a total of 4 doses, following the hospital protocol. In the present study, 25.5% of participants had premature rupture of membranes (PROM), 14.7% had placenta previa, 24.5% had a previous history of preterm labour, 14.7% had PIH, 2.9% had polyhydramnios, and 17.7% had preterm uterine contractions. In comparison, the study by Ahmed et al found that 60.3% of participants had PROM, 23.5% had placenta previa, and 16.2% had a history of preterm labour.[17] The differences in the indications for dexamethasone use highlight the need for

tailored approaches to manage the diverse conditions that can lead to preterm delivery.

In the present study, a significant decrease in foetal movement was observed from day 0 to 2, with a p<0.001, indicating a statistically significant reduction. However, this decrease was transient, as by day 4, the percentage of movements had nearly returned to baseline levels, with a p=0.498, indicating no significant change at this time point. This suggests that the effect of dexamethasone on foetal movement is short-lived. Studies by Ahmed et al and Rotmensch et al also observed changes in foetal movement, though they used different methodologies.[17,20] Ahmed et al employed quantitative measures and found a statistically significant decrease in foetal movement from day 0 to 2 (p<0.001).[17] However, by day 4, the p=0.7, indicating no significant change. Similarly, Rotmensch et al found a statistically significant decrease in foetal movement from day 0 to 2 (p<0.05), but by day 4, the change was not statistically significant indicating no sustained effect.[17] Chaurassia et al though evaluated the foetal kick count at baseline and after 48 hours also observed a trend similar to our study, however the difference was statistically not significant (p=0.062).[21]

The comparison of FHR variables before and after the first dose of dexamethasone administration across different studies (Ahmed et al, Rotmensch et al, Chaurassia et al and the present study) reveals a consistent finding: there were no significant changes in baseline FHR from day 0 to 2 or from day 0 to 4.[17,20,21] This suggests that dexamethasone administration does not have a substantial long-term impact on baseline FHR, which remains stable over the observed period. In terms of variability, both Ahmed et al and Rotmensch et al reported a significant decrease in variability from Day 0 to 2 (p=0.01), with this effect persisting, albeit less pronounced, by day 4 (p=0.5).[17,20] However, Rotmensch et al noted that this decrease in variability was significant only on day 2 (p<0.05), and by day 4, the effect was statistically not significant.[20] The present study also observed a significant reduction in moderate variability by day 2 (p<0.001), which almost normalized by day 4. Chaurassia et al also observed on day 2 that there was an increase in variability, although this change was not statistically significant (p=0.14).[21]

Regarding accelerations, both Ahmed et al and Rotmensch et al reported significant decreases in accelerations by day 2.[20] Ahmed et al found that accelerations largely normalized by day 4.[6,20] Similarly, the present study showed a significant decrease in accelerations by day 2, with normalization occurring by day 4. Chaurassia et al did not provide day 4 data, but no significant changes were observed by day 2.[21]

In summary, while there was a temporary reduction in foetal heart rate accelerations following dexamethasone administration, this effect generally diminished by day 4. The overall pattern across all studies indicates that dexamethasone administration leads to short-term changes in foetal heart rate variables, particularly in the variability and the accelerations, but these effects do not persist over time.

CONCLUSION

Dexamethasone administration has a transient effect on foetal kick count, foetal heart rate variability and accelerations. These effects are most pronounced within the first 48 hours, with significant decrease in foetal kick count, moderate variability and the presence of accelerations. However, by 96 hours post-administration, these changes are no longer statistically significant, suggesting a normalization or adaptation period following the initial dose of dexamethasone. Baseline FHR remains unaffected throughout the 96-hour period. While these changes are transient and short lived proper foetal monitoring and patient education are essential to manage these transient effects effectively so as to prevent iatrogenic preterm deliveries.

Recommendations

A normal FHR pattern is usually an indicator for reassuring foetal status, while an abnormal FHR pattern does not necessarily be related with hypoxia or acidosis. Clinicians should be aware of the expected changes in CTG following dexamethasone administration. This awareness helps in preventing unnecessary interventions due to misinterpretation of CTG changes. Continuous monitoring and repeated CTG assessment, doppler studies helps to distinguish between medication effects and actual foetal compromise. Increased frequency of CTG monitoring within the first 48 hours post-dexamethasone administration may be warranted to closely observe and understand the temporary changes in FHR patterns.

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