Effect of intramuscular administration of Dexamethasone on the duration of induction of labor in full term pregnancy

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Abstract: To evaluate the effect of dexamethasone administration on labor duration. In this controlled trial 120 women (pG-para1-para2) with a full-term pregnancy and a Bishop score of 7 or greater were randomly assigned to receive a single 8-mg dose of dexamethasone or placebo 6 hours before initiation of labor induction. The administration of dexamethasone was found to shorten labor duration.
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Key words: Dexamethasone, induction, labor duration and full term pregnancy.

1. Introduction:

The process of childbirth starts from the axis of the hypothalamus, the pituitary gland, and the adrenal glands. Steroid substances produced in the adrenal glands of the human fetus affect the placenta and the membranes and transform the myometrium from the static to the contractile state (1). The placenta may play a role in this process because it produces a lot of CRH (Corticotropinreleasing hormone). The adrenal glands of the fetus do not produce a considerable amount of cortisol until the third trimester. During the last weeks of pregnancy, the cortisol and DHEA –S (Dehydroepiandrosterone sulfate) contents of the fetus rise and this leads to an increase in maternal estrogens, a particularly sterol (1). Placental CRH is not under the influence of negative feedback from cortisol. The concentration of CRH in the fetus rises during the last 12 weeks of pregnancy. This results in modification of the contractility of the uterus (1), stimulation of the membranes to produce more prostaglandins (2), stimulation to produce Csteroids from placental adrenaline (3), and increase in the estrogen content (4). This will disturb the ratio of estrogen to progesterone and will cause expression of contractile proteins. In fact, the increase in CRH near the end of pregnancy confirms the presence of a placental-fetal clock (1).

Although administrating corticosteroids is a suggested method to shorten labor duration, the role of these agents in the process of labor is not well understood [2-6]. Several animal studies have shown the importance of corticosteroid secretion by the fetal adrenal glands on the beginning of labor[2-7], and infusing glucocorticoids in the lamb fetus was also shown to induce preterm labor [7]. These findings have led to the hypothesis that corticosteroids also had an effect on the labor of women [2,3]. Different studies have shown the paracrine and autocrine effects of corticosteroids on the human uterus, and receptors for these agents have been detected on the human amniotic membrane [2,8,9]. **Kalantaridou et al**. [10] have suggested that the corticotrophin-releasing hormone (CRH), which has been identified in various organ systems, including the female reproductive system, is the principal regulator of the hypothalamic–pituitary–adrenal axis. Circulating placental CRH is responsible for the physiologic hypercortisolism of the latter half of pregnancy and plays a role in the onset of labor. **O'Sullivan et al.** [11] reported that a prolonged gestation is more likely to occur when the fetus has congenital adrenal hyperplasia caused by 21-hydroxylase deficiency, which may be due to an impaired cortisol production.

All of these studies show the probable effects of corticosteroids on the labor process. Corticosteroids have been administered intravenously, intramuscularly, and by extra-amniotic infusion in various clinical trials [12-15].

Objective: To evaluate the effect of intramuscular dexamethasone administration on duration of induction of labor in full term pregnancy.

2. Patient and Method:

This prospective clinical interventional randomized case-controlled trial was conducted at EL Galaa Teaching Hospital during the period from April 2016 to January 2017.

The inclusion criteria in the study are:

Singleton pregnancy, Age: 18-30, Sure, reliable dates, BMI: 19.8-31.0, Nulliparous, Para 1 and Para 2,

Favorable cervix with Bishop score of 7 or greater, Longitudinal lie, Cephalic presentation (Vertex), Good pelvis.

The exclusion criteria in the study are:

Grand Mutli Para, Diabetes mellitus, Known contraindication or hypersensitivity to Dexamethasone, Twins, Non cephalic presentation, Unfavorable pelvis, The intake of any drugs besides iron or other, ordinary nutritional Supplements, active labor, Premature rupture of membranes, Intrauterine growth restriction, fetal distress, Significant vaginal bleeding, Any uterine anomaly or history of C.S.

Plan:

It included 120 participants whom are admitted for labor induction at ELGalaa Teaching Hospital.

The participants will be randomly assigned by computer list into Group I (Dexamethasone group) N=60 and Group II (Control group) N=60.

The participants of Group I (nulliparous = 20, Para1 =20 and Para2 = 20) will receive a prefilled syringe with two milliliters (8mg) of dexamethasone intra-muscular, and the participants of Group II will not receive dexamethasone or any other cervical ripening agent.

After six hours of the initial dose, the labor induction will start via Oxytocin using the following protocol:

A. Initial dose of oxytocin..... 1 to 2 mIU/min.
B. Increase interval..... 30 minutes.
C. Dosage increment..... 1 to 2 mIU.
D. Usual dose for good labour….. 8 to12 mIU/min.
E. Maximum dose..... 30 mIU/min.

No cervical ripping agent will be used for induction of labor in either group.

After approval of health committee in EL Galaa Teaching Hospital, a verbal consent was obtained from each candidate after explanation of the procedure in details.

3. Results:

The studied groups described as:

Study-0: Nulliparous cases recevied a single intramuscular administration of dexamethasone (N=20).

Study-1: Para-1 cases recevied a single intramuscular administration of dexamethasone (N=20).

Study-2: Para-2 cases recevied a single intramuscular administration of dexamethasone (N=20).

Control-0: Nulliparous cases did not recevie intramuscular administration of dexamethasone (N=20).

Control-1: Para-1 cases did not recevie intramuscular administration of dexamethasone (N=20).

Control-2: Para-2 cases did not recevie intramuscular administration of dexamethasone (N=20).

Table (1) show that: **Induction-delivery intervals** were significantly shorteramong study groups thanamong control groups at different parities.

Table (2) and show that: **Induction-delivery intervals** were significantly shortestamong para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups.

Table (3) show that: **Active phase durations** were significantly shorteramong study groups thanamongcontrol groups at different parities.

Table (4) show that: **Active phase durations** were significantly shortestamong para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups.

Table (5) shows that: **second stage durations** were significantly shorteramong study groups thanamong control groups at different parities.

Table (6) show that: **second stage durations** were significantly shortestamong para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups.

Table (1): Comparison between intervention groups regarding induction-delivery interval (hours)

Group	Measures	Study	Control	^P
Nullipar-ous	**Mean±SD**	3.9±0.5	4.9±0.4	**<0.001***
	Range	2.8–4.7	4.3–5.9	
Para-1	**Mean±SD**	3.2±1.2	4.0±1.0	**0.032***
	Range	1.4–4.9	1.9–5.6	
Para-2	**Mean±SD**	2.5±0.8	3.1±0.8	**0.017***
	Range	0.5–3.8	2.0–5.6	

Value of using cortisone		
Interval reduction	**Mean±SE**	**95% CI**
Nulliparous	1.0±0.1	0.7–1.3
Para-1	0.8±0.4	0.1–1.5
Para-2	0.6±0.3	0.1–1.1

^Independent t-test, *Significant, **CI:** Confidence interval

Table (2): Comparison between parity groups regarding induction-delivery interval (hours)

Group	Measures	Nulli	Para-1	Para-2	^P
Study	**Mean±SD**	3.9±0.5	3.2±1.2	2.5±0.8	**<0.001***
	Range	2.8–4.7	1.4–4.9	0.5–3.8	
	HG	A	B	c	
Control	**Mean±SD**	4.9±0.4	4.0±1.0	3.1±0.8	**<0.001***
	Range	4.3–5.9	1.9–5.6	2.0–5.6	
	HG	A	B	C	

^ANOVA test with post hoc Tukey test, *Significant, HG: Homogenous groups have the same letter

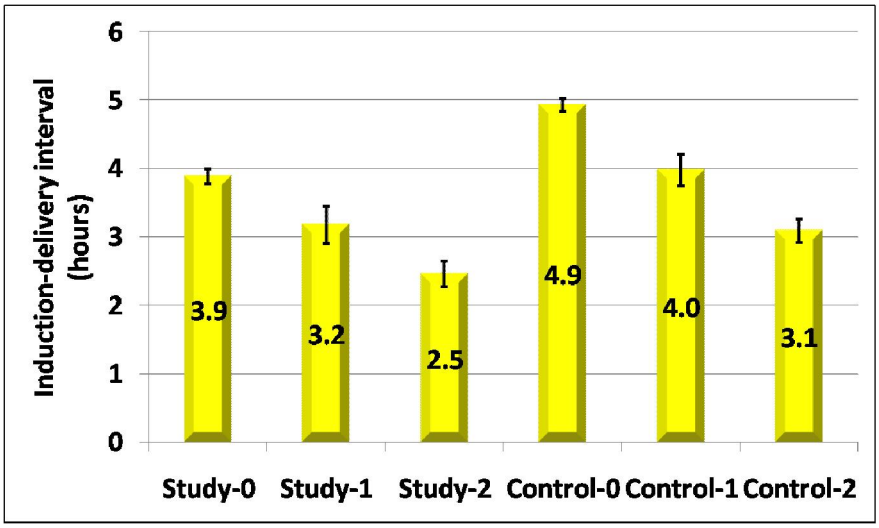

Figure (1): Comparison between study groups regarding induction-delivery interval

Table (3): Comparison between intervention groups regarding active phase duration (hours)

Group	Measures	Study	Control	^P
Nullipar-ous	**Mean±SD**	3.5±0.4	4.4±0.5	**<0.001***
	Range	2.5–4.2	3.7–5.7	
Para-1	**Mean±SD**	2.8±1.0	3.6±1.0	**0.020***
	Range	1.0–4.2	1.7–5.5	
Para-2	**Mean±SD**	2.2±0.7	2.8±0.8	**0.011***
	Range	0.7–3.1	1.8–5.4	
Value of using cortisone				
Interval reduction		**Mean±SE**	**95% CI**	
Nulliparous		1.0±0.2	0.7–1.3	
Para-1		0.8±0.3	0.1–1.4	
Para-2		0.6±0.2	0.2–1.1	

^Independent t-test, *Significant, **CI:** Confidence interval

Table (4): Comparison between parity groups regarding active phase duration (hours)

Group	Measures	Nulli	Para-1	Para-2	^P
Study	**Mean±SD**	3.5±0.4	2.8±1.0	2.2±0.7	**<0.001***
	Range	2.5–4.2	1.0–4.2	0.7–3.1	
	HG	A	B	c	
Control	**Mean±SD**	4.4±0.5	3.6±1.0	2.8±0.8	**<0.001***
	Range	3.7–5.7	1.7–5.5	1.8–5.4	
	HG	A	B	c	

^ANOVA test with post hoc Tukey test, *Significant, HG: Homogenous groups have the same letter

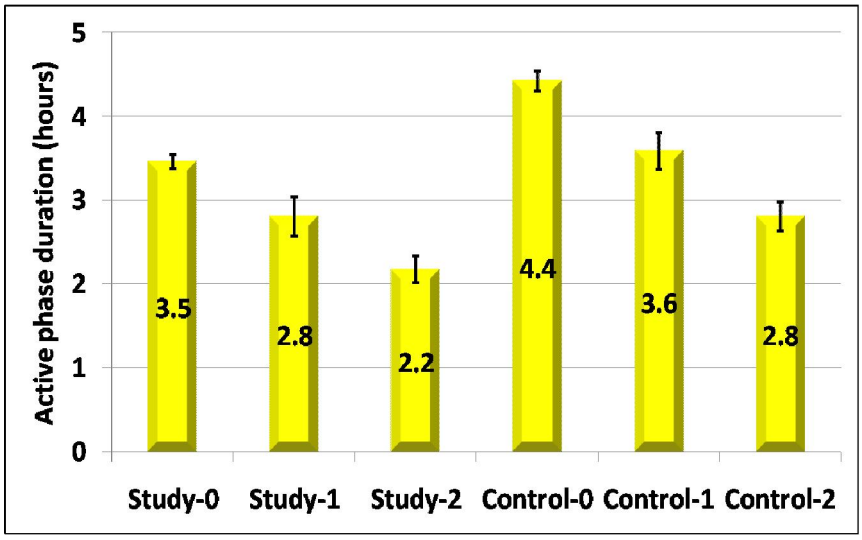

Figure (2): Comparison between study groups regarding active phase duratio

Table (5): Comparison between intervention groups regarding second stage duration (minutes)

Group	Measures	Study	Control	^P
Nullipar-ous	**Mean±SD**	26.8±1.5	31.2±2.8	**<0.001***
	Range	23.0–29.0	28.0–37.0	
Para-1	**Mean±SD**	23.8±4.4	27.3±4.3	**0.014***
	Range	15.0–30.0	20.0–36.0	
Para-2	**Mean±SD**	21.2±3.2	24.3±3.5	**0.006***
	Range	13.0–25.0	21.0–37.0	

Value of using cortisone		
Interval reduction	**Mean±SE**	**95% CI**
Nulliparous	4.4±0.7	3.0–5.8
Para-1	3.5±1.4	0.7–6.3
Para-2	3.1±1.1	1.0–5.2

^Independent t-test, *Significant, **CI:** Confidence interval

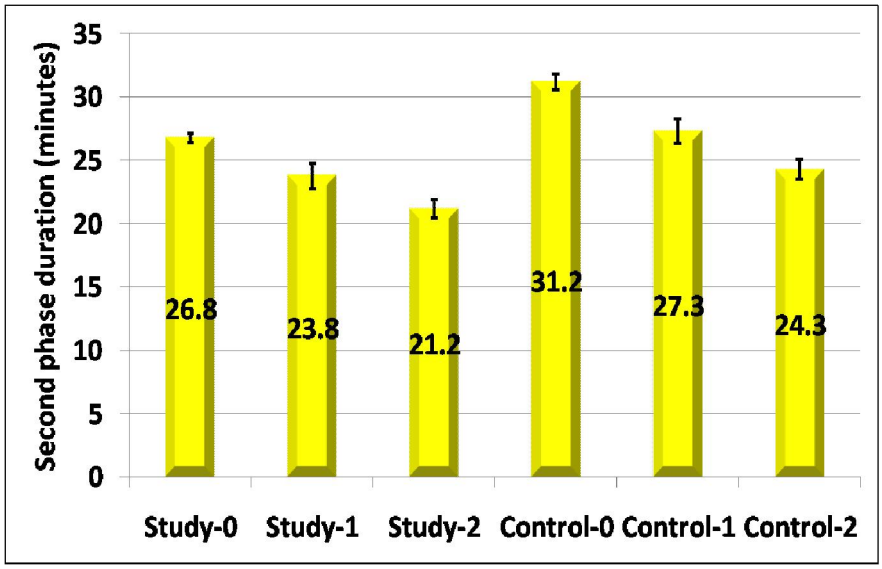

Figure (3): Comparison between study groups regarding second stage duration

Table (6): Comparison between parity groups regarding second stage duration (minutes)

Group	Measures	Nulli	Para-1	Para-2	^P
Study	**Mean±SD**	26.8±1.5	23.8±4.4	21.2±3.2	**<0.001***
	Range	23.0–29.0	15.0–30.0	13.0–25.0	
	HG	a	B	c	
Control	**Mean±SD**	31.2±2.8	27.3±4.3	24.3±3.5	**<0.001***
	Range	28.0–37.0	20.0–36.0	21.0–37.0	
	HG	a	B	c	

^ANOVA test with post hoc Tukey test, *Significant, HG: Homogenous groups have the same letter

4. Discussion

It is well known that glucocorticoids accelerate lung maturation by enhancing surfactant synthesis in the pulmonary alveolar cells. Evidence has been obtained from early studies that the phospholipid content of surfactant provides a source of arachidonic acid that can be used by the amnion for prostaglandin synthesis. Recently there is direct evidence pointing to surfactant protein A (SP-A) as the key link between the maturing fetus and the initiation of parturition in the mouse ***(Montalbano AP et al., 2013).***

Glucocorticoids derived from the maturing fetal hypothalamus-pituitary-adrenal axis play a crucial role in, triggering parturition ***(Challis el al., 2005).***

In humans, the placenta synthesizes corticotrophin-releasing hormone (CRH), and the exponential rise of this hormone in maternal plasma correlates with the timing of birth ***(Smith R et al., 2007).***

The corticotrophin-releasing hormone (CRH), which has been identified in various organ systems, including the female reproductive system, is the principal regulator of the hypothalamic-pituitary-adrenal axis. Circulating placental CRH is responsible for the physiologic hypercortisolism of the latter half of pregnancy and plays a role in the onset of labor ***(Kalantaridou et al., 2007).***

Cortisol increases the production of prostaglandins in the fetal membranes by either up regulating prostaglandin synthesis levels or down regulating 15-hydroxy prostaglandin dehydrogenase (PGDH) ***(Li Y et al., 2013).***

Therefore, glucocorticoids also play an important role in human parturition. In the fetal membranes, the actions of glucocorticoids are amplified by the actions of 11β-HSD steroid dehydrogenase type I (11β-HSD1), where 11β-HSD1 converts biologically inert cortisone to active cortisol thereby increasing the local levels of biologically active glucocorticoids. This cascade of events initiated by glucocorticoids may play an important role in the positive feed-forward mechanisms ***(Yang Z et al., 2007).***

This case controlled trial study was been conducted in the labor ward of El Galaa Teaching Hospital to evaluate the effect of intramuscular dexamethasone administration on the duration of induction of labor.

This study comprised 120 pregnant women with full term pregnancy, who admitted to the labor ward for induction of labor because of full-term pregnancy (gestational age ≥40 weeks).

Pregnant women were randomized (assigned) to receive dexamethasone sodium phosphate 8 mg (2 ml) or receive nothing or any other cervical ripening agent.

As regarding our results:

- The study showed there were no significant statistical difference between the two studied groups regarding the mean maternal age (years), Birth weight and body mass index (BMI).

In addition, there were non-significant statistical differences between the two groups as regard primary Bishop Score.

- The present study showed that a dexamethasone injection intramuscularly has suggestedno significant difference between the third stage of delivery, maternal complication and neonatal outcome (APGAR Scores – meconium aspiration – NIcu Admission).

It also shows that CS frequency was non-significantly less frequent among study groups than among control groups at different parities and It also shows that CS frequency was non-significantly least frequent among among para-2 groups, followed by para-1 groups and most frequent among nulliparous groups at both study and control groups.

- Administration of intramuscular dexamethasone shows that the **Third stage durations** were non-significantly shorter among study groups than among control groups at different parities **and** were non-significantly shortest among para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups.
- **The intervals** between the initiation of labor induction and the beginning of the active phase of labor were significantly shorter in study groups than control groups as in nulliparous were (3.9±0.5hrs vs. 4.9±0.4hrs) p=(**<0.001**), Para 1 groups were (3.2±1.2hrs vs. 4.0±1.0hrs) p=(**0.032)** and in Para 2 groups were **(**2.5±0.8hrs vs. 3.1±0.8hrs) p=(**0.017).**

The intervalsbetween the initiation of labor induction and the beginning of the active phase of labor were

significantly shortest among para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups. in study groups were (2.5±0.8hrs vs. 3.2±1.2hrs vs. 3.9±0.5hrs) p = (**<0.001)** and in control groups were **(**3.1±0.8hrs vs. 4.0±1.0hrs vs. 4.9±0.4hrs) p= **(<0.001).**

- **Active phase durations** were significantly shorter among study groups than among control groups at different parities as nulliparous (3.5±0.4 hrs. VS. 4.4±0.5 hrs.) p=**(<0.001),** Para 1 (2.8±1.0hrs vs. 3.6±1.0hrs) p= **(0.020) and** in Para2 **were (**2.2±0.7 vs. 2.8±0.8) p= (**0.011).**

On other side Active phase durations were significantly shortest among para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups. it was in study groups (2.2±0.7hrs vs. 2.8±1.0hrs vs. 3.5±0.4hrs) p = **(<0.001)** and in control group was (2.8±0.8hrs vs. 3.6±1.0hrs vs. 4.4±0.5hrs) p= **(<0.001).**

- **The second stage** of labor was shorter in the dexamethasone group than in control group at different parities as in nulliparous were (26.8±1.5 minutes vs. 31.2±2.8 minutes) p=**(<0.001),** in Para 1groups were **(**23.8±4.4 minutes vs. 27.3±4.3 minutes) p = (**0.014)** and in para2 groups (21.2±3.2 minutes vs. 24.3±3.5 minutes) p=**(0.006).**

The second stage durations were significantly shortest among para-2 groups, followed by para-1 groups and longest among nulliparous groups at both study and control groups. In study groups were (21.2±3.2 minutes vs. 23.8±4.4 minutes vs. 26.8±1.5 minutes) p = **(<0.001).** In control groups were **(**24.3±3.5mintes vs. 27.3±4.3mintes vs. 31.2±2.8mintes) p = **(<0.001).**

Our findings are in agreement with those observed by ***Maryam Kashanian et al., 2008*** who evaluated the effect of dexamethasone administration on labor duration. A controlled trial including 122 nulliparous women with a full-term pregnancy and a Bishop score of 7 or greater, were randomly assigned to receive a single 8 mg dose of dexamethasone for the case group or placebo for the control group 6 hours before initiation of labor induction.

They found that the interval between initiation of labor induction and beginning of the active phase of labor was shorter in the dexamethasone than in the control group. The duration of the second stage of labor was also shorter in the dexamethasone group. They concluded that the administration of dexamethasone was found to shorten labor duration by decreasing the interval between the induction and the beginning of the active phase, with no observed maternal or neonatal complications (***Maryam Kashanian et al., 2008).***

Kashanian et al., 2008 reported on the extra-amniotic infusion of a saline solution mixed with dexamethasone through a Foley catheter whose balloon was filled with 15 ml of water, and concluded that the procedure could shorten the duration of labor without significant maternal or fetal risk.

O'Sullivan et al., 2007 concluded that fetuses with congenital adrenal hyperplasia due to 21-hydroxylase deficiency were more likely to have a prolonged gestation, and this may be due to impaired cortisol production.

Hajivandi L et al., 2013 performed clinical trial on 100 eligible nulliparous women in their 40 to 42 weeks of gestation in 2009 who were admitted to Amir Hospital in Ahvaz. For the case group, 8 mg dexamethasone was administered 12 hours before induction and the controls were given 2 ml of normal saline at the same intervals.

There was no significant difference between the two groups in terms of age, demographic characteristics, initial Bishop score, first and fifth minute Apgar score, and meconium difference. There was a significant difference between the two groups (p =0.001) concerning the mean-time interval between the induction and the onset of active phase in the case group (3.1±0.68 hours) and in the control group it was (4.2±1.3 hours). They concluded that intra-muscular dexamethasone reduces the time duration from the induction to the onset of labor phase ***(Hajivandi L et al., 2013).***

In another study, conducted by ***Ziaee et al.,2003,*** thataimed to determine the effect of intra-muscular injection of dexamethasone on induction of labor. Women in 41 weeks gestational age and Bishop score greater than or equal to 7 received intramuscular injections of 10 mg dexamethasone in two doses with 12 hours interval, and the next day, induction was carried out using oxytocin. These patients were compared with patients in similar conditions, but receiving oxytocin.

In this study, more of the patients from dexamethasone group entered active phase than that in control group, and interval between induction and onset of active phase was shorter in this group than in control group. They reported that intra-muscular injection of dexamethasone before labor induction reduced the time between the induction and the active phase of labor (***Ziaei S et al., 2003).***

In another study conducted by ***Barakai et al., 1997*** with the aim to investigate the effect of extra-amniotic normal saline with dexamethasone for induction of labor, the interval between induction and onset of active labor in dexamethasone group was shorter than that in the group that received extra-amniotic normal saline only.

Also, 90.25% of dexamethasone group entered active phase, and 88.37% of control group, but the difference was insignificant. Mean onset of oxytocin to delivery was 7.25±2.86 hours in the case group, and 9.76±3.91 hours in the control group, with a significant difference between the two groups (p =0.002). Results of this study showed that injection of extra-amniotic normal saline was a suitable and inexpensive method for cervical ripening and response to induction. The addition of dexamethasone could help to shorten delivery process and that inducing labor by means of an extra-amniotic infusion of corticosteroids through an intra-cervical Foley balloon catheter reduced the time between induction of labor and delivery. This may indicate a possible role for corticosteroids in the parturition process (***Barakai et al., 1997).***

LigginsGC, 1968 found that ACTH infusion or cortisol into fetal sheep at more than 88 days of gestation causes parturition**.**

Elliot et al.,1995 showed that betamethazone administration in humans for fetal lung maturity in triplet and quadruplet births is associated with increase uterine contractions and preterm labor with cervical changes requiring tocolysis**.**

Mati et al., 1973 induced labor successfully in six post-date patients by giving 20 mg betamethazone into amniotic fluid. The mean time for onset of labor in the steroid group (67.4±24.3 hrs.) was shorter than the placebo-injected patients (312±142 hrs.), p less than 0.01. They concluded that it is clear that the betamethasone injections accelerated the onset of labor without any harmful effects on infants or mothers.

Moreover In agreement with ***M. Doganay et al., 2004***studied the relationship between maternal endogenous DHEAS, success of labor induction and Bishop score in post-term pregnancies. They found that DHEAS levels might be an important factor influencing the efficiency of labor and the success of labor induction in post-term pregnancies**.**

Goolsby et al., 1996 concluded that DHEAS levels are a factor influencing labor efficiency in women with term and post-term pregnancies.

Ali F. Al-Assadi et al., 2007 found that induction of labor using extra-amniotic Foley's catheter and dexamethasone reduces the ripening and induction delivery times; this may indicate a possible role for corticosteroids in the parturition process.

This goes with ***A. Sh. Zafarghandi et al., 2004*** who challenged the possible role of corticosteroids in induction of labor by extra-amniotic injection through an inflated intra-cervical Foley balloon catheter. This randomized trial was conducted on 44 women with a single pregnancy, intact membranes, and an unfavorable cervix.

They were randomly assigned to receive either 20 mg of dexamethasone in saline solution (study group, n=22) or saline solution only (control group, n=22) administered extra-amniotically through an intra-cervical inflated Foley balloon catheter. Eighteen (81.8%) patients in the study group and twenty (90.9%) in the control group entered the active phase of labor and were delivered vaginally. The mean time intervals between induction of labor to the active phase and between induction of labor to delivery were significantly shorter in the study group compared with those of the control group (3.3±2.1 hours vs. 9±4.7 hours, p<0.01, 5.7±3.4 hours vs. 6.9±4.7 hours, p<0.01, respectively). There was no maternal or fetal complication in study or control group.

They concluded that the intra-cervical Foley balloon catheter with extra-amniotic corticosteroids was more efficient in reducing the induction-to-delivery interval for termination of mid-trimester pregnancies than the same Foley catheter with saline solution only. Cervical ripening with extra-amniotic corticosteroids possesses the advantages of simplicity, low cost, lack of systemic or serious side effects ***(A. Sh. Zafarghandi et al., 2004)***.

This goes with ***M. Mansouri et al., 2003*** who tried to show the effect of extra-amniotic administration of corticosteroids to shorten the times to either active labor and/or delivery. This is a double blind randomized study. 65 patients who were candidates for the termination of pregnancy between the ages of 16-45, with intact membranes and unripe cervix were randomly divided into two groups, a study group (n=34) and a control group (n=31).

In the study group, 20 mg of dexamethasone was infused through a Foley catheter into the extra-amniotic space and the infusion was continued with normal saline in both groups. The result of the study showed that the interval of induction to active phase of labor was shorter in the study group compared to control group. The interval of induction to delivery was shorter in the study group compared to the control group. They concluded that corticosteroids might have a role in shortening the interval of induction to active phase of labor and the interval of induction to delivery (***M. Mansouri et al., 2003***).

Sciscioneet al., 2001 showed that using a Foley catheter is useful for pre-induction cervical ripening and advised to be used in outpatient versus inpatient setting.

In contrast to our results, **Kavanegh *et al.*, 2001&2006** in a review study on the effect of corticosteroids in cervical ripening and induction of labor concluded that, efficacy of corticosteroids in induction of labor was still unknown and required further studies. In 2006, they extended their studies, but arrived at the same conclusion.

Conclusion:

Single intra-muscular injection of two ml. (8mg.) of dexamethasone before induction of labor appears to shorten labor duration.

References:

1. Hoffman B, Schorge J, Schaffer J, et al, editors. Williams obstetrics. 23rd ed. New York: MacGrawhill books; 2012: p. 96-122.
2. Kavanagh J, Kelly AJ, Thomas J. Corticosteroids for cervical ripening and induction of labor. Cochrane Database Syst Rev 2006;(2): CD003100.
3. Kavanagh J, Kelly AJ, Thomas J. Corticosteroids for induction of labor. Cochrane Database Syst Rev 2001;(2): CD003100.
4. Goolsby L, Schlecht K, Racowsky C, et al. Maternal serum dehydroepiandrosterone sulfate levels and the efficiency of labor in young nulliparas. ObstetGynecol 1996;88(1):56- 9.
5. Doğanay M, Erdemoglu E, Avşar AF, Aksakal OS. Maternal serum levels of dehydroepiandrosterone sulfate and labor induction in postterm pregnancies. Int J GynecolObstet 2004;85(3):245—9.
6. McAuliffe F, Grimes H, Morrison JJ. Fetal cortisol in relation to labour, intrapartum events and mode of delivery. J ObstetGynaecol 2004;24(2):129—32.
7. Wood CE, Keller-Wood M. Induction of parturition by cortisol: effects on negative feedback sensitivity and plasma CRH. J DevPhysiol 1991;16(5):287—92.
8. Berkowitz GS, Lapinski RH, Lockwood CJ, Florio P, BlackmorePrince C, Petraglia F. Corticotrophin-releasing factor and its binding protein: maternal serum levels in term and preterm deliveries. Am J ObstetGynecol 1996;174(5):1477—83.
9. Campbell EA, Linton EA, Wolfe CD, Scraggs PR, Jones MT, Lowery PJ. Plasma corticotropin-releasing hormone concentrations during pregnancy and parturition. J ClinEndocrinolMetab 1987;64(5):1054—9.
10. Kalantaridou S, Makrigiannakis A, Zoumakis E, Chrousos GP. Peripheral corticotrophin-releasing hormone is produced in the immune and reproductive systems: actions, potential roles and clinical implications. Front Biosci 2007;12:572—80.
11. O'Sullivan J, Iyer S, Taylor N, Cheetham T. Congenital adrenal hyperplasia due to 21-hydroxylase deficiency is associated with a prolonged gestational age. Arch Dis Child 2007;92(8):690—2.
12. Barkai G, Cohen SB, Kees S, Lusky A, Margalit V, Mashiach S, Schiff E. Induction of labor with use of a Foley catheter and extraamniotic corticosteroids. Am J ObstetGynecol 1997;177(5): 1145—8.
13. Ziaei S, Rosebehani N, Kazeminejad A, Zafarghandi S. The effects of intramuscular administration of corticosteroids on the induction of parturition. J Perinat Med 2003;31(2):134—9.
14. Lindsay JR, Nieman LK. The hypothalamic–pituitary–adrenal axis in pregnancy: challenges in disease detection and treatment. Endocr Rev 2005;26(6):775—99.
15. McLean M, Smith R. Corticotrophin-releasing hormone and human parturition. Reproduction 2001;121(4):493—501.
16. Montalbano AP, Hawgood S, Mendelson CR (2012): Mice deficient in surfactant protein A (SP-A) and SP-D or in TLR2 manifest delayed parturition and decreased expression of inflammatory and contractile genes. Endocrinology, 154(1):483-498.
17. Challis JR, Bloomfield FH, Bocking AD, Casciani V, Chisaka H, Connor K, Li W (2005): Fetal signals and parturition. Journal of Obstetrics and Gynaecology Research, 31(6):492-499.
18. Challis JRG, Matthews SG, Gibb W, Lye SJ (2000): Endocrine and paracrine regulation of birth at term and preterm. Endocr Rev 21: 514-550.
19. Smith R, Smith JI, Shen X, Engel PJ, Bowman ME, McGrath SA, Smith DW (2009): Patterns of plasma corticotropin-releasing hormone, progesterone, estradiol, and estriol change and the onset of human labor. The Journal of Clinical Endocrinology & Metabolism, 94(6): 2066-2074.
20. Kalantaridou S, Makrigiannakis A, Zoumakis E, and Chrousos GP (2007): Peripheral corticotrophin-releasing hormone is produced in the immune and reproductive systems: actions, potential roles and clinical implications. Front Biosci; 12: 572-80.
21. Yang Z, Guo C, Zhu P, Li W, Myatt L, Sun K (2007): Role of glucocorticoid receptor and CCAAT/enhancer-binding protein-α in the feed-forward induction of 11β-hydroxysteroid dehydrogenase type 1 expression by cortisol in human amnion fibroblasts. J. Endocrinol.;195:241–253.
22. KashanianM, Fekrat M, Zarrin Z, AnsariNS (2008): A comparison between the effect of oxytocin only and oxtocin plus propranolol on the labor (a double blind randomized trial). Journal of Obstetrics and Gynaecology Research, 34(3):354-358.

23. O'sullivan J, Iyer S, Taylor N, Cheetham T (2007): Congenital adrenal hyperplasia due to 21-hydroxylase deficiency is associated with a prolonged gestational age. Archives of disease in childhood, 92(8): 690-692.
24. O'Sullivan J, Iyer S, and Taylor N, Cheetham T (2007): Congenital adrenal hyperplasia due to 21-hydroxylase deficiency is associated with a prolonged gestational age. Arch Dis Child; 92(8): 690-2.
25. Hajivandi L, Montazeri S, Iravani M, and Dawoodi M, Haghighizade MH (2013): Effect of intramuscular dexamethasone on onset of labor in postdates pregnancy. Journal of BabolUniversity of Medical Sciences, 15(3):24.
26. Hajivandi L, Montazeri S, Iravani M, Haghighizade MH (2013): The effects of intramuscular dexamethasone on cervical ripening and induction of labor. Journal of JahromUniversity of Medical Sciences, 11(3):11.
27. Ziaei S, Rosebehani N, Kazeminejad A, Zafarghandi S (2003): The effects of intramuscular administration of corticosteroids on the induction of parturition. J Perinat Med;31(2):134-9.
28. Barkai G, Cohen SB, Kees S, Lusky A, Margalit V, Mashiach S, Schiff E (1997): Induction of labor with use of a Foley catheter and extraamniotic corticosteroids. American journal of obstetrics and gynecology, 177(5):1145-1148.
29. Liggins GC (1968): Premature parturition after infusion of corticotrophin or cortisol into foetal lambs. J Endocrinol 42:323–329 - See more at: http://press.endocrine.org/doi/full/10.1210/endo.139.7.6086#sthash.7D5MTaAE.dpuf.
30. Elliott JP andRadin TG (1995): The effect of corticosteroid administration on uterine activity and preterm labor in high-order multiple gestations. Obstetrics & Gynecology, 85(2):250-254.
31. Mati JKG, Horrobin DF, Bramley PS (1973): Induction of labour in sheep and in humans by single doses of corticosteroids. Br Med J, 2(5859): 149-151.
32. Doğanay M, Erdemoglu E, Avşar AF, Aksakal OS (2004): Maternal serum levels of dehydroepiandrosterone sulfate and labor induction in post term pregnancies. Int J Gynaecol Obstet.; 85(3): 245-9.
33. Goolsby L, Schlecht K, Racowsky C, et al. (1996): Maternal serum dehydroepiandrosterone sulfate levels and the efficiency of labor in young nulliparas. ObstetGynecol; 88(1):56- 9.
34. Goolsby L, Schlecht K, Racowsky C, Gelety T, Reed K (1996): Maternal serum dehydroepiandrosterone sulfate levels and the efficiency of labor in young nulliparas. Obstetrics & Gynecology, 88(1):56-59.
35. ZafarghandiAS andBaghaii NZN (2004): Foley catheter cervical ripening with extraamniotic infusion of saline or corticosteroids: a double-blind, randomized controlled study. Acta MedicaIranica, 42(5):338-342.
36. Mansouri M, Pourjavad A, Panahi G (2003): Induction of labor with use of a Foley catheter and extraamniotic corticosteroids. Medical Journal of The Islamic Republic of Iran (MJIRI), 17(2):97-100.
37. Sciscione AC, Muench M, Pollock M, Jenkins TM, Tildon-Burton J, Colmorgen GH (2001): Transcervical Foley catheter for preinduction cervical ripening in an outpatient versus inpatient setting. Obstetrics & Gynecology, 98(5):751-756.

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