ORIGINAL RESEARCH

Antenatal corticosteroids in women at risk of late preterm delivery

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ABSTRACT

Background: One of the primary causes of early neonatal morbidity and mortality in preterm births is respiratory morbidity, which includes respiratory distress syndrome. The present study was conducted to assess the effect of antenatal corticosteroids in women at risk of late preterm delivery. **Materials & Methods:** 90women with a singleton gestation at high risk for late preterm delivery were divided into 2 groups of 45 each. Group I patients were given injections of 12 mg betamethasone and group II patients were given placebo 24 hours apart. In this study, we assessed neonatal and maternal outcome. **Results:** The indication for trial entry was preterm labor with intact membranes in 20 and 26 patients in group I and II and ruptured membranes in 25 and 19 in group I and II respectively. Gestational age at trial entry was ≤ 34 weeks seen in 10 and 12, 35 weeks in 19 and 18 and ≥36 weeks in 16 and 15 in group I and II respectively. The difference was significant (P< 0.05). Neonatal outcomeswas necrotizing enterocolitis in 2 and 1, hypoglycemia in 9 and 5, neonatal death in 0 and 2, proven neonatal sepsis in 4 and 2 and intraventricular hemorrhage in 2 and 1 patients in group I and group II respectively. Maternal outcomes was chorioamnionitis in 4 and 5 patients, cesarean delivery in 25 and 30, and postpartum endometritis in 2 and 3 patients respectively. The difference was significant (P< 0.05). **Conclusion:** When betamethasone was given to women who were at risk of a late preterm delivery, the rate of newborn respiratory morbidity was significantly reduced.

Key words: betamethasone, preterm, women

INTRODUCTION

One of the primary causes of early neonatal morbidity and mortality in preterm births is respiratory morbidity, which includes respiratory distress syndrome.[1] By improving the synthesis of surfactants, the administration of prenatal corticosteroids has been proven to minimize respiratory morbidities. It has also been found to lower the incidence of intraventricular hemorrhage and necrotizing enterocolitis in preterm newborns.[2] For women at risk of preterm birth, both betamethasone and dexamethasone are used as antenatal corticosteroid therapy; however, it is yet unknown which of the two is more advantageous for the mother and the fetus. It is crucial to investigate hemodynamic changes in fetal and uteroplacental circulation following pregnancy since some research have also shown that antenatal betamethasone reduced fetal body and breathing movements and varied fetal heart rate.[3]

For pregnancies at risk for an early preterm delivery, antenatal corticosteroids have been utilized extensively in practice. Due to a lack of evidence supporting use beyond 34 weeks and the fact that the neonatal survival of late preterm newborns is nearly equal to that of term infants, their use has historically been limited to pregnancies at less than 34 weeks gestation.[4] Eight percent of all deliveries take place during the late preterm period, which is defined as 34 weeks 0 days through 36 weeks 6 days. Although overall survival in the late preterm period is within 1% of that of term neonates, more recent research has shown that late preterm newborns have higher morbidities and long-term problems.[5]The present study was conducted to assess the effect of antenatal corticosteroids for women at risk of late preterm delivery.

MATERIALS & METHODS

The present study consisted of 90women with a singleton gestation at high risk for late preterm delivery. All gave their written consent to participate in the study.

Data such as name, age, etc. was recorded. Patients were divided into 2 groups of 45 each. Group I

patients were given injections of 12 mg betamethasone and group II patients were given placebo 24 hours apart. Parameters such as indication for trial entry, gestational age at trial entry, neonatal and maternal outcomes were recorded. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I: Baseline characteristics

Parameters	Variables	Group I	Group II	P value
Indication for trial entry	Preterm labor with intact membranes	20	26	0.38
	Ruptured membranes	25	19	
Gestational age at trial entry	≤ 34 weeks	10	12	0.64
	35 weeks	19	18	
	≥36 weeks	16	15	

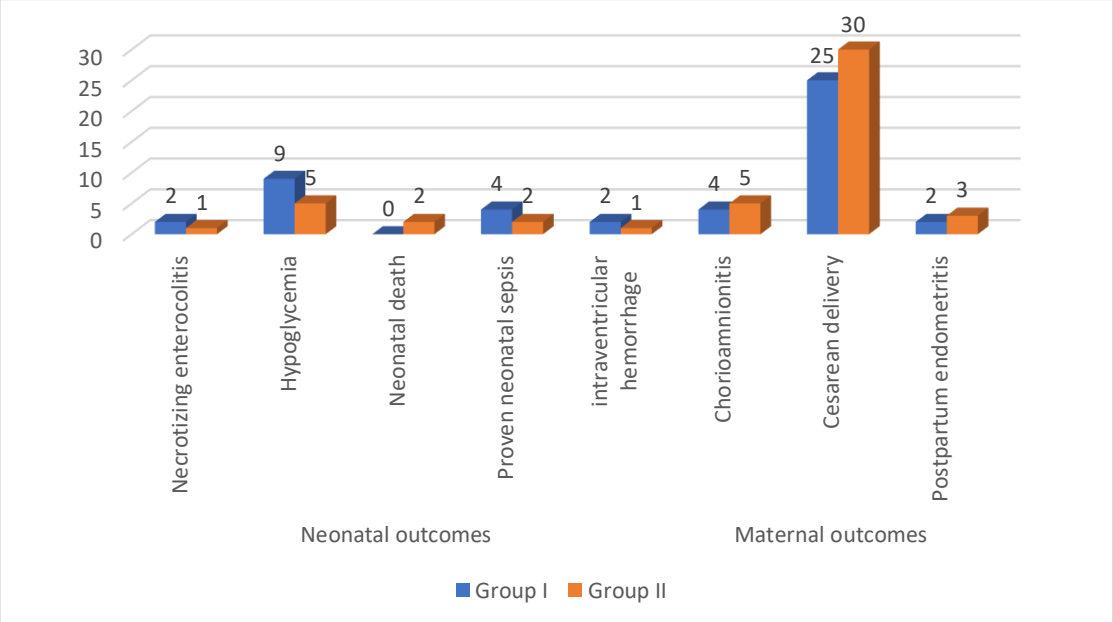
Table I shows that the indication for trial entry was preterm labor with intact membranes in 20 and 26 patients in group I and II and ruptured membranes in 25 and 19 in group I and II respectively. Gestational age at trial entry was ≤ 34 weeks seen in 10 and 12, 35 weeks in 19 and 18 and ≥36 weeks in 16 and 15 in group I and II respectively. The difference was significant (P< 0.05).

Table II: Neonatal and maternal outcomes

Parameters	Variables	Group I	Group II	P value
Neonatal outcomes	Necrotizing enterocolitis	2	1	0.05
	Hypoglycemia	9	5	
	Neonatal death	0	2	
	Proven neonatal sepsis	4	2	
	intraventricular hemorrhage	2	1	
Maternal outcomes	Chorioamnionitis	4	5	0.04
	Cesarean delivery	25	30	
	Postpartum endometritis	2	3	

Table II, graph I show that neonatal outcomes was necrotizing enterocolitis in 2 and 1, hypoglycemia in 9 and 5, neonatal death in 0 and 2, proven neonatal sepsis in 4 and 2 and intraventricular hemorrhage in 2 and 1 patients in group I and group IIrespectively. Maternal outcomes waschorioamnionitis in 4 and 5 patients, cesarean delivery in 25 and 30, and postpartum endometritis in 2 and 3 patients respectively. The difference was significant (P< 0.05).

Graph I: Neonatal and maternal outcomes



DISCUSSION

One of the most crucial prenatal treatments available to enhance neonatal outcomes is the prescription of corticosteroids before to an expected preterm birth. For pregnant women between 24 0/7 weeks and 33 6/7 weeks of gestation who are at risk of premature

delivery within 7 days, including those with ruptured membranes and multiple gestations, a single course of corticosteroids is advised.[6] Regardless of the number of fetuses or the state of membrane rupture, it may also be taken into consideration for pregnant women beginning at 23 0/7 weeks of gestation who are at danger of premature delivery within 7 days, depending on a family's decision regarding resuscitation.[7]The present study was conducted to assess the effect of antenatal corticosteroids for women at risk of late preterm delivery.

We found that the indication for trial entry was preterm labor with intact membranes in 20 and 26 patients in group I and II and ruptured membranes in 25 and 19 in group I and II respectively. Gestational age at trial entry was ≤ 34 weeks seen in 10 and 12, 35 weeks in 19 and 18 and ≥36 weeks in 16 and 15 in group I and II respectively. 26 pregnant women with singleton pregnancies who were deemed at risk for preterm birth at baseline had their umbilical and fetal cerebral artery flow velocity waveforms examined by Y Chitrit et al[8] in order to determine the effects of maternal dexamethasone administration. Fetoplacental vascular resistance was found to be normal in all pregnancies, and on the fourth day after maternal dexamethasone injection, there was a brief, notable, and inexplicable drop in fetal middle cerebral artery impedance in healthy fetuses.

We observed that neonatal outcomeswas necrotizing enterocolitis in 2 and 1, hypoglycemia in 9 and 5, neonatal death in 0 and 2, proven neonatal sepsis in 4 and 2 and intraventricular hemorrhage in 2 and 1 patients in group I and group II respectively. Maternal outcomes was chorioamnionitis in 4 and 5 patients, cesarean delivery in 25 and 30, and postpartum endometritis in 2 and 3 patients respectively. Cohlen et al[9]investigated whether there is evidence of circulatory changes in fetal, placental or uterine arteries, consistent with hypoxemia. Eighteen women at risk for preterm delivery received betamethasone to enhance fetal lung maturation. Doppler studies were performed before treatment, and 24 and 72 h after the second dose of betamethasone. Blood flow velocity waveforms were obtained from both uterine arteries, umbilical arteries, fetal descending aorta, fetal renal artery, and fetal cerebral arteries. No significant changes occurred in the pulsatility index of any of these blood vessels, suggesting that the transient reduction in fetal heart rate variation and fetal body and breathing movements following maternal betamethasone administration is not mediated through fetal hypoxemia.

In a study of 32 singleton pregnancies complicated by fetal growth restriction with absent end diastolic flow in UA, Alexandre M. Nozaki et al[10] investigated the effects of betamethasone administration on umbilical artery (UA), middle cerebral artery (MCA), and ductus venosus (DV) doppler flow. They discovered that there was a decrease in umbilical artery and ductus venosus pulsatility indices 24 hours after betamethasone administration, which persisted for 48 hours.

The limitation of the study is the small sample size.

CONCLUSION

Authors found that when betamethasone was given to women who were at risk of a late preterm delivery, the rate of newborn respiratory morbidity was significantly reduced.

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