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Original Article

Association of Maternal Hypertension with Intrauterine Growth Retardation

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Abstract

Background: To find out the association of intrauterine growth retardation (IUGR) with maternal hypertension.

Methods: In this case control study 124 cases and 249 controls (thus giving the case to control ratio of 1:2.) were enrolled. All were born full term and delivered in obstetrics department . After taking verbal consent for the study the mothers were interviewed for the presence or absence of hypertension during pregnancy and their antenatal records checked (if available). To rule out the confounders, a study proforma was used to record maternal hypertension, maternal height, weight, BMI, age, anemia, socioeconomic status, preeclampsia, eclampsia, number of children, age of last born, gender, mode and date of delivery. Cases and controls were identified and assigned an identification number. All the study cases were seen by the pediatrician on the first day of life, weighed properly, their length and head circumference noted and plotted on centile charts in accordance with their gender to note the presence or absence of intra uterine growth restriction (IUGR). A p-value of 0.05 or less was used to see the significance of the association.

Results: Mothers of small for gestational age (SGA) babies were more than two times likely to have hypertension. Both low maternal BMI and Anemia in mother were significantly associated with IUGR in both univariate and multivariate analysis. Socioeconomic condition was also showing significant association with IUGR. Other factors like gravidity, maternal age, parity were not showing any association with IUGR.

Conclusion: Maternal anemia and low BMI are showing strong association with IUGR while maternal hypertension is not showing a strong association.

Key words: Maternal hypertension, Intrauterine growth retardation, Small for gestational age

Introduction

The term intrauterine growth retardation (IUGR) or Small for gestational age (SGA) is generally used for fetuses weighing less than the tenth percentile for gestational age or less than two standard deviations below the mean for gestational age.[1] The factors responsible for IUGR are fetal, placental, environmental and maternal. IUGR affects 23.8% of new borns around the world and 75% of these affected infants are born in Asia. In Pakistan incidence of IUGR is around 25%.[2,3]

Among the maternal factors, hypertension is one of the main factors related with IUGR.[4] Both chronic hypertension and PIH are associated with low birth weight.[5] Even if the pregnant women with chronic hypertension do not develop pre eclampsia, hypertension in the presence of proteinuria will lead to restricted fetal growth.[6] The term maternal hypertension is used when a blood pressure of 140/90mmHg or increase in systolic pressure of at least 30mmHg or an increase of at least 15mmHg diastolic pressure over the baseline first trimester readings is observed.

The hypertension in pregnancy may be seen, as preeclampsia (hypertension with proteinuria), eclampsia (preeclampsia with seizure activity) and pregnancy induced hypertension (hypertension without proteinuria).Hypertension has its role in fetal growth restriction throughout the pregnancy. Usually from conception till 24 weeks of gestation, it is the chronic hypertension responsible for IUGR. From 24 weeks onwards, it is the pregnancy induced hypertension (PIH) that leads to IUGR.[7] PIH is one factor that is related to parity also. Chronic hypertension is associated with increased fetal risk.[8]

Studying the pathophysiology of IUGR reveals that maternal disorders like preeclampsia, eclampsia, chronic reno vascular disease and chronic hypertension lead to IUGR by causing uteroplacental insufficiency. Due to decreased oxygenation of tissues, the organ growth and muscular maturation is impaired. Preeclampsia can cause placental infarction

that disturbs the provision of nutrients and leads to IUGR and poor placental flow and hence poor oxygenation of tissues cause restricted fetal growth.[9] Preeclampsia occurring in the later part of pregnancy can lead to asymmetric form of IUGR because more of the blood is directed towards the vital organs like brain and the head is comparatively spared. As a result of chronic hypoxia due to placental insufficiency polycythemia occurs in the fetus. When scanning is being done to detect IUGR hypertension in the mother is one of the corroborative signs needed to support the diagnosis.[10]

The control of hypertension by various drugs like methyldopa, labetalol, calcium channel blockers like nifedipine and ACE inhibitors has a role in the outcome of pregnancy. Regular antenatal checkups are an important part of management regarding the detection of hypertension. The treatment of extreme preeclamptic hypertension includes the use of drugs like hydralazine, labetalol, nitroglycerine or sodium nitroprusside. Magnesium sulphate is also used for the treatment or prevention of preeclampsia.[11]

Complications of IUGR and SGA are many and include higher risk of perinatal mortality and sudden infant death syndrome. At any gestational age, the morbidity and mortality are increased among term infants whose birth weights are at or below 3rd percentile for gestational age. SGA babies are prone to perinatal asphyxia and hypoglycemia in the first twenty four hours after birth.[12]

Patients and Methods

Study was conducted in Paediatric department (Neonatal intensive care unit) and in Obstetrics and Gynecology department of Holy Family Hospital Rawalpindi from June 2006 to Feb 07. The sampling technique was Convenience sampling. Total 124 cases and 249 controls (thus giving the case to control ratio of 1:2.) were enrolled in this study, that are born full term and delivered in obstetrics department of Holy Family Hospital, Rawalpindi. Admitted newborns were seen in the Neonatal Intensive Care Unit while those being kept with the mother after delivery were seen in the Obstetrics and Gynecology Department. After taking verbal consent for the study the mothers were interviewed for the presence or absence of hypertension during pregnancy and their antenatal records checked (if available). To rule out the confounders, a study proforma was used to record maternal hypertension (as defined earlier) maternal height, weight, BMI, age, anemia, socioeconomic status, preeclamsia, eclampsia, number of children, age of last born, gender, mode and date of delivery.Cases and controls were identified and assigned an identification number. All the study cases were seen by the pediatrician on the first day of life, weighed properly, their length and head circumference noted and plotted on centile charts in accordance with their gender to note the presence or absence of IUGR.

Results

Majority were in age group 20 to 30 years (Table 1). Primigravida were 49 (39.2%) in case group and 100 (40%) in control group (Table 2). Out of the cases, 52 (42.6%) were delivered by SVD and 70 (57.4%) were delivered by LSCS. The number of controls delivered by SVD were 95 (38.2%) and 154 (61.8%) of the controls were delivered by LSCS. Out of the cases, 69 (55.2%) were males and 56 (44.8%) were females. Out of the controls, 157 (62.8%) were males and 93 (37.2%) were females**.** The Univariate analysis obtained showed association of IUGR with different maternal factors. Majority (61.6 %) of the cases and 211(84.4%) of the controls did not have maternal hypertension (OR 1).48 (38.4%) of the cases and 39 (15.6%) of the controls were having maternal hypertension (OR 2.2; p-value .004). Only 2 (1.6%) of the cases and 6 (2.4%) of the controls were having teenage mothers (OR 1.01) while 123 (98.4%) of the cases and 244 (97.6%) of the controls did not have teen age mothers (OR 1;p-value .989). Seventy three (58.4%) of the cases and 204 (81.6%) of the controls were having mothers with normal weight (OR 1). 52 (41.6%) of the cases and 46(18%) of the controls were having thin mothers (OR 2.9;p-value <.001). Thirty seven (29.6%) of the cases and 203 (81.2%) of controls were not having anemia in mothers (OR 1) while 88 (70.4%) of cases and 47 (18.8%) of the controls were having anemia in mothers (OR3.2;p-value <.001). 66 (52.8%) of the cases and 219 (87.6%) of the controls had mothers with normal (25+) BMI (OR 1) while 59 (47.2%) of the cases and 31 (12.4%) of the controls had thin mothers with a BMI of < 25(OR 3.7;p-value <.001) (Table 3).

In the univariate analysis, the weight of the newborn in our dataset ranged from 1.2kg to 3.8kg.A binary dependent variable termed as intrauterine growth retardation(IUGR) was created where newborns having birth weight <2.2kg were IUGR. Univariate logistic regression analysis was done with biological characteristics of the mother which were considered as possible risk factors for IUGR. Independent variables were converted into dichomotous variables for ease of analysis and clarity. In the univariate analysis cases compared to controls were more than two times likely to have their mothers having hypertension(OR

2.2,95%CI 1.3-3.6), and almost three times likely that their mothers would be underweight (OR 2.9,95% CI 1.7-4.8),over three times likely to have their mothers anemic (OR 3.2,95% CI 2.3-4.3)Table 6. Cases compared to controls showed that the mothers of the cases were four times likely to have a low BMI (OR 3.7, 95%CI 2.2- 6.2).

Table 1: Maternal age

Maternal age	Case	Control
17to 19years	2 (1.6%)	6 (2.4%)
20 to 30 years	107(85.6%)	219(87.2%)
31 to 35 years	13 (10.4%)	23 (9.2%)
36 years and above	3 (2.4%)	3 (1.2%)

Table 2: Number of children born

Characteristic	Cases	Controls	p-value (chi-sq)
Primigravida	49	100	0.777
2-4 children	54	113	
5-12 children	22	37	
Total	125	250	

Table 3: Univariate analyses showing association of IUGR with maternal factors

Variable	Cases (Number)	Controls (Number)	Odds ratio (OR)	p-value (95% CI)
Hypertension				
No	77	221	1	0.004 (1.3-3.6)
Yes	48	39	2.2	
Mothers' weight				
Normal (1-80 kg)	73	204	1	<0.001 (1.7-4.8)
Thin (upto 60 kg)	52	46	3.2	
Anaemia				
No	37	203	1	<0.001 (2.3-4.3)
Yes	88	47	3.2	
Mothers' BMI				
Normal (25+)	66	219	1	<0.001 (2.2-6.2)
Thin (<25)	59	31	3.7	

Hypertension however has shown a weak and non significant association and therefore has been discarded. A multivariate regression analysis model was developed by including those variables whose significance level was 0.02 or below. Many interactions were tried and variables were added to the model to look for confounding factors. Hypertension was not a significant factor and therefore was removed from the model. The risk factors associated with IUGR in the final model shows that cases compared to controls were greater than five times likely to have their mothers having anemia (Adjusted OR 5.17, p-value <.001, 95%CI 2.98-8.96), and their mothers would more than twice likely to be thin having BMI <20 (Adjusted OR 2.67, p-value .001, 95%CI 1.49-4.77) (Table 4).

Table 4: Multivariate logistic regression model showing association of independent variables to IUGR

Independent variable	Adjusted OR	p-value	95% CI
Maternal anaemia			
No	1	0.001	2.98-8.96
Yes	5.17		
Mothers' BMI			
Normal	1	0.001	1.49-4.77
Thin	2.67		

Discussion

The present study (a case- control study) shows that intrauterine growth retardation is poorly related to maternal hypertension. The other factors having a significant relation to intrauterine growth retardation were maternal anemia, maternal body mass index, maternal age and family income.[12-14]Intrauterine growth retardation was seen more in newborns with gestational age in the range of 37 to 38 weeks gestation as compared to those delivered at 39 to 41 weeks of gestation.

The results of the study differ from various studies conducted in various parts of the world which showed that maternal hypertension during pregnancy was associated with intrauterine growth retardation. [4,5,10] In present study maternal hypertension was poorly associated with intrauterine growth retardation. In the univariate analysis of our study, it was found that cases compared to controls were more than two times likely to have their mothers having hypertension. The calculated p-value for this maternal factor was .004. In our study maternal hypertension was found in 38.4% of the cases and 15.6% of the controls. The results of our study are also different from the study conducted in our country by Muhammad T et-al which showed preeclampsia and eclampsia as possible causes for low birth weight of the fetus.[15] The study by Mohammad T also stated that low birth weight of the newborn was associated with both primiparity and grand multi parity. In our study, out of the cases 39.2% had

primigravida mothers, those with 2 to 4 children were 43.2% and 17.6% of the cases had mothers with 5 to 12 children while among the controls, 40% were born to primigravida mothers, 45.2% were having mothers with 2 to 4 children and 14.8% were those whose mothers had 5 to 12 children. The p-value calculated for this characteristic was 0.777. As shown by the figures mentioned, our results did not show any significant difference in the two groups (cases and controls) regarding this demographic characteristic i.e. parity of the mother .

Our study has shown results different from those shown in a study by Ayaz et al in Abbottabad, their study showed that young age of the mother, maternal hypertension, and close birth spacing was the risk factors for low birth weight of the newborn.[16] Our results are different from this study because no significant association of growth retardation of the newborns was found with young age of the mother. Regarding the maternal age, in our study the univariate logistic regression analysis revealed that out of all the cases only 1.6% had mothers in the teenage group (17 to 19 years). The results of our study are also different from a study conducted abroad which showed that maternal age at delivery was significantly associated with poor pregnancy outcomes like low birth weight.[17] In our study no such association of maternal age with low birth weight of the newborns was seen.

The results of our study correspond to the study carried out by Khan MN et al which showed that maternal body mass index (BMI) was related to the birth weight of the newborn. [18] In this study it was recommended that pre pregnancy weight gain should be attained to reduce the incidence of low birth weight in newborns. In our study 47.2% of the cases were having mothers with BMI less than 25 while out of all the controls 12.4% were having mothers with a BMI of less than 25.52.8% of the cases were having a maternal BMI of 25 and above and 87.6% of the controls were having maternal BMI in the same range .

Body mass index is calculated by dividing the weight in kilograms by surface area of the body in meter squares. If we ignore the racial factors and consider malnutrition as the main factor in the causation of decreased weight then our results also correspond to the study done by Rehman et al where maternal malnutrition, anemia was considered a factor of unique importance for intrauterine growth retardation.[19]

The univariate logistic regression analysis revealed that the mothers of the cases were almost four times likely to have a low BMI (OR 3.7, 95% CI 2.2-6.2) The final multivariate model for detecting association of independent variables to intrauterine growth retardation showed that the mothers of cases would be more than twice likely to be thin having BMI less than 20 (Adjusted OR 2.67, p-value .001, 95% CI 1.49-4.77).

Intrauterine growth retardation was a perinatal outcome related to maternal anemia. [20] Our study also corresponds to a study done in Zimbabwe where it was shown that iron supplementation during pregnancy was associated with higher birth weights of newborns independent of other pregnancy care factors, nutritional status of the mother, smoke exposure and a number of demographic and socioeconomic factors.[21,22] Our study has shown strong correlation of intrauterine growth retardation with maternal anemia. 88% of the cases had mothers with anemia while 18.8% of the controls were having mothers with anemia. The univariate logistic regression analysis revealed that the cases are over three times likely that their mothers would be anemic (OR 3.2,95% CI 2.3-4.3). The results of our study are also corresponding with those of a study conducted By Moin A et al and Imdad A et al showing a significant correlation of birth weight of the newborn, maternal body weight and hemoglobin level. [23,24] The final multivariate analysis of our results revealed that cases compared to controls were greater than five times likely to have their mothers having anemia .

The results of our study resemble the study carried out by Fikree FF et al , which was a prospective study for determinants of low birth weight of the babies. [25] Their study showed that 46% of the low birth weight babies belonged to the low socioeconomic group. In our study when the various demographic characteristics of the cases and controls were compared it was found that the difference between cases and controls was significant (p- value<.001). 88% of the cases were having a family income in the range of 3001 to 10000 while 51.6% of the controls were having family income in this range. 28% of the cases were having a family income in the range of 10001 to 20000 and 46.4% of the controls were having family income in the same range.According to the results of the study, maternal anemia, low body mass index of mother and low socioeconomic conditions play a major role in causation of growth retardation of the neonates. Maternal factors like cigarette smoking, alcohol intake and drug abuse ,playing a definite role in the incidence of low birth weight of the newborn in the west, are not significant in our set up.

Conclusions

1. The maternal factors having a significant role in causation of intrauterine growth retardation are low body mass index of mother, anemia in the mother and poor socioeconomic conditions.
2. Intrauterine growth retardation is not related to parity or young age of the mother.
3. Hypertension is not strongly associated with intrauterine growth retardation.
4. Efforts should be made to ensure proper weight gain by the mother during pregnancy. This can be done by education of the mother through sessions related to nutrition during pregnancy, proper follow up and regular antenatal checkups.
5. Anaemia in the mother should be detected early through regular antenatal check ups and iron supplementation should be done in pregnant mothers. Nutritional programmes should be arranged in all centers to provide proper food and other micro nutrients to the mothers. Correction of anemia in the teenage before marriage can improve the situation.
6. Early antenatal diagnosis of intrauterine growth retardation through ultrasonic examination is necessary to reduce the fetal morbidity and mortality.
7. High quality obstetric and pediatric units should be established by the government to provide low cost and accessible services to the low income groups.
8.

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