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Corticosteroids Administration for Enhancement of Fetal Lung Maturity and its Effects on Doppler Indices

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Abstract

Background: Evaluation the effects of maternal corticosteroids administration on Doppler indices (RI, PI & S/D ratio) of umblical artery, fetal middle cerebral artery and fetal pulmonary trunk in third trimester and its correlation with pregnancy outcome.

Methods: One hundred and fourty (140) women were included in this study, and were divided in two groups, seventy (70) subjects as study group have a risk for preterm labour and seventy (70) subjects as control group with no risk of preterm labour, study group subjects received course of corticosteroids in third trimester. The Doppler examination of the Pulsatility Index (PI), Resistive Index (RI) and Systolic diastolic Ratio (S/D) of the Umbilical Artery (UA), the Middle Cerebral Artery (MCA) and Fetal Pulmonary Trunk (PT) were measured and comparison between values of both groups were done after zero, one, three and five weeks of corticosteroids administration of study group and same gestational age of control group.

Results: No significant change was observed in the mean values of the pulsatility, resistive indices and systolic-diastolic ratios of both groups in umbilical artery, fetal middle cerebral artery and fetal pulmonary trunk after one week after corticosteroids administration.

Conclusions: We demonstrated that dexamethasone administration did not cause changes in Doppler values and fetal biophysical profile scores after one week of its use.

Key Words: *Corticosteroids – Doppler – Preterm birth – Biophysical profile – Umbilical artery doppler – Middle cerebral artery doppler – Pulmonary trunk doppler.*

Introduction

PRETERM delivery is a leading cause of perinatal morbidity and mortality world wide and remains a significant problem in modern obstetrics [1]. Preterm infants are at risk for specific diseases such as respiratory distress syndrome, intraventricular hemorrhage, broncho-pulmonary dysplasia, patent ductus arteriosus, necrotizing enterocolitis, sepsis, apnea and retinopathy [2].

Synthetic corticosteroids have been successfully employed for more than 20 years to enhance fetal lung maturity in saturations where preterm delivery is anticipated [3].

Maternal administration of synthetic corticosteroids (betamethasone or dexamethasone), for accelerating the maturity of the fetal lung, reduces neonatal mortality, respiratory distress syndrome, intraventricular haemorrhage and necrotizing enterocolitis in preterm infants [4].

Serious side-effects on the neonate have not been described when prenatal treatment has been administered during the second half of pregnancy (RCOG, 2004). However, a transient reduction of fetal heat rate variation and fetal body and breathing movements following maternal betamethasone administration was recently reported by [5].

Monitoring of biophysical activities is a powerful tool for the assessment of fetal wellbeing. Some studies have provided preliminary evidence that antenatal steroids given for enhancement of fetal lung maturity may induce a transient suppression of fetal biophysical activities [6].

In fact, conflicting results concerning the effects of betamethasone and dexamethasone on fetal heart pattern have been reported [7].

Recently, Scheepens et al., [8] found that betamethasone and dexamethasone was associated with an increase in long term and short term variability and decreased fetal movements on the first day after steroid administration followed by a decline in fetal heart rate variability on the second day.

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Similarly [9], the trial of reported a transient decrease in fetal heart variability and in fetal body and breathing movement after betamethasone, conversely, a rise in fetal heart rate variability was noted on the first day after dexamethasone.

Doppler ultrasound has been used to measure the blood flow velocity in vessels during the cardiac cycle in the fetoplacental, uteroplacental circulation and has been focused on arteries for the evaluation of downstream distribution of cardiac output [10].

Doppler velocimetry does not only facilitate judgment in diagnosis and monitoring of fetal wellbeing during pregnancy and labour but also has a role in the early detection of fetal hypoxia [11].

Evaluation of fetal well being with Doppler waveform studies after maternal corticosteroid administration is therefore important knowledge of fetal haemodynamic effects after exogenous corticosteroids is limited [12].

Doppler flow velocimetry of the umbilical, fetal cerebral circulation and fetal pulmonary trunk serves as another modality for the assessment of fetal status. This technique can be helpful in identifying the compromised fetus and could be of clinical value in the differential diagnosis between drug-induced changes in fetal biophysical behavior and those due to fetal compromise [13].

Furthermore, antenatal administration of corticosteroids, given to accelerate the development of fetal maturity and improve perinatal outcome in pregnancies at risk of preterm delivery, has been shown to upregulate placental expression and secretion of Corticotrophin Releasing Hormone (CRH) which is a potent vasodilator of the fetal placental circulation, offering the possibility that corticosteroid treatment may alter placental blood flow [14].

Patients and Methods

This study was carried out at Obstetrics and Gynecology Department of Al-Hussien University Hospital from March 2015 to April 2017 on one hundred and forty (140) women with singleton pregnancies at a gestational age of 32 to 37 weeks divided into two groups. Group 1 : Seventy (70) subjects as study group have a risk for preterm labour. Group 2: Seventy (70) subjects as control group with no preterm labour.

Patient selection and inclusion criteria were, oral informed consent was obtained from all participating women, at the time of initial scanning all pregnancies had normal umbilical artery flow velocity waveforms values, preterm birth was anticipated on the basis of: History of previous preterm birth, preterm contractions of the uterus, history of recurrent spontaneous abortion, placenta previa and third trimester bleeding, preterm premature rupture of the membranes, polyhydramnios, patients with possible need for early termination of pregnancy including those with history of previous uterine rupture and hysterotomy.

Exclusion criteria were: Pregnancies with infants with major fetal structural malformations, women on corticosteroids treatment for another disease, any associated medical problem with pregnancy, patient's refusal.

An informed written consent was signed by every subject in study and control group. All patients enrolled in this study were subjected to full medical examination including general examination, local examination and special investigation (non stress test), intramuscular dexamethasone was given to group 1 subjects of the study following the setting protocol as 4 doses dexamethasone (6mg/12 hours for 48hrs), doppler ultrasound assessment of Umbilical (UA), fetal Middle Cerebral Artery (MCA) and fetal Pulmonary Trunk (PT) was carried out for all subjects in this study, using trans-abdominal probe 2.5:5MHz (Esaote C. class & Philips, clear, Vue, 350, C5-2), and evaluation of the pregnancy outcome as regards Apgar score, fetal birth and admission to NICU was done.

Evaluation of study group was done before (day 0) and after (1, 3, 5 weeks) of first dose of dexamethasone administration [15] by modified biophysical profile, doppler blood flow from Umbilical Artery (UA), fetal Middle Cerebral Artery (MCA) and fetal Pulmonary Trunk (PT) according to [4].

The examination was performed in a supine, slightly left lateral tilted position through the examination to avoid supine hypotension. Ultrasonographic and doppler Flow Velocity Waveforms (FVW) studies were done with pulsed-wave doppler after real time color flow localization of the fetal umbilical, middle cerebral arteries and pulmonary trunk [4].

Statistical analysis:

All the data collected were subjected to thorough statistical analysis.

Results

Table (1): Comparison between 2 groups according to characteristics.

Characteristics	Study	Control	*t*-test	*p*-value
Age (years):				
Mean ± SD	28.21±3.35	28.86±3.37	1.378	0.242
Range	20-36	20-36		
Parity:				
PG	18 (24.7%)	22 (30.6%)	1.033	0.793
P1	35 (47.9%)	29 (40.3%)		
P2	14 (19.2%)	14 (19.4%)		
P3	6 (8.2%)	7 (9.7%)		
H. PTL:				
Yes	11 (15.1%)	3 (4.2%)	4.939	0.026
No	62 (84.9%)	69 (95.8%)		

This table shows statistically significant difference between groups according to H. PTL.

Table (2): Comparison between study and control according to UA in all period of this study.

UA	Study	Control	*t*-test	*p*-value
RI:				
Mean ± SD	0.64±0.02	0.63±0.05	1.772	0.266
Range	0.58-0.69	0.58-0.69		
PI:				
Mean ± SD	0.94±0.07	0.94±0.04	1.016	0.152
Range	0.88-1.11	0.88-1.11		
S/D ratio:				
Mean ± SD	2.61±0.16	2.56±0.16	1.855	0.278
Range	2.27-2.91	2.27-2.91		

This table shows no statistically significant difference between groups according to UA.

Table (3): Comparison between study and control according to MCA in all period of this study.

MCA	Study	Control	*t*-test	*p*-value
RI:				
Mean ± SD	0.76±0.04	0.75±0.04	1.431	0.215
Range	0.67-0.85	0.67-0.85		
PI:				
Mean ± SD	1.71±0.19	1.68±0.15	0.669	0.100
Range	1.43-2.15	1.43-2.15		
S/D ratio:				
Mean ± SD	5.56±0.55	5.42±0.53	0.815	0.122
Range	4.34-7.11	4.34-7.11		

This table shows no statistically significant difference between groups according to MCA.

Table (4): Comparison between study and control according to PT in all period of this study.

PT	Study	Control	*t*-test	*p*-value
RI:				
Mean ± SD	0.86±0.09	0.84±0.08	1.008	0.151
Range	0.77-0.98	0.77-0.98		
PI:				
Mean ± SD	2.34±0.10	2.31±0.10	1.293	0.194
Range	2.18-2.51	2.18-2.51		
S/D ratio:				
Mean ± SD	9.49±1.45	9.18±1.37	1.036	0.155
Range	6.18-11.11	6.18-11.11		

This table shows no statistically significant difference between groups according to PT.

Table (5): Comparison between 0, 1, 3, 5. Wk after corticosteroid administration according to UA in study group.

UA	Before	1st wks.	3rd wks.	5th wks.	P1	P2	P3
• RI	0.65± 0.02	0.65± 0.02	0.63± 0.02	0.61± 0.01	0.306	0.247	0.031
• PI	0.98± 0.04	0.97± 0.04	0.91± 0.10	0.90± 0.02	0.175	0.115	0.014
• S/D ratio	2.73± 0.09	2.72± 0.09	2.53± 0.07	2.41± 0.06	0.320	0.140	0.018

P1: Difference between before and 1st wks.
P2: Difference between before and 3rd wks.
P3: Difference between before and 5th wks.

This table shows statistically significant difference between before and 5th wks according to UA.

Table (6): Comparison between GA (wks) according to UA in control.

UA	32wks.	35wks.	37wks.	P1	P2
RI	0.64±0.07	0.63±0.02	0.61±0.01	0.294	0.039
PI	0.98±0.05	0.94±0.02	0.90±0.01	0.158	0.028
S/D ratio	2.74±0.09	2.53±0.07	2.40±0.05	0.125	0.010

P1: Difference between 32wks and 35wks.
P2: Difference between 32wks and 37wks.

This table shows statistically significant difference between 32wks and 37wks according to UA.

Table (7): Comparison between 0, 1, 3, 5Wk after corticosteroid administration according to MCA in study group.

MCA	Before	1st wks.	3rd wks.	5th wks.	P1	P2	P3
• RI	0.79± 0.04	0.79± 0.03	0.73± 0.02	0.71± 0.02	0.174	0.481	0.022
• PI	1.83± 0.12	1.80± 0.21	1.69± 0.16	1.46± 0.06	0.223	0.733	0.028
• S/D ratio	5.96± 0.32	5.89± 0.55	5.66± 0.25	4.62± 0.24	0.178	0.598	0.022

P1: Difference between before and 1st wks.
P2: Difference between before and 3rd wks.
P3: Difference between before and 5th wks.

This table shows statistically significant difference between before and 5th wks. according to MCA.

Table (8): Comparison between GA (wks) according to MCA in control group.

MCA	32wks.	35wks.	37wks.	P1	P2
RI	0.80±0.02	0.75±0.02	0.71±0.02	0.311	0.037
PI	1.83±0.12	1.70±0.09	1.47±0.05	0.189	0.020
S/D ratio	5.99±0.32	5.64±0.25	4.67±0.23	0.138	0.016

P1: Difference between 32wks and 35wks.
P2: Difference between 32wks and 37wks.

This table shows statistically significant difference between 32wks and 37wks according to MCA.

Table (9): Comparison between 0, 1, 3, 5Wk after corticosteroid administration according to PT in Study group.

PT	Before	1st wks.	3rd wks.	5th wks.	P1	P2	P3
• RI	0.90± 0.12	0.91± 0.06	0.84± 0.06	0.78± 0.03	0.312	0.294	0.012
• PI	2.41± 0.08	2.41± 0.08	2.30± 0.06	2.23± 0.04	0.227	0.158	0.026
• S/D ratio	10.63 ±0.46	10.44 ±1.10	8.98± 0.90	7.73± 0.90	0.084	0.125	0.030

P1: Difference between before and 1st wks.
P2: Difference between before and 3rd wks.
P3: Difference between before and 5th wks.

This table shows statistically significant difference between before and 5th wks. according to PT.

Table (10): Comparison between GA (wks) according to PT in control.

PT	32wks.	35wks.	37wks.	P1	P2
RI	0.91±0.04	0.84±0.05	0.78±0.08	0.273	0.038
PI	2.41±0.08	2.30±0.06	2.22±0.06	0.286	0.022
S/D ratio	10.63±0.46	9.07±0.89	7.83±0.84	0.358	0.040

P1: Difference between 32wks and 35wks.
P2: Difference between 32wks and 37wks.

This table shows statistically significant difference between 32wks and 37wks according to PT.

Table (11): Comparison between study and control according to Apgar score.

Apgar score	Study	Control	*t*-test	*p*-value
1min:				
Median (QR)	6 (4)	6 (3)	0.017	0.898
Range	3-9	3-8		
5min:				
Median (QR)	7 (2)	7 (1.75)	0.417	0.520
Range	4-9	4-9		
10min:				
Median (QR)	8 (3)	8 (1)	1.459	0.271
Range	5-9	4-9		
60min:				
Median (QR)	9 (1)	9 (1)	1.743	0.197
Range	6-10	6-10		

This table shows no statistically significant difference between groups according to Apgar score.

Table (12): Comparison between study and control according to fetal weight and M. BPP.

	Study	Control	*t*-test	*p*-value
Fetal weight:				
Mean ± SD	2264.85±382.83	2357.55±428.75	6.426	0.012
Range	1390-3150	1420-3110		
M.BPP:				
Mean ± SD	7.72±0.70	7.74±0.67	0.117	0.732
Range	6-8	6-8		

This table shows statistically significant difference between groups according to fetal weight.

Table (13): Comparison between study and control according to NICU.

NICU	Study	Control	*t*-test	*p*-value
Yes	36 (49.3%)	32 (44.4%)	0.345	0.557
No	37 (50.7%)	40 (55.6%)		
Total	73 (100%)	72 (100%)		

This table shows no statistically significant difference between groups according to NICU.

Discussion

In this study Seventy study group women were subjected to standard dose of dexamethasone, followed by ultrasonographic assessment of fetal biophysical activities, fetal weight and doppler studies (pulsatility, resistive indices and systolic-diastolic ratios) for umbilical artery, fetal middle cerebral artery and fetal pulmonary trunk on baseline, and after one, three and five weeks of administration, also same ultrasonographic assessment and same doppler indices were done for seventy control group women at thirty two, thirty five and thirty seven weeks of gestation.

In a study performed by Rotmensch et al. [16], no change was observed on the biophysical profile scores after one week of steroid use. In this study, it was also observed that administrating dexamethasone for the mother did not lead to a significant decrease of biophysical profile scores in healthy preterm fetuses after one week, and no statistical significant difference of biophysical profile scores between both groups in same gestational age.

Jackson et al., [17] showed that the administration of dexamethasone decreases fetal movement and breathing and as a result the biophysical profile scores may be decreased. In their study, amniotic fluid was also decreased, but this result was not obtained in our study.

In the current study, there was not a statistically significant difference in the frequency of the findings in the pre-compared to post-dexamethasone measurements of modified biophysical profile score of the study patients, and also no statistical significant difference of biophysical profile scores between control group and study group patients and none of the biophysical profile scores was <6.

Another modality for evaluation of the fetal status is doppler velocimetry of the umbilical, fetal cerebral circulation and fetal pulmonary trunk **[18]**. In a previous study carried out by **[19]**, corticosteroids had no effect on doppler indices obtained from fetal middle cerebral artery, placental arteries or fetal pulmonary trunk. This findings has been subsequently confirmed by **[20]**, and with our study.

In a study done by Ustunyurt et al., **[15]** they noticed that the doppler indices after one week of administration were similar to those before treatment, and that also was noticed in our study group patients after one week of administration, and when these doppler values were compared with values of control group patients at the same gestational age no statistical significant difference were noticed.

Also when comparing doppler results of control and study patients obtained from umbilical, fetal middle cerebral arteries and fetal pulmonary trunk all over the period of study no statistical significant difference were noticed and and when comparing results of third week after steroid in study group with results of 35 th week in control group no statistical significant difference was noticed and thus was also noticed between results of 37 th week and results of 5 th week after steroid use.

But when comparing doppler results of before and 1 st week of steroid use with 5 th week results of study group there was a statistical significant difference was noticed, this significance was also noticed when comparing doppler results of 32 nd week with results of 37 th week in control group.

This significance may be explained by the overall increase in placental size and associated increase in number of chorionic villi throughout pregnancy which result in an expansion of the distal vascular pool of the umbilical artery, characterized with a decreasing values of vascular resistance **[10]**.

Yalti et al., **[21]** stated that umbilical velocimetry, is a test of placental function that does not always directly reflect fetal status.

Piazze et al., **[22]** and Urban et al., **[23]** added that there was no significant change observed in UA PI through administration of corticosteroids whether dexamethasone or betamethasone therapy. That is in accordance with the current study where no significant change was detected.

In the current study there was no significant changes in the mean values of the pulsatility , resistive indices, systolic-diastolic ratios or maximum velocity of flow in umbilical, fetal middle cerebral arteries and fetal pulmonary trunk pre and post corticosteroid injection in study group, and also no significant changes between control and study group values of same doppler indices for same vessels when compared in the same gestational age.

Kim et al., **[24]** observed doppler in fetuses which developed respiratory distress syndrome, in current study we follow-up pregnancy outcome of both groups by Apgar score and need to neonatal Intensive Care Unit admission, it was observed that no statistical significant difference in Apgar scores after one, five, ten and sixty minutes of delivery between study and control groups, and also no statistical significant difference between both groups in need for Neonatal Intensive Care Unit admission contrary to Kim et al., 2013.

Ustunyurt et al., **[15]** evaluated the effects of maternal corticosteroid administration on fetal MPA, significant changes in PI and RI were found 1 day and 2 days after corticosteroid administration. But after 7 days of treatment, the waveforms returned to the type of waveform found before treatment.

Thuring et al., **[25]** evaluated the effects of maternal corticosteroid administration on fetal and uteroplacental circulation in pregnancies at risk of preterm delivery, significant changes in PI and RI of both umbilical artery and fetal middle cerebral artery within 24h following corticosteroid therapy and maintained for up to 48h. But after 7 days of treatment the doppler results returned to the values before treatment.

Conclusion:

We demonstrated that dexamethasone administration did not cause changes in fetal biophysical profile scores after one week of its use.

No significant changes in the mean values of the doppler indices of umbilical, fetal middle cerebral arteries and fetal pulmonary trunk after one week of corticosteroid injection and also no significant changes between control and study

group values of same doppler indices for same vessels when compared in the same gestational age.

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تأثير تناول الأم للستيرويدات القشرية لتحسين نضج رئة الجنين وحيويته على دلالات الدوبلر

على الرغم من التقدم الذى حدث فى رعاية الحمل ورعاية الأطفال حديثى الولادة فإن متلازمة الضائقة التنفسية مازالت السبب الرئيسى لتدهور الصحة والوفاة فى الأطفال المبتسرين.

نقص نمو الجنين والولادة المبكرة هما العاملان الرئيسيان لحدوث ضيق بالتنفس عند المواليد لذلك فإن أكفأ وسيلة لتقليل حدوث متلازمة الضائقة التنفسية هو إطالة فترة الحمل حتى يكتمل وتجنب الولادة المبكرة ولكن هذا لا يمكن دائما الوصول إليه.

إن معدلات الولادة المبكرة مازالت تتراوح ما بين ٦٪-١٥٪ من كلا الولادات. مع زيادة هذا المعدل خلال السنوات الأخيرة وذلك نظرا للزيادة فى عدد حمل التوائم حيث أن للتوائم معدلات أعلى من الولادات المبكرة مقارنة بالحمل فى جنين واحد.

إستخدام مستحضرات الكورتيزون للسيدة الحامل المعرضة لحدوث ولادة مبكرة لتقليل شدة متلازمة الضائقة التنفسية لحديث الولادة هو تدخل طبى متعارف عليه، وقد كانت بداية هذه الممارسة فى عام ١٩٧٢ ميلادية عندما إستخدمها (ليجنزوهوى) وقد عرضا تقليل ملحوظ فى نسبة حدوث متلازمة الضائقة التنفسية بالأطفال المبتسرين المولودين لأمهات أعطيت الكورتيزون قبل الولادة.

وهذا العقار يعتقد أنه يحسن إنتاج المادة المقللة للتوتر السطحى داخل الحجيرات الهوائية ويصاحبه أيضا تقليل خطورة التعرض للنزيف داخل التجويف المبطن للمخ، والإلتهاب المعوى المصحوب بموت الأنسجة المعوية (إلتهاب الأمعاء الناخر)، وأيضا وفاه حديثى الولادة.

والكورتيزون للأم قبل وكذلك بعد الولادة للمولود يحسن عملية التنفس فى الأطفال المبتسرين ويسرع نضوج الرئة، ويقلل التوتر السطحى ويحسن الحالة والنشاط المضاد للأكسدة ويقلل مادة السيتوكاين التى تؤثر بالسلب على نمو الرئة على المدى الطويل وذلك فى بعض الدراسات التى أجريت على الحيوانات.

إن الديكساميثازون والبيتاميثازون نوعان من الكورتيزون يعبران المشيمة بسهولة ولذا يستخدمان لعلاج الأجنة، وقد أظهرت دراسات سابقة أن الستيرويدات لها تأثير على السلوك الجنينى الحيوى وتقلب فى معدل ضربات القلب للجنين. فى الواقع، تم وصف نتائج متضاربة بشأن آثار عقار البيتاميثازون والدكساميثازون فى نمط ضربات قلب الجنين مما أدى إلى حالات ولادة مبكرة غير مبررة.

هدف البحث:

تقييم آثار تناول الأمهات الديكساميثازون على السلوك الجنينى الحيوى وسرعة تدفق موجات الدوبلر فى حالات الحمل الطبيعى المعرضه للولادة المبكرة ونتائج الولاده.

طريقة البحث:

تم إجراء هذه الدراسة فى قسم النساء والتوليد بمستشفى الحسين الجامعى على مئة وأربعين (١٤٠) حالة ذوى حمل لجنين واحد ما بين الأسبوع ٣٢ و٣٧ مقسيمن إلى مجموعتين:

- (المجموعة الأولى): تم تجربة العقار على سبعين (٧٠) إمرأة تعانى من خطر الولادة المبكرة عن طريق حقنه فى العضل لأربع جرعات (٦مجم/١٢ ساعة/٤٨ ساعة) ثم تقييم بالأشعة فوق الصوتية للسلوك الجنينى الحيوى، ودراسات لدوبلر الشريان الجنينى السرى والدماغى الأوسط والرئوى فى اليوم الأول وبعد (١ و٣ و٥) إسبوع من أول جرعة ديكساميثازون.
- (المجموعة الثانية): سبعون إمرأة لا يعانون من خطر الولادة المبكرة ولم يتلقوا عقار الديكساميثازون، وتم أيضا تقييم السلوك الجنينى الحيوى بالأشعة فوق الصوتية ودراسات الدوبلر للشريان الجنينى السرى والدماغى الأوسط والرئوى.
- وتم مقارنة نتائج المجموعتين ومتابعة تأثير العقار على نتائج الولادة.
- ولتفادى الفرق فى التقييم تم إجراء القياسات عن طريق شخص واحد وتم تجميع النتائج فى جدوال ومناقشتها بطريقة إحصائية.

نتائج البحث:

- لعقار الديكساميثازون تأثير قوى وإن كان مؤقت على التنفس وحركات الجذع والأطراف، مما أدى إلى إنخفاض درجة السلوك الجنينى الحيوى.
- أثبتت الدراسة أن عقار الديسكاميثازون ليس له تأثير دال على التدفق الأنبساطى للدم فى الشريان السرى والدماغى الأوسط والرئوى بعد إسبوع من إستخدامه.
- معرفة هذا التأثير قد يجنب حدوث القرارات غير المبررة المتعلقة بالولادة المبكرة.