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Antenatal Corticosteroids in Preterm Infants: A Gestational Age-Based Comparison of Respiratory Outcomes

Reem Abdullah Ahmed Hassan
Department of Obstetrics and Gynecology
Faculty of Medicine, Taiz University, Taiz - Yemen
reemmoleki@gmail.com

Seena Baggash
Department of Obstetrics and Gynecology
Faculty of Medicine, Taiz University, Taiz - Yemen
seena.baggash 96@gmail.com

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Reem Abdullah Ahmed Hassan
Department of Obstetrics and Gynecology
Faculty of Medicine, Taiz University, Taiz - Yemen

Seena Baggash
Department of Obstetrics and Gynecology
Faculty of Medicine, Taiz University, Taiz - Yemen

Abstract

Background

Dexamethasone is an antenatal corticosteroid injected into mothers at risk of preterm labor (usually 24–34 weeks) to accelerate fetal lung maturation, significantly reducing risks of neonatal death, respiratory distress syndrome, and intraventricular hemorrhage. It acts by stimulating surfactant production.

Study Objectives

To determine the association between the administration of antenatal corticosteroids and the prevalence of respiratory distress syndrome among preterm neonates delivered at Al Mocha Hospital, Taiz Governorate (From January 1, 2023, to June 30, 2025).

Study Design

The research work was designed as a retrospective cohort study.

Subjects and Methods

A questionnaire was constructed for this study, which included information on maternal age, place of residence, gravidity, antenatal care visits, dexamethasone administration rate, gestational age at delivery, mode of delivery, diagnosis of RDS, and other neonatal complications. This information was recorded and analyzed using SPSS software.

Results

Two groups were studied: a study group (50) pregnant women who received dexamethasone upon hospital admission for preterm labor, and a

control group (50) women who did not receive dexamethasone. Their ages were between 20 and 30 years (70% in both groups). There were no statistically significant differences between the two groups in terms of place of residence or number of births. The number of women who did not seek antenatal care in the control group was twice that of the study group, 80% of the women in both groups experienced spontaneous preterm labor. The study group received four consecutive doses of dexamethasone (88%), two participants received two doses, and four participants received three consecutive doses. There were 86% of the women in both groups gave delivered between one and seven days after receiving the last dose of dexamethasone. Gestational age was between 28 and 32 weeks in 31% of the groups, and between 33 and 36 weeks in 69%, and 93% of the women delivered vaginally. The results of this study show that 32% of the neonates of the study group experienced respiratory distress syndrome compared to 54% of the control group. The statistical difference was significant (P=9.92) and 14% of neonates experienced severe RDS in the study group compared to 28% of the control group. All of these cases were admitted to the neonatal intensive care unit. Early neonatal deaths occurred in one child in the study group and two children in the control group, as 2% and 4% respectively.

Conclusion

The difference in the rate of RDS development between women exposed to steroids and the non-exposed group was statistically significant, and the majority completed their course of steroids. There was a stronger protective effect as when comparing infants of the same gestational age category, dexamethasone exposure reduces the odds of developing RDS by 69%. The severity of RDS among women not exposed to steroid was doubled compared with the exposed group. There was no increase in the rate of cesarean section among women delivered preterm. The Apgar score at 5 min. was better among the exposed group compared to the non-exposed group but did not reach to significant perinatal mortality was 30/1000 birth.

keywords: Preterm Infants, Antenatal Corticosteroids, RDS, dexamethasone.

Introduction

Preterm birth, defined as birth between 20 0/7 and 36 6/7 weeks of gestation, is the leading cause of neonatal mortality in non-anomalous fetuses and is the leading cause of long-term neurodevelopmental impairment [1-3]. It is estimated that 15 million preterm births occur globally each year [4-6]. Despite considerable investment in research and strategies to prevent preterm birth, little impact has been made on this goal, and there have not been substantial changes in the rates of preterm birth over the past decade [1,5].

Preterm birth is categorized based on gestational age at delivery. Births occurring between 34 weeks and 36 weeks and 6 days are classified as late preterm births, whereas those between 32 weeks and 33 weeks and 6 days are referred to as moderate preterm births. Births occurring before 32 weeks are categorizeed occurring births. In the United States, the majority of preterm births (approximately 71%) fall under the late preterm category, whereas about 16% occur before 32 weeks [4].

Preterm birth may be spontaneous or medically indicated [7-9]. In spontaneous preterm birth (representing approximately two-thirds of preterm births), painful contractions and progressive cervical effacement and dilation lead to early delivery, whereas in medically indicated preterm birth, the patient is delivered early due to complications of pregnancy or medical conditions in which the risk to the mother or fetus of continuing with the pregnancy is greater than the risk of delivery at that gestational age [9].

This article deals primarily with spontaneous preterm birth. However, there is some overlap between the conditions, and individuals who have one of these subtypes of preterm birth have increased risks both for recurrence and for the other subtype [8,9].

Pathophysiology

Over the past decades, extensive research into preterm labor and preterm delivery has shown that, rather than being in a single condition, preterm labor is a syndrome with multiple distinct causes, all culminating in a common pathway characterized by painful uterine contractions, cervical dilation, and vaginal delivery [10]. Some mechanisms that have been shown to result in preterm labor and preterm delivery include the following:

- Decidual hemorrhage and placental abruption.
- Uterine overdistension such as from multiple gestations or polyhydramnios.
- Cervical insufficiency.
- Uterine distortion.
- Maternal inflammation/stress.
- Uteroplacental insufficiency.
- Intrauterine infection and inflammation.

Bleeding in pregnancy has been shown to be a strong risk factor for preterm labor and delivery. Thrombin is a powerful uterotonic agent. In addition, bleeding greatly increases the risk for degradation and rupture of the membranes. It has been estimated that abruption contributes to at least 20% of spontaneous preterm births.

Uterine overdistension, such as from multiple gestations or polyhydramnios, significantly increases the risk of uterine contractions, cervical dilation, and preterm delivery. The higher prevalence of preterm delivery in multifetal pregnancies is primarily attributed to uterine overdistension. This understanding forms the rationale for multifetal reduction in higher-order multifetal pregnancies.

In some cases of multifetal pregnancies, the early spontaneous delivery of one or more fetuses alleviates uterine overdistension, often halting preterm labor and prolonging gestation for the remaining fetuses—a phenomenon known as delayed interval delivery [11].

A similar mechanism underlies the increased risk of preterm delivery in patients with uterine anomalies, such as a bicornuate or unicornuate uterus, uterus didelphys, or septate uterus [12,13]. These anomalies reduce uterine capacity, leading to earlier overdistension. For example, in patients with a bicornuate uterus, the classic presentation before the advent of ultrasonography was recurrent preterm birth, with gestational ages progressively increasing in subsequent pregnancies due to the "stretching" of the uterus with each pregnancy.

In some pregnancies, cervical insufficiency (previously called “cervical incompetence”) can lead to early painless cervical dilation, often resulting in premature rupture of membranes—particularly during the second trimester—and spontaneous preterm birth [14]. True cervical insufficiency may arise from trauma, such as cervical lacerations sustained during

spontaneous or instrumental vaginal delivery, or from cervical surgery, including extensive cervical cone biopsy or trachelectomy.

Congenital cervical insufficiency can also occur in patients with connective tissue disorders, such as Marfan syndrome and Ehlers-Danlos syndrome. However, the term “cervical insufficiency” is somewhat nonspecific and implies an inherent “weakness” of the cervix [14]. In reality, a significant proportion of patients with spontaneous, painless cervical dilation do not have a structurally weak cervix. Instead, cervical softening and dilation are often caused by intrauterine infection.

In some cases, preterm delivery associated with uterine anomalies may result from uterine overdistension. However, additional mechanisms may contribute, particularly to patients with distorted uterine cavities, such as those with uterine fibroids.

Maternal inflammation and stress have been strongly implicated in spontaneous preterm labor and birth, even in the absence of intrauterine infection.

Uteroplacental insufficiency and the syndrome known as “ischemic placental disease” encompass conditions such as early-onset preeclampsia, severe early-onset fetal growth restriction, and placental abruption. These conditions often overlap, and while they frequently result in medically indicated preterm births, they also carry an increased risk of spontaneous preterm birth.

Intrauterine infection has emerged as an important cause of spontaneous preterm birth and may contribute to at least 25-40% of preterm births [15–17]. The inciting agents are mainly Ureaplasma urealyticum and Mycoplasma hominis. The majority of these infections are subclinical with evidence of infection appearing only on evaluation of the amniotic fluid. Bacterial vaginosis has also been strongly implicated in preterm birth [18].

Finally, the fetus plays a role in the initiation of labor. In a simplistic sense, the fetus recognizes a hostile intrauterine environment and precipitates labor by premature activation of a fetal-placental parturition pathway. The aim of this study was to determine the association between the administration of antenatal corticosteroids and the prevalence of respiratory distress syndrome among preterm neonates delivered at Al Mocha Hospital, Taiz Governorate (From January 1st, 2023, to June 30th, 2025).

Subject and Methods

Study design

The research work was designed as a retrospective cohort study.

Study setting

The study was conducted in Al Mocha Hospital, Taiz Governorate, over a period (From January 1, 2023, to June 30, 2025).

Study population

The target population of this study were all preterm infants born at Mocha Hospital during the study period who met the inclusion criteria

Inclusion criteria

- Pregnant women between 28 weeks of gestation and 36 6/7 weeks.
- Delivered at hospital.
- Received at least one dose of ACS (betamethasone or dexamethasone) prior to delivery.
- Admitted to the NICU within 24 hours of birth.
- Availability of complete data on ACS administration and neonatal outcomes.

Exclusion criteria

- Major congenital anomalies affect respiratory function.
- Decision for non-active resuscitation/palliative care at birth.
- Incomplete critical data.
- Outborn infants transferred >24 hours after birth.
- Gestational age <28 weeks or birth weight <1000g

Sample size

The sample size of this study is 100 women who met our inclusion criteria. It is the available sample size during the study period. They were divided into 2 groups (exposed to steroid n=50) and non-exposed (n=50).

Sampling technique

All women who met the inclusion criteria enrolled in this study in consecutive manner by extracting their data from files. There was no specific technique used for this Study.

Data collection tool

A predesigned validated questionnaire was used for collecting data. The data included were:

i) Sociodemographic data: Age, residency, education, occupation etc.
ii) Obstetric data: Gravidity, parity, history of abortion, obstetric complication-if any-during the current pregnancy.
iii) The onset of developing PTL, Dexamethasone administration, timing and mode of delivery.
iv) Fetal/neonatal outcomes: Live born, SB, birth weight, Apgar score, RDS, ICH and other information.

Technique of data collection

All data of these women who enrolled in this study were extracted from their files and all personal information listed in the questionnaire were filled out. The clinical findings and routine investigation panel were obtained such as complete blood count (CBC) and urinalysis. Additional specific investigation is obtained if indicated by history and examination.

Steroid administration

Dexamethasone 6 mg IM every 12 hours for a total of 4 doses is administered for each woman of the preterm labor according to the hospital protocol.

After admission to obstetric unit (After 34 weeks of gestation) when there is a strong uterine contraction, a tocolytic agent in the form of $MgSO_4$ 4-gram initial dose followed by 1g/hour for 24 hours or till the uterine contractions are subsided. Alternatively, nifedipine tablet 30-20 mg initial dose followed by 10 mg every 6 hours is administered till the uterine contractions are subsided or 24 hours have passed since the last dose of dexamethasone. The women then are allowed for spontaneous delivery unless intervention in form of cesarean section or augmentation of labor process is indicated particularly when the amniotic fluid membranes are ruptured. The decision of mode of delivery is usually taken by the senior in charge.

Variables of study

Dependent variables:

Neonatal outcomes in the terms of:

i) RDS
ii) NE
iii) ICH
iv) Jaundice

Independent variables:

i) Maternal age
ii) Parity
iii) Gestational age
iv) Dexamethasone administration

Ethical consideration

The protocol of this study was approved by the hospital ethics committee. All patient identifiers were removed. Data were anonymized using unique study codes, with the linkage file stored separately and securely.

Data processing

Data analysis was performed using Statistical Package for Social Scientists (SPSS; Version 24.0; Armonk, NY; IBM Corp) software system. The variables were expressed as frequencies and percentages or mean ± standard deviation (SD). Logistic regression was performed to assess which of the variables affecting the dependent variable (RDS). P-value < 0.05 was considered statistically significant.

Table 1.
Distribution of sociodemographic variables among the steroid- exposed and non-exposed groups.

Item	Dexamethasone		Total	P-value
	Exposed	None		
Age (year)				
< 20	**7(14)**	**9(18)**	**16(16)**	
20 – 30	**34(68)**	**36(72)**	**70(70)**	**0.14**
> 30	**9(18)**	**5(10)**	**14(14)**	
Residence				
Urban	**11(22)**	**12(24)**	**23(23)**	
Rural	**39(78)**	**38(76)**	**77(77)**	**0.31**
Parity				
Primipara	**19(38)**	**17(34)**	**36(36)**	
Multipara	**28(56)**	**29(58)**	**67(67)**	**0.67**
Grand multipara	**3(6)**	**4(8)**	**7(7)**	
Subtotal	**50(50)**	**50(50)**	**100(100)**	

Results

100 pregnant women were subjected to analysis who were complicated by preterm birth. They were divided into 2 groups (exposed to steroid n=50)

and non-exposed (n=50). Table 1 summarizes the sociodemographic variables among the two groups.

There was no significant difference between exposed and non-exposed groups in relation to age, residence place, and parity (P>0.05).

Antenatal Care

Figures 1, and 2 show that 18% of women in the exposed group had no antenatal care, compared to double of this rate in non-exposed group.

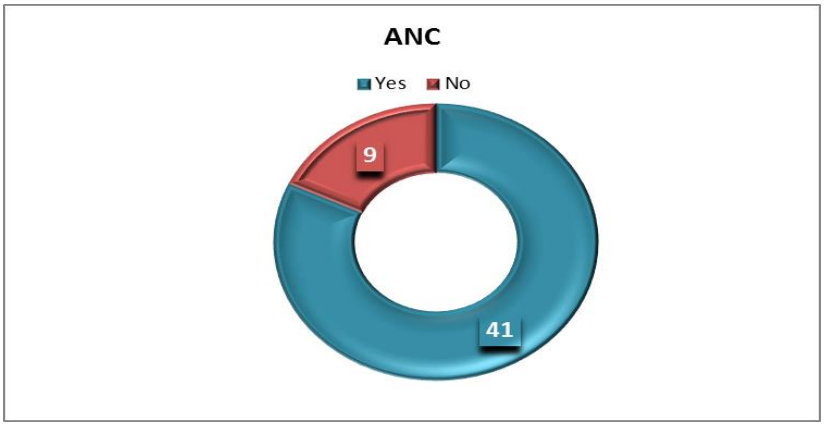

Figure 1: Antenatal care attendance of the exposed group.

Type of preterm birth among the two groups

By far, the commonest type of preterm labor was spontaneous preterm birth (84% and 76%) among the exposed and non-exposed group respectively. 13% of the two groups had induced labor for the two various indications. Only 7% had ended preterm birth by cesarean section (**Table 2**).

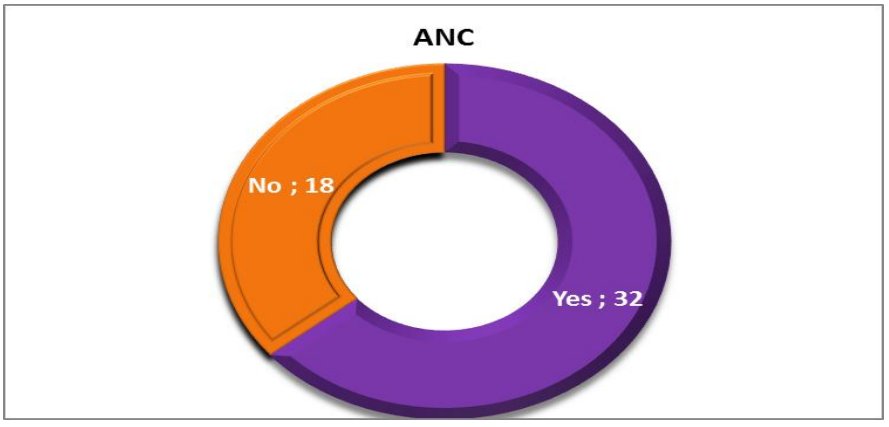

Figure 2: Antenatal care attendance of the non-exposed group.

Table 2.
Type of preterm birth among the two groups.

Preterm birth	Dexamethasone		Total	P-value
	Exposed	None		
Spontaneous	42(84)	38(76)	80(80.0)	
Induced	6(12)	7(14)	13(13)	0.05
Cesarean section	2(4)	5(10)	7(7.0)	
Subtotal	50(50)	50(50)	100(100)	

Regarding the exposed group, all women received dexamethasone, only 2 women had a dose, 4 women received 3 doses and the remaining 44 women (88%) received 4 doses and completed their course (88%) and 86% delivered after 24 hours in addition to one case delivered > 7 days after receiving the last dose of dexamethasone (Table 2).

Table 3.
Management-related variables of the exposed group.

Dexamethasone Dosing Parameters	Count	%	P-value
Corticosteroid type			
Dexamethasone	50	100	
Total dose received			
Two doses	2	4.0	0.00
Three doses	4	8.0	
Four doses	44	88	
Course completeness			
Incomplete	6	12.0	0.00
Complete	44	88	
Last dose-delivery			
< 24 (hour)	6	12.0	0.01
24 h 7 days	43	86	
> 7 days	1	2.0	

Birth-related characteristics

Table 4 shows that 31% of the total sample had gestational age between 28 to 32 weeks, and 69% their gestational age between 33-36 weeks the majority of women delivered vaginally with no statistically significant difference in the mode of delivery observed between the two groups (P=0.44).

Neonatal outcome

Table 5 summarizes the neonatal outcome according to the two groups. Those with birth weight < 2500 represented 84% of the exposed group

compared to 82% of the non-exposed group (P=0.56). There was no significant difference between the two

Table 4.
Birth-related characteristics.

Item	Dexamethasone		Total	P-value
	Exposed	None		
Gestational age (week)				
28 – 32	14(28)	17(34)	31(31)	0.51
33 – 36	36(62)	33(66)	69(69)	
Mode of delivery				
Cesarean section	2(4)	5(10)	7(7)	0.44
Vaginal delivery	48(96)	45(90)	93(93)	
Subtotal	50(50)	50(50)	100(100)	

Table 5.
Distribution of neonatal outcome among the two groups.

Item	Dexamethasone		Total	P-value
	Exposed	None		
Birth weight (g)				
< 1500	13(26)	12(24)	25(25)	0.56
1500 – 2000	13(26)	17(34)	30(30)	
2100 – 2500	16(32)	12(24)	28(28)	
> 2500	8(16)	9(18)	17(17)	
Infant sex				
Male	24(48)	22(44)	46(46)	
Female	26(52)	28(56)	54(54)	0.52
Apgar score at 5 min.				
1 – 5	14(28)	21(42)	35(35)	
6 – 10	36(72)	29(58)	65(65)	0.144
NICU admission				
Yes	16(32)	23(46)	39(39)	
No	34(68)	27(54)	61(61)	0.15
NICU stay duration				
1 – 7 days	7(14)	10(20)	17 (17)	
8 – 14 days	7(14)	9(18)	16(16)	0.6
> 14	2(4)	4(8)	6(6)	

groups in relation to infant sex, and NICU stay duration. There was no significant difference between the two groups regarding Apgar score at 5 min. despite those exposed group had better Apgar score compared to the non-exposed group.

Neonatal complications

The distribution of neonatal complications among the two groups is illustrated in Table 6. The calculated Odds Ratio (OR) is 0.4009. This indicates that the odds of a neonate developing RDS in the dexamethasone-exposed group are approximately 60% lower than the odds of a neonate developing RDS in the unexposed control group.

Table 6.

Distribution of neonatal complications among the two groups.

Item	Dexamethasone		Total	P-value
	Exposed	None		
RDS present				
Yes	**16(32)**	**27(54)**	**43(43)**	**0.02**
No	**34(68)**	**23(46)**	**57(57)**	
Severity of RDS				
Yes	**7(14)**	**14(28)**	**21(21)**	**0.08**
No	**43(86)**	**36(72)**	**79(79)**	
Respiratory support (O_2)				
Yes	**16 (32)**	**26(52)**	**42(42)**	**0.04**
No	**34(68)**	**24(48)**	**58(58)**	
Duration of respiratory support (day)				
1	**4(8)**	**3(6)**	**7(7)**	**0.12**
2	**1(2)**	**7(14)**	**8(8)**	
3	**4(8)**	**7(14)**	**11(11)**	
4	**5(10)**	**5(10)**	**10(10)**	
> 4	**2(4)**	**4(8)**	**6(6)**	
Outcome				
Alive	**49(98)**	**48(96)**	**97(97)**	**0.75**
ENND	**1(2)**	**2(4)**	**3(3)**	

Table 7.

Comparison between the two groups regarding RDS development.

Item	Dexamethasone		Total
	Exposed	None	
RDS present			
Yes	**16(32)**	**27(54)**	**43(43)**
No	**34(68)**	**23(46)**	**57(57)**

OR=0.4009, 95% Confidence Interval for the OR= 0.1776 to 0.9046.

The 95% Confidence Interval for the OR ranges from 0.1776 to 0.9046. Because the entire confidence interval sits below the null value of 1.0 (OR < 1.0), the finding is statistically significant. We can state with 95% confidence that the true population odds ratio lies within this interval, demonstrating a consistent protective effect. Absolute Risk Reduction (ARR =22.00%): The absolute incidence of RDS dropped from 54% in the control group to 32% in the treatment group (Table 7). This implies an absolute benefit of 22% across the sample. From a clinical perspective, this translates to a Number Needed to Treat (NNT) of approximately 5, meaning that for every 5 pregnant women in preterm labor treated with dexamethasone, 1 case of neonatal RDS is directly prevented.

Relative Risk Reduction, RRR=40.74%; dexamethasone administration successfully avoided 40.74% of the RDS cases that would have otherwise occurred had the intervention been withheld.

Adjustment of RDS occurrence according to gestational age

Table 8 shows the adjustment of RDS occurrence according to gestational age. It can be found that:

Stratum 1: Early Gestational Age (28–32 Weeks)

Exposed: 14 total women of them 10 neonates developed RDS (71.4 %), 4 did not. Non-exposed: 17 total women all 17 neonates developed RDS (100 %), 0 did not.

Table 8.

Adjustment of RDS occurrence according to gestational age.

Total	Exposed	Non-exposed	P-value
Gestational age			
28 – 32	**14**	**17**	**0.04**
RDS	**10(71.4)**	**17(100)**	
No RDS	**4(28.6)**	**0(0.0)**	
Gestational age			
33 – 36	**36**	**33**	**0.258**
RDS	**6(16.7)**	**10(30.3)**	
No RDS	**30(83.3)**	**23(69.7)**	

Crude OR 0.40, 95% CI 0.18-0.90, P=0.027 Adjusted OR MH: 0.31, 95% CI 0.10-0.94, P=0.043

Stratum 2: Late Gestational Age (33–36 Weeks)

Exposed: 36 total women of them 6 neonates developed RDS (16.7 %), 30 did not. Non-exposed: 33 total women of them 10 neonates developed RDS (30.3 %), 23 did not. Adjusted Statistical Results (Adjusted for Gestational Age) Adjusted Odds Ratio: 0.31, 95% Confidence Interval {95% CI}): [0.10, 0.94] Cochran-Mantel-Haenszel P-value: 0.0434 Statistically Significant, $P < 0.05$).

Relationship between the development of RDS and neonatal variables among the exposed group

Table 9 shows the relationship between neonatal variables and the occurrence of RDS among neonates who delivered to women exposed to steroid. It is detected that half of the neonates experienced RDS had birth weight >1500g with no significant difference with non-exposed group (P=0.31). No significant difference in relation to the infant's sex (P=0.12).

Table 9.

Relationship between the development of RDS and neonatal variables among the exposed group.

Neonatal outcome	RDS development		Total	P-value
	Yes	No		
Birth weight (g)				
< 1500	**8(16)**	**5(10)**	**13(26)**	**0.31**
1500 – 2000	**5(10)**	**8(16)**	**13(26)**	
2100 – 2500	**3(6)**	**13(26)**	**16(32)**	
> 2500	**0(0)**	**8(16)**	**8(16)**	
Infant sex				
Male	**7(14)**	**17(34)**	**24(48)**	**0.12**
Female	**9(18)**	**17(34)**	**26(52)**	
Apgar score at 5 min.				
1 – 5	**9(18)**	**5(10)**	**14(28)**	**0.00**
6 – 10	**7(14)**	**29(58)**	**36(72)**	
NICU admission				
Yes	**16(32)**	**0(0)**	**16(32)**	**0.05**
No	**0(0)**	**34(68)**	**34(68)**	
NICU stay duration				
1 – 7 days	**7(14)**	**16(32)**	**23(46)**	**0.41**
8 – 14 days	**7(14)**	**17(34)**	**24(48)**	
> 14	**2(4)**	**1(2)**	**3(6)**	
	16(32)	**34(68)**	**50(100)**	

Those neonates affected by RDS had 9 out of 16 (56.3%) low Apgar score compared to 5 out of 27 (18.5%) of the non-exposed group with significant difference (P=0.00). All affected neonates with RDS had been admitted to NICU.

Discussion

This study aimed to highlight the antenatal corticosteroids to women who delivered preterm and its effectiveness in reducing the rate of RDS. In the present study, maternal sociodemographic and obstetric characteristics were generally comparable between the dexamethasone-exposed and non-exposed groups. Most women in both groups were aged 20–30 years, multiparous, and presented with spontaneous preterm labor. The similarity of baseline characteristics between the two groups minimizes the effect of confounding variables and strengthens the validity of the comparison of neonatal outcomes. The majority of women in the dexamethasone group (88%) received the complete recommended course of four doses, and 86% delivered within 1–7 days after the last dose. This finding is important because the maximum benefit of antenatal corticosteroids is achieved when delivery occurs between 24 hours and 7 days after administration. WHO guidelines and several studies have demonstrated that this interval is associated with the greatest reduction in neonatal respiratory morbidity and mortality among preterm infants [19,20]. Regarding gestational age, approximately one-third of infants in both groups were delivered between 28 and 32 weeks, while about two-thirds were delivered between 33 and 36 weeks. These findings are comparable to studies evaluating antenatal corticosteroid therapy among women with threatened preterm labor, where most deliveries occurred in the moderate-to-late preterm period [21].

Birth weight distribution showed a lower proportion of very low birth weight infants (<1500g) in the dexamethasone group (16%) compared with the control group (24%). Although the difference was not statistically significant, it may partly explain the better neonatal outcomes observed among exposed infants. Similar findings were reported by Shittu et al., who found improved neonatal outcomes among corticosteroid-exposed preterm infants despite comparable gestational ages at delivery [21]. Apgar scores were better among neonates exposed to dexamethasone, with only 28% having scores between 1 and 5 compared with 42% in the non-exposed

group. Improved Apgar scores among corticosteroid-exposed infants have been reported in previous studies and are believed to result from enhanced pulmonary maturity and improved adaptation to extrauterine life [22]. One of the most important findings of the current study was the reduction in NICU admission among infants exposed to dexamethasone (32%) compared with controls (46%). Although NICU admission remained high because all infants were preterm, the reduction observed in the exposed group is consistent with evidence demonstrating that antenatal corticosteroids decrease neonatal complications requiring intensive care management [19,23]. The incidence of respiratory distress syndrome (RDS) was markedly lower among exposed infants (32%) than among non-exposed infants (54%). This finding agrees with the established biological effect of dexamethasone in accelerating fetal lung maturation and surfactant production. WHO evidence reviews and multiple randomized trials have consistently shown significant reductions in RDS among preterm neonates exposed to antenatal corticosteroids [19, 20, 23]. Similar reductions were reported in recent studies evaluating dexamethasone administration before anticipated preterm birth [21]. Antenatal corticosteroids (ACS) are routinely administered in patients at risk of preterm birth (PTB) before 34 weeks of gestation worldwide to reduce the risk of neonatal morbidity and mortality.1 This practice has been supported by consistent evidence demonstrating lower rates of adverse neonatal outcomes such as respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), and mortality among those exposed [24]. Another study reported that the short-term application of steroids is known to favourably influence lung development and avert neurological and gastrointestinal complications [25].

Furthermore, severe RDS occurred in only 14% of the dexamethasone group compared with 28% of the control group, representing a clinical halving of severe cases that did not reach statistical significance (P=0.08). This finding indicates that dexamethasone not only reduces the occurrence of RDS but also decreases its severity. Comparable observations have been reported in systematic reviews showing that antenatal corticosteroid therapy significantly reduces severe respiratory morbidity among preterm infants [19,23].

The need for respiratory support was also lower in the exposed group (32%) compared with the non-exposed group (52%). This result is consistent with studies demonstrating reduced requirements for oxygen therapy, Continuous Positive Airway Pressure (CPAP), and mechanical ventilation among infants exposed to antenatal corticosteroids before preterm delivery [21,23].

Early neonatal death occurred in only one infant in the dexamethasone group compared with two infants in the control group. Although the number of deaths was small and the difference was not statistically significant, the trend favors dexamethasone exposure. Previous meta-analyses have reported reductions in neonatal mortality following antenatal corticosteroid administration among women at risk of preterm birth [23,26-28].

Study limitations

This study has many limitations; the main one is the small sample size along with the small number of women who exposed to dexamethasone. Thus, a large cohort study is required to validate our data.

Conclusions

The difference in the rate of RDS development between women exposed to steroid and non-exposed group was statistically significant and the majority completed their course of steroids.

There was a stronger protective effect as when comparing infants of the same gestational age category, dexamethasone exposure reduces the odds of developing RDS by 69%.

The severity of RDS among women not exposed to steroid was doubled compared with the exposed group.

There was no increase in the rate of cesarean section among women delivered preterm.

The Apgar score at 5 min. was better among the exposed group compared to the non-exposed group but did not reach to significant perinatal mortality was 30/1000. birth.

Recommendations

Further studied are required to address corticosteroid administration for women with late preterm labor with large and powered sample size to confirm these results and assess potential long-term implications.

Antenatal corticosteroids use should be encouraged among all women who developed signs and symptoms of preterm labor

From 34 to 36 weeks gestation, antenatal corticosteroids should be considered, based on absolute harm, and benefits specific to the gestational week; this consideration may be facilitated by the clinician decision support tool.

Use a single course of antenatal corticosteroids in women at risk of preterm birth when birth is planned or expected within the next seven days even if birth is likely within 24 hours.

Antenatal corticosteroids are most effective in reducing RDS in pregnancies that deliver 24 hours after and up to 7 days after administration of the second dose of antenatal corticosteroids

There is no need for repeat of steroid dose for women with preterm labor till new recommendation is established.

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