Research Article

INTERNATIONAL JOURNAL OF CURRENT MEDICAL AND PHARMACEUTICAL RESEARCH

ISSN: 2395-6429, Impact Factor: 4.656
Available Online at www.journalcmpr.com
Volume 4; Issue 12(A); December 2018; Page No. 3956-3958
DOI: http://dx.doi.org/10.24327/23956429.ijcmpr201812589

PLACENTAL CHANGES IN BABIES WITH INTRAUTERINE GROWTH RESTRICTION

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ARTICLE INFO

Article History:
Received 10th September, 2018
Received in revised form 2nd October, 2018
Accepted 26th November, 2018
Published online 28th December, 2018

Key words:
Intraurerine growth restriction, Placenta, Fetus

ABSTRACT

Background and objective: The human placenta plays a crucial role in normal fetal growth. Any disturbance in placental development leads to pregnancy complications like IUGR. The aim of this study was to evaluate Histopathological changes of placenta in IUGR and compare them with that of AGA babies.
Methods: Sixty placentas each from IUGR & AGA were examined. The two groups were similar in terms of gestational age, sex of babies and socioeconomic status of family. IUGR was diagnosed based on doppler sonography. The collected placenta was preserved in 10 % formaline solution. Sections were taken from membranes, maternal and fetal surface of placenta and cord. Sections cut at 5 microns thickness and prepared for hematoxyline and eosine staining. Changes in placenta were compared using chi-square test.
Result: We observed lower placental weight in IUGR compared to AGA babies. (Placental infarction, intervillous haemorrhage, intervillous fibrin deposition and syncytial knots were observed more often in IUGR placentas (p value < 0.005).
Conclusion: There was significant difference between the placentas of IUGR and AGA babies. These observations may help in finding suitable steps for improving outcomes among IUGR babies.

INTRODUCTION

Intrauterine growth restriction (IUGR) is defined as the failure of the fetus to reach normal growth potential at particular gestational age (1,2). Fetal growth impairment is seen in about 10% of pregnancies, but most of such pregnancies will have a physiologically normal fetus called as small for gestational age (SGA) babies, in whom birth weight is below the tenth percentile for a given gestational age. But IUGR is a pathological condition leading to prematurity, prenatal mortality, neurological and respiratory morbidities and immediate problems of hypothermia, hypoglycemia, pulmonary hemorrhage and encephalopathy (2-4). In adulthood, it is suspected as an important risk factor in the development of obesity, type2 diabetes and cardiovascular diseases (5).The risk of mortality rate is 12 times higher in IUGR fetuses compared to normal birth weight babies (4).IUGR has many possible causes and placental abnormality could be one of them. Placental development plays a crucial role in the normal fetal growth as oxygen and nutrients are supplied to the developing embryo through it, hence disturbances in placental development affect both mother and fetus (6). In this study, we assessed the morphology and histopathological changes of IUGR placenta in comparasion to that of AGA babies.

MATERIAL AND METHODS

The present study was conducted in our tertiary care hospital from August 2017 to July 2018 after obtaining approval from Institute Ethics Committee of our institute. Written informed consent was obtained from pregnant women. The two groups were similar interms of gestational age, sex and socio-economic status of family. Sample size was calculated using open epi software based on a earlier study by Nayereh G *et al* recording intervillous fibrin deposition of 43.5 among IUGR compared to 17.4 among AGA babies, with the 95%α error and 80% power. Sample size was calculated as 56 and we included 60 placentas in each group. Pregnant women in whom the estimated foetal weight of less than 10^{th} percentile for the gestational age with the doppler sonography showing abnormal fetal blood flow were taken as IUGR (cases). Gestational age and sex matched babies with weight between $10\text{-}90^{th}$ percentile and normal doppler were taken as controls. Exclusion criteria were maternal history of infections and fetal malformations. Placenta were collected at the time of delivery and preserved in 10% formaline solution for evaluation. Placenta was examined and sections were taken from membranes, maternal and fetal surface and unbilicalcord. Sections cut at 5 microns thickness and stained with hematoxyline and eosine. The changes observed between the groups were recorded.

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Statistical analysis

All categorical data were expressed as numbers and percentages; data was expressed as mean ± standard deviation. The significant mean difference between IUGR and AGA was determined by independent student t-test. P <0.05 was considered as significant. Chi-square test was used to compare placental changes between IUGR and AGA groups.

RESULTS

We included sixty placenta each from IUGR & AGA pregnancies. Lower placental mean weight was observed in IUGR (323.5 ± 103.6g) compared to AGA (571.6 ± 110.3g) babies (P=0.001). Histopathalogical changes observed in placents are shown in figure. Placental abnormalities like microscopic infarction, intervilloushaemorrhage and intervillous fibrin deposition were significantly more in IUGR compared to AGA babies. Avascular villi, cord vasculities, membranitis, amnion nododsum, trophoblastic fusion and stromal collagenisation were not observed in both IUGR and AGA placentas (Table 1).

Table 1 Placental findings among IUGR and AGA babies

Variable	IUGR (n=60)	AGA (n=60)	
Placental weight in g (Mean ± SD)	323.5 ± 103.6	571.6 ± 110.3	P < 0.005
No. of umbilical cord vessels	3	3	
Microscopic infarction observed	48	13	P < 0.005
Intervillous fibrin	53	15	P < 0.005
Intervilloushemorrhage	54	18	P < 0.005

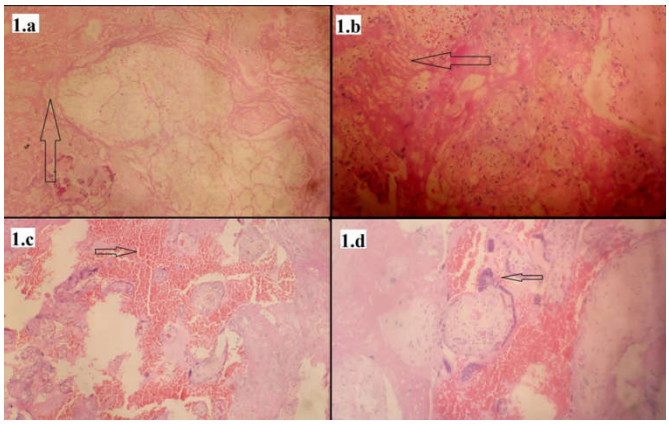

Fig 1 Histological findings in placenta from IUGR babies a.Microscopic infarction, b. Intervillous fibrin, c. Intervillous hemorrhage, d. Syncitial knots.

DISCUSSION

Placenta is crucial for normal foetal growth. It helps in suppling nutrients and oxygen to the developing embryo and also involved in removal of harmful substances and toxins from the foetus (7). Healthy dietary habits and avoiding harmful substances like alcohol, drugs and tobacco increase the chance of normal fetal growth (8). Foetal growth restriction contributes not only to neonatal morbidity and mortality but also leads to psychological problems like depression and sucide later in life (9). Placental pathology affects both mother and foetus. Disturbances in the placental implantation and function can result in IUGR (10).

Placental weight was significantly lower in IUGR compared to AGA group in our study similar to previous observations (11, 12). We recorded mean placental weight of 323.5 g and 571.5 g in IUGR and AGA groups respectively. Similar to earlier studies, placental infarction were higher in IUGR placentas compared to AGA group (13, 14). Infarction causes disturbance in normal placental blood flow and results in IUGR, preterm labour, miscarriage and intrauterine fetal death.

Intervillous fibrin deposition was observed in IUGR placenta. This indicates trapping of the villi by the fibrinoid material, which leads to the placental necrosis. Therefore increased intervillous fibrinoid deposition is a pathological feature of IUGR placenta (13, 15, 16). In our study we had also observed syncytial knots in IUGR placenta similar to that of Fard *et al* (17). Syncitial knots are indicators of compromise in fetal circulation and indicates reduced perfusion of the villi (18). A study conducted by afshari *et al* reported that chronic maternal diseases like diabetes are associated with increased formation of syncytial knots (19). Burton *et al* reported that intraplacental hypoxia as a causative factor for formation of syncitial knots which results in fetal hypoxia (20). Varli IH *et al* showed that placental pathological findings related to the fetal circulatory abnormalities are more common in IUGR than AGA group (20).

Some studies have shown placental ischemia, avascular villi, thrombosis, infarction, intervillous fibrosis in IUGR babies (11, 12). We did not find these changes in IUGR placentas.

We did not include cases with diabetes and preeclampsia and hence morphology of placenta with respect to specific etiologies cannot be commented. Multicentric larger sample size studies are required to find the relation between the placental morphology in different etiologies of IUGR.

The adverse consequences of altered fetal growth are not only limited to perinatal period but also adult onset of metabolic diseases.

Treatment and management of all pregnancy related studies not yet standardized. Extensive pathological and morphological research on placenta can be useful in identifying factors causing IUGR and consequently the treatment and preventive methods.

CONCLUSION

The placenta of IUGR was smaller than AGA group babies. Important pathological findings were placental infarction, intervillous haemorrhage, intervillous fibrin deposition and syncytial knots. Study of placenta can provide important information about pathophysiology and better treatment modality of IUGR.

Conflicts of interest: The authors declare no conflict of interest regarding the publication of this paper

Ethical clearance: This study was approved by Institute Ethics Committee of JIPMER hospital.

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How to cite this article:

Krishna Rao G *et al* (2018) 'Placental Changes in Babies with Intrauterine Growth Restriction', *International Journal of Current Medical And Pharmaceutical Research,* 04(12), pp. 3956-3958.

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