**Effect of Intramuscular Administration of Dexamethasone on the Duration of Labor in Full-Term Primigravida**

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**Abstract: Objectives:** to evaluate the effect of dexamethasone on labor duration and to establish whether dexamethasone plays a role in shorting the duration interval between initiation of labor induction and beginning of the active phase of labor in primigravida full-term pregnancy. **Study design:** Case control study included 200 primigravidae with full-term pregnancy classified into two groups: group I (cases) included 100 women assigned to receive a single 8-mg dose of dexamethasone intra-muscular and group II (control) included 100 women will not receive dexamethasone or any other cervical ripening agent. **Results:** The interval between initiation of labor induction and beginning of active phase of labor was shorter in the dexamethasone than in the control group **(2.54±0.94 hours vs. 3.59±0.86 hours; p=0.001).** Dexamethasone group shows shorter duration of active phase of labor than control group **(4.82±0.56 hrs. vs. 5.12±0.58 hrs.)**. Dexamethasone group shows shorter duration of first stage of labor than control group **(7.35±1.15 hrs. vs. 8.69±1.09 hrs.)**. Dexamethasone group shows faster rate of cervical dilatation than control group **(1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.)**. Dexamethasone group shows shorter duration of second stage of labor than control group **(25.09±12.99 minutes vs. 30.73±12.96 minutes).** Oxytocin requirement in dexamethasone group was less than in control group **(5.35±1.49 hrs. vs. 5.97±1.34 hrs.). Conclusions**: The administration of dexamethasone found to shorten labor duration.

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**Keywords:** Dexamethasone; post-term pregnancy; induction of labor.

**1. Introduction**

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 ***(Kavanagh et al., 2006)***. Several animal studies have shown the importance of corticosteroid secretion by the fetal adrenal glands on the beginning of labor ***(Kavanagh et al, 2006, Wood, Keller-Wood et al., 1991)***, and infusing glucocorticoids in the lamb fetus was also shown to induce preterm labor. These findings have led to the hypothesis that corticosteroids also had an effect on the labor of women ***(Kavanagh, Kelly, Thomas,2006, 2001).***

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 ***(Kalantaridou Kavanagh Campbell,2007).***

**Kalantaridou et al. (2007)** 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. (2007)*** 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 ***(Barkai et al,2007, McLean et al.,2001).***

Induction of labor refers to the process of artificially initiating uterine contractions prior to their spontaneous onset to effects progressive effacement and dilatation of the cervix and ultimately, delivery of the baby ***(Hayman, 2010).***

Induction of labor is one of the most common interventions practiced in modern obstetrics. In the developed World, the ability to induce labor has contributed to the reduction in maternal and perinatal mortality and morbidity ***(Subramanian and Penna, 2009).***

The goal of labor induction is to stimulate uterine contractions before the spontaneous onset of labor, resulting in vaginal delivery. The benefits of labor induction must be weighed against the potential maternal and fetal risks associated with this procedure. When the benefits of expeditious delivery are greater than the risks of continuing the pregnancy, inducing labor can be justified as a therapeutic intervention ***(Barclay, 2009).***

The success of induction and labor progression is dependent on the condition of the cervix before induction initiation ***(Barclay, 2009).***

In primigravidae, the mean time taken from induction to delivery is between 15 and 20 hours, of which up to 12 hours is spent in the cervical ripening phase before labor itself starts (***Stitely et al., 2000).***

About 10 percent of pregnancies may be prolonged. In general, the longer the truly post-term fetus stays in the uterus, the greater the risk of a severely compromised fetus and newborn infant. Therefore of major importance in handling compromised postdate pregnancies is the use of a suitable method of labor induction. A prolonged gestation is more likely to occur when the fetus has congenital adrenal hyperplasia caused by 2l-hydroxylase deficiency, which may be due to an impaired cortisol production ***(O'Sullivan et al., 2007).***

Glucocorticoids are now known to play key roles in fetal maturation for example in maturation of the lung in anticipation of extra-uterine life and in several species appear to be mediators in the initiation of labor. In humans, the placenta synthesizes CRH, and the exponential rise of this hormone in maternal plasma correlates with the timing of birth ***([Falah N](http://www.ncbi.nlm.nih.gov/pubmed/?term=Falah%20N%5BAuthor%5D&cauthor=true&cauthor_uid=25256192) et al., 2014).***

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

During pregnancy, large amounts of CRH are released from the placenta and fetal membranes. An increment in plasma CRH concentration occurs during spontaneous labor, with peak value at vaginal delivery ***(Riley & Challis, 2003).*** Placental CRH is also released into the fetal circulation, dehydroepiandrosterone and in vitro CRH directly stimulates sulfate (DHEA-S) production from the fetal zone of the fetal adrenal ***(Sirianni et al., 2005).***

This increase in fetal zone activity correlates with rising levels of maternal estrogen levels through the conversion of DHEA-S to estrogens within the placenta. The increase in the maternal estrogen to progesterone ratio may promote the expression of contraction-associated proteins in the myometrium, thus facilitating the initiation of parturition ***(Mastorakos and Ilias, 2003).***

Cortisol increases the production of prostaglandins in the fetal membranes by either up regulating prostaglandin synthesis (PGHS-2) levels or down regulating 15-hydroxy prostaglandin dehydrogenase (PGDH). It has been very well recognized that increased prostaglandin (PGE2 and PGF2) biosynthesis as a result of inflammation-like responses in intrauterine tissues is one of the key events leading to parturition in both term and preterm human labor because these compounds evoke uterine contractions as well as cervical softening and effacement.

**2. Patient and Methods**

This prospective clinical interventional randomized case-controlled trial was conducted at Al-Hussein university Hospital during the period from 2016 June to 2017 March.

**Methodology (plan):**

It included 200 participants whom are admitted for labor induction at Al-Hussein University Hospital.

The participants will be randomly assigned by computer list into Group I (Dexamethasone group) N=100 and Group II (Control group) N=100.

The participants of Group І 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.

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

After approval of health committee in Al-Hussein Hospital, a verbal consent was obtained from each candidate after explanation of the procedure in details.

**Statistical Analysis:**

All clinical and demographic data will be recorded on investigative report form. These data will be analyzed by IBM computer using SPSS (Statistical program for social science version 12) as follows:

Description of quantitative (numerical) variables will be performed in the form of mean, standard deviation (SD) and range.

Description of qualitative (categorical) variables will be performed in the form number of cases and percentage.

Chi-square test will be used to compare qualitative variables between groups.

Fisher exact test will be used instead of chi-square when one expected cell or more less than five.

Unpaired t-test will be used for comparison of quantities variables, in parametric data (SD<50%) of mean.

Paired t-test will be used to compare pre and post quantitative results in the same group.

***p*-value (level of significance):**

*p*>0.05= non-significant.

*p*<0.05= significant.

*P<*0.001= highly significant.

Data were graphically represented using Harvard Graphics program.

**3. Results**

Data were statistically described in terms of mean ± standard deviation (±SD), median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using Student *t* test for independent samples.

For comparing categorical data, Chi square (χ2) test was performed. Exact test was used instead when the expected frequency is less than 5. *p-*values less than 0.05 was considered statistically significant.

All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) release 15 for **Microsoft Windows (2006).**

**Table (1): Demographic characteristics of the patients (mean ± SD)**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **Dexamethasone group (n=100)** | **Control group (n=100)** | ***p-*value** | **Sig.** |
| **Age (years)** | 26±4.36 | 25.63±3.79 | 0.624\* | N.S. |
| **BMI (Kg/m2)** | 23.09±1.89 | 22.78±1.71 | 0.344\* | N.S. |
| **Gestational age on admission (weeks)** | 40±1.46 | 40±1.35 | 0.796\* | N.S. |

**Values are mean ±S.D. & number (percentage) Student t-test\***

**S.D.: Standard Deviation; N.S.: Non-significant**

There were non-significant statistical differences between the two studied groups as regard age, body mass index (BMI), gestational age.

**Table (2): Statistical comparison between the two studied groups as regards pulse and blood pressure (vital signs)**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p*-value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| N.S. | 0.746\* | 79.4±5.13 | 79.07±6.09 | **Pulse (bpm)** |
| N.S. | 1.000\* | 118±13.38 | 118±14.59 | **Systolic BP** |
| N.S. | 0.748\* | 73±8.69 | 72.5±8.31 | **Diastolic BP** |

**Values are Mean ± S.D. \*Student t-test**

**S.D.: Standard Deviation; N.S.: Non-significant; bpm: Beat per minutes; BP: Blood Pressure**

There were non-significant statistical differences between the two groups as regard pulse and blood pressure (vital signs).

**Table (3): Statistical comparison between the two studied groups as regards Bishop score at time of intervention**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p*-value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| N.S. | 0.589\* | 2.5±0.62 | 2.43±0.72 | **Cervical dilatation (cm)** |
| N.S. | 0.686\* | 44±10.61 | 43.33±7.05 | **Effacement (%)** |
| N.S. | 0.462\*\* | 6 (6%)  34 (34%)  60 (60%) | 4 (4%)  36 (36%)  60 (60%) | **Consistency**  **Firm**  **Intermediate**  **Soft** |
| N.S. | 0.930\*\* | 7 (7%)  53 (53%)  40 (40%) | 7 (7%)  53 (53%)  40 (40%) | **Position**  **Posterior**  **Central**  **Anterior** |
| N.S. | 0.094\*\* | 26(26%)  10 (10%)  54 (54%)  10 (10%) | 26 (26%)  10 (10%)  54 (54%)  10 (10%) | **Station of fetal head**  **-2**  **-1**  **0**  **+1** |
| N.S. | 1.000\* | 7.63±0.71 | 7.63±0.66 | **Total Bishop score** |

**Values are mean ± S.D. & number (%) \*Student t-test**

**S.D.: Standard Deviation; \*\*Chi-square test; N.S.: Non-significant**

There were non-significant statistical differences between the two groups as regard cervical

dilatation, effacement, cervical position, consistency, head station and total Bishop score.

**Table (4): Statistical comparison between the two studied groups as regards duration between induction of labor and active phase**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p*-value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| H.S. | 0.001\* | 3.59±0.86 | 2.54±0.94 | **Duration between induction of labor and active phase (hr.)** |

**Values are mean ± SD \*Student t-test S.D.: Standard Deviation; H.S.: Highly Significant**

Dexamethasone group shows shorter duration between labor induction and active phase of labor than control group (2.54±0.94 hr. vs. 3.59±0.86 hr.).

There was **a high significant statistical difference** between the two studied groups as regards duration between labor induction and active phase of labor (*p* less than 0.001).

**Table (5): Statistical comparison between the two studied groups as regards duration of active phase of labor**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| S | 0.006\* | 5.12±0.58 | 4.82±0.56 | **Duration of active phase of labor (hrs.)** |

**Values are mean ±S.D. \*Student t-test S.D.: Standard Deviation; S: Significant**

Dexamethasone group shows shorter duration of active phase of labor than control group (4.82±0.56 hr. vs. 5.12±0.58 hr.).

There was **a significant statistical difference** between the two studied groups as regards duration of active phase of labor (*p* less than 0.05).

**Table (6): Statistical comparison between the two studied groups as regards duration of 1st stage of labor**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| S | 0.001\* | 8.69±1.09 | 7.35±1.15 | Duration of 1st stage of labor (hr.) |

**Values are mean ± SD \*Student t-test S.D.: Standard Deviation; S: Significant**

Dexamethasone group shows shorter duration of first stage of labor than control group (7.35±1.15 hr. vs. 8.69±1.09 hr.).

There was **a significant statistical difference** between the two studied groups as regards duration of first stage of labor (*p* less than 0.001).

**Table (7): Statistical comparison between the two studied groups as regards rate of cervical dilatation**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| S. | 0.01\* | 1.28±0.17 | 1.37±0.18 | **Rate of cervical dilatation (cm/hour)** |

**Values are mean ± S.D. \*Student t-test S.D.: Standard Deviation; S.: Significant**

Dexamethasone group shows faster rate of cervical dilatation than control group (1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.).

There was **a significant statistical difference** between the two studied groups as regards rate of cervical dilatation (*p* less than 0.05).

**Table (8): Statistical comparison between the two studied groups as regards duration of 2nd stage of labor**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| S | 0.032\* | 30.73±12.96 | 25.09±12.99 | **Duration of 2nd stage of labor (minutes)** |

**Values are mean ±SD. \*Student t-test S.D.: Standard Deviation; S: Significant**

Dexamethasone group shows shorter duration of second stage of labor than control group (25.09±12.99 minutes vs. 30.73±12.96 minutes).

There was **a significant statistical difference** between the two studied groups as regards duration of second stage of labor (*p* less than 0.05).

**Table (9): Statistical comparison between the two studied groups as regards dose of oxytocin required**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group** | **Dexamethasone group** |  |
| S | 0.019\* | 5.97±1.34 | 5.35±1.49 | **Oxytocin requirement (hours)** |

**Values are mean ± S.D. \*Student t-test S.D.: Standard Deviation; S: Significant**

Oxytocin requirement in dexamethasone group was less than in control group (5.35±1.49 hours vs. 5.97±1.34hours).

There was **a significant statistical difference** between the two studied groups as regards dose of oxytocin required (*p* less than 0.05).

**Table (10): Statistical comparison between the two studied groups as regards duration of 3rd stage of labor**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| N.S. | 0.155\* | 9.52±2.99 | 8.57±3.63 | **Duration of 3rd stage of labor (minutes)** |

**Values are mean± SD & number Student t-test\* S.D.: Standard Deviation; N.S.: Non-significant**

There was no significant statistical difference detected between the two studied groups as regards duration of 3rd stage of labor.

**Table (11): Statistical comparison between the two studied groups as regards mode of delivery and its indication**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p*-value** | **Control group (n=100)** | **Dexamethasone group (n=100)** | **Mode of delivery and indication** |
| N.S. | 0.43\*\* | 77  (77 %) | 83  (83%) | SVD |
|  |  | 23(23%)  8(34.7%)  5(21.7%)  8(34.7%)  2 (8.6 %) | 17 (17 %)  4 (23.5 %)  4 (23.5 %)  7 (41.1 %)  2(11.7 %) | C.S.  Failed induction  Failure to progress  Fetal distress  Deep transverse arrest (direct occipito- transverse) |

**Values are numbers (percentage). \*\*Chi-square test**

**N.S.: Non-significant; SVD: Spontaneous vaginal delivery; C.S.: Caesarean section**

There was a non-significant statistical difference between the studied groups as regards mode of delivery.

**Table (12): Statistical comparison between the two studied groups as regards neonatal outcome**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Sig.** | ***p-*value** | **Control group (n=100)** | **Dexamethasone group (n=100)** |  |
| N.S. | 0.407\* | 3190±271.34 | 3150±255.12 | **Birth weight (gm.)** |
| N.S. | 0.283\* | 7.1±0.66 | 7.23±0.69 | **Apgar score at 1 minute** |
| N.S. | 0.702\* | 8.65±0.71 | 8.7±0.72 | **Apgar score at 5 minutes** |
| N.S. | 0.769\*\* | 6 (6 %)  94 (94 %) | 7 (7 %)  93 (93 %) | **Fetal heart rate disturbance**  **Yes**  **No** |
| N.S. | 0.697\*\* | 6 (6%)  94 (94 %) | 5 (5 %)  95 (95 %) | **Meconium-stained liquor**  **Yes**  **No** |
| N.S. |  | 8 (8 %)  92 (92 %) | 6 (6. %)  94 (94%) | **Admission to NICU**  **Yes**  **No** |

**Values are mean ± SD & number (%) \*Student t-test. \*\*Chi-square test**

**S.D.: Standard Deviation ; N.S.: Non-significant ; NICE: Neonatal intensive care unit**

There were non-significant statistical difference between the two studied groups as regards birth weight, Apgar score at 1 minute, Apgar score at 5 minutes, fetal heart rate disturbance, meconium stained liquor & admission to NICU.

**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](http://www.ncbi.nlm.nih.gov/pubmed/?term=Montalbano%20AP%5BAuthor%5D&cauthor=true&cauthor_uid=23183169) 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***](http://www.ncbi.nlm.nih.gov/pubmed/?term=Smith%20R%5BAuthor%5D&cauthor=true&cauthor_uid=17127348) ***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***](http://www.ncbi.nlm.nih.gov/pubmed/?term=Li%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=23506845)  ***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***](http://www.ncbi.nlm.nih.gov/pubmed/?term=Yang%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=17951535)  ***et al., 2007).***

This case controlled trial study was been conducted in the labor ward of Al-Hussein University Hospital to evaluate the effect of intramuscular dexamethasone administration on the duration of labor.

This study comprised 200 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), the gestational age (weeks) on admission, pulse (beat per minute) and blood pressure; No such difference was found regarding body mass index (BMI) and percentage of cesarean section between the two studied groups.

In addition, there were non-significant statistical differences between the two groups as regard primary Bishop score (cervical dilatation, effacement, cervical position, consistency, head station and total Bishop score).

The present study showed that a dexamethasone injection intramuscularly has suggested no significant difference between the 2 groups in the duration of the third stage of labor & the neonatal outcome (Birth weight, APGAR score at 1 minute and 5 minutes, number of cases with fetal heart rate disturbance, meconium stained liquor & neonatal admission to neonatal intensive care unit).

The first stage of labor was shorter in dexamethasone group than control group (7.35±1.15 hrs. vs. 8.69±1.09 hrs.) (*p*=0.001).

The second stage of labor was shorter in the dexamethasone group than in control group (25.09±12.99 minutes vs. 30.73± 12.96 minutes) (*p*=0.032). The interval between the initiation of labor induction and the beginning of the active phase of labor was 2.54±0.94 hours in the dexamethasone group and 3.59±0.86 hours in the control group, and the difference was significant (*p-*value less than 0.001).

The duration of active phase of labor was 4.82±0.56 hours in dexamethasone group and 5.12±0.58 hours in control group, and the difference was significant (*p* value less than 0.05).

The rate of cervical dilatation is faster in dexamethasone group than control group (1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.), the difference was significant (*p* =0.01).

The mean oxytocin dose consumption on entering active phase was 5.35±1.49 units for dexamethasone group and 5.97±1.34 unites for control group and the difference was significant (*p* =0.019).

***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)*.**

***Liggins*** ***GC, 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*.*

***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.

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