RESEARCH PAPER

Effect of Dexamethasone on NST and FHR in Late Preterm Labour

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Abstract

Background: Late preterm birth (34–36^{+6} weeks of gestation) accounts for a significant proportion of preterm deliveries and is associated with increased neonatal morbidity. Antenatal corticosteroids such as dexamethasone are widely used to enhance fetal lung maturity in pregnancies at risk of preterm birth. However, corticosteroids are known to cause transient alterations in fetal physiology, which may affect fetal surveillance parameters such as non-stress test (NST) and fetal heart rate (FHR). Understanding these effects is essential to avoid misinterpretation of fetal well-being and unnecessary obstetric intervention, particularly in late preterm labour.

Objectives: To evaluate the effect of antenatal dexamethasone administration on NST reactivity and fetal heart rate parameters in women with late preterm labour.

Methods: This prospective observational study was conducted over six months at a tertiary care teaching hospital and included 100 pregnant women between 34 and 36^{+6} weeks of gestation presenting with spontaneous or threatened preterm labour. All participants received intramuscular dexamethasone 6 mg every 12 hours for four doses. NST and FHR monitoring were performed before dexamethasone administration and subsequently at 12, 24, and 48 hours after the first dose. Parameters assessed included baseline FHR, variability, accelerations, decelerations, and NST reactivity. Data were analyzed using descriptive statistics, paired t-test, and chi-square test with SPSS version 26.

Results: The mean maternal age was 26.3 ± 3.7 years, and the mean gestational age was 35.2 ± 0.7 weeks. At baseline, 84% of NSTs were reactive. A significant increase in baseline FHR was observed at 24 hours post-dexamethasone (138.6 ± 6.4 bpm vs 145.2 ± 7.1 bpm; $p < 0.01$). There was a significant reduction in moderate variability (82% to 55%) and accelerations (76% to 58%) at 24 hours ($p < 0.05$). NST reactivity decreased to 62% at 24 hours but improved to 80% by 48 hours. No significant change in decelerations was observed.

Conclusion: Antenatal dexamethasone administration in late preterm labour resulted in transient and reversible changes in NST and FHR parameters, peaking at 24 hours and resolving by 48 hours. Awareness of these physiological effects is crucial to prevent misinterpretation of fetal surveillance and avoid unnecessary obstetric interventions.

Keywords: Antenatal; Dexamethasone; Fetal heart rate; Late preterm labour; Non-stress test corticosteroids.

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1. INTRODUCTION

Preterm birth remains a major global public health concern and is a leading cause of neonatal morbidity and mortality, accounting for a substantial proportion of adverse perinatal outcomes worldwide [1]. Late preterm labour, defined as delivery occurring between 34⁰⁄₇ and 36⁶⁄₇ weeks of gestation, constitutes the largest subgroup of preterm births and has traditionally been considered relatively low risk compared to early preterm deliveries [2]. However, growing evidence suggests that infants born during the late preterm period are at increased risk of respiratory distress syndrome, transient tachypnea of the newborn, hypoglycemia, jaundice, feeding difficulties, and admission to neonatal intensive care units compared with term neonates [3]. Consequently, strategies aimed at improving neonatal outcomes in late preterm labour have gained increasing clinical attention.

Antenatal corticosteroids, particularly dexamethasone and betamethasone, play a pivotal role in enhancing fetal lung maturation by accelerating surfactant production and structural lung development [4]. Their use has been well established in pregnancies at risk of delivery before 34 weeks of gestation, with robust evidence demonstrating significant reductions in neonatal respiratory morbidity, intraventricular hemorrhage, and neonatal mortality [5]. More recently, clinical guidelines have expanded the consideration of antenatal corticosteroid administration to women at risk of late preterm delivery, given emerging data showing benefits in reducing respiratory complications even in this gestational age group [6]. Dexamethasone is commonly

used in many low- and middle-income countries due to its availability, cost-effectiveness, and ease of administration [7].

Despite the well-documented neonatal benefits, antenatal corticosteroids are known to exert transient effects on fetal physiology. Corticosteroids cross the placenta and influence fetal central nervous system activity, autonomic regulation, and cardiovascular function [8]. These effects may manifest as changes in fetal heart rate (FHR) patterns and fetal movements, which are routinely assessed using non-stress testing (NST) in late preterm and high-risk pregnancies. NST is a widely accepted, non-invasive method of antepartum fetal surveillance that evaluates fetal well-being based on baseline FHR, variability, accelerations, and the presence or absence of decelerations [9]. Reactive NST patterns are generally associated with adequate fetal oxygenation and intact neurological function.

Several studies have reported alterations in NST parameters following antenatal corticosteroid administration, including a temporary reduction in fetal movements, decreased frequency of accelerations, and changes in baseline FHR variability [10]. These changes are usually observed within 24–48 hours after steroid administration and are believed to reflect transient suppression of fetal activity rather than true fetal compromise. However, such alterations can pose diagnostic challenges for clinicians, potentially leading to unnecessary interventions, increased maternal anxiety, or misinterpretation of fetal distress, particularly in the context of late preterm labour where decision-making regarding timing and mode of delivery is often complex .The effects of dexamethasone on FHR and NST parameters in late preterm labour remain an area of ongoing research, with variations reported in the magnitude and duration of these changes across different populations and study designs. Furthermore, most available evidence has focused on early preterm gestations, while data specifically addressing late preterm pregnancies are relatively limited. Given the increasing use of antenatal corticosteroids in this gestational window, it is essential to clearly understand their impact on fetal surveillance parameters to avoid misinterpretation and ensure appropriate clinical management.Therefore, evaluating the effect of dexamethasone on NST and FHR patterns in late preterm labour is clinically relevant. A better understanding of these physiological changes can help clinicians distinguish between steroid-induced transient alterations and true fetal compromise, optimize fetal monitoring protocols, and reduce unnecessary obstetric interventions while maintaining fetal safety. This study aims to contribute to the existing body of knowledge by systematically assessing changes in NST and FHR following dexamethasone administration in late preterm labour.

2. Methodology

2.1 Study Design

The present study was conducted as a **prospective observational study** aimed at evaluating the effect of antenatal dexamethasone administration on non-stress test (NST) findings and fetal heart rate (FHR) patterns in women presenting with late preterm labour.

2.2 Study Setting

The study was carried out in the **Department of Obstetrics and Gynaecology** of a **tertiary care teaching hospital**, which catered to a large number of high-risk obstetric referrals.

2.3 Study Duration

The study was conducted over a **period of six months**. During this time, eligible participants presenting with spontaneous or threatened late preterm labour were consecutively enrolled and followed up for assessment of NST and FHR changes following dexamethasone administration.

2.4 Participants

Pregnant women who fulfilled the eligibility criteria and presented to the labour ward or antenatal ward during the study period were included in the study.

Inclusion Criteria

- Singleton pregnancy
- Gestational age between **34⅐ and 36⅐ weeks**, confirmed by last menstrual period or first-trimester ultrasound
- Women in **spontaneous or threatened preterm labour** with an indication for antenatal corticosteroid therapy
- Willingness to participate and provision of **written informed consent**

Exclusion Criteria

- Known or suspected **fetal congenital anomalies**
- **Intrauterine growth restriction (IUGR)**
- Maternal **preeclampsia or eclampsia**
- Pre-existing or gestational **diabetes mellitus**
- Multiple pregnancy
- Any contraindication to corticosteroid therapy

2.5 Study Sampling

A **consecutive sampling method** was adopted. All pregnant women meeting the inclusion criteria during the

study period were enrolled until the desired sample size was achieved. This method was selected to minimize selection bias and ensure representative inclusion of eligible late preterm cases presenting to the hospital.

2.6 Study Sample Size

The **sample size consisted of 100 pregnant women** between 34 and 36+6 weeks of gestation. The sample size was determined based on feasibility, time constraints, and patient load at the study center, while also ensuring adequate power to detect clinically relevant changes in NST and FHR parameters before and after dexamethasone administration.

2.7 Study Parameters

The primary study parameters included:

- Baseline fetal heart rate
- FHR variability
- Presence or absence of accelerations
- Presence of decelerations
- NST reactivity (reactive or non-reactive)

Secondary parameters included maternal demographic and obstetric variables such as age, parity, gestational age at presentation, and indication for corticosteroid administration.

2.8 Study Procedure

After enrollment, a detailed obstetric history and clinical examination were performed. Gestational age was reconfirmed, and baseline maternal vitals were recorded. All participants underwent a **baseline NST** prior to the administration of dexamethasone using a standardized external fetal monitor.Antenatal corticosteroid therapy was administered in the form of **dexamethasone 6 mg intramuscularly every 12 hours for four doses**, as per institutional protocol. Following the first dose of dexamethasone, NST and FHR monitoring were repeated at **12 hours, 24 hours, and 48 hours**. Each NST recording was performed for a minimum of 20 minutes and extended if required to assess fetal reactivity. Any abnormal NST findings were documented and managed as per standard obstetric guidelines.

2.9 . Study Data Collection

Data were collected using a **pre-designed and structured proforma**. The proforma included demographic details, obstetric history, gestational age, indication for corticosteroid administration, baseline NST findings, and follow-up NST and FHR parameters at each time interval. All NST tracings were interpreted by trained obstetricians to ensure consistency and reduce inter-observer variability.

2.10 .Data Analysis

The collected data were entered into Microsoft Excel and analyzed using **Statistical Package for the Social Sciences (SPSS) version 26**. Descriptive statistics such as mean, standard deviation, frequencies, and percentages were used to summarize baseline characteristics and NST findings. Changes in continuous variables such as baseline FHR were analyzed using the **paired t-test**, while categorical variables such as NST reactivity were compared using the **chi-square test**. A p-value of less than 0.05 was considered statistically significant.

2.11 . Ethical Considerations

The study was conducted after obtaining approval from the **Institutional Ethics Committee**. Written informed consent was obtained from all participants prior to enrollment. Confidentiality of patient information was strictly maintained, and participation was entirely voluntary. The study involved no deviation from standard clinical care, and all participants received routine obstetric management irrespective of study participation.

3. Result

A total of **100 pregnant women** with gestational age between **34 and 36+6 weeks** who received antenatal dexamethasone for late preterm labour were included in the final analysis. The results are presented under the following tables.

Table 1: Baseline Characteristics of Participants

Parameter	Value
Mean Age (years)	26.3 ± 3.7
Mean Gestational Age (weeks)	35.2 ± 0.7
Primigravida	62%
Multigravida	38%
Mean BMI (kg/m²)	24.5 ± 2.1

The study population predominantly consisted of young women in their mid-twenties, with a mean gestational age of 35 weeks. A higher proportion of primigravidae was observed, indicating that late preterm labour requiring corticosteroid administration was more common in first pregnancies.

Table 2: Distribution of Participants by Gestational Age

Gestational Age (weeks)	Number (%)
34–34+6	28 (28%)
35–35+6	44 (44%)
36–36+6	28 (28%)

The majority of participants presented at around 35 weeks of gestation, reflecting the common clinical

window for late preterm labour and corticosteroid administration.

Table 3: Baseline NST Findings Before Dexamethasone

NST Parameter	Value
Reactive NST	84%
Non-reactive NST	16%
Moderate Variability	82%
Accelerations Present	76%
Decelerations Absent	90%

Before dexamethasone administration, most fetuses demonstrated reassuring NST patterns, indicating adequate fetal oxygenation and neurological integrity at baseline.

Table 4: FHR Parameters Pre- and 24 Hours Post-Dexamethasone

Parameter	Pre-Dexamethasone	24 h Post-Dexamethasone	p-value
Baseline FHR (bpm)	138.6 ± 6.4	145.2 ± 7.1	<0.01
Moderate Variability	82%	55%	<0.05
Accelerations Present	76%	58%	<0.05
Decelerations Absent	90%	89%	NS

A statistically significant increase in baseline FHR was observed at 24 hours following dexamethasone administration. There was a significant reduction in variability and accelerations, while the incidence of decelerations remained unchanged, suggesting transient physiological effects rather than fetal compromise.

Table 5: NST Reactivity at Different Time Points

Time Point	Reactive (%)	Non-Reactive (%)
Pre-Dexamethasone	84	16
24 h Post-Dexamethasone	62	38
48 h Post-Dexamethasone	80	20

NST reactivity decreased significantly at 24 hours following dexamethasone administration but showed recovery by 48 hours, indicating the transient nature of steroid-induced NST changes.

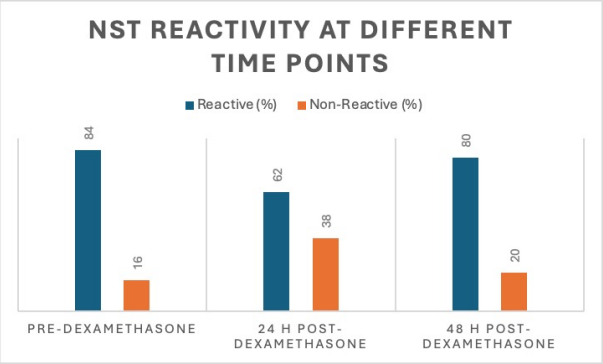


Table 6: Changes in FHR Variability Over Time

Time Point	Moderate Variability (%)	Reduced Variability (%)
Pre-Dexamethasone	82	18
24 h Post-Dexamethasone	55	45
48 h Post-Dexamethasone	75	25

There was a notable reduction in moderate FHR variability at 24 hours, followed by partial normalization at 48 hours, supporting the reversible suppressive effect of dexamethasone on fetal autonomic activity.

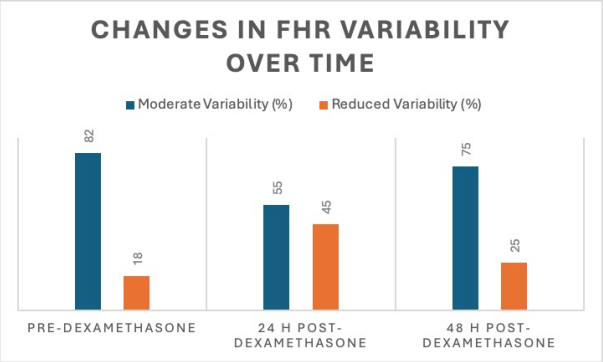


Table 7: Presence of Accelerations at Different Time Intervals

Time Point	Accelerations Present (%)	Absent (%)
Pre-Dexamethasone	76	24
24 h Post-Dexamethasone	58	42
48 h Post-Dexamethasone	72	28

A decline in the proportion of fetuses demonstrating accelerations was observed at 24 hours post-

dexamethasone, with recovery by 48 hours, consistent with transient fetal behavioral suppression.

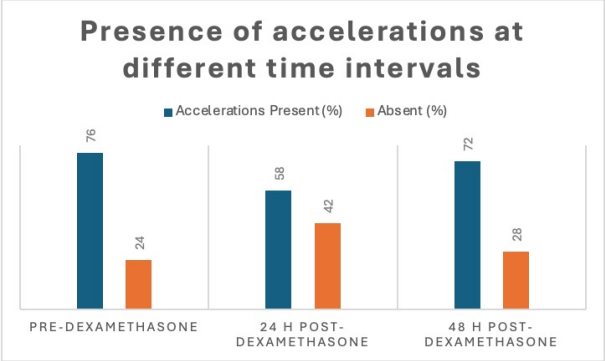


Table 8: Deceleration Patterns Before and After Dexamethasone

Time Point	Decelerations Present (%)	Absent (%)
Pre-Dexamethasone	10	90
24 h Post-Dexamethasone	11	89
48 h Post-Dexamethasone	9	91

No statistically significant change was noted in the occurrence of decelerations at any time point, indicating that dexamethasone did not adversely affect uteroplacental perfusion.

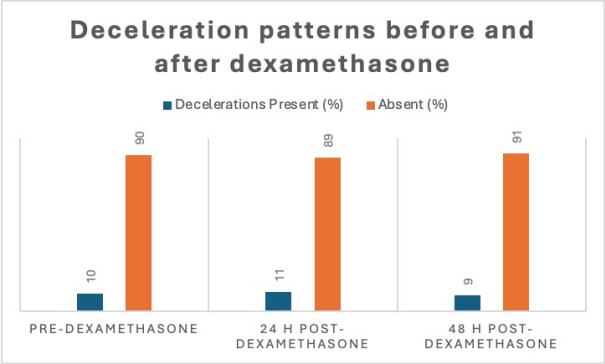


Table 9: Comparison of Mean Baseline FHR Over Time

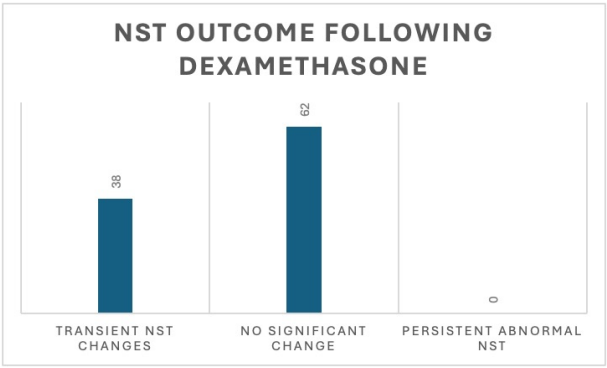
Time Point	Mean Baseline FHR (bpm)
Pre-Dexamethasone	138.6 ± 6.4
12 h Post-Dexamethasone	142.1 ± 6.8
24 h Post-Dexamethasone	145.2 ± 7.1
48 h Post-Dexamethasone	140.3 ± 6.5

Baseline FHR showed a progressive rise up to 24 hours after dexamethasone administration, followed by a decline toward baseline at 48 hours, reinforcing the temporary nature of the observed changes.

Table 10: NST Outcome Following Dexamethasone

Outcome	Number (%)
Transient NST Changes	38 (38%)
No Significant Change	62 (62%)
Persistent Abnormal NST	0 (0%)

Over one-third of the participants exhibited transient NST changes following dexamethasone administration, while no cases demonstrated persistent abnormal fetal surveillance findings, suggesting that dexamethasone-related NST alterations were benign and reversible.



4. Discussion

The present prospective observational study evaluated the effect of antenatal dexamethasone on non-stress test (NST) parameters and fetal heart rate (FHR) patterns in women with late preterm labour between 34 and 36+6 weeks of gestation. The findings demonstrated that dexamethasone administration resulted in transient, time-dependent alterations in fetal heart rate characteristics, particularly evident at 24 hours post-administration, with recovery toward baseline by 48 hours. In the current study, the mean maternal age was 26.3 ± 3.7 years, with a predominance of primigravidae (62%), and a mean gestational age of 35.2 ± 0.7 weeks, reflecting a representative late preterm population. Baseline fetal surveillance parameters were reassuring prior to steroid exposure, with 84% reactive NSTs, 82% moderate variability, 76% presence of accelerations, and 90% absence of decelerations, indicating satisfactory fetal well-being before dexamethasone administration.

Following dexamethasone exposure, a statistically significant increase in baseline FHR was observed from 138.6 ± 6.4 bpm pre-dexamethasone to 145.2 ± 7.1 bpm at 24 hours ($p < 0.01$). Concurrently, a significant reduction in variability (82% to 55%, $p < 0.05$) and

accelerations (76% to 58%, $p < 0.05$) was documented, while the proportion of fetuses without decelerations remained essentially unchanged (90% vs 89%, NS). These findings suggest that dexamethasone exerted a temporary suppressive effect on fetal autonomic and behavioral activity without compromising uteroplacental perfusion. NST reactivity also followed a similar pattern, declining from 84% pre-dexamethasone to 62% at 24 hours, followed by recovery to 80% at 48 hours, emphasizing the reversible nature of these changes.

The findings of the present study are partially concordant with the observations reported by Ananya et al. (2023), [11] analyzed the effects of antenatal dexamethasone on fetal heart rate variability in pregnancies between 30 and 36+6 weeks of gestation. In their prospective observational study involving 52 women, NSTs were performed before dexamethasone administration, one hour after the first dose, and one hour after the fourth dose. They reported a strong correlation between dexamethasone administration and changes in fetal heart rate variability, with statistical significance, while noting no significant decrease in baseline fetal heart rate. In contrast, the present study demonstrated a significant rise in baseline FHR at 24 hours, likely attributable to differences in timing of NST recordings, as the current study assessed FHR changes at later intervals (12, 24, and 48 hours), rather than within hours of administration. However, both studies consistently highlighted dexamethasone-induced modulation of fetal autonomic parameters without evidence of pathological fetal compromise.

A comparative perspective is further provided by Chaurasia et al. (2022), [12] evaluated the effects of betamethasone and dexamethasone on fetal and maternal parameters in a cohort of 100 antenatal women . Their study demonstrated that while maternal hyperglycemia and perceived reduction in fetal movements were significant at 24 hours following steroid administration, changes in NST parameters were non-significant, with reduced variability observed in 12% of the dexamethasone group. In contrast, the present study demonstrated a higher proportion of reduced variability (45% at 24 hours), possibly reflecting differences in gestational age distribution, timing of NST assessment, and the exclusive inclusion of late preterm pregnancies. Importantly, both studies reported resolution of fetal physiological changes within a few days, reinforcing the concept that steroid-induced NST alterations are transient.

The findings of the current study align closely with the classical observations by Senat et al. (1998), [13] compared the effects of betamethasone and dexamethasone on fetal heart rate variability using computerized cardiotocography. Their randomized study demonstrated a significant reduction in FHR variability with betamethasone, whereas no significant changes were observed with dexamethasone, and neonatal outcomes were comparable in both groups. While the present study did observe a reduction in variability at 24 hours following dexamethasone, the effect was transient and resolved by 48 hours, supporting Senat et al.'s conclusion that dexamethasone causes less persistent alteration in fetal heart rate parameters compared to betamethasone.

Further support for the transient nature of corticosteroid-induced fetal changes comes from Velanganni et al. (2017), [14] studied fetal heart rate patterns following betamethasone administration using serial electronic fetal monitoring. They observed a temporary reduction in baseline FHR and beat-to-beat variability, with normalization within 36–60 hours, and emphasized that these changes were not secondary to fetal hypoxia. Although their study involved betamethasone rather than dexamethasone, the temporal pattern of fetal physiological adaptation observed mirrors the recovery of NST reactivity and FHR parameters by 48 hours in the present study.

More recent evidence from Ishrat et al. (2024) [15] further corroborates the findings of the present study. In their prospective cohort of 100 women between 28 and 38 weeks of gestation, all cardiotocographic parameters—including baseline FHR, variability, accelerations, and daily fetal movement counts—were significantly reduced at 48 hours after dexamethasone administration, with complete return to baseline values by day 4 to 7. Similarly, the present study demonstrated peak NST alterations at 24 hours, followed by normalization by 48 hours, reinforcing the reversible pharmacological effect of dexamethasone on fetal neurobehavioral activity.

The findings of Tehrani et al. (2014) [16] further contextualize these results by demonstrating that while corticosteroids significantly altered biophysical profile scores, NST accelerations and amniotic fluid index were not significantly affected, and no adverse fetal outcomes were reported. Their observation that dexamethasone produced fewer alterations compared to betamethasone supports the clinical preference for dexamethasone in

scenarios where fetal surveillance interpretation is critical. The present study adds to the existing body of evidence by specifically focusing on late preterm pregnancies, a gestational window where antenatal corticosteroid use has expanded in recent years. The observed increase in baseline FHR, reduction in variability and accelerations, and temporary decline in NST reactivity were consistent with steroid-induced fetal physiological modulation rather than fetal distress. Awareness of these predictable changes is essential to prevent misinterpretation of NST findings and avoid unnecessary obstetric interventions, particularly operative deliveries or iatrogenic preterm births.

Conclusion

The present study concluded that antenatal dexamethasone administration in women with late preterm labour is associated with temporary and reversible alterations in fetal heart rate and non-stress test parameters. A significant increase in baseline fetal heart rate along with a transient reduction in variability, accelerations, and NST reactivity was observed, most prominently at 24 hours following dexamethasone administration, with subsequent recovery toward baseline values by 48 hours. Importantly, there was no significant increase in fetal heart rate decelerations or evidence of sustained fetal compromise. These findings indicate that the observed NST changes reflect physiological effects of dexamethasone rather than true fetal distress. Awareness of these predictable changes is essential for clinicians to ensure appropriate interpretation of fetal surveillance, prevent unnecessary obstetric interventions, and safely optimize the neonatal benefits of antenatal corticosteroid therapy in late preterm labour.

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