RESEARCH ARTICLE

Doppler Changes in IUGR Pregnancy Following Maternal Corticosteroids: A Prospective Observational Analysis at a Tertiary Care Hospital

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Abstract

Aims and objectives: To evaluate the impact of maternal administration of betamethsone on fetoplacental and uterine circulation in preterm pregnancies complicated with intrauterine growth restriction.

Materials and methods: Prospective observational study included 77 preterm pregnant women from 26- to 34-week period of gestation with IUGR. Color Doppler blood flow study (day 0) of fetoplacental and maternal circulation including umbilical artery middle cerebral artery (MCA), ductus venosus, maternal uterine arteries of all cases was being done. After day 0 Doppler, all received two doses of 12 mg of betamethasone intramuscularly 24 hours apart. Repeat Doppler (day 2) of the above-mentioned vessels was performed between 24 hours and 48 hours of first dose of betamethasone of all cases, and various indices were recorded. Third Doppler examination (day 4) of umbilical artery and MCA was done between 72 hours and 96 hours of first dose of betamethasone administration of all women (60 cases), and then Doppler indices values on day 4 were noted. Tabulated data were analyzed statistically by using appropriate statistical tests such as paired *t*-test, Pearson's Chi-square test, and ANOVA.

Results: A significant reduction in mean umbilical artery pulsatility index (PI) and S/D ratio was observed on day 2 following betamethasone administration (p <0.001), which was maintained till fourth day after first dose of betamethasone (p <0.05). A significant reduction in mean MCA PI and S/D ratio was obtained on day 2 following betamethasone administration (p <0.001) but the effect was transient and became insignificant by day 4. A significant decrease in mean ductus venosus PI was observed on day 2 following betamethasone administration (p <0.05). No significant change of maternal betamethasone administration in maternal uterine artery PI was observed (p >0.05).

Conclusion: Fetoplacental circulation of pregnancies with intrauterine growth restriction showed divergent response in terms of changes in various fetoplacental Doppler indices following antenatal betamethasone administration.

Clinical significance: This offers a unique model to explore the regulation of the fetoplacental vasculature. These findings are also important for fetal surveillance undertaken following corticosteroid administration to pregnant women with IUGR.

Keywords: Betamethasone, Doppler ultrasonography, IUGR-intrauterine growth restriction.

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Introduction

Pregnancy complicated with intrauterine growth restriction roughly constitutes 3–10% of all pregnancies.[1] The incidence is higher in developing countries, including India.[2] Intrauterine growth restriction leads to significantly higher morbidity and mortality in perinatal period and infancy.[1] Fetoplacental circulation abnormality has a pivotal role in causation of fetal growth restrictions. Doppler ultrasonography of fetoplacental and uteroplacental circulation plays an important role in fetal surveillance of these high-risk pregnancies.

Antenatal administration of betamethasone (corticosteroid) has been recommended before 34 weeks of gestation to ensure reduced neonatal complications in preterm newborns.[3–7] Few authors have reported a temporary reduction in fetal heart rate, fetal breathing movements, and fetal movement after the maternal administration of betamethasone.[8,9] This might prompt unwarranted delivery of premature fetus. Whether these transient effects really affect the fetus or not still remains controversial. It is prudent to ascertain whether corticosteroids (betamethasone) affect Doppler waveform patterns of fetoplacental circulation or not.

This study was designed and conducted to evaluate the impact of maternal administration of betamethasone on fetal, placental, and uterine circulation by evaluating various Doppler indices in preterm pregnancies complicated with intrauterine growth restriction.

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Aims and Objectives

- To study Doppler changes in the fetoplacental circulation after maternal betamethasone administration in preterm pregnancies with intrauterine growth restriction.
- To study Doppler changes in maternal uterine circulation following maternal betamethasone administration.

Materials and Methods

Preterm pregnant women with intrauterine growth restriction admitted in the Department of Obstetrics and Gynecology at a tertiary-level government health care hospital were recruited prospectively over a period of 1 year after approval from Institute Ethical Committee. It was mandatory to satisfy the following inclusion and exclusion criteria for recruitment into the study.

Inclusion Criteria

Women having singleton pregnancies with gestation age from 26 to 34 weeks complicated with intrauterine growth restriction.

Exclusion Criteria

Women with multiple pregnancies, women with singleton pregnancies with fetal anomalies, and women where two doses of corticosteroids could not be completed were excluded from the study.

- A written informed consent for participation was obtained from each woman included in this study at the time of enrollment.
- A detailed history, examination, and routine investigations of every woman enrolled in our study were being done. Cases with IUGR were subjected to obstetrical ultrasonography for fetal biometric parameters, estimation of gestational age, amniotic fluid index (AFI), estimated fetal weight (EFW), and for the detection of any congenital anomaly, placental localization, and placental maturity.
- Color Doppler blood flow study (day 0) of fetoplacental and maternal circulation, including umbilical artery (Umb.A), middle cerebral artery (MCA), and maternal uterine arteries (right and left) of all the cases, was being done. Doppler of fetal ductus venosus was also being done of all possible cases on day 0.
- The Doppler evaluations were done transabdominally with the fetus in a quite state, without fetal movements and without fetal breathing movements. The Doppler spectrums were recorded during voluntary apnea of mother. The Doppler examination was performed using ALT-HD1 5000 (SIEMENS) with 3- to 5-MHz transducer. Doppler waveforms from umbilical artery were recorded in the mid portion of free-floating umbilical cord.[10] The Doppler signals were recorded from the fetal MCA in its proximal third.[11] Ductus venosus was identified by following the intraabdominal portion of the umbilical vein. Color Doppler option was used to optimize the position of sample volume on the isthmus of ductus venosus.[12] Blood flow velocities were obtained from both maternal uterine arteries just above their crossing with the iliac artery.[13] Various Doppler indices (umbilical artery PI, umbilical artery S/D ratio, MCA PI value, MCA S/D ratio, and maternal uterine artery PI values) were recorded.
- Pulsatility Index = systolic peak velocity – end diastolic peak velocity/time averaged maximum velocity;
- S/D ratio = Systolic peak velocity/diastolic peak velocity.
- After the first Doppler (day 0), all women administered with two doses of 12 mg of betamethasone intramuscularly 24 hours apart. Women who could not complete the course of corticosteroid (betamethasone) were excluded from the study.
- Repeat Doppler examinations (day 2) of the above-mentioned vessels were performed between 24 hours and 48 hours (day 2) of the first dose of betamethasone of all the cases, and various indices, as noted on day 0 Doppler, were recorded.
- Wherever it could be possible, Doppler examination of ductus venosus was also done while doing day 0 and day 2 Doppler examination. Ductus venosus Doppler could be done in 62 cases out of total 77 cases, and pulsatility index (PI) values of ductus venosus were noted.
- A third Doppler examination (day 4) of umbilical artery and MCA was performed between 72 hours and 96 hours of the first dose of betamethasone administration in all the women (60 cases) who did not deliver within 72 hours of the first dose of betamethasone administration and their various Doppler indices values (PI and S/D ratio of umbilical artery and MCA, umbilical artery blood flow pattern) on day 4 were noted. Patients were managed as per protocol for IUGR.
- Tabulated data were analyzed statistically by using appropriate statistical tests, which included paired *t*-test, Pearson's chi-square test, and ANOVA. To evaluate the impact of betamethasone on fetoplacental circulation, various Doppler indices (PI and S/D ratio) values of umbilical artery, MCA, and ductus venosus obtained before (day 0) and after (day 2, day 4) betamethasone administration of all cases were compared and analyzed.
- To note any change in maternal uterine arteries' blood flow indices following betamethasone administration, PI values of right and left uterine arteries obtained before (day 0) and after (day 2) betamethasone administration of the cases were compared and analyzed.

Results

Seventy-seven women included in the study were between the age of 19 years and 34 years, and their mean age of cases was 25.10 ± 3.2 years (Fig. 1). Maximum women included in our study were between 20 years and 30 years of age, constituting around 80% of total cases. Seventy-five percentage of cases included in our

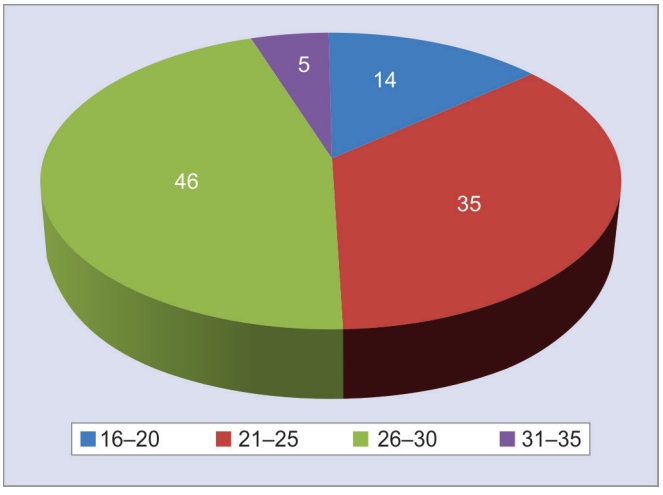

Fig. 1: Distribution of cases according to age (years)

study were between gestational age of 30 weeks and 34 weeks. Mean gestational age of our study cases was 31.58 ± 1.68 weeks (range 27.4–34 weeks) as depicted in Figure 2. Twenty-seven cases (40%) were primigravida, 16 cases (24%) were second gravida, and 16 cases (24%) were third gravida Figure 3. In our study group, 60% of multigravida women had a history of abortion, previous IUGR, and intrauterine death (IUD)/still birth.

Thirty-four (44.2%) women had pregnancy-induced hypertension. Out of these, 20 women had mild PIH and 14 women had severe PIH. Anemia was present in 26 (33.8%) women out of 77 women. No other medical illness was observed in our study group. Mean SFH lag [difference between period of gestation (weeks) and symphysio-fundal height] was 4.84 ± 1.01 cm (range 3.2–8.1 cm).

Ultrasonographic details of the study population are given in Table 1.

Out of 77 women, women who showed a decrease in PI of umbilical artery, MCA, and ductus venosus artery between 24 hours and 48 hours after the first dose of betamethasone administration are depicted in Table 2.

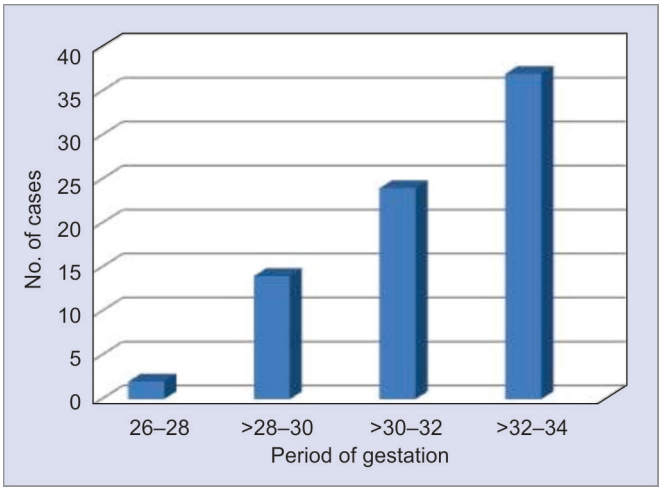

Fig. 2: Distribution of cases according to period of gestation (POG)

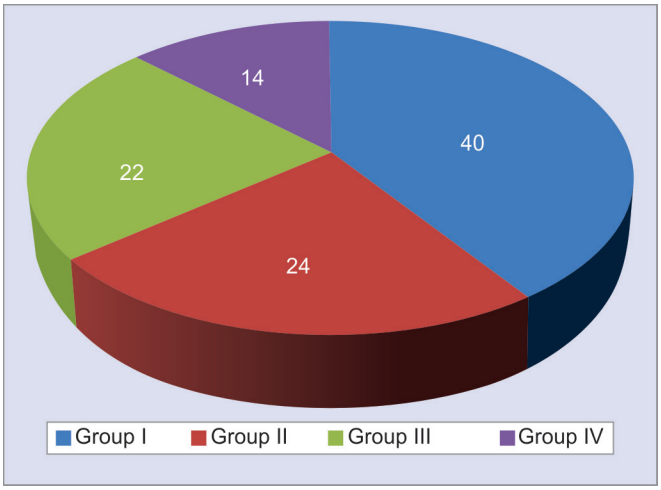

Fig. 3: Distribution of cases according to gravidity

Table 1: Ultrasonographic measurements of cases

Parameter	*Mean ± SD*	*Range*
Gestational age on admission by LMP (weeks)	32.1 ± 1.52	27.4–34
Gestational age by biparietal diameter (weeks)	31.3 ± 2.0	26.9–34.1
Gestational age by femoral length (weeks)	30.6 ± 1.7	28–33
Gestational age by abdominal circumference (weeks)	28.7 ± 0.93	27–30
Estimated fetal weight (g)	1207 ± 216	430–1769
Amniotic fluid index (cm)	6.14 ± 3.16	0–11

Table 2: Effect of betamethasone administration on fetoplacental circulation

	Change in pulsatility index following betamethasone (from day 0 to day 2) total cases n = 77		
Fetoplacental circulation	*Decrease in PI n (%)*	*No decrease in PI n (%)*	*p value*
Umbilical artery PI (%)	56 (73)	21 (27)	$p < 0.05$
Middle cerebral artery PI (%)	65 (84)	12 (16)	$p < 0.05$
Ductus venosus PI (%)	49 (79)	13 (21)	$p < 0.05$

Mean umbilical artery PI values of 77 cases on day 0 (before betamethasone) and day 2 (between 24 hours and 48 hours of first dose of betamethasone) were 1.73 ± 0.73 and 1.54 ± 0.76, respectively. Out of 77 cases, 24 cases had absent or reversed diastolic flow in the umbilical artery. Umbilical artery S/D ratio could not be calculated in these 24 cases. The remaining 53 cases with forward diastolic flow were included for umbilical artery S/D ratio analysis. Mean umbilical artery S/D ratio of 53 cases on day 0 (before betamethasone administration) and day 2 (between 24 hours and 48 hours of first dose of betamethasone) was 4.04 ± 1.07 and 3.56 ± 1.47, respectively. This decrease in umbilical artery PI and S/D ratio values following betamethasone administration (between 24 hours and 48 hours of its first dose) was statistically significant ($p < 0.001$) as depicted in Table 3.

Mean PI and S/D ratio values of MCA and ductus venosus of 77 cases on day 0 and day 2 are depicted in Table 3. Reduction in MCA PI, MCA S/D ratio, and ductus venosus PI following betamethasone administration was found to be statistically significant, as depicted in Table 3.

Out of the total 77 cases, 17 cases delivered within 72 hours of the first dose of betamethasone administration. So day 4 Doppler was done of the 60 undelivered cases only.

Mean umbilical artery PI values of undelivered 60 cases on day 0, day 2, and day 4 of 60 cases were 1.55 ± 0.61,1.33 ± 0.55, and 1.47 ± 0.63, respectively. Mean umbilical artery S/D ratio of 48 cases (as 12 cases had AEDF or REDF on day 0) on day 0, day 2, and day 4 was 3.93 ± 1.4, 3.42 ± 1.33, and 3.67 ± 1.39, respectively. On this continued observation, a significant increase in mean umbilical artery PI and mean S/D ratio from day 2 to day 4 was observed ($p < 0.001$), as depicted in Table 4.

It was observed that in spite of this rise in PI and S/D ratio from day 2 to day 4, umbilical artery mean PI value and mean S/D ratio of day 4 still remained lower than umbilical artery mean PI and

Table 3: Fetoplacental circulation Doppler indices before (day 0) and after (day 2) betamethasone administration

Doppler indices	*Day 0*	*Day 2*	*p value 0/2[c]*
Umbilical artery PI mean ± SD (range)	1.73 ± 0.73 (0.67–3.7)	1.54 ± 0.76 (0.53–3.78)	<0.001 (S)
Umbilical artery S/D mean ± SD (range) 53[a] cases	4.04 ± 1.07 (1.7–7.7)	3.56 ± 1.47 (1.73–8.2)	<0.001 (S)
Middle cerebral artery PI mean ± SD (range)	1.47 ± 0.39 (0.6–2.5)	1.26 ± 0.36 (0.6–2.2)	<0.001 (S)
Middle cerebral artery S/D mean ± SD (range)	3.71 ± 1.14 (2–7.7)	3.37 ± 1.08 (2–6.4)	<0.001 (S)
Ductus venosus PI mean ± SD (range) 62[b] cases	0.99 ± 0.29 (0.42–1.56)	0.88 ± 0.24 (0.48–1.5)	<0.05 (S)

[a]S/D ratio could be calculated in only 53 cases out of 77, 24 cases had absent and reversed end diastolic flow in umbilical artery; [b]Ductus venosus examined in 62 cases out of 77 cases. [c]0/2—*p* value of difference between day 0 and day 2 mean PI values; S, significant

Table 4: Fetoplacental Doppler indices before (day 0) and after (day 2 and day 4) betamethasone administration in undelivered 60 cases

Doppler index	*Day 0*	*Day 2*	*Day 4*	*p value 0/2[c]*	*2/4[c]*	*0/4[c]*
Umb. A PI mean ± SD	1.55 ± 0.61	1.33 ± 0.55	1.47 ± 0.62	<0.001 (S)	<0.001 (S)	<0.05 (S)
Umb. A S/D mean ± SD (48[a])	3.93 ± 1.4	3.42 ± 1.33	3.67 ± 1.39	<0.001 (S[b])	<0.001 (S)	<0.05 (S)
MCA PI mean ± SD	1.45 ± 0.38	1.29 ± 0.37	1.39 ± 0.40	<0.001 (S)	<0.001 (S)	>0.05 (NS[b])
MCA S/D mean ± SD	3.90 ± 1.14	3.56 ± 1.09	3.79 ± 1.08	<0.001 (S)	<0.001 (S)	>0.05 (NS)

[a]Forty-eight cases S/D ratio analyzed, rest 12 cases had absent end diastolic flow on day 0; [b]S, significant; NS, not significant; [c]*p* value 0/2-between day 0 and day 2 value; 2/4, between day 2 and day 4 value; 0/4, between day 0 and day 4 value

Table 5: Pulsatility index of maternal uterine arteries before (day 0) and after (day 2) betamethasone administration

Doppler indices	*Day 0*	*Day 2*	*p value 0/2*
Right uterine artery PI mean ± SD (range)	1.25 ± 0.69 (0.4–3.3)	1.20 ± 0.61 (0.4–3.3)	>0.05 (NS[a])
Left uterine artery PI mean ± SD (range)	1.25 ± 0.60 (0.44–3.2)	1.24 ± 0.69 (0.4–3.26)	>0.05 (NS)

mean S/D ratio on day 0. The difference in mean umbilical artery PI and mean S/D ratio between day 0 and day 4 was still statistically significant (p <0.05) (Table 4).

Mean MCA PI and S/D ratio of undelivered 60 cases are depicted in Table 4. After a statistically significant reduction in mean MCA PI and mean MCA S/D ratio between day 0 and day 2, a statistically significant rise in mean MCA PI and mean MCA S/D ratio between day 2 and day 4 was observed (p <0.001), making the difference in MCA PI and mean MCA S/D ratio values between day 0 and day 4 insignificant (p >0.05), as depicted in Table 4.

Mean PI values of right and left uterine arteries of all 77 cases on day 0 and day 2 are depicted in Table 5 (p >0.05).

Discussion

Antenatal administration of betamethasone is recommended before 34 weeks of gestation in pregnancies, which are at a risk of preterm delivery to decrease complications of prematurity in newborn.[3–7] Abnormal fetoplacental circulation plays an important role in causation of fetal growth restrictions. Few studies have reported a temporary reduction in fetal heart rate, fetal breathing movements, and fetal movement following maternal administration of betamethasone.[8,9] Hence, we carried out this study to evaluate the impact of betamethasone on fetoplacental and uterine circulation in preterm pregnancies complicated with IUGR.

This specific population was chosen because of the high rates of therapeutic preterm deliveries in this group and lack of much studies investigating the effects of corticosteroid administration on fetoplacental and maternal Doppler flow in such population. Umbilical artery, MCA ductus venosus, and uterine arteries were evaluated in this study. The venous circulation was studied owing to its association with the presence of acidosis at birth, and only few studies have evaluated the effect of betamethasone on this segment of the fetal circulation.[14–16]

Mean age of women included in our study was 25.36 ± 3.46 years. In this present study, we had women of gestational age ranging from 27.4 to 34 weeks, with a mean gestational age of 31.58 ± 1.68 weeks. However in the studies done by Edward et al., Robertson et al. and Smolin et al., the mean gestational age on admission was 28.5, 27.8, and 30.8 weeks, respectively.[14,15,17]

While analyzing our results, we observed that fetuses with intrauterine growth restriction showed divergent response in terms of changes in various fetoplacental Doppler indices following antenatal betamethasone administration.

A significant reduction in the mean PI and mean systolic/diastolic ratio of the umbilical artery was found between 24 hours and 48 hours (day 2) after the first dose of maternal betamethasone administration (Tables 2 to 4). A decrease in umbilical artery PI was observed in 56 cases (73%) out of total 77 cases, on day 2 following betamethasone administration. In this study, umbilical artery PI Mean ± SD values on day 0 (before), day 2, and day 4 (after betamethasone) of 60 cases were 1.73 ± 0.73, 1.54 ± 0.76, and 1.47 ± 0.62, respectively (Table 4). We found that a reduction in umbilical artery PI and S/D ratio was maintained up to day 4 (72 and 96 hours) after the first dose of betamethasone with a significant difference in values when compared with their respective day 0 values (Table 4).

Importantly, placebo or control group was not included owing to obvious ethical reasons, and it is quite possible that the changes observed might be due to other factors, such as bed rest or other medication. However, we believe that this is most unlikely because it has been previously shown that the umbilical artery Doppler changes were reproduced after the administration of second dose of steroids[18,19] and *in vitro* dexamethasone mitigates prostaglandin and KCl-induced vasoconstriction,[20] suggesting that the effect is a direct result of corticosteroid administration. While the women with hypertension were receiving antihypertensive medication, labetalol was used as a primary antihypertensive agent. Few studies have shown a vasoconstrictive effect of labetalol on fetoplacental circulation, whereas others researchers have reported no effect of labetalol on fetoplacental resistance.[20] So this decrease in mean PI value of umbilical artery cannot be because of the effect of antihypertensive medications like labetalol. Labetalol would be expected to increase rather than decrease the umbilical artery PI.[21]

As in the study by Wallace and Baker,[22] who found a significant decrease in umbilical artery PI along with return of flow in umbilical artery in all the cases with AEDF, we also observed that 73% cases (56 cases) with intrauterine growth restriction demonstrated an apparent improvement in umbilical artery Doppler flow parameters, such as reduction in umbilical artery PI and S/D ratio, which persisted up to the fourth day of the first dose of betamethasone in large number of cases as depicted in Tables 3 and 4.

Results were also comparable with the results of Thuring et al., who observed a significant decrease in umbilical PI on day 2 in 33 IUGR pregnancies and an improvement in umbilical artery flow velocity waveforms following betamethasone in cases who had AEDF or REDF before betamethasone.[16] In contrast, other researchers, like Wijnberger et al., Senat et al., and Piazze et al., have demonstrated no change in umbilical artery PI following betamethasone administration in cases of IUGR.[20,21,23] Cohlen et al. and Deren et al. failed to observe any change in umbilical artery PI in normal preterm fetuses following betamethasone administration.[24] There was no significant correlation found between the gestational age and Doppler changes in the umbilical artery following betamethasone administration. These results were similar to those of Thuring et al.[16]

A significant decrease in PI and systolic/diastolic ratio of MCA was observed on day 2 (between 24 hours and 48 hours) following the first dose of betamethasone (p <0.001) as depicted in Tables 3 and 4. These results were comparable to results of other studies done on intrauterine growth-restricted preterm fetuses by Senat et al., Baker and Wallace et al. and Muller et al., who observed a significant decrease in MCA PI following betamethasone administration.[21,22,25] Apart from these, Wijnberger et al., Chitrit et al., and Piazze have also observed a decrease in MCA PI following betamethasone administration similar to results obtained in our study.[20,23,26] In contrast to the present study, Nozaki et al. and Simchen et al. reported no effect of betamethasone on MCA PI.[27,28] In the last two studies, MCA PI values were low (in Nozaki's study MCA mean PI—1.16–1.21), suggesting a higher proportion of cases with redistribution of fetal circulation, while, in the current study, mean MCA PI before betamethasone was 1.47 ± 0.73.

In our study, mean MCA PI values showed a significant reduction between day 0 and day 2 followed by an increase between day 2 and day 4 following betamethasone administration. The values on day 4 were lower than day 0 MCA PI values but difference between day 0 and day 4 PI values of MCA PI was found to be insignificant (p >0.05). Comparable changes were observed in mean MCA S/D ratio values as depicted in Table 4. We concluded that betamethasone administration was associated with a significant but transient reduction in MCA PI and S/D ratio. Piazze et al. also obtained the same results as we did.[23] It has been postulated that fetal brain is very sensitive to overperfusion or changes in the pressure and it is efficiently protected through autoregulation of cerebral blood circulation, which might explain our findings.

A significant decrease in ductus venosus mean PI was noted following betamethasone administration as depicted in Tables 2 and 3. These results were similar to the results reported by Nozaki et al. and Thuring et al.[16,27] These findings differ from those reported by others like Edwards et al. and Mulder et al., where no effects of betamethasone were found on ductus venosus PI.[17,25] These authors evaluated a different population, including cases with only absent or reversed end diastolic flow in umbilical artery. Furthermore, that study used different corticosteroids (betamethasone in 3 cases and dexamethasone in 17 cases).

We observed that maternal uterine arteries' PIs did not show any significant influence of betamethasone therapy, as depicted in Table 5, a finding similar to other reports.[23,24]

The underlying mechanism of the alterations in the fetoplacental circulation following antenatal betamethasone administration still remains unclear. One of the plausible explanations for changes in fetoplacental circulation accompanied by reduced placental resistance is said to be because of an increased secretion of placental corticotropin-releasing hormone after exogenous administration of corticosteroids, which consecutively causes nitric oxide-mediated vasodilatation.[29–34] Another plausible reason might be related to an increase in fetal blood pressure, which might explain improved fetoplacental perfusion. Experimental studies on animal have shown that the administration of betamethasone to fetal sheep leads to an increase in fetal blood pressure.[35–38] *In vitro* study on human placentas, Clifton et al.[31] concluded that the mechanism behind the dexamethasone-induced vasodilatation might be an endothelium-independent mechanism as they did not find any involvement of endothelium-derived products like prostaglandin I_2 and nitric oxide.

Whether betamethasone-induced fetoplacental circulation changes are beneficial or detrimental for the fetus, it has still remained unclear and requires further research. It is likely that further elucidation of the clinical relevance, if any, of the Doppler responses to antenatal glucocorticoids in the fetus with intrauterine growth restriction will require a meta-analysis of several independent series. Accordingly, we would recommend other high-risk units to consider prospectively recording the responses to steroids in this cohort of pregnancies.

Conclusion

We conducted this observational study to evaluate the effect of maternal betamethasone administration on fetoplacental and uterine circulation in preterm pregnancy complicated with intrauterine growth restriction.

A significant reduction in mean umbilical artery PI and S/D ratio was observed on day 2 following betamethasone administration (p <0.001), which was maintained till fourth day after the first dose of betamethasone (p <0.05). A significant reduction in mean MCA

PI and S/D ratio was obtained on day 2 (24–48 hours) following betamethasone administration (p <0.001) but the effect was transient and became insignificant by day 4. A significant decrease in mean ductus venosus PI was also observed on day 2 following betamethasone administration (p <0.05). No significant change in maternal betamethasone administration in maternal uterine artery PI was observed (p >0.05).

Clinical Significance

Our observations are important for several reasons. First, it offers a new model to explore the regulation of the fetoplacental vasculature. Second, our findings are also important for fetal surveillance undertaken following corticosteroid administration to pregnant women with IUGR.

Another important thing to notify is that consideration should be given on getting a Doppler examination done prior to betamethasone administration in pregnancies with IUGR, because betamethasone administration changes various Doppler indices including umbilical artery and MCA PI and S/D ratio, which can be misleading for the obstetrician if first Doppler examination is done after betamethasone administration. Whether betamethasone-induced fetoplacental circulation changes are beneficial or detrimental for the fetus, it has still remained unclear and requires further research.

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