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Original Article

The Role of Intravenous Hydration and Amino Infusion in Intrauterine Growth Restriction and Oligohydramnios

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ABSTRACT

Background: - The study was carried out to evaluate the role of maternal intravenous infusions in increasing the fetal weight and liquor amount in patients of intrauterine growth restriction and Oligohydramnios. Methods: Women with sonographically confirmed IUGR and Oligohydramnios were divided into 4 groups of 100 each. Group I women were hospitalized but received no infusion and were kept on high protein diet with bed rest. Group II recieved IV infusions of normal saline, ringer lactate and 5% dextrose in ratio of 2:1:2 (5 pints) given on alternate days for 3 days in a week. Group III received amino acid drip 100 ml twice daily on alternate days for 3 days in a week. Group IV received Amino acid drip 100 ml twice daily on alternate days for 3 days in a week along with IV infusions of normal saline, ringer lactate, 5% dextrose in ratio of 2:1:2 (5 pints) given on alternate days for 3 days in a week. Clinical assessment for foetal weight was done by maintaining gravidogram and liquor was assessed clinically as well as sonographically. Ultrasonography was done after one week of therapy and was repeated after two weeks. Results: AFI increased significantly in the groups who were given intravenous fluids and aminoacids. In groups which received aminoacids weight gain of foetus was much more as compared to groups receiving no treatment or only intravenous fluids. Maximum foetal weight gain was in group 1V i.e. 40 % between 400 to 500 grams. Normal deliveries without complications were much more in group 111 and 1V which was statistically significant. Caesarean section rate was significantly reduced in group 11, 111 & 1V as compared to group 1. In group 111 & 1V foetal distress was significantly reduced, babies born had better weight & intrauterine deaths were significantly reduced. Conclusion: We conclude that IV infusion of aminoacids , 5 % dextrose and normal saline if given as a week regimen on alternate days increases short term AFI and also improves foetal weight and thus has a beneficial effect to both mother and foetus in Intrauterine Growth Restriction and Idiopathic oligohydramnios.

1. Introduction

Intrauterine growth restriction (IUGR) is a known entity since ages but Obstetricians are yet to find a concrete solution for it. Fetuses with intrauterine growth restriction are at increased risk of disease and death during antenatal, intrapartum, neonatal, pediatric, and adult life. When it is associated with oligohydramnios it further adds fuel to the fire. Developing countries report IUGR to be 8 – 10 % of all pregnancies and nearly 40 – 45 % are associated with oligohydramnios. IUGR fetus are prone to fetal distress and various neonatal complications leading to increased PMR. Nearly 50 -55 % of perinatal mortality is associated with IUGR babies constituting a major chunk in perinatal deaths . If oligohydramnios is associated with IUGR it further adds risk for the foetus during antepartum and intrapartum period [1-7].

Oligohydramnios per say effects 7 – 8 % of normal pregnancies and 12 % if postdatism is included. When it occurs in first half of pregnancy it is associated with birth defects while in second half it causes poor fetal growth, malpresentations , FHR decelerations , MAS , Fetal suicidal syndrome and increases cesarean section rates and PMR .

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Fetal growth restriction is defined as impaired growth and development of the embryo/fetus or its organs during pregnancy & is a major concern. It reduces neonatal survival, has a permanent stunting effect on postnatal growth and the efficiency of feed/forage utilization which negatively affects whole body composition and impairs long-term health and performance such as learning impairment. Uteroplacental insufficiency (UPI) remains the major cause of intrauterine growth restriction (IUGR) in developed nations Knowledge of the underlying mechanisms has important implications for the prevention of IUGR. Fetal growth within the uterus is a complex biological event influenced by genetic, epigenetic, and environmental factors, as well as maternal maturity. These factors impact on the size and functional capacity of the placenta, uteroplacental blood flows, transfer of nutrients and oxygen from mother to fetus, conceptus nutrient availability, the endocrine milieu, and metabolic pathways. Alterations in fetal nutrition and endocrine status may result in developmental adaptations that permanently change the structure, physiology, metabolism, and postnatal growth of the offspring. Impaired placental synthesis of nitric oxide (a major vasodilator and angiogenic factor) and polyamines (key regulators of DNA and protein synthesis) may provide a unified explanation for the etiology of IUGR in response to maternal undernutrition and overnutrition. There is growing evidence that maternal nutritional status can alter the epigenetic state (stable alterations of gene expression through DNA methylation and histone modifications) of the fetal genome. This may provide a molecular mechanism for the role of maternal nutrition on fetal. Thus undernutrition of various nutrients as aminoacids , carbohydrates, essential fatty acids play a great role in the development of IUGR [8].

Amino acids have multiple functions in fetoplacental development. The supply of amino acids to the fetus involves active transport across and metabolism within the trophoblast. Transport occurs through various amino acid transport systems located on both the maternal and fetal facing membranes, many of which have now been documented to be present human placentas. The capacity of the placenta to supply amino acids to the fetus develops during pregnancy through alterations in such factors as surface area and specific time-dependent transport system expression. In intrauterine growth restriction (IUGR), placental surface area and amino acid uptakes are decreased in human and experimental animal models. In an ovine model of IUGR, produced by hyperthermia-induced placental insufficiency (PI-IUGR), umbilical oxygen and essential amino acid uptake rates are significantly reduced in the most severe cases in concert with decreased fetal growth. These changes indicate that severe IUGR is likely associated with a shift in amino acid transport capacity and metabolic pathways within the fetoplacental unit. After transport across the trophoblast in normal conditions, amino acids are actively incorporated into tissue proteins or oxidized.. Potential changes may be occurring in the insulin/IGF-I signaling pathway that includes decreased production and/or activation of specific signaling proteins leading to a reduced protein synthesis in fetal tissues. Such observations in the placental insufficiency model of IUGR indicate that the combination of decreased fetoplacental amino acid uptake and disrupted insulin/IGF signaling in liver and muscle account for decreased fetal growth in IUGR.

Postnatal deficits in essential fatty acids have been associated with the neural and vascular complications of premature neonates. Total plasma concentrations of fatty acid fetal – maternal relationships in pregnancies associated with IUGR could be related to inadequate transplacental supply as well as to a fetal lack of the enzymes necessary for elaboration of these metabolically relevant conditionally essential fatty acids. These differences might have a role in determining the biochemical environment leading to the neural and vascular complications associated with IUGR.

IUGR associated with oligohydramnios pose dual threat in baby's survival. The amniotic fluid that bathes the fetus is necessary for its proper growth and development. It cushions the fetus from physical trauma, permits fetal lung growth, and provides a barrier against infection. An Inadequate volume of amniotic fluid, oligohydramnios, results in poor development of the lung tissue and can lead to fetal death. The importance of AF in fetal nutrition also is an acknowledged fact. Thus fetal malnutrition can be directly correlated to abnormalities of AF and its consequences on neonatal health.

To evaluate whether maternal intravenous infusions increase the fetal weight and liquor amount or not.

- To compare the efficacy of intravenous saline, ringer lactate and 5% dextrose solution given in ratio of 2:1:2 and amino acid infusion drip in increasing fetal weight and amniotic fluid.
- To assess whether after therapy is there any decrease in Caeserean section rate and whether Improvement in perinatal outcome occurs or not

2.Material and Methods

- Subjects were the women with IUGR and oligohydramnios confirmed clinicosonologically after 28 weeks of pregnancy .
- *Exclusion criterias*
  - ·Severe anemia
  - ·Heart disease
  - ·Premature rupture of membranes
  - ·Congenital malformation in baby
  - ·Women who did not give consent
- Women were divided into 4 groups

Group I - 100 - No infusion given , kept on high protein diet with bed rest but were hospitalized.

Group II - 100 women - IV infusions of normal saline, ringer lactate, 5% dextrose in ratio of 2:1:2 (5 pints) given on alternate days for 3 days in a week

Group III - 100 women - Amino acid drip 100 ml twice daily alternate days for 3 days in a week

Group IV – 100 women - Amino acid drip 100 ml twice daily alternate days for 3 days in a week along with IV infusions of normal saline, ringer lactate, 5% dextrose in ratio of 2:1:2 (5 pints) given on alternate days for 3 days in a week

- Clinical assessment for foetal weight was done by maintaining thegravidogram and liquor was assessed clinically as well as sonographically
- Ultrasonography was done after one week of therapy and was repeated after two weeks

3. Results

- Following were the observations

Table1. Age & Parity Socioeconomic Status

Table I

Age & Parity Socioeconomic Status

Age yrs	Parity	Socioeconomics Status																			
		Group I					Group II					Group III					Group IV				
		U	UM	M	LM	L	U	UM	M	LM	L	U	UM	M	LM	L	U	UM	M	LM	L
<19	Po	0	0	4	4	4	0	0	4	0	4	0	0	0	4	4	0	0	0	4	4
	P1	0	0	0	4	0	0	0	0	0	0	0	0	0	0	4	0	0	0	0	2
19 - <35	Po	0	6	0	4	4	8	4	4	8	8	4	8	0	8	8	4	8	0	8	4
	P1	4	0	6	6	4	4	0	0	8	8	0	8	0	8	8	0	8	10	8	6
	P2	4	8	0	4	0	0	0	2	8	10	0	0	2	8	8	0	0	2	6	4
	P3	0	0	4	8	6	4	4	0	0	4	0	0	4	4	4	0	0	4	4	8
>35	Po	0	0	0	0	4	0	0	0	0	4	0	0	0	0	0	0	0	0	4	0
	P1	0	0	0	0	8	0	0	0	0	4	0	0	0	0	4	0	0	0	4	0
	P2	0	4	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0
	P3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

U-Upper UM - Upper Middle M - Middle LM-Lower Middle L - Lower

Maximum lower middle & lower socioeconomics status.

Most between >19-35 yrs. Mean age 24 years

Maximum women were from lower middle and lower socioeconomic status ranging between 60 – 70 % in each group
Most of the women were between the age group 19 – 35 yrs age with parity one

Table 2.

Table II

Details With IUGR Oligohydramnios All Groups

IUGR	Olgohydramnios AFI in Cms	Division of Subjects			
		Group I No	Group II No	Group III No	Group IV No
Mild IUGR [<2 weeks lag]	Mild >8	12	8	4	8
	Moderate 8-5	6	8	4	8
	Severe < 5	6	4	4	4
Moderate IUGR [2-4 weeks lag]	Mild > 8	16	8	0	8
	Moderate 8-5	9	24	24	20
	Severe < 5	10	16	24	15
Severe IUGR [>4 weeks lag]	Mild <8	7	0 0	0	2
	Moderate 8-5	14	4	20	15
	Severe < 5	20	28	20	20

Although women wih moderate & severe IUGR were associated with moderate & severe oligohydramnios but there were 6%, 4%, 4% & 4% cases even with mild IUGR who had severe oligohydramnios in each group respectively.

Table 3

Table III

Post Infusion Changes In AFI And Fetal Weight

Amniotic fluid and fetal weight	AFI and fetal weight	Group I		Group II		Group III		Group IV	
		Ist USG After 7 days No	2nd USG After 15 days No	Ist USG After 7 days No	2nd USG After 15 days No	Ist USG After 7 days No	2nd USG After 15 days No	Ist USG After 7 days No	2nd USG After 15 days No
Increase in Amniotic Fluid in cms	≤2	76	88	12	48	80	88	80	88
	>2-4	16	12	80	48	20	12	20	12
	>4	8	0	8	4	0	0	0	0
Increase in fetal weight grms	≤ 100	72	80	4	20	4	8	4	8
	> 100-200	28	20	60	40	4	0	4	0
	>200-300	0	0	32	40	4	0	4	0
	>300-400	0	0	4	0	20	23	20	24
	> 400-500	0	0	0	0	48	39	48	40

The most interesting finding was that AFI increased significantly in the groups who were given intravenous fluids and aminoacids. Increase in AFI was much more after one week in USG, however it reduced further in second USG which was done after 15 days

The groups which received aminoacids weight gain of foetus was much more as compared to groups receiving no treatment or on intravenous fluids. Maximum women had foetal weight gain in group 1V i.e. 40 % between 400 to 500 grams & 34% between 300 to 400 grams.

Table 4.- Maternal Outcome IUGR Oligohydramnios

Table IV

Maternal Outcome IUGR Oligohydramnios

Mode of delivery	Group I 100 women No	Group II 100 women No	Group III 100 women No	Group IV 100 women No
Spontaneous normal delivery	24	48	68	60
Induced normal delivery	24	22	12	22
Instrumental delivery	12	16	4	10
Caesarian section	40	14	8	8

On evaluating the maternal outcome, it was observed that normal deliveries without complications were much more in group 111 and 1V which was statistically significant.

Caesarean section rate was significantly reduced in group 11, 111 & 1V as compared to group 1.

Table 5. Fetal outcome with IUGR and Oligohydramnios

Table V

Fetal outcome with IUGR and Oligohydramnios

Fetal complications	Group I 100 women No	Group II 100 women No	Group III 100 women No	Group IV 100 women No
Fetal Distress	48	24	12	8
Low birth weight	68	44	22	12
Intrauterine death	4	3	0	0
Neonatal death	3	1	1	0

On observing the foetal complications in various groups, in group 111 & 1Vfoetal distress was significantly reduced, babies born had better weight & intrauterine deaths were significantly reduced and were statistically significant.

In group 111 there was a neonatal death due to sepsis whereas 3 neonatal deaths were in group 1 due to severe birth asphyxia with extremely low birth weight. In group 11 also the neonatal death was due to low birth weight with septicaemia.

4.Discussion

FGR & oligohydramnios has always been a topic of discussion as no clear therapeutic modalities are developed for this entity. The lack of amniotic fluid allows compression of the fetal abdomen, which limits movement of its diaphragm. In addition to chest wall fixation, the lack of amniotic fluid flowing in and out of the fetal lung leads to pulmonary hypoplasia. Oligohydramnios is also associated with meconium staining of the amniotic fluid, fetal heart conduction abnormalities, umbilical cord compression, poor tolerance of labor, lower Apgar scores and fetal acidosis. In cases of intrauterine growth restriction (IUGR), the degree of oligohydramnios is often proportional to growth restriction, is frequently reflective of the extent of placental dysfunction, and is associated with a corresponding increase in the PMR. IUGR is a condition associated with placental insufficiency as a result of shallow trophoblast invasion during the early stages of gestation [1] Besides its relevance as an important cause of perinatal mortality and morbidity, a great number of studies have reported that low birth weight is a major determinant of cardiovascular disease and glucose intolerance in adult life [2-4]. Knowledge about this condition has been expanded in last decade by exploiting the possibility of obtaining fetal blood in utero by FBS. By this means, placental supply of amino acids has been shown to be significantly altered in pregnancies associated with IUGR [5,6], even when oxygenation and acid – base balance are not impaired [7].

During the third trimester intrauterine growth of the fetus is accompanied by a large deposition of fat tissue [8]. Fetal fat content increases from 0.7 to 3.1 g/ 100 g weight at 25 wk to 10.2 to 16.1 g/100 g weight at term . LC – PUFA are deposited in large amounts in the fetal brain during the period of maximum brain growth in the last trimester of pregnancy and first months of postnatal life (10-12). The incorporation of preformed AA and DHA into the developing brain is selective and more than 10 times faster than incorporation via the biosynthetic routes from LA and α LA . Because the placenta does not seem to desaturate LA and α LA, most of the ω - 3 and ω - 6 fatty acid structure acquired by the fetus in utero has to cross the placenta, and in normal pregnancies the biggest determinant of fatty acid delivery to the fetus is the concentration in the maternal circulation, which is strongly related to maternal fatty acid intake.

It is evident that pregnancies associated with IUGR and oligohydramnios are double threat to the foetus in intrauterine environment and these two are the main reasons for increased perinatal mortality. Globally various trails have been done to explore the possibility of increasing the liquor amount and foetal weight by giving intravenous fluids of variety in the form of dextrose, maltose, aminoacids etc.

A study was done to see whether maternal nutrition plays any role in maintaining the foetal environment in the Department of Obstetrics & Gynaecology at Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar. where 20 clinically and sonographically proven cases of oligohydramnios in third trimester were selected. Maternal nutrition was improved by parenteral amino acid infusion. A significant improvement was observed subsequently as the AF1 was seen to increase and less operative intervention was needed. A lesser perinatal mortality was as well seen. An important cause of IUGR and its associated complications are said to be to inadequate nutrition to mother , . So improving the maternal nutritional status during pregnancy shall improve the pregnancy outcome [1]. Various studies have tried intravenous infusion of large amounts of glucose and aminoacids to the mothers . Mothers carrying IUGR babies have been infused with 10% Fructodex and aminoacid solutions Pregnant patients with oligohydramnios also have been infused with aminoacids for improvement of foetal outcome . Improved maternal nutritional status by intravenous aminoacid infusion appears to improve the AFI. This improvement may not have been achieved with diet alone because of non-compliance and socioeconomicstatus [2,3]. According to Suzuki et al 10% Maltose can be used as an intravenous solution for increasing the amniotic fluid volume and there was significant increase in AFI with one week therapy3. American pregnancy association states clearly that maternal intravenous infusion is known to increase amniotic fluid volume [4].

According to Umber et al 2002 maternal hydration increased amniotic fluid volume (AFV) in women with oligohydramnios (mean change in amniotic fluid index 4.5 cm, 95% confidence interval 4.02 to 5.06; p-value<0.01); as well as in women with normal amniotic fluid volume (mean change in amniotic fluid index 2.7 cm, 95% confidence interval 2.23 to 3.21; p-value<0.01). The percentage increase in mean AFI was 58.6% in the oligohydramnios group, which was significantly greater ($p<0.05$) than the percentage increase of 28.4% in control group. Maternal hydration was associated with decrease in urinary specific gravity in both groups [5]. Hofmeyr et al too concluded in their study that

oligohydramnios (reduced amniotic fluid) may be responsible for malpresentation problems, umbilical cord compression, concentration of meconium in the liquor, and difficult or failed external cephalic version. Simple maternal hydration has been suggested as a way of increasing amniotic fluid volume in order to reduce some of these problems [6].

Fiank et al in their study concluded that there was a significant reduction in maternal plasma ($p < 0.05$) and urine osmolality ($p < 0.0001$) in both groups after short-term oral hydration. Hydration increased amniotic fluid volume in women with oligohydramnios (mean change in amniotic fluid index 3.2 cm, 95% confidence intervals 1.1 to 5.3; $p < 0.02$) but not in those with normal amniotic fluid volume (mean change in amniotic fluid index -2.0, 95% confidence intervals -4.1 to +0.2). The hourly fetal urine production rate, however, did not increase in either group (mean change in hourly fetal urine production rate 3.5 ml/hr, 95% confidence intervals -11.7 to +18.7 and -6.8 ml/hr, 95% confidence intervals -2.9 to -10.7, respectively). Hydration was associated with an increase in uterine artery mean velocity in the oligohydramnios group (mean change in mean velocity 16.7 cm/sec, 95% confidence intervals 8.0 to 25.3; $p < 0.006$) but not in controls (mean change in mean velocity 1.2 cm/sec, 95% confidence intervals -19.7 to +22.1). There was no change in pulsatility index or in velocity in any of the fetal vessels studied in either group. Thus short-term maternal hydration increases the amniotic fluid index in women with third-trimester oligohydramnios. Although the mechanism for this effect remains unclear, it could not be accounted for by fetal urination in this study but instead was associated with improved uteroplacental perfusion [7]. Oosterhof et al too did similar study and successful recordings were obtained in 10 of the 21 women with oligohydramnios.The hourly fetal urine production rate ncreased significantly after hypotonic rehydration (P <.02). Compared with the initial hourly fetal urine production rate after 4 hours of fluid deprivation, the hourly fetal urine production rate showed an increase of 63.2% after hypotonic rehydration, from 38.2 +/- 16.3 mL/h to 62.4 +/- 34.6

mL/h (mean +/- SD). After rehydration, the baseline fetal heart rate fell significantly, from 141 +/- 6 to 132 +/- 8 beats/min (mean +/- SD; P =.005).Thus the fetal urine production rate is augmented after acute maternal oral hypotonic rehydration after 4 hours of fluid deprivation. The current findings demonstrate that the near-term human fetus can handle such acute changes in fluid osmolality by increasing the urine production rate to maintain its fluid homeostasis. This mechanism implies that changes in maternal plasma osmolality and volume probably play an important role in determining amniotic fluid volume. Therefore the application of maternal hydration for the treatment of oligohydramnios should be further investigated [8]. Mesaki also concluded from their study that 10% Dextrose and 12 % AA increases fetal weight effectively. Cetin et al [8] also observed that aminoacids are essential for fetal growth specially valine, leucine isoleucine varieties. IV Aminoacid infusion in Oligohydramnios helps In Improving perinatal outcome Joshi, Sapre.

Similar findings were there in our study too. We found that there was significant difference in the improvement of AFI in the groups who received fluids and aminoacids as compared to the control group 1 as seen by USG after a week. However the fluid starts decreasing in the second week as seen in USG done after two weeks. An interesting finding was that foetal weight gain of more than 500 grams was seen more significantly in group 2 and 3 who were receiving aminoacids proving that it not only increases AFI but also increases foetal weight which is the need of present hour to reduce perinatal mortality indirectly. mL/h (mean +/- SD). After rehydration, the baseline fetal heart rate fell significantly, from 141 +/- 6 to 132 +/- 8 beats/min (mean +/- SD; P =.005).Thus the fetal urine production rate is augmented after acute maternal oral hypotonic rehydration after 4 hours of fluid deprivation.

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5.Conclusion

From the study we conclude that IVi nfusion of aminoacids , 5 % dextrose and normal saline if given as a week regimen on alternate days increase short term AFI and also improves foetal weight and thus has a beneficial effect to both mother and foetus in case of idiopathic oligohydramnios in developing countries. However, larger studies with controlled trial are needed to be done.These data demonstrate that IUGR oligohydramnios pregnancies are associated with significant changes in fetal – maternal fatty acid relationships. However, before recommending prophylactic measures in utero, which might perhaps reduce the risk of damages to the brain and retina, further studies are needed to elucidate the role of the placenta in Amino acids & fatty acid supply to the fetus and its relationships to fetal growth. In the meantime, IUGR infants could be considered for amino acid & other supplementations to restore a physiologic composition of their body pools.

Maternal IV Hydration combination of NS, RL & 5% D increases AF volume significantly

Amino acid drip increases foetal weight to great extent

A combined both IV Hydration on 1, 3 & 5 th day and Aminoacid drip on 2, 3 & 6th day will increase amniotic fluid as well as fetal weight

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